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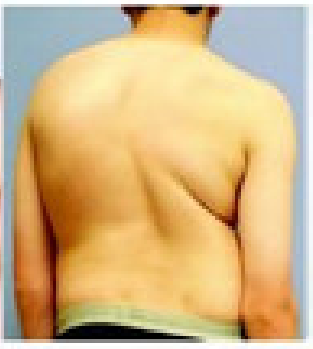
Case Discussion on Anesthesiology






Tulsi Nag

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ANESTHESIOLOGY

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Dedicated to

My beloved students

Preface

The textbooks of anesthesiology provide immense theoretical and clinical knowledge about various medical problems and surgical procedures, which help to improve the anesthetic management skill of the trainee anesthesiologists. But the vast and scattered information throughout the text is difficult to concise and memorize.

The objectives for the postgraduate trainees are to acquire the art of safe anesthesia as well as to attain success in final examination to obtain postgraduate degree for better establishment in future.

After being included in faculty, I had to conduct various departmental and university examinations (internal and external). During that period, my observation was that frequently examinees are not précised in their answers and fail to get-through. This tempted me to a desire of guiding the postgraduate trainees in preparation for practical examination. This book is a result of my much-awaited desire.

The case presentation, especially long cases, is of prime importance in practical examination. Hence, the contents of this book are in the form of question answer on different issues in patients commonly presented as long cases. I have tried my best to provide correct and current knowledge, gathered from different textbooks, about the discussed cases.

My dream will only become true if this book can help the postgraduate trainees to achieve success in their final examination.

Tulsi Nag

Acknowledgments

At the outset, I bow to The Almighty for making me able to prepare this book.

Next, I want to express my gratitude to my husband Dr Adinath Nag, without whose inspiration and cooperation it was not possible for me to compile this book.

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The list of my student supporters will be incomplete if I fail to mention the names of Dr Satyajit Maity, Dr Indranil Debnath and Dr Supriyo Bhattacharya.

Also, I must include in the list the names of Puja, Sonia, Kranti, Subhada, Sayani, Sweety, Shreya, Sohini, Saswata and Varun.

My special thanks go to Dr Kallol Chakraborty, for creation of the cover page of this book.

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Chapter

1

A Pregnant Woman Posted for Cesarean Section

Q. What are the important physiological changes that influence anesthetic techniques and how?

Increased extracellular fluid and vascular engorgement lead to upper airway compromise. Friable nature of mucous membrane during pregnancy can cause severe bleeding during insertion of nasopharyngeal airway, nasogastric tube and end orotracheal tube. Capillary engorement of the mucosa and edema of oropharynx, larynx and trachea lead to difficult intubation. Intubation of pregnant woman should be done gently with smaller size endotracheal tube (size 6.0 to 7.0). Nose should be avoided, if possible.

There is decreased functional residual capacity (FRC), which at term becomes <20% of pre-pregnancy values with closing capacity (CC) remaining unchanged. The resulting FRC/CC ratio causes faster small-airway closure when lung volume is reduced. Therefore, parturient can desaturate at a much faster rate as compared to non-pregnant women. The rapid development of hypooxia as a result of decreasing FRC, increased oxygen consumption during pregnancy and airway closure may be minimized by administration of 100% oxygen for 5 minutes before induction of anesthesia. In emergency setting, four vital capacity (VC) breaths with 100% oxygen should be sufficient.

From second trimester, aorto-caval compression by enlarged uterus becomes more important, reaching it maximum to 36–38 weeks. Cardiac output decreases during supine position due to obstruction of inferior vena cava by gravid uterus and the parturient shows signs of severe hypotension, which is known as supine hypotension syndrome. Compression of lower aorta in this position is generally not associated with material symptoms but associated with decreased placental circulation added to that due to maternal hypotension from various obstruction. Therefore, left uterine displacement of 15–20 degrees should be done by putting small pillow or wedge under right blocks during second and third trimester of pregnancy when the parturient assume supine position. Anesthetic drugs that cause vasodilatation and anesthetic techniques (e.g. neuraxial block) that cause sympthectomy, increase the impact of aortocaval compression and which is of concern for the anesthesiologists.

Enhanced progesterone production causes slower absorption of food, increased acidity due to gastric secretions and decreases lower esophageal sphincter tone. There remains the risk of pulmonary aspiration of gastric contents especially during emergency cesarean section under general anesthesia. A rapid sequence induction of anesthesia, application of cricoids pressure and intubation with a cuffed endotracheal tube are required for all pregnant women receiving general anesthesia from twelve weeks of gestation. Regional anesthetic technique can be used to avoid the disadvantages of general anesthesia. Antiaspiration prophylaxis may be used to minimize the risk of aspiration of gastric content and to reduce the effects of aspiration occurrence.

Drugs used are:

- Non-particulate antacid (0.3 M sodium citrate), 15–13 mL immediate before starting anesthesia. There is inadequate mixing with gastric contents and gastric volume.
- H_2 receptor antagonists (Ranitidine), 150 mg orally on night before surgery and repeated 3–4 hours before anesthesia. Effectively inhibits gastric acid secretion, rises intragastric pH and decreases gastric volume. Not effective in woman requiring emergency cesarean section.
- Metoclopramide, 10 mg IV before cesarean section, hastens gastric emptying and increases resting lower esophageal sphincter (LES) tone. However, this dopamine antagonist has side-effect as extrapyramidal reaction and transient neurological dysfunction.
- Altered drug responses:
  - Minimum alveolar concentration (MAC) for inhalational agents is decreased by 8–12 weeks. Gestation and is related to increase in progesterone levels.
  - The dose requirement of local anesthetic needed per dermatomal segment of epidural or spinal block, also decreases and is attributed to an increased spread of local anesthetic in the epidural and subarachnoid spaces due to epidural venous engorgement. An increased neuronal sensitivity to local anesthetics is also mediated by progesterone. Therefore, administration of anesthetic drugs should be well-titrated.
- Total plasma protein concentration declines to less than 6 g/dL at term. The abumin-globulin ratio declines due to relatively greater reduction in albumin concentration. Decrease in serum protein concentration is of clinical significance, in that the free fraction of protein bound drugs may be increased which needs proper adjustment of drug dose. The colloid oncotic pressure is also reduced due to low serum protein concentration during pregnancy which in combination with increased capillary permeability makes the parturient prone to develop pulmonary edema and is a concern for the anesthetists.
- Maternal blood volume begins to increase early in pregnancy and increase up to 45% at term, whereas RBC volume increases by only 30% leading to physiologic anemia of pregnancy. Parturient's body compensates for

this by cardiac output (CO), PaO_2 and rightward shift of oxyhemoglobin dissociation curve. Therefore, hyperventilation should be avoided during general anesthesia to prevent respiratory alkalosis which results left shift of oxyhemoglobin dissociation curve and uterine blood flow, thereby producing fetal hypoxia.

- Placental lactogen and cortisol increase the tendency towards hyperglycemia and ketosis. There is decreased ability for the pregnant patient to handle glucose load and transplacental passage of glucose may stimulate fetal secretion of insulin leading to neonatal hypoglycemia in immediate postparum period.

Q. What is supine hypotensive syndrome? How it is prevented?

When a pregnant woman is in supine position after second trimester onwards, the enlarged gravid uterus causes compression on IVC resulting in decreased venous return, thereby decreasing cardiac output. Parturient shows signs of severe hypotension, which is termed supine hypotension syndrome. As with this, there is also compression on lower aorta causing no maternal symptoms but profound decrease of uteroplacental circulation producing fetal hypoxia. Hypotension from decreased CO also leads to fetal hypoxia. As there occurs compression of both IVC and aorta, the supine hypotensive syndrome is also called aorto-caval syndrome.

The occurrence of supine hypotensive syndrome can be prevented by left uterine displacement of 15–20 degrees by putting small pillow or wedge under right buttock.

Q. How fetus is affected by supine hypotensive syndrome?

Fetus is affected by two ways:

1. Compression of IVC by gravid uterus produces venous obstruction which reduces venous return to heart, resulting in severe maternal hypotension. The hypotension reduces uteroplacental blood flow affecting fetal blood supply and oxygenation, venous obstruction due to compression of IVC results in venous stasis in uteroplacental unit affecting fetal blood supply and oxygenation leading to fetal asphyxia.
2. Compression of lower aorta by gravid uterus reduces blood flow through uterine arteries which decreases uteroplacental circulation. There occurs reduced fetal blood supply and oxygenation.

Q. Why parturients are at higher risk of regurgitation and pulmonary aspiration?

During pregnancy, enhanced progesterone production causes slower absorption of food, increases acidity of gastric secretions and lowers esophageal sphincter tone. Progesterone also relaxes smooth muscle impairing motility of intestine. The risk of regurgitation and pulmonary aspiration is a real concern in parturients, especially during emergency cesarean delivery under general anesthesia.

Q. What factors increase the risk of aspiration pneumonitis? How it can be prevented?

Upward displacement of stomach, delayed gastric emptying, relaxation of LES, decreased maternal laryngeal incompetence from analgesia and anesthesia, all increase the risk of pulmonary aspiration producing aspiration pneumonitis. Gastric juice volume >25 mL and pH <2.5 are critical factors for development of pulmonary damage. All pregnant women have large gastric juice volume (>25 mL) and low pH (<2.5) and are at increased risk of aspiration pneumonitis.

Prevention: Goal is to decreases gastric juice volume and increase gastric ph. Measures taken are:

- Preoperative fasting—reduces gastric volume.
- H_2 receptor antagonist (Ranitidine)—given at night before and morning of surgery. Decreases gastric acidity and is suitable for elective cesarean section.
- Metoclopramide, a dopamine antagonist—administered intravenously 30 minutes before anesthesia in dose of 10–20 mg. Increases gastric motility and LES tone.
- Nonparticulate antacid (0.3 M sodium citrate)—given 30 mL orally immediately before anesthesia.
- Rapid sequence, induction, cricoid pressure and intubation with cuffed endotracheal tube are the measures mandatory during general anesthesia to prevent gastric aspiration.
- Regional anesthesia is better choice for prevention of aspiration of gastric contents.

Q. What are the steps of rapid sequence induction?

The technique of rapid sequence condition is performed to gain control of airway in lest possible time after abolition of protective airway reflexes with induction of anesthesia.

Steps include:

- Preoxygenation with 100% oxygen.
- Administration of IV induction agent (Thiopentone, protocol) until abolition of eyelash reflex.
- Immediately followed by rapid acting neuromuscular blocking drug (succinylcholine)
- Direct laryngoscope and intubation reperformed quickly as soon as muscle relaxation is confirmed.
- Face mask ventilation is not undertaken for 40–90 seconds of time required for depute muscular relaxation.
- Cricoids (Sellick's maneuver) pressure is applied by assistant from the beginning of IV induction until confirmation of endotracheal tube placement and cuff inflation. Correctly applied cricoids pressure prevents regurgitation of gastric secretion.

Q. Why proxygention is mandatory in pregnant patient?

Hypoxemia may occur in time between induction of anesthesia and attainment of airway security. It is important to maximize the lung oxygen store before induction to prolong the period before the onset of hypoxemia, and is obtained with preoxygenation. Preoxygenation (commonly termed as denitrogention) should be practical when time permits. This procedure means the replacement of nitrogen volume of lung with oxygen to provide reservoir for diffusion into alveolar capillary blood after onset of apnea. Preoxygenation with 100% O_2 with tight fitting facemask for 5 minutes in spontaneously breathing patient can furnish up to 10 minutes of oxygen reserved following apnea. Less time consuming method of preoxygenation is by using four vital capacity breaths of 100% oxygen over 30 seconds.

During pregnancy, there is decrease in functional residue capacity (FRC) which, in turn, becomes <20% of prepregnancy value with closing capacity (CC) remaining unchanged. The decrease in FRC/cardio causes faster small airway closure, when lung volume is reduced. Therefore, the parturient can desaturate at much faster rate as compared to nonpregnant women. The rapid development of hypoxia during apnea, which is result of decreased FRC, increased oxygen consumption during pregnancy airway closure, is minimized by administration of 100% oxygen for 3–5 minutes before induction of anesthesia.

Changes during pregnancy make the parturient prone to develop rapid desaturation during apnea, preoxygention before induction of anesthesia is mandatory in pregnant patients to maximize the lung oxygen store so as to prolong the period before onset of hypoxemia between induction of anesthesia and attainment of airway security.

Q. How fetal well-being is monitored?

Fetal well-being is of major importance to both obstetrician and anesthesiologist. There are various biophysical and biochemical methods for evaluation of fetal well-being.

- *Fetal movement count:*
  - The parturient counts fetal movement from 9 AM till there are 10 movements, which should not be less than 10 counts in 12 hours.
  - Daily fetal movement count: The parturient counts the number of kicks for three counts, duration of each during morning, noon and evening. Total count multiplied by 4, gives daily count. Number of kicks <10 in 12 hours indicates fetal compromise.
- *Fetal cardiotocography:* Electronic fetal heart rate (FHR) monitoring provides continuous monitoring during labor on to paper chart. FHR can be monitored externally by placing the transducer on maternal abdomen (noninvisible) or internally by applying an electrode to the fetal scalp, after rupturing the membrane (invasive). For evalution of fetal well-being baseline heart rate, beat-to-beat variability, periodic patterns and uterine activity are to be considered.

The baseline FHR is measured between contractions and ranges between 120–160 beats/min in normal fetus. Acceleration of FHR in response to fetal stimulation during vaginal examination or fetal capillary blood sampling, proves that the fetus is not acidotic. Persistently elevated FHR may be associated with chronic fetal distress. Abnormally low FHR may be encountered as a late occurrence during the course of fetal hypoxia.

Baseline FHR variability of 5–25 beats/min is normal and reflects the beat-to-beat adjustment of parasympathetic and sympathetic nervous systems to variety of external and internal stimuli. Periodic FHR patterns consist of decelerations or accelerations of brief duration with uterine contractions. Early decelerations are transient with FHR not decreasing to less than 100 beats/min and are attributed to fetal head compression leading to increased vagal tone. Well-tolerated by foetus. Late decelerations begin 20–30 seconds or more after onset of uterine contraction, low point occurring well after the peak of contraction and are due to myocardial ischemia from uteroplacental insufficiency. Correction is done by improving fetal oxygenation with administration of oxygen to mother, correction of maternal hypotension or aortocaval compression or reducing uterine activity. Variable decelerations are most common periodic patterns, resulting from umbilical cord compression, onset is variable with FHR decreasing to less than 100 beats/min and >15 beats/min below baseline. Prolonged deceleration is defined as FHR decreased lasting for more than 2 minutes but less than 10 minutes.

- Fetal pulse oximetry evaluates intrapartum fetal oxygenation and is used when the electronic FHR monitoring shows nonassuring FHR tracing. Probe is inserted through the cervices and placed between the fetal cheek and uterine well. Fetal oxygen saturation between 30% and 70% is normal. Saturation consistently <30% for prolonged period (10–15 min) is suggestive of fetal acidemia. Fetal scalp blood sampling and for urgent obstetric intervention may be indicted.
- *Fetal scalp blood sampling:* Done to assess fetal acidotic status. Before labor, fetus is neither acidotic or alkalotic. During labor, various events like repeated uterine contractions, cord compression, maternal hypotension may decrease uteroplacental blood flow sufficient enough to produce fetal hypoxia and acidosis. Fetal acid-base status reflects the degree and duration of asphyxia.

  Assessment of fetal acid-base status is performed by using fetal capillary blood sampling technique. Blood is obtained from scalp or breech into a heparinized glass capillary tube of pH, PCO_2, PO_2 of base deficit, are determined immediately with an appropriate electrode system adapted to small simple size. Fetal capillary blood pH of 7.25 is the lowest limit or normal, pH <7.20 indicates fetal acidosis of urgent intervention. The pH values between 7.20 and 7.24 represent acidotic. The pH value >7.25 is reassuring of labor, progress is monitored using Apgar score, which at 1 or 2 minutes was found to be 7 or more when pH was >7.25. Fetal acidosis of maternal origin can be treated by correcting maternal acid-base imbalance.

Q. Describe obstetric pain pathways. How labor pain is produced?

Pain is the single most alarming feature of beginning of labor. Pain from the body of the uterus enters the spinal cord at T11 and T12. There are no conceptive afferents from the cervix, lower uterine segment, superior hypogastria plexuses and the aortic plexus. The no conceptive afferents then pass to the lumbar and lower thoracic sympathetic chain and terminate in the dorsal horn of the spinal cord at T10–L1 segments. Pain impulses from lower genital tract (pelvic floor, vaginal perineum) are transmitted primarily through the pudendal nerve, which is derived from the anterior division of several nerves (S2, S3, S4). Peripheral innervation of the perineum is provided by the ilioinguinal nerve and genital branch of the genitofemoral and posterior femoral cutaneous nerve. [*Reference:* Diagram of pain pathway in labor (DC Dutta, page 513)]. Additional pain pathway is involved during cesarean delivery. Skin incision involves T1 and T12 Intraperitoneal manipulation dissection involves purely localized visceral pain pathways and so requires a T4 level sensory block.

Mechanism of pain during labor: During the first stage of labor, pain results from dilatation of the cervix and contraction and detention of the body of the uterus. The pain in the first stage is referred to the dermatous supplied by the same spinal cord segments that receive impute from the body of uterus and cervix. During the early phase of first stage, the pain is limited to T11 and T12 dermatomes. As labor progresses, it spreads to T10 and L1 which overlie lower three lumbar vertebrae and upper half of the sacrum. Once the cervix is dilated, (i.e. second stage of labor), there is little or no stimulation from the structure, but the contraction and distension of the body of the uterus continue to cause pain. In addition to that there are new sources of pain like, impact of the presenting part on the pain-sensitive structures of the pelvis and distension of the outlet and perineum. Pain is referred to the sacral segments of pudendal nerve (S1, S3 and S4) and in part to the posterior cutaneous nerve of thigh (S2 and S3), genitofemoral nerve (L1)and ilioinguinal nerve (L1).

Q. What are the harmful effects of labor pain? What are the preventive measures?

- Painful uterine contraction of labor causes respiratory stimulation, increasing tidal volume, minute volume with much greater increase in alveolan ventilation. As a result of hyperventilation, there occurs material respiratory alkalosis, which results in leftward shift of oxyhemoglobin dissociation curve with increased maternal hemoglobin oxygen affinity and decreased release of oxygen.
- Hypocarbia due to hyperventilation also leads to hypoventilation between contructions, which may decrease maternal PaO_2.
- Oxygen consumption is increased during painful uterine contractions of labor.
- In addition to the above factors, hypobaric uteroplacental vasoconstriction are potential causes of fetal hypoxemia.

- Labor pain causes increased catecholamine level, mainly due to increased level of noradrenaline, which causes increased uterine contractions with decreased uterine blood flow. There occurs the creased uteroplacental circulation causing fetal hypoxemia.
- Cardiac output increases by 10–12% with each uterine contraction. As the labor progresses, cardiac output increases gradually attaining maximum value. Immediately after delivery, uterine contraction after delivery causes further increase in cardiac output. Half of this increase is caused by extrusion of blood from uterus and by increased venous return from relieved compression of IVC, (analogous to autotransfusion) and amounts to 300–500 mL, resulting in increased burden on heart.

  Fear, apprehension and anxiety further increase labor pain.

Though most of the matters and focuses do not suffer from these changes during, may be harmful for high-risk parturient (preeclampsia, cardiac disease) and those with marginal uteroplacental circulation and function.

Preventive measures include both psychological and physical preparation of childbirth along with systemic medications for effective analgesia and relief of anxiety. Regional anesthesia for labor and vaginal delivery is effective and safe. Ambulatory labor analgesia with least motor block provides advantage for parturient in labor.

Q. What are the methods of labor analgesia?

Labor analgesia options include: Psychoprophylaxis, transcutaneous electric nerve stimulation (TENS), systemic medications inhalational techniques, neuraxial block, and paracervical block are also used infrequently.

- *Psychoprophylaxis:* This method focuses on teaching methods to overcome the fear of childbirth. It also uses an education program, human support during labor, breathing techniques, relaxation techniques of voluntary muscles, a strong focus of attention and specific activities to concentrate on during contractions to block pain.
- *TENS:* TENS is thought to reduce pain by nonconceptive inhibition at presynaptic level in the dorsal horn by limiting central transmission. Placement of electrode pads over the lower back region, in the distribution of T10- L1 provides some analgesia for parturient in early labor.
- *Systemic medication:* Opioids are the most commonly used drugs for systemic medication in laboring women. All opioids have various side-effects, like respiratory depression, nausea, vomiting, euphoria and excessive sedation. All opioids have placental transfer causing respiratory depression in newborn.

  Meperidine: Most commonly used parenteral opioid analgesic during labor. Intramuscular dose—50–100 mg with peak onset of effect at 40–50 minutes. Intravenous dose is 25–50 mg to act within 5–10 minutes. The analgesic effect lasts up to 3–4 hours. Fetal exposure to meperidine is highest between 2 and 3 hours after maternal administration. It causes less respiratory depression in neonate than morphine.

Fentanyl: Fentanyl is an alternative analgesic option for parturient in whom neuraxial anesthesia is contraindicated. Its short half life makes if suitable for prolonged use in labor either as an intravenous bolus or by means of PCA delivery system. The usual dose of fentanyl for labour analgesia is 25 to50mcg. I.V. Peak effects occur after 2-3 minutes and has a duration of action 30-60 minutes. It can be administered in non-parenteral modalities like subcutaneously, orally and by patch but there have not been adequately evaluated in labouring patients.

Remifentanil: Remifentanil is a potent short acting drug that allows metabolism by nonspecific esterase throughout the blood and muscles with an extremely rapid plasma clearance and offset of action. Its half-life is 1.3 minutes and prolonged administration does not cause accumulation of this drug. Fetal exposure to the drug is minimized due to its rapid metabolism or redistribution or both. Therefore, it is an attractive alternative systemic analgesic in parturient in whom regional analgesia is contraindicated. An effective Dose of 0.4 mg with a lockout time of 1 minute for PCA with IV remifentanil or a continuous infusion of remifentanil at 0.05 mg/kg/min with a bolus dose of 25 mcg and a lockout time of 5 minutes, provides satisfactory labor analgesia.

Although primary reports are promising, further studies are needed to evaluate its safely, optimum administration technique and dosing.

Promethazine: It is most commonly used phenothiazine in obstetrics, When used with meperidine, it is given in doses of 25–50 mg to produce amnesia.

Ketamine: Ketamine has been used in subanesthetic doses (0.5–1 mg/kg or 10 mg every 2–5 min in a total of 1 mg/kg in 30 min) during labor. It can also be used in a dose of 25–50 mg to supplement an incomplete neuraxial blockade for cesarean section.

Disadvantages: Potential for hypertension and emergence reaction.

High doses (>2 mg/kg) produce psychomimetic effects and increased uterine tone which cause low Apgar scores and abnormalities in neonatal muscle tone.

Benzodiazepines: Diazepam, lorazepam and midazolam can be used as sedatives and anxiolytics. Administration during early pregnancy may lead to fetal malformation like cleft lip. The use of these drugs during labor causes no fetal malformations but are associated with problems like, sedation, hypnosis, and impaired metabolic responses to stress as these drugs are potent amnestic agents, the parturient may not remember her birthing experience.

Barbiturates: Secobarbital was once popular in obstetrics but now it is not in use because of anti-analgesic effects in mother and prolonged depressant effect in the neonate.

- *Inhaled analgesia:* It can be defined as administration of subanesthetic concentration of inhaled anesthetics to relieve pain during labor. It should not produce unconsciousness and loss of upper airway reflexes. Pain relief provided by it is not adequate for most parturient. Therefore, it may be

used as adjunct to neuraxial techniques or in parturient, where regional anesthesia is not possible. It can be administered intermittently (during contractions) or continuously. Scavenging of gases remains a concern.

Entonox: (50:50 N_2O/O_2 mixtures) may be used as a sole analgesic or an adjunct to systemic or regional techniques for relief of labor pain. The maximum analgesic effect occurs after 45–60 seconds, therefore, the parturient should use Entonox at the early onset of uterine contractions and discontinue its use after the peak of contraction. Side-effects: dizziness, nausea, dysphagia and lack of cooperation.

Desflurane (0.2%) of isoflurane (0.2% to 0.25%) may provide labor analgesia. Their effectiveness is comparable to N_2O. Most recent studies suggest sevoflurane as an effective labor analgesic. An inspired concentration of 0.8% appears to be acceptable and effective for labor analgesia. It provides superior pain relief than Entonox with more intense sedation, without any adverse effects and which is acceptable to parturient. The use of volatile agents is limited by drowsiness, unpleasant smell and high cost. Major risk is accidental overdose resulting in unconsciousness and loss of protective airway reflexes.

- *Regional analgesia:* A variety of regional techniques is used in obstetrics to provide optimum analgesia with minimum depressant effects on the mother and fetus. These techniques do not produce drug-induced depression on mother and fetus. Most commonly performed techniques are epidural, spinal and combined spinal-epidural blocks. Para cervical, pudendal and local perineal infiltration techniques are occasionally performed by obstetricians. Each technique has advantages and disadvantages.

Patient evaluation and preparation: All parturients should be assessed before placement of regional block with focus on medical and obstetric history, clinical examination and evaluation of airway. Informed consent must be obtained after explaining the procedure and potential complications of the technique by the anesthesiologist and full checkup of emergency equipment and resuscitative drugs should be performed to ensure immediate availability. Appropriate maternal and fetal monitoring should be placed before starting the procedure. Fetal well-being should be noted during preoperative assessment.

Epidural analgesia: Lumbar epidural analgesia is a safe and effective method of pain relief during labor, which may be extended to provide anesthesia for instrumental or operative delivery. Low doses of local anesthetic or combinations with opioids administered by infusion provide a continuous T10-L1 sensory block during first stage of labor. Further supplementation is required during late first stage and second stage to achieve a good block. Ultrasound is a useful aid for performing regional block, especially for epidural placement, particularly in obese parturient or in patients who have previous epidural placement.

Benefits of epidural analgesia include:

- Effective pain relief without appreciable motor block.
- Reduced maternal catecholamines.
- Rapid achievement of surgical anesthesia.
- Least depressant effects on the mother and fetus.

Contraindications of epidural analgesia include:

- Patient refusal.
- Overt maternal coagulopathy.
- Infection at the needle site.
- Maternal hemodynamic instability.

Epidural test dose: It is controversial.

Some believe it and some believe that it is unnecessary as ultradilute (0.0625%) solutions of bupivacaine is used.

Others maintain the importance of a test dose for detection of intrathecal or intravenous placement of epidural catheter, as catheter aspiration is not always predictive, especially when single orifice catheter is used. However, in laboring parturient, the heart rate variability from painful uterine contractions may confuse the interpretation of the heart rate response and intravenous epinephrine may have deleterious effects on uterine blood flow.

During continuous infusion of ultradilute solution of local anesthetic (minimum local anesthetic dose and minimum local anesthetic volume, i.e. 0.0625% bupivacaine at a rate of 5–6 mL/hr) is administered and patient remains comfortable for period without motor block, proper epidural catheter placement is ensured. If intravascular, patient would have inadequate pain relief and if subarachnoid, intense motor block would develop. Regardless of controversy, the safe practice of administering labor analgesia dictates the use of multiorifice epidural catheter, initial catheter aspiration, incremental injections and continuous monitoring. Continuous epidural infusion of dilute local anesthetic (0.0625%–0.125% bupivacaine, levobupivacaine, ropivacaine) alone or with opioids provide adequate labor analgesia.

Spinal analgesia: A single shot subarachnoid injection of local anesthetic or opioid provides effective and rapid onset of labor analgesia. It is particularly suitable for very early labor or in a distressed parturient, where epidural is difficult. Use of small-bore spinal ('micro') catheters have gained popularity in clinical practice because of convenience, fast onset of action of potential for decreased risk of postdural puncture headache.

Combined spinal-epidural analgesia: The CSE is widely used in obstetric practice to provide optimum analgesia for parturients. If offers effective, rapid-onset analgesia without motor block and with minimum risk of toxicity. This technique has the ability to prolong the duration of analgesia, through the use of epidural catheter as when required. The same catheter can be used to provide anesthesia if an operative delivery becomes necessary.

The onset of spinal analgesia is almost immediate and the duration is between 2 and 3 hours depending on the drug or drugs used. Addition of isobaric bupivacaine to the opioid produces intense sensory block with minimum motor block.

CSE technique reduces the incidence of several potential problems associated with conventional epidural technique, including incomplete (patchy) block, motor block and poor sacral spread.

Commonly used method to perform CSE technique is needle through needle which involves identification of epidural space and insertion of a long fine-bone atraumatic (pencil tip) spinal needle through the epidural needle until the tip of the spinal needle pierces the dura. Free flow of CSF confirms correct placement and the opioid alone or in combination with local anesthetic is then injected. After spinal injection, the spinal needle is withdrawn and the epidural catheter is placed 4–5 cm into the epidural space via the epidural needle.

Use of a continuous epidural infusion of dilate local anesthetic (e.g. 0.0625% to 0.1% bupivacaine) plus opioid (15 mg of fentanyl) provides sensory analgesia without motor block, permitting many parturient to ambulate during labor (walking epidural). Before ambulation, the parturient should be observed for 30 minutes to ensure maternal and fetal well-being.

Regional labor analgesia (mostly CSE) does not increase the rate of cesarean delivery. No emergency cesarean delivery for acute fetal distress is necessary in absence of obstetric indications, after regional labor analgesia.

Patient-controlled epidural analgesia (PCEA): PCEA is a safe and effective technique for providing labor analgesia. It reduces the total amount of local anesthetic used thereby, lessens unwanted effects like motor block and hypotension with excellent patient satisfaction. After establishing analgesia with spinal or epidural block, the catheter is connected to the PCEA device and the patient can then administer further boluses as required.

Paracervical and pudendal block: Paracervical block is an alternative technique for a pregnant woman who does not want or cannot receive neuraxial block. Relatively simple block to perform, provides pain relief for first stage of labor. Local anesthetic is injected submucosally into fornix of vagina, lateral to cervix. It blocks nerve transmission through the paracervical ganglion, which lies lateral and posterior to the junction of the cervix and uterus. As this block does not affect somatosensory fibers from perineum, it offers no pain relief for the second stage of labor.

Complications include:

a. Profound fetal bradycardia caused by decreased uterine blood flow
b. Systemic local anesthetic toxicity in mother
c. Postpartum neuropathy
d. Infection.

Pudendal nerves are derived from the lower sacral nerve roots (S_2—S_4) and provide sensory innervations for the lower part of vagina, vulva and perineum

with motor innervation to perineal muscles. These nerves are easily blocked by a transvaginal approach for deposition of local anesthetic behind each sacrospinal ligament. It provides satisfactory analgesia for vaginal delivery or outlet forceps delivery. It is not useful for labor analgesia.

Complications: Maternal complications include: (a) systemic local anesthetic toxicity (b) infection and (c) hematoma formation.

Lumbar sympathetic blocks: An alternative to central neuraxial block. Technically difficult to perform but is associated with fewer complications than paracervical block. It impedes transmission of pain from uterus during first stage of labor.

Q. What are the indications of epidural block in obstetric? How epidural labor analgesia is conducted?

- Labor analgesia and vaginal delivery are the primary indications of epidural block in obstetrics.
- It may be extended to provide anesthesia for cesarean section delivery and instrumental delivery.
- It can be extended to provide postoperative analgesia.
- Special indications include:
  - High-risk parturients with medical problems.
  - Pre-eclampsia—improved intervillous blood flow.
  - Previous cesarean section, anticipated difficult intubation.
  - Fetal indications include prematurity and small for date babies—excellent analgesia increases maternal oxygen saturation causing increased fetal oxygenation, improves acid-base status and causes no neonatal depression.

Steps of conduction of labor analgesia:

- A proper preanesthetic evaluation of the mother and fetal status should be done before regional block.
- A written informed consent obtained.
- Prehydration with 500 mL crystalloid done, which prevents hypotension due to sympathetic block produced by regional block.
- Monitors are attached.
- Resuscitation equipment and drugs should be ready for immediate availability.
- The patient is put in left lateral position.
- With all aseptic precautions, the patient's back is prepared with an antiseptic solution, the area is draped with sterile sheets and the desired space is located.
- The patient's back should be perpendicular to the table at the edge of the table.
- A wheal of local anesthetic is raised with a hypodermic needle at the desired interspace. A small amount of local anesthetic is injected into the subcutaneous tissue and interspinous region.

- Epidural needle is gradually inserted through the skin wheal until the needle is in the epidural space.
- Once it is certain that the needle is in the epidural space, the calculated dose of drugs is injected in incremental dose till satisfactory analgesia is achieved.
- The epidural needle is positioned in such a way that the bevel faces cephalad. The epidural catheter is inserted into the hub of the needle and pushed in. When the catheter has been inserted to desired length (usually 15 cm mark at the tub of the needle), the needle is gradually pulled out carefully without pulling the catheter.
- Once the desired length of catheter is introduced (usually 2–3 cm and not more than 5 cm), the connector assembly is fixed at the free-tip of the catheter. Sterile dressing is placed on the skin from where the catheter emerges.
- To obtain continuous analgesia top up doses are given as necessary.
- Use of ultradilute solution of local anesthetic with small dose of opioid provides effective analgesia without motor block.
- After the procedure, the parturient is positioned supine with left lateral tilt. Pulse, BP, respiration and level of sensory block is monitored. Fetal well-being is assessed.
- Effective analgesia for the first stage of labor is achieved by blocking T10–L1 dermatomes. For second stage of labor and delivery, block should be extended to include S_2–S_4 segments.
- Initial block is obtained with combination of 0.0625%–0.125% bupivacaine (10 mL) with 20–50 mcg fentanyl. Subsequent analgesia obtained with continuous infusion 8–15 mL/hr.
- Blood pressure should be checked every 1–2 min for first 10 min and then every 5–15 min during infusion + until block wears off.
- Conditions essential for initiation of epidural analgesia:
  - The parturient should be in active stage of labor with pain during uterine contractions.
  - Uterine contractions should be regular, of good intensity and occurring at intervals of 3 minutes and lasting for 30–40 seconds.
  - Presenting part should be engaged.
  - Cervix should be dilated 3–4 cm in multiparas and 4–5 cm in primiparas.

Q. When there is increased central spread of local anesthetic during regional block in pregnant patients?

When local anesthetic is injected for regional block, the spread of sensory block is more in term parturients. The dose of local anesthetic required to obtain any level of block is reduced to one-third in last trimester of pregnancy. Factors responsible for this increased sensitivity may be mechanical, hormonal and biochemical.

- *Mechanical factors:* The gravid uterus causing IVC compression may cause increased cephalad spread of local anesthetic solution due to distension of epidural veins. In addition, elevated intra-abdominal pressure, increased

intravascular volume and exaggerated lumbar lordosis may contribute to increased spread of local anesthetics. The increased blood flow to the highly vascular pia mater may result in increased penetration of local anesthetics to the nerve roots, resulting in more rapid onset of block.

- *Hormonal factors:* Studies reported increased spread of epidural anesthetic during first trimester of pregnancy despite minimum IVC compression by gravid uterus. This finding is contributable to hormonal changes which increase the sensitivity to local anesthetics. Hormones, principally progesterone during pregnancy increases neuronal sensitivity to local anesthetics.
- *Biochemical factors:* Hyperventilation associated with pregnancy results in alkalosis which the body tries to compensate leading lowered bicarbonate levels. This decreases buffering capacity allowing the local anesthetics to remain as salt for a longer time in epidural space. This decreased buffering capacity during pregnancy may account for both, the increased spread of local anesthetics and increased time needed to achieve analgesia.

Q. What are the disadvantages of bupivacaine? What are the complications of epidural analgesia?

The only disadvantage of bupivacaine is that accidental intravenous injection results in cardiac arrhythmias as VT + fibrillation convulsions. Bupivacaine is more cardiotoxic, high lipid solubility allows bupivacaine to enter the cell more easily. It blocks the sodium channels of nerve and heart which slows or stops conduction. Bupivacaine enters the sodium channels quickly but leaves slowly. This makes the resuscitation of patients in cardiac arrest from bupivacaine toxicity, difficult. High protein binding, particularly at low pH further contributes to this feature. CNS also plays a part to induce cardiac arrhythmias with bupivacaine. Factors lowering the cardiotoxic threshold of bupivacaine are: Pregnancy hyperkalemia, hypoxia, acidosis.

Complications of epidural analgesia:

- *Hypotension:* It is the most common complication with epidural analgesia in parturients due to decreased venous return to the heart resulted from compression of IVC by gravid uterus and peripartum blood loss in addition to sympathetic block. Moderate decrease in BP is tolerated by pregnant women with minimum effect on fetus. More severe hypotension (<20% decrease of BP or less than 100 mm Hg) results in significant decrease in uteroplacental blood flow which jeopardize the fetus. Patients with pregnancy-induced hypertension (PIH) are at particular risk of becoming hypotensive due to reduced intravascular blood volume. Treated with IV boluses of crystalloid solution and small doses of vasopressors.
- *Massive misplaced injection:*
  - ***Massive subdural injection:*** Large volume injection (Potential space between dura and arachnoid mater) intended for epidural use, produce unusual spread of analgesia characterized by slow onset of extensive but asymmetric sensory and motor block.

- ***Massive subarachnoid injection:*** Extensive cephaloid spread of local anesthetic may occur to block entire spinal cord or even brainstem leading to profound hypotension and bradycardia due to complete sympathetic block. Respiratory arrest may occur from paralysis of respiratory muscles or dysfunction of brainstem respiratory centers. Management includes left uterine displacement and administration of fluids, vasopressors and atropine to achieve hemodynamic stability. Rapid control of airway with endotracheal intubation (avoiding aspiration) and controlled ventilation with 100% oxygen is necessary to ensure oxygenation. If CVS and respiratory events are managed appropriately, total spinal block resolves without sequelae.

- *Massive intravascular injection:* It can result in convulsions and cardiac arrest. Due to compression of IVC by gravid uterus, pressure and flow in extradural and azygous venous system become higher.
- *Post-dural puncture headache:* Unintentional dural puncture by 16 or 18 gauge epidural needle are at risk of post-dural puncture headache (PDPH). The headache is mild or absent when the patient lies supine and head elevation leads to severe fronto-occipital headache. Headache results from loss of CSF from meningeal needle, whole leading to decreased buoyant support for the brain. In upright position, the brain sags putting traction on pain sensitive structures, causing pain. PDPH is self-limiting and often disappears within a week.

  Treatment includes:

  - Bed rest and hydration:
  - Analgesics (acetaminophen, codeine) provide some relief until headache resolves.
  - Caffeine: (Theophylline sumatriptan) has been used in managing PDPH. The cerebral vasoconstrictor activity of caffeine causes relief of symptoms.
  - Epidural blood patch: Severe headache that does not respond to conservative measures is best treated with autologous blood patch. With aseptic technique, 10–15 mL of patient's blood is drawn and injected into the epidural space close to the site of dural puncture. The procedure may be repeated as necessary. It presents CSF leak by forming a clot over the meningeal hole and effectively relieves symptoms from 1 to 24 hours.
- *Systemic local anesthetic toxicity:* CNS and cardiovascular toxicity may occur following accidental intravascular injection of local anesthetics during epidural anesthesia life-threatening convulsions and cardiovascular collapse may occur. Signs of CNS toxicity occur prior to CV events, seizure activity is treated with IV thiopentone (50–100 mg) midazolam (2–5 mg) or IV propofol (1 mg/kg). Material airway should be secured and oxygenation maintained. Bradycardia treated with atropine and hypotension treated with IV fluids and vasopressors. Profound CV depression and malignant dysrrhythmias should be prompty treated. Oxygenation and ventilation

immediately instituted with CPR if needed. Resuscitation is more difficult in pregnant patient and if efforts are not quickly successful, cesarean delivery may be required to relieve aortocaral compression and ensure effective cardiac massage. Administration of IV lipid solution is a novel and promising treatment for cardiac toxicity, by removing bupivacaine formed site of action. Ventricular dysrhythmias are treated with multiple electrical cardioversion, epinephrine, vasopressin and amiodarone. It is essential to avoid systemic local anesthetic toxicity adherence to recommended dosage, method of direct placement of needle and catheters and fractional administration of total amount.

- *Spinal hematoma:* Rare but potentially devastating complication of epidural anesthesia. Presents with numbness and lower extremity weakness. Early detection is essential because delay of more than 8 hours in decompressing the spine reduces chance of good recovery. Coagulation defects are principal risk factors for epidural hematoma. Patients receiving NSAIDs with antiplatelet effect or subcutaneous unfractionated heparin for DVT prophylaxis are not at increased risk of spinal hematoma. But patients taking antiplatelet drugs like clopidogrel should not be taken for epidural block. These drugs are to be discontinued 1 week before performing epidural block. $GPII_a/II_6$ have shorter duration of action and discontinued 24–48 hours before block. Patients receiving fractionated LMWH like enoxaparin are at increased risk of spinal hematoma. Patients receiving prophylactic dose should hold the drug for 10–12 hours before block and those receiving treatment doses should hold it for 24 hours before block. If an epidural catheter is already in place, it should not be given at least 2 hours after removal of the catheter. Patients who are fully anticoagulated (elevated PT or PPT) or who are receiving thrombolytic or fibrinolytic therapy are at increased risk of spinal hematoma and epidural block should be avoided.

Q. What is the objective of test dose during epidural block?

Once it is certain that needle is in epidural space, a small amount of local anesthetic (Bupivacaine 5 mg) and epinephrine 15 μg are administered as a test dose. The objective of test dose is to prevent inadvertent subarachnoid or intravascular injection of the total dose for epidural block, both of which have serious consequences. A test dose of local anesthetic produces a readily identifiable sensory and motor block if injected intrathecally. Addition of epinephrine with careful hemodynamic monitoring shows a transient increase in heart rate and blood pressure, following intravascular injection. However, in laboring patients, maternal heart rate variability from the pain of uterine contractions may confuse interpretation of hemodynamic response and intravenous epinephrine may reduce uteroplacental perfusion. Rapid injection of 1 mL of air into the epidural catheter with simultaneous precordial Doppler monitoring is a reliable indicator of intravascular catheter placement.

Regardless of technique used, the safe practice of administering labor epidural analgesia dictates initial catheter aspiration, administration of local anesthetic in incremental fractional doses and continuous monitoring for evidence of inadvertent subarachnoid or intravascular injections.

Q. What are the vasopressors used in obstetric patients?

Vasopressors are more reliable means of treating hypotension due to central neuraxial block. Drugs with both α and β action are superior to pure α-agonist for this purpose. Vasopressors used in parturients for treatment of hypotension are ephedrine and phenylephrine.

Ephedrine: The mixed α and β adrenergic effect of ephedrine causes an increase in BP by increasing cardiac output and systemic vascular resistance. It has positive inotropic effect. It increases BP with better preservation of uteroplacental blood flow. It has no detrimental effect on uterine blood flow and widely used as a vasopressor in hypotensive parturients. Due to β_1 adrenergic effects, it is helpful in treating moderate hypotension associated with bradycardia.

Phenylephrine: Vasopressor with direct α-adrenergic receptor activity which is a concern about the potential adverse effect on uterine blood flow. It can be used safely in healthy parturients and is as efficacious as ephedrine in maintaining maternal BP and fetal umbilical pH values. Studies comparing ephedrine and phenylephrine showed no difference in efficacy and also found that women given phenylephrine had neonates with high umbilical cord blood pH than those given ephedrine. Risk of true fetal acidosis (pH <7.20) was similar. As acidotic changes in umbilical arterial pH are sensitive indicator of decreased uteroplacental perfusion, this finding was indirect evidence that uterine blood flow may be better with phenylephrine. There are two reasons for better fetal acid-base status with phenylephrine than with ephedrine.

- Ephedrine readily crosses placenta and increases FHR. It may also stimulate fetal metabolism by direct β-adrenergic effect by stimulating release of endogenous fetal catecholamines, resulting in fetal acidosis.
- Sympathectomy from regional anesthesia shunts blood into mesenteric bed α-agonist like phenylephrine have a selective vasoconstrictive effect on mesenteric bed than on uteroplacental vasculature. This increase in cardiac preload leads to increased cardiac output with improved uteroplacental perfusion. Phenylephrine is commonly used to treat hypotension during regional anesthesia when vasoconstriction is needed. It also increases coronary perfusion pressure without chronotropic effect (useful in CAD and aortic stenosis). It has rapid onset of action with short duration (5–10 min) after IV administration. Bolus dose 40–100 μg, IV infusion 10–20 μg/min. Recommendation for hemodynamic treatment during neuraxial block: Blood pressure is treated when the decrease is 20%–30% below baseline or systolic BP falls below 90 mm Hg. Bradycardia is treated when heart rate falls below 60–50 beats/min.

Q. What is the anesthetic technique of choice for cesarean section? Why?

Neuraxial anesthetic techniques (spinal, epidural, CSE) have several advantages, including:

- A decreased risk of failed intubation and aspiration of gastric contents
- Avoidance of depressant agents
- Mother remains awake and enjoys the birth of her child
- Early bonding of mother with child
- Reduced blood loss during cesarean delivery.

For all the above advantages, the use of neuraxial anesthesia has dramatically increased and became the technique of choice for cesarean delivery.

Although epidural, spinal, continuous spinal and CSE techniques can all be used, most straightforward cesarean sections are now performed with single shot spinal anesthesia.

Spinal anesthesia: Spinal anesthesia is easy to perform. Needle placement is assured from free flow of CSF. It has very rapid onset and provides dense neural block. As small doses of drugs are used, there is little risk of local anesthetic toxicity and minimum transfer of drug to the fetus to cause toxicity. Disadvantages include limited duration of anesthesia and higher incidence of hypotension. A 0.5% solution of hyperbaric bupivacaine is most commonly used local anesthetic for spinal anesthesia in cesarean section delivery. Despite achieving an adequate block (T4), some parturient under spinal anesthesia experience some degree of visceral discomfort during cesarean section, particularly when the obstetrician exteriorises the uterus. The quality of spinal anesthesia be improved by addition of morphine and fentanyl to minimize the visceral discomfort. Continuous spinal anesthesia is advantageous in high-risk parturients with medical diseases and obesity and to reduce the risk of PDPH. The newer technique uses a 32G microcatheter inserted through a 26G spinal needle. As the catheter is placed, smaller dose can be given in an incremental fashion.

CSE technique: Provides rapid onset of dense surgical anesthesia and allows prolongation of block with epidural catheter. Problem with this technique is the possibility of failed epidural catheter after spinal injection. The advantage of this technique is that as block can be supplemented at any time using epidural catheter, smaller dose of spinal anesthetics is used which results in decreased incidence of high block and hypertension.

Epidural anesthesia

This technique is often chosen for:

1. Potentially prolonged cesarean delivery
2. In high-risk parturients, epidural catheter is placed earlier to be available for emergency cesarean section
3. Indwelling epidural catheter for labor analgesia can be used to provide anesthesis for cesarean section.

Because large volume of potentially toxic local anesthetics are used via epidural catheter for cesarean delivery, several measures should be taken to reduce the risk of local anesthetic toxicity.

- Catheter should be aspirated before use and appropriate test dose administered.
- The anesthetics should be administered in fractional doses.
- Newer amide local anesthetics (levobupivacaine and ropivacaine) should preferably be used.

Intraoperative conditions under epidural anesthesia may be improved by adding fentanyl (50–100 μg). Clonidine has also been used as an additive to epidural anesthesia but has side-effects like sedation, bradycardia and hypotension.

Q. What are the indications of general anesthesia for cesarean section? Outline the technique of general anesthesia for cesarean delivery.

Indications of general anesthesia for cesarean delivery:

- Maternal hemorrhage
- Life-threatening fetal compromise
- Maternal coagulopathies
- Acute maternal hypovolemia
- Inadequate regional anesthesia
- Infection at the site of needle injection for regional anesthesia.

Outline of general anesthesia:

- Administration of ranitidine 150 mg orally on the night before surgery and repeated 3–4 hours before induction of anesthesia, effectively inhibits gastric acid secretions, decreases gastric volume and raises gastric pH. Intravenous ranitidine 50 mg 30 min before induction along with 30 mL sodium citrate 10–15 min prior to induction reduces the risk of aspiration.
- Administration of 30 mL of 0.3M sodium citrate (a nonparticulate antacid) 10–15 min prior to induction neutralizes gastric acid and gastric content, if aspirated, causes least pulmonary damage.
- Administration of metoclopramide 10 mg IV or ondansetron 8 mg IV 15 min before induction increases gastric motility and hastens gastric emptying.
- Left lateral uterine displacement is maintained.
- Routine monitors including noninvasive blood pressure (NIBP), ECG, pulse oximeter, nerve stimulators, are applied.
- Suction apparatus checked and equipment to correct failed intubation are kept readily available. IV cannula inserted.
- Preoxygenation is done with 100% O_2 via tight fitting mask for 3–5 minutes or using 4 vital capacity breaths.
- After the drapes are applied and the surgeon is ready, rapid sequence induction with thiopentone 4 mg/kg and succinylcholine 1.5 mg/kg is done. Cricoids pressure is applied immediately after the patient is

unconscious and continued until correct position of endotracheal tube is verified with capnometer and the cuff is inflated.

- Intubation is done with smaller size (6 or 7 mm) endotracheal tube gently to prevent trauma.
- Controlled ventilation is done using Oxygen:Nitrous oxide 50:50% low concentration of volatile anesthetic (halothane 0.5%, isoflurane 0.6–0.75%. Hyperventilation is avoided as hypocapnea leads to uterine artery vasoconstriction with decreased intraplacental blood flow which jeopardises fetal well-being.
- After delivery, nitrous oxide is increased to 70%. An opioid (Fentanyl 2 μg/kg), midazolam 0.2 mg/kg or propofol 2 mg/kg are added with or without continuation of 0.5 MAC volatile agents.
- Muscle relaxant is administered when necessary (small dose of vecuronium or atracurium).
- Oxytocin is added to the intravenous fluid.
- Neuromuscular block is reversed at the completion of surgery.
- Extubation is done when anesthesia is adequately reversed with the patient awake and comfortable following verbal commands.
- The parturient is cared in PACU, with supplemental O_2, analgesic and monitoring of vitals.

Q. What are oxytocics? What are the commonly used oxytocics? What precautions are to be taken during administration of oxytocin and why?

Oxytocics are drugs having the power to excite contraction of uterine muscles and are commonly used in obstetrics to induce or accelerate labour or augment uterine contractility following delivery for prevention and treatment of postpartum hemorrhage.

Commonly used oxytocics are—oxytocin, ergometrine and prostaglandins.

Ergometrine: An ergot derivative, which increases contractile frequency and tone of uterine musculature by direct action on the myometrium. The uterus goes into a state of spasm and should only be administered after delivery of shoulder or following delivery of the baby. It is the drug of choice in uterine hemorrhage after expulsion of the fetus.

Contraindication: Due to vasoconstrictive effect, it results in hypertension and therefore, contraindicated in patients with heart disease, hypertension, preeclampsia and eclampsia. In these cases, oxytocin is a better choice.

Prostagalandins: Synthesized from fatty acid (arachidonic acid). PGE_2 and PGE_{2a} are extensively used in clinical practice. Prostaglandins promote myometrial contractions irrespective of the duration of pregnancy and so are indicated for medical termination of pregnancy. PGE_{2a} promotes myometrial contractility. PGE_{2a} helps cervical maturation. It is widely used because it is less toxic and more effective than PGE_{2a} but is more costly. Prostaglandins are used as an adjunct to oxytocin or ergometrine.

Oxytocin: A synthetic hormone, similar to posterior pituitary extracts but devoid of the direct vasoconstrictive or hypertensive response. Oxytocin acts through myometrial oxytocin receptors and voltage-mediated Ca^{++} channels to indicate myometrial contractions. It causes fundal contraction with relaxation of cervix. The sensitivity of uterus to this agent is maximum at the end of pregnancy, during labor and immediate postpartum. Oxytocin (syntocinon) ampoules contain 5 IU/mL. Routes of administration: (a) controlled IV infusion, (b) IM or slow IV bolus 10–15 units after birth of the baby. Infusion: To start with a low dose (4 mu/min) and to escalate at every 20 min intervals, if no response (2 units in 500 mL Ringer's solution with a drop rate of 60/min).

Complications: Due to direct relaxing effect on vascular smooth muscle, it causes decreased SVR hypotension and tachycardia. It has powerful vasoconstrictive effect on coronary vessels. Profound hypotension tachycardia and coronary vasoconstriction in combination leads to myocardial O_2 demand and supply mismatch resulting in myocardial ischemia. Though transient patients with hypovolemia and heart disease cannot compensate it. Oxytocin, therefore, should be used with caution, preferably in a low dose bolus very slowly or as a controlled infusion. In uterine atony, an infusion of oxytocin no more than 3 IU over 3–5 min or a bolus of up to 3 IU over 30 sec is recommended. In PPH infusion of 40 IU in 500 mL of IV fluid 125 mL/hr is recommended.

Two commonly used oxytocics are: (a) syntocinon (5–10 IU of oxytocin) (b) syntometrine (5 U oxytocin + 0.5 mg of ergometrine).

Q. How much blood returns to circulation from uterine bed immediately after delivery? When a laboring patient is more prone to develop pulmonary edema and why?

Following the delivery of the baby, the uterus contracts causing reduction in the volume of endometrial cavity. Hemostasis following delivery of the placenta is achieved by contraction of the interlacing bundles of myometrium, which constricts the blood vessels along with platelet aggregation and fibrin formation. The uterine contraction displaces about 500 mL of blood from uterine bed into the central circulation immediately after delivery of the baby (e.g. third stage of labor), which is analogous to autotransfusion.

A loboring woman is prone to develop pulmonary edema in third stage of labor, immediately after delivery of the baby and expulsion of placenta due to:

- Autotransfusion leading to burden to the heart.
- Intense uterine contraction and involution suddenly relieve IVC obstruction causing an enormous increase in cardiac load.
- Plasma protein concentration declines from the beginning of first trimester. Consistent with increase in plasma volume, the plasma protein concentration declines and serum albumin concentration falls, leading to decreased colloid oncotic pressure which is greatest at term.
- Increased capillary permeability leads to extravasation of fluid.

Q. What are the goals of anesthetic management of non obstetric surgery in a pregnant woman? Outline the anesthetic consideration.

Nonobstetric surgery in pregnant woman is gradually increasing. Most procedures are nonelective and life-threatening apart from trauma, the most common emergencies are torsion or rupture of ovarian cyst and acute appendicitis. Other less frequent situations include laparoscopic surgery, cardiac surgery under CPB, neurosurgery and most recently obstetric-related procedure intrauterine exchange transfusion and intrauterine fetal surgery.

The anesthetic management of pregnant patients is more complicated to mother but fetus is influenced by anesthetic technique. During 1st trimester, there is chance of abortion and teratogenicity. During 3rd trimester, there is chance of premature delivery. Therefore, nonemergent surgeries are best deferred until 2nd trimester or until after delivery (6 to 8 weeks postpartum).

When necessity for anesthesia arises, the anesthetic considerations include maternal risk factors resulting from physiological and anatomical changes of pregnancy, the teratogenic potential of anesthetic agents, maintenance of adequate uteroplacental blood flow, the direct and indirect effect of maternally administered anesthetic agents on fetus and potential for abortion or premature delivery. The risk must be balanced to provide most favorable outcome.

The goals of anesthetic management of nonobstetric surgery in pregnant woman at any week of gestation are:

- To assure maternal safety
- Avoidance of teratogenic drugs
- Avoidance of intrauterine fetal asphyxia
- Prevention of uterine stimulation and premature labor
- Maternal monitoring and monitoring of fetal well-being.

Anesthetic considerations in a pregnant women:

- Decreased FRC and increased oxygen consumption make a pregnant woman susceptible to arterial hypoxemia during apnea—preoxygenation is mandatory.
- Enlarged breast and increased anteroposterior diameter of thorax cause difficult intubation—laryngoscope with short handle is required.
- Capillary engorgement and edema of the upper airway cause need for smaller size endotracheal tube and careful manipulation during intubation to avoid trauma, resulting in bleeding.
- Aortocaval syndrome (maternal hypotension due to compression of IVC and lower aorta, after 20 weeks by gravid uterus in supine position) decreases fetal oxygenation by decreasing uteroplacental blood flow—left lateral displacement of uterus is maintained with left lateral tilt of 15° by placing a wedge under right buttock.
- Compression of IVC causes dialatation of azygous system and epidural venous plexus. Epidural venous engorgement reduces epidural and intrathecal space leading to increased cephaloid spread—drug doses should be reduced. Pregnant women are more sensitive to local anesthetics due to hormonal effects—reduced drug doses needed.

- Increased cardiac output and hyperdynamic maternal circulation hastens IV induction and slows down induction with inhalational anesthetics—caution should be taken during induction of general anesthesia.
- MAC of all commonly used inhalational agents is decreased—chance of overdose.
- Changes in GI system make pregnant women at increased risk of gastroesophageal reflux and pulmonary aspiration—anti-aspiration prophylaxis should be considered before anesthesia. Rapid sequence induction with cricoid pressure is to be done.
- Decreased plasma proteins during pregnancy causes decreased maternal protein binding with increased free drug concentration—chance of anesthetic drug overdose.
- Regional anesthesia causes minimum drug exposure and is preferable over general anesthesia.
- Maintenance of fetal oxygenation requires maintenance of maternal PaO_2 and $PaCO_2$, maternal blood pressure and uterine vascular resistance. Alkalosis from hyperventilation reduces uteroplacental perfusion by direct vasoconstriction and left shift of the oxyhemoglobin curve leading to decreased fetal oxygen supply should be avoided. Maternal hypotension decreases uterine blood flow. Deeper levels of inhalational agents cause maternal hypotension with fetal asphyxia and should be avoided. But lighter levels decrease uterine vascular resistance and increase uteroplacental blood flow.
- Use of anesthetic drugs with teratogenicity should be avoided.
- Preterm labor is associated with anesthesia and surgery during pregnancy, so uterine activity should be monitored closely during surgery and in immediate postoperative period and prompt treatment is instituted with tocolytic agents, if uterine contractions develop. Drugs used are calcium channel blocker (nifedipine), oxytocin receptor antagonist (atosiban, $MgSO_4$, beta-adrenergic agonists.
- Monitoring: Maternal blood pressure, oxygenation (pulse oximeter and inspired oxygen concentration) ventilation, ($EtCO_2$) and temperature. Blood sugar monitoring is done to avoid intraoperative hypoglycemia. Uterine activity should be monitored intraoperatively and in the immediate postoperative period using an external tocodynamometer, so that pharmacologic tocolysis can be instituted if uterine contractions develop.

  Monitoring of FHR and external Doppler device should be used after 16 weeks of gestation.

  Gestational age <16 weeks—elective surgery is postponed. If emergency surgery:

  a. Antiaspiration prophylaxis
  b. Maintenance of oxygenation, normocarbia
  c. Regional anesthesia preferred.

Gestational age >16 weeks:

a. Antiaspiration prophylaxis
b. Left uterine displacement throughout operation
c. Judicious use of tocolytics
d. Maternal monitoring with maintenance of oxygenation, normocarbia, normotension and euglycemia
e. Monitoring of FHR and uterine tone using external tocodynamometer.

Q. What is APGAR score? How APGAR score is evaluated ? What does 1 min APGAR score and 5 min APGAR score indicate and which one is of more value?

APGAR score was introduced by Virginia APGAR, an anesthesiologist in 1952. The score was developed to determine which baby may need resuscitation and also for studies of factors causing poor neonatal outcome. So APGAR score is the basis for neonatology and current practice of obstetric anesthesia. The universal acceptance of the scoring system is due to its simplicity and reproducibility.

Each of the five attributes of infant, namely, heart rate, respiratory effort, muscle tone, reflex irritability and color is assessed in numerical terms, zero, one or two. Addition of all 5 components provide final score with a maximum of 10 to avoid bias score and is better done by someone not involved in the care of the mother. The assessment is made generally at 1 min and 5 min to know the condition of the baby at birth. Sometimes done at 10 or 20 minutes to assess the response to resuscitation. APGAR score at 1 min is related to the status of acidosis and oxygenation of the fetus at or immediately after birth. Long-term neurological correlation is obtained at the 5 min score, which is of more value. If the score remains significantly low at 5 min, it is again evaluated at 15 min. The scoring is done in a newborn baby at 1 min, 5 min and 15 min. On the basis of scoring, the newborn babies are divided into three groups, which seem to correlate with acid-base measurement:

1. APGAR score >7, good—baby in good health.
2. APGAR score 4–6, fair—moderately depressed baby.
3. APGAR score 0–3, poor—combined metabolic and respiratory acidosis requiring immediate resuscitation.

Q. What is the anesthetic management of intrauterine fetal surgery?

Advances in prenatal diagnosis with use of modern technology (ultrasonography, fetal echocardiography, fetal MRI, amniocentesis, umbilical blood sampling and chorionic villous sampling) and the development of open fetal surgical techniques now permit the treatment of fetal conditions that would otherwise have caused progressive reversible damage and intrauterine fetal death, if left untreated, and allow normal development to proceed, if treated. Such anomalies include:

- Congenital diaphragmatic hernia
- Hydronephrosis

- Hydrothorax
- Twin-twin transfusion syndrome
- Hydrocephalus
- Meningomyelocele
- Sacrococcygeal syndrome.

Fetal surgery can be divided into three distinct procedure groups:

- *Ex-utero intrapartum treatment (EXIT):* These are also operations on placental supports and are performed on vaginal delivery or cesarean section. Only a portion of the fetus is delivered or brief procedures like endotracheal intubation or examination of neck mass done while the fetus is still connected to placenta through umbilical cord and oxygenated through placental transfer of O_2. After completion of the procedure, the fetus is delivered, the umbilical cord is divided and the baby placed in the care of neonatologist. The uterus is made involuted to prevent postpartum hemorrhage.
- *Mid gestation open procedures:* Recognition of fetal defect in early pregnancy allows intervention in mid gestation to prevent irreversible damage. Hysterotomy is required to access the fetus. The fetus is exteriorized for surgical intervention and then placed back in the uterus after completion of surgery for rest of the gestation. The uterus is closed. After this procedure, there is improved fetal survivability and enhanced post-gestation quality of life.
- *Minimally invasive mid-gestation procedures:* Fetoscopic surgery is a minimally invasive technique utilizing small diameter trocars and laparoscope placed percutaneously to access the uterus. This technique is commonly used for the evaluation and treatment of twin reverse arterial perfusion sequence, twin-twin transfusion syndromes, amniotic band syndrome and bladder outlet obstruction. During these procedures, electrocautery or lasers are used to ablate or cauterize vessels or tissues. This technique is considered, when fetal demise or severe fetal morbidity is imminent.

Anesthetic goals of fetal surgery:

- Maternal safety should be considered because many of the physiological changes during pregnancy increase anesthesia related risks.
  - Gastric content aspiration and hypoxemia should be prevented.
  - Left uterine displacement maintained to prevent aortocaval compression causing hypotension and decreased uteroplacental circulation.
  - Decreased doses of anesthetic agents required due to increased sensitivity during pregnancy.
  - Close attention to fluid balance needed in the postoperative period due to increased risk of pulmonary edema in pregnant women who received tocolytic therapy.
- Maintenance of fetal cardiovascular stability: Less cardiac contractile tissue leads to depression from volatile anesthetics. Surgical blood loss

poorly tolerated due to low blood volume and rate dependent cardiac output.
- Maintenance of uteroplacental circulation is vital.
- Avoidane of teratogenic agents.
- Avoidance of fetal asphyxia and kinking of umbilical cord.
- Prevention of preterm labor: Uterus must remain relaxed with tocolytic therapy (volatile anesthetic agents, $MgSO_4$, terbutaline, nitroglycerine). Uterine activity is monitored using external tocodynamometer.
- Fetal monitoring should be done using
  - Pulse oximeter probe placed on the limb and wrapped with foil (Normal saturation 60–70% and >40% adequate).
  - Echocardiography to monitor FHR and stroke volume.
  - Fetal arterial or venous sampling for acid-base balance.
- Prevention of fetal hypothermia—active warming required.

Anesthetic plan for fetal surgery: Regional anesthesia is a technique of choice for obstetric anesthesia. But because uterine relaxation is required for hysterotomy based fetal surgery, anesthesia is best provided with general anesthesia using high concentration of inhalation agents.

The patient is admitted on the day of surgery after being NPO. 0.3 M sodium bicarbonate is given orally and metoclopramide intravenously as prophylaxis for aspiration, 10–15 minutes prior to anesthesia. An indomethacin suppository is administered for postoperative tocolysis. Monitors include two pulse oximeters (maternal and fetal) and a maternal arterial transducer. Type specific packed red cells for the mother and O-negative packed red blood cells for the fetus are made available.

The operation room (OR) is warmed to 26.7°C. The parturient is then positioned on OR table with left lateral tilt to minimize supine hypotension. A large bore IV catheter is inserted. A lumbar epidural catheter is inserted and tested for postoperative analgesia.

Epinephrine 10 μg/kg, atropine 20 μg/kg, vecuronium 0.2 mg/kg and fentanyl 10–20 μg/kg are kept prepared in sterile 1 mL syringes for possible fetal IM administration.

A rapid sequence induction using IV sodium thiopentone and succinylcholine performed followed by tracheal intubation with cuffed endotracheal tube and application of cricoid pressure until accurate placement of endotracheal tube. Anesthesia is maintained with 0.5 MAC volatile anesthetic agents (isoflurane or sevoflurane) and 50% nitrous oxide in oxygen. A radial artery catheter, a second IV catheter, nasogastric tube and Foley catheter are inserted. Intravenous fluid infusion is restricted (500 mL total). Fetal status is monitored by sterile intraoperative echocardiography.

Open hysterotomy procedures require low uterine tone to maintain fetal perfusion and optimize fetal exposure. Before maternal skin incision, N_2O is turned off to improve fetal oxygenation and inhalation agent is increased to 2.0 MAC to provide uterine relaxation and fetal anesthesia. Maternal systolic blood pressure is also maintained within 10% of the baseline by

administering 5–10 mg ephedrine IV or 1–2 μg/kg phenylephrine IV, if necessary. Fetal anesthesia and analgesia are provided by placental transfer of inhalation agents and IM administration of opioids. Before fetal incision, the fetus receives fentanyl 20 μg/kg IM to supplement anesthesia and provide postoperative analgesia.

Fetal well-being is assessed by pulse oximeter to monitor fetal arterial saturation (>40% represent adequate oxygenation). Echocardiography is used to monitor heart rate and stroke volume. Fetal arterial or venous blood sampling for acid-base balance and fetal distress. O-negative blood can be administered to the fetus for correction of anemia intraoperatively. Following closure of uterus, anesthesia is converted to regional technique by administering local anesthetic and opioid through epidural catheter and decreasing volatile anesthetic. Tocolysis is instituted with a loading dose of $MgSO_4$ 6 grams IV followed by infusion at a rate of 2–3 grams per hour. After skin closure, the patient is adequately reversed, extubated and transferred to obstetric care unit for postoperative care. Magnesium sulfate is the drug of choice for tocolysis in the early postoperative period (up to 24 hours) and patient controlled epidural infusion is used for analgesia.

During ex-utero intrapartum therapy, tocolysis is not necessary as the procedure ends in delivery. Following delivery, uterine relaxation is quickly reversed. Volatile anesthetic is discontinued after cord clamping and administration of oxytocin, methergine or prostaglandin-F2α is done. Due to anesthetic-induced uterine relaxation, significant blood loss is a risk. So blood loss is monitored and cross-matched blood is administered, if needed. After securing the airway and assuring adequate fetal oxygenation with manual ventilation, the umbilical cord is clamped and the fetus delivered. Following delivery, two patients are to care for. The mother is transferred to a postpartum ward. The disposition of newborn depends on surgical need. A second OR should be available in case further surgery is needed. If surgery is not required, the newborn is resuscitated and transferred into intensive care.

In mid-gestation open fetal surgery, since the fetus is returned to the uterus after completion of intervention, the parturient requires tocolysis and fluid restriction. After the fetus is returned to the uterine cavity, the amniotic fluid is replaced by warm Ringer's solution containing antibiotics and the uterus is closed. Aggressive tocolysis is initiated after conclusion of fetal procedure with IV infusion of $MgSO_4$ to prevent reflex uterine contractions which reduces uteropalcental blood flow and results in premature labor. Postoperatively, the parturient is transferred to maternal-fetal intensive care unit. Uterine activity and fetal HR are continuously monitored by tocodynamometry. Use of epidural catheter reduces maternal stress response and incidence of early preterm labor.

Most of the minimally invasive surgeries can be done with a large bore needle with ultrasound imaging or a 5 mm trocar with a camera and a port for laser tube to coagulate placental vessels in twin-twin transfusion syndrome. In fetoscopy, normal saline irrigation is used inside the uterus. The maternal incision is small and can be performed under local anesthesia. Epidural,

spinal and combined spinal and epidural (CSE) anesthesia can be used. Intravenous sedation is given for maternal anxiolysis during fetoscopy. Light sedation is maintained with midazolam, fentanyl, remifentanil or propofol infusion. There is chance of pulmonary edema due to absorption of uterine irrigation fluid from peritoneal cavity along with tocolysis. This can be treated with diuretics. Most fetoscopy procedures involve manipulation placenta and umbilical cord. As there is no fetal incision, fetal anesthesia is not required. Most important aspect of postoperative management is tocolysis. Epidural catheter is removed after surgery. $MgSO_4$ followed by either nifedipine or terbutaline are the mainstay of tocolytic management. Discharge from hospital is expected on postoperative day 1 or 2.

Advantage of epidural anesthesia are minimal effect on fetal hemodynamics, on uteroplacental blood flow and postoperative uterine activity, yet it is not suitable for fetal surgery due to lack of uterine relaxation, lack of fetal anesthesia, difficult manipulation of the uterus and cord while the fetus is moving. This technique is used to provide postoperative analgesia and to reduce the incidence of preterm labor. A balanced general anesthesia with inhalation-opioid anesthesia has the advantage of allowing uterine with an immobile anesthetized fetus and less cardiovascular depression than deep inhalation anesthesia. General anesthesia also eliminates concerns associated with awake patient such as fear, anxiety, nausea and vomiting. Balanced anesthesia cannot provide full uterine relaxation to allow access to different cord position. Deep inhalation anesthesia causes profound uterine relaxation allowing externalization of the uterus and hysterotomy based procedures. But disadvantages of this technique are fetal cardiovascular depression and decreased uteroplacental blood flow.

Q. What is preeclampsia? What are the features of mild preeclampsia and severe preeclampsia?

- Preeclampsia is a multisystem disorder of unknown etiology, unique in pregnant women occurring after 20 weeks of gestation and characterized by hypertension, proteinuria, and edema which returns to normal within 3 months after delivery.

