



NAVODAYA
MEDICAL COLLEGE
HOSPITAL &
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PG EXCEL 2026

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NAVODAYA MEDICAL COLLEGE RAICHUR

June 13th & 14th, 2026

PG Excel -2026

Karnataka Postgraduate Conference

Hosted and Organized by:

Navodaya Medical College

Hospital and Research Centre Raichur

In Association with

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Raichur Chapter

SCIENTIFIC SCHEDULE

e-Book

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DAY -1 INDEX

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DAY -1

13th JUNE 2026

Time	Topic	Speakers
07:30AM - 08:00AM	Flag Hoisting	
08:00am - 09:00am	Registration and Breakfast	
09:00AM - 09:40AM	Anaesthetic management of patient with COPD with lung abscess posted for lobectomy/ pneumonectomy	Dr Milind Hatwalne KBN Medical College, Kalburgi Dr Gajendra Singh MRMC, Kalburgi
09:40AM - 10:20AM	Anaesthetic management of Primigravida with hypertensive disorder of pregnancy came for safe confinement	Dr Rajesh Mane JNMC, Belagavi Dr Gangadhar Siddhartha Medical College, Tumkur
10:20AM - 10:50AM	Diabetic patient with pyocele in septic shock posted for incision and drainage	Dr Natesh Rao Vydehi Institute of Medical Sciences and Research Centre, Bangalore Dr Manjula AIMS, Belur
10:50AM - 11:05AM	Tea Break	
11:05AM - 11:45PM	Anaesthetic management of patient with obstructive jaundice (CA head of pancreas) posted for whipple surgery	Dr Manjula BP JSS, Mysuru Dr Arathi B H KIDWAI Bangalore
11:45PM - 12:15PM	Patient with cardiac pacemaker with shaft of humerus fracture posted for ORIF	Dr Shivakumar MC Subbaiah Medical College, Shivamogga Dr Lavanya SDUMC, Kolar
12:15PM - 12:55PM	Anaesthetic management of a patient with RHD (severe MS) with Hollow viscous perforation posted for Emergency Laparotomy	Dr Netra SS BMCRI, Bengaluru Dr Karthik Jain St. Johns Medical College, Bengaluru
12:55PM - 01:35PM	Lunch Break	



Time	Topic	Speakers
01:35PM - 01:50PM	Live session: Clinical Mapping of the Brachial Plexus; Anatomy to Clinical practice (Demo on a Volunteer)	Dr Azam Danish Mahaveer Jain Hospitals, Bangalore
01:50PM - 02:05PM	Focus session: AI Driven Advances in Anaesthesia Practice	Dr Henjarappa KIDWAI Bangalore
02:05PM - 02:35PM	3 years old child with foreign body in bronchus posted for FB removal	Dr Vijayanand KIMS, Bengaluru Dr Ashwini KIMS, Bangalore
02:35PM - 03:05PM	Burns patient with neck contracture posted for contracture release	Dr Sripada G Mehandala KSHEMA, Mangalore Dr Liyakat GIMS, Kalburgi
03:05PM - 03:35PM	Pedagogy	Dr Chandrika Dutt Apollo Hospitals, Bengaluru Dr Harshavardhan Father Muller Medical College, Mangalore
03:55PM - 04:05PM	Panel: How to face Practical exam: Chat with experts	Moderator : Dr Devanand B Ballari Panelists : Dr Manjunath CIMS, Chamarajanagar Dr Srinivasulu D BMCRC, Ballari Dr Raveendra US AJ Shetty Medical College, Mangalore
04:05PM - 04:20PM	Tea Break	



Time	Topic	Speakers
05:00PM - 06:10PM Viva-voce		
04:20PM - 04:55PM	Anaesthesia Equipments	Dr Madhuri Kurudi KMCRI, Hubli Dr Prasad Kulkarni MVJMC, Bangalore
04:55PM - 05:30PM	Drugs	Dr Kiranchand N BMCRC, Ballari Dr Manjunath Bhat KIMS, Karwar
05:30PM - 06:20PM	Vijayanagara PG Quiz – 1 st Round	Dr Rashmi N R KIDWAI, Bangalore Dr Megha Chitradurga Medical College, Chitradurga Dr Krishna Rathod GIMS, Kalburgi
06:20PM - 06:50PM	Inauguration	
06:50PM - 07:30PM	Cultural Programme	
07:30PM Onwards	Dinner	



13th JUNE 2026
09:00AM - 09:40AM

ANAESTHETIC MANAGEMENT OF PATIENT WITH COPD WITH LUNG ABSCESS POSTED FOR LOBECTOMY/PNEUMONECTOMY

Case Summary:

A 45 year old male known smoker and alcoholic with history of cough with purulent expectoration, aggravated in right lateral position, on and off fever, breathlessness with stable vitals and clinical examination suggestive of right lower lobe consolidation with probable cavitation (infective etiology) posted for lobectomy / pneumonectomy.

1. What is chronic obstructive pulmonary disease and diseases incorporated in it?

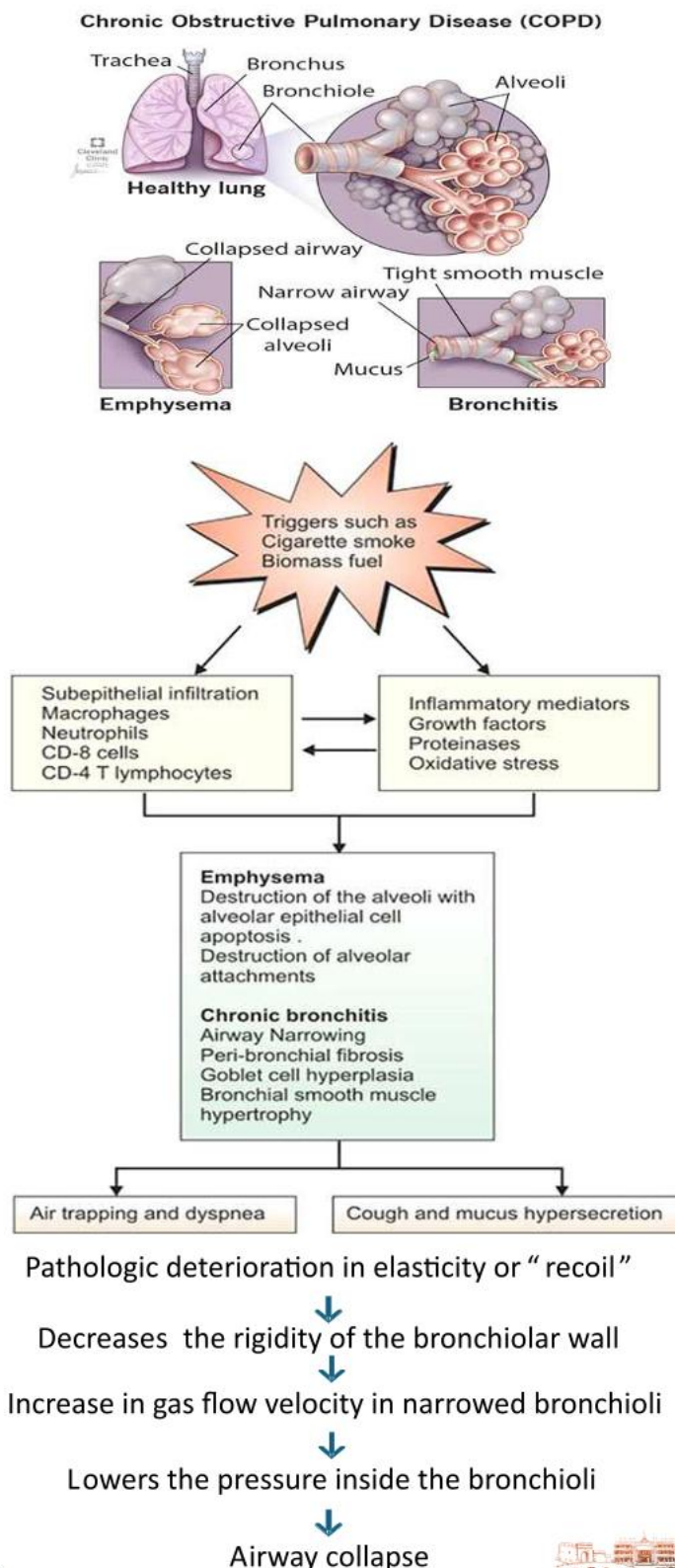
Chronic Obstructive Pulmonary Disease (COPD), a common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and co-morbidities contribute to the overall severity in individual patients.

Chronic bronchitis and emphysema are the two main diseases incorporated under COPD.

Chronic bronchitis is defined clinically as chronic productive cough for 3 months in a year for at least 2 successive years in a patient.

Emphysema is defined as the presence of permanent enlargement of the air spaces distal to the terminal bronchioles accompanied by destruction of their walls and without obvious fibrosis.

2. What is the pathophysiology in COPD?



"Pink Puffer" (Emphysema) & "Blue Bloater" (Chronic Bronchitis)

Features	Chronic Bronchitis	Emphysema
Mechanism of airway obstruction	Decreased lumen due to mucus & inflammation	Loss of elastic recoil
Dyspnea	Moderate	Severe
FEV1	Decreased	Decreased
PaO2	Markedly decreased (Blue bloater)	Mildly decreased (Pink puffer)
PaCO2	Increased	Normal or decreased
Diffusing capacity	Normal	Decreased
Hematocrit	Increased	Normal
Cor pulmonale	Marked	Mild
Prognosis	Poor	Good



Mechanisms underlying airflow limitation in COPD

Small airway disease	Parenchymal destruction
Airway inflammation	Loss of alveolar attachments
Airway fibrosis; luminal plugs	Decrease of elastic recoil
Increased airway resistance	

Auto-PEEP (intrinsic PEEP): It is the positive pressure remaining in the alveoli at end-expiration due to incomplete exhalation, causing air trapping and dynamic hyperinflation.