  Features of mild preeclampsia include:
  - Gestation >20 weeks.
  - Arterial blood pressure: Diastolic blood pressure >110 mm Hg on one occasion or >90 mm Hg on two or more occasions of at least 4 hours apart:
  - Proteinuria: >300 mg/24 hours.
  - Edema: Not required for diagnosis because it is detected in most of the normotensive healthy pregnant women.

  Features of mild preeclampsia include:
  - Systolic blood pressure >160–180 mm Hg, diastolic blood pressure >110 mm Hg on two occasions of at least 4 hours apart
  - Proteinuria: >5 g in 24 hour urine
  - Oliguria: <400–500 mL urine in 24 hours

- Cerebral or visual disturbances
- Pulmonary edema, cyanosis
- Right hypochondric or epigastric pain
- Impaired liver function tests
- Thrombocytopenia (<1,00,000/mm^3) or HELLP syndrome (Hemolysis, elevated liver enzyme and low platelet count)
- Fetal growth retardation.

Q. What is the pathophysiology of preeclampsia? What effects are produced on mother and fetus?

The precise cause of preeclampsia is unknown, making it disease of theories.

- Widespread endothelial cell injury is the principal underlying pathophysiology of preeclampsia. In preeclampsia, the normal endovascular invasion of cytotrophoblast into the spiral arteries fail to occur beyond decidua-myometrial junction, leaving the musculo-elastic portion of the arteries intact. Therefore, these arteries remain responsive to vasoconstrictor stimuli, resulting in decreased placental blood flow. The incomplete trophoblastic invasion also results in narrowed spiral arteries and subsequent placental ischemia. Subsequently, the abnormal placenta may release one or more factors which damage vascular endothelial cells throughout the body leading to multiorgan dysfunction. Endothelial cell injury and altered endothelial function result in increased secretion of vasoactive substances like endothelin-I, fibronectin, serotonin and thromboxane with decreased secretion of vasodilators like nitric oxide and prostacyclin and thromboxane. Majority of the symptoms associated with preeclampsia including placental ischemia, systemic vasoconstriction and increased platelet aggregation result from this imbalance.
- In preeclampsia, the stimulation of renin-aldosterone system does not occur, as during normal pregnancy and the adrenergic sensitivity is not blunted as in normal pregnancy. Angiotensin receptors remain up-regulated and circulating level of angiotensin may be excessive in relation to increased vascular sensitivity.
- Immunologic mechanism: It is supported for the abnormal levels of autoantibodies found in the maternal blood in preeclampsia and are thought to be responsible for the endothelial damage.
- Another hypothesis involving the role of calcium has been postulated. Hypocalcuria and reduced vitamin D levels are said to be responsible for endothelial injury.

Thus, there are variety of factors which favor increased vascular reactivity and tone in preeclampsia which include increased level of vasoconstrictors as thromboxane, endothelin, fibronectin and intracellular free calcium along with decreased levels of vasodilators as nitric oxide and prostacyclin.

Maternal effects: All Major organ systems are affected due to widespread vasospasm.

- *Hematodynamic profile:*
  - Total blood volume is reduced, being proportional to severity. Increase in plasma volume as during normal pregnancy is absent. Red cell volume is unchanged. So, hematocrit is elevated.
  - Blood pressure is elevated and is labile. There is also increased sensitivity to angiotensin II.
  - Cardiac output may be increased, normal or decreased with increased systemic vascular resistance (SVR). The hemodynamic findings in preeclampsia may be described as low volume, high pressure and high resistance.
  - Pulmonary artery pressure: Not increased and PVR is low to low-normal for pregnancy. Pulmonary artery wedge pressure (PAWP) in preeclampsia varies with the severity of change in SVR. If SVR is high, PAWP becomes low. There is poor correlation between CVP and PAWP.
  - Colloid oncotic pressure: Reduced to a greater extent than in normal pregnancy and is attributed to reduced capillary hydrostatic pressure, increased microvascular permeability to protein with reduced albumin concentration.

  Pulmonary edema in preeclamptic patient occurs due to three different mechanisms:
  - Altered capillary permeability.
  - Reduction in intravascular colloid oncotic pressure.
  - Left ventricular dysfunction—least common.

  Increased afterload causes left ventricular dysfunction and reduction in afterload corrects the situation. Therefore, pulmonary edema in preeclampsia may be cardiogenic or noncardiogenic. Heart failure may occur in severe cases due to peripheral vasoconstriction and increased viscosity.
- *Coagulation abnormalities:* Thrombocytopenia is a common finding in preeclampsia. This is caused by increased platelet consumption associated with low grade DIC. In preeclampsia, there is imbalance between prostacyclin and thromboxane, which is likely to contribute to enhanced platelet activity.

  Recently, investigations have been carried out using thromboelastograph to examine platelet function in preeclamptic patients with the conclusion that severe preeclamptic women with platelet count less than 1,00,000/mm^3 are hypercoagulable than healthy pregnant women. Richardson in 2000 claimed that a platelet count of 70,000/mm^3 is more worrisome if it represents a significant decline over hours than it has been stable for days.
- *Respiratory system changes:* Generalized edema involves upper airway including hypopharynx leading to difficult laryngoscopy and intubation. Smaller size ETT is needed. There may occur pulmonary edema.
- *Fluid therapy:* Though there is exaggerated retention of water and sodium, there occurs shift of fluid and protein from intravascular to extravascular compartment. Preeclampsia is associated with intravascular volume reduction, hemoconcentration and hypoproteinemia because plasma

volume should be corrected with fluid administration before vasodilatation with drugs or epidural analgesia/anesthesia.

The aim of fluid therapy is to raise CVP to 4–6 cm of H_2O or PAWP to 8–10 mm Hg and urine output to 1 mL/kg/hr. The current recommendation is to infuse crystalloids at a rate of 1–2 mL/kg/hr. If pulmonary edema develops treatment includes oxygen, diuretics, reduction in afterload/preload, fluid restriction and intermittent positive pressure ventilation (IPPV).

Prehydration before regional anesthesia should be done carefully with smaller volumes (200–300 mL bolus). Subsequent hypotension is treated with additional crystalloid bolus of 200–500 mL.

There is increased sensitivity to catecholamine and other vasoactive agents like ephedrine. Such drugs should be administered in titrated doses.

- *Central nervous system changes:* There is vasospasm and ischemia causing hypoxia. As a result, there is cerebral edema and small degenerative foci. Symptoms related to these changes include headache, hyperreflexia and convulsions.

  In eyes, intense arteriolar constriction results in blurred vision, even temporary blindness.
- *Liver:* There is impaired liver function which can affect drug metabolism. Decreased blood supply leads to periportal necrosis and subcapsular hemorrhage. There is elevated plasma levels of aspartate aminotransferase and lactate dehydrogenase, which may be a part of HELLP syndrome. This syndrome consists of hemolysis, elevated liver enzymes and low platelet count.
- *Renal:* Renal function is adversely affected. Arteriolar spasm leads to decreased renal perfusion. There is swelling of glomerular endothelial cells and deposition of fibrin leading to constriction of capillary lumina. Glomerular blood flow and filtration rate decrease leading to reduced uric acid clearance. Increased plasma uric acid level in preeclampsia reflects tissue ischemia and oxidative stress. Oliguria (<500 mL of urine in 24 hours) and proteinuria are the characteristic features of preeclampsia.
- The uterus becomes hyperreactive and more sensitive to oxytocin.

Fetal effects: Fetal effects are due to reduction in uterine and intervillous blood flow resulted from vasoconstriction or occlusive lesion in the decidual arteries. Reduced placental blood flow causes placental eclampsia and infarction leading to chronic fatal hypoxia and malnutrition. The risk of intrauterine growth retardation preeclampsia.

Q. What is the anesthetic technique in preeclamptic patients?

For labor analgesia, epidural technique is preferred choice and should be started early in labor room. It reduces blood pressure and improves uteroplacental circulation due to vasodilatation along with reduced stress responses and release of catecholamine due to pain. Platelet count <50,000/mm^3 is absolute contraindication for epidural analgesia and >70,000/mm^3 is acceptable and mixture of 1% bupivacaine with 2 kg/mL of fentanyl can

be used. 15 mL of the mixture is given intermittently in small (3 mL) doses in epidural route to avoid intravascular or subarachnoid injection. In CSE technique, 3 mL is administered through spinal route followed by 12 mL in epidural route.

For cesarean section, evidence has accumulated in favor of neuraxial block. Previously epidural technique was preferred because of the concern of rapid hypotension during subarachnoid block. But various studies and trails have demonstrated that epidural, spinal and CSE, all are safe in preeclampsia. Hypotension caused by spinal anesthesia is lesser in preeclamptic patients, which may be due to increased level of catecholamines. Sympatholysis produced by epidural technique improves uterine blood flow in preeclamptive woman during cesarean section and labor analgesia (in preeclampsia maternal uterine spiral arteries are abnormally responsive to sympathetic tone leading to vasoconstriction and decreased uterine blood flow). Unlike systemic vasodilators, epidural block induces segmental vasodilatation with reflex vasoconstriction in non-anesthetized areas and no redistribution of blood from uteroplacement circulation.

General anesthesia is avoided in preeclamptic parturient for cesarean section due to several reasons:

- Potentially difficult laryngoscopy of intubation respiratory tract during pregnancy.
- Increased risk of aspiration of gastric contents due to delayed gastric emptying during pregnancy.
- Increased sensitivity to muscle relaxants, especially in patients receiving $MgSO_4$.
- Exaggerated pressure response to laryngoscope of intubation.
- Impaired placental blood flow.

General anesthesia is reserved for:

- Patient refusal for regional anesthesia
- Coagulopathies
- Severe maternal hemorrhage
- Sepsis.

Procedure: Before induction, restoration of fluid volume of control of BP are done. Hemodynamic responses to laryngoscope of intubation are minimized, quick intubation (<30 sec). Antihypertensive drugs (Labetalol, hydralazine and GTN) given shortly before induction. Opioids (Remifentanil 1 μg/kg LV bolus) are given prior to induction. Awareness is a concern. B/S value does not improve much. Remifentanil during induction and sevoflurane during maintenance are useful. Monitoring is done. NSAIDs are avoided as some of these drugs constrict or close fetal PDA in late pregnancy.

Preeclamptic patient is a challenge because:

- Often limited time to optimize.
- Surge of BP during laryngoscope and intubation—chance of cerebral hemorrhage

- Low platelet count contraindicating
- neuraxial block
- Volume contracted but chance of pulmonary edema—judicious fluid administration
- Increased chance of seizures
- Increased chance of PPH.

Q. What are the common types of diabetes in pregnancy? What are the effects of pregnancy on diabetes? What are the effects of diabetes on pregnancy?

The common types of diabetes mellitus in pregnancy are gestational diabetes mellitus (GDM) or pregnancy-induced glucose intolerance and overt diabetes mellitus.

Gestational diabetes mellitus (GDM): First directed during present pregnancy. Oral glucose tolerance should come down to normal following delivery. These patients need more frequent antenatal supervision with periodic checkup of fasting blood glucose level which should be <95 mg/dL. Control of blood sugars is done by diet, exercise, with or without insulin.

Overt diabetes mellitus: A pregnant woman with symptoms of diabetes and random blood sugar level 200 mg/dL or none is considered as overt diabetic. This type of diabetes may preexist or detected for the first time during recent pregnancy. Diagnosis is positive when fasting plasma glucose level is >126 m/dL or 2 hours post glucose (75 g) value exceeds 200 mg/dl.

Effects of pregnancy on diabetes:

- During pregnancy, there is insulin antagonism due to placental hormones leading to increased insulin requirement with advances of pregnancy.
- Due to renal glycosuria, more glucose is out in urine. Hence, insulin dose cannot be adjusted by urine test and repeated blood glucose test is mandatory.
- Vascular changes produced by diabetes women during pregnancy.

Effects of diabetes on pregnancy:

Maternal effects (Hazards)

- Abortion—associated with uncontrolled diabetes.
- Preterm labor—due to infection and polyhydramnios.
- Increased incidence of preeclampsia.
- Polyhydramnios—common association due to large fetus, large placenta and fetal hyperglycemia irrigating amniotic epithelium.
- Maternal distress—due to large fetus, large placenta and polyhydramnios.
- Infection—common occurrence, especially urinary tract infection.
- Labor—prolonged labor and shoulder dystosia due to fetal macrosomia.
- All complications of diabetes mellitus, especially CAD, nephropathy, retinopathy, neuropathy and diabetic ketoacidosis, worsen during pregnancy.

Fetal effects (Hazards)

- Macrosome (>4 kg birth weight)
  Results from:
  - Maternal hyperglycemia, which stimulates fetal islets, increasing fetal insulin level. This stimulates carbohydrate metabolism and accumulation of fat in fetus.
  - Elevation of maternal free fatty acid, leading to increased transfer to fetus with accelerated triglyceride synthesis resulting fetal adiposity.
  - Insulin-like growth factors (1 GF-I and II) are also involved in exaggerated fetal growth and adiposity.
- Congenital malformations—related to the severity of diabetes affecting organogenesis, in the first trimester.
- Unexplained intrauterine fetal death due to hypoxia and lactic acidemia.
- Birth injuries (brachial plexus) due to prolonged labor and shoulder dystopia, caused by fetal macrosome.
- Increased perinatal mortality.

Neonatal death is due to hypoglycemia, RDS, hyperbilirubinemia, polycythemia and cardiomyopathy. In most of the diabetic mother, delivery is done by cesarean section. Epidural or spinal anesthesia is better choice.

Care of the fetus

- Observation in NICU for 48 hours to detect and treat complications like—asphyxia and hypoglycemia.
- Congenital anomalies are to be detected.
- Early breastfeeding, within 1 hour, to minimize hypoglycemia
- All neonates should receive 1 mg vitamin K_1.

Q. What are the types of anemia in pregnancy? What are the maternal and fetal effects of anemia? Outline anesthetic considerations of anemia in pregnancy.

Types of anemia during pregnancy may be:

1. Physiological anemia.
2. Pathological or acquired anemia.

Physiological anemia: Profound hematological changes occur during pregnancy. There is progressive increase in maternal blood volume mainly due to increase in plasma volume. Proportionately the increase in red cell mass is less than the increase in plasma volume leading to dilusional anemia in pregnancy. This dilusional anemia reduces the oxygen carrying capacity of blood, which is compensated by various changes like:

- Increase in maternal PaO_2.
- Decrease in blood viscosity.
- Rightward shift of oxyhemoglobin dissociation curve.

Anemia is defined as quantitative or qualitative reduction of hemoglobin or circulating RBC or both.

WHO defines anemia in pregnancy as a hemoglobin concentration of <10 g/dL or hematocrit <33 g/dL.

Pathological or acquired anemia:

- Nutritional anemia
  - Iron deficiency anemia due to poor dietary intake
  - Foliate deficiency due to decreased plasma folate concentration resulted from increased renal clearance of folic acid during pregnancy
- Hemorrhagic anemia
  - Hookworm infestation is the common cause
- Hemoglobinopathies
  - Sickle cell disease or thalassemia.
- Infections
  - Malaria causes hemolysis leading to anemia
  - HIV infection.

Treatment of anemia:

Treatment of underlying causes:

- Balanced diet rich in protein, vitamin and iron
- Iron therapy:
  - Oral therapy in ferrous form (ferrous sulfate)
  - Parenteral therapy by IM or IV administration
- Supplementation of folate. WHO recommends folate supplementation up to 5 mg/day orally.
- Recombinant human erythropoietin.
  - It is necessary for normal production of RBC
  - It is effective in hastening recovery of hematocrit
  - It reduces transfusion requirement in anemic elective surgical patients
  - It is expensive and requires frequent subcutaneous or intravenous administration.
- Blood transfusion—limited indications.

Maternal effects of anemia (complications)

- During pregnancy, increased incidence of
  - Preeclampsia
  - Heart failure
  - Preterm labor
  - Infection.
- During labor
  - Uterine inertia
  - Postpartum hemorrhage and shock
  - Cardiac failure.

Effects on fetus:

- IUD
- Low birth weight babies:
  - Baby born at term to severely anemic mother is not anemic at birth but due to low or no reserve of iron, anemia develops in the neonatal period.

Anesthetic considerations

Regional anesthesia: Spinal or epidural techniques need crystalloid infusion to fill up increased vascular bed due to sympathetic blocked. This may exacerbate anemia temporarily and may precipitate heart failure. Therefore, after infusion of 500 mL of crystalloid fluid, it is beneficial to administer vasoconstrictors to sustain blood pressure. Patients with megaloblastic anemia, may develop neurological symptoms postoperatively, so regional anesthesia should be preferably avoided in those case.

General anesthesia:

- Principal hypoxia is avoided
- Cardiovascular stability is maintained
- Factors causing alteration of rightward shift of oxygen dissociation curve should be minimized.

Procedure: Adequate preoxygenation before induction to have enough oxygen reserve to tide over apnea during intubation.

Intravenous inducing agents should be administered slowly to prevent cardiovascular disturbances.

Possibility of awareness should be considered as increased inspired oxygen concentration is used in anemic pregnant patients.

Disturbance in rightward shift of oxygen dissociation curve is prevented by avoiding alkalosis and hypothermia.

Ventilation provided to maintain normocapnea.

Spontaneous ventilation with high concentration of inhalational agents, depresses respiration and mycocardial function, adequate oxygen flux (the quantity of oxygen delivered to the tissues in each minute)—Controlled ventilation with minimum volatile agent concentration is desired.

Adequate tissue perfusion is judged by blanching ear lobule, nose, forehead and time taken for pallor to disappear.

Postoperative care: Extubation after adequate recovery from anesthetics and reversal of effect of muscle relaxants.

Observation is recovery room for 12–24 hours for monitoring of respiration and other vitals.

Oxygen enriched air given by face mask.

Shivering prevented (increases O_2 consumption), hemoglobin level checked and treated accordingly.

Pulse, BP, O_2 saturation should be monitored, fluid intake and output chart maintained.

Q. What are the effects of cardiovascular physiology during pregnancy on heart disease?

Effects of cardiovascular physiology during pregnancy on heart disease: During pregnancy, the cardiac output starts to increase from 5th week of pregnancy and reaches the peak (40–50%) at about 30–34 weeks and remains stage till term. The increase in cardiac output is attained by increase in stroke volume and increase in heart rate. The cardiac output increases further during labor (+50%) immediately after delivery (+70%) over problem values. There is squeezing out of blood from the uterus into material circulation (autotranfusion) during labor and immediate postpartum. These changes in cardiac output are well-alerted by pregnant women with normal heart or diseased heart with good cardiac reserve, but cardiac failure occurs in pregnant woman with damaged heart with poor cardiac reserve. Cardiac failure occurs during pregnancy around 30 weeks, during labor and mostly immediately following delivery.

Effects of Heart disease on pregnancy: In cyanotic heart disease, there is increased tendency of:

- Intrauterine growth retardation
- Prematurity
- Preterm delivery.

Q. What are the common heart diseases in pregnancy? What is the management of specific heart disease in pregnancy?

Commonest cardiac lesion is rheumatic origin followed by the congenital ones. Rheumatic valvular lesion predominantly includes mitral stenosis. Predominant cyanotic lesions include PDA, ASD, VSD, pulmonary stenosis, cortication of aorta and Fallots tetralogy.

Specific heart diseases

- *Rheumatic heart diseases:*
  - Mitral stenosis: Commonest heart lesion during pregnancy. Mortality is higher in symptomatic cases than asymptomatic cases. Normal mitral value area is between 4 and 6 cm.
  - Place of valvotomy: It is better to withheld elective cardiac surgery during pregnancy. It requires the best time for surgery which is between 14 and 18 weeks. Value replacement, commisurotomy, balloon valvotomy can be carried out in early second trimester. Atrial fibrillation is a complication and treated with digoxin, blocker and anticoagulant therapy.

  During labor, continuous epidural analgesia is ideal. Intravenous fluid overload is to be avoided.

  Fetal outcome is good in parturient with rheumatic heart lesion.
- *Congenital heart disease:* A pregnant woman with corrected congenital heart lesion poses little problem in obstetric. But when pregnancy occurs in a patient with uncorrected congenital heart lesion, problems are very

high. Mortality is low in acyanotic heart lesion but high with cyanotic group of heart lesion with increased pulmonary vascular resistance and Eisenmenger's syndrome. In cyanotic heart lesion, fetal loss increases due to abortion, prematurity and intrauterine growth retardation.

– ***Acyanotic congenital heart lesions (Left to right shunt):***
  - Patent ductus arteriosus (PDA): Most patients with PDA to tolerate pregnancy well. Pulmonary hypertension may cause maternal death Epidural and spinal anesthesia, better avoided to minimize shunt reversal due to hypotension from sympathetic blockade. Fetal loss is up to 7%.
  - Atrial septal defect (ASD): Most common congenital heart lesion during pregnancy. Even with in corrected ASD, a woman can well- tolerate pregnancy and labor. Congestive heart failure and unresponsive to medical therapy require surgical correction. Shunt reversal is a major risk due to hypotension resulting from hemorrhagic condition and injudicious administration of epidural or spinal anesthesia. Pulmonary hypertension is uncommon.
  - Ventricular septal defect (VSD): Pregnancy is well tolerated with small-to-moderate left-to-right shunt. Pulmonary hypertension and congestive heart failure do not occur. The major risk is shunt reversal leading to circulatory collapse and cyanosis. Hypotension is to be avoided. Fetal loss may be up to 20%.

– ***Cyanotic congenital heart lesion (Right-to-left shunt):***
  - Fallot's tetralogy: Most common form of cyanotic heart lesion met during pregnancy. It is a combination of ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy and overriding of aorta. Surgically incorrected patients are at increased risk. Bacterial endocarditis, brain abscess and cerebral embolism are the more common complications. Systemic hypotension may lead to death.
  - Eisenmenger's syndrome: Patients have pulmonary hypertension with right-to-left shunt through PDA, ASD, VSD. Heparin should be used throughout pregnancy, as there is risk of systemic and pulmonary thromboembolism. Epidural and spinal anesthesia are contraindicated.

Q. What are the changes during pregnancy that mimic cardiac disease?

The changes during pregnancy that mimic cardiac disease are:

- Fatigue, syncope, dyspnea and decreased exercise tolerance
- Tachycardia and hyperdynamic circulation
- The apex beat is shifted to the 4th intercostal space with outside the midclavicular line with slight rotation to left due to elevation of the diaphragm by enlarged gravid uterus
- Systolic ejection murmur audible in the apical or pulmonary area. This is due to increased blood flow through the aortic and pulmonary valves resulted from decreased blood viscosity and torsion of the great vessels

- A continuous hissing murmur, audible in left second and third intercostal spaces due to increased blood flow through internal mammary vessels (mammary murmur)
- Loud first sound with splitting.

Q. Outline the diagnosis of heart disease in pregnancy.

The approach to diagnosis of heart disease in pregnancy is as follows:

- *Symptoms:* Breathlessness, chest pain, syncope.
- *Signs:* Chest murmurs like pansystolic, late systolic, ejection systolic or diastolic associated with thrill, arrhythmias.
- *Chest radiograph:* Cardiomegaly, increased pulmonary vascular marking, enlargement of pulmonary veins.
- *Electrocardiography:* T wave inversion, dysrhythmias.
- *Echocardiography (color flow Doppler echocardiography) structural abnormalities:* Defects (like ASD,VSD), valve anatomy, valve area, cardiac function, left ventricular ejection fraction and pulmonary artery systolic pressure.

Q. What is New York Heart Association (NYHA) of heart disease?

NYHA classification of heart disease is done depending on the cardiac response to physical activity. It considers symptoms only but not anatomical type or severity of pathology.

Class 1—uncompromised heart disease with no symptoms, patients with cardiac disease but no limitation of physical activities.

Class 2—slightly compromised mild symptoms with extreme exertion. Patients with cardiac disease with slight limitation of physical activity. Patients are comfortable at rest but ordinary physical activity causes discomfort.

Class 3—markedly compromised, symptoms with minimum exertion, patients with cardiac disease with marked limitation of activity. Patients are comfortable at rest, but discomfort occurs with less than ordinary activity.

Class 4—severely compromised, symptoms at rest. Patients with cardiac disease with discomfort even at rests.

Q. What is peripartum cardiomyopathy? How it affects mother and fetus? Outline the treatment of cardiomyopathy.

Peripartum cardiomyopathy is defined as idiopathic cardiomyopathy causing deterioration of cardiac function anytime from last month of pregnancy till 5 months postpartum. It presents with heart failure due to left ventricular dysfunction in absence of any other cause of failure. The left ventricular ejection fraction (LVEF) is nearly always reduced to <45%.

The exact cause is unknown. The characteristic presenting features are:

- Dyspnea on exertion
- Chest pain, palpitations
- Arrthymias, pedal edema
- Raised jugular venous pressure

- Pulmonary rales
- Chest X-ray: Cardiomegaly and pulmonary congestion
- ECG: LVH and ST-T abnormality
- Echocardiography: Dilated left ventricle, increased, LVEF <45%

Maternal effects: Hypoxia, arrhythmias, cardiac failure and thromboembolism.
Fetal effects: Distress due to maternal hypoxia and placental hypoperfusion from:

- Decreased maternal cardiac output
- Maternal hypovolemia produced by diuretics
- Maternal hypotension due to afterload reduction.

Q. What is the treatment of peripartum cardiomyopathy? What are the anesthetic considerations?

Treatment of peripartum cardiomyopathy:

Goals:

- Improved oxygenation and maintenance of cardiac output
- Optimum decrease in pre- or afterload
- Improvement of cardiac contractility.

Rest and salt restriction:

- Oxygen administration via facemask or CPAP
- Diuretics to prevent water retention and decrease pulmonary congestion
- Hydralazine and nitrates to decrease afterload
- Anticoagulation in patients with LVEF <35% and fibrillation using low molecular weight heparins as there bioavailability is more predictable and have lower risk of thrombocytopenia
- BP and CVP monitoring done
- Noninvasive ventilation is done to maintain SPO_2 >95% nitroglycerine IV infusion at a rate of 10–20 mg/min to decrease overload
- Levosimendan IV infusion at a rate of 0.1–0.2 mg/kg/min to improve cardiac output and decrease mortality inotropes and inodilators
- Digoxin used.

Drugs not to be used:

- Calcium channel blockers are avoided due to negative inotropic effect
- ACE inhibitors are beneficial but are avoided due to risk of fetal toxicity
- Blockers are beneficial but are avoided due to increased incidence of low birth weight baby
- Nitroprusside is contraindicated in pregnancy due to cyanide toxicity in fetus
- Warfarin is teratogenic and causes fetal growth retardation and cerebral complications. So avoided
- Unfractional heparin is not used because of its low bioavailability in pregnancy and also causes thrombocytopenia.

Anesthetic considerations in peripartum cardiomyopathy:

Anesthesiologist must be vigilant regarding:

- Hypoxemia
- Hypotension
- Atrial fibrillation
- Pulmonary edema
- Electrolyte imbalance
- Thromboembolism
- Sudden death.

Vaginal delivery:

- Continuous hemodynamic monitoring with invasive monitors
- Labor analgesia preferred to decrease sympathetic nervous system activity with pain and anxiety, thereby preventing increase in cardiac output, increase in systematic vascular resistance with increased afterload and decrease in uteroplacental blood flow
- Regional analgesia is the method of choice, because sympathetic blockade decreases cardiac preload and afterload.
- Second stage labor should be shortened. Fluid overload is prevented.

Cesarean section:

- Goals: Cardiac preload and afterload are to be decreased
- Cardiac contractility should be maintained
- Sudden decrease in systematic vascular resistance is avoided
- Invasive monitoring (CVP and arterial BP) are needed

Regional anesthesia may be used in elective cesarean section delivery.

Problems:

- Excessive preload reduction resulting decreased cardiac output (CO).
- Excessive afterload reduction resulting in decreased coronary circulation.

General anesthesia

Indicated in:

- Emergency cesarean section
- Borderline cardiac decompensation
- Anticoagulated patients
- Propofol and emifentanil are preferred
- Target controlled infusion with propofol, remifentanil and reconium can be used
- Ergometrine avoided. Oxytocin infusion given slowly titrated to response
- Autotransfusion after delivery is treated with frusemide just before delivery of the baby.

Q. What are the considerations about trauma during pregnancy?

Trauma to pregnant women is associated with a high-risk for spontaneous abortion, preterm labor, premature delivery depending on the location and magnitude of maternal injury. Consultation with an obstretrician is desirable for the management of a pregnant trauma patient. The best treatment of

developing fetus consists of rapid and complete resuscitation of the mother. Trauma patients in the first trimester of gestation may not be aware of their pregnancy. Therefore, human chronic gonadotropin testing should be done in any injured woman of childbearing age. Serious trauma, during the period of fetal organogenesis may induce birth defects or miscarriage resulted from hemorragic shock with uterine ischemia, pelvic irradiation or the effects of medications. Indicated radiologic tests should be done after shielding of the pelvis. Parturients who do not spontaneously miscarry should be advised of the potential risks related to trauma and anesthesia. If miscarriage occurs, dilatation and curettage of the uterus is advisable to avoid toxicity from retained products of conception.

Trauma in second or third trimester of pregnancy necessitates early ultrasonographic examination to determine fetal age, size and viability. Monitoring of fetal heart rate is indicated when the pregnancy is sufficiently advanced to attain fetal viability. Preterm labor is very common in traumatized parturients and should be treated with β-agonist agents or magnesium, in consultation with obstretrician, to delay the delivery of the fetus until it is an unacceptable metabolic stress on the mother. Delivery by cesarean is indicated: (a) If uterus is hemorrhaging or (b) Surgical control of abdominal or pelvic hemorrage is impaired by gravid uterus. The Kleihauer-Betke blood test is done to determine if fetal blood has leaked into maternal circulation. (c) if, the anti-Rho immune globulinis administered to any Rh-negative mother carrying an Rh-positive fetus.

By the third trimester, the uterus is sufficiently enlarged to compress the IVC when the mother is in supine position, impairing venous return and leading to hypotension/Left lateral uterine displacement is indicated to treat the problem.

Q. What are the prehospital management of trauma in obstetric patients?

Abdominal injury with bowel injury is more likely than head-injury with trauma in pregnant women. Physiological and anatomical changes during pregnancy result in altered response to trauma. Changes in cardiovascular, pulmonary and gastrointestinal systems are especially relevant. Supine hypotensive syndrome, rapid maternal fetal hypoxia, rapid desaturation during laryngoscopy and intubation, increased risk of aspiration of gastric contents are among the changes during pregnancy, those are of concerns in trauma. Prehospital and ALTS protocol must, therefore, be modified in pregnant women with consideration of assessment of fetal well-being.

Prehospital care: Definite airway management, supplemental oxygen, spinal stabilization when indicated, large bore cannula for IV access, are of primary importance.

Management on arrival (0–5 min): Restrain and immobilization. Patient is rolled to left to avoid supine hypotension.

Initial survey (5–10 min): Focus of initial interventions are ABCDs (Airway, breathing, circulation, disability assessment). Intubation may be difficult and an alternative airway plan is kept ready. Administration of 100% oxygen (mother and fetus tolerate hypoxia very badly). Two large IV cannulae are inserted. Plasma volume is replaced and bleeding controlled. Disability assessment—AVPU is used for neurological assessment: Alert (A), Response to verbal stimuli (V), Response to painful stimuli (P) and Unresponsiveness (U). Pupils are checked for intracranial lesions.

Secondary survey (10–30 min): Physical examination from head-to-toe with frequent ABCDs re-evaluation. History obtained, if possible. NIBP, pulse, ECG and SpO_2 monitoring are done. Effective pain management is advocated.

Cardiographic monitoring beyond 24 weeks: It displays: (a) Continuous Doppler measurement of fetal cardiac activity (b) Continuous electronic recording of uterine activity.

Laboratory investigations:

- Urine for pregnancy test in all trauma patients of childbearing age
- Hematocrit—stat and serial to be done.
- Rh typing done in all cases of major and minor trauma in pregnant women.
- Acid-base status assessment.
- Coagulation studies may be helpful.
- Other tests: Radiology after shielding, CT scan, ultrasonography and diagnostic peritoneal lavage.

Q. What is the anesthetic management in a pregnant trauma patient?

Rapid assessment, hemodynamic stabilization and treatment of maternal injuries are essential for fetal survival. Shock in a pregnant patient may not be clinically evident until 20% to 30% of maternal blood volume is lost due to increased blood volume during pregnancy. At this point, the fetus may be already in jeopardy. In hemorragic shock, maternal blood is shunted away from the uterus to preserve perfusion of vital organs in mother at the cost of the fetus. This physiologic response causes fetal hypoxia and even death.

A preoperative assessment including airway evaluation should be performed. Choice of regional or general anesthetic technique depends on clinical status and psychological condition of the patient, surgical procedure and experience of the anesthesiologist.

Aspiration prophylaxis should be administered to all pregnant patients beyond 14 weeks of gestation because changes at the lower esophageal sphincter during pregnancy along with delayed gastric emptying increases the risk of aspiration of gastric contents. An H_2-antagonist (Ranitidine) should be given 1 hour before surgery, if possible. A non-particulate antacid (0.3 mg sodium citrate) is given just before induction of anesthesia. Use of a prokinetic agent (metoclopramide 10 mg IV) may enhance gastric emptying.

After the second trimester, it is imperative to position the patient with left lateral tilt to avoid aorto-caval compression by ground uterus, by placing a wedge under the right hip.

Monitoring of FHR is possible from 18th weeks onward and should be monitored perioperatively. Alteration in FHR may indicate adverse maternal conditions and such alterations encourage evaluation of maternal oxygenation, hemodynamics, acid-base status and compromise of uterine perfusion resulted from intervention at surgical field. FHR should be documented before and after institution of both regional and general anesthesia and on completion of surgery.

If general anesthesia is necessary, rapid sequence technique with adequate preoxygenetion, cricoid pressure and endotracheal intubation with cuffed ET, should be used to minimize the risk of gastric content aspiration for any pregnant woman after 16 weeks of gestation.

Drug administered should be chosen for their known safety in pregnancy, such as thiopentone depolarizing muscle relaxants, opiods (fentanyl, morphine, meperedine) inhaled agents and 50:50 N_2O:O_2 mixture, maternal $PaCO_2$ should be maintained in the normal range for pregnancy (30 mm Hg) because hyperventilation may reduce placental blood flow. The patient should be extubated until fully awake, because there is still risk of aspiration at the end of the procedure. Uterine activity should be monitored into the postoperative period and tocolytic drugs may be required. NSAIDs should be avoided after first trimester because some of these drugs may constrict or close the PDA in the later stages of pregnancy.

Q. What are the causes of cardiac arrest in pregnancy? What is the management of cardiac arrest during pregnancy?

Causes of cardiac arrest in pregnancy: Failed intubation, esophageal intubation, pulmonary aspiration, airway obstruction, hemorrhage, hypertensive disorders, myocardiac infarction, dysrythmias, idiopathic peripartum cardimyopathy, traumatic myocardial contusion and local anesthetic toxicity.

Contributing factors are:

- 5Hs (Hypovolemia, hypoxia, hydrogen ion (acidosis), hyper/Hypocatemia and hypothermia).
- 5Ts (Tamponade, tension pneumothorax, thrombosis (cardiac) thrombosis (Pulmonary) and toxins.

Management: During resuscitation in pregnant patients, two patients are to be treated—mother and fetus. Fetal monitoring throughout is essential.

Physiological changes during pregnancy make diagnosis and treatment of cardia arrest difficult in pregnant patients. Successful resuscitation of the mother requires prompt and effective CPR with some alterations in basic life support (BLS) and advanced cardiac life support (ACLS) principles.

The BLS Strategy- C-A-B (modification for pregnancy)

Defibrillation and chest compression

During compression (100 compressions/min): Left lateral uterine displacement of uterus is done in supine position with one handed technique from patient's right side or with two handed technique from patient's left side depending on the strength of resuscitation team to prevent supine hypotensive syndrome. Left lateral lift of 30 degrees is attained by placing a Cardiff wedge under right buttock. After 24 weeks of gestation, best survival rate for infant occurs if delivered with 5 min of cardiac arrest. Enlarged breasts hamper effective cardiac compression. As the diaphragm and abdominal contents are elevated by gravid uterus, chest compression should be performed at higher level, i.e. slightly above mid-point of sternum. Defibrillation- for VF and VT with immediate CPR and defibrillation within 3–5 min of cardiac arrest, survival rate is highest, defribrillation energy is 120–200 J for biphasic or 360 J for monophasic defibrillator. Defibrillation shocks have no adverse effect on fetal cardiac function. Fetal and maternal monitors should be removed before delivering any electric shocks.

Airways: A triple maneuver (Head lift, chin lift, jaw thrust) improves airway patency. Only jaw thrust is done in cervical spine surgery. Airway should be secured as early as possible in order to minimize the risk of regurgitation. The airway may be edematous and a small size tube is required. A pregnant patient develops hypoxemia at a quicker rate due to reduced FRC and in increased oxygen requirement. Quick tracheal intubation is needed. Sellicks maneuver (cricord pressure) should be applied during intubation. Tidal volume delivered needs to be reduced due to elevation of the diaphragm.

Breathing: Two breaths for 30 compression (compressions ventilation—30:2). With advanced airway 8–10 breaths/min is applied. Effective oxygenation is vital as pregnant patients develop hypoxemia quickly.

Perimortem cesarean section: It is done in pregnant patients in cardiac arrest if the fetal is potentially viable. As CPR is ineffective in third trimester, emptying of the uterus relieves aortocaval compression and improves venous return and consequently cardiac output. Maternal and fetal outcome are improved by initiation of peripartum cesarean section within 4 min of maternal cardiac arrest. The best chance of fetal survival is to assure maternal survival.

Advanced cardiac life support:

- Inj adrenaline: 1 mg IV energy 3–5 min is given.
- Inj vasopresssin: 40 U may replace 1st and 2nd doses of adrenaline.
  For peripheral channels: 20 mL of fluid bolus after each dose of drug administration.
- Amiodarone: 300 mg IV followed by 150 mg, considered if VF/VT are unresponsive to defibrillation and vasopressors.
- Earliest treatment of reversible (5H and 5T) contributing factors for cardiac arrest.

Post resuscitation:

- Adequate oxygenation (SPO_2>94%) and ventilation ($EtCO_2$ 35–40 mm Hg) ensured.
- Perfusion-SBP >90 mm Hg and MAP >65 mm Hg maintained with fluid administration followed by vasopressors (adrenaline/nonadrenaline, 0.1 to 0.5/kg/min or dopamine—5–10 mg/kg/min.

Chapter

2

Mitral Stenosis Patient Posted for Valve Replacement

Q. What is the etiology of mitral stenosis?

Rheumatic heart disease.

Q. What are the presenting features of mitral stenosis?

- Dyspnea on exertion
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Hemoptysis (in early stage) due to rupture of capillary between pulmonary capillary and bronchial veins.

In late stage as pulmonary hypertension increases, fibrosis of pulmonary capillary occurs and hemoptysis disappears.

Q. What is the pathophysiology in mitral stenosis?

Mitral stenosis → decreases orifice of mitral valve → obstruction to the left ventricular diastolic filling → increases left atrial volume and pressure → increases pulmonary venous pressure → fluid transudation in pulmonary interstitial space → decreases pulmonary compliance and increases work of breathing → progressive dyspnea on exertion.

Q. How mitral stenosis is diagnosed clinically?

- Characteristic opening snap early during diastole
- Rumbling diastolic murmur best heard at the cardiac apex.

Q. What is the cause of opening snap?

Due to vibration set in motion when mobile but stenosed valve initially opens.

Q. What does disappearance of opening snap indicate?

Calcification of the valve making it rigid.

Q. What investigations are done to confirm the diagnosis of mitral stenosis?

- *ECG*
  - Large biphasic P-wave indicating left atrial enlargement without fibrillation
  - Atrial fibrillation.
- *Chest X-ray—AP view:* Straightening of left heart border, widening of carinal angle indicating left atrial enlargement. (Ortner's syndrome—hoarseness due to LA compressing recurrent laryngeal nerve)
- *Lateral view:* Displacement of barium filled esophagus indicating left atrial enlargement.
- *Echocardiography:* Diagnostic tool for assessing the severity of mitral valve stenosis. Normal mitral valve orifice is 4–6 cm^2. Patient becomes symptomatic when decrease in size is 50%. When orifice is 1 cm^2, a mean atrial pressure of 25 mm Hg is required for adequate resting cardiac output.

Wilkin's MV score (2D echo)

- Leaflet mobility
- Leaflet thickness
- Leaflet calcification
- Subvalvular thickness

Score >8–10—severe suboptimal result of PMV.

Echo grouping

- Pliable noncalcified thin cordae >10 mm
- Pliable noncalcified thick cordae <10 mm
- Calcified mitral valve.

Q. When pulmonary hypertension develops?

Pulmonary edema develops when transvalvular pressure gradient is higher than 10 mm Hg (normal <5 mm Hg and there is addition of stress like sepsis, atrial fibrillation, pulmonary embolism and pregnancy.

Q. What is the treatment of mitral stenosis?

- *Medical*
  - Prophylaxis against infective endocarditis.
  - Diuretics: When symptoms of mitral stenosis develop, it decreases atrial pressure and relieves symptoms.
  - If fibrillation develops:
    - Heart rate is controlled by using beta-blocker or calcium channel blocker.
    - Anticoagulant therapy: As there is risk of systemic embolization.
- *Surgical:* Catheter balloon vulvotomy—early stage
  (Criteria: Significant symptoms, isolated MS, no MR, mobile noncalcified valve, left atrial (LA) free of thrombus).

- Open commissurotomy } After
- Valve reconstruction } heavy valvular
- Mitral valve replacement } calcification.

Q. What are the factors increasing pulmonary hypertension?

- Hypoxemia
- Hypercarbia
- Hyperinflation of lung.

Q. What is the treatment of increased pulmonary hypertension and right ventricular failure?

- Dopamine: 3–10 mcg/kg/min IV (inotropic support)
- Nitroprusside: 0.1–0.5 mcg/kg/min IV (pulmonary vasodilatation).

Q. What is the cause of atrial fibrillation?

Increased left atrial pressure → LA enlarges → atrial fibrillation.

Q. What should be the treatment of intraoperative atrial fibrillation?

- Cardioversion starting with 25 watt/sec
- IV esmolol to keep heart rate <110/min.

Q. What is the complication of atrial fibrillation?

Thrombus formation → systemic thrombi → stroke.

Q. What are the presenting features of CHF?

- Dysphagia
- Peripheral edema (pitting)
- Jugular venous distension
- Enlarged tender liver
- Chest X-ray
  - Hilus large with ill-defined margin
  - Kerley's lines (due to septal edema)
    - Upper lobe—Kerley's A line
    - Lower lung field—Kerley's B line
    - Basilar region—Kerley's C line (honey comb pattern)
    - Alveolar edema—butterfly densities.

Q. What is the treatment of CHF?

- ACE inhibitor
- Diuretics
- Peripheral vasodilator
- Digitalis preparation.

Q. What is ejection fraction?

- Stroke volume/end diastolic volume ratio
- Normal: 0.56 to 0.78, i.e. 56% to 78% of its volume during systole.

Q. What anesthetic considerations are given importance for noncardiac surgery?

- Avoid head down position—increases central blood volume
- Avoid sinus tachycardia
- Avoid fluid overloading—precipitate CHF, pulmonary edema, atrial fibrillation.
- Avoid drug-induced—decreases in systemic vascular resistance.
- Avoid arterial hypoxemia and hyperventilation → increases pulmonary hypertension → Right ventricular failure.

Q. Describe the basic circuit of cardiopulmonary bypass (CPB).

Patient → Reservior → filter → oxygenator and heat exchanger → pump → arterial line → patient.

Q. What are the functions of an oxygenator?

Oxygenation of blood and removal of CO_2.

Q. What are the types of oxygenator?

- *Bubble oxygenator:* Disadvantages include hemolysis, platelet destruction and microembolism.
- *Membrane oxygenator:* Microporous membrane. There is transient blood-gas interface.

Q. What is the purpose of a pump in CPB circuit?

The utility of a pump in CPB circuit is for circulation of blood through circuit tubing and back to the patient.

Q. What are the type of pumps in CPB circuit?

- Roller pump
  - Absent pulsatile blood flow→renal dysfunction
  - Increased need for ionotropic and mechanical support
  - Destruction of blood elements.
- Centrifugal pump
  - Less blood trauma
  - Lower line pressure
  - Less chance of massive air embolism.

Q. What are the flow rate and SBP during CPB?

- 50–60 mL/kg/min
- SBP 50–60 mm Hg for adequate vital organ perfusion during CPB.

Q. What is the function of heat exchanger in CPB circuit?

Active cooling and rewarming.

Q. Why systemic hypothermia is used in cardiac surgery?

Systemic hypothermia is used in cardiac surgery for myocardial and neurologic protection.

Q. What is the ideal prime solution and what is the average volume used?

Ringer's lactate is the ideal prime solution.

The average volume required is 1500–2500 mL. Albumin to decrease postoperative edema, mannitol to increase diuresis, calcium to prevent hypocalcemia, corticosteroids, heparin, blood in infant, children and anemic patient. Hematocrit tolerated in CPB is as low as 17%. Higher value is avoided to prevent renal and neurological consequences.

Q. Why anticoagulation is necessary in CPB and when it is done?

Anticoagulation in CPB is necessary to prevent thrombosis of CPB circuit and patient death. Anticoagulation is done prior to insertion of cannulation.

Q. What is the anticoagulation of choice? What is the dose used?

- Heparin is the anticoagulation of choice.
- Dose: 200–400 U/kg.

Q. How adequacy of perfusion is assessed during CPB?

Assessment of arterial pH and mixed venous oxygen saturation.

Q. How adequacy of heparinization is measured?

Adequacy of heparinization is measured by:

- Assessment of activated clotting time (ACT)
- Blood heparin concentration assessment
  ACT >480 sec is acceptable for initiation of CPB.

Q. What is the neutrilization ratio of heparin and protamine?

1 mg of protamine to 100 U of heparin.

Q. What is the method of myocardial protection during CPB?

Myocardial protection is done by:

- Intermittent hyperkalemic cold cardioplegia
- Moderate systemic hypothermia.
  Cold (10–15°C) blood or crystalloid with supranormal concentration of potassium is injected to coronary arteries or veins to induce diastolic electrical arrest.

Q. What are the stressful conditions during CPB?

Intubation, incision, sternotomy, pericardiotomy and manipulation of aorta.

Q. How the lung is treated during CPB?

- No need for ventilation
- Some disconnect the patient from anesthesia machine
- Others inflate the lung with low level of PEEP with 100% oxygen.

Q. How air from pulmonary veins is removed at the end of CPB?

By vigorously inflating the lungs.

Q. What are the prerequisites for separation from CPB?

- Patient should be normothermic
- Surgical field should be dry
- Appropriate laboratory values
- Adequate pulmonary compliance.

Q. How blood conservation is done during cardiac surgery?

- Intraoperative autologous hemodilution is a method of removing whole blood from patient prior to systemic heparinization and coagulopathy from CPB and administering crystalloid or colloid fluids to make up for the circulating volume removed. Following CPB, the blood is transfused returning the blood cells, active platelets and coagulation factors.
- Intraoperative blood salvage is used for blood conservation in cardiac surgery. Blood suctioned from surgical field is anticoagulated, filtered, and reinfused or use of cell washing which is composed of four steps:
  - Harvesting of patient's blood
  - Processing of shed blood
  - Removal of serum and storage of red cells
  - Reinfusion.
- *Administration of antifibrinolytic agents:* New techniques used to reduce need for homologous blood include administration of aminocaproic acid, tranexamic acid, aprotinin, desmopressin, ultrafiltration, blood fractionization and use of improved topical hemostatic agents. Antifibrinolytic agents like aprotinin and aminocaproic acid decreases bleeding after CPB and reduces risk of blood transfusion.
- *Retrograde autologous priming (RAP)*: Crystalloid prime contained within arterial and venous lines is drained into a recirculation bag prior to initiation of CPB and replaced by blood drained retrograde via the arterial cannula. This reduces hemodilution and drop in systemic vascular resistance associated with initiation of CPB.
- *Ultrafiltration:* Used to reduce postoperative bleeding and need for transfusion. During ultrafiltration/hemoconcentration, plasma water is separated from low molecular weight solute, transcellular cell components and plasma protein using a semipermeable membrane. It is initiated

during rewarming based on the volume within CPB circuit and exogenous fluid given. This by employing hemoconcentration levels of formed blood element, including erythrocytes, platelets and coagulation factors are augmented. There is increase in hemoglobin and hematocrit and decrease in postoperative bleeding and need for transfusion.

- *Blood fractionation:* Blood elements are removed and made into a concentrate that is removed from the patient pre-bypass. These then are returned to patient following CPB, avoiding dilutional coagulopathy and activation of blood elements while on CPB.
- *Blood substitutes:* Hemoglobin based compounds made from blood in which hemoglobin is chemically extracted for its oxygen carrying capacity. Self stable product, devoid of infectious complications of transfusion.

Q. Why transesophageal echocardiography (TEE) is useful during CPB?

Transesophageal echocardiography is an invaluable diagnostic and monitoring tool during cardiac surgery. TEE offers distinct advantages over other monitors of CV function and provides the anesthesiologist with unique diagnostic information. TEE permits assessment of ventricular volume, global and regional functions, estimation and quantitation of valvular pathology, measurement of valve gradient and calculation of filling pressure, visualization of thoracic aorta and detection of intracardiac air.

TEE is the only intraoperative monitor that provides information on function and structure of mitral, aortic, tricuspid and pulmonary valve. It is used to determine the etiology of acute hypotension in perioperative period. The early detection of the causes of hemodynamic instability allows appropriate therapy to be instituted (volume expansion, ionotropes, vasopressors).

Other applications specific to cardiac surgical patient are also useful. Following CPB, ventricular filling pressure, irrespective of site of measurement is a poor and often misleading indicator of ventricular volume status. Direct estimation of left ventricular volume with 2D TEE, more appropriately direct fluid infusion and selection of vasoactive drugs in patients who are difficult to wean from bypass. In addition, residual valve lesions, intracardiac air or new area of ischemia are readily identified. Global dysfunction suggesting residual cross-clamp effect, inadequate cardioplegia or reperfusion injury can be detected.

Q. What are the types and classification of prosthetic heart valve?

There are two types:

1. Bioprosthetic valve
2. Mechanical valve.

Bioprosthetic valve: Structurally resembles native valves.

- *Heterografts:* Composed of porcine or bovine tissues mounted on metal supports.
- *Aortic homografts:* Cryopreserved human aortic valves.

- *Autologous:* Pulmonary autograft for aortic valve replacement pericardial autografts.

These valves have low thrombogenic potential, long-term anticoagulation is not required in these patients. These valves often fail within 10–15 years and require replacement. These are preffered in patients who are elderly or who cannot take long-term anticoagulation medication.

Mechanical valves: Composed primarily of metal or carbon alloys.

Prosthetic heart valves are classified as:

- Single leaflet tilting disc valves
- Bileaflet tilting disc valves—most widely used
- Caged-ball valves.

Mechanical valves are highly durable, most lasting at least 20–30 years. These valves are thrombogenic so require long-term anticoagulation therapy. These valves are preffered for patients who are young or have life expectancy of more than 10–15 years and for those who require long-term anticoagulation therapy for reasons as atrial fibrillation.

Q. What are the criteria affecting valve selection?

- Age (>70 years)—Bioprosthetic valve:
- Bleeding diathesis—Bioprosthetic valve
- High risk of trauma—Bioprosthetic valve
- Poor patient compliance—bioprosthetic valve
- High-risk of endocarditis—Homograft preffered
- Multiple valve replacement—Mechanical valves
- Patients on chronic anticoagulations—Mechanical valves (atrial fibrillation)
- Females considering pregnancy—Homografts/mechanical valve.

Q. What are the complications peculiar to the prosthetic heart valves?

- Structural failure
- Valve obstruction
- Systemic embolization
- Bleeding
- Infection
- Hemolytic anemia.

Q. What are the preoperative assessments of a patient with a prosthetic heart valve?

- Evaluation of valve function
  - History
  - Physical examination
  - Echo-Doppler studies
- Anticoagulation
- Endocarditis prophylaxis

- Safety exercises: Trauma should be discouraged in patients on anticoagulation.
- Anticoagulation: Recommended in many patients with prosthetic heart valves to prevent valve thrombosis and thromboembolic events.

(ACC/AHA task force guidelines, 1998)

- For all patients with valve replacement unfractioned or low molecular weight heparin is used until international normalized ratio (INR) is stable with warfarin therapy.
- First three months after valve replacement, use warfarin to achieve target INR of 3
- More than 3 months after valve replacement:
  - Mechanical valves:
    - Aortic valve replacement with no risk factors—target INR 3
    - AVR with risk factor—target INR 3
    - Mitral valve replacement—target INR 3
  - Bioprosthetic valves:
    - AVR with risk factors—warfarin, target INR 2.5
    - AVR with no risk factors—aspirin, 80–100 mg /day
    - MVR with risk factors—warfarin, target INR 3
    - MVR with no risk factors—aspirin 80–100 mg/day.

Q. What are the methods of discontinuing anticoagulation for surgical procedure?

- For procedures with low-risk of bleeding, oral anticoagulation can be continued with gradual lowering of INR to 2
- Patients with low-risk factors:
  - For minor surgery, INR should not exceed normal range. Anticoagulation can be continued in most patients. If required, warfarin can be stopped 2 days prior to surgery and reinstituted after surgery.
  - For major surgery, anticoagulation can safely be stopped 3–4 days before surgery. After normal INR is obtained, surgery is performed and anticoagulation instituted after surgery.
- Patients with high-risk factors:
  - Minor surgery: Oral anticoagulation continued
  - Major surgery: Warfarin discontinued 3–5 days prior to surgery.

  IV heparin or LMWH is indicated when INR falls below the target range. Dose of heparin is adjusted to achieve an activated PTT of 2 times control.
- Heparin discontinued 4 hours and LMWH discontinued 12 hours prior to surgery.
- Heparin or LMWH restarted as soon as possible after surgery
- Warfarin reinstituted soon after surgery and heparin or LMWH discontinued once INR is in the therapeutic range.
- Endocarditis prophylaxis: Patients with prosthetic heart valves are at high-risk for endocarditis & should use prophylactic antibiotics.

Chapter

3

A Diabetic Woman Posted for Total Abdominal Hysterectomy

Q. What is diabetes mellitus?

Diabetes mellitus is dysregulation of glucose metabolism accompanied by long-term vascular and neurologic complications.

Q. What is the classification of diabetes mellitus?

- Insulin-dependent diabetes mellitus (IDDM)/Type 1.
- Noninsulin dependent diabetes mellitus (NIDDM)/Type II.
- Gestational diabetes mellitus (pregnancy).
- Secondary diabetes mellitus (pancreatic destruction, acromegaly, Cushing's disease, drugs like glucocorticoids).

Q. What is gestational diabetes mellitus?

Glucose intolerance with usual onset at 24–30 weeks of gestation and corrects after delivery. Maternal hyperglycemia increases fetal morbidity including congenital anomalies and stillbirth.

Q. How IDDM and NIDDM are differentiated?

IDDM

- Absolute insulin deficiency years
- Usual onset in youth (20 years of age)
- Ketosis prone
- Anti-islet cell antibodies

NIDDM

- Insulin resistance often in presence of adequate insulin secretion
- Usual onset after 40 years of age
- Ketosis resistant
- Obese.

Q. What are the metabolic abnormalities due to insulin deficiency?

- Hyperglycemia (glycogenolysis, gluconeogenesis and decreased skeletal muscle uptake of glucose)
- Increased serum fatty acid concentration (lipolysis)
- Increased serum ketone concentration (hepatic ketogenesis)
- Decreased protein synthesis
- Dehydration (glucose acting as osmotic diuretic).

Q. What are the diagnostic criteria for diabetes mellitus?

- Polyuria
- Polydipsia
- Weight loss
- Hyperglycemia—According to ADA 2014 guidelines
  - HbA1c >6.5
  - FBS >126 mg/dL
  - PPBS >200 mg/dL
  - RBS >200 mg/dL
- Abnormal glucose tolerance test.

Q. What is oral glucose tolerance test?

- Unrestricted carbohydrate diet for 3 days before test
- Fasted overnight (for at least 8 hours)
- Rest before test (30 min)
- Plasma glucose measured before and 2 hours after 75 g of oral glucose.

Diabetes impaired glucose tolerance test:

Fasting >110 mg /dL. Fasting < 110 mg/dL.
2 hours after glucose load >200 mg/dL 2 hours after glucose load—140–199 mg/dL.

Q. What does glycosylated hemoglobin indicate?

It indicates the mean blood glucose concentration over 60 days. It is the most accurate objective assessment of long-term blood glucose control and efficacy of therapeutic interventions.

- Non-diabetic range <6.5%.
- Goal of intensive therapy in patient with IDDM is to maintain range at <6.5% (ADA, 2014).

Q. What is the treatment of IDDM?

IDDM is characterized by an absolute insulin deficiency and so these group of patients depend on exogenous insulin for survival. They require self-monitoring and lifestyle adaptations including diet and exercise. The intensive exogenous insulin therapy is intended to maintain normal blood glucose concentration. HbAic level <6.5% decreases the development and progression of long-term diabetic complications. The disadvantage of more

intensive control of hyperglycemia, increases incidence of hypoglycemia. During pregnancy, management is particularly difficult because of changing insulin requirements.

Pancreatic transplant restores normal glucose metabolism. These patients do not require insulin to compensate stress response to surgery.

Q. What are the types of insulin?

- *Rapid acting (crystalline zinc insulin):* Subcutaneous injection does not produce a sharp peak. Must be administered 30–60 minutes before meal. Onset 0.5–1 hour; duration 6–8 hours.
- *Very rapid acting (lispro):* As a recombinant human insulin is more rapidly absorbed. Administered 10–15 mins before meal.
  Onset—0.25–0.5 hours; Duration: 4–6 hours.
- *Intermediate-acting (Lente and NPH):* Onset: 2–4 hours; Duration: 10–14 hours.
- *Long-acting (ultralente):* Because of long half-life, a new steady state is not achieved for 3–4 days after a change of dose. Onset: 8–14 hours; Duration: 18–24 hours.

Q. What is the treatment of NIDDM?

Clinical goals of therapy for NIDDM are same as for IDDM. Many NIDDM patients can be treated with diet and if successful, decreases CV risk factors and improve glucose tolerance. Diet is the most important treatment of NIDDM with weight loss in presence of obesity. Monitoring is less demanding as glucose profiles are relatively stable and risk of hypoglycemia is less. Risk of Ketoacidosis is low. Measurement of fasting blood glucose and HbA1c every 3–6 months is adequate. Hypoglycemic medications may be necessary in NIDDM patients when dietary therapy fails.

Q. What are the hypoglycemic medications?

Hypoglycemic medication is necessary in NIDDM patients when dietary therapy fails. Drug therapy is more effective when attention to diet is continued.

Oral medications are:

- *Sulfonylureas:* Acts by stimulating endogenous insulin secreation. It decreases insulin resistance and lowers HbA1c. Hypoglycemia is infrequent but when occurs it is more prolonged and dangerous than hypoglycemia secondary to insulin.
  Clinically useful sulfonylureas:
  - Glyburide
  - Glipizide
  - Glimepiride
  - Tolbutamide
  - Acetohexamide
  - Chlorpropamide—longest acting.

- *Biguanides:* Act by decreasing hyperglycemia (inhibit gluconeogenesis in liver and kidney) and increasing glucose uptake into skeletal muscle with low-risk of hypoglycemia.
  - Phenformin
  - Metformin—has a lower risk of causing lactic acidosis.
- *Meglitinides*—action is same as sulfonylureas with more prompt peak effect (1 hour) and shorter duration of action (4 hour).
- *Alpha-glycoside inhibitors:* Acarbose and miglitol decrease absorption of carbohydrate leading to blunted glycemic response during postprandial period.
- *Glucagon like peptide 1 receptor agonist (incretins):* Exenatide, liraglutide.
- *DPP4 inhibitors:* Gliptins (Dipeptidyl peptidase 4 inhibitor)
- *Peroxisome proliferator-activated receptor (PPAR) agonist.*

Q. Why combination therapy of oral hyperglycemic drugs is required?

In patient with persistent hyperglycemia, none of the oral hypoglycemic drugs can normalize HbA1c when used as monotherapy, because they act principally by correcting single abnormality. Combination therapy supports the logic of targeting two or more different causes of hyperglycemia simultaneously. On approach to decrease insulin resistance with metformin, while increasing insulin secretion with sulfonylureas or meglitinide. Exogenous insulin may also be a part of combination therapy. The primary aims of combination therapy are to decrease HbA1c and the secondary benefit is reduction in daily insulin dose.

Q. What are the complications of diabetes mellitus?

- Ketoacidosis
- Hyperosmolar hyperglycemic nonketotic coma
- Hypoglycemia
- Cardiovascular disease
- Peripheral neuropathy
- ANS neuropathy
- Nephropathy
- Retinopathy
- Stiff joint syndrome
- Diabetic scleroderma.

Q. What are the macrovascular events?

- Coronary artery disease
- Cerebrovascular disease
- Peripheral vascular disease.

Q. What are microvascular events?

- Retinopathy
- Nephropathy.

Q. What are the ophthalmic findings in diabetes mellitus?

- Microaneurysms
- Retinal hemorrhage
- Exudates
- Cotton wool spots
- Venous changes
- Neovascularization
- Preretinal hemorrhage
- Vitreous hemorrhage.

Q. What are the manifestations of autonomic neuropathy?

- Resting tachycardia
- Orthostatic hypotension
- Absent beat-to-beat variation in heart rate with deep breathing
- Cardiac dysarrhythmias (QT abnormalities)
- Sudden death syndrome
- Gastroparesis (vomiting, diarrhea, abdominal distension)—chances of aspiration during induction.

Q. What is sudden death syndrome?

Sudden unexpected, profound bradycardia in the perioperative period in a patient with autonomic neuropathy which is only responsive to IV epinephrine.

Q. Why autonomic neuropathy is most dangerous complication of diabetes mellitus?

Because autonomic neuropathy is associated with an increased incidence of postoperative cardiorespiratory arrest.

Q. What is hypoglycemic unawareness?

Absence of sympathetic nervous system responses (tachycardia, diaphoresis) to hypoglycemia (blood glucose level <50 mg%). It reflects autonomic neuropathy.

Q. What are the ophthalmic changes in autonomic neuropathy?

- Decreased pupil size
- Resistance to mydiatrics
- Decreased or absent reflexes to light.

Q. How assessment of autonomic nervous system done?

- Parasympathetic nervous system
  - Heart rate response to standing
  - Heart rate response to deep breathing
  - Heart rate responses to valsalva.

- Sympathetic nervous system
  - Blood pressure response to standing
  - Blood pressure response to sustained hand grip.

Heart rate response on standing: HR is measured as the patient changes from supine to standing position (increases maximum around 15th beat after standing and slowing maximum around 30th beat). Normal value: Ratio >1.04.

Heart rate response to deep breathing: Patient takes 6 deep breaths in 1 minute. The maximum and minimum HR during each cycle are measured and the mean of the differences (maximum HR – minimum HR) during three successive cycles is taken as the maximum – minimum HR. Normal value—mean difference >15 bps.

Heart rate response to valsalva: Patient blows into a mouth piece maintaining a pressure of 40 mm Hg for 15 seconds. The valsalva ratio is the ratio of the longest R-R interval on the electrocardiogram immediately after release, to the shortest R-R interval during the maneuver. Normal value—Ratio >1.21.

Blood pressure response to standing: Patient changes from supine to standing position and the standing systolic blood pressure is subtracted from the supine blood pressure. Normal value: difference <10 mm Hg.

Blood pressure response to sustained hand grip: Patient maintains a handgrip of 30% maximum squeeze for up to 5 minutes. The blood pressure is measured every 5 minute and the initial diastolic pressure is subtracted from the diastolic blood pressure just prior to release. Normal value—difference >16 mm Hg.

Q. What is classification of peripheral neuropathy?

- *Distal symmetric polyneuropathy:* Most common form of diabetic peripheral neuropathy. Loss of function appears in a stocking–glove pattern. Both sensory and sensory nerve conduction are delayed in peripheral nerve and ankle jerk may be absent. Larger nerves are vulnerable and so impact is on foot.
- *Isolated peripheral neuropathy:* Involvement of distribution of only one nerve (mononeuropathy) or several nerves (mononeuropathy multiplex). Sudden onset with subsequent recovery of all or most of the function. Cranial and femoral nerves are commonly involved and motor abnormalities predominate.
- *Painful diabetic neuropathy:* Hypersensitivity to light touch. Occassionaly severe burning pain at night.

Q. How diabetic foot ulcers are caused?

A combination of insensitive foot due to peripheral neuropathy and decreased blood supply due to peripheral vascular disease leads to foot ulceration, particularly at the site of pressure.

Q. How foot examination is done in a diabetic patient?

The feet of diabetic patients should be examined once in a year. Signs of deformity, callus, fungal infections between toes, nail care and ulceration should be carefully assessed. Peripheral pulses and nailfold refill should be assessed for signs of peripheral vascular disease. Nerve function should be assessed by testing vibration sense at the great toe, medial malleolus and knee. Fine touch is tested on toes, metatarsals heads, heels and dorsum of feet with a 10 g monofilament. Loss of ankle is also a sign of early diabetic peripheral sensory neuropathy. Charcot neuroarthropathy, a complication of diabetic peripheral neuropathy, usually affects ankle presenting with painless, swollen, hot red joint. When untreated, severe joint deformity develops putting the foot at high-risk of ulceration.

Q. What are the presenting features of diabetic ketoacidocis?

- Nausea and vomiting
- Dehydration
- Somnolence/coma
- Kussmaul's breathing (rapid, deep, sighing respiration—respiratory compensation for metabolic acidosis).
- Abdominal pain
- Ileus
- Hypotension
- Tachycardia.

Q. What are laboratory abnormalities in diabetic ketoacidosis?

- Hyperglycemia
- Metabolic acidosis (arterial pH <7.3, bicarbonate <15 meq/L)
- Ketones present in urine and blood
- Hyperosmolarity
- Hypokalemia
- Increased blood urea nitrogen and/or creatinine concentration.

Q. What is the treatment of diabetic ketoacidocis?

- Tracheal intubation if CNS depression is present
- Intravenous regular insulin (10 units followed by 5–10 units/hr)
- Restoration of intravenous fluid volume (normal saline 10 mL/kg/hr IV when blood glucose decreases to <250 mg/dL, 5% glucose is added)
- Restoration of total body potassium (0.3–0.5 meq/kg/hr) IV
- Blood glucose, serum K^+, arterial pH and urine ketones are monitored
- Cause is identified (sepsis, MI, poor compliance).

Q. What are the presenting features of hypoglycemia?

More common than ketoacidosis and potentially dangerous. Signs and symptoms of hypoglycemia reflect:

- Activation of sympathetic nervous system—diaphoresis, tremulousness and tachycardia.
- Neuroglycopenia due to effects of insufficient glucose delivery to brain—impaired cognition, confusion, headache. Irritability, retrograde amnesia, seizures and unconsciousness.

Q. How hypoglycemia is managed?

Oral ingestion of rapidly absorbed carbohydrates (15 g is sufficient). If patient cannot swallow 25 mL of 50% glucose is administered IV.

Q. What are the presenting features of hyperosmolar, hyperglycemic and nonketotic coma?

- Severe hyperglycaemia (>600 mg/dL)
- Hyperosmolality (>350 mOsm/L)
- Severe volume depletion (fluid deficit is 25% of total body water)
- Normal pH—no ketoacidosis
- Central nervous system depression (somnolence, coma)
- Osmotic dieresis (hypokalemia).

Q. What are the precipitating factors of hyperosmolar, hyperglycemic, nonketotic coma?

- Advanced age
- Sepsis
- Hyperalimentation using concentrated carbohydrate solution
- Drugs—corticosteroids.

Q. What is the treatment of hyperosmolar, hyperglycemic, nonketotic coma?

- Administration of hypotonic saline to correct hypovolemia and hyperosmolality.
- A blow dose intravenous insulin infusion to lower blood glucose level less than 300 mg/dL. Then exogenous insulin is not required.
- Potassium supplementation required to replace loss due to osmotic dieresis.

Q. How will you investigate a diabetic patient for surgery?

- Hemoglobin—anemia present with renal dysfunction. Baseline guide for intraoperative blood transfusion
- CBC—suggestive for infections
- Urine for microalbuminuria—nephropathy
- Serum creatinine—to detect renal dysfunction
- Fasting and postprandial blood glucose
- Glycosylated Hb: HbA1c of <7% implies good sugar control
- Serum electrolytes: Serum potassium level in patients on insulin

- ECG: To detect asymptomatic myocardial ischemia
- X-ray chest: Tuberculosis is common in diabetics.

Investigation on morning of surgery—serum electrolytes, fasting blood sugar, urine ketones.

Q. What are the principles of anesthetic management in a patient with diabetes mellitus?

- *Timing:* Diabetic patients should be placed first on the operation list. This shortens the preoperative fast and the risk of hypoglycemia and ketosis.
- *Fasting:* Delayed gastric emptying due to diabetic autonomic neuropathy is found in these patients. Undiagnosed gastroparesis prolongs retention of food in the stomach and increases the risk of regurgitation and aspiration. A 12 hour fasting may be beneficial.
- *Hydration:* Ringer's lactate solution is avoided, as lactate is converted to glucose.
- *Monitoring:* Frequent, rapid and accurate blood glucose measurement is essential in anesthetized patients as the requirements of glucose and insulin in this period are unpredictable and hypoglycemia may go undetected. Standard monitoring: ECG, SpO_2, BP, $EtCO_2$ and temperature should be instituted. Advanced monitoring is guided by presence of comorbidities.
- *Glycosylated hemoglobin (HBA1c)* measurement has no value in intra-operative or postoperative period but is a valuable guide to long-term glycemic control during preoperative evaluation.
- *Sugar control:* Postoperative wound healing and infection are influenced by adequacy of perioperative glycemic control. Diabetic patients receiving long-acting insulin are at risk of hypoglycemia if glucose is not supplemented.

  Glucose supplementation is required to safeguard against inadvertent hypoglycemia, excessive catabolism and starvation ketosis.
- *Anesthesia:*
  - Choice of induction agent depends on severity of systemic diseases.
  - A rapid sequence induction performed in patients with gastroparesis. Succinylcholine avoided in extensive peripheral neuropathy (risk of potassium release). Atracurium and mivacurium are preferred in presence of renal dysfunction. Rocuronium may be used for rapid sequence induction.
  - Maintenance of anesthesia with isoflurane or sevoflurane in air oxygen mixture. N_2O may be used.
  - Less severe cases can be reversed and extubated at the end of surgery. Patient should have adequate recovery of airway reflexes prior to extubation.
  - Bradycardia and hypotension develop suddenly in patients with ANS neuropathy and are unresponsive to atropine/ephedrine. IV epinephrine is more effective.