Incomplete emptying → Air trapping →
Hyperinflation → Auto-PEEP

3. What are the clinical features of patient with Lung abscess and COPD?

Symptoms:

A. Productive Cough with Foul-Smelling Sputum -

- Long-standing, usually smoker's cough
- Initially intermittent, later persistent, mucoid or mucopurulent sputum due to COPD
- Often worse in the morning
- May suddenly worsen with superadded infection

B. Fever - Moderate to high-grade fever

- Often associated with chills and rigors
- Suggests active suppurative infection

C. Progressive Dyspnea

- Gradual onset and slowly progressive
- Initially occurs only on exertion: Climbing stairs, Walking uphill, Heavy work
- Later with routine activities: Walking on level ground, Dressing/bathing
- Advanced disease: Dyspnea at rest, Difficulty speaking full sentences

Orthopnea: Dyspnea that worsens on lying flat and improves on sitting or standing. Seen in Left ventricular failure / congestive heart failure, Severe COPD, Obesity, Bilateral diaphragmatic paralysis, Massive ascites.

Platypnea: Dyspnea that worsens in upright posture and improves on lying down. Seen in Hepatopulmonary syndrome, Intracardiac shunts (PFO/ASD), Pulmonary AV malformations.

Trepopnea: Dyspnea occurring in one lateral decubitus position but not the other. Seen in Unilateral lung disease (consolidation, abscess), Pleural effusion, Congestive heart failure, Large unilateral mass.

D. Fatigue / Easy Tiredness -

Due to increased work of breathing
• Poor sleep quality

Signs: (explained in detail in later section)

- Tachypnea and increased work of breathing
- Fever
- Clubbing
- Cyanosis

Elaborate association of history of smoking and its anaesthetic importance.

History of smoking is of great importance in COPD patients and an important underlying factor. Duration and number of cigarettes per day are significant which is expressed as pack years.

PACK YEARS : Number of packs of cigarettes/day × number of years of smoking

One pack = 20 cigarettes

> 40 pack years is high risk for post operative pulmonary complications

SMOKING INDEX (SI) : Number of cigarettes /day × total duration (in years)

SI < 100 mild smoker

SI – 100 to 300 moderate smoker

SI > 300 severe smoker

17.25 Modified Medical Research Council (MRC) dyspnoea scale

Grade	Degree of breathlessness related to activities
0	No breathlessness, except with strenuous exercise
1	Breathlessness when hurrying on the level or walking up a slight hill
2	Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace
3	Stops for breath after walking about 100 m or after a few minutes on level ground
4	Too breathless to leave the house, or breathless when dressing or undressing



Anaesthetic importance of smoking :

Post operative pulmonary complications are very common and severe in smokers and can lead to increased morbidity and mortality.

Smoking increases the carboxyhaemoglobin levels due to increased carbon monoxide inhalation.

Increase in carboxy haemoglobin levels

- (a) Decrease the oxygen carrying capacity of haemoglobin,
- (b) Shifts the oxygen dissociation curve to the left and decrease the oxygen release to the tissues
- (c) Produces spuriously high pulse oximeter readings

(oxy-haemoglobin and carboxy-haemoglobin have the same wave length of 940 Nm and hence pulse oximeter reads carboxy-haemoglobin also as oxy haemoglobin. Only Co-oximeter can differentiate between the two and can give the correct saturation readings).

The half life of carboxy haemoglobin is 4-6 hours and stopping smoking for 24 hours is enough to reduce the problems associated with it.

Normal carboxy haemoglobin levels is < 2% in non-smokers.

Whereas it can be as high as 10% in smokers.

Effects of smoking on respiratory system.

- Smoking produces hypertrophy and hyperplasia of mucus secreting glands
- Impairs ciliary motility and mucociliary transport mechanism leading to infection, atelectasis and collapse.
- Inhibits the function of alveolar macrophages and suppresses immune mechanism
- Increases airway resistance due to bronchoconstriction
- Makes the airways Hyperactive .
- Increases Postoperative pulmonary complications
- Induces coronary vasoconstriction producing decreased coronary blood flow.

general physical examination ?

Body Mass Index :

- Obesity (BMI >30kg/m²) decreases FRC with relative increase in closing capacity producing increase in intra pulmonary shunts and decrease in pulmonary reserve.
- Also decreases thoracic and lung compliance producing restrictive lung disease and exaggerating the problems associated with obstructive disease.
- Increased post operative pulmonary complications due to hypoventilation.
- In elective surgeries, obese patient is asked to reduce body weight pre operatively in order to reduce post op pulmonary complications. In this patient BMI is 23kg/m² and hence normal.

Signs of respiratory distress : Tachypnoea, use of accessory muscles of respiration, and cyanosis.

Pallor: anaemia can exacerbate the respiratory problems by increasing the work of breathing due to anaemic hypoxia.

Cyanosis: If cyanosis is present, the arterial haemoglobin saturation with oxygen is 80% or less (Pao₂ <52 mmHg) which indicates an increased ventilation perfusion mismatch and limited margin of respiratory reserve. It may also indicate the presence of respiratory failure and requirement of oxygen therapy/mechanical ventilatory support.

Pedal oedema and raised JVP : Indication of Right heart failure as a result of cor pulmonale.

- Clubbing: May be present and should be graded :

Grade	Feature
Grade 1	Increased fluctuation (sponginess) of the nail bed
Grade 2	Obliteration of the angle between nail and nail bed
Grade 3	Parrot beak appearance (increased nail curvature)
Grade 4	Drumstick appearance (bulbous enlargement of fingertip)
Grade 5	Associated with hypertrophic osteoarthropathy

4. What are the main points to be considered in



VITALS:

Pulse rate : May be irregularly irregular if the patient is in Atrial fibrillation and missed beats which can occur as a result of chronic hypoxia.
Respiratory rate : Tachypnea may be present
Fever : Infection may be present

5. What important findings you would like to get from systemic examination?

Respiratory system :

Position of trachea : If collapse or fibrosis of the lung is present, the trachea may be pulled to the same side.

Respiratory rate and pattern: Rate more than 26 cpm with use of accessory muscles of respiration indicates respiratory distress.

Measurements of chest: Barrel chest in emphysematous patients.

Chest expansion is normally 5cms which can be reduced to < 2cms in COPD patients.

Breath sounds: Bronchial breath sounds may indicate pneumonic consolidation or cavity.
Distant sounds are an indication of emphysema
Decreased breath sounds heard over fibrosis, collapse, pleural effusion, and pneumothorax.

Added sounds -

• Wet sounds (crackles /coarse crepitations) are usually caused by excessive fluid in the airways and indicate sputum retention or edema.
Commonly heard over the areas of bronchiectasis.

• Dry sounds (wheezes/rhonchi) are produced by high-velocity gas flow through narrowed bronchi and are a sign of airway obstruction.

Cardiovascular system:

Pulmonary hypertension to be ruled out
Signs: Palpable P2, Narrowly split second heart sound, Increased intensity of the pulmonary component of the second heart sound and para sternal heave(right ventricular hypertrophy).

If present can be a bad prognostic sign.

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Pulmonary hypertension to be ruled out
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If present can be a bad prognostic sign.

Per abdomen examination:

In right heart failure - Epigastric pulsations – Tender hepatomegaly – Hepatojugular reflex

6. How do you diagnose COPD and what is GOLD'S Criteria for grading of severity of COPD?

A clinical diagnosis of COPD should be considered in any patient who has dyspnea, chronic cough or sputum production and a history of exposure to risk factors for the disease like Tobacco smoke (including popular local preparations).

Spirometry is required to make the diagnosis in this clinical context; the presence of a post-bronchodilator $FEV_1/FVC < 0.70$ confirms the presence of persistent airflow limitation and thus of COPD.

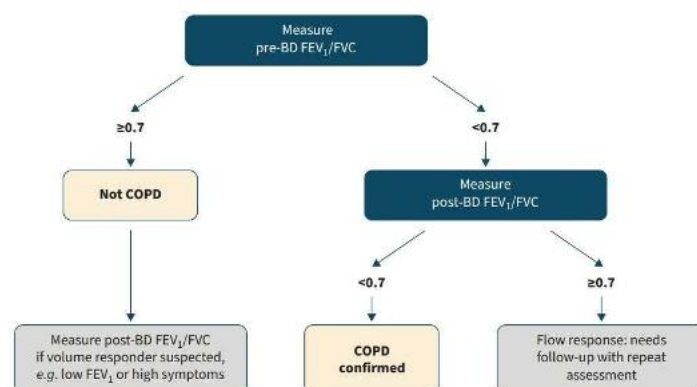


FIGURE 3 Global Initiative for Chronic Obstructive Lung Disease (GOLD) diagnostic pathway for spirometry using forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) ratio. BD: bronchodilator.

GOLD Grades and Severity of Airflow Obstruction in COPD (based on post-bronchodilator FEV_1)

In COPD patients ($FEV_1/FVC < 0.7$):

GOLD 1:	Mild	$FEV_1 > 80\%$ predicted
GOLD 2:	Moderate	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3:	Severe	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4:	Very Severe	$FEV_1 < 30\%$ predicted



7. What investigations should you ask for this patient and what are the ECG and Chest X ray findings ?

Routine Investigations

- Complete blood count : Anemia, Increased WBC count, Increased hematocrit,
- Absolute eosinophil count
 - 2000/cmm → suggests tropical eosinophilia
 - 300/cmm in asthmatics → indicates increased risk of perioperative bronchospasm
- Liver function tests, Renal function tests
- Random blood sugar
- Blood grouping, typing, and cross-matching
- Urine routine examination and Serology

Respiratory evaluation:

- Chest X-ray
- Sputum examination for culture & sensitivity and AFB
- Pulmonary Function Tests (PFTs) – bedside tests and spirometry
- ABG

Chest X-ray

- Emphysematous changes like horizontally placed ribs with wide intercostal spaces, flattened domes of the diaphragm, hyper lucent lung shadows and tubular heart will be present.
- Thick-walled cavitory lesion with a clear air-fluid level consistent with lung abscess.
- Surrounding patchy or dense consolidation around the cavitory lesion indicating active infective process.
- Commonly located in dependent segments (posterior upper lobe or superior lower lobe).
- Adjacent pleural thickening or pleural effusion may be present if associated pleural involvement/empyema exists.
- Possible mediastinal shift or volume loss depending on severity of surrounding collapse or fibrosis.

Preoperative PFT:

PFT with special reference to VC, FEV₁, PEFR.