Q. How you will manage perioperative blood sugar control?

Classic control: Measurement of blood glucose concentration every 1–2 hours during major surgery and in the postoperative period.

Blood glucose concentration is maintained between 100 mg/dL and 200 mg/dL by IV administration of regular insulin. Aim is to prevent hypoglycemia, ketoacidocis and hyperosmolar state.

50 units of regular insulin is mixed in 500 ml normal saline (1 unit/hr = 10 mL/hr).

IV infusion is initiated at 0.5–1 unit/hr.

Blood glucose concentration is measured every 1–2 hours and infusion rate adjusted accordingly.

- <80 mg/dl IV infusion is turned off for 30 minutes.
  25 mL of 50% glucose is administered.
  Blood glucose level assessed in 30 minutes.
- 80–120 mg%—decrease insulin infusion rate by 0.3 units/hr.
- 120–180 mg/dL—no change of insulin infusion rate.
- 180–220 mg/dL—increase insulin infusion rate by 0.3 units/hr.
- >220 mg/dL—increase insulin infusion rate by 0.5 unit/hr.

Sufficient glucose (5–10 g/hr—5% dextrose at 125 mL/hr/70 kg) and potassium (2–4 meq/L) are provided throughout operative period.

Q. What is tight control regimen?

Aim is to keep glucose level at 80–120 mg/dL to prevent wound infections and improve wound healing, neurological outcome after CNS ischemic insult and weaning from CPB.

On the evening before surgery, blood glucose level is estimated.

Intravenous infusion of 5% dextrose is started at 50 mL/hr/70 kg.

Infusion of regular insulin (50 units in 250 mL of normal saline solution) is piggybacked to dextrose infusion with an infusion pump.

Infusion rate of insulin is set using following equation:
Insulin (u/hr) = plasma glucose (mg/dL)/150.

- Denominator should be 100 if the patient is taking corticosteroids .
- Blood glucose measurement is repeated every 4 hours and insulin infusion rate is adjusted to obtain blood glucose level 100 to 200 mg/dL.
- On the day of surgery, fluid and electrolytes are managed by administration of nonglucose containing fluid.
- Blood glucose level is determined at the start of surgery and every 1–2 hours for the rest of 24 hours.
- Insulin dose is adjusted appropriately.

Q. How preoperative optimization is done in a patient with diabetes mellitus?

- These patients should be scheduled for surgery early in the day to limit duration of preoperative fasting.

- Well-controlled diet treated NIDDM patients do not require hospitalization.
- Well-controlled IDDM patients scheduled for brief outpatient surgery do not require any adjustment in usual s/c insulin regime.
- Oral hypoglycemic drugs are continued until the evening before surgery except metformin, which is discontinued 2 days before surgery (possibility of formation of lactic acidosis).
- Preadmission to the hospital is indicated in patients with poorly controlled IDDM.
- Evaluation and treatment of hypoglycemia, electrolyte disturbance and ketoacidosis are done before elective surgery.
- HbA1c concentration of <7.5% suggests adequate blood glucose control.
- Evidence of IHD, CV disease and renal dysfunction should be sought, in patients with IDDM.
- Review of ECG and search for proteinuria should be done.
- Signs of peripheral neuropathy and ANS neuropathy are noted.
- Patients with ANS dysfunction are at increased risk of aspiration during induction and are very prone to develop intraoperative cardiovascular lability.
- Evaluation of evidences of limited joint mobility is done to predict difficult laryngoscopy and intubation in patients with IDDM.
- Presence of scleredema should be sought to predict difficult laryngoscopy and intubation as well as IV cannulation.
- Presence of obesity in this patient population makes tracheal intubation and performance of regional anesthesia difficult.

Q. What airway problems are expected? Why?

Difficult neck extention, laryngoscopy and tracheal intubation are expected.

Causes:

- Stiff joint syndrome involving atlanto-occipital joint due to glycosylation of tissue protein.
- Diabetic scleredema causing thickening and hardening of skin over back of the neck, shoulder and upper back.
- Associated obesity in this patient population influences ease of tracheal intubation.

Q. What tests are done to predict airway difficulty?

- **Prayer sign:** Inability to approximate the palmer surfaces of the interpharangeal joints.
- **Palm print test:** A palm print is taken after painting the palm and fingers with ink.

Scoring

Grade 0—all phalangeal areas visible.

Grade 1—deficiency in interphalangeal areas of 2nd to 5th digit.

Grade 2—deficiency in interphalangeal areas of 2nd to 5th digit.

Grade 3—only the tips of the digits seen.

Q. What factors influence the use of regional anesthesia in a diabetic patient?

- Local anestheia requirements are lower and risk of nerve injury is higher in diabetic patients combining local anesthetics with epinephrine poses a greater risk for ischemic or exdematous nerve injury in diabetes.
- Increased incidence of peripheral neuropathies is a consideration when choosing regional anesthesia technique in diabetic patients.
- Systematic absorbption of local anesthetics, such as following brachial plexus block is associated with myocardial drpression in diabetics.
- Episodes of bradycardia or hypertension develop suddenly in diabetic patients with ANS dysfunction and are unresponsive to atropine or ephedrine. Considerations should be given to this when choosing spinal and epidural techniques in diabetic patients.
- In diabetic scleredema, due to deposition of collagen in epidural space, marked increase in epidural pressure occurs during epidural anesthesia. This may result in anterior spinal artery syndrome by restricting arterial blood supply to spinal cord.

Q. What are the risks to mother and baby due to gestational diabetes mellitus?

- *Risks to mother:* An increased incidence of preeclampsia, polyhydramnios and operative delivery, increased risk for developing hypertension, hyperlipidemia and ECG abnormalities.
  - Risk of developing diabetes in later life.
  - Mortality rate is also higher.
- *Risks to baby:* Increased risk of perinatal morbidity and mortality is larger for gestational age and increases shoulder dystosia and brachial plexus injury is common with macrosomia.
  Risk of neonatal hypoglycemia can be reduced by good maternal glycemic control during labor and birth and early feeding of the baby.
  Neurodevelopment of the fetus may be hampered. Risk of baby developing obesity and/or diabetes in later life.

Q. Why does the fetus develop macrosomia in a diabetic mother?

Maternal hyperglycemia leads to fetal hypergycemia and results in increased fetal growth particularly of fact and liver which are insulin sensitive.

Q. What special care is needed for babies born of diabetic mother?

- Major concern is hypoglycemia.
- Babies should be fed as soon as possible within 30 minutes & at 2–3 hourly interval.
- Babies with low blood sugar (<36 mg%) or clinical signs of hypoglycemia should receive IV dextrose.
- Blood glucose testing should be carried out to assess blood sugar level.

- Blood test for polycythemia, hyperbilirubinemia, hypocalcemia and hypomagnesemia should be carried out in babies with clinical signs of hypoglycemia.

Q. How does open heart surgery influence blood sugar control?

Insulin requirements are increased with cardiopulmonary bypass. The reason is insulin resistance caused by hypothermia, infusion of dead space of bypass machine with glucose solution and the hyperglycemia effects of adrenergic drugs.

Hyperglycemia and hyperosmolar coma are known in patients undergoing cardiac surgery and carries high mortality.

Tight sugar control has been shown to reduce risk of postoperative infection.

Chapter

4

A Kidney Transplanted Patient Put for Nontransplant Surgery

Q. What is the purpose of organ transplantation?

Organ transplantation is the treatment of choice for end stage organ failure from any of the wide variety of causes.

Q. What is meant by transplantation? How is it categorized?

Transplantation is the act of transferring organ, tissue or cell from one site to another. Transplantations are divided into three categories:

1. *Autotransplants:* Transplant of organ or tissue from one part of an individual to other part of the same individual. (Skin graft, vein graft for CABG). No immunosuppression is required.
2. *Allotransplant:* Transfer of solid organ from one individual to another individual of same species. Immunosuppression of the recipient is required to prevent graft rejection.
3. *Xenotransplants:* Involves transfer across species, barriers. Graft is taken from other species. Relegated to laboratory. Complex and potent immunological barrier needed.

Q. Why graft rejection occurs?

Graft rejection is due to an immune response in the host to foreign alloantigen after organ transplant from genetically non-identical donors.

It is a complex process involving T lymphocytes, B lymphocytes, macrophages, and cytokines with resultant local inflammatory injury and graft damage.

In humans, the main antigen involved in triggering rejection is the human leukocyte antigen HLA system. The HLA system initiates rejection and graft damage through humoral or cellular mechanisms. Cellular rejection is more common after organ transplants and mediated by T lymphocytes. Graft rejection reactions have various time courses:

- *Hyperacute:* Within minutes after transplanted organ is reperfused.
- *Acute:* Most common and occurs within days to a few months post-transplant, becoming less with moderate immunosuppression.
- *Chronic:* Occurs within months to years post-transplant.

Q. What specific measures are followed during laparoscopic procedures in a pregnant patient?

Concerns of laparoscopy during pregnancy:
- Risk of premature labor or miscarriage
- Risk of damaging gravid uterus.

Recommendations for safe laparoscopic procedure in pregnant patients:
- The operation should be done during second trimester, ideally before 23rd week of pregnancy to minimize the risk of miscarriage or preterm labor and maintain adequate intra-abdominal working room.
- Tocolytics are beneficial to arrest preterm labor.
- Uterine fundus reaches umbilicus at 20 weeks, which should be taken into consideration during abdominal access for creation of pneumoperitoneum. Direct peritoneal access (Hasson's technique) should be used to avoid damage to uterus or its blood supply.
- Pregnant patient should be positioned with left lateral tilt to avoid aortocaval syndrome.
- Pregnancy possesses risk of thromboembolism, so sequential compression devices are essential.
- Fetal monitoring should be performed using transvaginal ultrasound.
- Mechanical ventilation must be adjusted to maintain normal $EtCO_2$. Gasless laparoscopy is an alternative to avoid potential side effects of CO_2 pneumoperitoneum.

Q. How Graft rejection is prevented?

Graft rejection is prevented by immunosuppression. Immunosuppressive drugs are used to diminish immunological attack on graft and target T cell activation. Immunosuppressive drugs used for prevention and treatment of acute and chronic rejection of transplanted organ therapy must be maintained lifelong.

Success of transplantation is largely due to effective immunosuppression. A typical regimen consists of different phases:
- *Induction:* Drugs administered immediately post-transplant to induce immunosupression. Biologic agents consisting of antibody preparations that are directed at various cells or receptors involved in rejection process are commonly used in induction.
- *Maintenance:* Drugs administered to maintain immunosuppression after recovery of the recipient from operative procedure. Nonbiological agents form the mainstay of maintenance protocol.

Nonbiological agents are: Corticosteroids, azathioprine, cyclosporine, tacrolimus, sirolimus and mycophenolate mofelil.
Biological agents are: Polyclonal antibodies, monoclonal antibodies.

Q. What are the categories of organ donors?

Two categories: (1) Deceased donors and (2) living donors

1. *Deceased donors:* These donors are deceased individuals meeting the criteria of brain death whose organs are being perfused by life support measures till the procurement of the organ.
2. *Living donors:* These donors are generally very healthy and almost exclusively classified as ASA 1 or 2 patients. The surgeon generally operates on the individual to procure the organ. These donors should undergo an extensive medical, psychological and social evaluation. For renal transplant, laparoscopic live donor nephrectomy has completely replaced the traditional open approach. A major concern about living organ donation is the potential for harm to the entirely healthy individual who undergoes major surgery for the interest of other.

Q. Why and how organ preservation is done after procurement?

Organ harvesting and transplantation cause organ injury to the donor organ during three main periods:

1. Storage of recovered organ during transport
2. Implantation phase
3. Initial reperfusion phase with reintroduction of oxygen.

Hence, organ preservation is essential.

Most common method of preservation involves the use of hypothermia and pharmacological inhibition to slow down metabolic process in the organ after removal from the donor. Cold solutions have been developed to improve organ preservation by decreasing the detrimental effects of hypothermia alone. Most solutions are modified with additives (e.g. oxygen radical scavengers) that are believed to improve organ storage condition. Maximum cold preservation time varies among organs and solutions used. Kidneys can be stored in cold preservation solution for up to 24–30 hours before transplantation.

Q. What is end stage renal disease? What is physiology of ESRD?

End stage renal disease (ESRD) is the term used to describe a clinical syndrome characterized by multiple organ dysfunction that will prove fatal without dialysis. Patients with chronic renal failure are at increased risk of developing ESRD. They have glomerular filtration rates of <25% of normal with an increased blood nitrogen waste (urea) or retention of fluid and electrolytes.

Pathphysiology of ESRD: In chronic renal failure, there is extreme decrease of functioning nephrons and GFR which results in inability of kidney to

regulate the volume and composition of extracellular fluids and excrete waste products. The loss of homeostasis, accumulation of cellular toxins and inability of the kidney to maintain water balance causes multiple organ system dysfunction.

When urine production declines to less than 400 mL/day, patient becomes oliguric and fails to excrete dietary fluid and electrolyte load. Abnormalities in Na^+, K^+, Ca^{++}, Mg^{++} and phosphate levels develop, with most threatening being hyperkalemia. These patients require frequent or continuous dialysis.

Metabolic acidosis occurs in two forms: Hyperchloremic normal anionic gap acidosis and high gap acidosis due to inability to excrete titrable acids by the kidney.

Cardiovascular complications of ESRD are due to volume overload, high renin angiotensin activity, autonomic nervous system dysfunction, acidosis and electrolyte disturbances. Hypertension is almost a universal finding in ESRD contributing to development of myocardial dysfunction and heart failure (chronically elevated systemic pressure loads to left ventricular hypertrophy and increased myocardial oxygen requirement). Pericarditis may occur secondary to uremia. Dilated cardiomyopathy and concentric hypertrophy can develop in response to increase in intravascular volume and afterload. The accumulation of uremic toxins and metabolic acidosis contribute to poor myocardial performance. ESRD with depressed ventricular function is not contraindication to renal transplantation but may complicate anesthetic management. Renal failure also accelerates progression of atherosclerosis in coronary arteries. Arrhythmias may be encountered in patients with ESRD due to electrolyte abnormalities.

Pulmonary edema and restrictive pulmonary dysfunction are common patients with renal failure which are responsive to dialysis. Hypervolemia, heart failure, reduced serum oncotic pressure and increased pulmonary capillary permeability are factors contributing to pulmonary edema.

Anemia of chronic renal failure is due to reduced level of erythropoietin, red cell damage, and ongoing gastrointestinal blood loss and iron or vitamin deficiencies. It is of normochromic, normocytic variety and treated with recombinant erythropoietin.

A qualitative defect in the platelet function reduced by uremia aggravates blood loss, treated by cryoprecipitate and desmopressin acetate.

A thromboelastographic study of whole blood clotting found increasing coagulability and decreased fibrinolysis resulted from uremia. Uremia causes CNS disturbances as drowsiness, memory loss, myoclonus seizures, stupor and coma. Signs of peripheral or autonomic neuropathy are strong indications for dialysis.

Chronic uremia causes decreased gastric emptying. All presenting for kidney transplantation should be considered as full stomach.

Acquired defects in both cellular and humoral immunity account for high incidence of serious infection and high mortality due to sepsis in CRF patients.

Q. What is renal failure? How is it categorized?

The term renal failure is used to denote failure of renal excretion leading to retention of nitrogenous waste products of metabolism including creatinine and urea. Other aspects of renal function including regulation of fluid and electrolyte status and endocrine function of kidney also fail.

The most fundamental categorization of renal failure is acute and chronic type.

Q. What is acute renal failure?

Sudden inability of the kidney to vary the urine volume and content appropriately in response to homeostatic needs. There are three main types depending on etiology:

1. *Prerenal:* Results from decreased renal perfusion due to circulatory problems.
2. *Renal:* Results from primary or secondary renal disease, toxins and pigments.
3. *Post-renal:* Results from obstruction of urinary tract.

Pre-renal and Post-renal types are usually reversible with treatment. Renal type is most serious and often needs hemodialysis.

Classification according to urine flow rate: Oliguric, non-oliguric, polyuric renal failure.

In acute oliguric renal failure, there is progressive increase in creatinine and BUN concentration and urine flow <20 mL/hour.

Q. What are the presenting features of acute renal failure?

- Patients are oliguric (urine <500 mL/day)
- Anorexia, nausea, vomiting
- Muscle twitching
- Drowsiness, confusion and coma
- Increased respiratory rate due to acidosis, pulmonary edema and respiratory infection
- Anemia due to blood loss resulted from platelet dysfunction and disturbance of coagulation
- Severe infection due to depressed cellular and humoral immune mechanism
- Increased plasma creatinine and urea concentration. Hyperkalemia with metabolic acidosis.

Q. What is the management of acute renal failure?

- Hyperkalemia (K^+ >6 mmol/L) should be treated immediately
- Optimization of circulatory blood volume to be done for adequate renal perfusion using CVP and pulmonary wedge pressure (PWP) wedge monitoring
- Acidosis corrected with isotonic sodium bicarbonate
- Inotropic drug administration to restore effective blood pressure.

Treatment of hyperkalemia (K^+ >7 mmol/L):

Therapy	Mechanism
• Intravenous calcium gluconate (10 mL of 10% solution), if ECG changes suggest hyperkalemia	Stabilization of cell membrane potential
• Inhaled β_2 agonist e.g. salbutamol • Intravenous glucose (50 mL of 5% solution) and insulin 5U • Intravenous sodium bicarbonate (100 mL of 8.4% solution) if acidosis present	Shift K^+ into cells
• Intravenous furosemide and NS, if adequate residual renal function • Ion exchange resin • Dialysis	Remove K^+ from body

Early treatment cures the condition without any residual renal impairment. With delayed and ineffective treatment, renal failure is established and renal replacement therapy (RRT) required.

Q. What is chronic renal failure? What are the causes?

Chronic renal failure (CRF) is an irreversible deterioration of renal function which develops over a period of years. Initial manifestation is biochemical abnormality. Eventually loss of secretory, metabolic and endocrine functions of kidneys result, producing signs and symptoms of CRF (uremia).

When death is likely without renal replacement therapy, it is called end stage renal failure.

Causes: CRF is caused by any condition, which destroys the normal structure and functions of the kidney.

- Congenital (polycystic kidney)
- Renal artery stenosis
- Hypertension
- Glomerular diseases (IgA nephropathy is the most common)
- Diabetes mellitus.

Q. What investigations are done in a patient with CRF?

- Full blood count (anemia)
- Blood urea, creatinine and electrolyte concentrations
- Blood calcium, phosphate and albumin concentrations
- Blood lipid and glucose
- Renal ultrasound
- Chest X-ray (heart size and pulmonary edema)
- ECG in patients above 40 years and with risk factors for cardiac disease
- Renal artery imaging
- Hepatitis and HIV serology.

Q. What are the considerations for the management of CRF?

- Identification of underlying renal disease and their treatment
- Prevention of further renal damage by:
  - Control of systemic blood pressure to retard deterioration of CRF. The target BP is 130/85 mm Hg. ACE inhibitors are more effective in retarding the progress of renal failure
  - Moderate restriction of dietary protein to 60 g/day is recommended. This should be accompanied by adequate calorie intake to prevent malnutrition.

  Once the plasma creatinine exceeds 3.4 mg/dL, there is progressive deterioration of renal function irrespective of etiology
- Prevention of adverse effects due to loss of renal function
- Institution of renal replacement therapy (dialysis, transplantation) when appropriate.

Q. What are the adverse effects of CRF? How to limit them?

- *Anemia:* Correlates with the severity of renal failure and contributes to many of the nonspecific symptoms of CRF. Recombinant human erythropoietin is effective in curing anemia of CRF.
- *Fluid electrolyte imbalance:* Failing kidney is unable to concentrate urine, a relatively high volume is needed to excrete products of metabolism. Hence, a fluid intake of 3 L/day is desirable. Some patients with salt wasting disease require an intake of 5–10 g/day of sodium chloride.
- *Acidosis:* Declining renal function is associated with metabolic acidosis. Acidosis causes further reduction in renal function and increased tissue catabolism. Plasma bicarbonate should be maintained above 22 mmol/L.
- *Cardiovascular disease and lipids:* Atherosclerosis is common and accelerated by hypertension. Pericarditis is common in ESRF. Hypertension and increased triglyceride level are common in patients with CRF, should be reduced by appropriate medications.
- *Infection:* Cellular and humoral immunity are impaired with increased susceptibility to infection with CRF, needs prompt treatment.
- *Bleeding:* Patients with CRF have increased bleeding tendency, manifested as ecchymoses. Adequate dialysis corrects it partially.
- *Renal osteodystrophy:* It is metabolic bone disease in CRF that consists of osteomalacia, osteosclerosis, and hyperparathyroid bone disease. To minimize the effect of CRF on bone, plasma calcium and phosphate levels are kept nearer to normal values.
- *Myopathy:* Generalized myopathy is combined effect of poor nutrition, hyperparathyroidism, vitamin D deficiency and disorders of electrolyte metabolism. Proper correction is needed.
- *Other side-effects:* Neuropathy is due to demyelination of medulated fibers. Motor neuropathy—foot drop, autonomic neuropathy—delayed gastric emptying and postural hypotension.

  Neuropathy is a late manifestation of CRF and improves or resolves with dialysis.

Half-life of insulin is prolonged in end stage CRF due to decreased tubular metabolism and so insulin requirement declines in these patients. There is also insulin resistance.

GI manifestations are anorexia, nausea and vomiting.

Q. What is renal repacement therapy?

Routine treatment of ARF and CRF to replace some functions of the kidney artificially by dialysis. It does not replace endocrine and metabolic functions of the kidney but aims to maintain plasma biochemistry (uremic toxins, electrolytes and acid-base status) at acceptable level. Dialysis also removes fluid from circulation. Hemodialysis is common form of RRT.

Q. What are the common types of RRT in CRF?

Intermittent hemodialysis: Commonly used and standard blood purification therapy in ESRF. It is started when the patient has symptomatic advanced renal failure but before development of complications with plasma creatinine of 6.8–9 mg/dL. Vascular access is required, an arteriovenous fistula is formed usually in forearm or a prosthetic arteriovenous graft is inserted. Dialysis is carried out for 3–5 hours, three times weekly. The dialysis principle involves equilibriation of waste products in the patient's blood across a semipermeable membrane to the dialysis bath.

Both the patient and circuit are heparinized to prevent clotting. The signs and symptoms of uremia (volume overload, acid-base and electrolyte imbalance, abnormal mental function, muscle weakness, peripheral neuropathy and defective coagulation) are improved with this treatment. Hypertension is also improved.

If fluid and electrolyte shifts are too rapid, dialysis disequilibrium occurs with manifestations of nausea, vomiting, weakness, convulsion and coma.

Vascular access is done under local filtration, brachial plexus block (axillary) or general anesthetic uremic patients are debilated and should be given smaller doses of all drugs including local anesthetics. Brachial plexus block produces analgesia and vasodilatation facilitating insertion of cannula. Duration of block is shorter in uremic patients. Epinephrine content of local anesthetic should not be more than 1:200,000 and dilution of 1:400,000.

Problems during hemodialysis:

- *Hypotension:* Sudden decrease of BP due to fluid removal and hypovolemia. Treated by saline infusion.
- *Cardiac arrhythmias:* Decreased BP, chest pain due to acid-base balance shift. Dialysis is stopped and correction done.
- *Hemorrhage:* Due to anticoagulation. Dialysis is stopped and treatment instituted.
- *Dialysis hypersensitivity:* Due to allergic reaction to dialysis membrane, presenting with acute circulatory collapse. Dialysis is stopped.
  Emergencies between treatments:
  - Pulmonary edema (breathlessness)
  - Systemic sepsis (rigor, fever and decreased BP).

Continuous ambulatory peritoneal dialysis (CAPD): Patient's peritoneum acts as exchange membrane and vascular access is not needed. It is less efficient and dialysis time is longer. The dialysis involves insertion of a permanent silastic catheter into peritoneal cavity. Isotonic dialysis fluid is introduced and left in place for 6 hours and then drained. The cycle is repeated 4 times daily. Patient remains ambulatory. Concerns are pain during dialysis and chance of peritonitis. Automated peritoneal dialysis is now widespread, where the fluid exchange is performed at night with a mechanical device leaving the patient free or with a single exchange during the day.

Q. What are the indications of RRT in ARF?

Indications are:

- *Increased plasma urea and creatinine:* Urea >180 mg/dL, creatinine >6.8 mg/dL and progressive biochemical deterioration with little or no urine output.
- *Hyperkalemia:* Potassium level >6 mmol/L is hazardous and is usually reduced by medical treatment but dialysis is often required for definite control.
- *Metabolic acidosis:* Further raises plasma K^+ level
- *Fluid overload and pulmonary edema:* With presence of urine output, it is controlled by careful fluid balance and use of diuretics. In oliguria or anuria, RRT is indicated.
- Uremic pericarditis and uremic encephalopathy occur in severe, untreated RF and are strong indications for RRT, uncommon in ARF.
- *Options of RRT in acute renal failure:* Hemodialysis, high volume hemofiltration, continuous arteriovenous or venovenous hemofiltration and peritoneal dialysis.

Q. What is the indication of kidney transplantation? How is it performed and preserved?

Kidney transplantation is indicated in and offers best chance of long-term survival for patients with ESRD. It restores normal kidney function and corrects all metabolic abnormalities of CRF.

Renal transplantation with organs from living donors is increasing rapidly in recent years. The donor kidney is usually implanted in the iliac fossa with no need for native nephrectomy. The donor vessels are anastomosed to the recipient iliac artery and vein and the donor ureter to the bladder. ABO blood group compatibility between donor and recipient is essential. A cytotoxicity test for antibodies against HLA antigens and T and B cells, cross-matching tests are performed pretransplant. After transplant, immunosuppressive therapy is required to prevent graft rejection and is to be continued throughout life. Triple therapy with prednisolone, cyclosporin/tacrolimus and azathioprine is commonly used. Prolonged use of immunosuppression is associated with increased incidence of infection and increased risk of malignancy. Newer drugs such as mycophenolate mofetil, rapamycin are increasing in use. The goal of therapy of the newer drugs includes:

- Prevention of immune response
- Prevention of complications of immunosuppression as opportunistic infection and malignancy
- Minimization of drug-induced toxicity.

Q. What are the complications of chronic immune suppression?

Systems	Complications
Central nervous system	Lowered seizure threshold
Cardiovascular system	Hypertension, hyperlipidemia, diabetes
Renal/electrolyte	Decreased GFR, hyperkalemia, hypomagnesemia
Hematologic/immune	Increased risk of infection, increased risk of tumor, pancytopenia
Endocrine/others	Osteoporosis, poor wound healing

Q. What are the functions of the kidney?

- Regulation of volume and composition of body fluids by making large volume of ultrafiltrate of plasma (120 mL/min, 170 L/day) at glomerulus and selectively absorbing components from it at points along the nephron.
- Excretion of metabolic breakdown products (urea, creatinine and ammonia), drugs and toxins.
- Regulation of acid-base balance.
- Secretes rennin (from juxtaglomerular apparatus), which generates angiotensin II.
- The kidney is main source of erythropoietin.
- The kidney is essential for vitamin D metabolism.

Q. What are the tests for assessment of kidney function?

- *Urine specific gravity:* As renal function decreases, the ability of the kidney to concentrate urine decreases and is reflected by proportional change of specific gravity (normal: 1.012).
- *Serum creatinine level:* It is the end product of muscle creatine metabolism and excreted by kidney. It does not reflect early loss of renal function, because serum level remains normal until 50% loss of kidney function (Normal: 0.8 mg/dL).
- *Endogenous creatinine clearance rate:* It is the best measure of kidney function and is defined as the volume of plasma from which creatinine is completely removed per unit of time. (Normal: 90 to 110 mL/min). It provides a simple, inexpensive, bedside estimate of CRF. Small amount of creatinine is secreted by proximal tubules.
- *Inulin clearance:* Inulin is an inert polyfructose sugar that is completely filtered by the glomerulus and is neither secreted nor absorbed by renal tubules. Therefore, inulin clearance is the gold standard for measuring GFR, yet it is seldom used clinically as its accurate measurement is laborious and requires meticulous attention to detail.
  Normal values in males: 110 to 140 mL/min; in females: 95 to 125 mL/min.

Renal function is assessed preoperatively with serum electrolytes, blood urea nitrogen and creatinine measurements. Information about the size and perfusion are obtained by CT scan or aortogram.

Perioperative medication and perfusion strategies are adjusted based on renal function.

Q. What are the considerations in preoperative evaluation of a kidney transplanted patient?

When renal transplanted patients have subsequent surgery, the status of their renal function must be determined. Good graft function is determined by BUN and creatinine values and sufficient urine volume. Renal function must be assessed, as long-term immunosuppression leads to chronic renal insufficiency. The presence of graft rejection should be evaluated.

These patients are still at higher cardiac risk due to persistent risk factors as diabetes, hypertension, dyslipidemia and secondary hyperparathyrodism. Congestive heart failure (cardiomyopathy), left ventricular hypertrophy and ischemic heart disease remain important complications in renal transplanted patients. Therefore, a careful cardiac evaluation is essential.

Immunosuppressant therapy results in hyperglycemia, adrenal suppression (steroids), increased risk of infection, hypertension, renal insufficiency (cyclosporine, steroids and tacrolimus), myelosuppression with anemia, thrombocytopenia, and leukopenia (azathioprine, sirolimus). The side effects of immunosuppressants should be sought and close monitoring of blood glucose level and cardiovascular function is done.

Dosage scheduled for all medications should be noted and the patient is instructed to continue all the drugs.

Q. What are the considerations during anesthetic management of a kidney transplanted patient?

Both regional and general anesthesia are used successfully. Nasal intubation is avoided for risk of infection by nasal flora. Standard ASA monitors are used. Invasive monitoring not indicated solely because of previous transplantation and usually avoided due to increased risk of infection resulted from immunosuppression therapy.

A major consideration for renal transplanted recipient is maintenance of renal perfusion with adequate volume replacement. The CVP monitoring during volume replacement is useful for preventing prerenal damage to transplanted kidneys and the line should be placed using strict aseptic technique.

Antibiotics, antiviral, antifungal and immune suppression regimens should be disrupted as little as possible in the perioperative period.

The specific issue in renal transplanted patients is that, despite presence of normal creatinine level, GFR is generally decreased leading to electrolyte abnormalities and altered drug metabolism.

Nephrotoxic drugs, NSAIDs and COX-2 inhibitors should be avoided in renal transplanted patients.

Drug interaction between immunosuppressants and anesthetic drugs may occur. Cyclosporine alters barbiturate, fentanyl, and isoflurane requirement. It also enhances the effect of neuromuscular blocking (NMB) drugs leading to decreased requirement of non depolarizing muscle relaxants in patients taking cyclosporine.

Azathioprine causes small and transient antagonism to nondepolarizing muscle relaxants. Produces no effect on atracurium requirement.

Preoperative review of patient's medication focuses on possible side effects.

DVT prophylaxis should be considered in renal transplanted patients.

As there is increased risk of infection, patient cross-contamination should be avoided.

Chapter

5

TURP in an Elderly Patient with Hypertension and Ischemic Heart Disease

Q. What does ischemic heart disease reflect?

It refers to atherosclerosis in coronary artery.

Q. What are the first manifestations of IHD?

- Angina pectoris
- Acute myocardial infarction
- Sudden death.

Q. How atherosclerosis results angina pectoris?

Atherosclerosis impairs coronary blood flow leading to imbalance between coronary blood flow and myocardial oxygen consumption. This results myocardial ischemia and manifested as angina pectoris.

Q. How angina pectoris is diagnosed?

A typical retrosternal chest pain radiating to neck, left shoulder, left arm or lower jaw. Occasionally it radiates to back, down the right arm or down both arms. This discomfort is perceived as pain or often described as pressure or heaviness. Angina pectoris lasts for several hours and is promptly relieved by rest and/or nitroglycerine.

Q. What are the differences between stable and unstable angina?

Stable angina: Effort related chest pain is the hallmark of stable angina.

Symptoms over long-term: Occurs when there is imbalanced myocardial oxygen supply and demand. Fixed or stable coronary atheroma is the most common cause. May occur in aortic valve disease and hypertrophic cardiomyopathy.
Promptly relieved by rest or nitroglycerin.

Physical examination frequently negative, but risk factors may be present.

ECG: may show evidence of old myocardial infarction. T wave flattening or inversion in some leads providing nonspecific MI.

Exercise testing noting

- Amount of exercise tolerated
- Extent or degree of ST segment changes
- Amount of BP response.

Coronary angiography

Unstable angina: Characterized by a new-onset or rapidly increasing angina at minimal exertion or at rest. Prolonged ongoing in nature.

The causative lesion is usually a complex plaque with adherent platelet rich thrombus and local coronary artery spasm. The process is dynamic and the degree of obstruction may increase by changes in the plaque morphology or may regress temporarily by platelet disaggregation and endogenous fibrolysis.

Patient needs urgent evaluation to avoid complications like sudden death and myocardial infarction (MI). Hemodynamic compromise (hypotension, heart failure, ECG changes—ST elevation or depression) and biochemical marker of cardiac damage such as troponin I and T are most powerful indicators of short-term risk.

Q. How does the noncardiac chest pain differ from angina?

Noncardiac chest pains are usually transient, exacerbated by chest wall movement and associated with tenderness over the involved area which is often a costochondral joint.

Retrosternal sharp pain exacerbated by deep breathing, coughing and variation in body position suggests pericarditis.

Esophageal spasm can produce severe substernal pressure that may be confused with angina pectoris.

Q. What are the factors influencing myocardial oxygen demand and supply?

Myocardial oxygen extraction from arterial blood is maximum at rest. As increases with exercise or hemodynamic stress, the oxygen supply to myocardium also must increase.

Myocardial oxygen demand is influenced by contractile state of myocardium, afterload, preload and heart rate.

Oxygen supply is determined by oxygen content of arterial blood and coronary blood flow.

Oxygen content of blood depends mainly on the hemoglobin bound oxygen. The actual delivery of oxygen depends on the release of oxygen from hemoglobin according to oxyhemoglobin dissociation curve. A leftward shift of the curve decreases the release of oxygen and hence decreases oxygen supply, coronary blood flow and hence the myocardial oxygen supply is

affected by diameter of coronary arteries, left ventricular end diastolic pressure, aortic diastolic pressure and duration of diastole. 70%–80% of coronary arterial blood flow occurs during diastole. This is the reason for use of beta-blocker as anti-ischemic agent.

Q. What is the coronary perfusion pressure?

In the normal heart, the coronary perfusion pressure is the difference between the aortic diastolic pressure and the left ventricular end diastolic pressure. Thus, elevation of LVEDP reduces subendocardial blood flow.

Q. What are the risk factors for ischemic heart disease?

- Male gender
- Increasing age
- Hypercholesterolemia
- Systemic hypertension
- Cigarette smoking
- Diabetes mellitus
- Obesity
- Sedentary lifestyle
- Family history
  First two are the most important factors.

Q. What investigations are done in a patient with ischemic heart disease?

- *Resting ECG:* Reversible ST segment depression or elevation with or without T wave inversion at the time of symptoms is the most convincing ECG evidence of myocardial ischemia.
- *Exercise ECG:* Performed by using standard treadmill or bicycle ergometer protocol while monitoring patient's ECG, BP and general condition. The amount of exercise which can be tolerated and the extent and degree of any ST segment change provide a useful guide to the extent of coronary disease. Down-sloping of ST segment depression of 1 mm or more is indicative of ischemia.
- *Stress echocardiography:* Echocardiographic wall motion analysis performed after stressing the heart by dobutamine infusion. The ventricular wall motion abnormalities induced by stress correspond to the site of ischemia, thereby localizing the obstructive coronary lesion.
- *Nuclear stress imaging:* It is useful for assessing coronary perfusion. Tracer (e.g. Thallium) activity during stress is studied. Exercise increases the difference in tracer activity between normal and under perfused regions because coronary blood flow increases markedly with exercise except in the region distal to coronary artery obstruction. The magnitude of perfusion abnormality indicates prognosis.
- *Coronary angiography:* It provides detailed anatomical information about the extent and nature of coronary artery disease. Usually performed with

a view to CABG or PCI. Diagnostic coronary angiography is indicated when noninvasive tests fail to detect the cause of atypical chest pain. It is done under local anesthesia with cardiac monitoring using specialized radiological equipment by an experienced operating team.

Q. What are the stress testings in a patient with IHD?

Stress testing identifies ischemic heart disease and its severity but the positive predictive value for perioperative cardiac complication is less.

- *Exercise treadmill testing:* The test result is adequate when the patient can exercise to at least >85% of target heart rate (the target HR is 220 – age).
- *Dobutamine echocardiography and nuclear perfusion imaging:* These are useful in patients unable to exercise, patients with pacemakers, significant bradycardia or taking high dose beta-blocker.

Combination of exercise and imaging can be used in patients who are able to exercise and have significant abnormalities in ECG that may interfere with the interpretation of ischemia.

Dobutamine uncovers ischemia by increasing contractility, HR and BP. Therefore, is not the best test for the patient with pacemakers, significant bradycardia, aortic or cerebral aneurysms or poorly controlled hypertension.

Adenosine radionucleotide imaging uses the vasodilatory properties of adenosine and uptake of radioisotope by viable myocardium.

Echocardiography combined with exercise or pharmacologic agent is used to look for wall motion abnormalities. Abnormalities at rest (baseline) indicate previous infarction. Areas of myocardium that are normal at rest but show abnormalities with isotropy or chronotropy are indicative of stenotic lesion and limited blood flow.

Myocardium with limited blood flow is normal at rest but shows decreased uptake of isotope with exercise or adenosine administration.

Q. What is the treatment of ischemic heart disease?

Goal of management involves:

- Careful assessment of extent and severity of arterial disease.
- Identification and control of risk factors.
- Measurement to control symptoms.
- Treatment to improve life expectancy in high-risk patients.

In patient with stable angina, anxiety and pain should be treated.

Antiplatelet therapy: Low dose (75–150 mg) aspirin reduces the risk adverse event and should be prescribed for all with coronary artery disease.

Clopidogrel (75 mg daily) is equally effective anticoagulant agent.

Anti-anginal drug treatment: Drugs commonly used to relieve or prevent symptoms of angina are:

- *Nitrates:* Act directly on vascular smooth muscle to produce venous or arterial dilatation, beneficial effects are due to reduction in myocardial oxygen demand (lower preload and afterload) and an increase in myocardial oxygen supply (coronary vasodilatation).

- *Beta-blocker:* Lower myocardial oxygen demand by reducing heart rate, BP and contractility. These drugs should not be withdrawn abruptly as this may have rebound effect and precipitate dangerous arrhythmias, worsening angina and myocardial infarction (beta-blocker withdrawal syndrome).
- *Calcium antagonists:* Act particularly on cardiac arteriolar smooth muscle. They lower myocardial oxygen demand by reducing blood pressure and myocardial contractility.
- *Potassium channel activators:* These drugs cause arterial and venous dilatation. Exhibit no tolerance as seen with nitrates. Nicorandil (10–30 mg, 12 hourly orally) is the only drug currently available for clinical use.

No one drug is more effective than other. Conventionally, the treatment is started with low dose aspirin, sublingual GTN and beta-blocker and then a calcium channel antagonist is added or a long-acting nitrate. Revascularization is considered if an appropriate combination of two drugs fail to achieve a symptomatic response.

Invasive treatment: Most widely used invasive options for treatment of ischemic heart disease are:

- Percutaneous coronary intervention (PCI) including percutaneous transluminal coronary angioplasty (PTCA)
- Coronary artery bypass graft (CABG) surgery.

Percutaneous coronary intervention (PCI): Performed by passing a fine guide wire across a coronary stenosis under radiographic control to position a balloon, which is inflated to dilate the stenosis. A coronary stent is a piece of coated metallic scaffolding that is used to maximize and maintain dilatation of stenosed vessel. In combination with aspirin and heparin, adjunctive therapy with platelet inhibitors, clopidogrel improve the outcome of PCI.

Coronary artery bypass grafting (CABG): The internal mammary arteries, radial arteries or reversed segment of patient's own saphenous vein can be used to bypass coronary artery stenosis. It involves major surgery under CBP. Graft can be applied to beating heart, off pump surgery.

Aspirin (75–150 mg daily) and clopidogrel (75 mg daily) improve the graft patency; one or other should be prescribed indefinitely if well-tolerated.

Management of unstable angina: Patients should be admitted urgently to hospital as there is risk of death or acute myocardial infarction. Initial treatment should include:

- Bed rest
- Antiplatelet therapy: Aspirin (300 mg followed by 75 mg daily, long-term) and clopidogrel (300 mg followed by 75 mg for 12 months)
- Anticoagulant therapy: Heparin
- Beta-blocker: Atenolol (50–100 mg daily) or metoprolol (50–100 mg, 12 hourly)
- Calcium antagonist: Nifedipine or amlodipine can be added.
- If pain persists, IV or buccal nitrates are helpful.

Most low-risk patients stabilize with aspirin, clopidogrel, heparin and antianginal therapy.

Patients at moderate- or high-risk, patients who failed to settle on medical therapy, those with elevated plasma troponin need, angiography followed by PCI. Patients not suitable for PCI need urgent CABG.

Q. What are the factors that influence perioperative risk in a patient with IHD?

Cardiovascular risk is influenced by:

- Patient factor including functional capacity
- Surgical factors (nature of planned surgery).

Q. How patient factor is classified?

- Major risk predictors (marker of unstable coronary artery disease)
  - Recent MI (<1 month prior to surgery)
  - Unstable angina
  - Decompensated heart failure
  - High grade A-V block
  - Supraventricular tachycardia
  - Aortic/Mitral stenosis
  - CABG/PTCA (<6 weeks prior to surgery).
- Intermediate-risk predictors (markers of stable coronary artery disease)
  - Prior MI (>1 month prior to surgery)
  - Stable angina
  - Compensated heart failure
  - Abnormal renal function
  - Diabetes.
- Minor risk predictors (increased probability of heart disease)
  - Advanced physiological age
  - Abnormal ECG
  - Rhythm other than sinus
  - Low functional capacity
  - Previous stroke
  - Uncontrolled systemic hypertension.

Q. What is functional capacity?

Exercise tolerance is a major predictor of perioperative risk in patient with IHD. Ability to exercise is an excellent indicator of cardiovascular fitness and is usually expressed as metabolic equivalent (MET_S). Patients who cannot attain 4 METS of physical activity frequently have adverse outcome following high-risk surgery.

A metabolic equivalent or MET is equal to the amount of oxygen consumed by a 40-year-old 70 kg male sitting at rest is equal to 3.5 mL/kg/min of oxygen uptake.

Q. How METs are categorized?

- 1–4 METs—eating, dressing and walking around the house.
- 4–10 METs—climbing a flight of stairs, walking on level at >6 km/hr, running briefly.
- >10 METS—strenuous sports, swimming, etc.

Q. How surgical factors are categorized?

- High-risk
  - Major emergency surgery
  - Aortic/major vascular surgery
  - Prolonged surgery with large fluid shift.
- Intermediate-risk
  - Carotid endarterectomy
  - Head and neck surgery
  - Intraperitoneal and intrathoracic surgery
  - Orthopedic surgery
  - Prostate surgery.
- Low-risk
  - Minimally invasive endoscopic surgery
  - Cataract extraction
  - Superficial surgery.

Q. What is cardiopulmonary exercise testing?

Cardiopulmonary exercise testing (CPET) is a most capacity of the cardiopulmonary system. It is used to accurately assess risk prior to major surgery. There is increased global oxygen demand following major surgery which a heart with decreased functional reserve fails to supply, leading to imbalance of myocardial oxygen supply demand, a determinant of major cardiac complications in perioperative period in noncardiac surgery.

Cardiopulmonary exercise testing involves computerized analysis of gas exchange ECG date during exercise. It is noninvasive. It consists of an exercise test with a progressive graded work rate simultaneously breath-to-breath measurement at the month piece, of inspired expired concentrative of oxygen and carbon dioxide and inspiratory and expiration gas flow are assessed. With this information, oxygen uptake and carbon dioxide output are derived. In addition, 12 lead ECG is obtained continuously.

There are two commonly used modalities of exercise testing: The treadmill and the stationary cycle ergometer. In both methods the work rate is gradually increased in stepped or ramped manner until the subject is unable to continue or if ECG shows greater than 2 mm ST depression. Compared to treadmill, cycle ergometer has advantages of better isolation of lower limb musculature, less effect of movement artifacts and greater safety.

Other important piece of equipment required is the metabolic cart. The metabolic cart contains a gas analyzer, a computer and screens, which display the continuous 12-lead ECG, with ST segment analysis and graphical

displays of physiological charges occurring during exercise. The software of the metabolic cart is configured for continuous display of oxygen uptake, carbon dioxide output, work rate in watts, heart rate/VO_2 slope, ventilator equivalents for oxygen and carbon dioxide, minute and tidal volumes and flow volume loops and oxygen pulse, which is related to stroke volume.

In CPET, cardiopulmonary data is displayed in graphical form. As various disease states show characteristic patterns of physiological response to exercise, interpretation of CPET is more easily done by examination of graphical data.

Q. What is anaerobic threshold?

Anaerobic threshold (AT) is the primary determinant of cardiac function and is estimated by the V-slope method. The anaerobic threshold is the point of oxygen uptake where anaerobic ATP generation is needed to supplement aerobic metabolism. AT is used to define grading of cardiac failure.

Patients at risk of perioperative cardiac morbidity have an AT less than 11 mL/kg/min of oxygen uptake. In terms of AT, the range from no heart failure to significant heart failure is from 14 mL/kg/min to 8 mL/kg/min. An AT of 11 mL/kg/min of oxygen uptake is equal to 4 METs. If a patient on medication has an AT greater than 11 ml/kg/min, there is no need to stop or modify it. If the patient has a low AT, revealing a low cardiopulmonary rescue, is on potentially negative inotrope medication (e.g. α-blocker), the dose should be modified with consideration of postoperative cardiac failure versus risk of postoperative myocardial ischemia.

Myocardial ischemia is important only when it limits ventricular function. Various studies have shown that myocardial ischemia in presence of good functional capacity (i.e. AT greater than 11 mL/kg/min) was not associate with postoperative cardiac complications.

Risk factors for cardiac disease in general and myocardial ischemia have the common point of impaired ventricular function. Not the diagnosis of myocardial ischemia but the functional capacity of heart and lungs determines the ability to support increased postoperative oxygen demand and, thus, influences morbidity and mortality after major surgery. So measurement of functional capacity and identification of cardiac failure is of prime importance.

Q. What is revised cardiac risk index?

The revised cardiac risk index has been validated in several studies as the best scoring system to predict perioperative cardiac risk in patients undergoing noncardiac surgery. The most important risk factors for adverse events are:

- History of ischemic heart disease
- History of congestive heart failure
- History of cerebrovascular disease
- Diabetes mellitus
- Renal insufficiency (creatinine >2.0 mg/dL)

- High-risk surgery (intraperitoneal, intrathoracic and open vascular procedure)
- Advanced age.

Q. How cardiac evaluation is done for noncardiac surgery in a patient with IHD?

The goal for evaluation is to identify patients with heart disease who are at high-risk for perioperative cardiac morbidity or mortality or those with modified condition or risk. The guidelines for cardiac evaluation before noncardiac surgery published by the ACC/AHI have become the standard of care. These guidelines are recently revised with a marked reduction in recommendation for preparative stress testing and revascularization.

Simplified cardiac evaluation for noncardiac surgery:

Step 1: Emergency surgery	Proceed to surgery with risk reduction and perioperative surveillance
Step 2: Active cardiac conditions • Unstable coronary syndromes (unstable or severe angina, recent MI) • Decompensated HF • Significant arrhythmias (Mobitz II or 3rd degree heart block, SVT or AF, new VT bradycardia) • Servere valvular disease (severe AS or MS)	Postpone surgery until stabilized or corrected
Step 3: Low-risk surgery (risk <1%) • Superficial or endoscopic • Cataract or breast • Ambulatory.	Proceed to surgery
Step 4: Functional capacity • Good ≥4 MET (Can walk flight or stair without symptoms	Proceed to surgery
Step 5: Clinical predictors • Ischemic heart disease • Compensated or prior HF cerebrovascular disease (stroke or TIA) • Diabetes mellitus • Renal insufficiency	No clinical predictors – Proceed to surgery 1–2 clinical predictors – Vascular surgery – Intermediate risk surgery > 3 clinical predictors – Vascular surgery Consider testing of it will change management.

Q. What is NYHA classification of medical status of patients?

According to New York Heart Association, the medical status of patients is classified into following categories :

Class I: No limitation of physical activity. Ordinary activity does not cause fatigue, palpitation or syncope.

Class II: Slight limitation of physical activity. Ordinary activity results in fatigue, palpitation or syncope.

Class III: Marked limitation of physical activity. Less than ordinary activity results in fatigue, palpitation or syncope.

Class IV: Inability to perform any physical activity without discomfort; symptoms at rest.

Q. What is the role of percutaneous coronary intervention prior to noncardiac surgery?

A significantly lower rate of 30 days cardiac complications are noted in patients who undergo PCI at least 90 days before the noncardiac surgery, do not improve outcome, PCI may actually destabilize coronary plaque which becomes manifested in the days or weeks after noncardiac surgery.

Coronary revascularization is not indicated in patients with stable coronary artery disease.

Coronary revascularization is not indicated in patients with PTCA. It may be classified as:

Recent <6 months

Prior: >6 months and ≤ 2 years; Remote ≥ 2 years

Previous coronary revascularization (PTCA <2 years) may provide only moderate protection against adverse cardiac events of mortality. Individuals with remote revascularization (PTCA ≥ years) and nonrevascularized patients are stratified at high risk or intermediate/low risk.

PCI using coronary stenting poses several special issues. Perioperative cardiac events are greater in patients with recent stents (<35 days before surgery than in those who have PCI more than 90 days before surgery. The incidence of acute MI and death within 6 months is not significantly different in the stent group and in the PTCA group. Significantly more events occurred in the two groups when noncardiac surgery was done within 2 weeks of PCI. Therefore, it is recommended that elective noncardiac surgery after PCI with or without sent, should be delayed for 4 to 6 weeks. For patients with bare-metal stents in place for less than 30 days or drug eluting stents for less than 1 year, then new guide lines suggest continuing aspirin therapy in all and discontinuing clopidogrel for as short an interval as possible.

Q. What should be the preoperative therapy in patients with IHD?

The main goal of anesthesia in patients with IHD is to decrease determinants of myocardial oxygen demand heart rate, ventricular wall tension and

contractile performance with improved plaque stabilization. 50 medical management to preserve all viable myocardial tissue include the following:

- Continuation of β-adrenergic blocking drugs to avoid β-blocker withdrawal leading to an increased contractility and heart rate. Current ACC/AHC guidelines advocate that perioperative β-blocker is a Class I indication and should be used in patients previously taking β-blockers and those with positive stress test undergoing major surgery. Although acute administration without titration may be harmful.
- Vasodilatation (with nitroglycerine/nitroprusside) to decrease ventricular wall tension.
- Aspirin, statins, exercise and diet are indicated in many patients.
- Antihypertensive medications are to be continued through the morning of surgery.

Q. What is hypertension?

When systolic/diastolic blood pressures are 140/90 mm Hg or more on at least two occasions measured at least 1–2 weeks apart.

Q. What is the classification of essential hypertension?

It is classified into three stages:

Stage I (Mild): 140-159/9099 mm Hg

Stage II (Moderate): 160–179/100–109 mm Hg

Stage III (Severe): ≥180/≥110 mm Hg.

Several studies have shown that patients without significant cardiovascular comorbid conditions can proceed with surgery despite elevated blood pressure on the day of surgery (diastolic BP between 110 and 130 mm Hg).

Q. What is the difference between essential and secondary hypertension?

- In essential hypertension, cause cannot be identified.
- In secondary hypertension, etiology is known. Most common example is PIH.

Q. What is hyperlipidemia? What is the significance of it?

Raised plasma lipoprotein levels is termed as hyperlipidemia.

Plasma lipoprotein levels are major modifiable risk factor for cardiovascular disease. Increased level of atherogenic lipoprotein (LDL) contributes to the development of atherosclerosis. Measurement of plasma cholesterol alone is not sufficient for comprehensive assessment. Total cholesterol, triglyceride, HDL and LDL are to be assessed. Target of drug treatment is: HDL e > 38 mg/dL, TG < 180 mg/dL, LDL < 100 mg/dL and total cholesterol –190 mg/dL.

Treated with one or more of cholesterol lowering drugs.

Statin: Inhibits cholesterol synthesis and increases clearance of LDL. It is well-tolerated and has less side effects.

Q. What are the complications of poorly controlled essential hypertension?

- Left vermicular hypertrophy.
- Heart failure.
- Renal insufficiency: BUN and serum creatinine quantify renal function.
- Cerebrovascular disease.
- Retinopathy.

Q. What are the changes in the blood vessels due to hypertension?

- There is thickening of the internal elastic lamina, hypertrophy of smooth muscles and deposition of fibrous tissue in larger arteries >1 mm diameter. The vessels become dilated, tortuous and less compliant.
- Hyaline atherosclerosis is smaller arteries (<1 mm diameter). Widespread atheroma leads to coronary or cerebrovascular disease, increases systemic vascular resistance and decreases renal function. Decreased renal function further aggravates hypertension.

Q. What are the ophthalmoscopic findings of retinopathy due to hypertension?

Ophthalmoscopic findings are graded as follows :

Grade I: Arteriolar thickening and tortuosity.

Grade II: Grade I + Constriction of veins at arterial crossing (arteriovenous ripping).

Grade III: Grade II + retinal ischemia (flame shape or blot hemorrhage and cotton wool exudates.

Grade IV: Grade III + papilledema.

Q. What charges are produced in the heart?

Pressure load in the heart leads to left severe hypertension, resulting in left ventricular failure.

Q. What is white coat syndrome?

It is increase in systemic blood pressure on hospital admission reflecting patient's anxiety and subsequently becoming normal. Implication: These patients display exaggerated pressure responses to direct laryngoscopy for tracheal intubation. Perioperative myocardial ischemia is more likely.

Q. What are the risk factors for development of hypertension?

Risk factors are:

- Hyperlipidemia
- Diabetes mellitus
- Smoking
- Family history

- Age >60 years
- Sedentary life.

Q. What are the treatment modalities for essential hypertension?

Goal is not to decrease chronically increased blood pressure rapidly but over a period of several weeks. Aim is to decrease blood pressure less than 140/90 mm Hg by:

- Appropriate modification of lifestyle.
- Antihypertensive medication.
  Choice of drug is dictated by cost, convenience, response to treatment and free from side effects. Combination of drugs is often required to achieve optimum BP control, combination of drugs helps low dose therapy with less side effects. Comorbid conditions may influence the choice of drugs —cardioselective β-blockers are the best for preoperative treatment.
- Adjuncts to antihypertensive drugs: Aspirin, statin and anxiolytics.
- Diuretics: In combination with antihypertensive agents, produce most consistent and effective treatment of hypertension.

Q. What are the cardiovascular comorbid conditions?

These include:

- Previous MI
- Unstable or severe angina pectoris
- Renal failure
- Pregnancy-induced hypertension
- Left ventricular hypertrophy
- H/O PCI
- Dysrrhythmias
- Conduction block
- Stroke.

Q. What investigations are done in a patient with hypertension?

All hypertensive patients should undergo a limited number of investigations. Additional investigations are done in selected patients.

- Investigations for all patients :
  - Urine analysis for blood, protein and glucose
  - Blood urea, electrolytes and creatinine
  - Hypokalemic alkalosis indicates hyperaldosteronism, but is usually due to diuretic therapy.
  - Blood glucose
  - Serum total and high density lipoprotein (HDL) and cholesterol
  - 12-lead ECG (Left ventricular hypertrophy, coronary artery disease).
- Investigations for selected patients
  - Chest X-ray to detect cardiomegaly, heart failure, coarctation of aorta.
  - Ambulatory BP recording to assess border line or white coat hypertension.

- Echocardiogram to detect quantity of left ventricular hypertrophy.
- Renal angiography to detect or confirm presence of renal artery stenosis urinary catecholamine to detect possible pheochromocytoma.
- Urinary cortisol and dexamethasone suppression test to detect possible Cushing's syndrome
- Plasma semen activity and aldosterone detect possible primary aldosteronism.

Q. How preoperative preparation is done?

Adequacy of systemic blood pressure control is assessed. Elective surgery is delayed until BP is less than 180/110 mm Hg. Effective lowering of BP requires 6–8 weeks of therapy, hypotension and ischemia on ECG are more common in poorly controlled hypertensive patients.

Antihypertensive drugs are to be continued throughout the perioperative period. The pharmacological and potentially side effects of antihypertensive drugs should be reviewed.

ACE inhibitors cause exaggerated hypotension intraoperatively, which is treated with fluid and ephedrine or phenylephrine. So these drugs are with held on the morning of surgery.

Angiotensin II antagonist produces hypotension during anesthesia. So these should be discontinued from day before surgery.

β-blockers in chronically treated hypertensive patients reduce the risk of coronary artery disease, stroke, cardiovascular death and all causes of mortality and should be continued till the day of surgery. But acute administration of β-blockers results in increased incidence of stroke and death associated with increased hypotension in the intraoperative period.

Dietetics are given on the morning of surgery.

Q. What is the most crucial period during induction of anesthesia? Why?

During laryngoscopy and passage of endotracheal tube, these noxious stimuli provoke adverse responses in cardiovascular system, significant hypertension and tachycardia are associated with tracheal intubation under light anesthesia. The magnitude of response is greater with increasing force and duration. So prolonged attempts at laryngoscopy should be avoided.

Hemodynamic changes during laryngoscopy and tracheal intubation may result in myocardial ischemia and are undesirable in patients with cardiac disease.

There is also raised intracranial and intraocular pressures, with larygoscopy and intubation. Raised intracranial pressure and decreased intracranial compliance. Raised intraocular, pathologically high lop or an open eye injury.

Q. How the cardiovascular responses to laryngoscopy and intubation of ischemia can be prevented?

Several methods are used to alternate hemodynamic responses to laryngoscopy of intubation.

- An increased depth of anesthesia.
- Narcotics, such as fentanyl 6 μg/kg IV, 5 minutes before induction of anesthesia with inhaled anesthetics and N_2O.
- Esmolol: (Rapid onset and short-acting selective β-blocker). A dose of 0.5 mg/kg IV given 2 minutes before direct laryngoscopy tracheal intubation.
- Sodium nitroprusside: 1–2, 6 μg/kg IV as a rapid injection immediately before direct laryngoscopy for tracheal intubation offsets systolic blood pressure (SBP) increase.
- Aerosol or other application of topical anesthetics immediately before direct laryngoscopy, commonly used local anesthetic is 2% to 4% lignocaine.
- Lignocaine: 1 to 1.5 mg/kg administered 3–5 minute before induction of anesthesia.

Q. What should be the anesthetic goal during maintenance?

Perioperative hemodynamic fluctuations have some relation to morbidity. The goal is, therefore, to keep cardiovascular variables within the preoperative range and plan should be made before induction about the therapies to be used to achieve this goal. During maintenance, the depth of anesthesia should be adjusted to minimize fluctuations in SBP. The volatile anesthetics are useful for this purpose.

Intraoperative painful stimuli increase sympathetic nervous system activity leading to hypertension—attenuated by using narcotic analgesic as fantasy and volatile anesthetics.

Intraoperative hypotension treated by increasing rate of fluid infusion and IV sympathomimetics (ephedrine).

Q. What is the aim of TURP?

TURP is performed by inserting a resectoscope through urethra and prostatic tissue is resected using an electrically powered cutting coagulation metal loop. The aim is to resect as much prostatic tissue as possible, preserving the prostatic capsule.

Q. What happens if capsule is perforated?

Large amount of irrigation solution is absorbed into circulation and the periprostatic and retroperitoneal space.

Q. What are the characteristics of ideal irrigation fluid?

Ideally an irrigation fluid for TURP must be:

- Non-conductive—so that the diathermy current is concentrated at the cutting point.
- Isotonic—so that hemolysis does not occur if the fluid enters the circulation.
- Must have neutral visual density—so that the surgeries view is not distorted.
- Non toxic and inexpensive.

Q. What irrigation fluid is commonly used?

Most commonly used irrigant is glycine 1.5% in water.

Q. What other irrigation solutions can be used?

- Manitol 3% to 5%—expand blood volume and may cause pulmonary edema.
- Glucose 2.5 to 4%
- May cause severe hyperglycemia in diabetic patients.
- Sorbitol 3.5%
- Cytal (mixture of sorbitol 2.7% and manitol 0.54%)
- Urea 1%.

Q. What are the complications of TURP ?

Complications are:

- TURP syndrome—hypothermia and hyposmolality.
- Glycine toxicity—transient blindness.
- Ammonia toxicity—delayed awakening. Ammonia derived from glycine.
- Perforation of the bladder—upper abdominal, precordial or shoulder pain.
- Hypothermia—causes shivering.
- Bleeding and coagulopathy—vital signs and serial hematocrit value to be monitored, assess if blood transfusion is needed or not.
- Bacteremia and septicemia—chill, fever and tachycardia. (Prostrate harbors many bacteria).

Q. What is TURP syndrome?

TURP syndrome is a complication during TURP, caused by hypernatremia and hypoosmolality due to absorption of irrigation fluid, producing CNS symptoms like irritability, apprehension, confusion and headache at serum sodium level less than 120 mEq/L.

Q. What is the main cause of CNS signs in the TURP syndrome?

CNS signs of TURP syndrome are caused by acute serum hypo-osmolality that allows movement of water into the cells and causes cerebral edema.

Q. What are the signs and symptoms of TURP syndrome?

- Cardiorespiratory:
  - Hypotension
  - Brady or tachyarrhythmias
  - Congestive heart failure
  - Pulmonary and hypoxemia
  - Myocardial infarction.
- Central nervous system:
  - Sodium level <120 mEq/L—agitation/confusion
  - Sodium level ≤ 102 mEq/L—coma visual disturbances
  - Sodium level ≤ 100 mEq/L—unconsciousness, convulsion.

- ECG changes at:
  - Serum sodium level <115 nmEq/L
  - QRS widening and ST segment elevation.
- Others:
  - Hyponatremia
  - Hypo-osmolality

Q. What are the factors influencing absorption of irrigation fluid?

The prostate gland contains large venous sinuses. So it is inevitable that irrigation solution would be absorbed. On an average, 20 mL fluid is absorbed per minute with a total 1–1.5 liters but absorption up to 4–5 liters has been recorded.

The amount of absorption depends upon the following factors:

- The height of the container of irrigation solution determines the hydrostatic pressure during the fluid into prostatic venous sinuses. The container must be kept as low as possible to achieve adequate flow of fluid at minimum pressure, usually 60–70 cm and never more than 100 cm. Higher pressure increases absorption.
- Times of resection is proportional to the amount of fluid absorbed. Prolonged surgery—more absorption.
- Blood loss—large blood loss implies a large number of open viens with more absorption of fluid.

Q. What is the treatment of TURP syndrome?

- Surgery terminated as soon as possible.
- Bleeding points should be coagulated.
- Furosemide—40 mg IV followed by checking of serum sodium.
- Respiration is supported with oxygen
- Intubation and controlled ventilation, if required.
- Administration of anticonvulsant, if patient is fitting.
- Intravenous fluid is stopped.
- Administration of hypertonic saline (1.8 to 3%) to restore serum sodium around 125 mEq/L. Correction should ideally not be faster than 1.5–2 mEq/L/hr for 3–4 hours, then 1 mEq/L/hr until symptomatic improvement or serum sodium >125 mEq/L.
- Admit the patient to ICU/HDU for management and regular measurement of serum sodium.

Q. What should be the anesthetic technique of choice for TURP? Why?

Spinal anesthesia is most commonly used technique for TURP and is considered as the anesthetic technique of choice. The reasons behind are:

- It provides adequate anesthesia for patient.
- It provides good relaxation of pelvic floor and perineum for surgeon.

- Signs and symptoms of water intoxication and fluid overload are recognized early as the patient remains awake.
- Spiral anesthesia, resulting in a sensory block to T_{10} level, eliminates the discomfort caused by bladder distension and other aspects of TURP procedure.

Sensory block above T_9 level should not be attained because the capsular sign (i.e. pain on perforation of prostatic capsule) would not be detected, if perforation occurs.

Q. What are the advantages of spiral anesthesia over continuous epidural anesthesia in TURP?

Advantages of spinal over epidural anesthesia are:

- Easier to perform in elderly patients
- Duration of surgery is not very long
- Incomplete block of sacral nerve roots is caused by epidural anesthesia, but is avoided with spinal anesthesia.

Q. What are the advantages of regional anesthesia over general anesthesia?

Regional anesthesia offers several advantages over general anesthesia:

- The amount of operative blood loss is reduced due to:
  - Decreased systemic blood pressure.
  - Decreased peripheral venous pressure.
- There is decreased incidence of deep vein thrombosis due to:
  - Sympathetic blockade by regional anesthesia results in increased blood flow, reducing deep vein thrombosis formation.
  - Regional anesthesia decreases hypercoagulable tendency in postoperative period and helps to maintain normal coagulation and plate. Function, thereby, reducing deep vein thrombosis formation. These benefits are obtained through modulation of neuroendocrine response to tissue injury and homeostasis of neuroendocrine system is better preserved after regional than after general anesthesia.
- TURP syndrome can be detected early during regional anesthesia.
- Bladder perforation with symptoms like dyspnea abdominal and shoulder pain, can also be detected earlier during regional anesthesia.
- Glycine toxicity with visual disturbances and blurred vision are also recognized earlier during regional anesthesia.
- Capsular sign (i.e. pain or perforation of prostatic capsule). It can easily be detected under regional anesthesia.

Q. What is obturator spasm? How it is controlled?

Obturator spasm occurs when obturator nerve, which runs adjacent to the lateral walls of the bladder, is directly stimulated by the diathermy current. It causes adduction of the legs and can seriously impair surgical access, increasing the risk of bladder perforation.

It can usually be controlled by reducing the diathermy current.

Q. What is bladder spasm? How it is relieved?

Bladder spasm is a painful involuntary contraction of bladder, occurring after any cystoscopy technique, most commonly in patients who did not have an indwelling catheter preoperatively. Diagnosis is supported by the failure of irrigation fluid to flow freely in and out of the bladder.

It is relieved by small doses of IV benzodiazepine, e.g. diazepam 2.5 to 5 mg or buscopan (hyocine butylbromide) 20 mg IV/IM.

Chapter

6

Laparoscopic Cholecystectomy in a Middle-aged Man with Obstructive Jaundice

Q. What is jaundice?

Jaundice refers to the yellow appearance of the skin, sclera, and mucous membrane resulting from an increased bilirubin concentration in body fluid.

Q. When jaundice is clinically detectable?

It is usually clinically detectable when plasma bilirubin exceeds 3 mg/dL (normal serum bilirubin is 0.2–1.2 mg/dL).

Q. What are the sites for detection of jaundice?

Bulbar conjunctiva, inner surface of lips and under surface of the tongue. Examination should be done in bright daylight and not in artificial light.

Q. What are the types of jaundice?

There are mainly 3 types of jaundice:

1. *Hemolytic jaundice*: It results from increased destruction of red blood cells causing increased bilirubin production. Plasma bilirubin is usually less than 6 mg/dL and liver function tests (LFTs) are normal. It is mild type jaundice.
2. *Hepatocellular jaundice*: It results from an inability of the liver to transport bilirubin into the bile, due to parenchymal liver disease. In addition, swelling of the cells and edema due to disease cause obstruction of the biliary canaliculi. Concentration of both conjugated and unconjugated bilirubin in blood, increase.
3. *Cholestatic jaundice*: Cholestasis may be due to failure of the hepatocytes to generate bile flow, obstruction of bile flow in the bile ducts in the portal tracts or obstruction of bile flow in the extrahepatic bile ducts. Clinical features comprise those due to cholestasis, secondary infection and underlying disease.

Q. What is neonatal jaundice?

Jaundice is common after 48 hours in most preterm and some term neonates and considered physiological. Neonates developing jaundice before 48 hours or 7 days after birth needs to be investigated.

Q. What investigations are done in a patient with jaundice?

- Liver function test
- Hepatobiliary imaging: Demonstrates hepatomegaly, intrahepatic tumors and portal hypertension. This includes: (i) Ultrasonography, (ii) computed tomography scanning, (iii) magnetic resonance imaging (iv) endoscopic retrograde cholangiopancreatography.
- Liver biopsy: Precise nature of hepatic damage can be detected.

Q. What are the biochemical tests for liver function and their implications?

Tests	Normal range	Hemolytic jaundice	Hepatocellular jaundice	Obstructive jaundice
1. Serum bilirubin – Conjugated – Unconjugated	 0.1–0.3 mg/dL 0.2–0.7 mg/dL	 Increased (1–4 mg/dL)	 Increased --	 Increased Rarely exceeds 3.5 mg/dL
2. Serum albumin – Total protein	3.5 – 5.5 mg/dL 6.5 – 8.4 mg/dL	Normal	Decreased	Unchanged
3. Alkaline phosphatase	30–115 U/L	Normal	Increased +	Increased ++++
4. Prothrombin time	INR of 1.0 – 1.4 after vit K 10% increase in 24 hours	Normal	Prolonged does not respond to vit K	Prolonged response to parenteral vit K
5. Aminotranferases – ALT – AST	 5 – 35 units/L 5 – 40 units/L	Normal	Much increased	Minimally increased

Aminotransferases though included in LFTs, do not assess function of liver, rather are indicative of liver cell injury or dysfunction. Glutathione-S-transferase (GST) is another indicative of liver cell injury. GST is rapidly released into the circulation after hepatic injury and because of its short plasma half-life (90 min), monitoring of plasma concentration of GST permits rapid identification of continuing or resolving cellular damage.

Q. What are the indices of obstructed bile flow?

- *Alkaline phosphatase (AP):* Elevation of serum AP disproportionates to the changes of AST and ALT occurs with intrahepatic or extrahepatic obstruction to bile flow.

- *5'-nucleotidase (5'NT):* Elevated serum levels are solely hepatocellular in origin. It is markedly increased in intrahepatic or extrahepatic biliary obstruction. 5'NT correlates closely with AP levels and is so specific for liver disease. It is used to determine if an elevated AP level is hepatic in origin.

Q. How bilirubin is formed? What are the types of bilirubin? How it is detected? What does the serum bilirubin level indicate?

- *Conjugated bilirubin:* Water soluble direct reaching form.
- *Unconjugted bilirubin:* Lipid soluble indirect reaching form. Serum levels of bilirubin are determined by Van den Bergh reaction. Serum bilirubin levels confirm the severity of jaundice and determine the extent of its conjugation.

Q. What are the purposes of ERCP?

Purposes of Endoscopic retrograde cholangiopancreatography (ERCP) include:

- Rapid diagnosis and localization of different causes of jaundice due to obstruction of main bile duct. Needle or forceps biopsy and brush cytology give specific diagnosis of stricture of the biliary tree.
- Therapeutic role in the treatment of jaundice allowing removal of bile duct stone or replacement of stents which facilitates passage of bile in the duodenum.
- Sphincterotomy done using cutting diathermy wires opens ampulla and allows delivery of stone in bile duct.
- ERCP deploys entire pancreatic duct system, helping diagnosis of chronic pancreatitis and defining cases those are benefited from surgery.
- Needle biopsy or brush cytology of pancreatic duct can be done in pancreatic carcinoma.

Mortalities due to pancreatitis occur due to bleeding, perforation and infection.

Q. What are the functions of liver?

Functions of liver are:

- *Blood reservoir:* The liver is a vital reservoir of blood in human. It contains nearly 10–15% of total blood volume. With intense SNS stimulation, almost 80% of the blood content can be expelled within a second. Anesthetics suppressing SNS outflow impair this reservoir function.
- *Regulator of blood coagulation:* The liver is responsible for the synthesis of factors involved in coagulation, anticoagulation and fibrinolysis. All procoagulation factors are derived from liver. The liver also modulates production through the synthesis of thromboplastin. Precursor proteins for vitamin K-dependent coagulation factors are synthesized in the liver. Vitamin K catalyses the activation of these factors (factors II, VII, IX and X).

- *Endocrine function:* The liver has important endocrine function. It synthesizes and secretes essential hormones like insulin-like growth factor I, angiotensinogen and thromboplastin. Angiotensinogen is a precursor of angiotensin II, playing a major role in the regulation of fluid electrolyte balance. The liver is a principle site for hormone biotransformation and catabolism. Thyroxine (T4) is actively taken up by the liver and converted to triiodothyronine (T3). It also synthesizes thyroid binding globulin. Corticosteroids, aldosterone, estrogen, androgens, insulin and antidiuretic hormones are all inactivated by the liver.
- *Immunological function:* Liver is the largest reticuloendothelial organ in the human body. Hepatic macrophages (Kupffer cells) account for nearly 10% of the liver mass. Kupffer cells protect and defend the body against foreign intrusions. Before entering central circulation, the splanchnic venous blood is filtered by Kupffer cells, which degrade toxins, process antigens and phagocytic bacteria. Kupffer cells are also important modulators of inflammation. They attenuate inflammatory responses by removing inflicting substances from blood stream.
- *Excretion of bilirubin:* Bilirubin is the end product of heme degradation. About 75% bilirubins are derived from breakdown of heme moiety of hemoglobin from senescent erythrocytes by reticuloendothelial system and the remainder from breakdown of nonerythrocytic hemoproteins including cytochrome P_{450}. After separation of protein from heme moiety by reticuloendothelial cells,the heme is oxidized by heme oxygenase system to biliverdin. Biliverdin is rapidly converted to unconjugated bilirubin and is then released into blood stream where it combines to albumin. The albumin bilirubin complex is transported to the liver and passes through the fenestrations in the sinusoidal lining cells to come into direct contact with hepatocyte plasma membrane. Binding proteins on plasma membrane dissociate the unconjugated bilirubin from albumin before uptake into the hepatocyte where bilirubin binds to endoplasmic protein and transported to endoplasmic reticulum. The bilirubin is then conjugated with glucuronic acid. This water soluble conjugated bilirubin is actively excreted into the canaliculi and a small portion enters plasma. Conjugated bilirubin passes unchanged through the biliary tree and proximal small intestine. In the terminal ileum and large intestine, most of the conjugated bilirubin is converted to urobilinogen. Some urobilinogen is absorbed and reaches the liver by mesenteric blood. The liver extracts the urobilinogen and returns it to the intestine (enterohepatic circulation). Eighty-percent urobilinogens are excreted in the stool. Ten-percent excreted in the urine and the rest is reabsorbed.
- *Metabolic function:* The liver plays a major role in the regulation of carbohydrate, protein and lipid metabolism. It is also the major site for catabolism of various hormones involved in regulating metabolism including insulin, glucagon, glucocorticoid, thyroxin and growth hormones.

Carbohydrate metabolism: The liver plays an important role in maintaining euglycemia. After meal, hyperglycemia is prevented by insulin-mediated hepatic extraction of glucose from portal blood. Excess glucose in the liver is converted into glycogen. During fasting, hypoglycemia is prevented by glycogenolysis. Glycogen stores are mobilized and converted to glucose and released into circulation. When glycogen stores are depleted, the process of gluconeogenesis converts certain amino acids to glucose.

Protein metabolism: The liver is the major site for protein K amino acid metabolism. Ingested proteins are hydrolyzed in the digestive tract to form amino acids, which are absorbed, transported to liver via portal vein and finally reach hepatocytes. When necessary, liver is able to synthesize nonessential amino acids and use them to synthesize biologically essential proteins. Protein catabolism occurs primarily in the liver. Protein enters the hepatocytes which are then degraded into their component amino acids in the lysosome. These then form substrate for glucose production through gluconeogenesis, enter lipid metabolism pathways or are further catabolized via transamination and deamination to form urea which is excreted by kidney.

Synthesis of important proteins: The liver produces various proteins with important extrahepatic functions. Hepatocytes synthesize albumin, acute phase proteins, all coagulation factors and their inhibitors (except factor VIII), ceruloplasmin and most of alpha-globulin and beta-globulins. Albumin constitutes 10% of the proteins synthesized by liver and 60% of total plasma proteins. It is the primary determinant of colloid oncotic pressure. It is also an important transport vehicle for metabolites and hormones. Albumin binds to a variety of pharmacological agents and so hypoalbuminemia has important consequence for the pharmacokinetic and pharmacodynamics of commonly administered drugs.

Lipid metabolism: The liver is the major site for synthesis of fatty acids from excess of sugar, protein and lipid. Fatty acids are vital for cellular function because they represent major energy source for heart and skeletal muscle. Cholesterol is an essential constituent of biological membranes and an important precursor of steroids, hormones, vitamins and bile acids. In fasting state, fatty acids are oxidized to acetyl co-A in liver which is further oxidized to CO_2 and water or converted to ketone bodies, which are important fuel for extrahepatic organs (e.g. brain, muscle and kidney).

Pharmacokinetics: Drug metabolism primarily occurs in the liver. The liver influences the plasma concentration and systemic availability of most orally and parenterally administered drugs through synthesis of drug-binding proteins. Plasma proteins especially albumin and alpha acid glycoproteins act as sink to decrease free drug concentration. Changes in the concentration of plasma proteins often modify dose response relationship of drugs. Hepatic biotransformation is the metabolism of drugs by hepatocytes into inactive water soluble substances those can be excreted into the bile or urine for elimination from the body.

Q. What are the causes of cholestatic (obstructive) jaundice?

Causes of the cholestatic jaundice may be intrahepatic or extrahepatic.

Intrahepatic	Extrahepatic
1. Primary biliary cirrhosis 2. Primary sclerosing cholangitis 3. Alcohol 4. Drugs 5. Viral hepatitis 6. Postoperative 7. Pregnancy	1. Choledocholithiasis 2. Carcinoma—ampullary, pancreatic, bile duct, secondary 3. Traumatic biliary stricture 4. Parasitic infiltration

Q. What are the clinical features of cholestatic jaundice?

- ***Early features***:
  - Jaundice
  - Dark urine
  - Pale stool
  - Pruritus.
- ***Late features***:
  - Malabsorption:
  - Weight loss
  - Steatorrhea
  - Osteomalacia
  - Bleeding tendency
  - Xanthoma.

Q. What investigations are specific in a patient with obstructive jaundice?

- Alkaline phosphatase (AP) level
- 5-nucleotidase (5 NT) as sensitive as AP, but takes days to show change after biliary obstruction
- Gamma-glutaryl-transferase (GGT) level
- Aminotransferases levels to confirm diagnosis. In obstructive jaundice, there is greater elevation of alkaline phosphatase and Gamma-glutaryl transferase level compared to the aminotransferases
- Ultrasound—performed to identify any biliary dilatation
- Cholangiography or ERCP—If dilated bile duct is identified.

Q. What is the vascular supply of liver?

The liver receives about 25% of cardiac output and has an average blood flow of 100–130 mL/min/100 g. There are two major sources of blood to the liver—the hepatic artery and the portal vein.

The common hepatic artery arises from the celiac trunk, sends cystic artery and then enters liver. The hepatic artery delivers about 25% of the total hepatic blood flow but 50% of hepatic O_2 delivery. The portal vein is formed by the union of the splenic and superior mesenteric veins receiving blood from

entire digestive tract, spleen, pancreas, gall bladder. The portal vein provides 75% of total hepatic blood flow and 50% of hepatic O_2 delivery. The portal vein has numerous unimportant tributaries which form large porta hepatic shunts in cases of portal hypertension (e.g. esophageal varices). The portal vein and hepatic artery enter the liver at porta hepatis. Hepatic arterial pressure is similar to aortic pressure while the mean portal vein pressure is 6–10 mm Hg. These two afferent vascular systems merge in the sinusoid. From hepatic sinusoid drain into central vein and then flow through sublobular veins that join to form right, middle and left hepatic veins which are joined to inferior vena cava (IVC).

Q. How hepatic blood flow is regulated?

Intrinsic and extrinsic mechanisms play important role in the regulation of hepatic blood flow. Intrinsic mechanism, hepatic arterial buffer response (HABR), metabolic control and pressure flow autoregulations—work independently of neurohumoral factors.

Intrinsic regulation:

- *Hepatic arterial buffer response (HABR)*: It is the most important intrinsic mechanism for regulation of hepatic blood flow. With an intact HABR, changes in portal venous flow cause reciprocal changes in hepatic arterial flow. The HABR involves synthesis and washout of adenosine (vasodilator) from periportal region. With decreased portal venous flow, adenosine accumulates and lowers arteriolar resistance, increasing hepatic arterial flow with increased portal venous flow; adenosine is washed-out and arteriolar resistance increases resulting in decreased hepatic arterial blood flow. The HABR preserves hepatic oxygen supply most effectively and disorders that decrease or abolish the HABR render the liver more vulnerable to hypoxic injury.
- *Metabolic control*: Decrease in oxygen tension or the pH of portal venous blood flow lead to increase in hepatic arterial and portal venous flow. Post prandial hyperosmolality increases in hepatic arterial and portal venous flow. Arterial hypoxemia, hypercapnia, alkalosis also modulate distribution of blood flow within the liver.
- *Pressure flow autoregulation*: The mechanism involves myogenic responses of vascular smooth muscles to stretch. During a hypertensive episode, increase in transmural pressure stretches arterial smooth muscle, resulting increase in myogenic tone (vasoconstriction), which prevents increase in organ blood flow. Hypotension lowers transmural pressure and myogenic tone (vasodilatation), which helps to sustain organ perfusion during systemic hypotension. The hepatic artery exhibits pressure flow autoregulation in metabolically active liver (post prandial) but not in fasting state (prevalence status of surgical patients). Pressure flow autoregulation is nonexistent in the portal circulation. Therefore, decrease in systemic blood pressure (as occurs during anesthesia) leads to proportional decrease in portal venous flow.

Extrinsic regulation:

- *Neural control*: Fibers of vagus, phrenic and splanchnic (post ganglionic sympathetic fibers from T_6–T_{11}) nerves enter liver and innervate the terminal arterioles and venules. Sympathetic nervous system is an important regulator of splanchnic vasculature. When sympathetic tone decreases, splanchnic reservoir volume increases. Sympathoadrenal stimulation translocates blood volume from the splanchnic reservoir to the central circulation. Vagal stimulation causes redistribution of intrahepatic blood flow by altering the tone of presinusoidal sphincters.
- *Humoral control*: Injection of epinephrine into hepatic artery induces biphasic response—vasoconstriction (alpha stimulation) followed by vasodilation (beta stimulation). Injection of epinephrine into portal vein causes only vasoconstriction (alpha response). The dopamine released during sympathoadrenal activation has weak vasoactive effect in the liver. Glucagon induces relaxation of hepatic arterial smooth muscle. Angiotensin-II constricts hepatic arterial and portal venous beds. It markedly decreases both mesenteric arterial and portal venous flow. Vasopressin elevates splanchnic arterial resistance but lowers portal venous resistance.

Q. What are the effects of volatile agents on hepatic blood flow and oxygen delivery?

As all halogenated anesthetics depress cardiac output, it is predictable that hepatic blood flow and oxygen delivery will decrease during general anesthesia with volatile agents. The potential for causing hepatic ischemia differs with each agent.

Halothane: Various studies indicate that halothane is more likely than other inhaled anesthetics to produce liver injury because it causes most cardiovascular and respiratory depression as well as greatest reduction in hepatic arterial flow. Consequently halothane produces or exacerbates hepatic hypoxia when blood flow to the liver is critically limited and oxygen supply is inadequate.

Enflurane: Decreases hepatic blood flow and splenic perfusion more than any other halogenated vapor in clinical use, except halothane. It induces decrease in portal venous blood flow with decrease or unchanged hepatic arterial blood flow. Decrease in splanchnic perfusion is in parallel with decreased mean arterial pressure (MAP) and cardiac output. It also increases splanchnic oxygen extraction and lowers both hepatic venous and mixed venous oxygen saturation.

*Desflurane***:** Decreases hepatic blood flow. One MAC desflurane prior to skin incision decreases hepatic blood flow by 30%. It can markedly reduce oxygen delivery to the liver and small intestine and thereby decreases the oxygen reservoir capacity of those organs.

Isoflurane: Less-likely to cause or contribute to hepatic injury than halothane or enflurane. It preserves hepatic blood flow and oxygen delivery even during open laparotomy.

Sevoflurane: Preserves hepatic blood flow and oxygen delivery to the liver. During elective surgical procedures under sevoflurane anesthesia (1 or 2 MAC), there is significant reduction in MAP but hepatic blood flow is maintained at preanesthetic level due to intact hepatic arterial buffer response. Sevoflurane is most effectively inhaled anesthetic for maintaining both blood flow and oxygen delivery to the liver. Sevoflurane better preserves hepatic blood flow and oxygen delivery than halothane, enflurane and desflurane. Its effects on hepatic perfusion and metabolic function are similar to those of isoflurane. It is least toxic than all other inhaled anesthetics and induces least liver injury.

Q. How evaluation of liver is done?

- ***Clinical assessment***
  - *History*: Symptoms relevant to and major risk factors for liver dysfunction can be elicited from proper history taking.
- ***Physical examination*:** Clinical findings consistent with liver dysfunction can be detected by proper through examination of the patient.
- ***Laboratory tests***: Information obtained from history and physical examination of the patient directs to the laboratory test to be done for detection of the underlying cause.
  - *For detection of hepatocellular injury*:
    - Aminotransferases (ALT and AST): The usual cause of elevated levels is hepatic injury.
    - Lactate dehydrogenase (LDH): Elevated serum level indicates hepatocellular injury, extrahepatic disorders or both. Elevation in LDH is accompanied by elevation of AST and ALT levels and rarely provides information about liver injury beyond that provided by AST and ALT.
    - Glutathione-S-transferase (GST): Sensitive and specific test for drug-induced liver injury. AST and ALT reside in zone I. GST localizes in zone III (centrilobular region), containing hepatocytes with highest susceptibility to hypoxic injury. Therefore, GST is more sensitive than AST and ALT as a marker of liver injury.
  - *For detection of cholestatic disorders*:
    - Alkaline phosphatase (AP): Used to screen for disorders of liver or biliary tree. As AP exists in various organ (Bone and kidney) other than liver, it lacks specificity for hepatobiliary disease. Serum AP remains normal for 2 days after the onset of biliary obstruction and remains elevated for days after bile flow is restored due to long half-life. Extreme increases in AP suggest: (i) primary biliary cirrhosis and choledocolithiasis or (ii) hepatic malignancy compressing small intrahepatic bile duct.

- 5′ nucleotidase (5′NT): Most of the serum 5′NT is derived from the hepatobiliary tree. It is markedly increased with intrahepatic and extrahepatic biliary obstruction with modest increase in other hepatocellular disorders. It is very much specific for liver disease. Changes in the serum level of AP due to hepatic biliary disease are followed by changes in 5′NT level. 5′NT level determines if elevated AP level is hepatic in origin or not.
- Gamma- glutaryl transferase (GGT): Found in high concentration in epithelial cells lining biliary ductules. Serum GGT level is most sensitive indicator of biliary tract disease.
- Serum bilirubin: Most widely used for hepatic function. Serum bilirubin levels above 5 mg/dL in association with other LFT abnormalities signify presence of liver disease. Elevated levels of conjugated bilirubin indicate impaired intrahepatic excretion of bilirubin or extrahepatic obstruction.

– *For assessment of synthetic function of liver*: Measurement of serum albumin level and assay of the coagulation factor are most widely used methods for assessing hepatic synthetic function.
- Serum albumin: A decrease in serum albumin concentration indicates worsening hepatic function in presence of chronic liver disease in absence of other factors. Because half-life in serum is 20 days, the serum albumin level is not a reliable indicator of hepatic protein synthesis in acute liver disease.
- Prothrombin time (PT): Liver derived procoagulants have short half-life ranging from 4 hours for factor VII to 4 days for fibrinogen. PT or INR (International normalized ratio) are widely used to evaluate or monitor patient with acute liver dysfunction. Prolonged PT in liver dysfunction reflects low blood flow level of factor VII_a, having a shortest half-life. PT is also used as a prognostic indicator. PT depends on normal hepatic synthesis of clotting factors and sufficient uptake of vitamin K. In patients with obstructive jaundice, prolonged PT indicates vitamin K deficiency rather than impaired hepatic synthetic function.

– *For assessment of hepatic blood flow and metabolic capacity*: Estimation of the dye indocyanine green (ICG) from the blood provides an estimate of hepatic perfusion and hepatocellular function because it is highly extracted by liver following intravenous injection (about 70–90%).

- ***Hepatobiliary imaging***:
  – *Plain radiography*—limited role.
  – *Ultrasonography*—primary screening test for hepatic disease, gall stones, biliary tract disease—suspected from symptoms, jaundice, hepatomegaly, mass lesion and abnormal LFT.
  – *CT*—a complementary test to ultrasonography and provides information on liver texture, gall bladder disease, bile duct dilatation and mass lesion in liver and pancreas. Lesion can be biopsied under CT guidance.

- *MRI*—evolving method for evaluation of hepatocellular disease. Magnetic resonance cholangiopancreatography is used to visualize the bile and pancreatic ducts and to delineate the most proximal extent of biliary tract obstruction when planning for operative resection or drainage.
- *Percutaneous transhepatic cholangiography (THC)*—used to determine level and cause of biliary obstruction, presence of cholestasis and determines if the proximal cholangiocarcinoma is surgically resectable or not. THC is used for balloon dilatation of biliary stricture, for placement of an external drain.
- *ERCP*—endoscopic visualization of ampulla of Vater to inject selective contrast media into pancreatic and common bile duct which are then imaged radiographically. It is the imaging technique of choice in patients with choledocholithiasis because: (i) sphincterotomy and stone extraction can be done (ii) biopsies, brushing, balloon dilatation and stent insertion can also be done.
- *Liver biopsy*—it provides the only means of determining the precise nature of hepatic damage.

Q. What is Murphy's sign? How it is elicited?

Murphy's sign is a clinical finding in a patient with acute cholecystitis. The patient is asked to breathe deeply and gall bladder is palpated in normal way. At the height of inspiration, the breathing stops with a gasp as the mass is felt.

Q. What is Courvoisier's law?

Courvoisier's law states that in presence of obstruction jaundice, a palpable gall bladder makes gall stone obstruction of common bile duct an unlikely cause. A palpable gall bladder with obstructive jaundice usually signifies malignant disease.

Q. What are the advantages of laparoscopic surgery?

- Reduced wound size, tissue trauma and postoperative pain
- Reduced postoperative ileus, postoperative pulmonary complications facilitating faster recovery
- Early mobilization, more rapid return to normal work and shorter hospital stay reducing healthcare cost
- Improved cosmetic results and patient's satisfaction.

Q. What are the primary requisites for laparoscopic surgery?

- Creation of pneumoperitoneum by insufflation of gas (usually CO_2) to separate the abdominal wall from the viscera
- *Patients positioning*: Trendelenburg position for lower abdominal procedures. Reverse Trendelenburg position for upper abdominal procedures. Cholecystectomy usually involves change to steep reverse Trendelenburg with left lateral tilt to facilitate retraction of gall bladder

fundus and to minimize the diaphragmatic dysfunction associated with induction of pneumoperitoneum.

Q. Which gas is preferred for inducing pneumoperitoneum? Why CO_2 is chosen?

CO_2 is the insufflation gas of choice for inducing pneumoperitoneum during laparoscopic surgery for following reason:

- CO_2 does not support combustion and so can be used safely with diathermy or laser
- High blood solubility of CO_2 and its capability of pulmonary excretion reduces the risk of adverse outcome if gas embolism occurs
- CO_2 insufflation into the peritoneal cavity increase arterial CO_2 tension ($PaCO_2$), which is managed by increasing minute ventilation
- CO_2 is nontoxic.

CO_2 absorption is greater during extraperitoneal insufflations than during intraperitoneal insufflations. $PaCO_2$ progressively rises to reach a plateau 15–30 minutes after insufflations. Any significant subsequent increase in $PaCO_2$ indicates CO_2 subcutaneous emphysema.

Q. What are the monitoring techniques used during laparoscopic surgery?

Standard intraoperative monitoring is recommended for all patients undergoing laparoscopic surgery. $EtCO_2$ is most commonly used, noninvasive indicator of $PaCO_2$ in assessing the adequacy of ventilation during laparoscopic procedure.

$PaCO_2$ is maintained close to free insufflation levels by increasing minute ventilation by 12–16% and $EtCO_2$ provides a reasonable approximation of $PaCO_2$ in healthy patients undergoing laparoscopic surgery.

Patients with pre-existing cardiopulmonary disease sustain significant increase in $PaCO_2$ during CO_2 insufflation, which is not reflected by comparable increase in $EtCO_2$. Therefore, $EtCO_2$ may not be a reliable index of $PaCO_2$ during CO_2 insufflation in these patients and $PaCO_2$ should be monitored at times during the procedure to avoid adverse outcome.

Q. What is Hasson technique for gas insufflations?

The Hasson technique is the method for open insertion of the trocar for gas insufflation, thereby guaranteeing pneumoperitoneum and avoiding dangers of blind trocar insertion. The surgeon makes a small incision just below the umbilicus and locates abdominal fascia under direct vision. A small incision is made through the fascia and underlying peritoneum. A finger is placed into the abdomen to make sure that there is no adherent abdominal viscera. Pneumoperitoneum is then created using a specialized trocar.

Q. Why capnography is most essential during laparoscopic surgery?

Capnography is most essential because:

- The $EtCO_2$ is a noninvasive indicator of $PaCO_2$ in assessing the adequacy of ventilation during laparoscopic procedure
- Subcutaneous emphysema may occur as a complication during CO_2 insufflation, which is detected by a sudden rise of $EtCO_2$ due to absorption of CO_2. CO_2 absorption is greater during extra peritoneal insufflations
- Gas embolism may occur due to inadvertent intravenous passage of CO_2 into open vessels during insufflations, which is detected by decrease in $EtCO_2$ due to reduction in pulmonary blood flow.

Q. Why and what cardiorespiratory changes occur during laparoscopic surgery?

The cardiorespiratory changes occurring during laparoscopic surgery relate to combined effects of intra-abdominal insufflations of CO_2 to create pneumoperitoneum (alteration of patient's position) and the effect of systemic absorption of CO_2.

- *Problems related to pneumoperitoneum:*
  - ***Cardiovascular effects due to pneumoperitoneum:*** Hemodynamic changes observed during laparoscopy result from combined effect of pneumoperitoneum, patient's position, anesthesia, and hypercapnia from the absorbed CO_2. In addition, reflex increases in vagal tone and arrhythmias can also develop.

    Peritoneal insufflations to intra-abdominal pressure (IAP) higher than 10 mm Hg induce significant alteration of hemodynamics. This disturbances are—decreased cardiac output, increased arterial pressures and elevation of systemic and pulmonary resistance. Heart rate (HR) remains unchanged or increases slightly. Decrease in cardiac output is proportional to increase in IAP. Fall of cardiac output is up to 10–30% during peritoneal insufflations with both head down and head up positions. The decrease in cardiac output is due to decreased venous return resulted from compression of IVC by pneumoperitonem, pooling of blood in the legs and increased venous resistance. The reduction in venous return and cardiac output can be attenuated by increasing circulating volume or tilting the patient to slightly head down position before creation of pneumoperitoneum. Ejection fraction of left ventricle does not decrease till IAP increases to 15 mm Hg. Systemic vascular resistance increases during pneumoperitonem causing increase in afterload. This increased afterload is tolerated by patients with normal heart but can be deleterious in patients with cardiac disease. Increase in SVR is attenuated by Trendelenburg position and aggravated by reverse Trendelenburg position. Increased SVR can be corrected by vasodilating anesthetic agents—isoflurane or direct vasodilating drugs like nitroglycerine and nicardipine. Both mechanical and neurohumoral factors are involved in mediating the increase in SVR and, thereby, increased afterload. During pneumoperitoneum, there occurs release of catecholamines, the rennin-angiotensin system and especially vasopressin resulting increased afterload. Mechanical stimulation of

peritoneal receptors also results in increased vasopressin release, SVR and arterial blood pressure. IAP <12 mm Hg causes minimum alteration in hemodynamic function. Use of alpha-adrenergic agonists like clonidine or dexmedetomidine and beta-blocking agents significantly reduces hemodynamic changes and anesthetic requirement. Use of high doses of remifentanyl almost completely prevents the hemodynamic changes. Increased IAP and head up position—lower limb venous stasis.

- ***Effects of pneumoperitoneum on renal function are:*** Decreased urine output, renal plasma flow and GFR. Cardiac arrhythmias during laparoscopy result from reflex increases of vagal tone from sudden stretching of the peritoneum during creation of pneumoperitoneum. Bradycardia, cardiac arrhythmias and asystole can develop. Vagal stimulation is accentuated in light plane of anesthesia or the when the patient is on beta-blocker. Treatment: Interruption of insufflations, atropine administration and deepening of anesthesia after recovery of heart rate.

  Effect of ventilator changes due to pneumoperitoneum: Pneumoperitoneum decreases thoracopulmonary compliance and increase in peak airway pressure. Reduction in functional residual capacity (FRC), development of atelectasis due to elevation of diaphragm and changes in the distribution of pulmonary ventilation and perfusion from increased airway pressure may result. Hypoxemia due to reduction in FRC is uncommon in healthy patients but significant hypoxemia because of ventilation-perfusion mismatch and intrapulmonary shunting may occur in obese patients and in patients with pre-existing pulmonary disease. During CO_2 pneumoperitoneum, there is increase in $PaCO_2$ due to absorption of CO_2 from peritoneal cavity, impairment of pulmonary ventilation and perfusion by abdominal distension, patient's position and volume controlled mechanical ventilation. In healthy patients, main cause of increased $PaCO_2$ is absorption of CO_2 from abdominal cavity but in patients with cardiorespiratory problems, ventilator changes also contribute to increasing $PaCO_2$. PaO_2 values and intrapulmonary shunts do not significantly change during laparoscopy. $PaCO_2$ should be maintained within physiological range by adjusting mechanical ventilation. Increase in $PaCO_2$ can be corrected easily by a 10%–20% increase in alveolar ventilation.

- *Problems related to patient position:* Patient positioning depends on the site of surgery. Head down tilt is used for lower abdominal and pelvic surgeries. Head up position is required for upper abdominal surgery. The steepness of the tilt affects the magnitude of the pathophysiologic changes produced by the positions.
  - ***Cardiovascular effect:*** In normotensive subjects, the head-down position results in an increase in CVP and cardiac output. The baroreceptor reflex response to increased hydrostatic pressure consists of systemic vasodilatation and bradycardia. These hemodynamic

changes remain insignificant in healthy patients, but are of greater magnitude in patients with coronary artery disease with poor ventricular function leading to deleterious myocardial oxygen demand. Trendelenburg position affects cerebral circulation in patients with low intracranial compliance leading to increased intraocular venous pressure which can worsen acute glaucoma. With head up position, a decrease in cardiac output and MAP results from reduction in venous return. This decrease in cardiac output is added to hemodynamic changes due to pneumoperitonem. Reverse Trendelenburg position results in venous stasis in the legs and pneumoperitoneum further increases blood pooling in the legs.

- ***Respiratory changes:*** The head down position results in atelectasis. Steep head down position decreases FRC, total lung volume and pulmonary compliance, which are more marked in obese, elderly and cardiorespiratory compromised patients but are tolerated by healthy subjects. The head up position is usually considered to be more favorable to respiration.

Q. What are the complications of laparoscopic surgery?

The intraoperative complication during laparoscopic surgery results from creation and maintenance of CO_2 pneumoperitoneum. These are:

- *Injuries*
  - Major vascular injury during insertion of Veress needle or trocar. Hemorrhage can occur due to insertion of Veress needle into major intra-abdominal vessels. Concealed bleeding into retroperitoneal space is diagnosed late and associated with unexplained hypotension.
  - Gastrointestinal tract perforation, hepatic and splenic tears and mesenteric laceration may also occur during insertion of Veress needle.
- *Subcutaneous emphysema*
  - Extraperitoneal insufflations of CO_2 may occur if the Veress needle tip lies in subcutaneous, preperitoneal or retroperitoneal tissue during insufflations. Indicated by development of crepitus over abdominal wall and sudden rise in $EtCO_2$ due to increased CO_2 absorption.
- *Gas embolism*: Gas embolism during laparoscopic procedures occurs due to inadvertent intravenous placement of Veress needle with passage of CO_2 into vessels during insufflations. This is associated with profound hypotension, cardiovascular collapse, hypoxemia, mill wheel murmur, and decrease in $EtCO_2$ due to reduced pulmonary blood flow.
  Management:
  - Head down with left lateral position of the patient
  - Surgeon notified
  - Gas insufflation is stopped.
  - Nitrous oxide is discontinued
  - 100% oxygen administered
  - Right heart catheter is aspirated

  - Vasopressors/inotropes
  - Chest compression.
- *Cardiac arrhythmia*: Arrhythmias during laparoscopic surgery occur due to hypercapnia resulted from intraperitoneal CO_2 insufflation and increased vagal tone following peritoneal stretching, especially in light plane of anesthesia. Cardiac arrhythmias, bradycardia and asystole all may occur.
- *Endobronchial intubation*: Cephalad displacement of diaphragm during pneumoperitoneum results in cephalad movement of carina leading to endobronchial intubation, resulting decreased SpO_2 with increased airway pressure.
- Aspiration of gastric content due to increased IAP.
- Pneumothorax, pneumomediastinum and pneumopericardium.
  Pneumothorax: Although rare, is a life-threatening complication during laparoscopic procedures. The suggested mechanism includes trekking of insufflated CO_2 around the aortic and esophageal hiatus of the diaphragm into the mediastinum and then rupture into pleural space. Gas may also enter through anatomic defects in the diaphragm at outer crust and through a congenital defect at pleura-peritoneal hiatus. Presenting features are unexplained increase in airway pressure, hypoxemia, hypercapnia, and cardiorespiratory compromise with hypotension.
  Treatment: Immediate abdominal deflation and chest tube decompression followed by chest radiography.
  Pneumomediastinum and pneumopericardium also can result during laparoscopic procedures.
  High IAP during insufflations contributes to rise these complications.

Q. What should be the anesthetic plan for the patient undergoing laparoscopic surgery?

- General anesthesia with endotracheal intubation and controlled ventilation is the safest and most commonly used anesthetic technique. It reduces the increase in $PaCO_2$ and avoids ventilator compromise due to pneumoperitoneum and Trendelenburg position. Controlled ventilation is adjusted to maintain $EtCO_2$ between 35 and40 mm Hg by a 15%–25% increase of minute ventilation.
- Nitrous oxide should better be avoided as it causes increased postoperative nausea and vomiting (PONV), bowel distension, and size of CO_2.
- High intra-abdominal pressure increases the risk of aspiration of gastric contents. Hence, cuffed endotracheal tube is preferred. ProSeal laryngeal mass airway (LMA) may be an alternative. IAP should be monitored and kept as low as possible to reduce hemodynamic and respiratory changes. Recommended IAP is 10–12 mm Hg. Standard intraoperative monitoring is done. $EtCO_2$ is most commonly used as a noninvasive indicator of $PaCO_2$ in assessing the adequacy of ventilation during laparoscopic procedures.
- Propofol can be used for induction of anesthesia. Choice of neuromuscular blocking drugs depends on the duration of surgery.

- Remifentanyl reduces the hemodynamic effect produced by pneumoperitoneum.
- Atropine should be readily available as there is reflex increase in vagal tone during laparoscopy. Reversal of residual neuromuscular blockade is done using neostigmine at completion of surgery.
- Liberal use of per-operative intravenous fluid reduces hemodynamic changes from pneumoperitoneum and PONV improving postoperative recovery.
- Hemodynamic monitoring should be continued in post-anesthetic care unit (PACU). Oxygen should be administered to all patients in PACU.

Q. What is the cause of shoulder pain after laparoscopic surgery and how it is prevented?

Shoulder pain after laparoscopic procedures is due to irritation of the diaphragm by residual gas in the abdominal cavity. It can be minimized by complete evacuation of gas from the peritoneal cavity before closure of the abdomen.

Q. What are the contraindications for laparoscopic surgery?

Absolute contraindications of laparoscopic surgery are rare. Pneumoperitoneum is undesirable in:

- Patients with increased intracranial pressure
- Patients with hypovolemia
- Patients with ventriculoperitoneal and peritoneal jugular shunts
- Patients with glaucoma
- Patients with congestive cardiac failure
- Patients with terminal valvular insufficiency
- Patients with compromised ventricular function
- Patients with renal failure.

Q. What are the alternatives to pneumoperitoneum for laparoscopic procedures?

The gasless laparoscopic technique avoids any gas for insufflations instead relying on an abdominal lift with a fan retractor to create an intra-abdominal space at atmospheric pressure. This can be considered for elderly patients or those with cardiopulmonary problems. Hand-assisted laparoscopic access can also be used. It increases technical difficulty and compromised surgical exposure combined with abdominal wall lifting and low pressure (5 mm Hg) pneumoperitoneum improves surgical conditions.

Q. What is the place of local and regional anesthesia in laparoscopic surgery?

Local anesthesia offers quicker recovery, decreased PONV, early diagnosis of complications and fewer hemodynamic changes. However, it is associated with patient anxiety and discomfort during intra-abdominal manipulation,

requiring supplementation with IV sedation. The combined effect of pneumoperitoneum and sedation can cause hypoventilation and arterial oxygen desaturation. Therefore, complex laparoscopic procedures cannot be managed with local anesthesia.

Regional anesthesia including epidural and spinal technique combined with Trendelenburg position can be used for gynecological laparoscopy. Laparoscopic cholecystectomy can be performed successfully in COPD patients using epidural anesthesia. Regional anesthesia reduces metabolic response, need for sedation and narcotics, produces better muscle relaxation and can be used for laparoscopic procedures. Shoulder tip pain from diaphragmatic irritation and discomfort from abdominal distension are completely alleviated using epidural anesthesia. Extensive sensory block (T_4 to L_5) is necessary for surgical laparoscopy.

Q. What is the management of patients with cardiac disease for laparoscopic surgery?

Anesthetic management

- *Pre-operative evaluation*: Echocardiography is done to see if the left ventricular ejection fraction is <30%.
- Intra-operative monitoring:
  - Intra-arterial line
  - Pulmonary artery catheter
  - Trans-esophageal echocardiography
  - Continuous ST segment analysis.

Intra-operative management

- Slow insufflations of gas
- Intra-abdominal pressure as low as possible
- Gasless laparoscopy may be a useful alternative to pneumoperitoneum
- Hemodynamic optimization is done before creation of pneumoperitonium by preload augmentation
- Patient is tilted after insufflation of gas.

Anesthesia: Remifentanyl, vasodilatation anesthetics and drugs (nicardipine and nitro-glycerine), cardiotonic agents.

Postoperative care: Slow recovery from anesthesia, clonidine is beneficial.

Q. What is the anesthetic management for a patient with COPD posted for laparoscopic cholecystectomy?

Preoperative respiratory optimization with bronchodilator therapy, treatment of super added infections with antibiotics and chest physiotherapy should be done. Controlled ventilation must be adjusted to maintain $EtCO_2$ between 35 and 40 mm Hg by increasing minute ventilation up to 15–25%. In patients with COPD, increase of respiratory rate rather than tidal volume is preferable for increasing minute ventilation.

If preoperative pulmonary function test shows low forced expiratory volume and vital capacity, the patient is likely to develop hypercapnia and acidosis during laparoscopic cholecystectomy. Increase in $PaCO_2$ during CO_2 insufflation is also not reflected by comparable increase in $EtCO_2$. Hence, it is essential to monitor $PaCO_2$ at times during the procedure to avoid adverse outcome.

Chapter

7

Child Aged 2 Years Posted for Repair of Cleft Lip and Palate

Q. Why these cases are challenge to the anesthesiologist?

Presents at young age:

- Associated with other congenital anomalies
- Varying degree of difficult airway
- Airway has to be shared with surgeons.

Q. What are the pathophysiological features of cleft lip and palate?

- Pharynx communicates with nasal fossae and oral cavity—impaired swallowing, breathing, hearing and speech
- Cleft → difficulty in creation of negative pressure → neonate cannot suck
- Difficulty in feeding, sucking, swallowing since birth → inadequate weight gain, anemia, nutritional deficiencies
- Lower respiratory tract infection secondary to regurgitation of fliud and food
- Abnormal anatomy of nasopharynx → Eustachian tube function is affected → middle ear effusion → hearing loss → communication problem
- Problem with phonation of P, R, T and D, etc.
- Secondary defect of tooth development
- Associated with congenital anomalies/syndromes with their special problems
- Psychological problem in school age.

Q. What are the syndromes associated with cleft lip and palate?

- Pierre–Robin sequence—difficult airway, postoperative respiratory obstruction
- Treacher Collin's syndrome—difficult airway
- Goldenhaur syndrome—difficult intubation, airway obstruction, cervical spine laxity, CHD (antibiotics needed).
- Klippel–Feil syndrome—difficult intubation, CHD—antibiotics needed.

Q. What are the nonsyndrome anomalies associated with cleft lip and palate?

Umbilical hernia, club foot, limb/ear deformities, CHD
V = Vertebral anomalies—spina bifida
A = Anal atresia
C = Cardiovascular anomalies—PDA, ASD, VSD
T = Tracheoesophageal fistula
E = Esophageal atresia
L = Limb anomalies.

Q. What monitors are used during surgery?

NIBP, ECG, pulse oximetry, end tidal capnometry, temperature probe, precordial stethoscope.

Q. What is the ideal anesthetic management?

Anesthetic management

- Preoxygenation.
- Induction with sevoflurane with N_2O to speed up induction—preferrable IV line is set up.
- Atropine premedication to decrease secretion immediately after IV line.
- No neuromuscular blocking agent should be given until one is sure that mask ventilation is possible.
- Lubricated dental role or gauge is placed in the gap of the palate to prevent airway obstruction by impacted tongue and tissue trauma with laryngoscope blade.
- Intubation done with RAE tube as it lies flat against tongue and chin—less kinking and dislodgement.
- Muscle relaxant (Atracurium) administered and ventilation controlled. Analgesia provided with fentanyl.
- ET tube is secured in the midline over the chin without distortion.
- Eyes are shut to prevent trauma.
- Positioning of the patient done carefully.
- Throat pack inserted by surgeon to prevent aspiration of blood and also to secure the position of ET tube
- ET tube position checked and confirmed
  - Immediately after intubation
  - Immediately after patient positioning
  - Immediately after throat pack
  - Immediately after insertion and opening of mouth gag (malposition of gag can lead to partial or complete obstruction of tube)
- Anesthesia is maintained with controlled ventilation using O_2, N_2O, fentanyl, atracurium, sevo/isoflurane to maintain normocapnea.
- Hypothermia should be prevented.
- At the end of the procedure, throat pack should be removed before extubation and oropharynx sucked to remove pooled blood and secretion. Traction suture placed through tongue and loosely tied.

- Neuromuscular blockade should be reversed.
- Extubation is done when child is awake, warm, comfortable with full control of airway reflexes and good limb movements.
- Elbow restrains are routinely used to keep child's hand away from the face. Child is kept in tonsillar position to make the airway clean and allow drainage of oral secretions.
- Postoperative analgesia produced with paracetamol/diclofenec suppository. Infraorbital nerve block in lip surgery and palatine nerve block I palate surgery. Local infiltration of the wound with LA can be done.
- Close monitoring for airway obstruction should be done at least for 24 hours.

Q. What are the causes of postoperative airway obstruction?

- After palate surgery reduction in airway size.
- Edema of tongue due to pressure of mouth gag.
- Mucosal swelling in the hypopharynx and flap edema.
- Increased oral secreation.
- Posterior displacement of the tongue.
- Laryngeal edema following ET intubation—subglottic edema.
- Residual effects of anesthetics and opioids.
- Overlooked throat pack.

Q. What positioning is recommended for cleft palate repair?

Maximum head extension. Folded blanket is placed under the shoulder in intrascapular region allowing the head to drop back, hyperextended into a head ring. Purpose is good surgical exposure, flow of blood away from the larynx and collect in the nasopharynx from where it is removed by suction.

Q. What are the complications of palate surgery?

- Obstruction of ET tube with blood, secretions or gag
- Inadvertent extubation during surgery
- Postoperative airway obstruction
- Bleeding with or without aspiration.

Q. How to protect the airway?

- Suctioning of pharynx and nasopharynx before removal of gag and extubation.
- Traction suture placed through the tongue and tied, loosely → traction on the suture stimulates respiration and clears airway.
- Lateral position with head dependent, turned to the side and hyperextended → blood and mucus accumulate in the dependent portion and roll out of mouth leading to decreased chance of aspiration.
- No oral or nasal airway.
- Elbow restraints.

Q. Where no throat pack is used during palate repair?

When palate repair is required, pharyngeal flap or pharyngoplasty is done, pack interferes neck which ensures blood pools in the posterior nasopharynx and sucked out prior to extubation.

Exaggerated neck extension can bring ET tube up and out of trachea.

Q. When lip and palate repair is ideally done?

Lip repair as early as 1st 3 months of age (can be done in neonate, if no airway problem) for better cosmetic, improved ability to suck and relieves parental anxiety.

Cleft palate repair is done before dentition and speech development.

Killian's rule of Ten

- For cleft lip—10 weeks, 10 pounds and 10 g Hb.
- For cleft palate—10 months, 10 kg and 10 g Hb.
- Restoration of normal speech and prevention of maxillary growth retardation.

Q. What is the consideration regarding premedication?

Sedative premedication:

- <6 months—not needed (no separation anxiety)
- >6 months—pedicloryl syrup (50 mg/kg), midazolam syrup (0.5 mg/kg)
- 30 minutes prior to surgery.

Q. What is the fasting guideline of ASA?

- Clear fluids – 2 hours
- Breast milk – 4 hours
- Infant formula, nonhuman milk, light meal – 6 hours
- Full meal – 8 hours

Q. What are the purposes of reconstruction surgery in lip and cleft palate?

- Cosmetic
- Psychological
- Functional:
  - Separates nasal and oral cavities
  - Improves swallowing mechanism
  - Improves speech mechanism
  - Prevents middle ear disease, improves hearing
  - Provides normal dental occlusion.

Q. How to replace the perioperative fluid in children?

Fluid management has three key issues:

- Fluid deficit due to food and fluid restriction.
  Maintenance fluid × hours of restriction.
  50% replaced in 1st hour and 25% in each of next 2 hours.

- Maintenance fluid Holliday-Segar formula (4:2:1)
  0–10 kg—4 mL/kg/hr
  Next 10–20 kg—add 2 mL/kg/hr
  >20 kg—add 1 mL/kg/hr for above 20 kg.

  24 hours requirement
  0–10 kg—100 mL/kg
  10–20 kg—1000 mL + 50 mL/kg for above 10 kg
  >20 kg—1500 + 20 mL/kg for above 20 kg.
- Third space loss—depends on surgical procedure and varies from 1 mL/kg/hr for minor surgery to 15 mL/kg/hr for major abdominal surgery.
- Blood loss.

Q. How blood volume in pediatric age group is calculated?

- Preterm—100–120 mL/kg
- Full term—90 mL/kg
- 3–12 months child—80 mL/kg
- >1 year—70 mL/kg
  EBV = Blood volume × Body weight (kg)

Q. How MABL is calculated ?

MABL = EBV (Starting hematocrit – Target hematocrit)/Starting hematocrit

Q. What is the significance of MABL?

If blood loss is equal to MABL and no further loss is anticipated—no need for transfusion of blood, otherwise consideration is to be made.

If blood is less than MABL—replacement is done with 3 mL of Ringer's lactate per mL of blood loss.

In children, fall in BP is a late sign of hypervolemia.

Q. What type of fluid is preferred in pediatric patient ?

A balanced salt solution for all deficits and third space loss.

Five-percent dextrose in 0.45% normal saline (NS) by piggyback infusion at maintenance rate satisfies concern for unrecognized hypoglycemia—blood glucose monitoring done.

High glucose level—chance of hypoxic brain damage.

Q. What precautions are taken if adrenaline containing solutions are used for hemostasis ?

Chance of arrhythmia with use of halothane, which is limited by maintaining normocapnea with IPPV. $EtCO_2$ monitoring is helpful. Lowering CO_2 level also helps to prevent vasodilation and excessive bleeding. Risk can also be reduced by using 0.25–1% lignocaine with 1 in 200,000 to 1 in 400,000 adrenaline, up to 10 μg/kg of adrenaline with lignocaine can be used in a normocapnic child.

Q. What are the important points noted in the preoperative period?

- Gestational age
- Feeding habit
- Sleep apnea
- Otitis media
- Nutritional anemia
- Associated congenital anomalies
- Chronic rhinorrhea should be differentiated from active respiratory infection
- Lung infection.

Q. How infraorbital nerve block is done ?

- Direct cutaneous injection surrounding the infraorbital foramina to surround the nerve.
- Intraoral injection through the superior labial sulcus at the apex of the canine fossa.

Q. Why bilateral clefts of lips and alveolus makes intubation difficult?

Protrusion of free premaxilla makes visualization of larynx difficult.

Premaxilla is supplied by an end artery and its injury may lead to tissue necrosis. So its protection from injury by laryngoscope blade makes the procedure difficult.

Chapter

8

A Case of Scoliosis Put up for Corrective Surgery

Q. What is scoliosis? How scoliosis is classified? How structural and nonstructural scolioses are differentiated?

The spine normally curves posteriorly in thoracic (kyphosis) and anteriorly in lumbar (lordosis) region. Scoliosis is abnormal lateral curvature of spine (commonly thoracic), associated with rotation of vertebrae.

Classification of Scoliosis

Idiopathic scoliosis: Cause is not clearly known. This is further divided into: (i) Infantile (0– 3 years); (ii) Juvenile (4–10 years) and adolescent (10 years or more) types.

- *Congenital scoliosis*: Occurs due to an insult during intrauterine period and is associated with other congenital anomalies (cardiac and renal).
- *Acquired scoliosis*: Occurs due to various reasons, such as:
  - Neuromuscular (muscular dystrophy, cerebral palsy)
  - Paralytic, following attack of poliomyelitis
  - Traumatic injury to spine
  - Pulmonary, following lung disease, chest deformity
  - Postural seen in patients with osteoporosis and obesity.

Primary or structural scoliosis is fixed asymmetrical, progressive with C-shaped scoliosis, lacks normal flexibility and do not correct with bending towards the convexity or in supine position.

Secondary or nonstructural scoliosis: Compensatory or additive. Curve resolves correctly with bending towards the convexity or in supine position.

Q. What is the aim of surgery for scoliosis? Why anesthesia for correction of scoliosis is a challenge to the anesthesiologists?

Aim of surgery:

- Correction of the curve of scoliosis
- Fusion of the spine

- Improvement of posture
- Arrest progression of pulmonary dysfunction.

Correction of scoliosis is a challenge to anesthesiologists because of:

- Prolonged anesthesia in prone position
- Major blood loss requires massive blood transfusion
- Electrolyte imbalance
- Associated problems like malignant hyperthermia, restrictive lung disease, pulmonary hypertension.

Also because:

- Patients are associated with frequent comorbidities
- Surgery is extensive in nature
- Constraints on anesthetic techniques because of intraoperative spinal cord monitoring.

Q. How preoperative assessment is done? How the severity of scoliosis is measured?

Pre-anesthetic evaluation includes:

- Patient history
- Physical examination of the patient
- Etiology of scoliosis
- Location of scoliosis
- Degree of scoliosis.

The focus in preoperative medical assessment is on cardiorespiratory function. For most patients with idiopathic scoliosis, a good exercise tolerance is best guide to cardiopulmonary status.

Respiratory changes: Restrictive lung disease with:

- *Reduced vital capacity*: If preoperative vital capacity is less than 30–35% of the predicted value, patient may require postoperative ventilation.
- Reduced total lung capacity
- VA/Q mismatch
- Residual volume normally unchanged.

Severity of respiratory compromise depends on:

- Angle of scoliosis-Cobb's angle
- Number of vertebrae affected
- Cephalad location of the curve
- Loss of normal thoracic kyphosis.

Independent factors likely to require postoperative ventilation are neuromuscular scoliosis and FEV <40% of predicted value. FEV <40% predicted value, VC <60% predicted value, IC 30 mL/kg, TLC <60% predicted and neuromuscular disease, each corelate with prolonged postoperative mechanical ventilation.

Surgery on thorax (anterior, combined or posterior) is associated with an initial decline in FVC, FEV and TLC at 3months. Exclusive posterior approach is required for improvements in pulmonary function test (PFT) at 3 months and better at 2 years.

Changes in cardiovascular system:

- *Direct myocardial involvement*:-Cardiomyopathy complicates many progressive muscular diseases like Duschenne's muscular dystrophy and myotonic dystrophy.
- *Secondary cardiac involvements*:
  - Mediastinal distortion
  - Pulmonary hypertension
  - Corpulmonale: Secondary to hypoxia and pulmonary hypertension.
- Increased incidence of congenital heart disease and mitral regurgitation.

The severity (degree) of scoliosis is measured by Cobb's angle obtained using:

Lippmann–Cobb method: The uppermost and lowermost vertebrae, that are most severely tilted towards the concavity, are determined. Two horizontal lines are drawn, one at the upper border of upper end vertebra and another at the lower border of the lower end vertebra, perpendiculars to these two lines are drawn and the intersecting angles are measured. This method is called Lippmann–Cobb method and the angle as Cobb's angle. It is the most commonly used method to access severity of scoliosis.

Another method is Risser–Ferguson method: in this method, the degree of scoliosis is measured from the angle formed by the intersection of lines drawn from the middle of upper and lower end vertebrae to the center of optical vertebra.

As the curvature of scoliosis increases, rotation progresses and the chest cavity becomes narrowed, resulting in restrictive lung defect. This is rarely significant for the curves of <65 degree. With severe curves, restriction increases.

VA/Q mismatching occurs and pulmonary hypertension and respiratory failure may result. Surgical correction does not reverse the restrictive lung defect but will arrest the progression.

Severity of sign/symptoms proportional to the degree of deformity:

- When Cobb's angle >40 degree—indication for surgery
- When Cobb's angle >65 degree:
  - Restrictive lung disease
  - Dyspnea on exertion.
- When Cobb's angle is >100 degree:
  - Respiratory failure
  - Pulmonary hypertension
  - Right heart failure.

Q. What are the preoperative investigations required in patients put for corrective surgery for scoliosis?

Routine investigations	Additional investigations
• Blood tests - Full blood count: Hb% to rule out polyurea, creatinine, electrolytes coagulation screening - Blood cross matching and grouping	• ECG: RVH, pulmonary HT
• Plain chest X-Ray • Lung compression & size of heart	• Echocardiography - Nonidiopathic scoliosis can have subclinical cardiomyopathy - Low left ventricular ejection fraction
• Pulmonary function tests (to assess the restrictive airway disease.) - Bedside PFTS - Spirometry	• Arterial blood gases.
	• RBS, LFT and RFT

Q. What are the important aspects of anesthesia in corrective surgery for scoliosis? How to optimize respiratory changes in these patients?

Important aspects of anesthetic management for corrective surgery for scoliosis:

- Preanesthetic evaluation
- Technique of anesthesia to be used
- Positioning of the patient during surgery
- Standard monitoring and temperature control. Urine output measurement
- Neurological monitoring
  - Neurological (SSEP, MEP)
  - Wake up test.
- Blood conservation
  - Minimizing blood loss
  - Autologous blood transfusion.
- CVP catheter insertion.
- If severe cardiac impairment
  - Transesophageal echocardiography
  - Pulmonary catheter insertion.
- Postoperative management
  - Pain management—adequate analgesia allows good cough and removal of retained secretion.
  - Fluid management

Preoperative optimization of respiratory changes in patients with scoliosis:
Reversible cause of pulmonary dysfunction, including infection is done with antibiotics, physiotherapy and nebulized bronchodilators. Patients are taught to perform exercises, breath holding techniques and incentive spirometry. Hydration is necessary to loosen pulmonary secretions.

Associated congenital heart disease and mitral value regurgitation are ruled out. Presence of preoperative neurodeficit must be carefully documented to prevent postoperative medicolegal issues. Presence of musculoskeletal abnormalities makes these patients prone to abnormal response to muscle relaxant and should be detected preoperatively.

Q. How anesthesia is conducted for surgical correction of scoliosis?

Anesthesia technique: The primary aim is to provide stable depth of anesthesia, which allows intraoperative neurologic monitoring.

Premedication:
- Bronchodilators to optimize respiratory function.
- Anticholinergics—atropine or glycopyrrolate, especially if fiberoptic-guided intubation is planned.
- Aspiration prophylaxis—H_2 blockers, proton pump inhibitors
- Anxiolytics—midazolam
- Opioids—fentanyl.

Adequate quantity of cross-matched blood should be readily available.

Two peripheral IV lines with 18g cannula are done. Central lines instituted to monitor CVP. Arterial line done.

General anesthesia is induced on trolley and then the patient is positioned on supporting table in prone position.

Induction is done by using:
- IV inhalation agents (preferred)
  - Thiopentone
  - Propofol.
- Inhalational agents — IV or inhalational. Induction technique is guided by the patient's preoperative condition and the ease of intubation
  - Halothane .
  - Sevoflurane.

Intubation is done when:
- Awake with fiberoptic bronchoscopy
- Asleep using anesthetic agents and direct vision laryngocopy.

A proper size armored cuff ET-tube should be used to prevent kinking. After confirming the correct placement of the ET tube, it is adequately secured with dynaplast and a bite-blocker is placed.

As most patients with idiopathic scoliosis present for surgical correction, succinylcholine is still the preferred agent for rapid sequence intubation (RSI). Succinylcholine is contraindicated in patients with muscle dystrophies as it may cause rhabdomyolysis and hyperkalemia leading to cardiac arrest.

Rocuronium is preferred alternative to succinylcholine.

Eyes are covered with eye pad before making patient prone.

Problems of prone position: Though prone position is associated with more uniform distribution of pulmonary blood flow and improved ventilation/perfusion, problems associated with it are:

- Dislodgement of endotrachcal tube
- Pressure on various parts:
  - IV lines—eyes—risk of eye ball injury
  - Arterial line—arms—arterial or venous occlusion of upper arms.
  - ECG leads—breast—soft tissue injury
  - Genital organ injury
  - Pulse oximeter probe—ETT—kinking of the tube
  - Temperature probe.
- Neural injury:
  - Supraorbital nerve injury
  - Brachial plexus injury
  - Ulnar and peroneal nerve injury.
- Compression of IVC leads to elevated pressure in the thoracolumbar venous plexus leading to venous congestion, which increases surgical bleeding. Bolsters are placed below chest and pelvis to keep abdomen free during ventilation to minimize blood loss.

Maintenance:

- Stable depth of anesthesia is maintained with minimum interference to interpret in somatosensory evoked pressure (SSEP) and motor evoked potential (MEP).
- 60% N_2O, <0.5 MAC isoflurane, relaxant (vecuronium or atracurium) and opioid technique is commonly used. Atracurium for neuromuscular blockade makes patients easily arousable for wake up test.
- Methylprednisolone is administered in bolus and in infusion.
- For hypotensive anesthesia to minimize blood loss, halothane and esmolol or nitroglycerine infusion may be used. Induced hypotension makes the patient vulnerable to spinal ischemia and should not be done.
- A controlled mechanical ventilation is provided during maintenance of anesthesia.
- During closure, thoracotomy drain with underwater seal is placed.
- An intrapleural or paravertebral catheter may also be placed at this time to be used for postoperative analgesia.
- Proper recovery is done and the need for postoperative ventilation is carefully decided.

As the surgery is extensive, the occurrence of major blood loss is 20–50 mL/kg. Blood loss depends on the number of spinal vertebra fused, being 250 mL blood loss per each vertebra. Increased intra-abdominal pressure increases blood loss, which is associated with delayed wound healing and infection, increased operative time and massive blood transfusion. Therefore blood conservation is done by:

- *Minimizing blood loss with*:
  - Careful positioning to avoid venous congestion
  - Prevention of hypothermia as it increases bleeding
  - Correction of coagulopathy
  - Use of antifibrinolytics like aprotinin inhibits fibrinolysis
  - Tranexamic—Anti-fibrinolytic agent—Dose–15–25 mg/kg infusion.
  - Aminocaproic acid-
  - Use of good surgical technique.
- *Autologous blood transfusion*: Methods of autologous blood transfusion are:
  - Preoperative autologous transfusion:
  - Normally not possible as the patients commonly belong to pediatric age group
  - Requires regular visits
  - Requires erythropoietin injections for 4 weeks
  - Hemoglobin should be >11 g%.
  - Intraoperative hemodilution
  - Hemoglobin should be >10 g% if no comorbidities
  - Done in operating table after induction of anesthesia
  - Target hemoglobin 7 g%
  - Volume of blood to be removed is:

  Estimated blood volume × (initial Hct – Target Hct – Mean Hct)
  - Blood volume is replaced with colloids.
  - *Intraoperative cell salvage*
  - Blood lost by the patient during surgery is collected and then reinfused
  - Survival and oxygen transport properties of RBC are equivalent to those of stored allogeneic RBC
  - Recovered blood may contain tissue debris, small blood clots and bone fragments—microaggregate filters (40 μm) are to be used
  - Hemolysis may occur during suctioning of lost blood
  - Chance of bacterial infection
  - Special machines are required to collect shed blood wash—it concentrates the RBCs.

Q. What are the complications of corrective surgery for scoliosis?

- ***Intraoperative complications***:
  - Hypothermia or hyperthermia: Malignant hyperthermia is common in scoliosis patients having associated muscle dystrophy.
  - Bleeding: Extensive surgery causes major blood transfusion associated with side effects.
  - Tachycardia and cardiac arrhythmias
  - Hypotension and hypertension
  - Signs of venous air or fat embolism
  - Compression neuropathies from positioning.

- ***Postoperative complications*:**
  - Motor deficit or paraplegia caused by:
    - Direct contusion of the spinal cord by implants or instruments
    - Reduction of spinal cord blood flow by stretching or compression of vessels
    - Destruction injury of the spinal cord
    - Epidural hematoma can be reduced with proper neurological monitoring
  - Various other ophthalmological complications may occur—Transient or permanent ophthalmoplegia, orbital compartment syndrome, dislocation of intraocular lens.
  - Syndrome of inappropriate ADH secretion causing hyponatremia and serum hyposmolality.
  - Bleeding
  - Pneumothorax
  - Hyperpyrexia, hypothermia.

Q. What intraoperative monitoring is essential during anesthesia for corrective surgery for scoliosis?

Monitoring during anesthesia for corrective surgery for scoliosis includes:

- Standard monitoring including ECG, pulse oximetry, $EtCO_2$ monitoring (to detect circuit disconnection and leak during prone position), noninvasive blood pressure amplifier (NIBP) and invasive blood pressure (IBP) (for ABG analysis), urinary catheter for hourly monitoring may be misleading.
  - Indicator of right ventricular end-diastolic volume (RVEDV) and left ventricular end-diastolic volume (LVEDV)
  - Pulmonary artery pressure monitoring
  - Temperature monitoring (to detect hypothermia and hyperthermia)
  - Neuromuscular block monitoring.
- Spinal cord integrity is monitored using:
  - Ankle clonus test
  - Wakeup test
  - Somatosensory evoked potential (SSEP)
  - Motor evoked potential (MEP)

Ankle clonus test: It is done during emergency or during wakeup test. It is easy to elicit and has high sensitivity and specificity. Injury of spinal cord is indicated by complete absence of clonus. Absence of clonus could be due to inadequate depth or too deep anesthesia. Presence of clonus does not exclude spinal cord damage as other parts of the cord may be damaged leaving the ankle clonus stretch intact.

Wakeup test: During the test, anesthesia is lightened intraoperatively and the patient is asked to do a specified motor response of lower limbs. Once the patient is able to perform the actions, sleep is induced once again. Volatile anesthetic and midazolam based anesthesia technique is commonly used. Propofol and remifentanil infusion may be used. This test evaluates grossly

the motor function, but not the sensory function. Patient's cooperation is needed. Therefore, preoperative counseling is needed to assure the patient that he/she will be awake but there will be no pain or discomfort and will not have recall of the test.

The test enables early detection of spinal cord injury after correction of deformity of the spine. Gross patient movement may result in accidental extubation or loss of IV access. While inspiratory effort may promote venous air embolism. It is not continuous intraoperative monitoring and is valid only up to the time the test is performed. Damage occurring later is missed.

Somatosensory evoked potential (SSEP): It involves stimulation of a peripheral nerve, usually the posterior tibial nerve, and detecting a spinal response with epidural electrode or critical response with scalp electrodes.

Only the dorsal sensory pathways are monitored, not the most vulnerable anterior motor pathways. Baseline data are obtained after stem incision. During surgery, responses are recorded repeatedly so that it provides continuous intraoperative monitoring of posterior column integrity.

Functional integrity of dorsal sensory pathways is determined by comparing the amplitude and latency change of the responses during surgery to baseline values.

Reduction in amplitude response by 50% and an increase in latency by 10% are considered significant.

Factor affecting SSEP are:

- Anemia, hypoxia, hypercarbia, hypotension, hypothermia and anesthetic drugs.
- Inhalational and N_2O-amplitude and latency
  - N_2O 60% with 0.5 MAC isoflurane is compatible with effective SSEP monitoring.
  - False positive has been attributed to an increased concentration of inhalational agents.
  - Intravenous agents produce same effects as inhalational agents but to a lesser degree.

Motor evoked potential (MEP): MEP monitors the more vulnerable anterior cord. It involves stimulations of the motor cortex by electrical or magnetic stimulation transcranially and detecting the resulting signal at spinal level with epidural electrode or from muscle as a compound muscle action potential (CMAP). Stimulation cranial to the surgery causes stimulation of the motor tracts in the spinal cord and peripheral nerve and muscle caudal to the surgery.

Any perturbation of the motor pathway leads to amplitude and latency.

Recorded responses: Myogenic/neurogenic.

Neurogenic responses can be recorded under complete NM block and are more reliable in terms of amplitude, latency and morphology.

Stimulation of the motor cortex results in direct stimulation of the pyramidal cells and conduction down spinal pathways.

Summation of these wakes result in firing of the anterior horn cells and peripheral transmission via motor nerves to muscle. Muscle depolarization results in CMAP.

Effects of anesthetic agents on MEP:

- Propofol is powerful suppressant of MEP
- 2 mg/kg abolishes all cortical responses
- Volatile anesthetics—similar effect at 0.8 MAC of isoflurane
- Midazolam/etomidate—cause lesser depression; opioids have little or no effect.
- Ketamine, fentanyl and remifentanil based techniques are reported to be the best.

Combined SSEP and neurogenic MEP monitoring during idiopathic scoliosis surgery represent a standard of care to monitor spinal cord damage. This combination obviates the need of wakeup test, once reliable data are obtained and maintained. SSEP and MEP are complimentary.

Intraoperative wakeup test is preserved, where both SSEP and MEP are not possible or responses are significantly perturbed.

Effects of anesthetic agents on neurophysiologic monitoring:

- Volatile anesthetic agents and propofol depress SSEPs and MEPs in a dose-dependent manner
- N_2O profoundly depresses SSEPs and MEPs
- Opioids have little effect on either modality.
- Muscle relaxants reduce background noise by enhancing SSEP recordings. Profound degrees of muscle relaxation abolish CAMPs but not epidural MEPs.
- Ketamine and etomidate may enhance recordings

Q. What are the risk factors for intraoperative spinal cord damage?

- Length and type of surgical procedure
- Pressure, retraction and manipulation of neural tissues
- Intraoperative drop in spinal cord perfusion pressure (SCPP = MAP – CSFP)
- Hematoma in spinal canal — careful hemostasis and proper adjustment of anticoagulant medication perioperatively.

Q. What are the considerations during postoperative care after corrective surgery for scoliosis?

Special considerations are given on:

- Need for postoperative mechanical ventilation
- Significant pain till 4 days — adequate analgesia is to be provided. Good pain relief implies good and early recovery
- Prevention of complications, such as:
  - ARDS
  - Pneumonitis—physiotherapy, incentive spirometry
  - Atelectasis

- Pulmonary embolism—complications.
- Cerebrovascular accidents

Need for postoperative mechanical ventilation: Patients who undergo corrective surgery for scoliosis are at risk for postoperative complications as long as one week postoperatively, 1–2 months after surgery.

Postoperative ventilation may be needed in:

- Lengthy procedure
- Occurrence of hypothermia
- Blood loss >30 mL/kg
- Metabolic derangement
- Presenting systemic disease
- Right ventricular failure
- FVC <35%.

Independent factors likely to require postoperative ventilation are:

- Neuromuscular scoliosis
- FEV1 <40% predicted—each correlated with prolonged postoperative mechanical ventilation
- VC <60% predicated—ventilations
- IC <30 mL/kg
- TLC <60% predicated.

Need for postoperative analgesia: Significant postoperative pain occurs till 4 days following corrective surgery for scoliosis, so proper pain management is needed. Adequate pain relief provides good and early recovery. Methods used for postoperative analgesia:

- Opioids:
  - arenteral opioids: IV/IM/infusions
  - CA with or without background infusions
- Inherent opioid disadvantages:
  - Respiratory depression
  - Sedation, nausea, vomiting.
  - Gastrointestinal ileus.
- Non-steroidal anti-inflammatory drugs:
  - Nonselective cyclo-oxygenase inhibitors
  - Selective cyclo-oxygenase 2 inhibitors
- Epidural analgesia—intraoperative placement

There is mounting evidence that epidural morphine is safe and postoperative analgesia following spine surgery.

A continuous epidural infusion of local anesthetics with morphine delivered through a single epidural catheter provides improved analgesia, compared to IV morphine.

However, many patients still experience significant pain in the upper and lower portion of the surgical site. In these cases, two placements following completion of surgery and prior to wound closure should be done. One inserted at T_6 level with tip directed cephalad to T_{1-4} and another inserted at T_{12} with tip pointed at the L_{1-4} levels, provides adequate analgesia.

After wound closure, fentanyl is administered through the catheters followed by continuous infusion of 1% ropivacaine and fentanyl. Use of shorting opioid (remifentanil) avoids respiratory depression.

- Intrathecal analgesia: Low-dose (2–5 mg) intrathecal morphine administered in operating room supplemented by PCA morphine provides better analgesia than PCA morphine alone.
- Intrapleural analgesia is provided by placing catheter in intrapleural space before chest closure.
- Nerve blocks: Intercostal nerve blocks provide analgesia after thoracotomy.

Chapter

9

A Middle-aged Female with Huge Thyroid Swelling Posted for Thyroidectomy

Q. Classify thyroid swellings. What is meant by goiter? What is the important causative factor of endemic goiter?

Classification of thyroid swellings:

- Simple goiter (euthyroid)
  - Diffuse hyperplastic goiter
  - Multinodular goiter.
- Toxic goiter
  - Diffuse goiter
  - Multinodular goiter
  - Toxic adenoma.
- Neoplastic goiter
  - Benign
  - Malignant.
- Inflammatory goiter
  - Chronic lymphocytic thyroiditis.
  - Hashimoto's disease.

Simple goiter may develop as a result of stimulation of the thyroid gland by TSH, either as a result of inappropriate secretion from the microadenoma in the anterior pituitary (rare) or in response to a low level of circulating thyroid hormones. The most important factor causing endemic goiter is dietary deficiency of iodine.

The term goiter is used to describe generalized enlargement of thyroid gland. Normal thyroid gland is impalpable.

Q. What are the hormones secreted by thyroid gland? Which thyroid hormone is physiologically active? What are the steps of thyroid hormone synthesis?

The follicular epithelial cells synthesize thyroid hormones by incorporating iodine into amino acid tyrosine on the surface of thyroglobulin. Iodine is the

key substance for thyroid hormone synthesis; a dietary intake in excess of 100 mg/day is required to maintain thyroid function in adults.

The thyroid secretes predominantly thyroxine (T_4) and only a small amount of triiodothyronine (T_3). Approximately 85% of T_3 in blood is produced from T_4, which can be regarded as prohormone. T_4 has longer half-life in blood (1 week.) than T_3 (18 hours.); and binds and activates thyroid hormone receptors less effectively than T_3, which is more effective.

T_3 and T_4 circulated in plasma, almost entirely bound to transport proteins, mainly thyroxin binding globulin (TBG). It is the unbound or free hormones, which diffuse into tissues and exert metabolic action.

- Tyrosine
- Monoiodotyrosine
- Thyroxine (T_4)
- Triiodothyronine (T_3)
- Diiodotyrosine.

Iodine is a new material essential for thyroid hormone synthesis. Ingested iodine is converted to iodide and absorbed. The thyroid concentrates iodide by actively transferring it from circulation to the colloid. The transport mechanism is frequently called 'iodine trapping' or 'iodine pump'. In the thyroid gland, iodide is oxidized to iodine, which is instantly bound to the 3rd position of tyrosine molecules attached to thyroglobulin to form monoiodotyrosine. The enzyme responsible for oxidation and binding of iodine is thyroid peroxidase.