Criteria which indicates increased mortality & morbidity after lung resection:

FVC <50% of predicted value

FEV₁ <50% of FVC

Maximum breathing capacity <50% of predicted value

Preoperative ABG:

1. Mild to moderate stages: Acute alveolar hyperventilation with hypoxemia (Respiratory alkalosis) - Increased pH, decreased PaCO₂, decreased PaO₂, decreased HCO₃
2. Severe stages: Chronic ventilatory failure with hypoxemia (Compensated respiratory acidosis) Normal pH, Increased PaCO₂, Increased HCO₃ (significantly), Decreased PaO₂

8. Enumerate the static and dynamic lung function tests

Static Tests

- Force is not used and tests are not time-based
- Divided into:
 - o 4 lung volumes
 - o 4 lung capacities
- Two or more lung volumes together form a capacity

Lung Volumes:

1. Tidal volume (TV)
2. Inspiratory reserve volume (IRV)
3. Expiratory reserve volume (ERV)
4. Residual volume (RV)

Lung Capacities:

1. Vital capacity (VC)
2. Total lung capacity (TLC)
3. Functional residual capacity (FRC)
4. Inspiratory capacity (IC)

Dynamic Tests

- Force is used and tests are time-based
- Includes:
 - Maximum breathing capacity (MBC)
 - Forced vital capacity (FVC)
 - Forced expiratory volume in first second (FEV₁)
 - Maximum mid-expiratory flow rate (MMEFR)
 - Peak expiratory flow rate (PEFR)
 - Respiratory muscle strength

9. Describe the bed side lung function tests?

1. Sabrazes Breath Holding Test:

- Used to assess cardiopulmonary reserve



- Patient takes a deep inspiration and holds breath as long as possible
- Examiner listens over trachea for onset of expiration

Interpretation:

- Normal: > 40 seconds
- < 25 seconds → Decreased ventilatory capacity
- < 15 seconds → Poor cardiopulmonary reserve

2. Snider's Match Blowing Test:

- Assesses expiratory capacity and maximum breathing capacity (MBC)
- Patient blows out a lighted candle placed 6 inches away (mouth open, no pursing of lips)

Interpretation:

- Cannot blow out at 6 inches → MBC < 100 L/min, FEV₁ < 1.6 L
- Can blow at 9 inches → MBC > 150 L/min
- Can blow only at 3 inches → MBC ≈ 50 L/min

3. De Bono's Whistle Test:

- Used to estimate Peak Expiratory Flow Rate (PEFR)
- Whistle with adjustable aperture
- Patient blows with aperture fully open

If unable:

- Reduce aperture size gradually
- Note the smallest aperture through which patient can blow
- Corresponding PEFR values are marked on the device

4. Forced Expiratory Time (Tracheal Auscultation):

- Performed after deep inspiration with forced expiration
- Stethoscope placed over trachea; stopwatch used

Interpretation:

- Normal: 3–4 seconds
- 6 seconds → Obstructive airway disease (FEV₁ < 50%)

5. Wright's Respirometer:

- Measures:
 - Tidal volume
 - Minute ventilation

6. Wright's Peak Flow Meter:

- Measures PEFR (Peak Expiratory Flow Rate)

Normal values:

- Males: 450–700 L/min
- Females: 350–500 L/min

Clinical significance:

- < 200 L/min → Inadequate cough efficiency

7. Greene & Berowitz Cough Test:

- Patient takes deep inspiration and coughs once

Assesses:

- Ability to cough
- Strength
- Effectiveness

Inadequate cough if:

- FVC < 20 mL/kg
- FEV₁ < 15 mL/kg
- PEFR < 200 L/min
- Effective cough requires VC ≈ 3 × tidal volume
- Wet/productive cough → risk of pulmonary complications

8. Wheeze Test:

- Patient takes 5 deep breaths
- Auscultation done over posterior lung fields
- Detects presence or absence of wheezing

9. Single Breath Count Test:

- Patient counts numbers aloud after maximal inspiration

Interpretation:

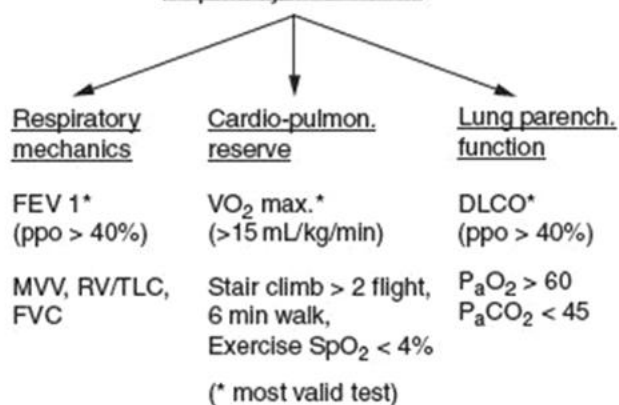
- Normal: ≥ 50
- < 15 → Severe reduction in vital capacity

10. What is the three legged stool assessment of pulmonary function ?

The "three-legged stool" of pre-thoracotomy respiratory assessment is a foundational framework used to estimate a patient's risk of pulmonary complications after thoracic surgery. It involves evaluating three distinct aspects: lung mechanics (spirometry), parenchymal function (gas exchange), and cardiopulmonary interaction (exercise capacity).



The "three-legged" stool of pre-thoracotomy respiratory assessment



11. What is the importance of VO₂ max , 6 min walk test and stairs climbing ?

VO₂ max:

- Formal cardiopulmonary exercise testing is the gold standard for assessing cardiopulmonary function.
- VO₂ max is the most useful predictor of post-thoracotomy outcome.

Key Points:

- Preoperative VO₂ max < 15 mL/kg/min** → high risk of morbidity and mortality
- Predicted postoperative VO₂ max (ppoVO₂ max < 10 mL/kg/min)** → absolute contraindication to pulmonary resection (very high mortality)

6-Minute Walk Test (6MWT):

- Distance walked correlates well with VO₂ max
- Simple test requiring minimal equipment

Interpretation:

- Distance < 610 m (2000 ft) → correlates with VO₂ max < 15 mL/kg/min

Stair Climbing Test:

- Performed at patient's own pace without stopping
- Measured in number of flights climbed

Note:

- 1 flight ≈ 20 steps (each ~6 inches)

Performance	VO ₂ Max Equivalent	Interpretation
> 5 flights of stairs	> 20 mL/kg/min	Low mortality after pneumonectomy (FEV ₁ > 2 L)
> 3 flights of stairs	> 15 mL/kg/min	Low mortality after lobectomy (FEV ₁ ≈ 1.7 L)
< 2 flights of stairs	< 12 mL/kg/min	High mortality risk
< 1 flight of stairs	< 10 mL/kg/min	Very high risk
6-minute walk = 610 m	≈ 15 mL/kg/min	Acceptable functional capacity

12. What is predicted postoperative FEV1 (ppoFEV1) and how is it calculated?

Predicted postoperative FEV1 (ppoFEV1) is the estimated lung function remaining after lung resection surgery (lobectomy/pneumonectomy).

It helps determine:

- Operability
- Perioperative pulmonary risk
- Whether patient can tolerate resection

$$\text{ppoFEV1} = \text{Preoperative FEV1} \times \left(\frac{\text{Remaining functional segments}}{\text{Total functional segments}} \right)$$

Example : Right Upper Lobectomy:

Right upper lobe = 3 segments removed

Remaining segments = 19 – 3 = 16

ppoFEV1 = Pre-op FEV1 × (16/19)

From perfusion scan:

ppoFEV1 = Pre-op FEV1 × (1 – fraction of perfusion to resected lung)

- ppoFEV1 > 60% predicted: Low risk
- ppoFEV1 30–60%: Moderate risk → Further testing (DLCO, CPET)
- ppoFEV1 < 30%: High risk / major surgery often contraindicated



13. How do you prepare this patient for surgery ?

1. Abstinence from Smoking

- Stop smoking for > 4–8 weeks → reduces postoperative complications
- Within 12–48 hours:
 - ↓ COHb levels
 - ↑ Oxygen carrying capacity
 - Oxygen dissociation curve shifts to normal

Time Course	Beneficial Effect
12–24 hours	↓ CO & nicotine levels
48–72 hours	COHb normalizes, ciliary function improves
1–2 weeks	↓ sputum production
4–6 weeks	Pulmonary function tests improve
6–8 weeks	Immune function & metabolism normalize
8–12 weeks	↓ postoperative morbidity & mortality

2. Dilating Airways

Drugs:

- Bronchodilators & sympathomimetics
- Parasympatholytics
- PDE inhibitors

Aminophylline

- Loading dose: 5 mg/kg
- Smokers: ↑ dose required (hepatic enzyme induction)
- Under GA: reduce dose by 2 mg/kg

Infusion:

- Normal: 0.5 mg/kg/hr
- Smokers: 0.7 mg/kg/hr
- Under GA: reduce by 0.2 mg/kg/hr
- Therapeutic level: 10–20 mg/L

Advantages:

- Stimulates respiratory center
- Increases diaphragmatic contractility

- Direct bronchodilation
- Bronchodilation via PDE inhibition (↑ cAMP, ↑ catecholamines, anti-adenosine effect)
- Improves cardiac inotropy
- ↓ Peripheral vascular resistance → ↑ cardiac output

Disadvantages:

- ↑ Cardiac automaticity → risk of arrhythmias

Important Notes:

- Interacts with halothane → cardiac dysrhythmias
- Halothane can be used 4 hours after last dose
- Not routinely used now due to dysrhythmic effects

Other Drugs:

- Steroids
- Cromolyn sodium (prophylactic)

3. Loosening Secretions

- Mucolytics: N-acetylcysteine, potassium iodide
- Airway hydration
- Humidification (ultrasonic nebulizers)
- Systemic hydration (correct hypovolemia & electrolyte imbalance)

4. Removing Secretions

- Postural drainage
- Coughing
- Chest percussion & vibration
- Forced expiration technique

5. Adjunct Medications

- Antibiotics (after culture & sensitivity)
- Antacids (H₂ blockers / proton pump inhibitors) if reflux present

6. Measures to Increase Patient Motivation for surgery

- Psychological preparation
- Preoperative pulmonary care training
 - Incentive spirometry
 - Secretion removal techniques
- Preoperative exercises
- Weight optimization (loss/gain)
- Stabilization of comorbid conditions



14. What are the Anesthetic goals and considerations?

Anaesthetic goals:

- Maintain oxygenation
- Minimize intra-pulmonary shunt
- Lung-protective ventilation
- Optimal surgical exposure
(Ensure complete collapse of operative lung)
- Prevent contamination
- Maintain hemodynamic stability
- Provide effective analgesia.

Anaesthetic considerations:

- Airway: One lung ventilation
- Risk: aspiration, pus contamination, hypoxia during OLV
- Ventilation: lung-protective strategy with FiO_2 titration
- Position: left lateral decubitus
- Monitoring: standard ASA monitors + arterial line
- Analgesia: Thoracic epidural, Paravertebral block (discussed later)

15. Discuss about intra-operative management.

A. Secure Venous Access

- At least one large-bore IV (14G/16G)

B. Monitoring

- Invasive BP monitoring - Beat to beat monitoring
 - Indicated in:
 - Major resections
 - Poor pulmonary reserve
 - Cardiovascular disease
- Central Venous Access
 - Used in:
 - Pneumonectomy
 - Note:
 - Place on same side as thoracotomy
 - Prevent pneumothorax on ventilated lung side
- Special Situations
 - TEE (Transesophageal Echo):
 - CAD
 - Pulmonary hypertension
- Arterial Blood Gas (ABG)
 - Ensures:

- Adequate oxygenation
- Adequate ventilation

5. End-Tidal CO_2 (ET CO_2)

- Useful for trend monitoring
- Limitation:
 - Inaccurate due to $\uparrow$ dead space
 - Variable PaCO_2 -ET CO_2 gradient

A. Induction of Anesthesia

- Based on patient's condition and co-morbidities
- Ensure:
 - Adequate preoxygenation
 - Adequate depth of anesthesia
- Prevent:
 - Bronchospasm
 - Hemodynamic response to laryngoscopy
- Incremental induction agent dosing
- Opioids
- Volatile agents (useful in reactive airway disease)
- Additional Benefits
 - Volatile agents:
 - May protect lungs during OLV

D. Positioning (Lateral Decubitus Position)

- Most thoracic surgeries use this position
- Lower arm: flexed
- Upper arm: extended forward
- Pillows:
 - Between arms
 - Between legs
- Axillary roll:
 - Placed under dependent axilla
 - Reduces shoulder pressure
- Care:
 - Protect eyes
 - Protect dependent ear
 - Avoid brachial plexus injury

A. Maintenance of Anesthesia

- Provide high inspired oxygen concentration
- Allow rapid adjustment of anesthetic depth
- Maintain hemodynamic stability
- Suppress airway reflexes

a. Inhalational Agents (Halogenated Agents)

- Dose-dependent bronchodilation
- Depression of airway reflexes



- Minimal effect on hypoxic pulmonary vasoconstriction (HPV) at <1 MAC

b. Opioids

Advantages:

1. Minimal hemodynamic effects
2. Suppress airway reflexes
3. Provide postoperative analgesia

Important Note:

- If epidural/intrathecal opioids planned →
→ Minimize intraoperative IV opioids
→ Prevent postoperative respiratory depression

c. Neuromuscular Blockade

- Use nondepolarizing muscle relaxants (NDMR)

Benefits:

- Facilitates rib retraction
- Improves surgical exposure
- Enhances ventilation control

d. Fluid Management

Risks of Excess Fluid:

- Acute Lung Injury (ALI)
- Lower Lung Syndrome:
 - Fluid shifts into dependent lung (lateral position)
 - ↑ shunt → hypoxemia

Current Approach:

- Goal-directed fluid therapy (GDFT)
 - Avoid both overload and hypovolemia

e. Lung Protection During Surgery

- Collapsed lung prone to:
 - Ischemia-reperfusion injury
 - Surgical trauma

Protective Lung Ventilation Strategy

Ventilation Parameters:

- Low tidal volume: <6 mL/kg
- FiO₂: 50–80% (avoid 100% unless necessary)
- Plateau pressure: <25 cm H₂O
- Peak airway pressure: <35 cm H₂O

Ventilation Mode:

- Pressure-controlled ventilation (PCV) preferred

Advantages:

- Limits barotrauma
- Better tidal volume distribution
- Reduces dead space ventilation

Lung Reinflation (End of Surgery):

- Gradual inflation:
 - Peak pressure <30 cm H₂O
- Prevent:
 - Staple line disruption

Technique Tip:

- Clamp dependent lung lumen → prevent overdistention

f. Surgical Steps Relevant to Anesthesia

- Bronchial stump:
 - Checked for air leak at 30 cm H₂O pressure
- Before closure:
 - Manual lung expansion under vision
- Post-expansion:
 - Resume controlled ventilation
 - Connect chest drains to suction

16. What are the physiological changes in the lateral decubitus position (one-lung ventilation)?

1. Blood Flow (Perfusion):

- Gravity creates a vertical gradient in pulmonary blood flow.
- Dependent lung → receives more perfusion
- Nondependent lung → receives less perfusion
- Distribution (approx.):
 - Dependent lung: ~60%
 - Nondependent lung: ~40%
- Within each lung:
 - Lower zones > upper zones (Zone 3 > Zone 2 > Zone 1)
- In open chest:
 - Distribution usually unchanged
 - However, if nondependent lung compliance ↑ significantly → vascular resistance ↓ → may ↑ nondependent perfusion

1. Ventilation :

- Ventilation is greater in the dependent lung in awake, spontaneous breathing patients due to:
 - More negative pleural pressure gradient
 - Better diaphragmatic excursion (dependent diaphragm more efficient)
- Dependent alveoli lie on a steeper part of compliance curve → receive more tidal volume



- Nondependent alveoli are more expanded at baseline → less compliant → less ventilation
- **In anaesthetised patients**, contrast to the awake state, ventilation shifts predominantly to the **nondependent lung**.

• Causes:

- General anaesthesia ↓ FRC and lung volumes
- Dependent lung moves to **low-volume, less compliant (flat) part** of pressure–volume curve
- Nondependent lung moves to **more compliant (steep) part** of curve
- Compression of dependent lung by mediastinum, abdominal contents, and positioning
- Reduced diaphragmatic function
- Poor mucociliary clearance → risk of **atelectasis**

3. Ventilation–Perfusion (V/Q) Relationship:

- In awake state:
 - V/Q matching is relatively good (both ventilation and perfusion favor dependent lung)
- **Major V/Q mismatch develops in anesthetised patients:**
 - Nondependent lung → well ventilated but poorly perfused (**dead space effect**)
 - Dependent lung → well perfused but poorly ventilated (**shunt effect**)

Leads to hypoxemia

4. Diaphragm Mechanics:

- Dependent hemidiaphragm:
 - More curved
 - Contracts more effectively
- Nondependent hemidiaphragm:
 - Less efficient movement

1. Pleural Pressure Gradient:

- More negative at apex, less negative (or positive) at base
- Causes regional differences in alveolar size and compliance

2. Effect of Open Chest and Paralysis:

- Open chest ↑ compliance of **nondependent**

lung → further ↑ its ventilation

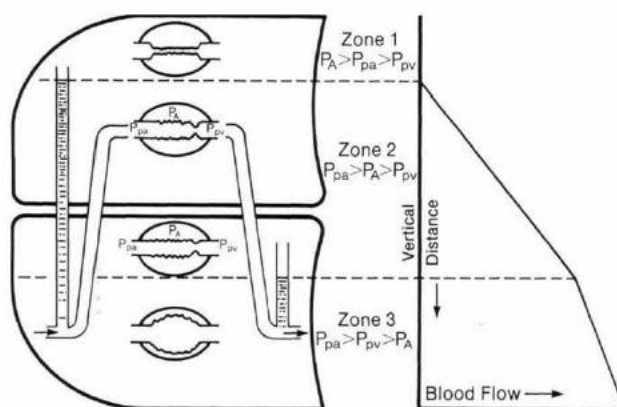
- Paralysis reduces diaphragmatic resistance → ventilation favors nondependent lung
- Dependent lung ventilation further decreases

3. Effect during One-Lung Ventilation:

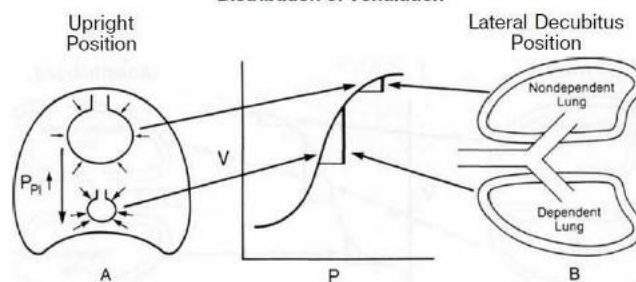
(Anesthetized Patient – important)

- Ventilation shifts to nondependent lung (due to mechanical ventilation)
- Perfusion remains higher in dependent lung
- Leads to V/Q mismatch and shunt
- Hypoxic pulmonary vasoconstriction (HPV) helps reduce shunt by diverting blood away from nonventilated lung

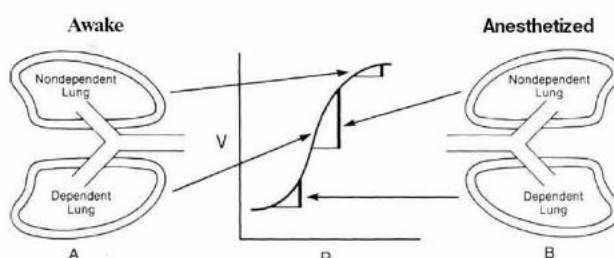
Distribution of Blood Flow
Lateral Decubitus Position

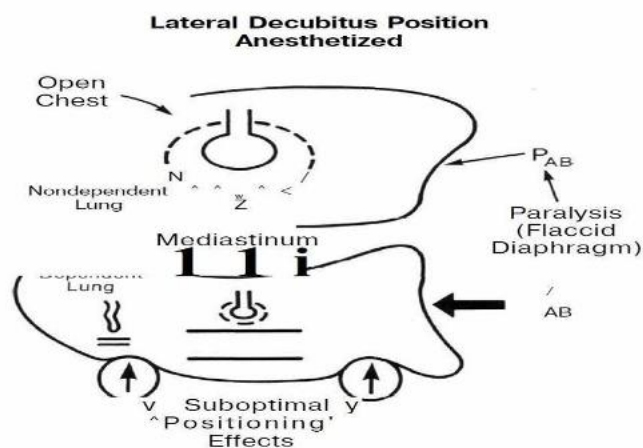
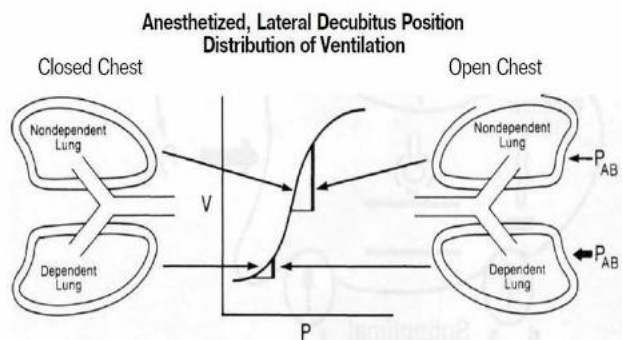


Awake, Closed Chest
Distribution of Ventilation



Closed Chest, Lateral Decubitus Position
Distribution of Ventilation





17. What is One lung ventilation and what are the physiological changes affecting oxygenation and shunt during one-lung ventilation in an anaesthetized patient?