Monoiodotyrosine is next iodinated in 5-position to form diiodotyrosine. Two diiodotyrosine molecules then undergo oxidation condensation to form thyroxin (T_4).

Q. How examination of thyroid gland is done clinically?

- Inspection—from front to side
- Palpitation—from behind
- Thyroid movement on swallowing, retrosternal extension
- Cervical Lymph nodes
- Tracheal deviation
- Auscultation for bruit
- Patient is asked to hold breath
- If bruit is present, checking for radiating murmur is done
- Percussion—for retrosternal goiter.

Abnormal findings:

- Diffuse soft goiter with bruit (indicating increased blood supply)
  - Graves' disease
- Diffuse firm goiter
  - Hashimoto's thyroiditis
- Diffuse tender goiter
  - Subacute thyroiditis
  - Multinodular goiter.

Q. What are the presenting features of retrosternal goiter? What are the complications of retrosternal goiter? How the retrosternal extension of goiter mass is assented?

Very few retrosternal goiters arise from ectopic thyroid. Most of them arise from the lower lobe of the thyroid gland. If the neck is short and the muscles, are strong, the negative pressure intends to draw these nodules into superior mediastinum.

Often the retrosternal extension of the goiter is discovered on routine chest radiography. There may, however, be severe symptoms like:

- Dyspnea, particularly at night
- Cough and stridor (harsh sound on inspiration)
- Dysphagia
- Dysphonia from recurrent laryngeal nerve paralysis is rare.
- Engorgement of the neck veins and superficial veins on the chest wall. In severe cases, there may be obstruction of the superior vena cava

Complication*:* Superior mediastinal obstruction may occur with large goiter leading to venous obstruction in the thoracic inlet. Signs of compression can be induced in case of large retrosternal goiter. When the patient's arms are raised above the head (Pemberton's sign),there occurs flushing of face and dilatation of cervical veins with giddiness and syncope.

Assessment of retrosternal extension of goiter mass:

- To observe whether it is possible to get the palpating fingers below the midline enlarged thyroid gland, when the patient swallows a little water (attachment of thyroid to pretracheal fascia dictates that it moves superiorly on swallowing). This draws the partial retrosternal goiter above the examining finger but the inferior border is not palpable if the retrosternal extension of goiter is complete into the superior mediastinum.
- To observe the features of Pemberton's sign.
- To observe the findings in the radiograph of neck thoracic inlet — it shows a soft tissue shadow in the superior mediastinum, which often causes deviation and compression of trachea.

Q. What is superior vena cava syndrome? What are the presenting features of it?

Superior vena cava syndrome is a group of symptoms that develop in patients with superior mediastinal tumor, leading to obstruction of the venous drainage of upper thorax. Initially it presents as bilateral engorgement of jugular veins. Increased venous pressure leads to: (i) dilatation of collateral veins in the thorax and neck, (ii) an edema and cyanosis of face, neck, arms of upper chest, (iii) conjunctival edema, (iv) evidence of increased ICP including headache, nausea, seizures and attend mental state, (v) hoarseness may reflect edema of the vocal cords.

Q. How tracheal displacement and compress are assessed?

Radiograph of neck and thoracic inlet shows the displacement and compression trachea by superior mediastinal tumor (retrosternal goiter).

- AP view — denotes displacement of trachea
- Lateral view denotes compression of trachea. CT and MRI scans give excellent anatomical detail of thyroid swellings but have no role in first-line of investigation.

Q. What investigations are to be done before thyroidectomy?

- *Evaluation of thyroid function*: Estimation of thyroidism is associated with a discrete swelling manifestation of 'toxic adenoma' or a manifestation of toxic modular goiter.
- *Auto antibody titer*: Thyroid antibody levels do not indicate thyroid function. It is important in determining which swelling may be a manifestation of chronic lymphatic thyroiditis (autoimmune thyroiditis).
- *Isotope scan*: It is main day of investigations of functional activity related to the surrounding gland according to isotope uptake. On scanning, swellings are categorized as hot (overacting), warm (active) or cold (underactive). A hot nodule takes up isotope, while the surrounding tissue does not. The nodule produces such a high level of thyroid hormones that TSH secretion is suppressed and surrounding thyroid tissue is inactive. A warm nodule takes up isotope and so does the normal thyroid tissue around it. A cold nodule takes up no isotope.
- *Ultrasonography*: It can demonstrate subclinical nodularity and cyst formation but the former is clinically irrelevant and the later appears at aspiration, which should be a routine in all discrete swellings.
- *Fine needle aspiration cytology (FNAC)*: It is the investigation of choice in discrete thyroid swelling. FNAC has excellent patient compliance, is simplex quick to perform in the OPD and can be repeated. Thyroid conditions those may be diagnosed by FNAC include colloid nodule, thyroiditis, papillary carcinoma, medullary carcinoma and anaplastic carcinoma lymphoma. [FNAC cannot distinguish between a benign follicular adenoma and follicular carcinoma as this distinction is not dependent on cytology but on histological criteria, which include capsular and vascular invasion].
- *Radiology*: Neck and thoracic in bet radiographs are only necessary when there is clinical evidence of tracheal deviation and compression or retrosternal extension.
- *Other scans*: CT and MRI scans give excellent anatomical detail of thyroid swelling, but have no role in first-line of investigation.
- *Indirect laryngoscopy*: To determine the mobility of the vocal cords, preoperatively for medicolegal rather than clinical reasons.
- *Large-bone needle (Trucut) biopsy*: It has a high diagnostic accuracy but has poor patient compliance and may be associated with complications like pain, bleeding, tracheal and recurrent laryngeal nerve damage.

Q. How thyroid function is evaluated? What is hyperthyroidism? What is hypothyroidism?

Mild-to-moderate thyroid dysfunction probably has minimum impact perioperatively. Significant hyperthyroidism appears to increase perioperative risks. Patients with a history of chronic thyroid disease need thyroid function tests before surgery. Tests include:

- *Serum thyroxine (T_4):* Serum thyroxine (T_4) assay is the standard screening test for evaluating thyroid gland function. The total T_4 is elevated in most patients with hyperthyroidism and it is low in those who have hypothyroid. T_4 is a prohormone product of thyroid gland.
- *Serum triiodothyronine* (T_3): Serum triiodothyronine (T_3) levels are often determinate to detect disease in patients with clinical evidence of hyperthyroidism in absence of elevations of T_4. T_3 may be the only thyroid hormone produced in excess. T_3 concentration may be depressed by factors that impair the peripheral conversion of T_4 to T_3. In 50% of hypothyroid patients, the serum T_3 concentration is low and in remaining 50%, it is normal. The T_3 is a product of both thyroid and extrathyroid enzyme deiodination of T_4 and the effect of thyroid hormone is mediated by the T_3, which is more potent. The T_4 functions only as prohormone.
- *Thyroid stimulating hormone (TSH)*: TSH is usually regarded as the most useful investigation of thyroid function. A rising TSH level is a sensitive indicator of failing thyroid function. Normal range of TSH is 0.3–4.5 mU/L. TSH value of 5–15 mU/L is characteristic of hypothyroidism. The goal of thyroid replacement therapy is to normalize TSH levels. Current assay is sensitive enough to diagnose hyperthyroidism by depressed level of TSH. Production of the major thyroid hormones, thyroxine (T_4) and triiodothyronine (T_3) is stimulated by thyroid stimulating hormone (TSH) released from anterior pituitary which, in turn, is regulated by secretion of thyrotropin-releasing hormone (TRH) in the hypothalamus. There is a negative feedback of thyroid hormones on hypothalamus and anterior pituitary such that in thyrotoxicosis when the plasma concentration of T_3 and T_4 are raised, TSH secretion is suppressed. Conversely in hypothyroidism, the low T_3 and T_4 are associated with high circulating TSH levels.
- *Thyroid antibodies*: Thyroid antibodies include antithyroglobulin antimicrosomal or antithyroid peroxides, thyroid stimulating immunoglobulin (TSI). Anti-Tg and anti-TPO antibody levels do not determine thyroid function, instead they indicate an autoimmune thyroiditis.
- *Serum thyroglobulin*: It is not normally released; it increases dramatically in thyroiditis or Grave's disease and toxic multinodular goiter. The most important use for serum thyroglobulin levels is in monitoring patients with differentiated thyroid cancer for recurrence. Because only thyroid tissues produce thyroglobulin, the levels of this protein should be low after total thyroidectomy.

- *Radioisotope imaging*: Both iodine - 123 (^{123}I) and iodine – 131 (^{131}I) are used to image the thyroid gland. The former emits low-dose radiation and is used to image lingual thyroids or goiters. In contrast ^{131}I emits higher dose radiation exposure and is used to screen and treat patients with differentiated thyroid cancers. The areas that trap less radioactivity than the surrounding gland are termed 'cold', whereas the areas that demonstrate in opposed activity are termed 'hot'. The risk of malignancy is higher in cold lesion than in 'hot' or 'warm' lesions.
- *Ultrasound*: It is helpful in evaluating thyroid nodules, distinguishing solid from cystic ones and providing information about size multicentricity. It can guide the needle aspiration biopsy.
- *Needle aspiration biopsy*: Investigation of choice in discrete thyroid swellings, which include colloid nodule, thyroiditis, lymphoma and different carcinomas.

Hyperthyroidism: It is due to over-activity of thyroid gland with symphony produced due to actions of thyroid hormones. It has various causes, however, the most common cause is Grave's disease, which is an autoimmune disease, more common in women, where the antibodies to the TSH receptor stimulate the receptor. This produces marked T_4 and T_3 secretion and enlargement of thyroid gland (Goiter). However, due to the negative feedback effects of T_4 and T_3, plasma TSH is low, not high. Another hallmark of Grave's disease is the occurrence of swelling of tissues in the orbits producing protection of the eyeball (exophthalmos). In Hashimoto thyroiditis, autoimmune antibodies ultimately destroy the thyroid gland but during the early stage of inflammation of the gland causes excess thyroid hormone secretion and thyrotoxicosis similar to that seen in Grave's disease. Other causes of hyperthyroidism include solitary toxic adenomas, toxic multinodular goiter and TSH secreting pituitary tumor.

The presenting features are:

- Weight loss, sweating, anxiety, palpitations, diarrhea, heat intolerance, weakness, fatigue irritability and menstrual irregularities.
- Warm and moist skin, stare-look, tachycardia, arrhythmias, fine tremor of the outstretched fingers and raised basal metabolic rate (BMR).
- In Grave's disease-goiter (often with bruit), ophthalmopathy.
- Suppressed TSH in primary hyperthyroidism with increased T_4 and T_3.

Hypothyroidism: It may be the end result of a number of diseases of thyroid gland or it may be secondary to pituitary or hypothalamus failure. In the latter two conditions, the thyroid remains able to respond to TSH. Thyroid function may be reduced by a number of conditions. When the dietary iodine intake falls below 50 mg/day, thyroid hormones synthesis is inadequate and secretion declines. As a result of increased TSH secretion, the thyroid hypertrophies produce iodine deficiency goiter. Drugs may also inhibit thyroid function, either by interfering with the iodine-trapping mechanism or by blocking the organic binding of iodine. In both the cases, TSH secretion is stimulated by the decline in thyroid hormones and a goiter is produced.

Iodine itself in large doses inhibits thyroid hormone. The presenting features of hypothyroidism are: Organic building of iodine weakness, lethargy, fatigue, cold intolerance, constipation, weight gain, depression, menorrhagia and impaired ventilator response to hypoxia and hypercapnia. T_4 level is usually low and TSH level elevated in primary hypothyroidism.

Primary:
- T_4
- TSH.

Secondary:
- T_4
- TSH.

Amiodarone, because of its high iodine content causes clinically significant hypothyroidism in patients who receive it. T_4 level is normal or low and TSH level is elevated. Cardiac patients with amiodarone-induced symptomatic hypothyroidism are treated with just enough thyroxin to relieve symptoms. Patients receiving amiodarone need special consideration in the perioperative period due to arrhythmias that lead to such therapy and also for the concern for thyroid dysfunction.

Q. What is thyroid storm? What are the manifestations and treatment of it?

Thyrotoxic crisis or thyroid storm is an extreme form of thyrotoxicosis most commonly precipitated by stressful illness like infection in a patient with unrecognized or inadequately treated thyrotoxicosis. It may also develop shortly after subtotal thyroidectomy in an ill-prepared patient or within few days of ^{131}I therapy. It is a life-threatening medical emergency and despite early recognition and treatment, the mortality rate is high.

Most prominent signs are hyperpyrexia (very similar to malignant hyperthermia), marked delirium, agitation, confusion, severe tachycardia or atrial fibrillation and in older patients, cardiac failure. There is also vomiting, diarrhea and dehydration.

Treatment: NSAIDs or aspirin are avoided as they displace T_4 from thyroxine-binding globulin (TBG), raising FT_4 serum level.

Treatment modalities include:
- Oxygen supplementation done
- Active cooling to reduce hyperthermia and paracetamol
- Patient should be rehydrated carefully with saline and glucose
- A broad spectrum antibiotic is given
- Hydrocortisone is usually given in a dose of 100–200 mg intravenously 6 hourly to treat adrenal insufficiency and decrease T_4 release and conversion to T_3 at very high levels.
- Propranolol is given IV in 1 mg increments up to 10 mg. with CVS monitoring to decrease pulse rate to <90/min. Alternatively esmolol is given as 250–500 mg/kg loading IV dose followed by 50–100 mg/kg/min infusion.

- Propylthiouracil is given as 1 g loading dose via nasogastric tube followed by 200–800 mg hourly. This inhibits thyroid hormone release and also decreases peripheral conversion of T_4 to T_3.
- After blocked by propylthiouracil, sodium iodide is given IV in a dose of 500 mg hourly or potassium iodide in a dose of 5 drops 6 hourly via nasogastric tube or Lugol's iodine in a dose of 5–10 drops 6 hourly via nasogastric tube.
- Carbimazole in a daily dose of 40–60 mg orally inhibits the synthesis of new thyroid hormone. When the patient is unconscious or incooperative, carbimazole can be administered rectally with good effect using the oral dose as no preparation is available for parenteral use.
- Definitive treatment with ^{131}I or surgery is delayed until the patient is euthyroid.

Q. What is myxedema coma? What are the manifestations and treatment of it?

Myxedema coma is a rare form of severe, life-threatening hypothyroidism. It is often induced by an underlying infection; cardiac, respiratory or cause illness, cold exposure and trauma. It is most often manisfested in elderly women who have had a stroke or have stopped taking their thyroxin medication. Mortality rate is high. The manifestations of hypothyroidism are present in a more complex form—affected patients have impaired cognition, ranging from confusion to somnolence to coma convulsions and abnormal CNS signs may occur. Patients have severe hypothermia, hypoventilation, hypernatremia, hypoglycemia and bradycardia, rhabdomyolysis and acute kidney injury may occur.

Treatment: Myxedema coma is a medical emergency and treatment must begin before biochemical confirmation of the diagnosis. Treatment modalities include:

- Rehydration with intravenous fluids, containing saline and glucose, is done carefully.
- Patient is warmed with only blankets, as sudden warming may lead to extreme peripheral vasodilatation, precipitating cardiovascular collapse.
- Infection must be detected and treated aggressively.
- Patients with hypercapnea require intubation and assisted mechanical ventilation.
- Patient with concomitant adrenal insufficiency are treated with hydrocortisone 100 mg. IV followed by 25–50 mg. every 8 hourly.
- Stabilization of CVS and pulmonary systems is done as necessary.
- Myxedema coma requires a large initial dose of levothyroxine intravenously. Sodium 400 mg is given IV as a loading dose, followed by 50–100 mg IV daily. Lower dose is given in patients with suspected coronary insufficiency.
- In patient with myxedema coma, liothyronine (T_3) can be given IV in a dose of 5–10 kg every 8 hours for the first 48 hours.

Patients are treated in ICU. Myxedematous patients are usually sensitive to opioids and may die from average doses. Patients with severe myxedema may have hyponatremia that is severe and refractory to treatment.

Q. What are the types of thyrotoxicosis? What are the differences between primary and secondary thyrotoxicosis?

The term thyrotoxicosis refers to the clinical manifestations associated with serum levels of T_4 or T_3 that are excessive (5–15 times normal) for the individual (hyperthyroidism). TSH levels are suppressed in primary hyperthyroidism.

Varieties of thyrotoxicosis are:

- Associated with thyroid hyperfunction:
  - Grave's disease
  - Hashimoto's disease
  - Hyperfunctioning adenoma (toxic nodule)
  - Toxic multinodular goiter.
- Not associated with thyroid hyperfunction:
  - Disorders of hormone storage—thyroiditis
  - Extrathyroid source of hormone
- Ectopic thyroid.

In hyperthyroidism, hyperfunction of the thyroid gravid is reflected in an increase in radioactive intake iodine test (RAIU), whereas in nonhyperthyroid toxic states, thyroid function (as reflected in RAIU) is subnormal.

Treatment of thyrotoxicosis by means intended to decrease hormone synthesis (Antithyroid agents, radioiodide, and surgery) is appropriate in hyperthyroidism but inappropriate/ineffective in other forms of thyrotoxicosis.

In primary thyrotoxicosis, hyperthyroidism is more severe than in secondary thyrotoxicosis, but cardiac failure is rare. The goiter is diffused and vascular with a thrill and a bruit.

In secondary thyrotoxicosis, the goiter is nodular. The onset is insidious and may present with cardiac failure or atrial fibrillation. It is characteristic that hyperthyroidism is not severe. Eye signs other than lid lag and lid spasm are rare.

Manifestations of thyrotoxicosis are not due to hyperthyroidism per se, e.g. orbital proptosis, ophthalmoplegia and pretibial myxedema may occur in primary thyrotoxicosis but not in secondary thyrotoxicosis.

Ophthalmopathy of primary thyrotoxicosis may be divided into two categories—the spastic and the mechanical. The former includes stare-look, lid lag, lid retraction and accounts for the "frightened facies".The mechanical component includes proptosis of varying degrees with ophthalmoplegia and congestive oculopathy, characterized by chemosis,.conjunctivitis, periorbital swelling, corneal ulceration, optic neuritis and optic atrophy.

Hyperdynamic circulation in primary thyrotoxicosis suggests excessive activity of the sympathetic nervous system.

Cardiac rhythm: A fast heart rate which persists during sleep is characteristic of primary thyrotoxicosis. Cardiac arrhythmias are superimposed on the sinus tachycardia with progress of the disease and are common in elderly patients with thyrotoxicosis because of the prevalence of co-incidental heart diseases.

Stages of development of thyrotoxic arrhythmias are:
- Multiple extra systoles
- Paroxysmal atrial tachycardia
- Paroxysmal atrial fibrillation
- Persistent atrial fibrillation not responding to digoxin.

Q. What is Grave's disease? What are the characteristic manifestations of Grave's disease?

Grave's disease is the most common cause of thyrotoxicosis. It is an autoimmune disorder affecting thyroid gland, characterized by increased synthesis and release of thyroid hormone; the gland is rapidly enlarged. Grave's disease is much more common in women between the age of 20 and 40 years. It may be accompanied by infiltrative ophthalmopathy (exophthalmos) and less commonly by infiltrative dermopathy (preitibial myxedema). Grave's disease may also be associated with other systemic autoimmune disorders such as pernicious anemia, myasthenia gravis and diabetes mellitus. The pathogenesis of hyperthyroidism of Grave's disease involves the formation of antibodies that bind to TSH receptors in thyroid cell membranes and stimulate the gland to hyperfunction.

Clinical manifestations:
- Nervousness, restlessness, weakness, fatigue, weight loss, heat intolerance, increased sweating, diarrhea, palpitation or angina pectoris.
- Lid lag with downward gaze (Von Graefe's sign), upper eye lid retraction (Dalrymple sign) and a staring appearance (Kocher's sign), fine resting finger tremor, moist and warm skin, hyperreflexia.

Cardiac manifestations: Sinus tachycardia persisting during sleep, forceful heart beat and premature atrial contraction; atrial fibrillation and atrial tachycardia occur in elderly males and patients with ischemic heart disease or valvular heart disease. Thyrotoxicosis itself may cause cardiomyopathy and onset of atrial fibrillation may precipitate congestive heart failure.

Ophthalmopathy: Characteristic of Grave's disease. It consists of conjunctival edema (chemosis), conjunctivitis and mild exophthalmos (proptosis). More severe lymphocytic infiltration of the eye muscles occur, pushing the eye forward, producing clinical exophthalmos and sometimes diplopia due to extraocular muscle entrapment. There may be weakness of upper gaze (Stellwag's sign). The optic nerve may be compressed causing progressive visual disturbances. Diplopia can be caused by ocular myasthenia gravis, which is more common in Grave's disease.

Grave's dermopathy: Occurs usually in pretibial region. Glycosaminoglycan accumulation and lymphoid infiltration occur in affected skin, which becomes erythematous with thickened and rough in texture.

Thyroid acropachy: It is an extreme and unusual manifestation of Grave's Disease. It presents with digital clubbing, swelling of fingers and toes and periosteal reaction of extremity bones. It is an indication of severity of autoimmunity. Patients with thyroid acropachy are at greater risk of having Grave's dermopathy and severe ophthalmopathy.

Q. How hyperthyroid patients are treated? Which antithyroid drugs are in common use?

Management of hyperthyroidism: Symptoms of thyrotoxicosis respond to beta-blockers but definitive treatment requires control of thyroid hormone secretion. The different options are antithyroid drugs (carbimazole, propylthiouracil), subtotal thyroidectomy and radioiodine.

In all patients with thyrotoxicosis, a nonselective beta-blocker such as propranolol (160 mg daily) or nadolol (40–80 mg daily) will alleviate but not abolish symptoms within 24–48 hours. Beta-blockers cannot be recommended for long-term but they are extremely useful in short-term treatment.

Antithyroid drugs: The most commonly used drug is carbimazole. Propylthiouracil is equally effective. These drugs reduce the synthesis of new thyroid hormones by inhibiting iodination of tyrosine. Carbimazole also has an immunosuppressive action, leading to a reduction in serum TRAb concentrations, but it is not sufficient to influence the natural history of thyrotoxicosis significantly. Initial dose of carbimazole is 40–60 mg daily.

Propylthiouracil is the drug of choice in breastfeeding as it is not concentrated in milk. It is also favored during pregnancy as it causes fewer problems in newborn. It is given orally in initial doses of 300–600 mg daily in four divided doses. Drug dose and frequency are reduced as symptoms of hyperthyroidism resolve and T_4 level approaches normal.

Antithyroid drugs are generally used for patients with mild thyrotoxicosis, small goiters or fear of isotopes. These drugs are also useful in preparing hyperthyroid patients for surgery and elderly patients for radioactive iodine (RAI) treatment. These drugs do not permanently damage thyroid gland and are associated with a lower chance of post-treatment hypothyroidism; side effects—agranulocytosis, pruritus, allergic dermatitis, nausea and dyspepsia.

Antithyroid drugs are introduced at high doses—carbimazole 40–60 mg daily and propylthiouracil 400–600 mg daily. There is subjective improvement within 10–14 days and the patient is clinically or biologically euthyroid at 3–4 weeks, when the dose can be reduced. The maintenance is determined by measurement of T_4 and TSH, keeping both in their respective reference range.

Q. What is the perioperative management of subtotal thyroidectomy in hyperthyroid or hypothyroid patients?

Patients must be rendered euthyroid with antithyroid drugs before operation. Potassium iodide 60 mg 8 hourly orally is often added for 2 weeks before surgery to inhibit thyroid hormone release and reduce the size and vascularity of the gland making surgical procedure easier.

In hyperthyroid patients, beta-blocker is continued perioperatively to reduce the possibility of thyroid storm. Propranolol is generally used for symptomatic relief until hyperthyroidism is resolved. It effectively relieves tachycardia, tremor, diaphoresis and anxiety that occur in hyperthyroidism. Treatment is usually begun with propranolol 60 mg orally twice daily with dose increasing every two days. It is given once daily as hyperthyroidism improves.

In hypothyroid patients:

- As low metabolic rate predisposes to hypothermia—active warming is done
- As susceptible to profound hypotension—all drugs are given slowly.
- As drug metabolism is slow, drug doses are reduced, especially muscle relaxants and opioids.

Virtually all anesthetic drugs and techniques have been used without any adverse effects. Some recommend avoidance of atropine but as the patients are euthyroid, it can be used. Excessive sedation is to be avoided. Majority of cases are straight forward even when there is some tracheal deviation or compression. A firm armored ectopic thyroid tissue (ETT) will negotiate most distorted trachea and permit optimum head positioning. The ETT should be passed beyond the point of extrinsic compression. Airway is established in awake patient. Preoxygenation should be followed by IV induction and a neuromuscular blocking drug after checking the proper placement of ETT. Standard monitoring is done. Extubation should be performed under optimal circumstances for reintubation in the event of tracheal collapse.

Q. What are the postoperative complications of thyroidectomy surgery?

Postoperative complications following thyroidectomy surgery are:

- *Hemorrhage with tense swelling of the neck*: Clot is removed by removing clips from skin and sutures from platysma and strap muscles. In extreme case, this is done at the bedside or the patient is brought to operation room (OR) without delay. Wound dressing is placed in crossing fashion in hematoma.
- *Tracheomalacia*: Long standing goiter may cause tracheal collapse. Immediate reintubation and tracheostomy may be needed. Probability of tracheal collapse prior to extubation of trachea can be detected by noting the amount of cuff inflation required to seal the trachea after intubation. Prior to extubation, oropharynx is sucked and ETT is deflated. Then it

is noted whether leak to positive pressure ventilation returned or not. If no leak of gases, there is probability of tracheal collapse around ETT. Extubation is deferred for few hours.
(Leak Test: The valve in the breathing circuit is partially closed. The bag is squeezed with increasing pressure till an audible leak is detected around the ETT).

- *Recurrent laryngeal nerve injury*:
  - Bilateral recurrent laryngeal nerve injury causes stridor and laryngeal obstruction resulted from unopposed adduction of the vocal cords and closure of the glottis aperture. Immediate tracheal intubation is required followed by tracheostomy to ensure an adequate airway.
  - Unilateral recurrent laryngeal nerve injury often goes unnoticed because of compensatory over adduction of the uninvolved cord.
    Vocal cord function is tested preoperatively and postoperatively by asking the patient to say 'e' or 'moon'. In unilateral nerve injury, there is hoarseness and in bilateral nerve injury, there is aphonia.
  - Selective injury of the adductor fiber of both the recurrent laryngeal nerve leaves abductor muscle unopposed and pulmonary aspiration is a risk.
  - Selective injury of the abductor fibres of both the recurrent laryngeal nerve leaves the adductor muscle unopposed and airway obstruction can occur.
- *Hypocalcemia*: Intimate involvement of parathyroid gland with thyroid gland may result in inadvertent removal of parathyroid gland during thyroid surgery leading to hypocalcemia. Serum calcium should be checked at 24 hours and again daily if low.
  Presenting features are signs of neuromuscular excitability, tingling around the mouth and tetany. May progress to fits or ventricular arrhythmias.
  Diagnosis is done by:
  - Carpopedal spasm (flexed wrist with fingers drawn together) may be precipitated by inflation of BP cuff—Trousseau's sign.
  - Facial twitching precipitated by tapping over the facial nerve at the parotid gland—Chvostek's sign.
  - Prolonged QT interval on ECG.

  Treatment: Serum calcium levels with below 2 mmol/L should be treated urgently with 10 mL of 10% calcium gluconate over 10 minutes followed by 40 mL in 1 liter saline over 8 hours. Serum calcium is checked after 4 hours. Calcium infusion is considered if level is still low. If hypocalcemic but level is >2 mmol/L, treatment with oral supplement calcium is done.
- Thyroid crisis or thyroid storm— discussed previously.
- Myxedema coma— discussed previously.

Q. When a hyperthyroid patient is considered euthyroid and safe for anesthesia?

When there is: (i) H/O relief of symptoms; (ii) sleeping pulse rate below 85/min; (iii) iodine uptake returned towards normal; (iv) tremor relieved

(v) disappearance of cardiac murmurs and hyperreflexia. Anesthesia is performed under optimal circumstances for reintubation in tracheal collapse.

Q. What are the implications of pregnancy in hyperthyroidism and hypothyroidism?

Hyperthyroidism in pregnancy: Pregnant women with hyperthyroidism are treated with propylthiouracil as carbimazole may cause fetal abnormalities. Whichever thiourea is used, it should be given in smallest dose, permitting mild hyperthyroidism, which is well-tolerated by pregnant women. Both carbimazole and propylthiouracil cross the placenta and can induce hypothyroidism with fetal, TSH hypersecretion and goiter. Thyroid hormone administration to mother does not prevent hypothyroidism in fetus, since T_4 and T_3 do not cross the placenta. Fetal hypothyroidism is rare if the mother's hyperthyroidism is controlled with small dose of propylthiouracil (50–150 mg/day orally) or carbimazole (5–15 mg/day orally). Thyroidectomy is reserved for women who are allergic to or are resistant to antithyroid drug or who have very large goiter. Fetal ultrasound at 32 weeks gestation can visualize any fetal goiter which can be treated.

The use of either propylthiouracil or carbimazole during breastfeeding does not significantly affect the infant's thyroid hormone levels and both drugs are approved for nursing mother.

If subtotal thyroidectomy is necessary during pregnancy, it is most safely performed in the second trimester. Radioactive iodine is absolutely contraindicated in pregnant women as it invariably leads to fetal hypothyroidism.

Hypothyroidism in pregnancy: Most pregnant women with primary hypothyroidism require an increase in the dose of thyroxin, about 50 μg daily to maintain normal TSH levels. This reflects increased metabolism of thyroxin by placenta and increased thyroxin binding globulin during pregnancy, resulting in an increase in total thyroid hormone pool to maintain the same T_4 and T_3 concentrations. Recent researches suggest that inadequate maternal T_4 therapy is associated with impaired cognitive development in their offsprings. Serum TSH and free T_4 should be measured during each trimester and the dose of thyroxin adjusted to maintain a normal TSH.

Q. What are the presenting features of hypothyroidism? How hypothyroid patients are treated?

Manifestations of hypothyroidism:

Symptoms: Weight gain, fatigue, lethargy, weakness, depression, dyspnea on exertion, arthralgia, cold intolerance, constipation, dry skin, headache, carpal tunnel syndrome, aches and pain, muscle cramps and menorrhagia.

Physical findings: Bradycardia, diastolic hypertension, hoarse voice, goiter, myxedema, anemia, ascites, pleural effusion, pericardial effusion and delayed relaxation of deep tendon reflexes and palpable enlarged thyroid (goiter).

Serum concentration of thyroid hormone is in normal range and only serum TSH levels are raised (normal range of TSH is 0.3–4.5 mu/L). TSH values of 5–15 mu/L are characteristic of hypothyroidism.

Complications:

- Hypothermia, stupor or myxedema coma may develop in patients with severe hypothyroidism.
- Delayed gastric emptying, increased chance of gastric content aspiration during induction of general anesthesia.
- Significant electrolyte imbalance, hyponatremia.
- Associated amyloidosis causes enlarged tongue, which hampers laryngoscopy and tracheal intubation.
- Prolonged postoperative intubation and ventilatory support may be needed as there is depression of ventilatory response to hypoxia and hypercapnia.
- Pregnancy in a woman with untreated hypothyroidism often results in miscarriage and may cause mental and physical abnormalities in offspring.

Treatment: Levothyroxine (thyroxin, T_4) is the treatment of choice. It is partially converted to T_3 in the body, the active thyroid hormone. Most of the patients require life-long thyroxin therapy. Treatment is started slowly. A dose of 50 μg/day should be given for three weeks, increasing thereafter to 100 μg/day for a further 3 weeks and finally in a maintenance dose, usually 100–150 μg/day. Thyroxin should always be taken as a single daily dose and at least 6 weeks should pass before repeating thyroid function tests and adjusting the dose in increment of 25 μg/day. The correct dose of thyroxin in the long-term is that which restores serum TSH to normal range. To achieve this serum level, T_4 is to be in the upper part of normal range or slightly raised because the T_3 required for receptor activation is derived exclusively from conversion of T_4 within the target tissues. Once the dose of thyroxin has been established, it is important to measure the thyroid function every 1–2 years.

Q. What is the anesthetic management in a patient with hypothyroidism?

There is little reason to postpone elective surgery in patients who have mild-to-moderate hypothyroidism. Thyroid replacement therapy is indicated in patients with severe hypothyroidism or myxedema coma and for pregnant patients who have hypothyroid. A number of anesthetic medications have been used without difficulty in hypothyroid patients. Although ketamine has been proposed as the ideal inducing, thiopentone has been used in hypothyroid patients. Maintenance of anesthesia is safely achieved with either intravenous or inhaled anesthetics. There is very little decrease in MAC for volatile agents. As drug metabolism is slow, drug doses are reduced especially for muscle relaxants and opioids. As the patients are susceptible to profound hypotension, all drugs are administered slowly.

Regional anesthesia is a good choice in hypothyroid patients provided the intravascular volume is well-maintained.

Monitoring is directed towards the early recognition of hypotension, congestive heart failure and hypothermia. Scrupulous attention should be paid to maintain normal body temperature. Ventilatory responsiveness to hypoxia and hypercapnia is depressed in hypothyroid patients. This depression is potentiated by sedatives, opioids and general anesthetics. Postoperative ventilatory failure requiring prolonged ventilatory support is rare in hypothyroid patients in absence of co-existing lung disease, obesity or myxedema coma.

Decreased gastrointestinal motility can compound the effect of postoperative ileus.

In long standing cases, stress response may be blunted and adrenal depression may occur.

The recommendation is that all hypothyroid surgical patients must be restored to a euthyroid state before surgery because most of the hypothyroid patients are sensitive to anesthetic drugs, have prolonged recovery time or have higher incidence of cardiovascular instability or collapse. Surgery in severely hypothyroid patients should be postponed where possible until these patients are at least partially treated.

Several studies have found no differences in frequency of intraoperative and postoperative complications when mild or moderately hypothyroid patients underwent cardiac surgery. In symptomatic and unstable patients with cardiac ischemia, thyroid replacement is delayed until after coronary revascularization.

Chapter

10

Total Hip Replacement in an Elderly Patient with COPD

Q. What is chronic obstructive pulmonary disease (COPD)?

Chronic obstructive pulmonary disease is characterized by the progressive development of expiratory airflow obstruction which is not fully reversible.

Q. What pathological conditions are associated with COPD?

- Chronic obstructive bronchitis
- Emphysema and mucus plugging

Q. What are the presenting features of COPD?

The presenting features of COPD include chronic productive cough and progressive exercise limitation due to dyspnea from expiratory airflow obstruction.

Wheezing is common in presence of mucus accumulation in airway and may mimic asthma.

Q. How COPD is differentiated from asthma?

Asthma is characterized by chronic airway inflammation, reversible expiratory airway obstruction and airway hypersensitivity. COPD patients exhibit fixed obstruction to expiratory airflow and airway hyperactivity is not unique.

Q. How the diagnosis of COPD is confirmed?

- Pulmonary function testing (spirometry): Decrease in FEV1, FEV1/FVC ratio, PEFR and greater decrease in forced expiratory flow between 25% and 75% of vital capacity.
  Normal values: FEV1: 3 L in man FEV1/FVC >70%
  2 L in woman PEFR >200 L/min
- Arterial blood gas analysis
- Bed side pulmonary function tests (PFTs).

Q. What are bedside PFTs?

- Ability to talk continuously for 2 minutes (normal)
- Match blowing test: Lighted match held at 6 inches away from the patient. If can be extinguished by blowing with mouth wide open––normal). If not, FRC is less due to the disease process.
- Breath holding time: Deep breath and holding:
  - Normal >30 seconds
  - Chance of pulmonary complications <15 seconds
- Expiratory time: Put stethoscope over trachea at suprasternal notch
  - Normal: 4 seconds
  - Prolonged: COPD, shortened-hyperventilation
- Sphygmomanometer test: Patient is asked to blow and raise column of Mercury (Hg)
  - Normal: Raise of column up to 40–50 mm Hg

  Note the duration for which the patient can hold it at that level.
- Wright's respirometer:
  - Normal values
    - Tidal volume (TV): 7 mL/kg
    - Minute volume (MV): 8–10 L/min
    - Respiratory rate (RR): 12–16/min
- Peak flow meter: It measures the peak expiratory flow rate (PEFR)
  - Normal values
    - Adult: 300–400 L/min
    - Children: 200–250 L/min.

Q. What are the categories of COPD patients?

Using arterial blood gases analysis, COPD patients are categorized into the following:

- Blue bloaters with PaO_2 lower than 65 mm Hg and $PaCO_2$ more than 45 mm Hg. They exhibit recurrent episodes of cor pulmonale.
- Pink puffers with PaO_2 usually higher than 65 mm Hg and $PaCO_2$ are normal or slightly decreased. Cor pulmonale develops only rarely.

Q. Why cor pulmonale develops in blue bloaters?

Arterial hypoxemia and respiratory acidosis evoke pulmonary vascular constriction leading to chronic pulmonary hypertension, and in response to it, cor pulmonale develops (Sustained pulmonary vasoconstriction, hypertrophy of vascular smooth muscle, irreversible increased in PVR, chronic pulmonary hypertension—Cor pulmonale).

Q. What are the clinical features of cor pulmonale?

- Jugular venous distension (enlargement)
- Peripheral edema
- Passive hepatic congestion (enlarged liver).

All the features are due to systemic venous hypertension, which resulted from right ventricular failure.

Q. What is the treatment of cor pulmonale?

- Supplemental O_2 to maintain PaO_2 >60 mm Hg
- Diuretics
- Digitalis
- Antibiotics.

Q. What is the most important precipitating factor of COPD?

The most important precipitating factor of COPD is infection.

Q. What is the treatment of COPD?

- Supplemental O_2 if PaO_2 <55 mm Hg or hematocrit >55%. The goal is to maintain PaO_2 at 60–80 mm Hg by giving O_2 through nasal cannula at a rate of 2 L/min. Oxygen therapy can dangerously elevate $PaCO_2$ and precipitate respiratory failure. Abolition of hypoxic drive by elevation of $PaCO_2$ to normal leads to severe hypoventilation.
- Beta agonist
- Anticholinergic drugs
- Inhaled corticosteroids
- Broad-spectrum antibiotics
- Diuretics (If right-sided heart failure present)
- Cessation of cigarette smoking.

Q. What is the most important predisposing factor of COPD?

The most important predisposing factor of COPD is chronic cigarette smoking.

Q. What are the ill-effects of cigarette smoking?

- Decreased O_2 carrying capacity due to increased level of carboxyhemoglobin resulted from carbon monoxide
- Sympathomimetic effect of nicotine on heart
- Mucous hyper secretion, impaired mucociliary transport activity and narrowing of small airways
- Interfere with normal immune response leading to pulmonary infection anesthesia and surgery
- Stimulate hepatic enzymes which could alter postoperative analgesic requirement.

  All these increase the incidence of postoperative pulmonary complications.

Q. How ill-effects of cigarette smoking are corrected?

- Abstinence of 6 hours increases P_{50} from 22.9 to 26.3
- Smoke-free interval of 12–18 hours improves oxygen carrying capacity
- Abstinence from cigarette smoking for 6–8 weeks is needed to decrease the incidence of postoperative pulmonary complications.

Q. What are the risk factors for postoperative pulmonary complications?

- Pre-existing pulmonary disease
- Advanced age (>60 years)
- Smoking
- Thoracic and upper abdominal surgery
- Prolonged general anesthesia (>3 hours)
- Poor general health
- Incision >30 cm.

Q. How postoperative pulmonary complications are reduced in COPD patient?

Preoperative:

- Encourage cessation of smoking for at least 6–8 weeks
- Treatment of expiratory airflow obstruction with bronchodilators (beta adrenergic agonist, glucocorticoids, ipratropium)
- Treatment of infection with antibiotics
- Patient education about lung volume expansion maneuvers (deep breathing, incentive spirometry, chest physiotherapy)
  For operation, regional anesthesia is considered.

Postoperative:

- Maximum pain relief (neuraxial block, PCA, nerve block)
- Institution of lung volume expansion maneuvers.

Q. What is the utility of PFT and arterial blood gas analysis (ABG) analysis?

They help as a management tool to optimize preoperative pulmonary function but not as a means to assess postoperative risk.

Q. How preoperative assessment and optimization of COPD patient is done?

Assessment:

- History (exercise tolerance, chronic cough and dyspnea)
- Physical examination (decreased breath sounds, wheezing, prolonged expiratory phase)
- PFT and ABG analysis.

Optimization: Optimization is done by treating existing signs and symptoms and therapy improving the pulmonary functions.

Q. What preoperative medication is done?

- Sedation with benzodiazepines
- Bronchodilators are continued up to the time of surgery
- Hydrocortisone (50–100 mg preoperatively in patient on long-term steroid therapy).

Q. What should be the aims of anesthetic management in this case?

- Preoperative effort to optimize pulmonary functions
- Intraoperative management to minimize residual depressant effect of anesthetic drugs on respiration
- Postoperative pain management to improve ventilation and oxygenation.

Q. Why regional anesthesia is preferred?

- Alteration in pulmonary physiology is usually minimum with neuraxial block
- Instrumentation of airway and positive pressure ventilation can be avoided
- Residual depressant effect of anesthetic drugs on breathing can be avoided
- Postoperative pain relief can be produced adequately to improve ventilation and oxygenation
- Early mobilization reduces chance of DVT and pulmonary embolism
  All these help reducing postoperative pulmonary complications.

Q. What are the immediate complications that can occur during placing epidural needle and catheter?

- Intravascular injection affecting CNS and CVS
- Total spinal anesthesia due to inadvertent intrathecal injection.

These complications can be avoided by:

- Carefully aspirating before every injection
- Using test dose
- Injecting local anesthetic in incremental doses
- Close observation of early signs (tinnitus and lingual sensation in intravascular injection. Loss of sensation in spinal anesthesia).

Q. What should be the postoperative management in this case?

- Relief of postoperative pain with neuraxial opioid.
- Early mobilization
- Lung volume expansion technique
- Prevention of acute respiratory failure.

Q. What are the problems associated with hip replacement surgery?

- Blood loss
- Pain
- Deep vein thrombosis (DVT)and pulmonary embolism
- Bone cement implantation syndrome leading to hypotension, hypoxia, or cardiac arrest

Treatment:

- Oxygenation
- Volume replacement
- Vasopressors.

Q. How blood loss can be reduced during surgical procedure?

- Patient should avoid antiplatelet drugs (NSAIDs, Aspirin) prior to surgery
- Anesthetic technique to reduce MAP (<55 mm Hg)
- Patient should be positioned to minimize venous pressure
- Hypothermia should be avoided
- Surgical techniques should be efficient and meticulous to reduce time
- Topical application of bone glue, bone wax
- Administration of desmopressin, tranexamic acid and aprotinin.

Q. How the risk of homologous transfusion can be minimized?

- Presurgical banking of autologous blood
- Immediate preoperative autodonation by hemodilution
- Salvage of patient's intraoperative blood loss.

Q. What measures are adopted for postoperative pain management?

- IV/IM narcotic analgesics until oral administration
- PCA morphine is used
- Regional (epidural/spinal) administration of opioid
- Intra-articular injection of local anesthetics
- COX-2 selective NSAIDs (new drugs can be used parenterally)
- Multimodal analgesic regimens.

SECTION 2

Short Cases

Section Outline

- Cystic Hygroma
- Tracheostomy
- TOF with Shunt
- Inguinal Hernia
- Congenital Talipes Equinovarus
- Temporomandibular Joint Ankylosis
- Patent Ductus Arteriosus
- Intercostal Drain
- Hydrocephalus
- Meningomyelocele
- Cataract
- Strabismus
- Diabetic Foot Complications
- Neck Contracture Following Upper Body Burns

Chapter

11

Cystic Hygroma

Q. What is cystic hygroma? What are the presenting features of it?

Cystic hygroma is benign, multilolcular, lymphatic tumor of neck/oral cavity/ tongue. Many being detected at birth and majority appearing in 1st 2 years of life. They distort local anatomy. They invade tissue planes involving skin, subcutaneous tissue, muscle and the structures of pharynx and larynx and may present with upper airway obstruction. Obstructed airway in awake patients may totally obstruct on induction, oral intubation often impossible due to enlarged tongue and tracheostomy is complicated with submandibular involvement.

Investigations: Radiography of neck and chest to demonstrate displacement of airway and mediastinal involvement.

Treatment: Surgical excision is the treatment of choice and should be done as early as possible, once the diagnosis is made. Respiratory difficulties are an indication of urgent treatment often requiring tracheostomy.

Q. What is airway? What is upper airway? What is lower airway?

Airway is the passage through which the air passes during respiration. Upper airway comprises of mouth, nasopharynx, oropharynx, pharynx and larynx.

Functions: It serves to filter, warm and humidify the air/ gas before entering lower airway.

Lower airway includes trachea, bronchi or bronchioles which terminate in the alveoli.

Q. What is meant by: difficult airway; difficult mask ventilation; difficult endotracheal intubation; difficult laryngoscopy?

Difficult airway: ASA defined difficult airway as the clinical situation in which a conventionally trained anesthesiologist experiences difficulty with mask ventilation, difficulty with tracheal intubation or both.

Difficult mask ventilation: It is the situation when it is not possible for unassisted anesthesiologist to maintain oxygen saturation >90% using 100% oxygen of positive pressure mask ventilation in a patient whose oxygen saturation was >90% before anesthesia.

Difficult endotracheal intubation: ASA Task Force defined it to be occurring when insertion of tracheal tube with conventional laryngoscopy requires more than three attempts or more than 10 minutes.

Difficult laryngoscopy: ASA Task Force defined it to be occurring when it is not possible to visualize any portion of the vocal cords with conventional laryngoscopy.

Q. What is Cormack and Lehane's gradation of view and its anesthetic implication?

Cormack and Lehane's gradation of laryngoscopic view:
Easy intubation:
- Grade I: Visualization of entire vocal cords
- Grade II: Visualization of posterior part of laryngeal aperture.

May not be intubated. The alternative techniques required are:
- Grade III: Visualization of epiglottis only
- Grade IV: No glottis structures seen.

Q. How does the child's airway differ from that of an adult?

The child's airway differs from adult in many ways including size, shape and position.

In newborn, the upper airway is smaller. Tongue is relatively large and occupies greater portion of oral cavity and oropharynx causing difficult laryngoscopy. They are obligate nasal breathers, so nasal obstruction is hazardous. Tonsils and adenoid appear in 2nd year, attaining largest size by 4–7 years and snoring and sleep apnea.

Larynx is higher and more anterior: Mid C_3 for preterm, C_3–C_4 for full term and C_4–C_5 for older children. It is funnel-shaped in children below 8 years. Tracheal position is downward and posterior, making application of cricoid pressure more effective in infants. The distance between vocal cords and tracheal bifurcation is 4–5 cm. So the endotracheal tube (ETT) must be carefully positioned and fixed. The right main bronchus is less angulated than the left. So ETT slips most often into it, as also the inhaled foreign bodies. The neonates and infants have large head which tends to flex their small and weak neck.

Highly compliant chest wall and horizontally placed ribs without bucket handle movement, increases work of breathing.

The Diaphragm is the major muscle of respiration, but its muscle fibers are immature having less type I, fatigue-resistant muscle fibers; hence, are less efficient leading to respiratory failure or apnea with significant increase in work of breathing, e.g. airway obstruction.

Oxygen consumption is approximately 6 mL /kg (compared to 3 mL/kg in adults). Airway obstruction produces hypoxia more rapidly in infants and neonates.

Q. What are the classifications of pediatric difficult airway?

The classification of difficult airway in pediatric patients is as follows:

- *Congenital:*
  - Craniofacial abnormality: Pierre Robin, Treacher Collins, Goldenhar
  - Cervical spine: Down syndrome, Klippel–Feil
  - Laryngotracheal: Tracheomalacia
- *Structural:* Aspirated foreign body, trauma, tracheal stenosis, post-intubation edema temporomandibular joint (TMJ) ankylosis, post-burn contracture.
- *Inflammatory:* Croup, epiglottis, laryngeal papillomatosis, retropharyngeal abscess, peritonsillar abscess.
- *Neoplastic:* Cystic hygroma, tumors, encephalocele

Q. How to assess the difficult pediatric airway? What are the problems and their management?

Mallampati classification and thyromental distance are not validated in pediatrc patients. Mouth opening, neck and jaw mobility are difficult to assess in non-cooperative child. Assessment begins with comprehensive history and physical examination. History of snoring, apnea, stridor, hoarse voice and prior anesthetic experience are important. Whether breathing is easier or not, whether opens mouth during crying or not, all are important.

Appearance: Agitation, dyspnea, chest retraction, weak or absence cry or drooling of saliva are significant.

Crowing on inspiration is indicative of extrathoracic airway obstruction. Noise on exhalation is due to intrathoracic lesions.

Distress of noise during both inspiration of expiration are due to lesion at or just above the thoracic inlet.

A clinical classification based on symptoms of presentation is useful in management of difficult airway. These children are divided into four main types:

Type I patients: These children present with normal respiratory frequency, mild respiratory distress, normal oxygen saturation and airway having normal look with minimum or non-existent sternal retraction.

Type II patients: These children have significant airway disease and moderate airway distress but have known airway, as they have under gone and surgical procedure.

Type III patients: These children have definite physical abnormality with or without respiratory distress. It Includes: micrognathia, macroglossia and severe palatofacial deformity or tumors displacing the airway like cystic hygroma and anterior mediastinal tumor.

Type IV patients: These children present with significant airway obstruction with symptoms like stridor, sternal retraction, drooling, cyanosis, low oxygen saturation or signs of fatigue.

There are two main problems encountered with these patients: (1) Effective control of airway; (2) Intubation.

The technique of induction of anesthesia depends on the severity of pathology in airway and degree of respiratory difficulty. An alternative plan for securing airway should always be ready.

Type I and Type II children may be given mild sedative premedication.

Induction is done with halothane or sevoflurane. The child is kept on spontaneous breathing until adequate depth of anesthesia is reached, assessed by laxity of the jaw, regularity of respiration and eye signs (Eyes centrally placed, loss of conjunctival reflex and pupils normal or small). Positive pressure is applied gradually and possibility of ventilating the child is confirmed. Positive pressure also decreases the obstruction caused by the soft tissues. If ventilation is easy, a muscle relaxant may be given of the child intubated. If ventilation is not adequate, muscle relaxant should not be given, as it may lead to complete airway obstruction due to loss of muscle tone of tongue and pharynx. Induction is attempted on spontaneous ventilation.

The same principle as above is observed for Type III and Type IV children with special requirement of personnel and equipment in anticipation of difficult airway. A surgeon should be standing nearby to establish surgical airway, if necessary, and also an anesthesiologist. These children are not sedated preoperatively if there is a fear of losing the airway.

Q. What are the alternative techniques to secure the airway?

There are several alternative techniques to secure the airway. They are:

- Oropharyngeal of nasopharyngeal airways: These can improve mask ventilation. The size measurement of orotracheal airway is done from angle of the mouth to angle of the mandible.
  If the size is too large, it passes into esophagus, impinge on the epiglottis, close the glottis a traumatize uvula.
  If the size is too small, it will push the base of the tongue and worsen airway obstruction.
- Laryngeal mask airway: It is used as definitive airway for many surgical procedures, as a conduct for intubation and as temporary airway during establishment of surgical airway. It cannot substitute ETT.
- Fiberoptic intubation:
  - May be done in older children under sedation with midazolam or propofol after topicalization of the airway.
  - Younger children are kept asleep under general anesthesia using nasopharyngeal airway of fiberoptic-guided intubation performed through other nostril. Useful in children with limited mouth opening as in TMJ ankylosis.
- A blind nasal intubation may also be attempted under deep general anesthesia.
- Jet ventilation through a stylet may be used to establish ventilation, with a path for egress of gases to prevent barotraumas.

- Percutaneous needle cricothyroidotomy: Emergency airway of choice in "cannot ventilate, cannot intubate" situation.
  Procedure: The head is extended over shoulder. After locating cricothyroid membrane, a 12 or 14 gauge IV cannula is inserted into it. Air is aspirated, the needle is withdrawn and the cannula is advanced downward. The cannula is connected to standard circuit source of oxygen via a 15 mm connector from a 3.0 mm ETT or through a barrel of 2.5 mL syringe, connected to a 15 mm connector 7.5 mm ETT. Alternatively, a jet ventilation system can be connected directly to the cannula.
  Complications: Barotraumas with pneumothorax, subcutaneous or mediastinal emphysema, vascular injury, esophageal puncture, hypercarbia and catheter kinking or dislodgement.
  Low flow (1L/min) of oxygen is all that is needed to support oxygenation and this will reduce the risk of barotrauma.
- Dilatational percutaneous tracheostomy or surgical tracheostomy: Methods of establishing surgical airway.

Q. What is "cannot ventilate, cannot intubate" situation?

"Cannot ventilate" situation may be defined as very poor or non-existent mask ventilation (failure to keep SaO_2 >90% on 100% oxygen) despite two persons giving simultaneous jaw thrust and applying face mask with appropriate size oropharyngeal or nasopharyngeal airways. The first person gives jaw thrust and holds the mask, the second person only squeezes the bag.

"Cannot intubate" situation is considered when an experienced anesthetist using direct laryngoscopy fails to intubate the trachea, despite taking intubation attempts under optimum condition (adequate relaxation, sniffing position, change of laryngoscope blade).

Patients to be considered while selecting ETT for infants:

- Cricoid is the narrowest part of the trachea and is circular; so too large tube produces ischemia of the tracheal mucosa (adult-vocal cord is the narrowest part and not circular).
- In pediatric age group, the pseudostratified ciliated epithelium is loosely bound to the underlying layers, so trauma to the airway results in edema. 1mm edema causes 75% reduction in cross-sectional area and 16 folds increase in resistance (adult—44% reduction in cross-sectional area or 3 folds increase in resistance).
- Small difference in radius makes big difference in flow through ETT by increasing airway resistance leading to increased work of breathing.

Available versions are: (1) PVC (cuffed/uncuffed); (2) Reinforced armored tube for head and neck surgery; (3) Preformed ETT for cleft palate surgery—Oxford, Ring Adair Elwin (RAE) tubes; (4) Double lumen tube—Smaller size available is 26 Fr Left; (5) Single lumen bronchial blocker (Arndt blocker).

Types of tube of correct sizes:

- It should be large enough to allow controlled or spontaneous ventilation but not too large to damage trachea.

- It should seal the trachea against aspiration size selection:
  - Measuring the diameter of the distal joint of the index finger of the child or lumen of external nares.
  - Calculation based on child's age: 3 mm for newborn, 4 mm for 1 year, 5 mm for 2 years. For older children, it is calculated by the formula:

$$\frac{\text{Age} + 16}{4} \text{(uncuffed)}$$

  - Penlington's formula:

$$< 6 \text{ year} - \frac{\text{Age in years}}{3} 3.5$$

$$< 6 \text{ year} - \frac{\text{Age in years}}{4} + 4.5$$

  Tips regarding mask ventilation of intubation Mask ventilation: The child is positioned with a small roll under shoulder (except in older children) as the neck may be hyperflexed due to the large head. Overextension of the head is avoided as it may worsen the obstruction. In older children, small pillow or folded blanket is placed under head to cause anterior displacement and the chin is pointed upward to extend the neck, so as to bring oral, pharyngeal and tracheal axes into straight line.
- Some children maintain airway in lateral position
- A mask with minimum dead space is used and applied over the face so that it does not occlude nostrils or exert pressure on eyes
- A jaw thrust applied by pressure behind the angle of the mandible provides an open airway in most cases. Pressure should not be applied on submandibular soft tissues, as this pushes the tongue up towards the palate which increases obstruction
- An appropriate size of oropharyngeal airway may help. Insertion done in light planes of anesthesia may cause laryngospasm and induce vomiting.

Laryngoscopy and intubation:

- Continued with roll under the shoulder in the neonates and infants
- Straight blade of laryngoscope is more useful as the larynx is high with anterior inclination. Large floppy U-shaped epiglottis can be picked up by the straight blade of the laryngoscope. Often it is not necessary to pick up the epiglottis and intubation may be accomplished as in adult
- As tracheal direction is downwards of posterior in infants, external pressure on the larynx pushing it backward markedly improves the view
- In older child, folded blanket, towel or small firm pillow under the head with extension of neck, bring the glottis into view
- Since the vocal cords are angled such that anterior commissure is caudal to the posterior commissure, the tracheal tube hitches the anterior commissure. Slight withdrawal or rotation of the tube or flexion of the neck will encourage the tube to slip in

- During laryngoscopy and placement of tracheal tube, it should be checked that the tube is at mid-tracheal level. The position is checked with auscultation for bilateral air entry, both with the neck flexed and extended, carefully preventing accidental extubation. The length of the tube at lips is noted and fixed carefully.
- Intubation guides, stylets, gum elastic bougies, curved anteriorly to a hockey stick configuration are helpful in case of anterior larynx.

Extubation: Extubation in cases of difficult airway may cause problems because:

- The cause of difficult airway may still be present, e.g. a craniofacial abnormality
- Surgery may have worsened the situation, e.g. wiring of the jaws
- Repeated attempts at intubation or use of too large ETT may lead to trauma and laryngeal edema, increasing the airway resistance.

Treatment:

- Humidified oxygen
- Nebulized adrenaline (racemic adrenaline)
- Intravenous or inhaled steroids.

In all those cases:

- Extubation is not done if there is any doubt regarding the ability to resecure the airway if needed
- After extubation the child is kept in OR or high dependency unit (HDU) until the airway and respiration are satisfactory
- The child is kept as comfortable as possible rapport is developed with the child and parents
- If IV access is to secure prior to procedure as in Type III and IV children, it should be done using topical analgesia as local anesthetic cream or a small injection of local anesthetic. Muscle relaxant should not be administered without being definite that ventilation can be done.

Chapter

12

Tracheostomy

Q. What is a percutaneous airway? What are the types of percutaneous airway?

A percutaneous (transcutaneous) airway connects the trachea and lower airway to the atmosphere, anesthesia circuit or other device through a surgically created airway in front of the neck that bypasses the larynx of upper airway. Emergency percutaneous airway is necessary when noninvasive technique fails to relieve. They cannot ventilate and cannot intubate situation and severe increasing hypoxemia develops. Creation of percutaneous airway involves significant hazards. There are two types of percutaneous airways: (i) Tracheostomy, (ii) cricothyroidotomy or (iii) cricothyrotomy.

Tracheostomy is insertion of a tracheal tube though neck incision.

Cricothyroidotomy is insertion of a 14 gauge catheter over needle device into the trachea through the cricothyroid membrane.

Cricothyrotomy uses surgical technique to insert a cuffed tube into the trachea.

Q. What are the indications of tracheostomy? How tracheostomy is done?

Indications of tracheostomy are*:*

- To maintain an open airway in presence of obstruction to the pharynx or larynx, which prevents endotracheal intubation
- To protect the airway from aspiration of gastric or pharyngeal contents in patients whose protective mechanisms are absent or poor
- To facilitate suction of secretions from within the tracheobronchial tree (pulmonary toileting)
- To provide prolonged mechanical ventilator support
- Often used for the temporary management of airway in the preoperative period during head and neck surgery
- Following surgical resection of oral cavity and oropharyngeal cancer, bleeding into the sublingual and submaxillary soft tissue spaces may result in airway compromise and elective tracheostomy is indicated to prevent loss of airway.

Anesthesia: The tracheostomy can be done safely with local infiltration supplemented with sedation/opioid drugs appropriate to general condition of the patient. This allows for the administration of high level of inspired oxygen. In patients with stable cardiovascular system, general anesthesia (GA) with or without supplemental local infiltration are satisfactory. ECG, BP, pulse oximetry and end tidal CO_2 monitoring are mandatory.

Procedure: Patient is placed supine with neck extended with roll towel or small pillow underneath the shoulder. Skin preparation and surgical draping are done aseptically. Small skin incision is made at the level of 2nd tracheal ring. Strap muscles are separated in midline Thyroid isthmus is divided and sutured to stop bleeding. The 2nd and 3rd tracheal rings are opened vertically in midline. A proper sized tracheostomy tube is placed after retraction of the lateral tracheal wall. The tube position is confirmed by positive pressure ventilation of inspection of chest wall expansion, confirmed by CO_2. The previous ETT is removed. The wound is closed and skin suture placed.

A high tracheostomy is always preferable as it enables the tip of the tracheostomy tube to be well above carina. It is best to use larger tube that can be comfortably accommodated by the trachea.

Early tracheostomy has benefits over late tracheostomy. Advantage of early tracheostomy are decreased duration of mechanical ventilation, decreased incidence of pneumonia, enhancement of ventilator wearing, decreased ICU stay and decreased hospital cost. Early tracheostomy should be performed in patients who will encounter difficult and prolonged weaning. Timing of tracheostomy does not alter the mortality or increase the risk of hospital acquired pneumonia.

Q. What are the advantages of tracheostomy?

Advantages of tracheostomy are*:*

- It requires less skilled care
- Allows a more secured and manageable airway
- Provides improved patient comfort and less sedation and analgesia are required
- Provides improved ability to communicate and improved lip reading
- There is less laryngeal damage causing reduced laryngeal stenosis and less voice damage
- There occurs less oral injury and oral hygiene is better maintained
- Provides better secretion removal with suctioning (pulmonary toileting)
- There is preservation of glottis competence, resulting decreased risk of aspiration
- There is better preservation of swallowing allowing earlier oral feeding.
- Prolonged orotracheal and nasotracheal intubation is avoided
- Provides less tube dead space, lower resistance to GADS flow and lower work of spontaneous breathing
- There is lower incidence of tube obstruction
- There is lower incidence of ventilator associated pneumonia

- Reinsertion of displaced tube is easy, after the tract is matured
- Allows more rapid wearing from mechanical ventilation
- There is decreased length of ICU and hospital stay with decreased hospital cost.

Placement of a tracheostomy tube does not obligate a patient to loss of speech. When a large cuffed tracheostomy tube is in place, expecting a patient to be capable of normal speech is impractical.

However, after the patient is downsized to an uncuffed tracheostomy tube, intermittent finger occlusion or Passy–Muir value placement will allow a patient to communicate while still using the tracheostomy to bypass upper airway for inhalation.

Q. What are the complications of tracheostomy?

Complications of tracheostomy: Tracheostomies are now performed open or percutaneous, with or without bronchoscopy and with or without Doppler guidance and get complications still arise.

- Hemorrhage in the immediate postoperative period, after a tracheostomy, is usually from local vessels in the incision (anterior jugular and inferior thyroid veins)
- Massive hemorrhage 1–6 weeks postoperative is mostly caused by trachea-innominate artery fistula, which is the most serious complication of tracheostomy. This occurs rarely but has a high mortality rate
- Significant lobar collapse can occur from copious tracheal secretions or mechanical obstruction
- Cardiorespiratory collapse occurs immediately following emergency tracheostomy done to relieve upper airway obstruction, where sympathetic stimulation occurs with catecholamine liberation. Sudden release after emergency tracheostomy leads to collapse. The risk decreases when done in intubated patients who are preoxygenated before tracheostomy
- Pneumothorax or pneumomediastinum and recurrent laryngeal nerve injury may occur during the procedure of tracheostomy
- Misplacement of tracheostomy tube may occur. It may slip out into the pretracheal plane leading to surgical emphysema when the patient is on mechanical ventilation
- Obstruction of tracheostomy tube may occur with inspissated secretion
- Obstruction below the lower end of the tube may occur by over inflated cuff herniating over the lower end
- Ulceration with pressure necrosis of trachea — especially in patients with bleeding tendency
- Respiratory infection — every tracheal aspiration should be performed as an aseptic surgical procedure
- Tracheal stenosis occurs any time from 1 week to 2 years following tracheostomy. Stenosis occurs at tracheostomy site, cuff site and point where the tip of the tube irritates formation of tracheal wall. Granulation tissue formation also may occur

- Tracheal dilatation occurs over the area of cuff pressure and is avoided by using proper cuff pressure (14–24 cm H_2O)
- Tracheomalacia may occur after prolonged use of tracheostomy tube. It leads to inspiratory collapse of the trachea with obstruction of the airway following extubation.

Q. What are the points of consideration for care of tracheostomy?

Points for care of tracheostomy*:*

- Sterile gloves are used for handling tracheostomy tube
- Minimum occluding volume is used for inflating the cuff — minimum leak technique.
- Cuff pressure is measured daily and kept at acceptable limit — 14–24 cm H_2O
- Cuff is deflated periodically, except when contraindicated
- No touch sterile technique is used during suction of secretions
- Preoxygenation with high FiO_2 is done in hemodynamically unstable patients before suction
- Suction should not be done more than 10 seconds
- Large bore suction catheter should not be used and powerful suction force should collapse. Suction catheter should not be more than half the size of tracheostomy tube
- Suction should be stopped if bradycardia or hypotension occurs and FiO_2 is increased to 100% for a short time
- Viscid secretions are liquefied with normal saline and inspired gas is humidified. Physiotherapy is used
- Central position of the tracheostomy tube is assured and blockage of the tube carefully prevented. Tube is changed every 4–5 days
- Tracheostomy wound is covered with sterile dressing of antibiotic ointment.

Complications during suctioning: Cardiac arrest, arrhythmias, hypotension, massive lung collapse, introduction of infection and mechanical irritation of the tracheal mucosa.

Tracheostomy tube change:

- A tracheostomy tube change in a patient with prolonged tracheostomy is usually uncomplicated because the tract has been stable and can be done at bedside
- Changing a fresh tracheostomy tube within the first week can be dangerous as the tract is unstable of soft tissue and can easily close after removal of tracheostomy tube. Attempts to force the tube post the tract can result in false passage subcutaneous emphysema, inability to oxygenate and a critical situation.
- Early tube changes should be avoided but may be necessary to optimize the size and reduce the leak. This early tube change is done using airway exchange catheters after preoxygenation.

Q. What are the indications for removal of the tracheostomy tube?

Removal of tracheostomy tube is considered safe when the reasons for doing tracheostomy no longer persist and patient can tolerate capping of the tracheostomy tube for more than 24 hours. Patient has normal protective reflexes and can handle upper respiratory secretion and swallow well. Patient can breathe spontaneously and cough effectively. Therefore, the tracheostomy tube is removed when it is no longer needed. The tracheostomy orifice is closed with sterile dressing before removing the tube. The tracheal stoma usually heals and closes within 3–7 days if extubation.

If an upper airway mass or tissue reconstruction was the indication for the tracheostomy, pre-decannulation flexible bronchoscopic examination of the airway is recommended.

Q. What are the types of tracheostomy? What are the indications and advantages of them?

Types of tracheostomy are:

- Surgical tracheostomy or open tracheostomy — described above.
- Percutaneous tracheostomy — alternative to open surgical approach.
- Mini tracheostomy — provides a permanent access to the trachea for suction while avoiding to the trachea for suction while avoiding disadvantages of conventional methods.

Percutaneous tracheostomy (PCT)

Types of percutaneous tracheostomy:

- Ciaglia technique
- Griggs technique
- Blue rhino/Rhino horn technique
- Fantony's technique
- Pere twist technique.

Advantages of percutaneous tracheostomy:

- Fever personnel requirement
- Done at bedside. Smaller skin incision
- Quicker than surgical tracheostomy
- Decreased incidence of surgical complications tissue trauma of wound infection
- Transferring patient to OT is avoided
- Expense of OT resources avoided, so cost-effective.

Most popular technique is known as percutaneous dilatational tracheostomy (PDT), described by Ciaglia.

Principle: Use of serial dilators is introduced into the trachea over a guidewire and is generally done with bronchoscopic visualization. Several commercial PDT kits are available providing all the necessary equipment for efficient or expedient application.

Exclusion criteria for PCT:
- Emergency tracheostomy
- Suspicion or evidence of difficult airway
- Previous airway problems
- Coagulopathy
- Previous tracheostomy.

Through safe and effective, is favorable only for a particular subset of ICU patients.

In following situations, surgical tracheostomy is preferable to PCT:
- Coagulation abnormalities
- High level of ventilator support
- FiO_2 > 0.7 and PEEP >10 cm H_2O
- Unstable/fragile cervical spine
- Neck injury, obesity
- Unfavorable neck anatomy (Previous surgery or tracheostomy).

Minitracheostomy

It provides a permanent access to the trachea for suction while avoiding the disadvantages of conventional methods. The procedure of minitracheostomy is simple and consists of percutaneous tracheal cannulation using 4 mm protex pediatric ETT inserted through a 1 cm incision in the cricothyroid membrane under local anesthesia.

Procedure: Patient is placed in routine position for tracheostomy. One cm stab incision is made through cricothyroid membrane. The cannula is passed over introducer into the tracheal and then introducer is removed.

Advantages of minitracheostomy:
- Allows a constant tracheal access for suction
- Natural mechanisms are retained
- Function of glottis is preserved
- Cough reflex to catheter is preserved of supplements internal clearing mechanisms
- Early use prevents onset of respiratory failure
- Used for humidified air /O_2 mixture or nebulized drugs
- Used for management of airway obstruction and faciomaxillary surgery
- Used for high frequency jet ventilation
- Simple to perform
- Quick healing and minimum scaring
- Retention of voice and ability to communicate.

The use of cricothyrotomy as an alternative to tracheostomy for patients who require prolonged intubation has been associated with a higher incidence of vocal cord dysfunction and subglottic stenosis. When cricothyrotomy is used in the setting of establishing an emergency airway, conversion to a standard tracheostomy should be considered, if decannulation is not anticipated within 5–7 days.

Chapter

13

TOF with Shunt

Q. What is the classification of congenital heart disease?

Congenital cardiac defects are classified into:
- Acyanotic defects
- Cyanotic defects.

Acyanotic defects: The acyanotic congenital heart diseases are characterized by a left-to-right intracardiac shunt and include the following defects:
- Ventricular septal defect
- Atrial septal defect
- Patent ductus arteriosus
- Pulmonary stenosis
- Aortic stenosis
- Coarctation of aortas.

Cyanotic defects: The cyanotic congenital heart diseases are characterized by a right-to-left intracardiac shunt with associated decreases in pulmonary blood flow and development of arterial hypoxemia. The defects included are:
- Tetralogy of Fallot
- Eisenmenger syndrome
- Ebstein's anomaly (malformation of tricuspid vulve)
- Tricuspid stenosis
- Transposition of great vessels.

Q. What are the signs and symptoms of congenital heart disease?

The signs and symptoms of congenital heart disease varies in infants and children. In infants, the signs and symptoms include:
- Tachypnea
- Failure to gain weight
- Heart rate > 100 beats/min
- Heart murmur
- Congestive heart failure
- Cyanosis.