One-lung ventilation (OLV) is a specialized anesthetic technique in which one lung is intentionally ventilated while the other lung is isolated and allowed to collapse.

1. Shunt Formation:

- During one-lung ventilation (OLV), the nondependent lung is not ventilated but still perfused, leading to a right-to-left intrapulmonary shunt
- Shunt fraction may increase significantly ($\approx 20\text{--}30\%$) $\rightarrow$ major cause of hypoxemia

2. Effect of Anaesthetic Agents:

- Volatile anaesthetics (e.g., isoflurane, sevoflurane) inhibit hypoxic pulmonary vasoconstriction (HPV)
- This increases blood flow to the nonventilated lung $\rightarrow$ worsens shunt and hypoxemia
- Higher MAC $\rightarrow$ greater inhibition of HPV

3. Hypoxic Pulmonary Vasoconstriction (HPV):

- Protective mechanism that diverts blood from nonventilated lung $\rightarrow$ ventilated lung
- Reduces shunt fraction and improves oxygenation
- Blunted by:
 - Volatile agents (dose dependent)
 - Excessive vasodilators
 - High pulmonary artery pressures

4. Lung Volumes and Compliance:

- Dependent lung:
 - $\downarrow$ FRC due to anaesthesia, muscle relaxation, and compression
 - $\uparrow$ risk of atelectasis $\rightarrow$ $\downarrow$ ventilation
- Nondependent lung:
 - More compliant $\rightarrow$ receives ventilation (if ventilated) but may be collapsed during OLV

5. Pressure–Volume Relationship:

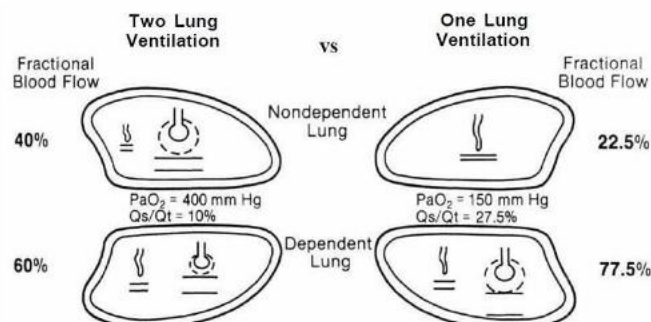
- Dependent lung operates on low-compliance (flat) portion of curve
- Nondependent lung operates on high-compliance (steep) portion
- Leads to preferential ventilation away from dependent lung

6. V/Q Mismatch:

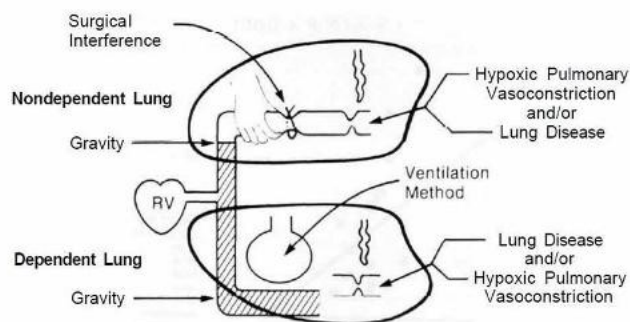
- Dependent lung $\rightarrow$ low V/Q (shunt-like)
- Nondependent lung $\rightarrow$ high V/Q or no ventilation (true shunt during OLV)
- Overall effect $\rightarrow$ impaired oxygenation

7. Factors Worsening Oxygenation:

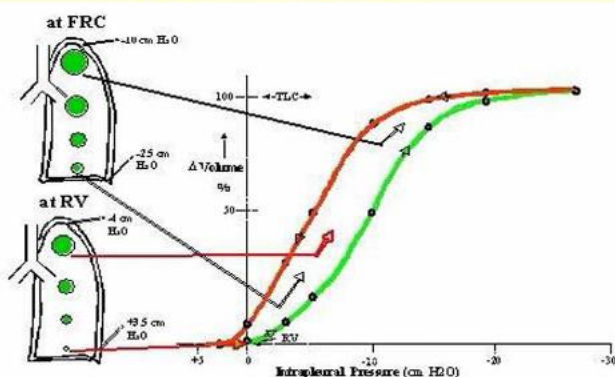
- High $\text{FiO}_2 \rightarrow$ absorption atelectasis
- Poor positioning
- Low PEEP in dependent lung
- Excessive airway pressures
- Open chest conditions



One Lung Ventilation: Determinants Of Blood Flow Distribution



Compliance of Top Alveoli and Bottom Alveoli at Different Lung Volumes



18. What are the indications for lung isolation?

Absolute Indications:

A. Patient-related:

- Protection of healthy lung from contamination (infection, massive hemoptysis)
- Need for independent lung ventilation
 - Bronchopleural fistula
 - Tracheobronchial injury
 - Cyst or bulla
- Severe hypoxemia due to unilateral lung disease

B. Procedure-related:

- Surgical opening of major airways
- Repair of bronchial disruption
- Thoracic aortic aneurysm surgery
- Esophageal surgery
- Bronchoalveolar lavage (one lung)
- Anterior approach to thoracic spine

Relative Indications:

- Need for lung collapse to improve surgical exposure:
 - Thoracoscopy
 - Pleural surgery
 - Lobectomy
 - Pneumonectomy
 - Bullectomy
 - Lung volume reduction surgery
 - Descending thoracic aorta surgery
 - Pulmonary endarterectomy

19. What are the different lung isolation techniques ?

Methods of Lung Isolation:

- Double-lumen endobronchial tubes (DLT):
 - Left-sided (commonly used), Right-sided
 - Types: Carlens, Robertshaw
- Bronchial blockers (BB):
 - Passed through a single-lumen tube to occlude a main bronchus
- Single-lumen endobronchial intubation (EBT):
 - Advancing a standard tube into one main bronchus

Methods in Children:

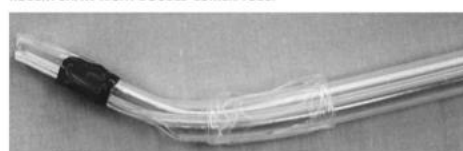
- 0–6 months: Single-lumen endobronchial intubation (preferred)
- 2–8 years: Bronchial blocker (coaxial technique)
- Alternatives:
 - Fogarty catheter
 - Miller atrioseptostomy catheter
- 8–18 years: Marraro double-lumen tube

There is no double-lumen tube small enough for a child less than about 8 years of age.

MARRARO'S BILUMEN PAEDIATRIC ENDOTRACHEAL TUBE:

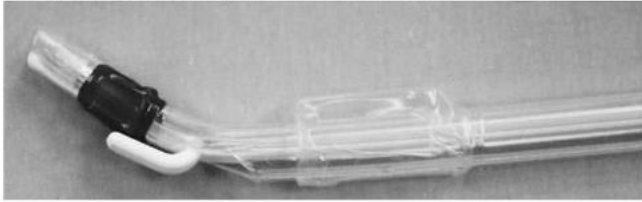


ROBERT SHAW RIGHT DOUBLE - LUMEN TUBE:

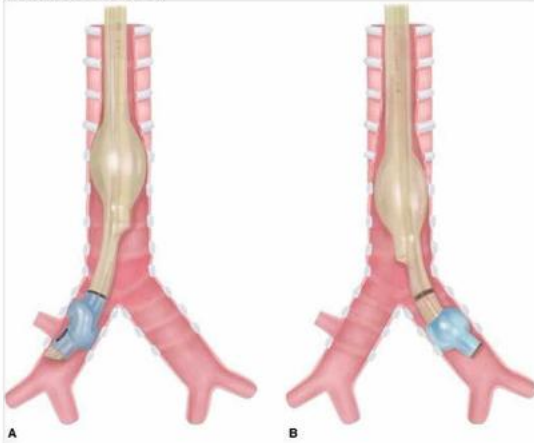


The bronchial portion of the Robertshaw right DLT is angled at 20 degrees. The bronchial cuff has a slotted opening in its lateral aspect. The Robertshaw left DLT - The angle of the bronchial portion is 40 degrees

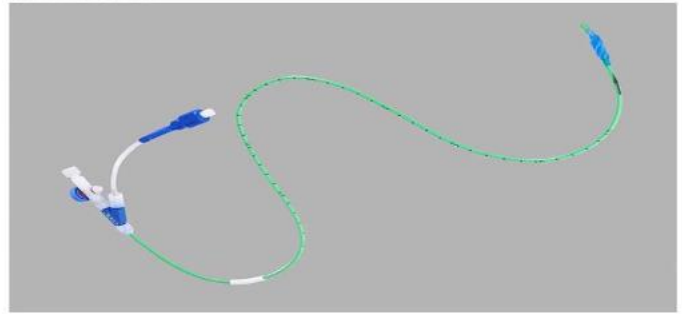
CARLENS DOUBLE-LUMEN TUBE:



RIGHT AND (B) LEFT POLYVINYL CHLORIDE (PVC) (MALLINCKRODT) DOUBLE-LUMEN TUBES (DLT)