In children, the signs and symptoms include:
- Dyspnea
- Slow physical development

- Decreased exercise tolerance
- Heart murmur
- Congestive heart failure
- Cyanosis
- Clubbing of digits
- Squatting
- Hypertension.

Q. How is the diagnosis of congenital heart disease made?

The diagnosis of congenital heart disease is apparent during the first week of life in about 50% of affected neonates and generally 90% before 5 years of age.

Following imaging modalities are helpful in diagnosing congenital heart disease:

- Echocardiography is the initial diagnostic step if congenital heart disease is suspected. Transthoracic and transesophageal echocardiography have facilitated early and accurate diagnosis of congenital heart disease.
- Fetal cardiac ultrasonography has permitted early diagnosis of congenital heart defects, allowing subsequent perinatal management in specialized tertiary care centers.
- Cardiac magnetic resonance imaging.
- Three dimensional echocardiography have increased the understanding of complex cardiac malformations, and allowed visualization of blood flow and vascular structures.
- Cardiac catheterization and angiography are most definitive diagnostic procedures available for use in patients with congenital heart disease.

Q. What is cardiac catheterization? What are the utilities of this procedure?

Cardiac catheterization is one of the special procedures used for the investigation of cardiovascular disease. In this, a catheter is inserted into a vein or artery or is advanced into the heart under X-ray guidance. This allows the operator to measure intracardiac pressures, take blood samples from individual cardiac chambers and to obtain angiogram by injecting contrast media into a chamber or blood vessel.

Left heart catheterization is mainly used to assess coronary artery disease, but is also used to evaluate the disease of the mitral valve, aortic valve, aorta. Left ventriculography is used to determine the size and function of the left ventricle, coronary angiography is used to detect stenosis to guide revascularization procedures such as balloon angioplasty or stenting.

Right heart catheterization is used to assess pulmonary artery pressure and can also be used to detect intracardiac shunts by measuring oxygen saturation in the different chambers. A step-up in oxygen saturation from 65% in the right atrium to 80% in the pulmonary artery is indicative of a large left-to-right shunt that might be due to a ventricular septal defect. Cardiac output can be measured using thermodilution techniques. Left atrial pressure can be measured directly by puncturing the interatrial septum from right atrium,

with a special catheter cardiac catheterization permits measurement of transvalvular pressure gradient across bioprosthetic valve and the effective orifice area allowing assessment of prosthetic value function.

Placing a catheter in right atrium is helpful for guiding rate of fluid infusion. This catheter may be important for aspirating air from the chamber of the heart, should a venous air embolism occur. The junction of superior vena cava with right atrium is the ideal location for catheter tip, because this position provides most rapid aspiration of air.

Q. What are the common problems associated with congenital heart disease?

Certain complications which are likely to accompany the presence of congenital heart disease are:

- Infective endocarditis
- Cardiac arrhythmias
- Complete heart block
- Hypertension (systemic or pulmonary)
- Erythrocytosis
- Thromboembolism
- Coagulopathy
- Brain abscess
- Sudden death.

Q. How is a pediatric cardiac surgery patient different from an adult cardiac surgery patient?

- The cardiovascular system of infant has reduced myocardial reserve due to: Limited or restricted number of beta-receptors, high resting level of circulating catecholamine, reduced ventricular compliance.
  The stroke volume does not increase with preload augmentation. They are more dependent or heart role to increase the cardiac output.
- Pediatric patients are physically smaller than adults. Invasive monitoring establishment is challenging in these small patients. One may have to rely on transthoracic echo during surgery, as well as, specialized monitoring such as transesophageal or epicardial echocardiography with Doppler color flow imaging.
- During cardiopulmonary bypass (CPB), these patients are cooled to 15°–18°C; hemodilution occurs to more than 50% of this extracellular volume. They may undergo periods (>1 hour) of deep hypothermic cardiac arrest. They have exaggerated inflammatory response to CPB due to the disproportionate exposure to non-endothelialized surface of the CPB circuit per body surface area as compared to adults.

Q. What is tetralogy of Fallot?

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart defect, characterized by:

- A large single VSD
- An aorta that overrides the right and left ventricles
- Obstruction to the right ventricular outflow tract (RVOTO) (Subvalvular, valvular and supravalvular). The RVOTO may be infundibular spasm, pulmonary stenosis or pulmonary atresia
- Right ventricular hypertrophy.

Several anomalies may occur in association with tetralogy of Fallot, including right aortic arch, ASD (pentalogy of Fallot) and coronary arterial anomalies. Right ventricular hypotrophy occurs as the VSD permits continuous exposure of the right ventricle to the high pressures present in the left ventricle.

Right to left intracardiac shunting occurs because of increased resistance to flow in the right ventricular outflow tract, the severity of which, determines the magnitude of the shunt. Because the resistance to flow across the right ventricular outflow tract is relatively fixed, changes in systemic vascular resistance (drug-induced) may affect the magnitude of the shunt. Decreases in systemic vascular resistance increases right-to-left intracardiac shunting and systemic vascular resistance (squatting) decreases right-to-left intracardiac shunting with resultant increase in pulmonary blood flow.

TOF with pulmonary atresia is much more severe form of congenital heart disease and cyanosis develops almost immediately after birth and worsen progressively as the ductus arteriosus closes. These patients depend on the ductus arteriosus until definitive corrective surgery is done. Prostaglandins are added to maintain the patency of ductus arteriosus.

Q. What are the presenting features of tetralogy of Fallot?

- Most patients with TOF have cyanosis from birth or beginning during the first year of life. The most common auscultatory finding is an ejection systolic murmur, heard along the left sternal border, resulting from blood flow across the stenotic pulmonary value.
- Congestive heart failure rarely develops because the large VSD permits equilibration of intraventricular pressure and cardiac workload.
- Chest radiography shows evidence of decreased lung vascularity and the heart is 'boot-shaped'.
- The ECG is characterized by changes of right axis deviation of right ventricular hypertrophy.
- Arterial oxygen desaturation is present even when breathing 100% oxygen (PaO_2 is usually <50 mm). Compensatory erythropoiesis is proportional to the magnitude of the arterial hypoxemia.
- $PaCO_2$ and arterial pH are usually normal.
- Squatting is a common feature of the children with TOF. Squatting increases the systemic vascular resistance by kinking the large arteries in the inguinal area. The resulting increase in systemic vascular resistance tends to decrease the magnitude of the right-to-left intracardiac shunt, which leads to increased pulmonary blood flow and subsequent improvement in arterial oxygenation.

- Hypercyanotic attacks: Sudden spells arterial of hypoxemia with worsening cyanosis, tachypnea and sometimes apnea, loss of consciousness, seizures, cerebrovascular accidents and even death. These attacks may occur without provocation but are often associated with feeding or attacks, most likely caused by a sudden decrease in pulmonary blood flow due to spasm of the infundibular cardiac muscle or decreased systemic vascular resistance. The subvalvular component of the RVOTO is dynamic and may increase suddenly under adrenergic stimulation. These attacks are called Fallot's spell or Tet spell.
- Treatment of hypercyanotic attacks depend on the cause of RVOTO. When symptoms reflect dynamic infundibular obstruction (spasm), the appropriate treatment is administration of beta-adrenergic antagonists like esmolol or propranolol. Chronic oral propranolol is indicated in children having recurrent hypercyanotic attacks due to spasm of outflow tract muscle. If cause is decreased SVR, treatment is IV fluid administration and/or phenylephrine.
- In older children, Fallot's spell are uncommon but cyanosis become increasingly apparent with stunting of growth, digital clubbing of nail bed, polycythemia. Cyanosis may be absent in the newborn or in children with only mild right ventricular outflow tract obstruction (acyanotic tetralogy of Fallot). In these cases, blood flow to the pulmonary circulation is sufficient to maintain the deoxygenated hemoglobin concentration less than 5 g% and so these children are acyanotic. This is also known as 'pink tet' or 'pink TOF'. These patients are asymptomatic or may show signs of congestive heart failure from large left-to-right shunt. Medical management includes diuretics (frusemide), after load reduction with angiotensin, converting enzyme inhibiters (captopril).
- Adults with TOF manifest dyspnea and limited exercise tolerance. They may also have complications of chronic cyanosis including erythrocytosis, hyperviscosity, abnormalities of hemostasis, cerebral abscess, stroke and infective endocarditis.
- Cerebral vascular accident (CVA): Common in children with severe TOF due to cerebrovascular thrombosis and severe arterial hypoxemia. Dehydration and polycythemia may contribute to thrombosis. Hemoglobin concentrations exceeding 20 g% are common in these children.
- Cerebral abscess: Abrupt onset of headache, fever of lethargy followed by persistent emesis, appearance of seizure activity.
- Infective endocarditis: Constant danger with patient with TOF, associated with high mortality. Antibiotics should be administered before dental or surgical procedures in these patients.

Q. How is the diagnosis of TOF made?

- Most children with TOF have cyanosis from birth or beginning during the first year of life.
- The patient usually gives the history of the hypercyanotic attacks in the child.

- Most common auscultatory finding is an ejection systolic murmur in the pulmonary area.
- Chest radiographs show evidence of decreased lung vascularity and boot-shaped cardiac shadow.
- ECG reveals right ventricular hypertrophy.
- Echocardiography is used to assess the level severity of RVOTO, the size of main pulmonary artery of the location of VSD.
- Color Doppler imaging is used to visualize the right-to-left shunting through the VSD.
- Cardiac catheterization further confirms the diagnosis along with confirmation of the anatomic and hemodynamic data.
- MRI can also provide much of this information.

Q. What is the treatment of TOF?

The definitive treatment of TOF is complete surgical correction of the defect (closure of VSD with a Dacron patch, relief of RVOTO by placing a synthetic graft) when the patients are extremely young.

A major complication of complete surgical repair is difficulty in achieving surgical hemostasis. Platelet dysfunction, hypofibrinogenemia are common in these children which may contribute to postoperative bleeding problems.

In earlier days, corrective surgery of TOF was associated with high morbidity and mortality rate. In recent days with the development of deep hypothermic cardiac arrest, low flow hypothermic CPB, advances in anesthetic management of infants and children. Of their postoperative care, the mortality rate has decreased dramatically after early one stage corrective surgery.

The ideal timing for corrective surgery in asymptomatic children is between 4–6 months of age. Some centers prefer to operate in early neonatal periods and others wait until 1 year of age symptomatic children or those refractory to medical management, require urgent surgery irrespective of age.

One stage repair is preferred as it avoids second surgery and eliminates complications of shunt operation. These patients depend on the patent ductus arteriosus, to increase pulmonary blood flow, until corrective surgery is done, therefore prostaglandins are added to maintain the patency of the ductus arteriosus.

Q. What are the indications of aim of palliative surgery in TOF? What are the varieties of shut done?

In the past, palliative procedures were done in the children with TOF in whom risk of complete repair was considered greater than the cumulative risk of two stage repair.

All palliative procedures involve anastomosis of a systemic artery to a pulmonary artery in an effort to increase pulmonary blood flow of improve arterial oxygenation. Shunt surgery also promotes pulmonary vascular growth but predisposes the child to right ventricular failure, secondary to increased pulmonary blood-flow.

Three palliative procedures were in common use:

1. Waterston procedure: Side-to-side anastomosis of the ascending aorta of the right pulmonary artery.
2. Potts procedure: Side-to-side anastomosis of the descending aorta to the left pulmonary artery.
   Both the Waterston and Pott's shunts resulted in excessive pulmonary blood low, distortion of pulmonary artery of problems during subsequent complete surgical repair of TOF. Therefore, they are no longer used.
3. Blalock-Taussig (BT) procedure: Direct end-to-end anastomosis of subclavian artery to pulmonary artery. Professor Mare modified the original BT shunt using an interposition conduit between subclavian artery and pulmonary artery, and is known as modified BT shunt, which is most commonly used systemic to pulmonary artery shunt.

Q. What is the anesthetic considerations in a child with TOF put for modified BT shunt surgery?

Premedication: The goal is to achieve adequate sedation maintaining respiratory and hemodynamic stability allowing separation from parents. Oral premedication is preferred. Syrup midazolam 0.5 mg/kg given orally 20 minutes before anesthesia.

All syringes and intravenous tubing must be bubble-free to avoid paradoxical air embolism.

All resuscitative drugs must be ready and labeled. Monitors should be placed after induction to avoid crying as it leads to precipitation of Fallot's spell. Monitoring includes noninvasive blood pressure (NIBP), pulse oximetry, ECG, temperature (esophageal stethoscope), urine output, arterial and central venous catheter (Arterial line should not be placed on the side of shunt surgery), capnometry. End tidal capnometry is useful in identifying a tet spell intraoperatively. As the pulmonary blood flow decreases, so does the end tidal CO_2, this is followed by arterial desaturation.

The anesthetic concerns are to maintain SVR, reduce pulmonary vascular resistance (PVR) and prevent hypercyanotic attacks intraoperatively. Endocarditis prophylaxis is given. Ketamine is preferred as induction agent as it increases the SVR.

Both hypercapnea and acidosis are avoided as they increase PVR. Positive pressure ventilation and positive end-expiratory pressure (PEEP) do not cause clinically significant increase in PVR in children with severe pulmonary stenosis.

Nitrous oxide and halothane can be used. Halothane relieves infundibular spasm during hypercyanotic spell and is preferred.

Opioids are used carefully to maintain hemodynamic stability. Euvolemia is maintained to prevent dynamic RVOTO. As soon as the shunt is open, arterial saturation improves dramatically and diastolic BP reduces drastically which may precipitate MI. Heparin infusion and aspirin are started to maintain shunt patency.

Postoperatively, the child is ventilated mechanically for some time and opioids may be need for analgesia. The shunt flow assessment is done as follows:

- If oxygen saturation is around 80% then pulmonary blood is good
- If oxygen saturation is >80%, it implies excessive pulmonary blood flow and congestion, which can lead to unilateral pulmonary edema on the side of the shunt
- If oxygen saturation is <80%, it implies poor pulmonary blood flow.

Q. What is the anesthetic management of a child undergoing corrective surgery for TOF?

The anesthetic considerations are understanding of the events after administration of drugs that alter the magnitude of the right-to-left intracardiac shunt. When shunt is acutely increased, there occurs a decreased pulmonary blood flow and decreased arterial oxygenation. The magnitude of right-to-left shut increases by: (1) decreased SVR; (2) Increased PVR; (3) Increased myocardial contractility which accentuates infundibular obstruction to ejection of blood by right ventricle. Volatile anesthetics also decreases SVR.

Preoperative preparation: Oral feeding is maintained to avoid dehydration.

Oral premedication is preferred to avoid crying from needle prick, which precipitates hypercyanotic attacks.

β-adrenergic antagonists should be continued until induction of anesthesia in children using these drugs for prophylaxis against tet spell.

Induction is done with IV ketamine (1–2 mg/kg) as if results increased SVR, increased pulmonary blood flow improved arterial oxygenation onset of action of drugs administered by IV route may be more rapid due to right-to-left shunt, as dilution effect in the lungs is decreased.

Induction with volatile anesthetic, sevoflurane or halothane can be done with caution and careful monitoring of $SPO_{2.}$ Decreased pulmonary blood flow speeds achievement of anesthetic concentration but the hazards of decreased SBP and decreased SVR are great.

Maintenance of anesthesia is achieved with 5% N_2O oxygen plus ketamine.

Opioids or benzodiazepines may be used during maintenance with adjustment of dose and rate to minimize decrease in SVR.

Pancuronium is preferred to provide muscle relaxation as it maintains SBP and SVA. The increased heart rate produced is helpful for maintaining left ventricular cardiac output.

Ventilation is controlled with optimum airway pressure to avoid increased resistance to pulmonary blood flow.

IV fluid administration is done to maintain fluid volume status as hypovolemia increases right-to-left shunt. As there is associated erythrocytosis, blood transfusion is not needed until about 20% of patient's blood volume is lost. Meticulous care is taken to avoid transfusion of air through tubing used for IV fluid administration, which will lead to systemic air embolism.

Phenylephrine must be available to trat decreased SBP due to decreased SVR.

The child posted for corrective surgery of TOF requires cardiopulmonary bypass (CPB) with total body heparinization. Following complete repair, the arterial saturation improves to 100%. The effect of heparin is reversed with protamine as the child comes of CPB. Inotropes may be required intraoperatively and in the postoperative period. Postoperatively, the child is mechanically ventilated for some period.

Q. What is Eisenmenger syndrome?

When a left-to-right intracardiac shunt is reversed, as a result of increased PVR, to the level that equals or exceeds the SVR is known as Eisenmenger syndrome. Exposure of the pulmonary vasculature to increased blood flow pressure as in VSD and ASD, results in pulmonary hypertension. When PVR equals or exceeds, SVR and intracardiac shunt is reversed. The murmur associated with these cardiac defects disappears when Eisenmenger syndrome develops.

Q. What is the anesthetic consideration in a child with TOF for noncardiac surgery?

Understanding the pathophysiology of the lesion, its hemodynamics effect of anesthetic drugs, ability of the anesthesiologist to tailor anesthetics for the child.

- Evaluation of the child's heart and lungs:
  - Cardiac function: Child's activity, sweating when nursing, slow to finish bottle
  - Rule out congestive heart failure (CHF), puffy eyes, enlarged liver (lungs may sound clear)
  - Effect of cyanotic spells.
- Appropriate tailoring of anesthetics:
  - Oral premedication sedation: Midazolam with ketamine is a good option
  - Promoting left-to-right shunt or reducing right-to-left shunt
  - Reducing pulmonary vascular resistance, maintaining SVR and blood pressure
  - Avoidance of hypoxia, hypercarbia, acidosis, hyperthermia, pain, inadequate depth of anesthesia
  - Maintaining proper hydration if the child is polycythemic
  - IV induction in presence of right-to-left shunt may be faster with inhalation anesthetic
  - Avoidance of entry of air bubbles from syringes and tubing for infusion.
- If palliative shunt is in place:
  - Consideration of appropriate site for monitoring NIBP and IBP, etc.
  - Antibiotic prophylaxis
  - Maintaining SVR to promote pulmonary blood flow.

- Postoperative pain management:
  - Appropriate analgesia reduces stress on the child hence reduces hypercyanotic spells
  - Regional blocks may be considered for analgesia
  - Paracetamol or NSAIDS may be used, if suitable.

Chapter

14

Inguinal Hernia

Q. What is hernia? What are its common types and the clinical classification?

A hernia occurs when an internal part of the body pushes through a weakness in the muscle or surrounding tissue.

The common types are:
1. Inguinal hernia
2. Femoral hernia
3. Incisional hernia
4. Umbilical hernia
5. Hiatus hernia
6. Epigastric hernia.

The common areas of hernia are:
1. Groin—inguinal hernia
2. Belly button—umbilical hernia
3. Sites of previous operations—incisional hernia.

The abdominal wall has natural areas of potential weakness. Hernia can develop at these due to heavy strain on abdominal wall, aging, injury, an old incision or a weakness present from birth. It can occur at any age. Most hernias in children are congenital. A bulge is noticed under the skin. Pain is felt during coughing, straining, prolonged standing and lifting heavy weights. Pain may be sharp or dull aching, that gets worse at the end of the day. Bulge disappears on lying down. Severe continuous pain, redness and tenderness are signs of strangulation and need immediate medical consultation.

Clinical classification:

- *Reducible hernia:* It can be pushed back into the abdomen by manual pressure.
- *Irreducible hernia:* This cannot be pushed back into the abdomen. It is further classified as:
  - ***Obstructed hernia:*** The lumen of the hernia is obstructed but the blood supply to the gut is intact causing intestinal obstruction
  - ***Incarcerated hernia:*** Adhesion developed between the wall of the hernia sac and the intestine resulting in irreducible hernia

– ***Strangulated hernia:*** With the progress of hernia, intestine can descend into hernia sac and may become obstructed causing intestinal obstruction. If blood supply of the obstructed intestine is compromised, the hernia is termed as strangulated hernia causing ischemia and gangrene. Emergency surgery is needed.

Q. What is inguinal hernia? What are the types of inguinal hernia?

When the inside layer of abdominal muscles have weakened, the inner lining of the abdomen pushes through the weakened area of abdominal wall to form a small bulge which can allow a loop of intestine or abdominal tissue to push into the sac, is known as inguinal hernia. An inguinal hernia is the most common type of hernia. It appears as a swelling in the groin or as an enlarged scrotum. The lump appears on straining, coughing or prolonged standing and disappears on lying down. Inguinal hernia may develop at birth or over time, does not get relieved by itself.

There are 2 types of inguinal hernia—indirect and direct, which are defined by their relationship to the inferior epigastric vessels.

Direct inguinal hernia occurs medial to the inferior epigastric vessels, when abdominal contents herniate through a weak spot in the fascia of the posterior wall of inguinal canal, is formed by the transversalis fascia (Hesselbach triangle). This triangle is within the myofascial orifice that refers to an anatomical area in the groin through which all inguinal hernia occurs. The inguinal ligament forms the base of the triangle, edge of the rectus abdominis is the medial border and inferior epigastric vessels are the superolateral border. Direct inguinal hernia is less common, occurring commonly in men over 40 years of age. The two major factors for the development of direct inguinal hernia are:

1. Increased intra-abdominal pressure.
2. Weakness of posterior inguinal wall.

Indirect inguinal hernia: It is the most common inguinal hernia and occurs when abdominal contents protrude through the deep inguinal ring, lateral to the inferior epigastric vessels. This is caused by the failure of embryonic closure of the processus vaginalis after the passage of testicles through deep inguinal ring.

In men, indirect inguinal hernia follows the same route as the descending testis, which migrates from abdomen into scrotum during the development of urinary and reproductive organs. The larger size of the inguinal canal, which transmits testicles and accommodates the structures of spermatic cord, is one reason why men are more likely to have indirect inguinal hernia than women. Increased intra-abdominal pressure further stretches and weakens the internal ring allowing additional organ to herniate through the orifice.

Indirect inguinal hernia can appear before 1 year of age and often before 30 years, though usually in later life. Premature infants have higher chance of developing indirect inguinal hernia.

Sliding inguinal hernia: It is defined as any inguinal hernia in which part of the hernia sac is the wall of a viscus. It is more common after the age of 70 years. If the hernia is on the right side, the cecum, ascending colon or appendix more commonly are involved. If on left side, the sigmoid colon is involved. The uterus, fallopian tubes, ovaries, ureter and bladder can be involved on either side. The sliding components usually are found on the posterolateral side of the internal ring. The importance of this condition has lessened with the realization that it is not necessary to resect hernia sacs and that simple reduction into the preperitoneal space is sufficient. This eliminates the primary danger of injury to the viscus during high ligation and sac excision.

Q. What is inguinal canal? What are the structures of spermatic cord?

The inguinal canal is a passage through the lower abdominal wall. In males, the spermatic cords pass through the inguinal canal and connect to the testicle in the scrotum. In females, round ligaments which support the uterus, pass through the inguinal canal. The spermatic cord contains blood vessels, nerves and spermatic ducts that carry sperm from the testicle to the penis.

Q. What are the signs and symptoms of inguinal hernia? How inguinal hernia is diagnosed?

Signs and symptoms: The signs and symptoms of inguinal hernia include (Indirect hernia produce more symptoms):

- First sign is a small bulge on one or rarely on both sides of the groin. The bulge gradually increases in size over time. It disappears when lying down
- Discomfort or pain in the groin, especially on bending over, coughing, straining or weight lifting which improves on resting
- Heaviness or dragging sensation in the groin
- A burning, gurgling or aching sensation in the bulge
- Pain or swelling around the testicle, when the protruding intestine descends into the scrotum.

The bulge can be seen or felt. It is usually more obvious when patient stands up and especially coughs or strains. Indirect and direct inguinal hernias may slide in and out of the abdomen. Patient is, therefore, able to gently and easily push the hernia back into abdominal cavity when lying down. If not, applying icepack to the area of the bulge, may reduce the swelling enough and the hernia slides in easily. Lying with the pelvis higher than the head may also help.

Diagnosis of inguinal hernia:

- Symptoms described by the patient suggest that of inguinal hernia
- Physical examination is the best way to determine the presence or absence of inguinal hernia. During physical examination, the patient is asked to stand and cough or strain to feel the bulge caused by hernia as it moves into groin or scrotum. Gentle massage is done to push the hernia back into

the abdomen. While straining, an indirect inguinal hernia pushes against the fingertip, and direct inguinal hernia pushes against the pulp of the finger. Pressure applied over mid-inguinal point prevents protrusion of indirect inguinal hernia, when patient stands but direct inguinal hernia is not affected.

Imaging tests are done when:

- Inguinal hernia cannot be felt during examination, especially in obese patients
- An incarcerated or strangulated hernia is to be diagnosed
- Differentiating from other conditions causing groin swelling.

Test includes:

- Abdominal X-ray: During X-ray, patient lies on a table then stands
  - Hernias are visualized as abnormal ballooning of the anteroposterior diameter of inguinal canal and/or simultaneous protrusion of fat or bowel within the inguinal canal
  - Dilated loops of bowels with air fluid levels. Absence of bowel gas distal to obstruction and bowel shadow in the region of the hernia, more clearly demonstrated in lateral view.
- CT scan
- Abdominal ultrasound
- MRI

Q. What is the differential diagnosis of inguinal hernia? What are the complications of inguinal hernia?

Differential diagnosis: The differential diagnosis includes femoral hernia, testicular torsion, undescended testis, hydrocele, groin abscess and inguinal adenopathy. Complications that can develop in inguinal hernia include incarceration, bowel obstruction and strangulation.

- *Incarceration*: It is the irreducible hernia, occurs when part of omentum or small intestine from inside the abdominal cavity becomes stuck in the groin or scrotum and cannot be massaged back into abdomen. It is not a surgical emergency as chronic states of incarcerations are common because of the size of the neck of hernia in relation to its content or because of adhesions to the hernia sac.

  Treatment: Surgical repair but no emergency as it is not a life-threatening complication. Incarcerated inguinal hernia may exhibit signs of bowel obstruction or may develop as an acute incarceration associated with tenderness. Patient may have vomiting.
- *Bowel obstruction*: An incarcerated hernia can lead to bowel obstruction, when a section of the bowel becomes stuck in the inguinal canal causing nausea, vomiting, pain abdomen and painful lump in the groin. Emergency surgery is always needed.
- *Strangulation*: It occurs when blood supply of the loop of intestine is cutoff in an obstructed inguinal hernia, causing the section of the intestine to die, creating an emergency situation. Emergency surgery is needed within

hours to release trapped tissue and restore blood supply to the gut. It is a serious life-threatening condition as the hernia contents become ischemic and nonviable.

Symptoms:

- Extreme tenderness or painful redness in the area of the bulge in the groin.
- Sudden pain that worsen quickly and doesnot go away
- Absence of bowel movement and passage of gas
- Nausea and vomiting
- Fever and tachycardia
- Patient is toxic and dehydrated
- Leukocytosis
- Arterial blood gas (ABG) shows metabolic acidosis.

Treatment: Rapid resuscitation with IV fluids, nasogastric (NG) suction and IV antibiotics are essential.

- Urgent surgery needed after resuscitation
- If bowel is viable, it is reduced into the abdomen prior to repairing the hernia
- If bowel is found to be gangrenous, more bowel is pulled into hernia so that viable bowel can be transected and gangrenous portion removed. An end-to-end anastomosis is done and the bowel is reduced into the abdominal cavity followed by hernia repair.

Q. What is the management of inguinal hernia?

There is no medical recommendation for conservative management of inguinal hernia as recently elective surgery is the recommended treatment. Use of hernia truss is rarely prescribed as it is usually ineffective. The hernia truss is intended to contain a reducible inguinal hernia within the abdominal cavity. Most modern variety of truss is made with nonintrusive flat pads and holds the hernia secure during all activities. Such devices cannot prevent inguinal hernia from progressing but provide greater confidence and comfort during physically demanding task. Truss increases complications like strangulation of hernia, atrophy of spermatic cord and fascial margins and the defect enlarges making subsequent repair of hernia difficult.

In incarcerated hernia, the initial treatment is taxis, in absence of signs of strangulation. Taxis is performed with patient sedated and placed in Trendelenburg position. The neck of the hernia sac is grasped with one hand and pressure is applied on the most distal part of hernia by other hand. The goal is to elongate the neck of hernia, so that the contents may be guided back into abdominal cavity using a rocking movement. Not more than one or two gentle attempts should be made.

Reduction en-masse of hernia is defined as reduction of hernia with peritoneal sac and constricting neck into the abdomen without relief of incarceration.

Nonoperative treatment like watchful waiting is only applicable in asymptomatic or minimally symptomatic hernia.

Surgical repair of all inguinal hernia at diagnosis is the standard recommendation. Due to improvement in surgical techniques and use of prosthetics, there is decreased rate of recurrence after herniorrhaphy. The mesh herniorrhaphy has decreased the recurrence rate when compared to nonmesh repair. The incidence of chronic post herniorrhaphy groin pain is also less with a prosthetic repair. The material for mesh suitable for use in hernia surgery includes: (1) Polypropylene, either monofilament or polyfilament; (2) Polytetrafluoroethylene; (3) Dacron.

Surgery is needed: (1) Soon after diagnosis of hernia; (2) When hernia causes severe or persistent symptoms; (3) When complications develop.

Surgical repair pushes the bulge back into the abdominal cavity and strengthen the weakness in the abdominal wall.

Surgical repair of inguinal hernia is done on an outpatient basis. Recovery time varies with the size of hernia, surgical technique used, age and health of the patient.

Surgical repair (herniorrhaphy) is the only treatment of inguinal hernia and can prevent incarceration or strangulation. Patients with inguinal hernia causing few or no symptoms may delay surgery but should watch for symptoms. Surgery is usually recommended for infants or children to prevent incarceration or strangulation. Emergency surgery is needed for strangulated hernia.

Surgical techniques include: (1) Open repair with or without mesh; (2) Laparoscopic hernia repair.

Open repair with or without mesh: Open approach is done from outside through a 3–4 inch incision in the groin or the area of the hernia. The incision extends through skin, subcutaneous fat allowing the surgeon to get through the level of defect. Surgeon may choose a small piece of surgical mesh to repair the defect or hole. This is usually done under LA and sedation but may be done under spinal or GA.

Steps of open repair of inguinal hernia:

1. *Skin incision:* An oblique incision between ASIS and pubic tubercle.
2. *Mobilization of cord structure:* After dissection of external oblique aponeurosis off the internal oblique muscle and isolation of iliohypogastric nerve, the cord structures are lifted at the pubic tubercle.
3. *Division of cremasteric muscle:* To facilitate sac identification when dealing with indirect inguinal hernia and to lengthen the cord for better visualization of inguinal floor.
4. *Management of inguinal scrotal hernia sac:* A better approach is to divide indirect inguinal hernia sac in the mid-inguinal canal, to avoid trauma to the testicular blood vessels.
5. *Releasing incision:* It divides the anterior rectus sheath, extending from pubic tubercle superiorly for a variable distance. This allows various components of abdominal wall to displace laterally and inferiorly.
6. *Wound closure:* External oblique fascia is closed, reconstructing the superficial ring loosely to prevent strangulation of cord structures. Scarpa fascia and skin are closed to complete the surgery.

Conventional anterior prosthetic herniorrhaphy: For indirect hernia, the cremasteric muscle is incised longitudinally and the sac is dissected, freed and reduced into the preperitoneal space.

Direct hernias are separated from the cord and other surrounding structures and then reduced back into the preperitoneal space. Mesh prosthesis with a minimum size of 15 × 8 cm for adult is positioned over inguinal floor.

Laparoscopic inguinal herniorrhaphy: A laparoscope connected to a special camera is inserted through a cannula allowing the surgeon to view the hernia and surrounding tissue on a video screen. The hernia is repaired from behind the abdominal wall. Surgical mesh is placed over the hernia defect and held in place with small surgical staplers usually done under GA, because of pneumoperitoneum.

Factors increasing the possibility of choosing or converting to open procedure include: Obesity, prior abdominal surgery causing adhesion and bleeding during operation. The decision to convert to open procedure is strictly based on patient's safety.

Indications:
- Recurrent inguinal hernia
- Bilateral inguinal hernia using the same small laparoscopic incision
- Inguinal hernia in a patient requiring laparoscopy for another procedure.

Contraindications:
- Signs of intra-abdominal infection
- Intra-abdominal adhesions
- Severe underlying medical illness
- Incarcerated sliding scrotal hernia
- Coagulopathy.

Advantages:
- Less persisting pain or numbness
- Quick return to normal activity
- Less invasive
- Several small cuts are made to introduce laparoscope.

Q. What are the preoperative preparations? What are the postoperative management and complications of inguinal herniorrhaphy?

Preoperative preparations:
- History, physical exam and investigations like full blood count, chest X-ray, ECG and medical evaluation depending on age
- Written informed consent from patients or parents after explaining the risks and benefits
- Laxative is given night before surgery for bowel clearance
- Fasting for solid food from midnight
- Medicines can be taken with sips of water and clear fluids are permissible up to 2 hours before surgery

- Drugs like aspirin, anti-inflammatory medication should be stopped for a week before surgery
- Smoking should be stopped 6 weeks before surgery
- Most of the inguinal hernia repair is done on OPD basis.

Postoperative management:
- Patient transfer to postanesthesia care unit (PACU) for 1–2 hours until patient is fully awake
- Supplemental oxygen and adequate analgesia
- Patient is discharged as early as possible
- Patient is encouraged to intubate from the day after surgery
- Promoting an easy bowel motion
- Scheduled follow-up done within 2 weeks after surgery.

Complications:
- Risk from surgery:
  - Bleeding and infection as any other surgery
  - Slight risk of injury to bladder, intestine, blood vessels, nerves or spermatic cord. Difficulty in urination requires catheterization.
- Early complications:
  - Blood and fluid bulging up the space left by hernia
  - Painful swelling and bruise of the testicle or base of the penis
  - Pain or numbness in the groin due to nerve damage
  - Recurrence of hernia.

  Complications are more in patients above 50 years of age, having heart or lung disease.
- Late complications:
  - Chronic groin pain
  - Ischemic orchitis
  - Testicular atrophy.

Q. What anesthesia is used for groin herniorrhaphy?

Open surgical repair can be done using:
- Local anesthesia (LA) of abdominal wall with sedation
- Spinal block with sedation
- General anesthesia (GA): Laparoscopic repair is usually done under GA. Open repair in patient with severe comorbidities are also done under GA.

Most inguinal herniorrhaphies can be done under LA or regional anesthesia. Laparoscopy needs GA due to pneumoperitoneum.

Use of 100 mL of 0.5% xylocaine with adrenaline or 0.25% bupivacaine with epinephrine or a combination of the two is most common. A 70 mL of solution is injected by surgeon in an adult patient prior to preparation of draping of the patient. A 10 mL is placed medial to the ASIS to block the ilioinguinal nerve. The other 60 mL is used as a field block along the orientation of eventual incision in the subcutaneous and deeper tissues. The remaining 30 mL is reserved for the discretionary use during the procedure. With this technique, endotracheal intubation is avoided and patient can be aroused from sedation at intervals to perform valsalva maneuver and test the adequacy of repair.

Q. Describe the anatomy of groin?

Anatomy from skin to abdominal cavity is as follows:

- Skin
- The Camper and Scarpa fascia in subcutaneous tissue beneath the skin
- Next to fascia is the aponeurosis of the external oblique muscle. The muscle arises from the posterior aspect of lower 8 ribs
  - Posterior portion of the muscle, oriented vertically and inserts on the crest of the ilium
  - The anterior portion courses inferiorly in an oblique direction towards the midline and pubis
  - The obliquely oriented anterior inferior fibers of the external oblique aponeurosis fold back on themselves to form inguinal ligament. Laterally, the inguinal ligament attaches on the ASIS. Medially the insertion of the ligament is dual
    - One portion insert to pubic tubercle and pubic bone
    - Second one folds back as lacunar ligament
  - The more medial fibers of external oblique muscle divides into medial and lateral creases to form the external superficial inguinal ring, through which spermatic cord or round ligament and branches of ilioinguinal and genitofemoral nerves pass.
- The internal oblique muscle fibers fan out, following the shape of iliac crest.
  - The superior fibers course obliquely upwards to be inserted into the distal ends of 3–4 ribs
  - The lower fibers run inferomedially towards the pubis, parallel to the external oblique aponeurotic fibers and arch over the spermatic cord or round ligament, forming superficial part of internal (deep) inguinal ring.
- The transversus abdominis muscle arises from the inguinal ligament, inner side of iliac crest, endoabdominal fascia and lower six costal cartilages and ribs. The medial aponeurotic fibers contribute to the rectus sheath and is inserted on the pubis and pubic crest forming falx inguinalis.

Nerve supplies of groin are ilioinguinal and iliohypogastric nerves and divisions of L1 nerve.

Q. What is femoral hernia? How is it presented? Why femoral hernia carries more risk?

Groin hernias also include femoral hernia. A femoral hernia is not a protrusion of abdominal cavity contents via inguinal canal but via femoral canal, which normally allows passage of femoral vessels from pelvis to leg. Femoral ring is bounded by superior pubic ramus inferiorly and femoral vein laterally. The illiopubic tract with curved insertion on to the pubic ramus is the anteromedial border. The canal normally contents preperitoneal fat, connective tissue and lymph nodes.

The femoral presents as a swelling below the inguinal ligament, just to the pubic tubercle and medial to the femoral vessels usually asymptomatic and disappears when the patient lies down.

The factors which contribute to the development of femoral hernia are:

Size and shape of the femoral ring: Increased intra-abdominal pressure. The femoral hernias are particularly dangerous because the rigid structures that make up the femoral ring, slightest edema at the ring can produce gangrenous changes in the sac contents, continuing distally into the femoral canal and the thigh.

Q. What is the cause of pediatric hernias? How is it presented? Why incidence of pediatric hernia is more? How is it treated?

Most pediatric hernias are indirect inguinal hernias, due to persistent and patent processus vaginalis, a weakness in abdominal wall. Many children are born with or develop an inguinal hernia after birth.

Sometimes the hernia is visible only when the infant cries, coughs or strains during bowel movements. It is also visible when an older child stands for a long time. Infants or children may also present with a mass in the groin or scrotum. The incidence of inguinal hernia is more in preterm infants and low birthweight baby. The conditions associated with increased incidence of pediatric hernia are:

- Undescended testis
- Cryptorchidism
- Hypospadias/epispadias
- VP shunt
- Prematurity.

Silk globe sign: It reflects the way the hernia sac fills as it is palpated over the cord structures. Reliable diagnosis is an acceptable indication for surgery than taking the risk of strangulation.

Incarceration is a more serious problem in the pediatric patient than adult and present with a hard, tender groin mass. This can be successfully reduced initially using sedation, Trendelenburg position, icepack application and gentle taxis. If not reduced within 6 hours or there are signs of peritonitis and systemic toxicity, immediate operation is appropriate.

Most pediatric hernias are repaired with high ligation of sac. The external oblique aponeurosis is opened for a short distance from external inguinal ring. The hernia sac is then gently dissected away from the cord structure, proximally until the internal inguinal ring is reached. The sac is then twisted, the structure ligated and amputated. Most pediatric hernia is repaired under GA.

Chapter

15

Congenital Talipes Equinovarus

Q. What is congenital talipes equinovarus?

Congenital talipes equinovarus (CTEV) is one of the most common congenital structural foot deformities in children, being the most common pediatric orthopedic condition. It is a serious emotional issue for the parents.

The heel tilts in and down with forefoot also turned in. The affected foot is smaller than the unaffected foot. The calf is also smaller on the affected side. One or both feet may be affected. The upper limb is normal in idiopathic CTEV. Without t treatment a child with CTEV will walk on the outer side of the foot leading to pain and/or deformity.

The CTEV is commonly known as club foot as the deformity makes the leg and foot look-like a club. It manifests from mild varus position to a significant pathology that can result in an inability to walk or even stand.

The CTEV is syndromic, when in association with genetic syndromes, and is idiopathic when in isolation. Syndromic CTEV arises in many neurological or neuromascular disorders (spina bifida or spinal mascular atrophy). Idiopathic CTEV is more common.

Idiopathic (Nonsyndromic) CTEV or clubfoot is a common developmental disorder of lower limb in children. It is defined as a fixation of the foot in a hand-like orientation—in adduction, supination & varus with concomitant soft tissue abnormalities (i.e. inclined inwards, axially rotated outwards and pointing downwards). Calcaneus, navicular and cuboid bones are medially rotated in relation to talus and are held in adduction and inversion by ligaments and tendons. The foot is supinated and the front of the foot is pronated in relation to back of the foot causing cavus. The first metatarsal is more planter fixed.

Presentation of CTEV is of a planter fixed, inverted and adducted position of the foot. At the nadir of the deformity is the talar neck, which is positioned in varus and planter grade. Without successful correction, in adulthood presents a serious pressure problem on the planter surface. Pressures are maldistributed across the lateral border of the foot and present as serious callosities, often painful.

Q. What are the causes of ICTEV (clubfoot)?

Idiopathic congenital talipes equinovarus (ICTEV) is associated with joint laxity, dislocation of hip, tibial torsion, oligodactyly and absence of some tarsal bone.

The exact cause of the clubfoot is unknown. It cannot be prevented but treatable.

There is strong evidence for a genetic component of the etiology of ICTEV. Genetic background is important. ICTEV is 2–2.5 times more common in males than females.

There is an association ICTEV with maternal smoking during pregnancy. There is an interaction between genetic factor and tobacco exposure.

Amniocentesis carried out early (11–12 weeks) in pregnancy was found to be associated with ICTEV. There may be a critical point in development between 11 and 12 weeks when there is increased susceptibility to ICTEV.

Proposed mechanisms are uterine restriction, abnormalities of joint and/or bone formation, connective tissue, distal limb vasculature, neurological development, muscle migration, an underlying developmental abnormality or developmental arrest. Basis of evidences for each of these theories is:

- *The 'mechanical forces' or 'positional hypothesis':* Hypothesis of uterine restriction—restriction of fetal foot movements by the uterus causes ICTEV. It arises from oligohydramnios sequence, i.e. reduced amniotic fluid volume is itself a cause.
- *The bone/joint hypothesis*: This hypothesis postulates that positional bony abnormalities underly the anomaly. Hippocrates wrote "The deformity involves the entire combination of bones which make up the skeleton of the foot. All the changes seen in the soft part are secondary. Endrochondrial ossification of the foot is disturbed and its coordination with perichondrial ossification is also disturbed".
- *Connective tissue hypothesis*: This hypothesis suggests that primary abnormality of the connective tissue is responsible for ICTEV, supported by association of ICTEV with joint laxity. Presence of increased fibrous tissue in the muscles, fascia, ligaments and tendon sheaths, supports that a restricting fibrosis might be a primary etiological factor.
- *The vascular hypothesis*: Allen et al documented vascular abnormalities in all deformed feet of 12 fetuses. At the level of sinus tarsi, one or more branches of the vascular tree foot, are blocked resulting wasting of the ipsilateral calf from decreased perfusion though anterior tibial artery in development.
- *Support for a neurological hypothesis*: CTEV is a feature of many neurological syndromes, often seen in association with neurological abnormalities that are secondary to spina bifida.
- *Developmental arrest hypothesis*: During late normal human limb development (9–38 weeks), chondrification of the foot is completed, ossification commences, joint cavitation and ligament formation is completed. The distal limb rotates medially allowing the sole of the foot to be placed flat on the ground, rather than being oriented with sole facing

inwards as seen in the late embryonic period. Pronation continues beyond birth and into postnatal development. A severe clubfoot resembles an embryonic foot at the beginning of the second month and the deformity is accompanied by underdevelopment of bones and muscles. Clubfoot may arise due to the arrest of the normal medial rotation of the foot in the late fetal development.

Conclusion: Genetic and environmental factors are important in case of ICTEV. There is evidence that development of bone, joint, connective tissue, innervation and muscle may each be implicated in the pathophysiology. Disturbances of overall process of medial rotation of the fetal foot may be the common pathway link to all these aspects of development. There are more than one different causes and the abnormality may occur as a result of a threshold effect of different factors acting together.

Q. How ICTEV is treated? What is the goal of treatment?

There are two types of ICTEV:

1. Those who respond to strapping and casting (nonsurgical techniques)
2. Resistant types those require surgical correction.

The goal of treatment of the ICTEV is to have a functional pain-free foot with good mobility and strength.

Q. What are the nonsurgical techniques of management of ICTEV?

Nonsurgical techniques are used in newborn and young infants. The first consists of daily stretching, taping and splinting by a physical therapist and parent. The second consists of weekly cast changing by an orthopedist, followed by bracing.

- *Stretching, taping and splinting program*: This is a specialized physical therapy program consisting of stretching stimulation and taping of the foot, which is performed daily. Parents can learn the technique in few months and can continue the treatment at home. Children may continue to wear the plastic splint at night until they are at least two years old.
- *Serial (Ponseti) casting program*: This involves positioning of the foot, followed by application of a cast, which extends from toes to the top of the thigh. They are removed and changed every one or two weeks in the clinic. After casting is complete, patients wear a special brace full-time for three months and then at night until two or three years of age.

Q. How Ponseti method of serial casting is applied to the ICTEV deformity?

Ponseti method of serial casting includes several steps:

Step 1: The foot is adducted and planter flexed at the subtalar joint and the goal is to abduct and dorsiflex the foot.

Step 2: Foot cavus increases when the foot is pronated. If cavus is present, the first step in the manipulation process is to supinate the forefoot by gently

lifting the dropped first metatarsal to correct the cavus. Once the cavus is corrected, the forefoot can be abducted as outlined in the step one.

Step 3: Pronation of the foot also causes the calcaneum to jam under the talus and stay in vasus. At the end of step one, the foot is maximally abducted but never pronated.

Step 4: After the foot is manipulated, a long leg cast is applied to hold the correction. The cast should be snug with minimum but adequate padding. The cast must incorporate the toes right up to the tips but not squeeze the toes or obliterate transverse arch. The cast is molded to contour around the heel while abducting the forefoot against counter pressure on the lateral aspect of the head of the talus. The knee is flexed to 90° for the long leg component.

Step 5: The cases should be separately treated as outlined in step 2 and the equinus should be corrected without causing a mid-foot break. It generally takes up to 4–7 casts to achieve maximum foot abduction. The casts are changed weekly. After maximum foot abduction is obtained, most cases require a percutaneous Achilles tenotomy alone under local anesthesia. The final cast is applied with the foot in maximum dorsiflexion and held in cast for 2–3 weeks.

Step 6: Following manipulation and casting phase, the feet are fitted with open toed, straight laced shoes attached to a Dennis–Brown bar. The shoes are worn for 23 hours a day for 3 months and worn at night and during naps for up to 3 years.

Step 7: In 10–30% cases, a tibialis anterior tendon transfer to lateral cuneiform is performed when the child is almost 3 years of age. This gives lasting correction of the forefoot preventing metatarsal adduction and foot inversion.

Q. What is the basis of Ponseti method of manipulative treatment of ICTEV deformity?

The Ponseti method of manipulative treatment of ICTEV deformity is based on the inherent properties of the connective tissue, cartilage and bone, which respond to the proper mechanical stimuli created by the gradual reduction of the deformity.

The ligaments, joint capsules and the tendons are stretched under gentle manipulation. A planter cast is applied after each manipulation to retain the degree of correction and soften the ligaments. The displaced bones are, thus, gradually brought into the correct alignment with their joint surfaces progressively remodeling yet maintaining congruency.

Q. What are the factors influencing the result of nonsurgical management?

Following factors affect the result of nonsurgical management:

- Nonoperative treatment succeeds better if it is started a few days or weeks after birth

- The earlier is begun and the more continuous the treatment, the better is the result
- With continuous plaster dressing, correction is more quickly attained, reforming of articular facet is more certain and maintenance of position is absolutely under control
- Treatment result is better with plaster of Paris dressing
- A well applied plaster dressing, allows the child to walk more comfortably and easily
- The treatment kept under the control of the surgeon offers better result
- Cleanliness and avoidance of skin irritation also have influence on outcome.

Q. What are the indications of surgical treatment? What procedures are done?

When nonsurgical treatment cannot completely correct the foot deformity or when the foot deformity recurs, an operation will usually be successful in correcting the foot. Most patients wear a cast for one to three months following surgery, which may need to be changed during this period. After the cast is removed, the child may need to wear a special brace to help prevent the clubfoot from recurring.

When tendon Achilles is tenotomized, an important resisting point towards the correction of the deformity is removed.

The division of the planter and internal ligaments removes an important element in correction of abnormal contour of the bones. Open division of structures produces scar tissue, resulting in an added element towards relapse.

Chapter

16

Temporomandibular Joint Ankylosis

Q. What is temporomandibular joint?

Temporomandibular joints are the dual articulation of mandible to temporal bone, located in front of the ears. These joints are ginglymoarthrodial joints since these are both ginglymus (hinging joints) and arthrodial (sliding) joints. It involves the condylar process of the mandible below and articular fossa (or glenoid fossa) of the temporal bone above. Between these articular surfaces is the articular disc (miniscus) which is biconcave, transversely oval disc composed of fibrous connective tissue. Each TMJ is covered with fibrous capsule. There are two joint capsules creating an upper joint space and a lower joint space with articular disc in between. The synovial membrane of the TMJ lines the inside of the fibrous capsule and articular surface of the disc. This membrane secretes synovial fluid which is both lubricant of means to convey nutrients to the tissues inside the joint.

The TMJs are one of the few synovial joints in human body with an articular disc. The disc divides each joint into two, which are synovial cavities consisting of an upper and a lower synovial cavity. The central area of the disc is avascular and lacks innervation. The peripheral region has both blood vessels and nerves. The central part is thinner but of denser consistency than the peripheral region, which is thicker but more cushioned consistency. The avascular disc is the fibrous extension of the capsule between the two bones of the joint. Synovial fluid provides nutrition for the avascular central area of the disc.

The TMJ is different from other joints of the body. The combination of hinge and sliding motion makes this joint most complicated. The tissues that make up the TMJ also differ from other load bearing joints like knee or hip. Because of its complex movements and unique make up, the TMJ poses a tremendous challenge to both patients and care providers, when problems arise.

Q. What are the functions and movements of the temporomandibular joints?

As the temporomandibular joints are flexible, the joint can move smoothly up and down and side-to-side, enabling us to talk, chew and yawn. Muscles attached to and surrounding the TMJ, control its position and movements.

Jaw movements: Normal full jaw opening is 40–50 mm as measured from edge of the cover front teeth to the edge of the upper front teeth. During jaw movement, only the mandible moves.

Normal movements of mandible during functions, such as mastication and chewing are excursions. There are two lateral excursions (left and right) and forward excursion, known as protrusion. The reversal of protrusion is retrusion.

When the mouth is opened, initial movement of the mandibular condyle is rotational which involves mainly the lower joint space and when the mouth is opened further, the movement of the condyle is translation involving mainly the upper joint space. This translational movement is achieved by the condylar head sliding down the articular eminence, which constitutes the front border of articular fossa and limits the forward movement of the condyle. The ligaments of the TMJ restrict the extreme movements of the joint.

The inferior compartment allows for the rotation of the condylar head around an instantaneous axis of rotation, corresponding to the first 20 mm or so of the opening of mouth. After that, the mouth can no longer open without the superior compartment of TMJ becoming active. At this point if the mouth continues to open, the condylar head rotating within the lower compartment of TMJ with entire apparatus (condylar head and articular disc) translates. This translation actually amounts to a rotation around another axis which effectively produces a development, termed the resultant axis of the mandibular rotation, allowing for a low tension environment for the vasculature and innervation of the mandible.

Q. What are the muscle controlling the movements of the temporomandibular joints?

Muscles attached to and surrounding the TMJ control its position and movements.

Muscles of mastication: These muscles are painted on each side and together work to produce the movements of the mandible. The main muscles involved are masseter, temporalis and medial and lateral pterygoid muscles. Most of these muscles have more than one type of movements. Protrusion—lateral and medial pterygoid; retraction—posterior fibers of temporalis. Lateral movement—medial and lateral pterygoid. Each lateral pterygoid muscle is composed of two heads, the upper or superior head of the lower or inferior heads, with different origins and insertions. The two heads of lateral pterygoid have different actions. The lower head contracts during mouth-opening and the upper head contracts during mouthclosing. The function of the lower head is to steady the articular disc, as it moves back with the condyle into the articular fossa. It is relaxed during mouth closure.

Normal TMJ function depends upon the coordinated contraction of masticatory muscles acting on intact condyle-disc complex. Opening of mouth is a passive process involving relaxation of most masticatory muscles except lateral pterygoid and suprahyoid muscles which contract and pull the

mandibular condyle forward. Initially suprahyoid muscles contract causing rotation between the condyle and the inferior surface of the articular disc, allowing first 20 mm or so of opening. Then a forward translational movement of superior disc surface and condyle, aided by lateral pterygoid muscle, allows a further 25 mm of opening. Closing of the mouth is accompanied by contraction of the elevator muscles (medial pterygoid, superficial fibers of masseter muscle and anterior fibers of temporalis muscles). Activation of posterior temporalis and deep masseter muscle fibers produce mandibular retrusion. Contraction of the lateral pterygoid also acts to pull the disc and condyle forward within their glenoid fossa and down the articular eminence and serves to protrude the jaw.

Q. What are the blood supply and innervations of TMJ?

The arterial supply is provided by branches of external carotid artery, predominantly superficial temporal branch. Other branches are deep articular, anterior tympanic, ascending pharyngeal and maxillary arteries.

Q. What are TMJ disorders? What are the causes of it? What are the categories of TMJ disorders?

TMJ disorders are problems affecting the joint usually pain or reduced movements of the joint. The prevailing modern view is that TMJ disorder is a cluster of related disorders with many common features. The TMJ dysfunction is described as the musculoskeletal disorders affecting TMJs and their associated musculature. It is a collective term representing a diverse group of pathologies involving TMJ and muscles of mastication or both. The TMJ disorders are taken to mean any disorders that affect the TMJ. The TMJ dysfunction is taken to mean any symptomatic (pain, limitation of movements, clicking) dysfunction of TMJ. There is no single globally accepted term or definition for this topic.

The TMJ disorders occur as a result of problems with the jaw joint and surrounding facial muscles that control chewing and moving the jaw. Causes include injury to TMJs.

- Grinding and clenching of the teeth causing lots of pressure on the TMJs
- Dislocation of soft cushion or disc between the ball and socket
- Presence of osteoarthritis or rheumatoid arthritis in the TMJs.

Seen more commonly between 20 and 40 years of age. More common in women than men.

TMJ disorders fall into three main categories:

1. *Myofascial pain*: It involves discomfort or pain of the muscles that control jaw function.
2. *Internal derangement of the joint*: It involves a displaced disc, dislocated jaw or injury to the condyle.
3. *Arthritis*: It refers to group of degenerative or inflammatory joint disorders that can affect the TMJ.

The problems linked to the muscles controlling the jaw functions are caused by overactivity of the muscles or increased sensitivity to pain.

The problems inside the joint itself are caused by wear and tear inside the joint (affect older people), arthritis or injury to TMJ or its cartilages. TMJ disorders may be classified as:

- Post-traumatic (following trauma)
- Idiopathic (unknown cause)
- Myogenous (muscle related)
- Arthrogenous (joint related).

} Based on whether muscles of mastication or TMJs themselves are predominantly involved

Most people have relatively mild disorders. Their symptoms improve significantly or disappear completely within weeks or months. For others, the condition causes long-term, persistant and disabling pain.

Q. What are the signs or symptoms of TMJ disorders?

The symptoms usually involve more than one of the various components of masticatory system muscles, tendons, ligaments, bones, connective tissues and nerves.

Three classically described cardinal signs or symptoms of TMJ disorders are:

1. *Pain and tenderness on palpation in the muscles of mastication or joint itself (preauricular pain)*: Pain is defining feature of TMJ disorders and is aggravated by manipulation or functions, such as chewing, clenching, and yawning and is worse on waking. Pain is dull aching, poorly localized and intermittent, though sometimes be constant, usually unilateral.
2. Noise from joint during mandibular movements, described as clicking, popping or crepitus (grating).
3. *Limited range of mandibular movements* which cause difficult eating or talking. There may be locking of the jaw or stiffness in the jaw muscles and joint, especially on waking. There may be inco-ordination, asymmetry or deviation of the mandibular movement.

Pain particularly in chewing muscles and/or jaw joint is the most common symptom. Other symptoms include radiating pain to face, jaw or neck; jaw muscles stiffness; limited movement or locking of the jaw; painful clicking, popping or grating in jaw joint during opening and closing of the mouth, the upper or lower teeth not fitting together properly.

Pain in the joint area or nearby, located just in front of the ear, may spread to the cheek, the ear itself and to the temple. Jaw movements may be reduced and very rarely the jaw may get locked in open or closed mouth position. As ear is very close to TMJ, some patients get ear symptoms as noise in the ear, sensitivity to sound and dizziness (vertigo).

TMJ pain is generally due to:

- Myofascial pain dysfunction syndrome involving muscles of mastication
- Disc displacement
- Osteoarthritis (degenerative joint disease)
- Temporal arteritis (Pain is most diagnostic criteria).

Q. What is the classification of TMJ disorders?

- ***Muscle disorders***:
  - *Splinting*: Hypertonicity in the masticatory muscles. Mouth opening is only restricted due to pain. Symptoms are of acute onset and short duration. If untreated, leads to muscle spasm.
  - *Spasm*: More severe and protracted state of hypertonic state of masticatory muscles.
  - *Inflammation*: Masticatory muscles can become stiff and swollen as consequence of trauma or untreated muscle spasm. Prolonged muscle inflammation can cause a degree of fixed restriction of mouth opening.
- ***Disorders of disc-condyle complex***: Tissue damage or structural incompatibilities of disc-condyle complex may lead to hyper- or hypomobility of TMJ.
- ***Inflammatory disorders*:** An inflammatory process in the TMJ producing sign. Symptoms like synovitis or arthritis affecting the synovial joints. Joint pain, swelling, limited movements and radiological changes in joint space or surrounding tissues may be present.
- ***Dislocation*:** Mandibular condyle prolapses anteriorly over the articular eminence and is completely displaced forward of the articular fossa. During wide opening of mouth or yawning, the ligaments supporting the disc are lax making joint vulnerable to dislocate. Reduction of dislocation is done under heavy sedation or general anesthesia. Downward and slightly forward pressure is exerted on external oblique ridge of the mandible.
  - *Disc displacement*: Initially moderated anterior disc displacement, results in painful clicking joint. Mouth opening is restricted due to pain. With progress, more anterior disc displacement produces a closed joint with a fixed resistance to opening of the mouth that cannot be overcome by manipulation of the jaw.
- ***Chronic hypermobility (fixed restriction to mouth opening)***: Elevator muscle contracture results from prolonged inability to open the mouth, leading to decreased length of these muscles. Infection in the maxillofacial region leads to fibrosis with restriction of muscle mobility and may become permanent unless treated.

Q. How the diagnosis of TMJ disorders is made?

Diagnosis*:*

- Patient's description of symptoms:
  - *Pain*: Preauricular, intra-auricular, facial, temporal/frontal pain and headache.
  - *Joint noises*: Clicking, popping.
  - Diminished mouth opening.
  - *Locking*: Open/closed.
  - History of previous trauma.
- Detailed medical and dental history.

- **Examination***:*
  - *Inspection*: Facial asymmetry, alteration in jaw opening, range of motion of TMJ (full, restricted, none, hypermobility), pain on opening (straight, deviated, deflected), swelling of erythema.
  - *Palpation*: Tenderness (localized over joint, diffused over muscles), crepitus on opening the joint.
  - *Auscultation*: Joint noises (clicking, popping, crepitus). Examination of the problem area including head, neck, face and jaw.

Ruling out other known causes of pain such as sinusitis, ear infection and various types of headaches and facial neuralgias.

Diagnosis is made on the basis of symptoms and examinations. No tests are necessary if the patient is healthy or has typical symptoms of TMJ disorders. Possible tests are:

- Blood tests—signs of inflammation.
- An MRI scan—gives a detailed picture of the joint.
- X-rays are used less often as MRI scan is available.
- Arthroscopy—when other tests do not show the cause of pain.
- A diagnostic nerve block—local anesthetic is injected near the nerve supplying the TMJ. If this relieves pain, it is suggestive of pain originating from the joint itself.

Examination: To palpate the joint and its associated muscles effectively, the patient is asked to go through all the movements of the mandible in relationship to the TMJ and bilaterally palpating the joint just anterior to the external acoustic meatus of each ear. The patient is asked to open or close the mouth several times or then to move the open jaw to the right and then forward. To further assess the mandible moving at TMJ, use digital palpation by gently placing a finger into the outer part of the external acoustic meatus. Auscultation of joint is also done.

Q. What is the management of TMJ disorders? What surgical procedures are done?

Generally the outcome of TMJ disorders is good. Most disorders improve overtime and do not get worse. Most patients do not need injections or surgery and get improved with simple treatment and time.

Experts strongly recommend most conservative or reversible treatments. Conservative treatments do not invade the tissue of the face, jaw or joint nor involve surgery. Reversible treatments do not cause permanent changes in the structure or position of the jaw or teeth. Simple treatments may be all that is necessary—self-care practice, pain medications and stabilization splints. Surgical treatments are controversial and should be avoided, if possible. Before undertaking any surgery on the jaw, it is important to get other options and to fully understand the risks.

Since the disorders involve several disciplines, particularly dentistry and neurology, the treatment involves multiple approaches or is multidisciplinary.

- *Psychological or behavioral interventions:* Treatment of factors that modulate pain sensitivity, such as mood disorders, anxiety, fatigue, is important in treatment of TMJ disorders. Cognitive behavioral therapy is also effective. Hypnosis is appropriate and has comparable effects to relaxation techniques. Relaxation techniques include progressive muscle relaxation, yoga and meditation. These techniques cause reduced sympathetic activity, muscle relaxation, reduced sensitivity to external stimuli and reduced anxiety with sense of well-being.
- *Occlusal splints*: These are often used by dentists to treat TMJ disorders (bite plates/intraoral appliances). Hard stabilization appliances, when adjusted properly have modest efficacy compared to non-occlusive appliances, in the treatment of TMJ disorders. Soft stabilization appliances, anterior positioning appliances and anterior bite appliances also have efficacy in reducing pain in TMJ disorders. However, the potential for adverse events with these appliances is higher and need close monitoring during use.
- *Medications:* Main method of management of pain in TMJ disorders. Drugs used to treat pain are analgesics, benzodiazepine, anticonvulsants (e.g. gabapentin), muscle relaxants. Analgesics are NSAIDs (piroxicam, diclofenac) of cyclo-oxygenase 2 inhibitors (celecoxib). Long-term use of opioid analgesic has been suggested. Injection of local anesthetic produces temporary pain relief and steroids inhibit proinflammatory cytokines. Steroids and other medications are also injected directly into the joint.
- *Physiotherapy, biofeedback and similar noninvasive measures: Physiotherapy* is used as an adjunct to other methods of treatment in TMJ disorders. The simplest method is regular stretching within pain tolerance, using thumb of a finger in scissors maneuver. Commercial devices are also available for stretching exercise. Over time the mouth opening possible without pain can be increased.

  Massage therapy for TMJ disorders improves both subjective and objective health status. Friction massage uses surface pressure to cause temporary ischemia and subsequent hyperemia in the muscle and inactivates trigger points and disrupts small fibrous adhesions within the muscle formed due to restricted movements.

  Osteomanipulative treatment is also used to treat TMJ disorders. It uses precise forces applied by physician to improve the functions of the muscles and ligaments and to optimize blood flow. It improves symptoms of TMJ disorders, maximum mouth opening or mouth opening velocity. It is effective in reducing pain and muscle relaxants use for TENS may override pain by stimulation of superficial nerve fibers, due to release of endorphins.

  Use of *ultrasound* is recommended to produce tissue healing, altered blood flow of metabolic activity at deeper level.

  Low level *laser therapy* is effective in reducing TMJ pain via a promotion of cellular and tissue alterations, which increase recovery and healing potential of tissue, triggered by metabolic activation, increased vascularization and fibroblast formation.

- *Surgery:* These techniques are reserved for most cases where other orthopedic modalities have failed. American Society of Maxillofacial Surgery recommends a conservative, nonsurgical approach first. Only 20% of patients need to proceed to surgery. Surgical procedures are:
  - ***Arthrocentesis*:** Refers to lavage (flushing out) of upper joint space with saline via the introduction of two cannulas, carried out under local anesthesia and also under general anesthesia.
  - ***Arthroscopy*:** Introduction of arthoscope (thin, flexible camera) into the joint via single cannula through a small incision just in front of the ear. Arthroscopy is done purely as diagnostic procedure or combined with surgical interventions within the joint as release of adhesions, release of disc or biopsies, carried out under general anesthesia.
  - ***Intra-articular injection*:** Both sodium hyaluronate and gluco-corticoids are injected into the joint. Its function is to lubricate and maintain internal environment of the cut.

Q. How airway management is done in patients with TMJ disorders?

In patients with TMJ disorders, the presence of pain, restricted movements, hypermobility or swelling in one or both TMJs need detailed history taking and joint movements examined closely before any intervention in the upper airway.

The airway management problems and the treatment plan should be explained to the patient. Informed written consent is obtained.

Patients without fixed restriction to mouth opening have muscle disorders, disc-condyle disorders, joint inflammation or hypermobility. Mouth opening is restricted by pain and the restriction will be overcome by induction of general anesthesia. Normal procedures may be followed during direct laryngoscopy, including jaw thrust. Mouth should be opened not more than is necessary or jaw manipulation should be firm but not forceful.

In surgical patients with fixed restriction of mouth opening:

- Regional anesthetic techniques should be chosen and manipulation of upper airway avoided, if possible.
- For prolonged procedures, the use of LMA is considered if the incisal opening is sufficient to permit its introduction.
- If tracheal intubation is indicated and mouth opening is marginally sufficient for laryngoscopy, special laryngoscopes like Bullard laryngoscope should be considered.
- If mouth opening is less than 25 mm, direct laryngoscopy is not possible for visualization of laryngeal parts for tracheal intubation. So, oral or nasal fiberoptic bronchoscopy (FOB) assisted intubation should be performed under sedation or inhalational anesthetics with patient breathing spontaneous, after anesthetizing the patient's upper airway with topical local anesthetic spray or gurgle.
- *Alternative to FOB*: Blind nasal intubation can be attempted. Retrograde tracheal intubation can also be used.

Q. What problems can occur in critical care patients with restricted mouth opening?

Problems that can occur in critically ill patients with restricted mouth opening are:

- *Acute upper airway obstruction* can be caused by trauma, deep space infection, large tumors or gross anatomical distortion. In such cases, it is safer to perform tracheostomy or cricothyrotomy under local infiltration in awake patients. Oxygenation should be done with face mask and oxygen saturation monitored during the procedure.
- *Acute ventilatory insufficiency* results from lung trauma, pneumonia, pulmonary edema and ARDS. Elective tracheostomy or cricothyrotomy is appropriate for a patient having restricted mouth opening with relatively normal upper airway but severe acute ventilatory insufficiency.

Q. What is temporomandibular joint ankylosis and how it is caused? What are the consequences of it?

Temporomandibular joint ankylosis is stiffness of the TMJ which results in inability of mouth opening either partial or complete. It occurs between 2 and 60 years of age.

Trauma or infection is the main cause of TMJ ankylosis.

Consequences of TMJ ankylosis are:

- Facial asymmetry
- Malocclusion
- Anemia and malnutrition.

It also leads to:

- Airway obstruction
- Obstructive sleep apnea
- Cor pulmonale

All structural deformities lead to difficulty in ventilation, intubation and extubation.

Q. What are the findings in unilateral and bilateral TMJ ankylosis?

Unilateral TMJ ankylosis:

- Facial asymmetry
- Deviation of the mandible and chin on affected side
- Hypoplastic mandible with receding chin
- Fullness of mouth on the affected side.

Bilateral TMJ ankylosis:

- Facial symmetry maintained
- Micrognathia is present
- Bird face deformity present
- Receding chin
- Narrow maxilla
- Protruding upper incisors
- Nil or few mm mouth opening

Untreated cases lead to:

- Anemia, Malnutrition
- Poor oral hygiene
- Respiratory distress
- Increased airway resistance
- Cor-pulmonale.

Q. What is the treatment of TMJ ankylosis? What are the safer techniques to secure airway in these patients?

Treatment is always surgical.

Due to nil or limited mouth opening, nasotracheal intubation with blind or guided by fiberoptic bronchoscope, retrograde tracheal intubation or tracheostomy is the safer technique of securing the airway. Awake, fiberoptic scope-guided nasotracheal intubation is the safest technique of securing the airway.

As TMJ ankylosis is common in pediatric patients, awake intubation is difficult to perform. Blind nasal intubation is the technique of choice, when pediatric fiberoptic bronchoscope is not available. In patients with facial asymmetry and with onset of ankylosis in early childhood, intubation is difficult due to structural deformity of larynx and trachea, which are shifted to the affected side. In children, general anesthesia is necessary before making an attempt to secure airway.

Q. What is the core of airway assessment?

Combination of mouth opening, jaw protrusion and head extension is the core of airway assessment. *Mouth opening* is measured by inter-incisor distance and a value of 4 cm (2 fingers breath) indicates difficult intubation. *Prognathic* ability of mandible depends on the size and shape of the mandible in relation to maxilla and TMJ function. Prognathic inability of mandible (the mandibular incisors cannot be brought in line with maxillary incisors) indicates difficult intubation. Limited head extension (occipito-atlanto axial) impairs direct laryngoscopy and intubation.

Assessment of mouth opening, prognathic ability, head extension, thyromental distance and mallampati test can be performed rapidly and are most important of tests included in ASA guidelines.

Airway evaluation gives some indication of potential difficulty and anesthesiologists can make a judgment of whether direct laryngoscopy, mask ventilation and percutaneous rescue are likely to be successful or not.

Q. What is awake intubation? What are the indications of awake intubation?

Awake intubation is tracheal intubation of a conscious patient allowing spontaneous respiration and airway protection while avoiding the risk of airway maintenance inherent with general anesthesia.

Use of a flexible fiberoptic laryngoscope for tracheal intubation under topical anesthesia was a milestone in safe airway management because intubation of a conscious patient could now be achieved with minimum discomfort. This technique has become the standard for management of anticipated difficult airway.

Indications:

- Anticipated difficult tracheal intubation
- Anticipated difficult mask ventilation (sleep apnea)
- Anticipated difficult rescue technique
- Cervical spine instability
- Position of double lumen tube and bronchial blocker
- Diagnosis of malfunction of supraglottic airway
- Confirmation of tracheal tube position
- Tracheal tube change between nasal and oral routes
- Intensive care use—aspiration of secretion—confirmation of dilatational tracheostomy site.