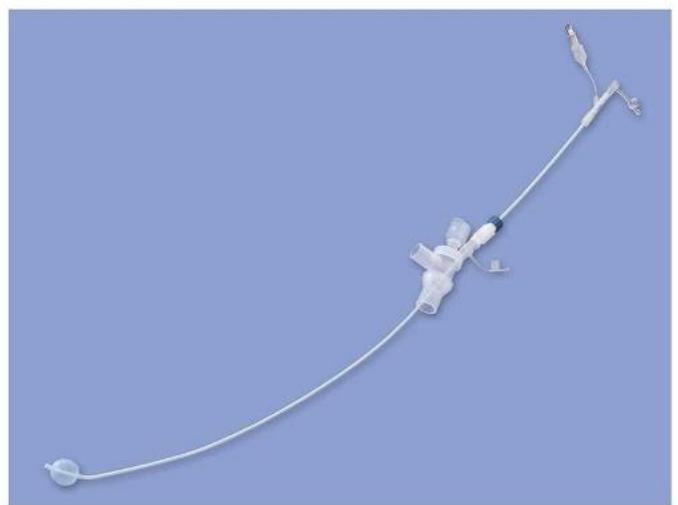


COHEN BLOCKER:



- The Cohen blocker has a proximal wheel that controls the direction of the distal tip
- The tip is pre-angled to help enter the desired bronchus

FUJI UNIBLOCKER:



EZ BLOCKER:

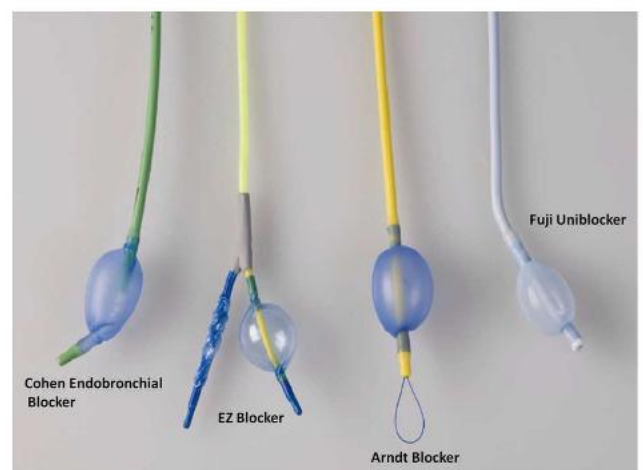
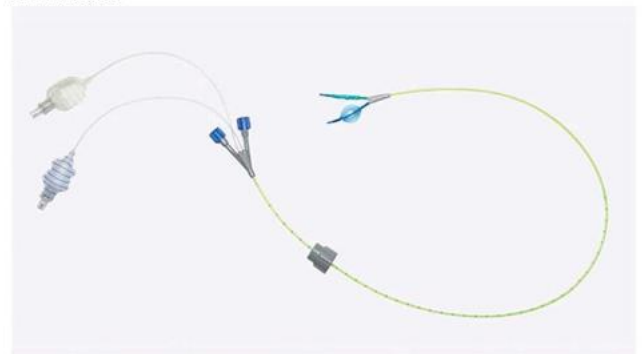


Table 49.7

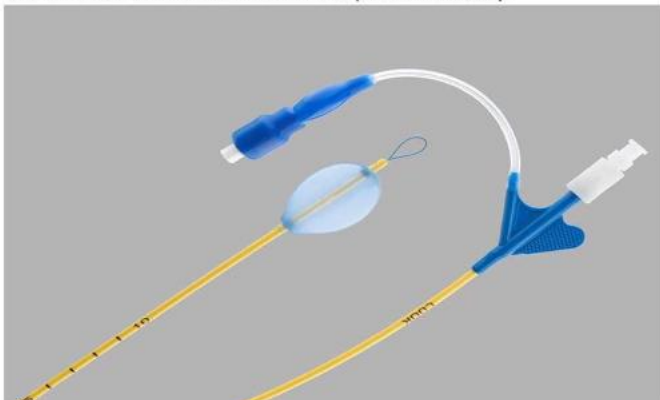
Selection of Double-Lumen Tube (DLT) Size Based on Adult Patients' Sex and Height

Sex	Height (cm)	Size of DLT (Fr)
Female	<160 (63 in.)*	35
Female	>160	37
Male	<170 (67 in.)**	39
Male	>170	41

* For females of short stature (<152 cm or 60 in.), examine bronchial diameter on CT scan, consider 32 Fr.

** For males of short stature (<160 cm), consider 37 Fr.

WIRE-GUIDED ENDOBRONCHIAL BLOCKER (ARNDT BLOCKER):



The Arndt blocker has a retractable loop that is placed over the fiberoptic bronchoscope, which is then used to guide the blocker into place.

20. What are the advantages and disadvantages of double-lumen tubes (DLT) and endobronchial blockers (BB)?

1. Double-Lumen Tubes (DLT):

Advantages:

- Ability to ventilate or collapse either lung independently
- Allows rapid lung isolation
- Provides access to both lungs for:
 - Endotracheal suction
 - Application of CPAP to nondependent lung

Disadvantages:

- Malposition is common
- Risk of laryngeal and bronchial trauma
- Bronchial cuff (low-volume, high-pressure) → mucosal injury
- Tracheal cuff may tear during insertion
- Difficult insertion in poor laryngoscopic view
- Requires exchange to single-lumen tube if postoperative ventilation needed

1. Endobronchial Blockers (BB):

Advantages:

- Useful in children or small patients (where DLT not feasible)
- Can be used in emergency with existing endotracheal tube (ETT)
- Allows selective lobar blockade
- Safer in difficult airway
- No need to change tube for postoperative ventilation

Disadvantages:

- Requires fiberoptic bronchoscopy (FOB) for placement
- Suctioning difficult (balloon must be deflated)
- More prone to dislodgement during positioning or surgery

21. Describe the procedure of one lung ventilation with DLTs.

For laryngoscopy, the shoulder of a Macintosh blade provides better tongue displacement and more space through which to insert the tube.

The use of a video laryngoscope has also been described as effective and potentially time-saving.

The bronchial tip of the DLT is placed through the cords, and the stylet is then removed to prevent trauma.

After the bronchial portion has passed the cords, the DLT must be rotated 90 degrees toward the selected side of the bronchial lumen to sit properly.

If resistance is encountered on rotating or advancing the tube, the use of a smaller tube needs to be considered. The average depth of insertion is 29 cm for a 170-cm individual.

For each 10-cm increase or decrease in height, the tube depth is increased or decreased by 1 cm, respectively.

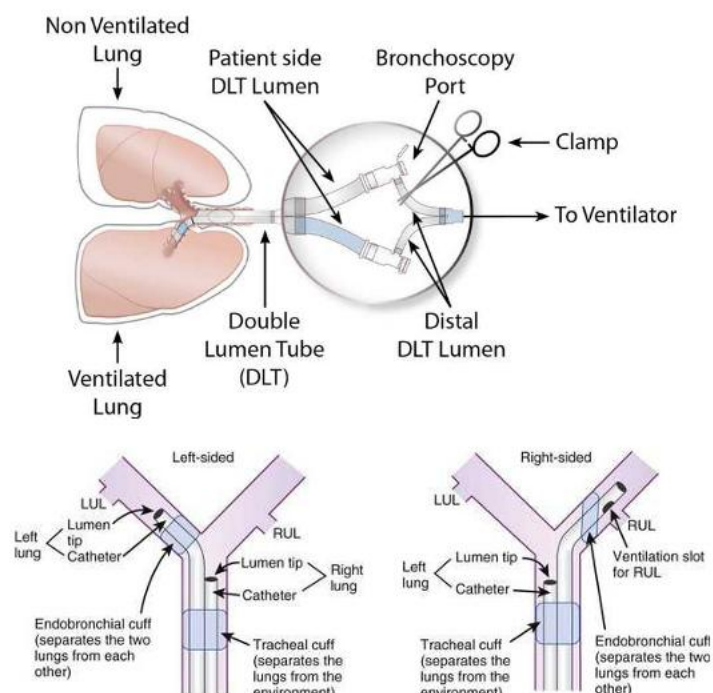
When the tube depth is reached, the tracheal cuff is inflated, and the patient is connected to the ventilator.

Then using Fiberoptic bronchoscope, the position is confirmed and the balloon of the bronchial lumen should be inflated under direct vision and should lie just distal to the carina.

Left sided tube is mostly preferred for one-lung ventilation:

The distance between the right upper lobe bronchus and the right mainstem bronchus is 2.1 cm in females and 2.3 cm in males. The distance between the left upper lobe bronchus and the left mainstem bronchus is 5 cm in females and 5.3 cm in males.

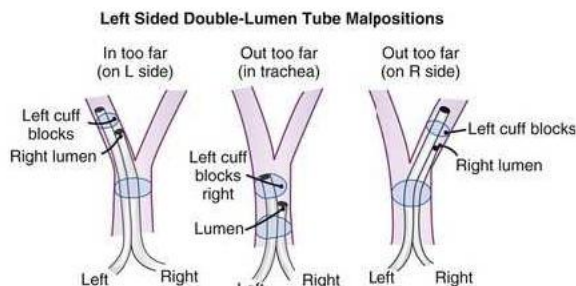
Therefore, it is less likely one will inadvertently obstruct the left upper lobe with the bronchial cuff of a left-sided tube than the possibility that one will inadvertently obstruct the right upper lobe with the bronchial cuff of a right-sided tube.