Q. What are the advantages of awake intubation?

Advantages

- Well-tolerated by awake patients
- Spontaneous breathing continues
- Oxygenation and muscle tone preserved
- Anatomy and muscle tone preserved
- Intubation is easier and safer
- Airway protection preserved
- Phonation used as a guide to identify larynx
- High success rate is achieved

Q. What is the technique of awake intubation?

Technique

- Rapport is established with patient
- Full explanation of the procedure is given
- Patient position may be supine, semi-sitting or sitting
- Drying agent is administered
- Effective topical anesthetic provided
- Equipment checking is done
- Tracheal tube is mounted on the flexible fiberoptic laryngoscope (FFL)
- Insertion cord is kept straight and the scope maneuvered in 3 planes (tip flexion-extension, rotation and advance-withdrawal)
- Secretions aspirated
- Targets (epiglottis, vocal cords, tracheal cartilages and carina) kept in center of view as FFL is advanced
- Advanced close to carina and tracheal tube passed over FFL
- Tracheal tube position is confirmed and secured
- Anesthesia induced.

Q. What are the techniques of airway anesthesia?

Techniques of airway anesthesia:

- *Nebulizers*: Entire airway (well-tolerated)
- *Topical spray and gels*: Upper airway (nose, mouth and pharynx). Four-percent xylocaine spray is popular.
- *Transtracheal injection*: Larynx and trachea.–Rapidly produces good topical anesthesia.
- *Spray as you go*: Larynx and trachea
- *Nerve blocks*: Distribution of nerve supply.– More profound and long lasting anesthesia. Superior laryngeal nerve block produces good anesthesia of the area between vocal cords or epiglottis.

Combination of any of the above: Block of the superior laryngeal nerve bilaterally along with transtracheal injection of local anesthetics provides anesthesia of the airway from the infraglottic area to epiglottis. Additional application of local anesthetic to oral or nasal mucosa, along with appropriate sedation provides satisfactory analgesia for endoscopic procedures.

Nerve blocks:

- *Superior laryngeal nerve*: Patient placed supine with neck extended. Hyoid bone is displaced towards the side to be blocked. A 25 gauge, 2.5 cm needle is walked off the greater cornu of the hyoid bone inferiorly and advanced 2–3 mm. A loss of resistance is felt as the needle passes through the thyrohyoid membrane and 3 mL of local anesthetic solution is injected superficial and deep to this structure. The block is then repeated on the opposite side. This produces anesthesia from the inferior aspects of the epiglottis to the vocal cords.
- *Translaryngeal block*: Produces anesthesia of the trachea below the vocal cords. Patient placed in supine position with neck extended. The cricothyroid membrane is located. A 20 or 22 gauge of 3–5 cm plastic catheter over a needle is introduced in the midline. The needle is removed and plastic catheter held firmly in place. Aspiration of air confirms correct catheter placement. 3–5 mL of 4% lignocaine solution is injected rapidly and the vigorous cough produced helps spread of solution within the trachea.
- *Glossopharyngeal nerve block*: This nerve provides sensory supply to the posterior third of tongue, the pharynx and the superior surface of epiglottis. It can be blocked intra-orally by injecting 5 mL of local anesthetic into the base of each posterior tonsillar pillar, visualized by laryngoscopy after topical anesthetic applied to the tongue. A 22 gauge spiral needle with stylet removed and bending the distal 1 cm is used for this block. As carotid artery is in proximity, aspiration before injection is mandatory.

Q. What is the prime importance to successful direct laryngoscopy?

The tongue and epiglottis are the anatomic structures that intrude into the line of sight during direct laryngoscopy. Management of the tongue and epiglottis is, therefore, the prime importance to successful direct laryngoscopy.

Patient is placed in 'sniff' position. Direct laryngoscopy is then used to displace tongue to the left, out of line of sight. The hyoid bone and the attached tissues are moved anteriorly and the epiglottis elevated directly or indirectly to reveal the larynx.

The head neck position used for direct laryngoscopy need to align the axes of oral cavity, pharynx and larynx. Management of tongue and epiglottis is more important than axes alignment to improve direct laryngoscopy.

In 'sniff' position, cervical spine below C_5 is straight with increased flexion from C_4–C_2 and the head fully extended (occipito-atlanto-axial complex). Neck flexion between C_2 and C_4 is obtained by elevation of head. Head extension facilitates insertion of laryngoscope, full mouth opening and improved view of the larynx. Head extension should be used unless contraindicated.

Q. What are the maneuvers used to optimize the view at direct laryngoscopy? How tracheal intubation is confirmed?

Maneuvers to optimize the view at direct laryngoscopy are:

- Maximum head extension
- Tongue entirely to the left of the laryngoscope
- Optimum depth of insertion of laryngoscope
- Strong lifting force applied in correct direction to the laryngoscope
- External laryngeal manipulation.

After insertion, the tracheal tube is advanced until the cuff is about 2 cm distal to the vocal cords. More proximal position can cause cuff leak and pressure on recurrent laryngeal nerve.

Confirmation of tracheal intubation:

- Visual confirmation of passage of tracheal tube between vocal cords
- Air is easily aspirated from trachea but not from esophagus
- Acceptable breath sounds in both lung fields. Symmetrical expansion of chest on both sides with ventilation
- Identification of CO_2 in expired air is a standard for verification of proper tracheal placement of characteristic waveform over several breaths on capnography is confirmatory finding
- Identification of tracheal sings and carina through flexible fiberoptic laryngoscope
- Auscultation over epigastrium and axilla
- A gurgling sound with first inflation suggests esophageal intubation.

Q. What is the physiological response to direct laryngoscopy and tracheal intubation? How it is prevented?

Direct laryngoscopy and insertion of tracheal tube are noxious stimuli, providing adverse response in cardiovascular, respiratory and other systems. Significant hypertension of tachycardia occurs with tracheal intubation under light plane of anesthesia. Elevation of blood pressure starts within 5 seconds of laryngoscopy, peaks in 1–2 minute and returns to control level within 5 minutes. Such hemodynamic changes can cause myocardial ischemia,

causing little or no harm to healthy patients but are undesirable in patients with cardiac disease.

Prevention:

- Increased depth of anesthesia (N_2O with volatile agents is used)
- Large dose of narcotics (not morphine), fentanyl 6 mcg/kg suppresses hemodynamic response but prolongs respiratory depression
- Aerosol or other application of topical anesthetics is beneficial
- Combination of local anesthetics and opioids is useful
- Esmolol in combination with narcotic is recommended
- Awake flexible fiberoptic laryngoscopy with effective topical anesthesia reduces hemodynamic responses

Q. What are the essential components of the strategy for extubation? What are the complications of extubation?

Airway tube should be removed when airway protection is no longer needed. Essential components of the strategy for extubation include:

- Continued administration of oxygen
- Continued ventilation
- Strategy to facilitate reintubation.

Complications of extubation:

- Hypoventilation (residual effect of anesthetic drugs and neuromuscular blocking agents)
- Upper airway obstruction
- Laryngospasm, bronchospasm and coughing
- Impaired laryngeal competence of pulmonary aspiration
- Hypotension, tachycardia and arrhythmias
- Myocardial ischemia.

Q. What is the strategy extubation for patients with difficult airway?

Important management issues for extubation of patients with difficult airway include airway risk assessment and location, time and technique or extubation. Principal extubation risk assessment tests are leak test, visual inspection and imaging of airway swelling.

Leak test determines whether gas can pass between tracheal tube and wall of the trachea after cuff deflation. Positive test indicates absence of swelling and predicts airway patency after removal of tracheal tube.

Whenever any doubt about risks arises, flexible fiberoptic endoscope should be used to assess swelling or other causes of upper airway obstruction.

An extubation risk patient should remain intubated in intensive therapy unit (ITU) until the risk is resolved. Extubation should not be done in patients with increased risk of vomiting or regurgitation.

Extubation should be performed in OT or ITU in awake patients after breathing 100% oxygen. Noninvasive ventilation and continuous positive airway pressure (CPAP) reduce the need for reintubation.

Q. What are the modes of extubation?

Extubation can be performed at different depth of anesthesia with the terms: Awake extubation, light anesthesia extubation, deep anesthesia extubation.

- *Deep anesthesia extubation*: Straining is less. Avoids reflexes caused by presence of tracheal tube and its removal.
  - Hypoventilation and upper airway obstruction are risks.
  - Absence of protective airway reflexes.
- *Light anesthesia extubation*: Recovery of protective airway reflexes. Adverse hemodynamic of respiratory reflexes are risks.
- *Awake extubation:* There is appropriate response to verbal stimuli.

Chapter

17

Patent Ductus Arteriosus

Q. What is patent ductus arteriosus? What is the pathophysiology of patent ductus arteriosus?

Patent ductus arteriosus (PDA) is present when the ductus arteriosus (which arises just distal to the left subclavian artery and connects the descending aorta to left pulmonary artery) fails to close spontaneously shortly after birth. In fetus, the ductus arteriosus permits pulmonary arterial blood to bypass the deflated lungs and enter the descending aorta for oxygenation in the placenta. In full term neonates, functional closure of the ductus arteriosus may occur soon after the establishment of pulmonary circulation, but the anatomical obliteration takes about 1–3 months. Within few hours of starting of respiration, the muscle wall of ductus arteriosus contracts probably in response to rising oxygen tension of blood flowing through the ductus arteriosus, mediated through the action of prostaglandins.

Pathophysiology: In PDA blood flow is from the high pressure aorta into the pulmonary artery. The resulting aorto-pulmonary artery shunt (left to right shunt) leads to increased pulmonary blood flow. The pulmonary/systemic blood flow ratio depends on the pressure gradient from the aorta to pulmonary artery, the pulmonary/systemic vascular resistance ratio, and the diameter and length of PDA. A decrease in systemic vascular resistance or an increase in pulmonary vascular resistance decreases the magnitude of the shunt through the PDA.

The left to right shunting of blood through PDA results in pulmonary circulatory overload left heart volume overload. Increased pulmonary circulation decreases lung compliance and increases work of breathing. Volume over load of left heart results in increased end diastolic pressure. Initially stroke volume increases and in time left ventricle hypertrophies. Finally there is sub-endothelial ischemia.

Q. Describe fetal circulation. What changes occur in fetal circulation at birth?

The umbilical vein carrying the oxygenated blood (80% saturated) from the placenta enters the fetus at the umbilicus and goes to the liver. In the liver, it gives off branches to the left lobe of the liver and receives the deoxygenated

blood from the portal vein. Greater portion of the oxygenated blood, mixed with some portal venous blood, short circuits the liver through ductus venosus to enter the inferior vena cava and goes to right atrium of the heart. In the right atrium, most of the blood (75%) is guided towards the foramen ovale by the value of the inferior vena cava and crista dividens and passes into left atrium. Here, the blood mixes with small amount of pulmonary venous blood and passes through mitral opening to the left ventricle.

Remaining lesser amount of blood (25%), after reaching the right atrium via superior and inferior vena cava (carrying venous blood from the cephalic and caudal parts of the fetus, respectively), passes through the tricuspid opening into right ventricle.

During ventricular systole, the left ventricular blood is pumped into the ascending and arch of the aorta to be distributed through their branches to heart, head, neck, brain and arms. The right ventricular blood is discharged into the pulmonary trunk. Due to high resistance in pulmonary arteries in fetal lungs, main portion of the blood passes through ductus arteriosus into the descending aorta, bypassing the lungs, and mixes with blood from proximal aorta. The mixed blood is distributed by the descending aorta and leaves the body by two umbilical arteries, to reach the placenta where it is oxygenated and gets ready for recirculation. In fetal circulation, three shunts exist—ductus venosus, ductus arteriosus and foramen ovale.

Several changes in fetal circulation occur due to:
- Cessation of placental blood flow
- Initiation of respiration.

Changes are:
- *Closure of the umbilical arteries*: Functional closure is almost instantaneous preventing fetal blood to drain out. Anatomical closure takes about 3–4 months.
- *Closure of the umbilical vein*: Obliteration occurs a little later than the arteries allowing few extra volume of blood (80–100 mL) to be received by the fetus from the placenta.
- *Closure of the ductus venosus*: It occurs soon after cessation of placental blood flow. The venous pressure of inferior vena cava falls and so the right ventricular pressure.
- *Closure of the ductus arteriosus*: It occurs within few hours of establishment of respiration, the muscle wall of the ductus arteriosus contracts in response to rising oxygen tension of blood flowing through the ductus arteriosus and the changes mediated through the action of prostaglandins leading to functional closure. Anatomical obliteration takes about 1–3 months.
- *Closure of the foramen ovale*: This is caused by an increased pressure of the left atrium combined with decreased pressure of the right atrium. Functional closure occurs after birth but anatomical closure occurs in about 1 year time. During first few days, the closure may be reversible.

Q. What are the clinical presentations of a patient with PDA?

Rarely the child with PDA is asymptomatic and the cardiac defect is detected during a routine physical examination or the child may present with the followings:

- Poor growth
- Breathlessness
- Early fatigability
- Tachycardia
- Frequent lung infections.

Physical examination reveals:

- Prominent or bounding peripheral pulses
- Systolic hypertension and low diastolic pressure due to increased left ventricular stroke volume and flow from aorta to pulmonary circulation.

The first heart sound (S_1) is normal. The second heart sound (S_2) is often masked by the murmur.

Continuous machinery murmur is best heard along the upper left sternal border, radiating down the left side of sternum and to the back. A thrill may be present.

Apical flow rumble: The pulmonary blood flow exceeds the systemic blood flow, causing left sided volume overload leading to increased blood flow from left atrium to left ventricle across mitral value causing a flow rumble.

Cyanosis: Babies with Eisenmenger's syndrome present with differential cyanosis (cyanosis and clubbing of the toes but not the fingers), because the right to left ductal shunting is distal to the subclavian arteries.

Q. What investigations are done in a child with PDA?

Investigations done in a child with PDA include:

- *Chest radiography*:
  - In significant left-to-right shunt, cardiomegaly with left atrial and left ventricular enlargement is present.
  - If pulmonary hypertension develops, right ventricular hypertrophy is apparent.
  - With marked increase in pulmonary blood flow, signs of pulmonary edema are seen.

  May be normal with small PDA.
- *Electrocardiography*:
  - Sinus tachycardia or atrial fibrillation
  - Small PDA—the ECG findings may be normal
  - Large PDA— left ventricular hypertrophy.
  - In neonate, especially premature neonate with large PDA—T-wave inversion and ST segment depression suggesting ischemia or supply demand mismatch.
- *Two dimentional and Doppler echocardiography*:
  - It helps to confirm the diagnosis of PDA
  - It is useful in describing the PDA as silent, small, moderate or large.

- *M-mode echocardiography*:
  - It is used to measure the cardiac chamber sizes to quantify left ventricular systolic function
  - Small PDA—chamber sizes are usually normal although mild left atrial and/or left ventricular enlargement may be present.
  - Moderate to large PDA, the left atrium or left ventricle is enlarged.
- *Cardiac catheterization*: Therapeutic catheterization is the treatment of choice for PDA. It helps in complete diagnostic evaluation of hemodynamics of the patient as it is important to assess the pulmonary vascular resistance and the amount of shunting before transcatheter closure.

  It also helps in assessing the response of vasodilating agents as oxygen, nitric-oxide, sildenafil, nifedipine or prostacyclin in patients with raised pulmonary vascular resistance.
- *MRI and CT scan*: Demonstrate the abnormality and assess the size of pulmonary arteries.

Q. What are the treatment options of PDA?

The treatment options for PDA are pharmacological or mechanical closure of the ductus.

Pharmacological treatments in neonatal period

The risks of surgical closure are significant and include, intracranial hemorrhage, infection, and recurrent laryngeal nerve paralysis, especially in infants born at less than 28 weeks of gestation (premature neonate).

When the ductus is structurally intact, inhibition of prostaglandin synthesis with nonselective cyclo-oxygenase inhibitors (cox-1, cox-2) is an effective alternative to surgery for closure of PDA in first week of neonatal life.

Indomethacin: A prostaglandin synthetase inhibitor is the preferred therapy for closure of PDA and has replaced surgery in early neonatal period. Adverse side effects are: Decreased mesenteric, renal, cerebral blood flow.

Ibuprofen: A prostaglandin synthetase inhibitor that can be used effectively to close PDA in early neonatal period and has less effect on organ blood flow than indomethacin.

Mechanical closure is done once diagnosis is established in term infants as they are unresponsive to pharmacological therapy. Closure should be undertaken in infancy if the shunt is significant and pulmonary resistance is not elevated.

Mechanical closure may be undertaken with surgery or catheter-based therapy.

- *Catheter-based therapy*: Transluminal placement of occlusive devices is done. Complications include thrombo-embolism, endocarditis, incomplete occlusion, vascular injury and hemorrhage from perforation. Not applicable to very young infant as peripheral vessels do not provide adequate access for delivery devices.

- *Surgical procedures*:
  - Video-assisted thoracoscopic: Occlusion using metal clips. Preterm newborn and children do well with thoracoscopic technique. It has few advantages over standard surgical technique.
  - *Coil occlusion*: Indicated in older children (>5 years) and those with smalls duet (<3 mm).
  - Standard surgical approach involves:
    - Triple ligation of PDA with placement of suture, through left anterior or posterior thoracotomy.
    - Division between clamps with overscrewing of both the ends, when the ductus is short and broad (diameter approaching length).

Q. What are the pathophysiologic problems created by left-to-right shunt? Why the increased cardiac workload is concern?

If the shunt is nonrestrictive and does not impede blood flowing freely in both direction then the main determinant of blood is the pulmonary and systemic vascular resistance. Left-to-right shunt results in increased pulmonary blood flow creating following problems:

- Volume overload of pulmonary circulation
- Increased cardiac work of left ventricle which is required to increase stroke volume and heart rate to ensure adequate systemic perfusion
- Excessive pulmonary blood flow resulting in progressive elevation of PVR.

Increased cardiac workload is a concern because the immature heart of the baby is:

- Relatively noncompliant
- Has relatively restricted ability to change stroke volume
- Plateau of pressure volume curve reaches early
- Cardiac output is more rate-dependent.

Q. What is the anesthetic management in a child with PDA?

Anesthetic goal in a patient with PDA whether for corrective surgery or noncardiac surgery is to:

- Prevent increase in shunt (left-to-right) or reversal of shunt (right-to-left)
- Avoid increase in pulmonary vascular resistance by avoiding hypoxia, hypercarbia, acidosis, nitrous oxide and pain-related catecholamine release
- Prevent decrease in systemic vascular resistance, particularly drug-induced fall in cardiac output
- Prevent hypoglycemia which is a frequent concern in neonates during perioperative period
- Infective endocarditis prophylaxis is not required in babies undergoing corrective surgery (AHA, 2008)
- Antibiotic prophylaxis for protection against infective endocarditis is recommended PDA scheduled for noncardiac surgery.

Anesthetic procedure:

Premedication: The goal is to achieve adequate sedation, maintain respiratory and hemodynamic stability. In a premature neonate, sedation is not required.

Anesthetic induction: Preoxygenation with 100% oxygen. Inhalational induction is done with sevoflurane, which decreases systemic vascular resistance and improves systemic blood flow by decreasing the magnitude of left-to-right shunt. Intubation is done with appropriate sized ETT.

Maintenance of anesthesia: Analgesia with fentanyl 1–3 mg/kg. High-dose opioids are not tolerated by neonates. Muscle relaxation can be with either vecuronium or atracurium.

- Ventilation: Positive pressure ventilation is well-tolerated as a rise in airway pressure increases pulmonary vascular resistance decreasing the shunt.
- Maintenance of anesthesia with air: Oxygen mixture and volatile anesthetics. Anesthesia machine should have the capacity to provide air as well as oxygen and nitrous oxide to help balance pulmonary or systemic blood flow.

When surgical closure of the PDA is planned through left thoracotomy, appropriate preparation must be taken in anticipation of possibility of large blood loss during attempted ligation. Anesthesia with volatile agents is useful, as these drugs tend to lower systemic blood pressure, lessening the danger of the PDA escaping from vascular clamp or tearing as it is being divided. The decreased systemic vascular resistance produced by volatile anesthetics may improve systemic blood flow by decreasing the magnitude of left-to-right shunt. Increase in systemic vascular resistance or decrease in pulmonary vascular resistance, increases the magnitude of left-to-right shunt and are to be avoided. Continuous monitoring of systemic blood pressure by a catheter placed in a peripheral artery is helpful during operation.

Reversal: Postoperative ventilation may be needed due to the prematurity of the baby.

Ligation of PDA is often associated with significant systemic hypertension during the postoperative period. This can be managed with continuous infusion of nitroprusside. Long-acting antihypertensive drugs can be gradually substituted for nitroprusside, if systemic hypertension persists.

Monitoring during anesthesia for ligation of PDA through left thoracotomy.

Pre-induction:

- Precordial stethoscope
- Pulse oximetry
- ECG
- NIBP

Preincision:

- Invasive blood pressure
- Hourly urine output
- Esophageal stethoscope
- Temperature monitoring
- CVP monitoring— may or may not be used
- $EtCO_2$.

Other monitors that may be used are:

- Doppler transducer
- Arterial blood gas (ABG).

Chapter

18

Intercostal Drain

Q. Why chest drains are inserted? How the chest drains function?

Purposes: Chest drains or pleural tubes are inserted for two reasons:

- To drain fluid, thereby, preventing pleural fluid accumulation
- To evacuate air if an air leak is present.

The chest chain systems work by a combination of followings:

- When patient breaths out deeply or coughs, the expiratory positive pressure helps to push air and fluid out of the pleural space
- Positioning the collection chamber of the chest drain below the level of the chest allows fluid drainage by gravity
- Application of suction to the collection chamber hastens drainage from the pleural space. Suction should be applied with caution.

Q. What are the common indications for the placement of pleural tube?

Placement of pleural tube is indicated for the following conditions:

- Pneumothorax
  - In mechanically ventilated patients
  - In tension pneumothorax after initial decompression by needle insertion
  - In persistent or recurrent pneumothorax after simple aspiration
  - In large or symptomatic pneumothorax.
- Large or symptomatic pleural infusion
- In patients with other pleural collections
  - Pus in the pleural space (empyema)
  - Blood in the pleural space (hemothorax)
  - Style in the pleural cavity (chylothorax)
- Postoperative placement after thoracotomy or thoracoscopy.

Q. What is pneumothorax? What are the clinical features of pneumothorax?

Pneumothorax is the presence of air in the pleural space. It occurs either spontaneously through rupture of subpleural emphysematous bulla, from

iatrogenic injury following thoracic surgery or biopsy or from trauma to the lung or chest wall.

Patient presents with sudden onset of unilateral pleuritic chest pain or breathlessness. A large pneumothorax (>15% of hemothorax) results in decreased or absent breath sound.

Combination of absent breath sound and resonant percussion note is diagnostic of pneumothorax clinical features.

Inspection—tachypnea (Pain and deflation reflex).

Palpation—decreased expansion of the affected side of the chest.

Percussion—resonant or hyperresonant note on the affected side of the chest.

Auscultation—absent breath sound on the affected side of the chest.

Tension pneumothorax also causes deviation of trachea to unaffected side, associated with tachycardia and hypotension.

Q. What are the types of spontaneous pneumothorax?

Spontaneous pneumothorax are of the followings types:

- *Closed pneumothorax*: Here the communication between lung and pleural space seal off as the lung deflates and does not reopen. The mean pleural pressure remains negative. Spontaneous reabsorption of air and re-expansion of lung occurs over few days or weeks. Infection is uncommon.
- *Open pneumothorax*: Here the communication between the lung and pleural space fails to seal or air continuously transfers freely between lung and pleural space. Commonly seen following rupture of emphysematous bulla, tuberculosis cavity, lung abscess in pleural space. The mean pleural pressure is atmospheric. For example, bronchopleural fistula which causes transmission of infection from air passage to pleural space, leading to empyema.
- *Tension pneumothorax*: Here the communication between airway and pleura is small, which acts as one way value allowing air to enter the pleural space during inspiration but cannot escape during expiration. Large amount of trapped air accumulates in the pleural space and intrapleural pressure rises well above atmospheric levels. Pressure causes mediastinal displacement towards the unaffected side with compression of the normal lung and impaired systemic venous return, causing hypotension, tachycardia and cardiovascular compromise. Findings are: Rapidly progressive breathlessness, marked tachycardia, hypotension, cyanosis of tracheal displacement to opposite side.

Intrapleural space is between lung and the chest wall, containing thin layer of fluid. The intrapleural pressure at the base of the lung is normally about 2.5 mm Hg (relative to atmospheric) or decreases to about 6 mm Hg at the start of inspiration.

Q. What is the management of pneumothorax?

Chest radiography shows sharply defined edge of the deflated lung with complete translucency (no lung marking) between lung edge and chest wall. The extent of mediastinal shifting is also seen.

Primary pneumothorax with lung edge less than 2 cm from the chest wall of the patient not breathless, resolves without intervention.

In young patient presenting with moderate or large spontaneous primary pneumothorax, percutaneous needle aspiration of air is a simple and well-tolerated alternative to intercostal drainage.

Patients with underlying lung disease require intercostal tube drainage or observation even with small secondary pneumothorax. Intercostal space in midaxillary line following blunt dissection through parietal pleura. It is connected to a water seal drain or one way Heimlich valve. The water seal chain is removed after the lung has fully reinflated and bubbling stopped.

Continued bubbling after 5–7 days is indication for surgery.

Bubbling stopped prior to re-expansion of lung indicates that the tube is blocked, kinked or displaced.

All these patients are given supplemental O_2 which accelerates rate of absorption of air by pleural.

Q. What is pleural effusion? What are the clinical features of pleural effusion?

Pleural effusion is accumulation of serous fluid within the pleural space. In general, pleural fluid accumulates as a result of either increased hydrostatic pressure or decreased oncotic pressure or from increased microvascular pressure due to disease of the pleural surface itself or injury in the adjacent lung (exudative effusion).

Clinical features on examination of patient with large pleural effusion are as follows:

- Breathlessness is the only symptoms
- Inspection—tachypnea
- Palpation—decreased expansion of chest on the affected side
- Percussion—stony dull note in the mid and lower zones of chest on affected side
- Auscultation—absent breath sound and vocal resonance on the base of the affected side of the chest. Crackles heard above effusion.

Q. What investigations are done in a patient with pleural effusion and how it is managed?

Investigations

- *Chest X-ray* (PA view) shows curved shadow at the lung base, blunting the costophrenic angle and ascending towards the axilla.
- *Ultrasound or CT scan* is required to identify smaller pleural effusion.

Management

Therapeutic aspiration is done to palliate breathlessness. Removing more than 1.5 L in one episode may lead to re-expansion of pulmonary edema or is not advisable.

Treatment of the underlying causes results in resolution of the effusion.

Other pleural collection includes:
- Pus (empyema)
- Blood (hemothorax)
- Chyle (chylothorax).

Q. What is empyema? What are the clinical features of empyema?

Empyema is the presence of pus in the pleural space. Pus may be as thin as serous fluid or as thick that cannot be aspirated through a wide bore needle. It may involve the whole pleural space or only a part of it (loculated or encysted empyema) or is almost invariably unilateral.

The pas in the pleural cavity is often under considerable pressure or if not treated adequately may rupture into bronchus leading to bronchopleural fistula and pyopneumothorax or track through the chest wall forming subcutaneous abscess or sinus.

The clinical features of empyema are as follows:
- Systemic features
  - Pyrexia (high and remittent)
  - Rigors, seating, malaise or weight loss
  - Polymorphonuclear leukocytosis.
- Local features
  - Pleural pain, breathlessness, cough and sputum due to underlying lung disease or from bronchopleural fistula
  - Clinical signs of fluid in pleural space.

Q. What is the management of empyema?

Finding of chest radiograph is indistinguishable from pleural effusion. If air is present with empyema (pyopneumothorax), a horizontal fluid level marks the interface between liquid and air.

The only way to heal empyema is eradication of infection, obliteration of empyema space, or apposition of visceral and parietal pleural layers. This cannot occur unless re-expansion of the compressed lung is required in an early stage by removal of all the pus from the pleural space.

When the patient is acutely ill and the pus is thin, an intercostal tube is inserted under ultrasound or CT guidance into the most dependent part of the empyema space or connected to a water seal drain stem. If initial aspirate reveals turbid fluid or frank pus or if locations seen in ultrasound, the tube is put on suction (5–10 cm H_2O) and flushed regularly with 20 mL normal saline. Finally an antibiotic-directed against the organism causing empyema is given for 2–4 weeks.

If the intercostal tube does not provide adequate drainage due to thick or loculated pus, surgical intervention is required. The empyema cavity is cleaned of pus and adhesion and a wide bore tube is inserted to allow optimum drainage.

If gross thickening of visceral pleural develops, preventing re-expansion of lung, surgical decortication of the lung is required.

Q. How chest drain is inserted? What are the complications associated with chest drain insertion? How to review the drainage system?

Tube thoracotomy or chest tube insertion is done for pneumothorax, hemothorax, pleural effusion or empyema.

Chest tube can be easily placed using a combination of local anesthesia and high conscious sedation.

Common complications include:

- Inadequate analgesia or sedation, incomplete penetration of the pleura with formation of subcutaneous tract for the tube, laceration to the lung or diaphragm, intraperitoneal placement of the tube through diaphragm and blooding related to these various lacerations or injury to the pleural adhesions. Additional problems include slippage of the tube out of position and mechanical problems related to drainage system.
- All these complications can be avoided with proper initial insertion techniques plus a daily review of the drainage system and follow-up of radiographs.
- Tube removal can create a residual pneumothorax if patient does not maintain positive intrapleural pressure by valsalva maneuver during tube removal and dressing application.
- A drainage volume of 150 mL or less over 24 hours is necessary to safely remove the chest tube. Suctions at level of 20 cm H_2O are routinely used after pulmonary surgery to eradicate residual air leak.
- Routine use of water seal (off suction) actually promotes more rapid healing of parenchymal air leak.
- Routine use of water seal (off suction) actually promotes more rapid healing of parenchymal air leak. The main guidelines for use of water seal are the air leak and degree of expansion of the remaining lung. If leak is significant enough to induce atelectasis or collapse during use or water seal (off suction), suction should be used to achieve lung expansion.

Process of assessing an air leak:

- The chest tube and its attached tubing are examined to exclude kinks or mechanical obstructions. The patient is asked to cough. If bubbles pass through water seal chamber, an air leak is presumed.
- During voluntary cough, the fluid level in the water seal chamber should move up and down with cough and deep inspiration reflecting the pleural pressure changes during these maneuvers. A stationary fluid level indicates mechanical blockage by external tube compression or clot or debris in the tube.

Q. How bronchopleural fistula is caused? How diagnosis is done?

Bronchopleural fistula is an abnormal connection between the intrathoracic respiratory system and the pleural cavity and caused by:

- Rupture of a lung abscess bulla, cyst, bronchus or parenchymal tissue into pleural space

- Erosion of bronchus by carcinoma or chronic inflammatory disease.
- Most fistulas are postoperative complications of pneumonectomy and lobectomy resulted from stump dehiscence of a bronchial suture line.

Clinical features: Productive cough, hemoptysis, fever, dyspnea and subcutaneous emphysema. Severity of symptoms is proportional to the size of the fistula. Big size with large air leak causes severe dyspnea necessitating urgent ventilator support. Patients are debilitated. Respiratory function is compromised by infection and prior lung resection.

Diagnosis After pneumonectomy, sudden onset of dyspnea, subcutaneous emphysema, contralateral deviation of trachea, and decrease of fluid level on serial radiograph of the chest are seen.

In lobectomy patients, persistent air leak, purulent drainage and expectoration of purulent material are seen.

After removal of chest drain, fever purulent sputum, new air fluid level in intrapleural cavity on chest X-ray.

Diagnosis is confirmed by bronchoscopic examination.

- If disruption occurs in the early postoperative period, resuture of the stump is possible.
- Late or chronic pneumonectomy bronchial disruption—open pleural drainage or use of muscle flap to reinforce the bronchial stump.
- In non-pneumonectomy cases:
  - If the lung expands to fill the thoracic cavity, the air leak can be controlled.
  - If fistula is large and significant air leak through a large persistent pleural space occurs, the fistula is unlikely to close or surgical resection is necessary.

Q. What are the intraoperative challenges during repair of bronchopleural fistula? What are the anesthetic considerations for repair of bronchopleural fistula?

Intraoperative challenges in a patient with bronchopleural fistula are:

- Need for lung isolation to protect the healthy lung region
- Possibility of tension pneumothorax with the pressure ventilation
- Possibility to have inadequate ventilation due to loss of tidal volume from the fistula.

Preoperative estimation of loss of tidal volume:

- When chest drain is present:
  - Intermittent flow of air bubbles indicates small fistula
  - Continuous flow of air bubbles indicates large fistula or bronchus.
- Quantified by assessing the difference between inhaled and exhaled tidal volume:
  - In non-intubated patients, with tight fitting mask and fast responding spirometer

- In intubated patients by direct attachment of spirometer to endotracheal tube. The larger the air leak, the greater the need for isolation of fistula with double lumen tube (DLT) of blocker.

Anesthetic management

- To protect the healthy lung from contamination—control distribution of ventilation
- Airway pressure minimized during ventilation
- Invasive arterial BP monitoring obtained.

Delivery of adequate positive pressure ventilation intraoperatively is considered before surgery. A chest drain is placed before induction to avoid possibility of tension pneumothorax with positive pressure ventilation.

In small, chronic, uninfected fistula, single lumen tube (SLT) can be used.

If leak is significant, DLT is best choice for delivering positive pressure ventilation. DLT provides the pressure to normal lung without loss of minute volume and prevents contamination of healthy lung.

Lung isolation is obtained safely with awake fibreoptic intubation with DLT. It needs cooperative patients of excellent topical anesthesia.

Alternative method is to maintain spontaneous ventilation during induction and intubation, until lung isolation is secured—not tolerated by older patients with comorbidities.

In post pneumonectomy fistula, DLT or SLT is guided by direct vision with FOB into the existing lung.

Alternative method of ventilation is high frequency oscillatory ventilation with permissive hypercapnea—avoids barotraumas to healthy lung, decreases air leak or optimizes the operative outcome. It uses lower peak airway pressure or decreases air leak. Early extubation in OR should be done in all patients undergoing fistula repair.

High dependency unit (HDU)/ICU care is needed in postoperative period.

Q. What are the indications of lung isolation? What are the methods of lung isolation?

Lung isolation is performed to facilitate one lung ventilation (OLV) in patients undergoing cardiac, thoracic, mediastinal, vascular, esophageal or orthopedic procedures involving chest cavity. It is also needed in the management of patients with unilateral lung injury. The purpose of lung isolation is to: (i) protect the healthy lung from soiling (ii) provide differential pattern of ventilation.

Lung isolation is achieved by three different methods:

- With use of DLT
- With use of bronchial blockers
- With use of SLT.

DLT

- *Carlen's tube*: Red rubber double lumen tube.
  - It is first DLT introduced for long isolation.

Disadvantages: (i) high flow resistance due to narrow lumen (ii) hazards associated with carinal hook.

- *Robertshaw tube*: Separate left- and right-sided DLT. PVC tube with larger lumen and carinal hook removed. There are radiographic markers surrounding endotracheal and endobronchial cuffs or also surrounding the ventilation slot for right upper lobe bronchus. Right main bronchus is shorter (1.5 cm). Right upper lobe bronchus originates at a distance of 1.2–2 cm from the carina. So there is possibility of obstruction of right upper lobe bronchus by endobronchial tube placed on right main bronchus. To overcome this complication, right-sided Robertshaw DLT incorporates a modified slot with cuff on the endobronchial side to allow ventilation of right upper lobe.

Left-sided tube is commonly used during practice. Bright blue, low resistance, low pressure endobronchial cuffs are incorporated for easier visualization through FOB.

Insertion: There are two techniques: (i) Blind technique: The tube is introduced with direct laryngoscopy and then turned 90° counter clockwise (for left DLT placement) after the endobronchial cuff passes the vocal cord and then negotiated into the main bronchus. (ii) Direct vision technique using FOB: The tip of the endobronchial lumen is guided into correct bronchus after DLT passes vocal cord.

Problems of DLT:

- Malposition and airway trauma.

Bronchial blockers: Alternative method to achieve lung separation involves blockade of mainstem bronchus to allow lung collapse distal to the occlusion. Blocker can also be used to achieve lobar collapse.

Types of devices are:

- Within a modified SLT as an enclosed bronchial blocker
- One used independently with conventional SLT
- Arndt wire-guided endobronchial blocker
- Cohen tip deflecting endobronchial blocker
- Fuji uniblocker.

Torque control blocker (univent) –

Placement: Straight forward, fully retracted into standard lumen of the tube. Conventional endotracheal tube intubation technique is used.

Q. What are the modes of mechanical ventilation?

Followings are the modes of mechanical ventilation:

- *Controlled mechanical ventilation*: The main characteristic is that the variable used by the ventilator to trigger and to cycle off breath is time. May be flow and volume controlled mechanical ventilation or pressure controlled ventilation.
- *Assisted mechanical ventilation*: Provides ventilatory support while patient's Pmus is preserved. The patient is able to contribute some degree

of ventilator support. Preserving diaphragmatic activity is to reduce the need for sedation of paralysis, avoid disuse atrophy of respiratory muscles of minimize the cardiovascular effects of controlled mechanical ventilation.

- Positive end expiratory pressure (PEEP) is the positive pressure applied at the end of expiration during mechanical ventilation.
- Continuous positive airway pressure (CPAP) is a constant level of pressure applied to a spontaneously breathing cycle.

PEEP/CPAP improve arterial oxygenation by re-expanding functionally collapsed alveoli, redistributing lung water and thereby reducing V/Q mismatch.

PEEP/CPAP lead to increase in compliance (alveolar recruitment), decrease in the compliance (lung over distension). There is increased intrathoracic pressure and reduced venous return.

Mechanical ventilation is applied through endotracheal tube or tracheostomy tube. Noninvasive ventilation applied to the patient's mouth or nose restores respiratory function minimizing the side effects associated with invasive ventilation. It avoids endotracheal intubation and patient discomfort. There is decreased need for sedation or incidence of ventilator-related pneumonia and sepsis. Conventional ventilator support delivers a tidal volume higher than the dead space volume and applies positive pressure at airway opening.

Non-conventional modalities of ventilation support are based on application of a tidal volume lower than the dead space volume (high frequency oscillator) and on the use of external gas exchangers that bypass the lung and heart or provide ventilator support without the need of positive pressure (extracorporeal support).

High frequency oscillator (HFO) is an open ventilator system that maintains continuous exchange with the atmosphere and escape off ventilated gas through an open port. A bulk flow provides fresh air to the circuit of small tidal volume delivered by the strokes of the device, sustain alveolar ventilation. HFO provides active expiration. Inspiration and expiration are independent of level of applied pressure, CO_2 removal and oxygenation are not coupled to inflation or deflation of the lung. Alveolar gas exchange is, therefore, entirely due to the bulk of fresh air delivered through the circuit.

Q. What are the components of chest drainage system? What are the components of underwater seal?

Components of the chest drain systems are:

- A chest tube—inserted into pleural space or mediastinal cavity to allow contents (air, fluid) to leave the chest.
- A length of flexible tubing connecting the chest tube—the drainage system.
- A drainage system consisting of a chamber that collects fluid. There is a one way seal (fluid level or mechanical value) that prevents outside air from entering the thoracic cavity during inspiration.

Under water seal acts as a one way valve through which air is expelled from the pleural space during expiration and prevented from reentering during the next inspiration.

Q. What are the types of chest tube drainage system? How they function?

Types of chest tube drainage system are:
- Single bottle drainage system
- Two bottle drainage system
- Three bottle drainage system
- Multifunction chest drainage system.

Single bottle drainage system: It consists of a collecting chamber of approximately 20 cm diameter, in which the chest drainage tube is submerged to a depth of 1–2 cm of water. When the patient inspires, water is drawn up to the tube to a height equal to negative intrathoracic pressure (usually up to 20 cm H_2O). So the collection chamber must be kept far enough below the patient to prevent water from being sucked up into the chest. Usually a height of 100 cm is sufficient to allow for negative pressure as high as 80 cm H_2O. The portion of the tubing that is under water offers resistance to expiration. This should be no more than 2–3 cm. The end of the tube in the underwater seal bottle must remain covered with water at all times. Collected air escapes through a side vent in the chamber.

The disadvantage of single bottle drainage system is that as fluid drains from the chest, the level of fluid in the water seal chamber rises, resulting in increase in resistance against which the patient has to breathe and can obstruct further outflow of fluid from the chest.

Two bottle drainage system: To overcome the disadvantage of single bottle system, a second bottle is introduced between the chest tube and water seal chamber. This trap bottle collects the fluid draining out of the chest, while air passes to the water seal chamber and is vented to the atmosphere. This keeps the water level in water seal chamber constant.

Three bottle drainage system: When application of suction (i.e. negative pressure) is desired, a third bottle is added between the water seal chamber and suction device. This third bottle has an inlet connected to the vent of the water seal chamber, an outlet connected to the suction device and a control tube open to the atmosphere on the other side and submerged 20 cm under water on the other side.

When suction is applied, air is drawn down the atmospheric vent in the third bottle, equal to the pressure inside the bottle that is decreased by the vacuum. This low pressure suction is transmitted to the water seal chamber and then into the pleural cavity resulting in evacuation of contents and allowing quicker re-expansion of the underlying lung.

Continuous low pressure suction (recommended level of suction is 5–20 cm H_2O) may be applied to ICD bottle. Suction pressure >20 cm H_2O can

damage lung tissue. Suction is avoided when there is ongoing air leak (air leak may worsen) and to post pneumonectomy patients.

Three bottle drainage system provides stable water seal level, allows documentation of drainage and also controlled suction. But it is bulky and does not allow easy transport or ambulation of patients.

Multifunction chest drainage system: Commercially available multifunction drainage systems incorporate all the components of the three bottle system into a single unit. The chance of accidental disconnections of different components is minimized.

Q. When an ICD can be removed?

Intercostal drainage tube can be removed when:

- Drainage diminishes to little or nil
- Air leak has stopped
- Fluctuations of water C in water seal chamber stop.
- Patient is breathing normally without any signs of respiratory distress
- Breath sounds are equal and at baseline for the patient
- Chest X-ray shows complete re-expansion of lung and absent residual air or fluid in the pleural space.

Chapter

19

Hydrocephalus

Hydrocephalus itself is not a disease but is the consequence of various congenital or acquired pathological conditions with many variations. It is always characterized by an increased volume of CSF within the ventricular system and it is mostly caused by obstruction of CSF circulation or reduced absorption. In Arnold–Chiari malformations, the brainstem is displaced downwards with an abnormal vermis extend below the foramen magnum and into the cervical canal. The fourth ventricle becomes compressed and its foramina become obstructed. Most children with meningomyelocele have an associated Arnold–Chiari malformation and ultimately develop hydrocephalus usually in the first month of life.

Q. How CSF circulation occurs?

The cerebral ventricles contain choroid plexus. This produces CSF which cushions the brain within the cranium. CSF flows from the lateral or 3rd ventricle through the aqueduct to the 4th ventricle. It then exists the 4th ventricle through foramina of Luschka and Magendie flowing over the hemisphere and down around the spinal cord and roots in the subarachnoid space. It is then absorbed into venous sinuses via the arachnoids villa, as a result of the hydrostatic pressure difference between CSF pressure and venous sinus pressure, Hence, the rate of CSF absorption increases with increasing ICP. At any time, there is approximately 150 mL of CSF, 50 mL within ventricular system and 100 mL in the subarachnoid space.

Q. What are the functions of CSF?

The primary function of CSF is to provide buoyancy to allow the brain to float, protecting it from repeated trauma on each occasion the head moves. It also serves as a transport medium allowing circulation of various neurotransmitters, local hormones, and as a modified lymphatic system for brain and spinal cord.

Q. What are the causes of hydrocephalus?

Four basic causes are involved in the causation of hydrocephalus:

1. Congenital anomalies (e.g. Arnold–Chiari malformation).
2. Neoplasm.

3. Inflammatory conditions.
4. Overproduction of CSF (choroid plexus papilloma).

Q. What is the classification of hydrocephalus?

The classification of hydrocephalus is:

- Obstructive hydrocephalus: It is caused by blockage somewhere in the fluid pathway of the CSF. It may be:
  - Communicating: The ventricular system communicating with the subarachnoid system, e.g. when the fluid pathway to the subarachnoid space is open as in tuberculous arachnoiditis.
  - Noncommunicating when the fluid pathway proximal to the subarachnoid space is obstructed, e.g. aqueductal stenosis.
- Nonobstructive hydrocephalus: It is caused by a reduction in the volume of brain substance with secondary dilatation of the ventricles, by overproduction of CSF (choroid plexus papilloma) or diminished reabsorption due to scarring.

Hydrocephalus frequently develops in premature infants who have had an intraventricular hemorrhage.

Noncommunicating hydrocephalus occurs from scaring around the aqueduct of Sylvius. More often, hydrocephalus is the consequence of diminished CSF reabsoption from the arachnoid granulations from scar or fibrin deposition and it is, therefore, communicating hydrocephalus. Neonatal meningitis produces hydrocephalus by a similar mechanism.

Q. What is active hydrocephalus? What are the varieties of it?

Hydrocephalus is not a single entity but is the end result of a diverse group of conditions that result in accumulation of CSF within the cranium which is usually under raised pressure and associated with ventricular enlargement (Normal CSF pressure (ICP) is 15 mm Hg. CPP = MAP-ICP should be maintained above 70 mm Hg).

The term active hydrocephalus is used when the intraventricular pressure is elevated. Active hydrocephalus may be:

- *Progressive:* Elevation of intraventricular pressure is sufficient to cause progressive ventricular enlargement and progressively worsening clinical syndrome of uncontrolled hydrocephalus.
- *Compensated:* Ventricles are enlarged but no longer progressively dilating despite moderate elevation of ICP and the absence of clinical signs.

Q. What is the pathophysiology of hydrocephalus?

Hydrocephalus is a dynamic disorder of CSF circulation. There is either a congenital or acquired imbalance between the production and absorption of CSF, resulting in accumulation of CSF and secondary ventricular enlargement. Obstruction to CSF flow is the hallmark of hydrocephalus. Obstruction to flow may occur at any point in the CSF pathway. Conditions that predispose hydrocephalus can be classified into three basic disease processes:

1. Congenital causes.
2. Space occupying lesions.
3. Inflammatory.

Congenital hydrocephalus typically presents during the neonatal period or in early of infancy. Common congenital anomalies of the CSF pathway include stenosis, occlusion or forking of the aqueduct of Sylvius, fourth ventricle outlet obstruction etc. In 80% of children with spina bifida, hydrocephalus usually results from a combination of Arnold–Chiari malformation and basal cistern arachnoid fibrosis.

Clinical features:

Symptoms: Poor feeding and lethargy, increased irritability, Gait disturbances, decreased level of consciousness, headache on awakening, and Diplopia.

Signs: Cranial enlargement and deformity, bulging anterior fontanel, dilated scalp veins "sunset sign", ptosis, strabismus, papilloedema and absent pulsation in retinal vessels.

Motor deficit of lower extremity: In the neonates, rapid head enlargement crossing percentile lines are the usual clinical findings. Most neonates develop hydrocephalus after birth. Most infants with hydrocephalus are asymptomatic. Late in the process they develop signs of irritability, decreased appetite, vomiting and poor head control. The usual clinical findings are of a full fontanel, dilated scalp veins and separate sutures. The usual clinical findings are of full fontanels, dilated scalp veins and separated sutures. The setting sun sign (paralysis of upward gaze and the prominence of the upper part of the sclera) is the late sign. Slow development of hydrocephalus in newborn is accompanied by increasing skull diameter to accommodate the increased in CSF volume and thus normal ICP is maintained. Rapidly developing hydrocephalus outpaces gradual skull growth and results in rapid elevation of ICP. Rapidly developing hydrocephalus outpaces gradual skull growth and results in rapid elevation if ICP and cerebral herniation unless it is treated. A tense anterior fontanel, irritability, vomiting and ophthalmoplegia suggest intracranial hypertension. Vomiting bradycardia, hypertension develop as late features of raised ICP and parallel the clinical signs. To confirm the diagnosis of hydrocephalus, following measures are undertaken:

- Serial recording of head circumference will show an increase of more than 1 cm every two weeks in the first three months
- CT scan and ultrasound can be used to assess ventricular size
- MRI will delineate the site of obstruction.

Normal changes in head circumference after birth:

0–3 months	2 cm/month
3–6 months	1 cm/month
6–9 months	0.5 cm/month
9–12 months	0.5 cm/month
1–3 years	0.25 cm/month
4–6 years	1 cm/year

Q. What is the surgical management of hydrocephalus?

Unless the etiology of hydrocephalus can be definitively treated, the treatment necessitates surgical placement of an extracranial shunts are of three types:

1. Ventriculoperitoneal shunt.
2. Ventriculoatrial shunt.
3. Ventriculopleural shunt.

Most children are now treated with a ventriculoperitoneal (VP) shunt. In absence of abdominal pathology, the VP shunt is preferred. The peritoneal catheter is tunneled and then inserted into the peritoneum in the right upper quadrant with spare tubing to allow the child to grow. This is a significant advantage of peritoneal cavity over the atrium for the sitting of the distal end of the shunt. Repeated or multiple previous abdominal procedures with subsequent adhesions may indicate that a ventriculoatrial (VA) shunt is the better option.

Q. What are the shunt complications?

Shunt complications include:

1. Underdrainage (malfunction).
2. Infection.
3. Overdrainage.

These children may present to the operating room multiple times for shunt revision and for shunt replacement surgery. Hydrocephalus children with VP or VA shunts frequently experience shunt malfunction or failure came to medical attention when intracranial pressure become elevated. Early symptoms of shunt malfunction are headache and irritability and later symptoms include lethargy, vomiting seizures and ophthalmoplegia (the downward gaze, i.e. the setting sun sign). Most commonly, the shunt malfunction occurs in the distal shunt tubing within the atrium or peritoneum or in the valve in the scalp. Shunt malfunction is usually detected by failure of the shunt reservoir to fill after decompression along with presence of symptoms and signs of raised intracranial pressure. If the malfunction is not in the proximal intraventricular portion of the shunt, neurosurgeon can place a needle in the shunt reservoir (which is easily palpable under the scalp) and drawn an aliquot of CSF, thereby lowering ICP. The maneuver may be life-saving.

Anesthetic technique depends upon whether intracranial hypertension exists. If so, no premedication is given and an IV induction with thiopentone and non-depolarizing muscle relaxant is ideal. Hyperventilation and avoidance of potent inhalation agent may be beneficial and help to control raised ICP.

Shunt and the pediatric general surgery: Endoscopic surgery is increasingly being used to treat abdominal conditions that previously required laparotomy. Endoscopic surgery requires the insufflation of the peritoneal cavity with carbons dioxide to a pressure of 30 cm H_2O, often combined with Trendelenburg position. The ability of various VP shunts to prevent

retrograde flow of carbon dioxide into the cranial cavity has not been tested. So, temporary occlusion of the peritoneal end during the procedure may be the safest option.

Q. What are the preanesthetic considerations in a child with hydrocephalus?

Children who present for CSF shunting procedure may exhibit a broad-spectrum of symptoms of clinical signs ranging from an apparently healthy child with minimal disability to a seriously ill, comatose child for whom surgery is urgent.

Preoperative considerations include assessment of:

- *Level of consciousness (which is an indicator ICP):* Children presenting for primary shunting shunt revision or malfunction may exhibit severe elevation of ICP that requires aggressive treatment. The increased ICP caused by shunt malfunction can be reduced acutely by tapping the proximal reservoir. The needle can be left in place to monitor ICP during induction.
- *Full stomach:* Evidence of vomiting suggests increased ICP and are indications to take precautions against aspiration of gastric contents, e.g. a rapid sequence induction. Placement of a nasogastric tube may precipitate coughing, bucking and increase in ICP.
- *Coexisting pathology:* Other organ system compromise. A child with cerebral palsy frequently aspirates.
- Age-related pathophysiology like anoxic spell, poor pulmonary compliance, immature renal and cardiac function.
- Drug history, especially of steroids and anticonvulsants.
- Surgical position required, to assess its impact on anesthetic management.

Q. What is the anesthetic management for surgical placement of extracranial shunt in hydrocephalus?

Anesthetic management depends on whether or not ICP is elevated. If there is no clinical evidence of elevated ICP, anesthesia can be induced by inhalational agents or IV drugs. Inhalation agents or IV drugs. Inhalation agent of choice is sevoflurane, nonirritant, cardiostable and stable cerebral hemodynamics. If there is increased ICP and delayed gastric emptying, rapid sequence induction is done with nondepolarizing muscle relaxant. Cricoid pressure is applied till the correct placement of endotracheal tube (ETT). The child is hyperventilated at low peak inspiratory pressure to minimize the risk of barotrauma. Since laryngoscopy is a potent stimulus for increasing ICP, an endotracheal tube is placed as smoothly and as rapidly as possible.

Maintenance:

- *Positioning:* Children are placed in supine position with the head turned or in a slightly lateral position. Children with increased ICP should be placed in a 30°head up position with minimal neck rotation or flexion to improve cerebral venous drainage. During intubation the main aim is to

stabilize the head and trunk. This can be achieved by elevating the trunk over a large pillow and supporting the head with a large head ring. Attempt should not be done to hang the child's head over the edge of the table as stability is poor.

- *Ventilation:* After the airway is secured, children with increased ICP are hyperventilated to a $PaCO_2$ of 25--30 mm Hg .Children with normal ICP are maintained at normocapnea. Controlled ventilation is beneficial because it reduces the risk of pneumothorax in ventriculopleural shunt. Controlled ventilation is mandatory in children with poor lung compliance and apneic spell and also when the cranium is open.
  In premature infants undergoing VP shunt placement, the inspired oxygen fraction should be limited to maintain PaO_2 and arterial oxygen saturation at 70 mm Hg and 95% to 97%, respectively. These levels minimize the risk of retinopathy of prematurity. In addition, the ventilation should be controlled with careful attention to inflation pressure, to minimize the risk of barotrauma. Measures should be taken to conserve body heat.
- *Anesthetic agents:* Anesthesia is usually maintained with N_2O, O_2, low concentration of isoflurane or sevoflurane or minimal narcotic supplementation. In patients with elevated ICP, halogenated anesthetics are used in low concentration or avoided entirely until the CSF is drained. Muscle relaxation is usually maintained with intermediate-acting muscle relaxant.
- *Fluid management:* Ventricular shunt operations are usually not associated with significant blood loss or third space losses. Fluid management centers around replacement of intravascular volume associated with emesis and drug-induced diuresis.
- *Maintenance of body temperature:* Body temperature may decrease during shunt procedures due to exposure of a large body surface area specially in ventriculoperitoneal shunt. Preparation solution may cause infant to cool rapidly. Use of an overhead warmer when anesthesia is commenced, conserves body heat. The limbs are covered, warm preparation solutions are used. Child is covered with sterile plastic drops which help to prevent cooling and evaporation.
- *Monitoring:* Precordial stethoscope, noninvasive blood pressure (NIBP), electrocardiogram (ECG), pulse oximeter, capnography, urinary catheter (required for long surgical procedure of is mandatory if osmotic diuretics are administered).
- *Emergence:* Adequate time for elimination of anesthetic agents and adequate reversal of neuromuscular blockade should be ensured before extubation of the trachea, suctioning of stomach contents before extubation in children suspected to have increased stomach contains may decrease the chance of regurgitation. The child should be fully awake and have adequate protective reflexes present before extubation. Hypothermic child should be rewarmed before extubation.
- *Postoperative management:* Supplemental oxygen should be given. Analgesics should be used judiciously in neurologically impaired

children. Infiltration of the skin incision sites with local analgesics at the time of closure substantially reduces the requirement of postoperative analgesia, children without preoperative neurological impairment can be given postoperative narcotics. Body temperature should be maintained. Premature infants less than 50 weeks postconceptual age requires cardiorespiratory monitoring to detect post operation apnea.

Chapter

20

Meningomyelocele

Q. What is spina bifida?

Spina bifida is a limited delete in the vertebral column, consisting absence of vertebral arches, through which the spinal membranes, with or without spinal cord tissue, protrude.

Spina bifida occulta: It is the congenital absence of posterior vertebral elements, including spinous process and laminae. Children are neurologically normal. It is common but asymptomatic. There is no herniation of meninges or neural elements through the vertebral defect. Superficial sign of this lesion may include a tuff of hair, cutaneous angiomas, lipoma or skin dimple.

Spina bifida with myelomeningocele: Congenital absence of posterior vertebral segment with protrusion of meninges through the defect of underlying neural structure abnormalities. Common findings are weakness and atrophy of the lower extremities, gait disturbances, urinary incontinence and deformities of the foot. Myelomeningocele arising from the high lumbar cord usually caused total paralysis and incontinence, although those arising from sacral cord may have only clawing of the foot and partial urinary function. Myelomeningocele patients always have 'Chiari II malformations' (abnormal downward herniation of the cerebellum and brainstem through the foramen magnum).

Treatment includes surgical closure of dura, fascia and skin over the defect.

Q. What is the classification of CNS malformation associated with spina bifida?

CNS malformations are clarified into different groups depending on the severity of the lesion. It is also important to define the level of the defect, i.e. cervical, thoracic, thoracolumbar, lumbar, lumbosacral or sacral.

- *Meningocele:* Herniation of coverings of the spinal cord (dura and pia mater and pia-arachnoid) through the bifid neural arch usually covered by dura and skin. The neural tube of fully developed with no neurological deficit.
- *Myelocele*: Open lesion with neural plate exposed. The central canal opens onto the surface at the upper end of the neural groove. The dura mater

is deficit and stops at the margin of the defect. The defects are usually thoracolumbar or lumbar and the neurological defect is often severe.

- *Meningomylocele or myelomeningocele*: The terms are synonymous. The dura mater and skin are deficient over the posterior aspect of the defect but the cord is still protected by the pia-arachnoid usually there is evidence of a neurological deficit. They are usually lumbar or lumbosacral in position. It constitutes primary neural tube defect, which is due to primary failure of closure of neural tube or possibly disruption of already closed neural tube between 18–28 days of gestation.
- *Encephalocele*: Herniation of brain encased is meninges through skull that forms an extracranial mass, is referred to as encephalocele. Most occurs over the convexity of the skull commonly in the occipital region.

Treatment: Excision of the herniated tissue and closure of the defect. Most patients with myelomeningocele of encephalocele have impaired cognitive development. Nasal encephalocele repair may require postoperative ventilation.

Q. What are the associated anomalies in myelomeningocele?

Associated anomalies are of two types:

1. Primary anomalies.
2. Secondary anomalies due to neurological dysfunction.

The primary anomalies are:

- Vertebral anomalies which may result in the deformities of kyphosis of kyphoscoliosis
- Arnold—Chiari malformation, resulting in hydrocephalus is almost a universal findin
- Congenital heart disease and other serious defects are relatively rare.

The secondary anomalies are:

- Motor (paralysis)
- Sensory (Loss of touch, temperature, etc.)
- Autonomic (sphincter paralysis of the urinary bladder and bowel)

Treatment includes surgical closure of the back defect depending on different criteria.

Q. What is the embryologic basis of these defects?

The development of neural tube occurs in two stages:

1. Primary neurulation.
2. Secondary neurulation.

Primary neurulation: The neural folds elevate and approximate each other, and start closing, thus forming the neural tube. The point of initial closure occurs at the caudal rhombencephalon or cranial spinal cord. The cutaneous ectoderm fuses first, followed by the neuroectoderm. The caudal neuropore closes between T_4 of S_2. Parallel to this process, the cutaneous ectoderm separates from the neuroectoderm to from the overlying skin, while the lateral

mesoderm migrates between the 2 ectodermal layers to form the posterior vertebral arches.

Secondary neurulation: This comprises further neural development occurring caudal to the caudal neuropore after the termination of conus medullaris from a poorly differentiated cell mass of the medial eminence. Because of differential growth between the vertebral column of the spinal cord, the conus becomes more rostral during later development.

Meningomyelocele constitutes primary neural tube defect, which is due to primary failure of closure of neural tube or possibly disruption of already closed neural tube between 18–28 days of gestation.

Q. What is myelodysplasia?

Myelodysplasia is a congenital failure of the neural tube to close, a process that normally occurs at 28 days of gestation. In its most common form, fusion fails in the middle or caudal neural groove, resulting in thoracic or lumbosacral meningomyelocele. When the site of failure is more cephaloid, encephaloceles result with meningomyelocele, the defective neural tissue is often in communication with the environment. Neurologic function very frequently is a similar phenomenon usually involving occipital skull of the upper cevical spine.

After closure of the neural tube, mesodermal and ectodermal structures complete the formation of the spinal column, skeletal muscles and skin of the back. A defect in this process may result in herniation of dural elements posteriorly producing a meningocele contains no neural tissue unlike meningomyelocele. Although neurologic function is normal in children with meningomyelocele, the spinal cord is often tethered (restrained with certain limit) caudally by the sacral neural roots. The tethered cord results in orthopedic or urologic symptoms in later childhood if it is not surgically corrected.

Myelodysplasia causes exposure of CNS tissue and places the child at risk of infection and death. The defect is repaired within 48 hours of birth. Delayed in surgical closure of the defect increases the likelihood of progressive neural damage of decreased motor function. For these reasons, myelodysplasia is regarded as a surgical emergency, and most neonates present for surgery in the first 24 hours of life.

Q. What is spinal cord? What is the vascular supply of it?

Spinal cord is the extension of CNS into the upper 2/3rd of the vertebral canal. It is 45 cm long in average adults, extending from the upper border of the atlas to the upper border of the 2nd lumbar vertebra. At the upper end, it is continuous with medulla oblongata and below with the conus medullaris, from which filum terminale descends as far as coccyx.

In the fetus, the spinal cord is the same length as the vertebral canal. In the first trimester the spinal cord extends from the foramen magnum to the end of spiral column. During growth and development of fetus, the vertebral

canal grows more rapidly than the spinal cord such that at birth the spinal card ends at the level of 3rd lumbar vertebra. In adults, the caudate tip of the spinal cord lies at the level of 1st lumbar vertebra. The nerve roots that pass out transversely in early fetal life become more oblique. In adults, the cauda equina consists of vertical lumbar and sacral nerves, bathed in CSF, that descend to meet their respective foramina.

Vascular supply: The spinal cord receives its vascular supply from 3 arteries, one anterior and two posterior. The anterior spinal artery, a single vessel lying in the substance of the pia mater overlying the anterior median tissue, arises at the level of foramina magnum from the function of a small branch from each vertebral artery. It receives communications from the intercostal, lumbar of other small arteries. It supplies the lateral columns, comprising 3/4th of the substance of the spinal cord. Thrombosis of this artery leads to anterior spinal artery syndrome.

The posterior spinal arteries, two on each side, branch from the posterior, inferior cerebellar arteries at the level of foramen magnum. They supply the posterior columns that carry fibers responsible for position, touch and vibration sense. Communicating branches at the level of 1st and 11th thoracic vertebrae, longer than the others, help to supply cervical of thoracic enlargement of spinal cord. The artery at 11th thoracic vertebra supplies the cord both upwards of downwards from this level.

Q. What are the coverings of the spinal cord? What are the spaces in between the meninges and their clinical implications?

The spinal cord is ensheathed by three membranes: Dura mater, arachnoid mater and pia mater. The dura mater begins at foramen magnum and end at S_2, fusing with the filum terminalae. The duramater is the outermost membrane. It is a strong fibrous sheath consisting of collagen and elastin fibers. The fibrous bands forming the structure of the duramater have hitherto been regards as longitudinal and clinical teaching has recommended insertion of spinal needle bevels parallel to the dural fibers and to separate rather than to cut the dural tissue. However, recent electron microscopy have revealed a more complex arrangement, dural fibers are actually arranged in a complex overlapping lattice, suggesting that position of the needle bevel has little influence on the size and shape of dural hole.

Within the cranium, the dura is composed of an outer end steal component that lies against the bone of cranium and an inner meningeal layer. Both layers are lightly adherent except where they divide to form venous sinuses.

Within the vertebral column, the dural layers separate. The inner meningeal layer of cerebral dura mater forms the spinal dura mater and cranial endosteal layer continues as periosteum lying in the vertebral canal, thus forming extradural space, closed off from the cranial vault. The extradural space communicates freely with the paravertebral space through the intervertebral foramina of caudally ends of the sacral hiatus.

Dilated extradural veins pose many problems:

- Risk of entering veins during epidural catheter insertion and inadvertent IV injection of the local anesthetics
- Decreases extradural space volume, causing more extensive spread of local anesthetics increasing the extent of block
- Increases the risk of local anesthetics toxicity due to greater absorption from.

Arachnoid mater is membrane of flat overlapping cells with tight junctions, closely applied to the dura mater. It presents the greatest barrier to drug transport from extradural space to CSF. The potential space between the dura and arachnoid is termed the subdural space. Occasionally, drug injected for epidural or subarachnoid space is injected into the subdural space. If suspected, the extradural catheter is removed, as migration through thin arachnoid mater into CSF can easily occur, which may precipitate total spinal anesthesia. Injection into subdural space during spinal anesthesia explains failed spinal.

Pia mater is a fenestrated single layer of epithelial cells lightly adherent to spinal cord and sends delicate septa into its substance. Pia mater is separated from the arachnoid by CSF within the subarachnoid space, which is a continuation of ventricular system CSF at the base of the brain. CSF lies between arachnoid and piamater within the spinal canal.

Q. How intrauterine and postnatal diagnosis of myelomeningocele is made?

Intrauterine diagnosis of myelomeningocele is made by laboratory investigations of imaging studies.

The laboratory investigations are:

- Elevated maternal serum alpha-fetoprotein level in second trimester Amniotic fluid alpha-fetoprotein assay
- Presence of acetylcholinesterase (a nerve-specific enzyme) in amniotic fluid.

Imaging studies: Fetal ultrasound at about 18th week gestational age. Postnatal diagnosis of myelomeningocele is immediately obvious at birth. After delivery MRI of spine helps accurate assessment. Serial cranial ultrasounds, CT scan and MRI of brain are after needed in cases of associated hydrocephalus.

Q. What are the surgical considerations in a child with myelomeningocele?

Surgery in-utero is now available at many centers and the benefit of early correction has been evaluated at these centers.

When intrauterine diagnosis has been done but in-utero correction has not been done, the pediatric team should be present to do corrective surgery immediately after delivery which should always be by cesarean section.

Early closure of myelomeningocele defect is not associated with improvement of neurological function but leads to lower infection rate and

arrests progress of neurological damage. The defect should be closed within 24 hours whether or not membrane is intact.

In children with clinically overt hydrocephalus at birth, myelomeningocele repair of Ventriculoperitoneal (VP) shunt may be done in the same sitting without increased risk of infection. If VP shunt is not performed an increased risk of myelomeningocele repair breakdown is present.

Q. What are the preanesthetic considerations in a child presented for surgical correction of myelomeningocele?

- Congenital lesions in the central nervous system, unlike other type of congenital anomalies are generally not associated with an increased incidence of associated anomalies of other organ systems. Especially, the frequency of congenital heart disease is not increased in children with CS lesions. Therefore, routine preoperative screening need not include a cardiologic evaluation.
- Most children with myelomeningocele also have an associated Arnold-Chiari malformation, of hydrocephalus ultimately develops, necessitating a CSF shunt surgery.
- Meningocele and myelomeningocele usually are repaired within the first day of life to minimize bacterial contamination of the exposed spinal cord, and subsequent sepsis, which is the most common cause of death in this population during the newborn period.
- These infants rarely exhibit increased Intracranial pressure (ICP). They can have a variety of neurological deficits depending on the level of the lesion.
- Age related pathophysiology should be given considerations, especially presence of prematurity.
- Airway management is problematic due to positioning of the child giving protection of the neuroplaque.
- As there is high third space losses from the skin defect, volume status of the infant should be carefully assessed and corrected.
- Potential for hypothermia due to exposure of large body surface area and loss of third space fluid. Measures should be taken to prevent hypothermia.

Q. What is the anesthetic management of surgical correction of myelomeningocele in an infant?

Positioning the child during induction of intubation is a major concern. Intubation of the trachea may pose a challenge to the anesthesiologist. Anesthesia for infants with thoracolumbar or thoracic myelomeningocele can be induced either in left lateral position with head in the midline by an assistant or supine position with neural sac protected by a cushioned ring. The shoulder of head of the infant may require additional elevation.

In newborn, in which difficult airway is suspected, an awakened intubation may be performed after atropine premedication (20 mg/kg, minimum dose 0.1 mg) and preoxygenation. Otherwise, the induction may proceed by mask inhalation of halothane or intravenous administration of this thiopentone or

propofol and muscle relaxants. Ether a nondepolarizing muscle relaxant or succinylcholine (SCh) can be used safely.

Maintenance of anesthesi: After tracheal intubation of proper fixation of the endotracheal tube, the infant is torched to prone position. Injury to the exposed neural tissue is prevented. Chest of hip rolls are placed to ensure that the abdomen is free, to facilitate ventilation and to reduce intra-abdominal pressure and decrease bleeding from epidural plexus.

Since most of the infants have an Arnold-Chiari malformation, excessive rotation of the neck should be avoided. The extremities should be in a relaxed position be well-padded. Anesthesia can be maintained with halothane or isoflurane with or without nitrous oxide. Higher dose of narcotics and ketamine may cause postoperative apnea. Nondepolarizing neuromuscular blocking agents may be used if surgeon does not expect to use nerve stimulator to help identify and space neurologic tissue. The lungs are mechanically ventilated avoiding barotrauma in the immature lungs. Premature infants (especially those <32 weeks' gestation) are at increased risk of retinopathy of prematurity and lung injury from prolonged exposure to high oxygen concentration. Therefore inspired O_2 fraction should be limited to maintain PaO_2 and SPO_2 at 70 mm Hg and 95% to 97%, respectively. Routine monitoring is necessary during anesthesia. Blood loss during myelomeningocele repair is usually not excessive, averaging about 30 mL or 10% of the blood volume of the infant. But incidious blood loss occurs especially if neural sac is large and significant undermining of skin, relaxing incisions or skin grafting is required for closure of the defect. Blood transfusion may be necessary. Conservation of body heat is important for infants with myelomeningocele, particularly because automatic control below the level of the defect is abnormal. The operating room should be warmed to 27°C (80°F) during surgery. Radiant heat lamps are used during positioning and skin preparation until the baby is draped and again at the end of surgery. Humidification of inspired gases will further prevent heat loss, as well as, minimize pulmonary complications.

The large areas of exposed tissue liberal use of cold surgical preparation solution increase the risk of hypothermia in these infants.

Emergence: The considerations are-:

- Complete elimination of anesthetic agents
- Complete reversal of neuromuscular blockade
- Assessment of airway patency: Neonates at risk of apnea after anesthesia, should be extubated fully awake.

Postoperative management: Similar as in hydrocephalus.

Chapter

21

Cataract

Q. What is the crystalline lens? What is its function?

The crystalline lens is a biconvex structure approx 4 mm thick and 9 mm in diameter, situated behind the iris, being suspended in position from ciliary body by zonules, it is transparent, colorless, and avascular. Anteriorly it is in contact with the aqueous humor through the pupil and posteriorly lays the vitreous.

Function

The sole function of the lens is to focus the rays of light on the retina. When viewing an object at a distance, the ciliary muscles relax, causing tightening of the zonules and decreasing the AP diameter of the lens and parallel rays of light pass through it to get focus on the retina. When viewing a near object, the ciliary muscle contracts and the zonules relax leading to a decrease in tension of the lens capsule and modules the elastic lens in to a more spherical form. Hence, the rays from a near object get focused onto the retina. This mechanism is known as accommodation.

Q. What is accommodation? What is the range and amplitude of accommodation?

Accommodation is the process of change in the focus of eye by change in the refractive power of the crystalline lens. It is a process which enables us to see objects clearly at various distances from the eye. The two factors responsible for it are:

1. Ability of the lens to alter the shape
2. Power of the ciliary muscle.

There are two types of accommodation:

1. Physiological
2. Physical.

Physiological accommodation is related to ciliary muscle and depends on a contractile power. In nerve palsies, at any age (3rd cranial nerve), the power of ciliary muscle fails and so does the accommodation.

Physical accommodation is related to the crystalline lens and is an expression of its actual physical deformation. When lens become hard,

accommodation fails and is a manifestation at around 40 years of age (Presbyopia).

Presbyopia is the physiological aging process when the near point gradually recedes beyond the normal reading or working distance leading to presenting feature of gradual difficulty in reading small prints and inability to perform near work meticulously.

Treatment: Use of appropriate convex lens, so that accommodation is reinforced and the near point is brought within workable distance.

Range of accommodation

- The far point is the furthest point at which object can be focused on the retina with ciilary muscle relaxed, decreasing the AP diameter of the lens.
- The near point is the nearest point at which small objects can be seen clearly due to contraction of the ciliary muscle making the shape of the lens more spherical.
  The range of accommodation is the difference between these two distances.

Amplitude of accommodation

It is the difference between the refractive power of the eyes between far point and near point.

Q. What is cataract? What is its classification?

Any opacity of the lens or its capsule causing visual impairment is called cataract.

Classification of cataract is:

- Congenital or developmental cataract: Present at birth or develop soon after birth.
- Acquired cataract: These are of various forms:
  - Senile cataract: The aging lens tends to become opaque after the age of 50 years. This is divided into two types: Cortical or soft, nuclear or hard cataract.
  - Traumatic cataract
  - Complicated cataract (due to some ocular disease)
  - secondary cataract (due to some systemic disease like DM)
  - Toxic cataract (due to drugs)
  - Syndrome associated cataract (Down's).

Cataract can also be classified as follows:

- According to etiology: Senile
- According to age: Congenital
- According to location of opacity: Nuclear
- According to progress.

Q. What is senile cataract? What are the types and its symptoms?

The aging lens tends to become opaque after the age of 50 years leading to senile cataract.

Senile cataract may be of two types:
1. Cortical or soft
2. Nuclear or hard.

Manifestations: Early symptoms are spots before the eyes and blurring of vision. Frequent changes of glasses, with progress there is decreased vision.

Q. What is the management of senile cataract and what are the indications of surgery?

Only treatment is surgical removal of lens.

Indications of surgery:
- For visual improvement
- For secondary complications like glaucoma
- For therapeutic reasons (fundal view, retinal detachment in diabetic retinopathy needing photocoagulation)
- For cosmesis.

Q. What are the surgical techniques used for removal of lens?

Main techniques used for extraction of lens are:
- ECCE (Extracapsular cataract extraction)
- ICCE (Intracapsular cataract extraction)
- Phacoemulsification.

Q. What anesthetic techniques are used for surgery?

Most ophthalmic operations can be performed under local anesthesia with or without premedications. General anesthesia is indicated in children, apprensive patients and mentally ill patients. In intraocular operations, anesthesia as well as temporary akinesia is needed. Akinesia includes functions of both the orbicularis oculi muscle and all the extraocular muscles, so that squeezing of the lids and movement of the eyeball are prevented during surgery.

Retrobulbar block leads to decrease in IOP, anesthesia of the deeper structures (iris) and total external and internal ophthalmoplegia.

Retrobulbar block: One injection of local anesthetic solution at the junction of middle one-third and lateral one-third of inferior orbital margin.

Peribulbar block: Two injections of the LA solutions are administered, one injection at the junction of the medial two-thirds and lateral one-third of the lower lid, another injection at the junction of the medial one-third and lateral two-thirds of the upper lid.

Facial block: One injection of LA solution given at the neck of mandible just below the condyle.

Q. What is congenital cataract and what are the surgical procedures done for it?

Congenital or developmental cataract presents at birth or develops soon after birth.

Surgical procedures are:
- Simple dissection or needling operation: Not used now
- Pars plana lensectomy with vitrectomy is most suitable
- Planned ECCE may be considered after age of three years.

Q. What is diabetic cataract? How it is formed and treated?

DM is associated with two types of cataracts: (i) early onset senile cataract, (ii) true diabetic cataract—occurs in poorly controlled juvenile DM patients with gross disturbances in water balance.

In diabetic, cataract opacities are bilateral and cortical, predominantly involving anterior and posterior subcapsular region. The opacities consist of minute white dots of various sizes like snowflakes and are usually called snowstorm cataract. It may progress to complete maturity in less than 72 hours.

Mechanism: Excess of glucose in the lens→ metabolized to sorbitol→ increased osmolality of lens→ formation of vacuoles in the cortex→ opacification.

This cataract is reversible in the early stage but becomes irreversible after coagulation of protein.

Treatment: Surgical extraction of lens.

Q. What is subluxation of lens? How it is treated?

Subluxation of lens is the condition of the lens in which a portion of the supporting zonules is absent and the lens lax support in the quadrant. Lens remains in the pupillary area with tilting or displacement in any meridian.

Treatment: If the lens remains clear without symptoms, glasses are prescribed against phakic part to correct lenticular myopic astigmatism. If not clear and associated with symptoms, extraction of lens is done.

Q. What is dislocation of the lens and how it is treated?

In dislocation of the lens, the crystalline lens is completely unsupported by the zonules and is completely displaced from the pupillary area.
- In anterior dislocation, treatment includes immediate intracapsular extraction of the lens and vitrectomy with or without implantation of anterior chamber IOL.
- In posterior dislocation, treatment includes:
  - If no inflammatory signs, the lens is kept as such and only aphakic glasses prescribed.
  - If there are complications, the lens has to be extracted (lensectomy) along with vitrectomy, a scleral fixation IOL can be implanted by an expert.

Q. What is pupil and what are its functions?

Pupil is an aperture present in the center of the iris. Normally it is almost circular in shape. Examination of the pupil includes size, shape, position and reaction.

Functions

- The pupil limits amount of light reaching retina and protects against excessive bleaching of the visual pigment
- It controls the chromatic and spherical aberration in retinal image
- It helps in light and dark adaptation, i.e. minimizes visual acuity at different light level. Large pupil allows more light and greater acuity in deemed illumination. Smaller pupil allows less light and greater acuity in bright illumination.

Q. What are the pupillary reflexes?

- *Light reflex*: When light is held in front of the eye both pupils constrict and the change in size and speed of the change are observed. The pupil to which light is shown is called direct light reflex and that of the other pupil is called indirect reflex. To access indirect reflex, two eyes are dissociated with an occluder. Light is shown to one eye, observing the change of size and speed of change in the other eye.
- *Near reflex*: Changes in the size of the pupil and speed of the change observed with change of fixation from distance to near and from near to distance. Fixation from the distance to near consists of increased accommodation, convergence of visual axis and constriction of pupil.
- *Darkness reflex*: When someone goes from lighted environment to darkness, the pupil dilates due to abolition of light reflex with relaxation of sphincter pupillae and constriction of the dilator pupillae.
- *Lid closure reflex*: Lid closure may be accompanied by pupillary constriction or dilatation. Constriction of the pupil with blinking and dilatation with lid closure on touching the cornea.
- *Psychosensory reflexes*: Dilation of the pupil in response to sensory and psychic stimuli, seen in infants.

Q. What are miotics and what are mydriatics?

Drugs which constrict the pupils are known as miotics. Two groups—parasympathetic and sympathetic.

- Parasympathetic includes drugs such as pilocarpine, physostigmine (reversible) and ecothiopate (irreversible).
- Sympathetic drugs: Alpha adrenergic blockers like phenoxybenzamine and tolazoline.
- Other miotics are histamine, morphine.

Drugs which dilate the pupil are known as mydriatics. Two groups—parasympathetic and sympathetic.

- Parasympathetic drugs include atropine, tropicamide which are quick and short-acting. Homatropine which is quicker than atropine.
- Sympathetic drugs include adrenaline, phenylephrine and ephedrine.

Q. What is IOP? What are the characteristics, functions and factors responsible for short-term changes in IOP?

The pressure exerted by the intraocular contents on the coats of eyeball is known as IOP. Normal IOP is maintained by an outflow and episcleral venous pressure. IOP is distributed evenly throughout the eye, so that the pressure is always the same in the posterior vitreous as it is in the anterior humor.

Normal IOP varies between 10.5 and 20.5 mm Hg with mean pressure of 15.5 mm Hg. IOP is created by aqueous formation .Normal IOP is pulsatile, reflecting its vascular origin and effect of blood flow on the internal ocular structures. IOP is dynamic, any single measurement being just a momentary sample. IOP is important in maintaining the shape of the eyeball and also the optical integrity. It senses a tissue pressure of the vascularized internal structure of the eye and is much higher than tissue pressure elsewhere in the body (5 mm Hg).

Factors responsible for short-term changes in the IOP:

- Arterial pressure (BP): No effect by physiological changes, sudden large swings may affect IOP.
- Systemic venous pressure: Changes in SBP cause profound effect on IOP (compression of SVC).
- Mechanical pressure on the globe from outside.
- Blood pH: Acidosis lowers IOP.
- Hyperthermia increases IOP and hypothermia decreases.
- Effect of general anesthesia:
  - Hypercapnea and hypoxia increase IOP
  - Hypocapnea and hyperoxia decrease IOP.
  - Premedications (diazepam, morphine) decrease IOP.
  - Thiopentone and volatile anesthetic agents reduce IOP.
  - Ketamine increases IOP.
  - Parenteral atropine does not increase IOP.
  - Succinyl choline causes rise in IOP.

Q. How examination of the fundus is done and what is observed?

Examination of fundus is done by ophthalmoscopy after dilatation of the pupil. Ophthalmoscopy are of two types:

1. *Direct:* Hand-held direct ophthalmoscope provides direct magnified view of the fundus.
2. *Indirect:* Used by specialist, with a head mounted prism directed light source coupled with use of a high condensing lens.

Fundal examination includes:

- Fundal glow—good, poor or absent.
- Optic disc—margin, color, shape, venous pulsation or any abnormal vessels.

- Retinal vessels—vascular reflexes, arteriovenous reflexes, arteriovenous crossing and any abnormal or new vessels.
- General fundus—abnormalities like exudates, hemorrhage, patch, tumor, new vessels, etc.
- Macula and foveal reflex—abnormalities like cyst, hole, scar, edema, hemorrhage, etc.
- Choroidal blood vessels—normally not visible; in sclerosis, they appear like ribbons.

Q. What is papilledema and what are the ophthalmoscopic features of it?

Papilledema is bilateral, non-inflammatory, passive swelling of the optic disc due to raised ICP and malignant hypertension. Ophthalmoscopic findings are different in different stages:

- *Early papilledema*: Earliest change is blurring of the superior and inferior margins of the disc.
  Disc hyperemia and dilated capillaries, absent spontaneous venous pulsation, sphincter hemorrhage at or just of the disc margin. Normal optic disc cup is preserved.
- *Established papilledema*: Disc margin indistinct and central cup obliterated. Disc surface is elevated above retinal plane. Venous engorgement and peripapillary edema, flame shaped hemorrhage and cotton wool spots, radiating folds around macula (macular star) are manifested.
- *Chronic papilledema*: Central cup obliterated, hemorrhagic and exudative components gradually resolve. Optic disc appears like 'champagne cork'.
- *Atrophic papilledema*: Retinal vessels attenuated with perivascular sheathing. Dirty white optic disc due to reactive gliosis leading to secondary optic atrophy.

Q. What are the different parts of eye?

The exposed part of eye consists of a central transparent portion, cornea; surrounding opaque portion, the sclera; and their junction area, the limbus. This part is protected by eyelids .These eyelids also limit the amount of light entering the eye and help to distribute tears over the surface of eyeball. The movements of the eye are controlled by six extraocular muscles (four reti muscles and two oblique muscles). The reti muscles have their insertion anterior to the equator and the oblique muscles insert into the sclera, post to the equator. Ciliary muscles, sphincter and dilators of iris form the intrinsic muscles.

The globe consists of three concentric layers (Tunica) and three chambers.

Concentric layers are:

1. Outer supporting layer (tunica).
2. Middle vascular layer (uvea).
3. Inner neural layer (retina).

Crystalline lens: A transparent structure located immediately behind the iris and supported by zonules, sole function is to focus the rays of light into the retina.

Chambers of eyeball are:
1. Anterior chamber.
2. Posterior chamber.
3. Vitreous cavity.

Cornea: Central, transparent part of tunica, constitutes the anterior one-sixth of the eye. It is avascular but the corneoscleral limbus is supplied by branches of anterior ciliary arteries forming perilimbal plexus. Nerve supply is by long and short ciliary branches of ophthalmic division of 3rd cranial nerve.

Sclera: Dense tuff fibrous envelope covering posterior 5/6th of the eyeball.

Limbus: Transitional zone between cornea and sclera.

Uvea: Three parts:
1. Anterior—iris with a central opening, pupil.
2. Intermediate—ciliary body.
3. Posterior choroid.

Retina: Membranous light sensitive coat of eyeball (outer pigmented layer and inner sensory layer).

Blood supply: Outer portion by choriocapillaries. Inner portion by central retinal artery.

Conjunctiva: A transparent mucous membrane covering the inner surface of eyelids.

Parts: Palpebral (lining the lids), bulbar (over sclera, movable), fornix (cul-de-sac).

Blood supply: Anterior ciliary artery, lacrimal artery and palpebral branch of nasal artery.

Nerve supply: Ophthalmic division of 3rd cranial nerve.

Eyelid: Thin curtain of skin, muscle, fibrous tissue and mucous membrane. Muscles are orbicularis oculi, Muller's muscle (upper and lower) and LPS.

Q. What are the blood supply and nerve supply of the eyeball?

Nerve supply:

- *Visual-optic nerve*: Optic nerve→ optic chiasm→ optic tract→ lateral geniculate body→ optic radiation→ visual cortex.
- Sensory-trigeminal via ophthalmic and maxillary branch.
- *Motor*: **Occulomotor nerve** having 3 divisions namely superior, inferior and short root of ciliary ganglia supplying all extraocular muscles except superior oblique (**trochlear nerve**) and lateral rectus (**abducens nerve**).
- *Mixed motor and sensory*: Facial nerve (motor to orbicularis oculi, secretory to lacrimal glands).

- *Autonomic supply*: (i) Parasympathetic: Oculomotor nerve to inferior oblique, ciliary, sphincter pupillae muscles and ciliary ganglion, facial nerve to lacrimal gland. (ii) Sympathetic supply: Posterior ganglionic fibers from superior cervical ganglion enter the ciliary body and iris to supply dilator pupillae muscle.

Blood supply: Mainly from the ophthalmic artery.

Q. What anesthetic techniques are used for ophthalmic surgeries?

Most of the ocular surgeries in adults can be performed under local anesthesia (LA), as the nerve supply to the eye is easily accessible. LA can be given in two forms:

1. *Topical*: Used for superficial procedures on conjunctiva and cornea, lignocaine and amethocaine are used.
2. *Infitration*: Subconjunctival injection, retrobulbar injection, peribulbar injection, local injection into lids and lacrimal apparatus and facial nerve block. Drugs used—lignocaine and bupivacaine. Addition of hyaluronidase encourages the spread of anesthetic solution and shortens the onset of action. Mixture of lignocaine and bupivacaine can be used for dual advantage of rapid onset and longer duration of action.

General anesthesia is indicated in children, apprehensive patient and mentally ill patients. In intraocular operations, anesthesia as well as temporary akinesia is needed. Akinesia includes function of both orbicularis oculi and all the extraocular muscles so that squeezing of lids and movement of eyeballs are prevented during surgery.

Chapter

22

Strabismus

Q. What is strabismus?

Normally the visual axis of two eyes is essentially parallel in all direction of ocular movement. The visual axis of both eyes is directed simultaneously to the same object in space, so that the image of the object falls on the fovea of each eye.

Strabismus is genetic term applied to those conditions in which the eyes are so positioned that the visual axis of both eyes is not directed simultaneously to the same object in space and the image of the object falls upon the fovea of one eye but not on the other causing deviating or squinting eye. In squint, one of the visual axes is directed towards the fixation object and the other deviated from this point.

Q. What are the mechanisms for ocular alignment? What is visual pathway?

Two mechanisms are involved in ocular alignment:

1. *Sensory:* Formation of image in the eye and is central perception.
2. *Motor:* Movement of the eye by various extraocular muscles.

An abnormality in either of the two leads to faulty alignment of the eye or strabismus.

Visual pathway: Each of the eyeballs perceives the image and relays the sensations to the brain (occipital cortex) via the visual pathway. It consists of:

- *Optic nerve*: Starts from the optic disc and extends up to optic chiasm where the two nerves meet.
- *Optic chiasm*: Lies over diaphragma sellae, so visual field defects occur in a patient with pituitary tumor.
- *Optic tracts*: Cylindrical bundle of nerve fibers running outward and backward from optic chiasm. Each tract contains fiber from temporal half of the retina of same eye and nasal half of the opposite eye. Each tract ends posteriorly in lateral geniculate body.
- *Optic radiations*: Extent from lateral geniculate body to visual cortex on medial aspect of occipital lobe.

Q. What are the extraocular muscles? What are their nerve and blood supply?

A set of 6 extraocular muscles (4 recti and 2 oblique) control the movements of each eye. Recti are: Superior rectus, inferior rectus, medial rectus and lateral rectus. The oblique muscles include superior and inferior oblique.

Four rectus muscles originate from a common tendinous ring which is attached to the apex of the orbit, encircling the optic foramina and medial part of the superior orbital fissure. All four recti run forward around the eyeball and inserted into the sclera at different distance from the limbus.

Superior oblique muscle arises from the bone above and medial to optic foramen, moves forward between the roof and medial to the wall of the orbit, to be inserted on the upper and outer part of the sclera behind the equator. Inferior oblique muscle arises by rounded tendon from a shallow depression on the orbital plate of maxilla. It passes laterally and backward to be inserted by a short tendon in the lower and outer part of sclera behind the equator.

Superior oblique is longest and thinnest muscle and inferior oblique is shortest muscle. All muscles are ensheathed by a fascia which is continuous with tenon's capsule near the point of insertion. Fascial condensation to orbit is also present which serves as check ligaments to limit ocular rotation.

Nerve supply: Ocular muscles are supplied by third, fourth and sixth cranial nerves. Third cranial nerve supplies the inferior oblique and superior, inferior and medial recti muscles, entirely motor in function. Fourth cranial nerve innervates superior oblique muscle, entirely motor in function. Sixth cranial nerve innervates the lateral rectus, entirely motor in function.

Blood supply: Vascular supply is by muscular artery (medial and lateral) which are branches of ophthalmic artery. Medial muscular artery supplies medial and inferior rectus muscle and inferior oblique muscle. Lateral muscular artery supplies lateral and superior recti and superior oblique muscle.

Q. What are the actions of extraocular muscles?

There are three categories of action in primary position of gaze—primary, secondary and tertiary action.

Muscle	Primary action	Secondary action	Tertiary action
Medial rectus	Adduction	–	–
Lateral rectus	Abduction	–	–
Superior rectus	Elevation	Intorsion	Adduction
Inferior rectus	Depression	Extorsion	Adduction
Superior oblique	Intorsion	Depression	Abduction
Inferior oblique	Extorsion	Elevation	Abduction

Q. What are the signs and symptoms of squint?

- Usually no symptoms.
- No limitations of eye movements. Visual acuity is poor.
- Amblyopia denotes a unilateral or a bilateral reduction in form vision without any detectable organic ocular lesion (reduction in visual acuity). Strabismic amblyopia develops in a squinting eye and persists when deviated eye is forced to fixate.

Q. What is the aim of treatment for squint? What are the procedures involved in the treatment?

Aim of treatment for squint is to make the eye straight and ensure binocular single vision, which indicates coordinated use of two eyes to produce single unifixed image in three dimensions.

Procedures involved in the treatment are:

1. Optical correction of refractive error by suitable spectacles.
2. Occlusion therapy to treat the amblyopia. Aim is to occlude the sound eye and to force the use the amblyopic eye.
3. Orthoptic exercises (defective binocular vision)
   Above are the initial steps to restore binocular single vision earlier before surgery.
4. Operative measures—undertaken when there is no improvement of vision in the squinting eye after initial occlusion therapy.

Q. What is oculocardiac reflex? How it is treated?

Oculocardiac reflex is a decrease in pulse rate associated with traction applied to extraocular muscles and/or compression of the eyeball. The reflex is mediated by nerve connections between the ophthalmic branch of trigeminal nerve via ciliary ganglion and vagus nerve of parasympathetic nervous system. Nerve fibers from the maxillary and mandibular division of trigeminal nerve have also been documented. These afferent synapses with the visceral motor nucleus of the vagus nerve from the cardiovascular center of the medulla to the heart, increased stimulation of which leads to decreased output of the SA node. This reflex is especially sensitive in neonates and children, particularly during strabismus correction surgery. However, this reflex may also occur in adult. Bradycardia, junctional rhythm and asystole, all of which are life-threatening can be induced through this reflex.

Treatment: The reflex can be blocked by injecting peribulbar or retrobulbar LA prior to stimulation or with intravenous injection of anticholinergic agents such as atropine or glycopyrrolate.

If bradycardia occurs, removal of the stimulus is immediately indicated, which after results in restoration of normal sinus rhythm of the heart. If not, the use of anticholinergics will usually be successful and permit continuation of surgical procedure. In extreme cases, such as asystole, CPR is required.

Q. What is force duction test?

It is used to differentiate defective ocular movements due to physical restriction, from a muscle paralysis, done under topical anesthesia. Insertion of affected muscle is grasped with forceps and gently attempted to rotate the eyeball in the field of action of the weak muscle.

FDT positive—means difficulty to move the globe with forceps (contracture of muscle).

FDT negative—in case of muscle paralysis.

Q. What are the types of squint?

Types

- *Apparent squint (pseudo strabismus)*: Visual axes of the two eyes are parallel in all direction of ocular movements, but the eyes seem to have squint.
- *Latent squint*: Tendency of the eyes is to deviate but is prevented by fusion mechanism for the interest of binocular single vision.
- *Paralytic squint:* Misalignment of visual axes of two eyes, as a result of paresis or paralysis of one or more extraocular muscles.

Chapter

23

Diabetic Foot Complications

Q. What is diabetes mellitus? What are the types of diabetes mellitus?

Diabetes mellitus is characterized by metabolic dysregulation, mostly that of glucose metabolism, accompanied by long-term vascular and neurologic complications.

Diabetes mellitus has several clinical forms, each with distinct etiology, clinical presentation and course.

1. Insulin dependent diabetes mellitus (IDDM)
   - Occurs most often in patients younger than 20 years of age
   - Patients are thin and prone to keto-acidosis
   - Absolute insulin deficiency
   - Anti-islet cell antibodies
   - Presenting feature:
     - Hyperglycemia
     - Polyuria
     - Polydipsia
     - Weight loss.
2. Non-insulin dependent diabetes mellitus (NIDDM)
   - Usual onset after 40 years of age
   - Patients are obese and with sedentary lifestyle and resistant to keto-acidosis
   - Insulin resistance often, in presence of adequate insulin secretion
   - Presenting feature same as IDDM
   - Fasting plasma glucose >140 mg/dL
   - Abnormal glucose tolerance test.
3. Gestational diabetes mellitus
   - Usual onset 24–30 weeks of gestation
   - Glucose intolerance which corrects after delivery
   - Increased perinatal complications
   - NIDDM develops within 10 years (in 30–50% of cases).
4. Secondary diabetes mellitus
   - Pancreatic disease
   - Cushing's disease
   - Acromegaly

- Drugs (glucocorticoid)
- Diagnostic criteria same as IDDM.

Q. What are the complications of diabetes mellitus?

The most serious acute complication of diabetes mellitus is ketoacidosis.

Hypoglycemia in IDDM is more common than ketoacidosis and potentially as dangerous as ketoacidosis. Late complications of diabetes mellitus may manifest as macrovascular events (coronary artery disease, cerebrovascular disease, and peripheral vascular disease), microvascular events (retinopathy, nephropathy) and disorders of the nervous system (autonomic nervous system neuropathy, peripheral neuropathy), stiff joint syndrome and diabetic scleroderma.

Q. What are the signs and symptoms of diabetic ketoacidosis? How is it treated?

Signs and symptoms

- Nausea, vomiting
- Dehydration, hypotension, tachycardia
- Kussmaul's respiration—usually deep, rapid respiration —'air hunger' due to metabolic acidosis
- Abdominal pain
- Somnolence or coma.

Laboratory findings:

- Hyperglycemia
- Metabolic acidosis
  - Arterial pH <7.3
  - Bicarbonate <15 mEq/L
- Ketones present in urine and blood
- Hyperosmolality, hypokalemia
- Increased blood urea nitrogen and creatinine.

Treatment of diabetic ketoacidosis

- Tracheal intubation if CNS depression is present
- Administration of IV regular insulin (10 units followed by 5–10 units/hour)
- Restoration of intravascular fluid volume (normal saline 5–10 mL/kg/hour IV and 5% glucose when blood glucose concentration decreases to <250 mg/dL)
- Restoration of total body potassium (0.3–0.5 mEq/kg/hour IV)
- Monitoring of blood glucose, serum potassium, arterial pH and urine ketones)
- Identification of cause (infection, myocardial infarction, poor compliance).

Q. What are the signs and symptoms of hypoglycemia? How is it treated?

Signs and symptoms of hypoglycemia reflect activation of sympathetic nervous system (diaphoresis, tremulousness and tachycardia), neuroglycopenia due

to the effect of insufficient glucose delivery to the brain (impaired cognition, confusion, headache, irritability, seizures, unconsciousness).

Patients should recognize the episodes of hypoglycemia and promptly treat themselves by oral ingestion of rapidly absorbed carbohydrates (50 g is sufficient and provided by 180 mL orange juice).

If patient cannot swallow, treatment is intravenous administration of 25 mL of 50% glucose.

Glucagon, 1 mg IM or IV is an alternative to IV glucose and produces response in 15–30 minutes.

Q. What is diabetic foot? What are the most serious foot complications in diabetes mellitus?

A diabetic foot is a foot that exhibits any pathology that results directly from diabetes mellitus or long-term (chronic) complications of diabetes mellitus. The most serious foot complications in diabetes mellitus are ulceration, infection and neuropathic osteoarthropathy.

Q. What are the causes of diabetic foot?

Foot complications are common in people with both diabetes I and II. In diabetes mellitus, high blood sugar levels over time can damage nerves, blood vessels, eyes and kidneys. It can also decrease body's ability to fight infection. Foot problems develop and can quickly become serious.

Organ damage and impairment of immune system result when diabetes in not well controlled. Though treatment of diabetic foot problems has improved, good control of blood sugar level remains the best way to prevent diabetic complications.

Foot problems in diabetic patients result from interaction of a number of causes:

- *Peripheral neuropathy*: Loss of sensation in feet results in injuries to go unnoticed leading to ulceration.
- *Foot deformity*: Results in formation of calluses on weight bearing areas of feet due to Charcot's joint associated with peripheral neuropathy.
- *Trauma*: Small, repetitive trauma of feet goes unnoticed due to peripheral neuropathy.
- *Arterial insufficiency*: Poor blood flow results in impaired wound healing and increased risk of infection.
- *Impaired resistance to infection*: Results in severe infection and gangrene.

With damage to the nerves due to neuropathy, a person with diabetes mellitus may not be able to feel his/her feet properly, leading to abnormal pressure on the skin of the foot and sores may develop.

Damage to blood vessels and impairment of immune system make it difficult to heal wounds. Bacterial infection of the skin, connective tissues, muscles and bones can occur, leading to gangrene. Antibiotics cannot reach the site of infection due to poor blood supply and often the only treatment is amputation of the foot or leg.

Contributing or predisposing factors for foot problems:

- Poor vision, especially those with diabetic retinopathy, may result in falls and foot injury. Poor vision also causes difficult toe-nail management. Deformed, overgrown nails result in skin trauma.
- Comorbidities frequently result in reduced motility, unsteadiness and fall causing foot injury. Cannot practice self-care and prevent trauma.
- Patients having diabetes for >10 years or poor glucose control develop reduced immune function and poor healing power from chronic hyperglycemia with increased risk infection.
- Depression due to chronic diabetes results in lack of self-care leading to skin infection and injury.
- Cardiovascular and renal complications of diabetes also increase the risk of infection.

The incidence of diabetic foot problems increases with age >40 years and most common in those aged 50 years or more. However, duration and control of diabetes are greater predictors. Men with diabetes are at increased risk of foot ulcer.

Q. What are the cardinal features of diabetic foot?

Cardinal features of diabetic foot are:

- Diabetic peripheral neuropathy is the major problem leading to foot problems.
  - Patients with type II diabetes are more prone to peripheral neuropathy.
  - Peripheral neuropathy affects sensory, motor and/or autonomic nerves leading to deformity and abnormal gait with calluses formation over pressure point on foot.
  - Patients with neuropathy cannot feel discomfort and continue to walk on injured foot resulting in breakdown of skin and chronic ulcer.
  - Charcot's joint—rare but serious complication in diabetic patients with peripheral neuropathy. Charcot's joint results in destruction and deformity of articulation of the foot, which is made worse due to absence of pain sensation from peripheral neuropathy. If patient continues to walk on it, the foot may become injured and deformed leading to ulceration and amputation. Typical early sign of Charcot's joint —one foot or part of one foot being warmer than the other with swelling and redness.
  - Another major complication of diabetes is peripheral vascular disease leading to painful ischemic ulcer following minor injuries. Lower limb ischemia slows healing process.
  - It is not uncommon for patients to have both neuropathy and vascular disease. So the ulceration may not be painful.
  - Local infection can result in callosities, deep abscess formation and gangrene.
  - In cases of deep ulceration, osteomyelitis may occur hampering wound healing.

As it is not always possible to achieve good diabetic control and as severe neuropathy and peripheral vascular disease cannot be easily reversed, many people with diabetes are at risk of developing foot problems.

Q. What are the different types of foot infection? What are the different intensities of treatment required?

Types of foot infection and the intensity of treatment required are:

- Localized foot infection is recognized by redness, heat and swelling confined to an area. Foot as a whole is not swollen. Oral antibiotics for a few days eliminate infection.
- In localized foot infection associated with neuropathy, the patients feel no pain or discomfort. Oral antibiotics are unsatisfactory and need to be continued until the ulcer is completely healed.
- *Generalized foot ulceration*: The whole foot is red and swollen. Oral antibiotics in higher doses are started and if no response within 24 hours, IV antibiotics are administered. Sometimes, surgical interventions are urgently required, as infected tissue is likely to be deep inside the foot.
- *Foot infection complicated with osteomyelitis*: In foot ulcer, there is risk of underlying bone involvement. X-ray may show bone involvement and bone destruction. Much higher dose of antibiotics for a longer period (several months) is often required to eradicate infection. Sometimes, infected bone needs to be surgically removed to help foot healing.

Q. What abnormalities of foot shape make the effect of neuropathy and vascular disease worse?

Sometimes the foot shape abnormality is a part of diabetic neuropathy or other disease process. Some of the abnormalities are:

- Clawed toes
- Rocker bottom foot
- Abnormal toe nails.

Clawed toes: Occur as a result of imbalance of muscles in the feet due to diabetic neuropathy. This increases pressure at the tip or apex of the toes, which become ulcer prone in presence of neuropathy.

Rocker bottom deformity: Occurs due to Charcot's joint, a complication of diabetic neuropathy.

Abnormal toe nails: Toe nails become infected, thickened and deformed.

Q. What are the different foot problems that develop in diabetic patients?

Foot problems most often occur when there is neuropathy. It causes tingling, pain (burning or stinging) or weakness in foot. It also causes loss of sensation in the foot resulting injury, poor blood flow and change in shape of feet or toes also causes problems.

Foot problems in diabetic patients include:

- *Neuropathy*: Produces loss of sensation making patient prone to foot injury without notice until skin breaks down and become infected. It also changes the shape of the foot or toes requiring special therapeutic shoes.
- *Skin changes*: Diabetes can cause changes in foot which become dry. Skin may peel and crack due to nerve damage.
- *Calluses*: Calluses occur often and build up faster on the feet of diabetic patients, as there are high pressure areas on foot. Too much calluses mean requirement for therapeutic shoes and inserts.
- *Foot ulcers*: Most often ulcers occur on the ball of the foot or bottom of the big toe. Ulcer should be seen by healthcare provider immediately. Neglecting ulcer results in infection.

Q. How the degree of diabetic neuropathy is detected?

The degree of diabetic neuropathy can be detected by testing if the patient can feel pain of a pinprick or touch of a cotton wool or vibration of a tuning fork. Standardization of the procedures and results is difficult. To overcome these problems, two other methods are used:

1. *Testing vibration sensation with biothesiometer*: In this test, a probe is applied to part of the foot, usually on big toe. The probe is made to vibrate at increasing intensity by turning a dial. The patient indicates as soon as he/she can feel the vibration and the reading is recorded. Biothesiometer can have a reading from 0–50 volts. Reading is low in younger people and become higher as age increases. The risk of developing a neuropathic foot ulcer is much higher if the reading is greater than 30–40 volts.
2. *Testing touch pressure sensation with monofilament*: A standardized filament is pressed against part of the foot. When the filament bends, its tip exerts a pressure of 10 g (referred to as 10 g monofilament). When the patient cannot feel the monofilament at certain specific sites on the foot, enough sensation is lost, to be at risk of developing a neuropathic foot ulcer.

   Monofilament is cheaper than biothesiometer but needs to be calibrated to make that it give an exerting force of 10 g.

If a patient has a high biothesiometer reading (40 volts) and cannot feel monofilament, there is high risk of developing neuropathic foot ulceration, especially if other risk factors are also present and they need intensive foot care education. Presence or absence of pain is not a major factor in determining the risk of neuropathic foot ulceration.

Q. How the severity of peripheral vascular disease predisposing to foot ulceration is detected?

Diabetes mellitus causes blood vessels of the feet and legs to narrow and harden to peripheral vascular disease. If peripheral vascular disease is more severe, there is less circulation to bring enough oxygen to the tissues, resulting ischemic damage.

- When a patient with diabetes complains of intermittent claudication or rest pain (especially the latter), is an indication of sufficiently severe peripheral vascular disease to predispose vascular diabetic foot ulceration.
  Intermittent claudication: Ischemic pain of leg that occurs because blood flow cannot keep up with demand as when walking fast, up a hill or on a hard surface. The pain is severe, cramping and usually located in the calf muscles. It may be unilateral or bilateral. Stopping to rest for a few moments relieves pain. In this case, (i) smoking must be stopped (ii) walking program to be started (iii) exercise is good for poor circulation as it stimulates blood flow in the legs and feet (iv) walking in hurry, good fitting and comfortable shoes is helpful.
  Rest pain: Classically rest pain due to vascular insufficiency is confined to the dorsum of the foot at the area of metatarsal heads. The pain develops about an hour after the patient goes to bed at night, as the effect of gravity is lost and blood pressure and cardiac output fall during sleep. The pain is severe, usually burning in character and wakens the patient from sleep. The pain is relieved by hanging the limb over the edge of the bed, which increases blood flow to the foot. Dependent rubor of the foot may be prominent with pallor on elevation. The skin of the feet is cool, atrophic and hairless. In contrast to IC, patients with rest pain are at increased risk of development of ulcer and gangrene. Even trivial injury fails to heal and entry of bacteria leads to infection.
- If the feet look purplish in color and feel cold on touch, indicates important clue that circulation is impaired.
- If pulses in the foot (dorsalis pedis artery pulse and posterior tibial artery pulse) can be clearly felt, the risk of ulceration due to vascular disease is small. When foot pulses are very weak or not palpable, noninvasive vascular tests to assess the risk of foot ulceration are necessary.
  These tests are easily done by measuring:
  - Ankle brachial index: BP is taken in the arm (brachial pressure) and BP is taken in the ankle (dorsalis pedis or posterior tibial artery) – (ankle pressure). Ankle brachial index = Ankle pressure/Brachial pressure.
  - Normal—0.9–1.2—risk of vascular foot ulcer is small
  - Definite vascular disease —0.6–0.9—moderate risk of vascular ulcer, depending on other risk factors.
  - Severe vascular disease—less than 0.6—risk of vascular foot ulcer is very high.
  - In case of calcification of ankle arteries, more information is obtained by measuring toe pressure. Toe brachial index less than 0.5 indicates presence of peripheral vascular disease.

When significant vascular disease is established, a duplex scan is performed to locate the block and assess the severity. It is a combination of an ultrasound test and a Doppler test.

Q. What steps are to be taken by diabetic patients to avoid foot problems?

People with diabetes mellitus are more prone to infection. They also develop neuropathy (damaged nerves) or peripheral vascular disease (blocked arteries) of legs and either can lead to foot ulceration. Poor diabetic control increases infection and impair wound healing. The person with very poor control of diabetes mellitus (HbA1c >10%) is most at risk. Steps taken to avoid foot problem are:

- Good diabetic control is very important and most essential as high blood glucose levels make it hard to fight infection.
- More care should be taken to prevent foot trauma. Keeping off the feet is very important—walking on ulcer increases ulcer size and forces infection deeper. Special shoes, brace or cast is put by healthcare provider, for foot protection.
- Good foot hygiene is also crucial.
  - Feet are checked every day.
  - Feet are washed and dried everyday.
  - Skin of feet is kept soft and smooth. Dry skin cracks lead to infection.
  - After bathing, feet are dried and the remaining moisture is sealed with thin film of plain petroleum or unscented hand cream. Extra moisture can lead to infection. No oil or cream is put between toes.
  - Calluses or corns should not be cut but are gently smoothened to avoid ulcer or infection. Calluses or corns are not to be removed by chemical agents, as these can burn the skin. Using pumice stone daily helps keep calluses under control. It is best used on wet skin. Lotion is put on just after the use of pumice stone.
  - Patients should trim toe-nails regularly or podiatrist is asked to trim them.

More care is taken to increase chance to avoid foot ulceration:

- Socks and shoes should be worn all times
- Feet are protected from hot and cold. Cold feet in diabetic patient are easy to burn with hot water bottle or heating pads. Best way to help cold feet is wear warm socks
- Measures should be taken to keep blood flowing to the feet
- Smoking is avoided as it hardens arteries faster
- BP and cholesterol should be under control to prevent vascular disease.

Q. What steps are to be taken immediately in case of limb infection?

Steps to be taken immediately in limb infection are:

- Metabolic assessment
- Optimization of glycemic control
- Institution of broad spectrum antibiotics
- Immediate surgical consultation.

Q What steps are to be taken in case of deep foot ulcer with or without osteomyelitis?

Urgent steps to be taken in deep foot ulcer with or without osteomyelitis are:

- Metabolic assessment
- Optimum glycemic control
- Assessment of the limb lesion
- Debridement of devitalization tissues
- Treatment of infection
- After foot ulcer heals, foot is treated carefully. Special shoes are needed to protect the area and prevent recurrence.

Chapter

24

Neck Contracture Following Upper Body Burns

Q. What are the types of burns?

The types of burns are:

- *Thermal burns*:
  - Scalds usually from hot water are the most common cause of burns.
  - Flame burns are the second most common mechanism of thermal injury.
  - Flash burns are next in frequency. Explosions of natural gas and electrical arcs cause intense heat for a brief time period.
  - Contact burns result from contact with hot metal, glass or hot coals.
- *Electrical injury and burns*: These are particularly dangerous as they can be instantaneously fatal. Severity of injury depends on amperage of the current, the pathway of current through the victim's body and the duration of contact with the source. Low voltage causes local contact burns. There is no associated deep tissue damage. High voltage (>500 volts) causes flash burns or deep tissue damage due to current transmission. Neurologic complications are very common.
- *Chemical burns*: Strong acids and alkalis cause most chemical burns. Typically are associated with industrial accidents, assaults, or improper use of harsh household solvents and cleaners. Hands of upper limbs are most frequently affected areas. The burnt area should be irrigated with water for several hours.

Chemical burns are influenced by duration of contact, concentration of chemical and the amount of the agent. Alkali burns are more serious than acid burns.

Q. How the severity of burns is assessed?

Severity of burns can be assessed in several ways. There are three commonly used methods for measuring a burn:

1. *Wallace rule of nines:* It is used to assess the percentage of body surface area burnt. The total body surface area is divided into multiples of nine and is used as a rule of thumb to quickly calculate total body surface area burnt.

Head and neck	9%
Upper extremities	9% each
Chest (anterior and posterior)	9% each
Abdomen	9%
Lower back	9%
Lower extremities	18% each
Perineum	1%

2. *Palmar surface*: The palmar surface of the patient's hand, with fingers very slightly spread, equals to approximately 1% of the patient's TBSA. This is a quick and reliable method of assessing patchy areas of burns up to 15% of TBSA. It is not very accurate in estimating moderate burns.
3. Lund and Browder chart: Currently it is the most accurate method of assessing the burn area in children.
   The children have relatively larger heads and smaller limb as compared to adults. Due to the different body proportions, the "rule of nine" is inaccurate in children. Age adjusted nomogram are a better method, because they reflect the gradual change in the relative body surface areas with age. Accurate assessment of burn area in children is done by Lund and Browder chart.
   Chart for calculation of percentage of BSA burn in children (age in years)

Area	0	1–4	5–9	10–14	15
1. Half of head	9½	8½	6½	5½	4½
2. Half of one thigh	2¾	3¼	4	4¼	4¼
3. Half of one lower leg	2½	2½	2½	3	3¼

Q. How burn injury is classified?

Classification of burn injury can be done based on the degree of burns.

Degree of burns	Depth of injury	Outcome and treatment
First degree (superficial)	Epidermis	Erythematous, minimum tissue damage, heals without scar
Second degree (partial thickness)	Epidermis of varying portion of dermis	Blistering, excruciating pain, heals with minimum scaring, may need grafting
Third degree (full thickness)	Destruction of epidermis or dermis.	Dry leathery skin with firm consistency and pearly white discoloration, not very painful, will require incision and grafting

Further classification can be done as follows:

Minor	Superficial burns of <15% TBSA
Moderate	Superficial burns of >15% TBSA in children.
	Full thickness burns of <10% not involving the eyes, face, hands, feet or perineum

Major	Third degree burns involving >5% of TBSA.
	Second degree burns involving >10% of TBSA.
	Burns involving face, hands, feet, perineum and major joints.
	Electrical, chemical and inhalational burns.
	Burns in children with serious preexisting medical disease.

Q. What are the major layers comprising the healthy skin? What are the functions of the skin?

Normal healthy skin is divided into three layers:
1. The epidermis
2. The basement membrane
3. The dermis.

Epidermis: The outer most layer of the skin, a stratified squamous epithelium and predominantly composed of keratinocytes with very little extracellular matrix. It is attached to but separated from the underlying dermis by the basement membrane.

Basement membrane: Acts as an anchor for the epidermis but allows the movement of the cells and nutrients between the dermis and epidermis.

Dermis: It supports the epidermis structurally and nutritionally. The dermis contains and supports blood vessels, nerves and epidermal derived structures, such as appendageal (hair follicle and sweat glands). The prominent cells of the dermis are fibroblasts. The accellular part of the dermis consists predominantly of collagen, elastin of reticulin. Support is provided by an amorphous ground substance. Below the dermis is a layer of adipose tissue (the subcutis). This fat layer provides mechanical and cushioning as well as thermal insulation for the underlying structures. The skin has various important functions:
- The skin protects against most noxious agents like chemical (by impermeability of the epidermis), infecting agents (through efficient immune surveillance), physically deforming forces (by the durability of dermis) and solar radiation (by means of pigmentation)
- Its efficient ability to conserve or disperse heat makes the skin the major organ of thermoregulation
- Fluid and electrolyte homeostasis
- Touch, temperature and pain sensations
- Vitamin D metabolism.

Q. How major burn is defined?

Definition of major burns is based on body surface area burnt and the area of the body burnt.
- Third degree (full thickness) burn injury involving >10% of TBSA

- Second degree (Partial thickness) burn injuries involving > 20% of TBSA at extremes of age and >25% of TBSA in adults
- Burns involving face, hands, feet, genitalia, perineum and major joints
- Inhalational injuries
- Chemical burn injuries
- Electrical burn injuries
- Burn injuries in patients with coexisting medical disease
- Burns associated with trauma.

Q. What is the role of anesthesiologist during the management of a burnt patient?

- Anesthesiologist may be involved in the initial resuscitation of a burnt patient, especially if there is hypoxemia and airway compromise, which is life-threatening.
- A burnt patient may need general anesthesia for various surgical procedures like:
  - Early excision of damaged tissues
  - Change of dressings
  - Excision of granulation tissue or subsequent skin grafting
  - Reconstructive plastic surgical procedures to release contracture, correct deformities or restore limb function.

Q. How will you resuscitate a patient with thermal burns?

The burn patients have the same priorities as all other trauma patients. They should undergo a quick primary survey and more comprehensive secondary survey to assess the damage while management is instituted.

All burn patients should receive 100% oxygen with a reservoir mask. If there is any sign of airway obstruction or any suspicion of inhalational injury, immediate endotracheal intubation is done. The burnt area should be rinsed with tapid water (at 15°C) for 20–30 minutes. Ice should not be used as it causes vasoconstriction leading to further tissue damage and hypothermia. The patient is then covered with warm, clean and dry linens to prevent hyperthermia.

Primary surgery

A = Airway maintenance with cervical spine control.
B = Breathing and ventilation.
C = Circulation with hemorrhage control.
D = Disability—neurological status.
E = Exposure and environmental control.
F = Fluid resuscitation proportional to burn size.

Airway maintenance with cervical spine control: Control of airway and cervical spine. Identification of inhalational injury in all burnt patients. If any, history of trauma in cervical spine should be immobilized. Airway can be supported with simple manual techniques or use of basic airway adjuncts. 100% oxygen is administered via reservoir mask. Endotracheal intubation

and mechanical ventilation is needed in patients who are unconscious from associated trauma, who have developed acute respiratory failure (ARF) due to smoke inhalation and patients with major burns.

Breathing: Chest is kept exposed to ensure that the chest expansion is adequate and equal and to look for signs of respiratory distress. Circumferential burns to chest may restrict breathing and impair gaseous exchange. An escharotomy is required if breathing is compromised. Carbon monoxide poisoning should be ruled out by measuring HbCO level and if present high flow oxygen at 100% should be administered via non rebreathing mask.

Circulation: Rapid assessment of volume status should be done. Pulse, BP and capillary refill are monitored. In burnt upper limbs, BP is monitored by invasive method. Escharotomy is required if there is poor perfusion of the limb due to circumferential full thickness burns.

Disability—(neurological status): Level of consciousness and pupillary response to light are assessed. Baseline GCS should be assessed in all burnt patients.

Exposure and environmental control: Jewelries and clothing should be removed. Patient is kept warm. They easily become hypothermic due to loss of protective thermoregulatory skin and evaporation of fluid from exposed tissues.

Fluid resuscitation: Following tissue burns, there is large fluid shift due to increased capillary permeability. Fluid depletion is greatest in the first few hours of early fluid resuscitation. It is essential to avoid hypovolemic shock and ARF. During fluid resuscitation in burn patients, urinary catheter is inserted to monitor urine output as a function of organ perfusion (0.5 mL/kg/hour in adult and 1 mL/kg/hour in children). Warm RL solution is commonly used parkland formula and is widely used for resuscitation of burnt patients.

4 mL/kg/%age BSA of burns. Half the amount of total fluid is given in first 8 hours and the remaining over the next 16 hours. In children, maintenance of IV fluid should contain glucose in addition to burn formula.

Clinical assessment is done by pulse, BP, capillary refill, CVP, urine output, peripheral and core temperature and mental status. Blood should be collected for grouping and cross-matching.

Investigations: CBC, blood sugar, serum electrolytes, serum urea and creatinine, COHb levels and ABG analysis.

Urine is tested for myoglobin/hemoglobin and chest X-ray for ARDS.

Pain management: A severely burnt patient is restless and anxious due to hypoxemia and hypovolemia, rather than pain. So they respond better to oxygen and fluid administration, rather than narcotic analgesics and sedatives, which may mask the signs of hypoxemia or hypovolemia. Narcotic analgesics and sedatives should be administered in small frequent doses by IV route only.

Secondary surgery

Once the burn patient has been stabilized, a thorough examination is done to detect any concomitant injuries. After assessing the size and depth, the burnt area is washed with tapid water and then covered with transparent dressing to protect the wound and to reduce heat and evaporative losses.

Nasogastric tube is inserted to decompress the stomach, if there is nausea and vomiting. As burnt patients are in a highly catabolic state, starting enteral feeding within 4 hours of admission improves the outcome and promotes normal gut function. Prophylactic antibiotics are not indicated.

Q. What is the pathophysiology of inhalational injury?

Inhalation injury occurs as a result of direct thermal injury to mucosa of the respiratory tract or from chemical tracheobronchitis due to inhalation of incomplete products of combustion.

Airway injury above the larynx: Direct thermal injury particularly occurs in the upper respiratory tract only, because these occur in reflex closure or the glottis. Usually there is upper airway edema, which may progress to complete airway obstruction. Stridor and respiratory distress indicate inhalation injury. Intubation is performed immediately if airway obstruction is suspected, because the pharyngeal mucosa swells rapidly after fluid resuscitation.

Airway injury below the larynx: The lower airway is rarely burnt by exposure to heat.

Chemical tracheobronchitis: Inhalation of products or incomplete combustion leads to pulmonary inflammatory response similar to that seen after gastric acid aspiration. Water soluble gases such as ammonia, nitrogen-dioxide, sulfur-dioxide and chlorine combine with water in the respiratory tract, producing strong acids and alkalis. These chemical products act as direct irritants causing bronchospasm, edema and mucous membrane ulceration. The production of surfactant is impaired and ventilation perfusion mismatch develops as the alveolar injury evolves.

Systemic intoxication injuries: Toxins and chemicals like carbon monoxide (CO), hydrogen cyanide, ammonia, hydrofluoric acid and phosgene that reach the alveoli can be absorbed into the systemic circulation. Severe systemic intoxication often results in metabolic acidity. CO interferes with tissue oxygenation by binding to hemoglobin and preventing oxygen from binding to hemoglobin. It also inhibits mitochondrial function at cellular level. Combustion of plastic, wool, silk, nylon, rubber and paper products leads to production of cyanide gas, which interferes with cellular metabolism by binding to the ferric iron in cytochrome A_3. Due to decreased oxygen utilization, there is anaerobic metabolism leading to lactic acidosis.

Q. How inhalational injuries are diagnosed and treated?

Diagnosis

A proper history, a good clinical examination and laboratory testing will help the diagnosis of inhalational injury.

On examination, there would be darkened or reddened oral and/or nasal mucosa, burns on the face, lips and nares and presence of carbon or soot in mouth or throat. Hoarse voice and productive cough indicate inhalational injury. Tracheal tug, inspiratory stridor and inability to clear secretion indicate impending airway occlusion and early intubation is indicated.

Arterial blood gases (showing high lactate level in hemodynamically stable patient) and carbon monoxide levels (measuring using co-oximetry) may be useful in determining the degree of insult.

Currently the presence of inhalational injury is diagnosed by bronchoscopy or xenon lung scan. Fiberoptic bronchoscopy is performed for diagnosis and treatment.

Mild injury leads to erythema or edema of the mucosa.

In severe inhalational injury, FDB shows grey colored mucosa, ulceration and/or desquamation.

Management

- *Primary supportive*: Early tracheal intubation ventilator support and aggressive pulmonary toilet, bronchodilator therapy or bronchoscopic lavage, all play an important part in the treatment of inhalational injury.
- The ABC (airway, breathing and circulation) approach should be used for managing patients with inhalational injury. Early intubation is considered if there is stridor, GCS <8, hypoxia, hypercarbia, full thickness neck burn, deep facial burns and oropharyngeal edema.
- With abnormal airway or upper airway obstruction, the safest airway is with the patient awake, after effective topical analgesia, proper patient positioning along with supplemental oxygen administration. Alert patient can be given IV opioids for pain relief but sedation should be used continuously as it may worsen airway obstruction.
- Fiberoptic intubation is not useful in patients with swollen upper airway as it is impossible to anesthetize the airway or instrumentation may cause trauma or swelling.
- Alternatives include direct laryngoscopy or laryngeal mask airway.
- GA with inhalational induction may be needed in uncooperative patients where oral intubation is preferred.
- A direct airway is indicated, when the upper airway is badly damaged and endotracheal intubation is not possible, but should be considered only as a last resort. Options include a needle cricothyroidotomy, surgical cricothyroidotomy or tracheostomy.
- Mild smoke inhalation is managed supportively with high flow oxygen, humidified inspired gases, bronchodilators and physiotherapy.
- Patients with more severe injury need ventilator support for deteriorating lung function. FOB should be done in all intubated patients with inhalational injury for better diagnosis and aggressive pulmonary toileting.
- Fluid restriction does not prevent pulmonary edema. Under resuscitation makes pulmonary damage worse.
- Ventilatory management includes low tidal volume (6 mL/kg) and low pressure (plateau pressure <30 cm H_2O).

- Bronchodilators may be helpful in bronchospasm. Mucolytics like N-acetyl cysteine helps in airway clearance of mucus plugs and improving PaO_2/FiO_2 ratios.
- Steroids of prophylactic antibiotics are not indicated.

Q. What is the mechanism of carbon monoxide (CO) poisoning? What symptoms are produced in CO poisoning? How the patient with CO poisoning is treated?

Commonest cause of fire-related death is smoke inhalation of CO poisoning.

Mechanism: CO reduces the oxygen carrying capacity of blood resulting in tissue hypoxia. The PaO_2 is normal. CO binds avidly to other heme containing compounds, especially the cytochrome system. Possible mechanism of toxicity includes:

- Decrease in oxygen carrying capacity of blood.
- Leftward shift of the oxyhemoglobin dissociation curve, further decreases oxygen delivery to the tissues.
- Binding with cytochrome A_3 results in decrease in cellular respiration.
- Binding to myoglobin, potentially causes myocardial or skeletal muscle dysfunction.

Incapacitation of the cellular mechanisms to utilize oxygen is one of the most important toxic actions of CO, which results in chemical asphyxiation and hypoxia.

CO binds with oxygen binding sites of hemoglobin, as it has 250 times more affinity for hemoglobin than oxygen. Therefore, oxygen cannot bind with red cells resulting in histotoxic hypoxia. Carboxyhemoglobin causes leftward shift of oxygen dissociation curve, preventing release of oxygen to the tissues. CO also binds to cytochrome oxidase in the cells, making them unable to utilize oxygen.

CO also binds to cardiac and skeleton myoglobin. Carboxymyoglobin dissociation is slower than COHb due to increased affinity of CO for myoglobin.

Fetal hemoglobin binds to CO more avidly than hemoglobin A, and with slow transplacental transport, fetal hemoglobin level decreases much more slowly than in mother. This accounts for the occurrence of fetal death in nonfatal maternal exposure.

Signs/Symptoms: As oxygen is required by the cells in the body to function normally, all body systems are affected by CO poisoning. Early signs and symptoms are due to impairment of respiratory, cardiovascular and nervous systems. CO poisoning often mimics a viral illness with symptoms like fatigue, lethargy, somnolence, malaise, nausea, vomiting or dizziness. Cardiac contractility is decreased, leading to further decrease in tissue oxygen delivery (low flow hypoxia). Clinical effects are seen due to side-effects of spread of ischemia including rhabdomyolysis, pulmonary edema, multiorgan failure, DIC and renal failure. The severity of symptoms depends on the level, which is <10%. Patient becomes symptomatic when the level is

20%. With level >40%, coma and seizures occur due to cerebral edema. Death is likely with level above 60%.

Treatment: Patient is removed from the exposed environment to terminate the exposure and supportive measures are taken. As the binding of CO with hemoglobin is reversible, the mainstay of therapy for CO poisoning is supplemental oxygen, ventilator support and monitoring of cardiac arrhythmias. Oxygen is given at a rate of 15 L/min via a non-rebreathing mask. Half-life of COHb is 250 mins at atmospheric pressure, 40–80 mins while breathing 100% O_2 and 22 mins in hyperbaric chamber at 2.5 atm. Oxygen therapy should be continued, because secondary peak of COHb occurs after 24 hours due to washout of CO from the cytochrome.

Roll of hyperbaric oxygen (HBO) therapy in CO poisoning:
- HBO therapy reduces half-life of COHb to 20–30 minutes.
- HBO also increases the amount of oxygen dissolved in the blood from 0.3 mL/dL with isobaric therapy (FiO_2 100%) to 5.5–6.4 mL/dL (2.4–2.8 atm).
- HBO induces cerebral vasoconstriction, which may reduce ICP and cerebral edema.
- HBO results in more rapid dissociation of CO from respiratory cytochromes.
- HBO may antagonize the oxidative injury that occurs after CO poisoning.

Q. How cyanide poisoning is caused? What is the pathophysiology of cyanide poisoning? What is the management of cyanide poisoning?

Cyanide comes from a wide range of natural and manmade sources.
- Cyanide is used in industries including metallurgy, electroplating and metal cleaning.
- Cyanide is used in the recovery of gold and silver from minerals and silver from photographic materials.
- Other sources of cyanide include emissions from production of iron and steel, coal burning, vehicle exhaust and cigarette smoke.
- Combustion substances like nylon, wool and cotton can result in production of hydrogen cyanide.
- Nitrites can produce hydrogen cyanide when they are metabolized in the body after absorption through skin and GI tract.
- Some drugs like nitroprusside, can generate cyanide in the body when metabolized.

Pathophysiology

Cyanide causes tissue asphyxia by inhibiting intracellular cytochrome oxidase activity, preventing mitochondrial oxygen consumption. Affected cells can only generate adenosine triphosphate (ATP) via anaerobic metabolism as a result there is lactic acidosis. Cyanide poisoning is suspected in burnt patients when there is persistent lactic acidosis in spite of fluid resuscitation.

With a concentration of 50 parts per million (ppm), symptoms include headache, dizziness, tachycardia and tachypnea.

Above 100 ppm, there is lethargy, seizures and respiratory failure.

These patients have high anion gap acidosis, which does not respond to oxygen administration. Mixed venous oxygen saturation is also high in these patients.

Plasma lactate levels are useful alternative diagnostic tool as they correlate with cyanide levels.

Treatment

Cyanide is normally metabolized in the liver to thiocyanate with thiosulfate as a substrate, but it is a slow process.

Administration of additional thiosulfate hastens the hepatic metabolism of cyanide to produce nontoxic thiocyanate, which is excreted in urine.

Nonhepatic metabolic pathway to remove cyanide includes the combination of cyanide with methemoglobin and hydroxycobalamin. Administration of amyl sodium nitrite results in oxidation of hemoglobin to methemoglobin, which combines with cyanide to form cyanomethemoglobin.

Q. What are the anesthetic considerations for excision or grafting of major burn injuries?

Burnt patients are posted for wound excision and skin grafting after a period of 48 hours of initial stabilization.

A complete history about the burns should be taken and a thorough physical examination should be done. History reveals the possibility of inhalational injury.

- The percent TBSA of burns should be assessed to know the physiologic condition of the patient.
- The adequacy of resuscitation should be assessed.
- The airway assessment should include Mallampati class thyromental distance of head-neck mobility.
- The presence of facial burns makes mask ventilation difficult.
- Commonly used IV access sites may be unavailable.

Investigations

- Blood grouping and cross-matching
- Complete blood count
- Blood glucose
- Serum electrolytes
- Serum urea and creatinine
- Arterial blood gas analysis to know the COHb levels
- Chest X-ray
- ECG in patients above 40years of age
- Adequate blood should be kept ready as sufficient blood loss occurs in excision and grafting of major burn injuries.

Preoperative optimization

Prolonged period of fasting is avoided because in major burns patients are in hypercatabolic state and adequate nutritional status is difficult to attain.

These patients receive large doses of analgesics for pain relief and should be continued throughout perioperative period.

Depending on the surgical excision, adequate sized IV cannula should be inserted.

Monitoring

Intraoperative monitoring includes ECG, BP, $EtCO_2$, pulse oximetry, core temperature, neuromuscular and urine output monitoring. In cutaneous burns, placement of ECG electrodes or BP cuff is difficult. Invasive BP monitoring is preferable.

It may be difficult to get pulse oximetry trace due to peripheral burns or vasoconstriction.

Alternative sites like ear, nose and tongue can be used. Carboxyhemoglobin gives spurious results, hence, blood gas analysis is done when indicated. Blood gas analysis is also done to monitor ventilator settings. In patients with extensive burns, CVP catheter is needed.

Temperature monitoring is essential, because hypothermia in these patients is common and difficult to prevent. Heat loss is due to evaporation. In addition, cutaneous vessels in burnt area cannot constrict to prevent heat loss by radiation.

The operating room temperature should be 28°C–32°C and all topical and IV fluids should be warmed. Nonoperative sites should be covered or forced air warming devices should be used.

Induction of anesthesia

General anesthesia with combination of an opioid, muscle relaxant or a volatile agent is the most commonly used technique for burn excision and grafting. Laryngeal mask airway (LMA) is effective in maintaining airway both in adults and children with burns.

Ketamine is used extensively as a primary agent for both general anesthesia and analgesia for burn dressing, because it has a lot of advantages. It has analgesic property. It provides stable hemodynamics.

Antisialagogue premedication is a must, diazepam or midazolam is used to control emergence hallucinations.

Etomidate is an alternative to ketamine in hemodynamically unstable patients.

Thiopentone can be used in volume resuscitated patients. Succinylcholine (Sch) should be avoided 24 hours after burn injury.

Maintenance of anesthesia

When LMA is used, the patient is kept on spontaneous ventilation with O_2, N_2O and inhalational agents. As these patients may require repeated anesthesia in short period of time, use of halothane should be avoided to reduce the risk or halothane hepatitis.

When the patient is intubated, non-depolarizing muscle relaxants atracurium or vecuronium) can be used for controlled ventilation.

These patients have increased minute ventilation requirement due to increased metabolic rate and hyperalimentation. Patients with ARDS due to inhalational injury may be PEEP dependent.

As these patients receive opioids for various procedures or routine pain management, they develop tolerance and need large doses of opioids in perioperative period for pain relief.

Regional anesthesia is useful in minor burns. Not very useful in major burns as skin has to be harvested from extensive areas which may not be effectively covered by regional blockade and there is also chance of massive blood loss and hypotension during extensive debridement. With regional technique through burnt tissues, there is chance of spread of infection. Blood loss during excision and grafting procedures can be minimized by topical application of epinephrine. Bandages are soaked in 1:10,000 adrenalines after excision of burnt skin graft. As after burn injury cardiovascular responses to catecholamine are attenuated, there are minimum changes in BP and heart rate after topical administration of such high concentration of adrenaline.

These patients need large amount of fluid administration intraoperatively, which can cause considerable soft tissue edema. If patient is in prone position and if there is significant facial edema, extubation should be delayed till the swelling subsides.

Postoperative care

Patient controlled analgesia is used in the postoperative period using various opioids, NSAIDs or paracetamol may be used depending on presence or absence of renal or liver dysfunction.

Q. What are the considerations for using muscle relaxants in a patient with burns?

Suxamethonium can be used in the first 24 hours of burns to facilitate endotracheal intubation.

The post junctional receptors increase in number after burns and cause prolonged depolarization with marked release of potassium. The most dangerous period is between 4 days and 10 weeks after thermal burns, as receptor proliferation takes several days to develop. How long the hypokalemic response to suxamethonium persists is unclear and the recommendation is to avoid use of suxamethonium in burnt patients for up to 2 years post burn. Suxamethonium is also contraindicated in patients with extensive muscle damage as it causes release of large amount of potassium sufficient to cause cardiac arrest.

Rocuronium is an alternative to suxamethonium.

Patients with thermal injury are resistant to the action of nondepolarizing muscle relaxants (NDMR). This resistance develops by 1 week and usually persists for 8 weeks. Mark resistance occurs when the burn is >30% TBSA. Proliferation of the acetylcholine (Ach) receptor is responsible for resistance to NDMR. Therefore, the burnt patients require larger than normal doses of NDMR to achieve a desired effect and the duration of action is shorter than

normal. Hence, neuromuscular junction monitoring is used to assess the adequacy of neuromuscular blockade and reversal with anti-cholinesterase.

Q. What is the mechanism of burn pain? What is the management of chronic pain after burn injury?

Mechanism

Skin consists of three layers—outer epidermis, inner dermis and hypodermis. The sensory structures are contained within the dermis and consist of free nerve endings (for pain, temperature or touch), Meissner's corpuscles (light discriminatory touch) and A delta fibers (fast pain) and nonmyelinated C fibers (slow and chronic pain) to synapse in the substantia gelatin of the dorsal horn of the spinal cord. Fibers then cross the midline of the spinal cord to ascend to the thalamus in the lateral spinothalamic tracts and from there to the postcentral gyrus, where conscious perception of the stimulus may occur.

The instant pain that follows a burn injury is due to stimulation of skin nociceptors that respond to heat (thermoreceptors) or mechanical distortion (mechanoreceptors). Exogenous (hydrofluoric acid) or endogenous (histamine, serotonin, bradykinin, leukotriene and prostaglandins) chemical stimuli are also responsible for pain after burn injury. Nerve endings those are entirely destroyed cannot transmit pain, but those remaining undamaged and exposed generate pain. The immediate pain sensations are carried by unmyelinated C and thinly myelinated A delta fibers.

Subsequently, there is primary and secondary hyperalgesia.

Primary hyperalgesia: Following burns, there is a massive inflammatory response resulting in release of inflammatory mediators which sensitizes the active nociceptors at the site of injury. Due to this, the wound or the skin immediately adjacent to it becomes sensitive to mechanical stimuli (touch, rubbing, debridement) and chemical stimuli (antiseptics and other topical applications).

Secondary hyperalgesia: It is the increased sensitivity in the surrounding unburnt areas resulting from continuous or repeated peripheral stimulation of nociceptive afferent fibers. This is mediated by the mechanical stimulation that occurs as a result of frequent dressings changes.

Different sizes and degrees of burns can result in differing amount of pain. Larger the burnt area more the pain, deeper the burnt area lesser is the pain, as there is greater destruction of nerve endings.

Management

Intravenous morphine remains the gold standard for management of pain in burnt patients, who are not at risk of airway obstruction. As these patients often require multiple dressing changes and other procedures, they very rapidly develop tolerance to opioid and use of other analgesics.

Initial first-aid cooling with tapid water reduces pain. NSAIDs can be used in patients with minor burns. They act by inhibiting formation of inflammatory mediators like prostaglandins, which should be used with caution in patients with shock.

For postoperative analgesia, multimodal analgesic technique combining paracetamol, NSAIDs, local anesthetics and opioids (IV morphine PCA) can be used.

For regional blocks, there are practical difficulties like infection close to the site of insertion, generalized sepsis or coagulation abnormalities.

Q. How the pharmacology of drugs is affected by burns?

Pharmacokinetic effects: The pathophysiologic changes occurring after thermal injury alter the pharmacokinetic parameters like absorption, bioavailability, protein binding, volume of distribution and clearance. The extent of these changes depends on the magnitude of injury or the time between injury and drug administration.

In the acute phase after burn, organ blood flow is reduced due to hypervolemia and decreased cardiac output. Therefore, drugs administration other than IV have delayed absorption.

Plasma albumin concentrations decrease and alpha1-acid-glycoprotein levels increase. Plasma protein binding of drugs like benzodiazepine is decreased resulting in an increase in the free fraction with decreased dose requirement.

Raised fibrinogen and alpha 1-acid glycoprotein reduce the free fraction of basic drugs (local anesthetics, muscle relaxants and propranolol) with altered pharmacological response.

The fluid loss to burnt wound and edema elsewhere decreases plasma concentration of many drugs below those in healthy patients.

After initial resuscitation, phase cardiac output increases as hypermetabolic state develops. This increases renal and hepatic blood with increased drug clearance. However, after burns there is wide patient-to-patient variability in renal and hepatic functions and drug therapy must be tailored to each patient.

Volume of distribution of drugs is altered by changes in protein binding and in extracellular fluid volume. Changes in loading dose are required with small volume of distribution of drug or narrow therapeutic range. Total plasma clearance should be considered during maintenance doses and dose intervals.

Pharmacodynamic changes: Changes in drug receptor interaction are common after burns and account for many clinically important alterations in anesthetic pharmacology.

Burn injury causes proliferation of extrajunctional receptors leading to resistance to non-depolarizing muscle relaxants (NDMRs) and hyperactivity to depolarizing muscle relaxants (DMRs). Effect occurs within a week and persists up to a year, being proportional to TBSA. These patients develop tolerance to sedatives, analgesic and inotropic medications.

Q. What are the considerations in the management for release of neck contracture following upper body burn?

- Complete medical and surgical history including a thorough examination of the patient is essential.
- Main concerns would be difficult airway due to limited neck mobility and microstomia.
- Due to facial scarring, it may not be possible to get good mask seal making mask ventilation difficult.
- Microstomia and distortion of oral cavity make use of LMA or oral fiberoptic intubation difficult.
- Blind nasotracheal intubation is unlikely to succeed if neck movements are restricted; it is fixed in flexed position.
- Retrograde intubation is not possible due to extensive scarring on the anterior surface of neck distorting the surface anatomy of larynx.
- Therefore, the technique of choice is awake fiberoptic intubation under airway local anesthesia. However, patency of the external naris has to be checked.
- IV access in the upper limb may be difficult due to scarring.
- Since, surgery does not require muscle relaxation, patient can be maintained on spontaneous ventilation using inhalational agents.

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