23. What are the methods of confirming correct placement of a double-lumen tube (DLT)?

A. Clinical Methods:

- Auscultation:
 - Clamp tracheal lumen → check unilateral (left/right) breath sounds
 - Clamp bronchial lumen → confirm ventilation of opposite lung



Procedure	Breath sounds heard		
Clamp right lumen; both cuffs inflated	Left	Left and right	Right
Clamp left lumen; both cuffs inflated	None or faint right	None or very	None or faint left
Clamp left lumen; deflate left cuff	Left only or left and faint right	Left and right	Right only or right and faint left

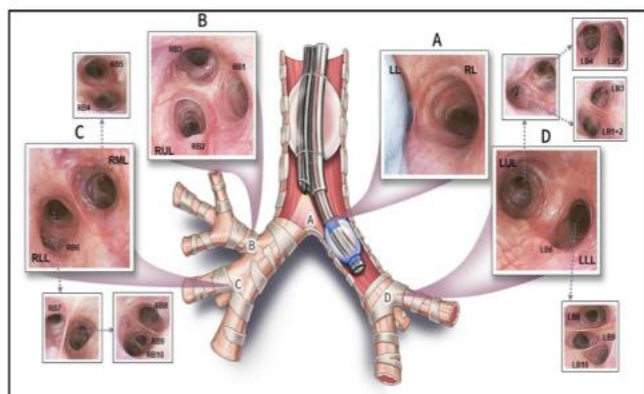
- Observe chest movement
- Confirm CO₂ on capnography

Stepwise Check:

1. Inflate tracheal cuff → confirm bilateral ventilation
2. Inflate bronchial cuff → isolate lung
3. Clamp lumens alternately → check selective ventilation
4. Reassess after lateral positioning

B. Best Method (Gold Standard):

- Fiberoptic bronchoscopy (FOB):
 - Tracheal lumen → see carina
 - Bronchial lumen → confirm entry into correct bronchus
 - Visualize bronchial cuff just below carina



24. What are the complications that can occur with one-lung ventilation despite correct tube placement?

- Hypoxemia
- Hypercapnia
- Increasing airway pressure
- Air trapping
- Pneumothorax
- Arrhythmias
- Hypotension

1. Hypoxemia (most common):

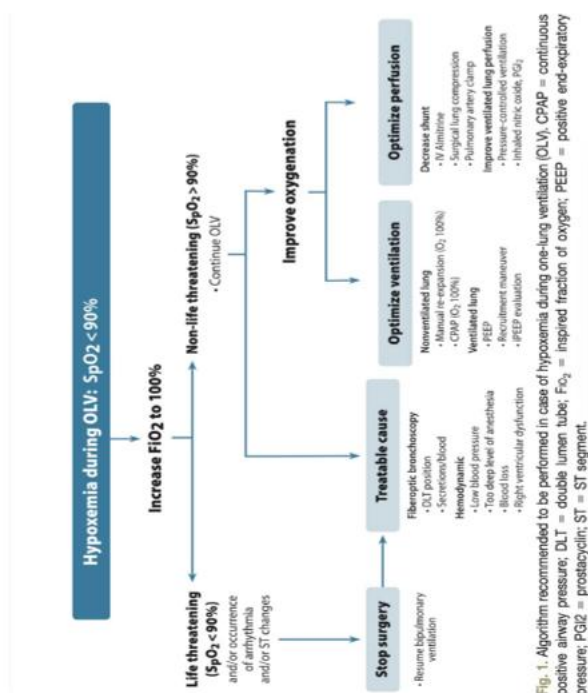
Cause:

- Shunt through the non-ventilated lung
- V/Q mismatch

Management:

- Check tube position (first step) → malposition is the most common reversible cause
- Increase FiO₂ to 100%
- Apply PEEP to dependent lung → improves oxygenation
- Apply CPAP (1–5 cm H₂O) to non-dependent lung → reduces shunt
- Ensure adequate tidal volume (protective: 4–6 mL/kg)
- Intermittent two-lung ventilation if severe

Key idea: Improve oxygenation by reducing shunt and improving ventilation-perfusion matching.



2. Hypercapnia

Cause:

- Reduced ventilated lung surface area
- Hypoventilation

Management:

- Increase respiratory rate (preferred over tidal volume)
- Slightly increase tidal volume if safe
- Check for airway obstruction or kinking
- Accept permissive hypercapnia (if mild and patient stable)

3. Increasing Airway Pressure

Cause:

- Tube obstruction/kinking
- Secretions
- Bronchospasm
- Reduced lung compliance

Management:

- Suction airway
- Check tube position with fiberoptic bronchoscope
- Treat bronchospasm → bronchodilators
- Reduce tidal volume
- Assess for pneumothorax

4. Air Trapping (Auto-PEEP):

Cause:

- Inadequate expiratory time
- Obstructive airway disease

Management:

- Increase expiratory time (reduce RR)
- Reduce tidal volume
- Treat bronchospasm
- Monitor intrinsic PEEP

5. Pneumothorax:

Cause:

- Barotrauma
- Surgical injury

Management:

- Suspect if sudden hypoxia + ↑ airway pressure + hypotension
- Immediate needle decompression
- Insert chest tube
- Switch to two-lung ventilation

6. Arrhythmias

Cause:

- Hypoxia
- Hypercapnia
- Surgical manipulation

Management:

- Correct hypoxia and hypercapnia
- Treat electrolyte imbalance
- Use antiarrhythmics if needed
- Ensure adequate anesthesia depth

7. Hypotension

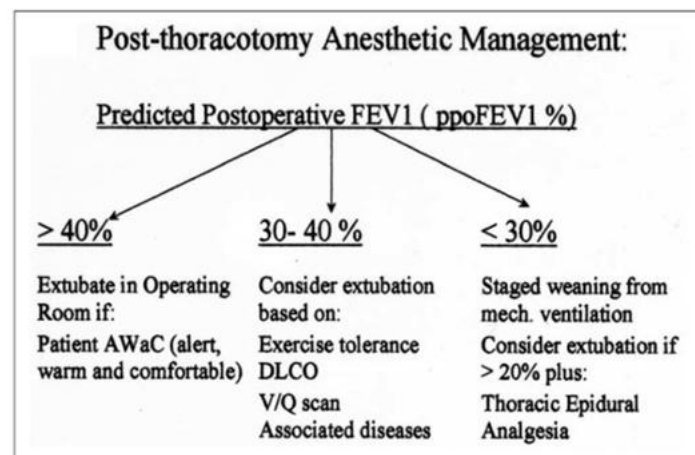
Cause:

- Reduced venous return (PEEP, high airway pressures)
- Anesthetic drugs
- Blood loss

Management:

- Reduce airway pressures/PEEP if excessive
- Fluid resuscitation
- Vasopressors (e.g., norepinephrine)
- Control bleeding

25. What is the criteria for extubation post-operatively?



26. What are the common post operative complications and causes of post operative hypoxemia ?

1. Pulmonary Complications:

- Infections:
 - Atelectasis
 - Pneumonia
 - Empyema
 - Mediastinitis
- Pneumothorax and tension pneumothorax
- Post-pneumonectomy pulmonary edema
- Torsion of residual lobe



- Postoperative respiratory failure

2. Cardiovascular Complications:

- Right-sided heart failure
- Dysrhythmias
- Right-to-left shunting (through foramen ovale)
- Myocardial ischemia and infarction
- Postoperative hypotension
- Herniation of heart

3. Anatomical Structure Injuries:

- Phrenic nerve injury
- Recurrent laryngeal nerve injury
- Spinal cord injury
- Brachial plexus injury
- Thoracic duct injury
- Injuries related to lateral decubitus position

1. Wound Infection and Sepsis

Causes of Postoperative Hypoxia:

1. Diffusion Hypoxia (Fink Effect)

2. Increased V/Q Mismatch

- Anaesthesia ↓ FRC due to:
 - ↓ tone of diaphragm
 - Diaphragm positioned higher in thorax (↑ intra-abdominal pressure)
- More pronounced in:
 - Elderly
 - Smokers
- ↑ Closing capacity may exceed FRC → ↑ intrapulmonary shunt

3. Reduced Cardiac Output

- ↓ Oxygen delivery (O_2 flux) → may not meet metabolic demand
- Worse in conditions like:
 - Shivering
- ↓ Mixed venous PO_2 → ↓ arterial PO_2
- Further worsened by:
 - Anaemia

4. Hypoventilation

(a) Drugs

- Most anaesthetic drugs depress ventilation
- Residual effects of:

- Premedication
- Induction agents
- Maintenance agents
- Analgesics

• Common causes:

- Opioids
- Muscle relaxants

(b) Airway Obstruction

- Partial obstruction in postoperative period
- Often not recognized early

© Pain

• Prevents:

- Deep breathing
- Effective coughing

• Leads to:

- Hypoventilation
- Atelectasis
- Alveolar collapse

(d) Intraoperative Hyperventilation

- Causes ↓ CO_2 (CO_2 deficit)
- Leads to hypoventilation when patient resumes spontaneous breathing

(e) External Factors

- Tight abdominal binders after surgery

Post-pneumonectomy syndrome: It is a rare but serious late complication following pneumonectomy caused by excessive mediastinal shift and rotation into the empty hemithorax.

- This mediastinal displacement leads to compression of the trachea or main bronchus, producing central airway obstruction.
- It is more commonly seen after right pneumonectomy, where the left main bronchus may be compressed between the aorta and pulmonary artery.
- Clinical presentation: May occur months to years after surgery and includes progressive dyspnea, stridor, wheeze, recurrent respiratory infections, and respiratory distress.
- Diagnosis: Chest X-ray, CT imaging, and bronchoscopy demonstrating marked mediastinal shift with airway compression.



- Differential diagnoses : tumor recurrence, bronchial stenosis, and vocal cord dysfunction.
- Definitive treatment is surgical repositioning of the mediastinum with insertion of saline or silicone tissue expanders into the post-pneumonectomy space. Early recognition and intervention are crucial to prevent severe airway compromise and improve long-term respiratory outcomes.

27. What are the special considerations for patients undergoing lung resection?

1. Massive Pulmonary Hemorrhage

Definition & Causes:

- Defined as massive amount (around 500–600 mL) blood expelled in 24 hours
- Causes:
 - Tuberculosis
 - Bronchiectasis
 - Neoplasm
 - Biopsy complications

Key Risk:

- Death mainly due to asphyxia (airway obstruction by blood/clot)

Management:

- Maintain airway + give 100% O₂
- Avoid sedation in non-intubated hypoxic patients
- Rapid sequence induction (RSI) (full stomach)
- Use:
 - Double-lumen tube (DLT) → protects normal lung
 - Or single-lumen tube + bronchial blocker
- Bronchial blocker/Fogarty catheter → tamponade bleeding
- Rigid bronchoscopy / embolization may be done
- Secure large-bore IV access

2. Pulmonary Cyst & Bulla:

Problem:

- Risk of rupture → tension pneumothorax during ventilation

Management:

- Maintain spontaneous ventilation during induction
- Avoid positive pressure until lung isolation
- Use DLT or chest tube before controlled ventilation
- Avoid nitrous oxide (expands cyst)
- Treat rupture immediately:
 - Chest tube insertion

3. Lung Abscess:

Problem:

- Risk of contamination of healthy lung

Management:

- Rapid sequence induction + DLT
- Inflate cuffs immediately after intubation
- Position:
 - Diseased lung → nondependent
- Frequent suctioning of affected lung

4. Bronchopleural Fistula:

Problems:

- Large air leak → difficult ventilation
- Risk of:
 - Tension pneumothorax
 - Contamination (if empyema present)

Management:

- Use double-lumen tube → isolates fistula
- Prefer one-lung ventilation (healthy lung)
- Drain empyema before closure
- Early extubation after surgery



28. Explain the various methods for post-operative pain management.

Opioid-sparing / multimodal analgesia

Modality	Drug / Dose	Mechanism	Advantages	Disadvantages / Complications
Thoracic Epidural Analgesia (TEA) - Gold standard	- Bupivacaine 0.125–0.25% (5–10 ml) - Infusion: 0.1–0.125% + Fentanyl 2 mcg/ml @ 5–10 ml/hr	Blocks thoracic nerve roots (T4–T8), sympathetic block	- Superior analgesia - decreased pulmonary complications - early mobilization	- Hypotension (sympathetic block) - Bradycardia - Epidural hematoma (in anticoagulated pts) - Infection/abscess - Respiratory depression (with opioid)
Paravertebral Block (PVB)	- Bolus: 15–20 ml LA - Infusion: 0.2% Ropivacaine @ 5–10 ml/hr	Unilateral somatic + sympathetic block	Comparable analgesia to TEA, less hypotension	- Pneumothorax - Vascular puncture - LA toxicity
Intercostal Nerve Block	3–5 ml LA per level (multiple levels needed)	Blocks intercostal nerves	Simple, effective immediate relief	- Short duration - LA toxicity (multiple injections) - Pneumothorax
Erector Spinae Plane Block (ESPB)	20–30 ml 0.25% Bupivacaine	Fascial plane spread → dorsal & ventral rami	Easy, safe, USG-guided	- Variable efficacy - Incomplete visceral pain relief
Serratus Anterior Plane Block	20–30 ml LA	Blocks lateral thoracic wall nerves	Good for lateral thoracotomy	- Limited visceral analgesia
IV Opioids (PCA)	- Morphine PCA: 1 mg bolus, lockout 5–10 min - Fentanyl: 20–25 mcg bolus	μ-receptor agonist	Strong analgesia, easy titration	- Respiratory depression (very important post lung surgery) - Sedation - Nausea/vomiting - Pruritus
Paracetamol	Paracetamol 1 g IV/PO every 6–8 hr (max 4 g/day)	Central COX inhibition	Safe, opioid-sparing	- Hepatotoxicity (overdose)
NSAIDs	Diclofenac 75 mg IM/IV BD Ibuprofen 400–600 mg PO	COX inhibition	Good adjunct, ↓ opioid use	- Renal impairment (↓ renal perfusion) - Gastric ulcer/bleeding - Platelet dysfunction → bleeding risk
Local Wound Infiltration	0.25% Bupivacaine	Peripheral nerve block	Simple, safe	- Short duration



29. Note on newer advances.

A. Enhanced Recovery After Surgery (ERAS) protocols

Integration of prehabilitation, smoking cessation, nutritional optimization, opioid-sparing anesthesia, early extubation, chest physiotherapy, digital chest drainage, and early ambulation has significantly improved outcomes after thoracotomy.

B. Advanced lung isolation devices

Contemporary double-lumen tubes, video double-lumen tubes, bronchial blockers, VivaSight systems, and routine fiberoptic bronchoscopy guidance have improved accuracy of lung separation, reduced malposition, and enhanced safety in complex thoracotomy cases



Vivasight system

A. Non-intubated thoracic surgery (NIVATS)

Selected thoracic procedures can now be performed under regional anesthesia with spontaneous ventilation, reducing airway trauma and ventilator-associated complication in carefully chosen patients. Components of NIVATS:

1. Regional Anesthesia:

Thoracic epidural anesthesia
Thoracic paravertebral block
Intercostal nerve block
Erector spinae plane block

2. Cough Suppression:

Thoracoscopic vagal nerve block:

Right side: near lower trachea/azygos arch
Left side: aortopulmonary window

3. Sedation:

Dexmedetomidine

Propofol

Remifentanyl

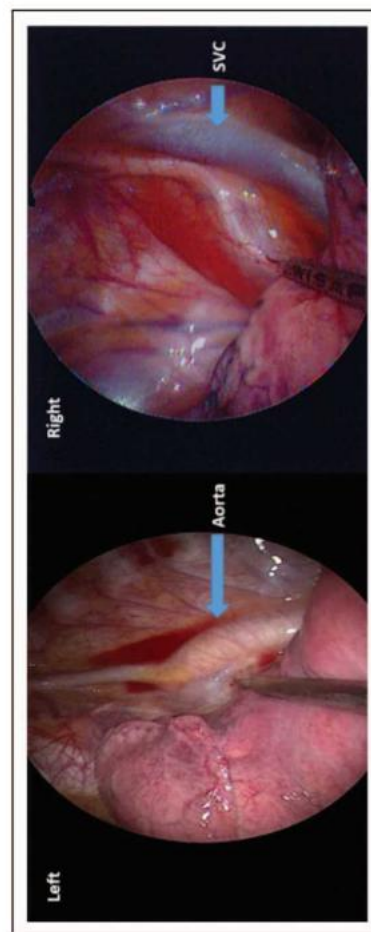


FIGURE 1. The injection site of left and right thoracoscopic vagal nerve blocks. SVC, superior vena cava.

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13th JUNE 2026

09:40AM - 10:20AM

PRIMIGRAVIDA WITH HYPERTENSIVE DISORDER FOR SAFE CONFINEMENT

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DEMOGRAPHIC DETAILS

Name: Mrs. A

Age: 24 years / Female

Primigravida, at 36+4 weeks gestation, booked case, presented for safe confinement.

CHIEF COMPLAINTS

- Headache since 5 days
- Swelling of both feet since 10 days
- Blurring of vision since 1 day

HISTORY OF PRESENTING ILLNESS

Patient was apparently doing well till ➤ **At 32 weeks**

28 weeks of gestation:

- BP: 150/100 mmHg

• **Developed:**

Pedal edema

Mild headache

- Urine albumin: 1+

➤ **At 28 weeks**

- BP noted: 140/90 mmHg.
- No other symptoms noted.
- No proteinuria

Progression → Pre-eclampsia

Diagnosed as **gestational hypertension**

➤ **At 35–36 weeks (CURRENT PHASE):**

- BP: 168/112 mmHg
- Complaints of Persistent headache Visual disturbances Epigastric discomfort
- Urine albumin: **3+**

This clearly indicates: **Severe pre-eclampsia**

PAST HISTORY

No history of:

- Chronic hypertension
- Type 2 Diabetes Mellitus
- Renal disease

MENSTRUAL & OBSTETRIC HISTORY

- LMP consistent with gestational age
- Regular antenatal visits done
- Immunized with TT
- Iron + calcium supplements taken

ANTENATAL HISTORY

FIRST TRIMESTER (0–12 weeks)

- Booked case at 8 weeks of gestation.
- BP: 110/70 mmHg
- Baseline labs normal: Hb: 11.5 g/dL Platelets: 2.5 lakh/dl RFT/LFT normal Urine routine: no protein

SECOND TRIMESTER (13–28 weeks)

- Up to 28 weeks:
- BP gradually increased to **140/90mmHg**
- No proteinuria initially
- Transition phase → **gestational hypertension**

THIRD TRIMESTER (29–36 weeks)

- BP progressively rising:
- 32 weeks → 150/100 mmHg
- 36 weeks → 168/112 mmHg
- **Proteinuria appeared and increased**
- **CNS & hepatic symptoms appeared**

FINAL DIAGNOSIS TREND:

Gestational HTN → Pre-eclampsia → Severe Pre-eclampsia

RISK FACTORS

Primigravida

- Age < 25 years (relative risk)
- Possibly endothelial maladaptation

RED FLAG SIGNS

- Headache
- Visual disturbances
- Epigastric pain



- Oliguria
- Falling platelets

These indicate **impending eclampsia**

GENERAL PHYSICAL EXAMINATION

1. Conscious, oriented
2. BMI: 28 kg/m²

3. Vitals:

- BP: **170/110 mmHg**
- HR: 92/min
- RR: 18/min
- SpO₂: 98% on room air

4. Findings:

- Pedal edema: +++
- Facial puffiness present
- Reflexes: brisk

SYSTEMIC EXAMINATION

1. CNS

- No focal deficits
- Headache present

2. CVS- S1 S2 heard; no murmurs

3. RS- Bilateral NVBS heard; no added sounds

4. Per Abdomen (OB EXAM)

- Fundal height: corresponds to 34 weeks
Suggests **FGR**
- Fetal heart rate: 140/min
- Cephalic presentation

INVESTIGATIONS

1. Hematology

- Hb: 10.2 g/dL
- Platelets: **90,000/mm³** ⚠️ 3
- 24-hour protein: >2 g
- Suggests: Severe pre-eclampsia & Pos

2. 🧪

- Serum creatinine: **1.4 mg/dL**
- Uric acid: elevated

3. 🩺

involvement

4. Urine Routine

- Protein: 3+



- Fetal growth restriction
- Estimated fetal weight <10th percentile Renal

7. Doppler:

Liver Function

- Increased resistance in uterine artery
- AST/ALT: elevated
- 🩺 hepatic involvement

CLINICAL DIAGNOSIS

Primigravida at 36+4 weeks with severe pre-eclampsia with:

- Severe hypertension (>160/110)
- Proteinuria 3+
- CNS symptoms (headache, vision)
- Renal involvement
- Hepatic involvement
- Thrombocytopenia
- Fetal growth restriction

DIFFERENTIAL DIAGNOSIS

- Chronic hypertension with superimposed pre-eclampsia
- HELLP syndrome
- Acute fatty liver of pregnancy

SUMMARY

24-year-old primigravida at 36+4 weeks with progressively increasing BP from 28 weeks, now presenting with severe hypertension, proteinuria, CNS symptoms, thrombocytopenia, renal and hepatic involvement, along with fetal growth restriction-consistent with **severe pre-eclampsia with maternal and fetal complications**

QUESTIONS

1. How do you define Gestational Hypertension, Pre-eclampsia, Eclampsia?

GESTATIONAL HYPERTENSION: It is new-onset hypertension (BP ≥140/90 mmHg on at least 2 occasions, ≥4 hours apart); After 20 weeks of gestation; Without proteinuria or features of end-organ dysfunction, in a previously normotensive woman.

PRE-ECLAMPSIA: New-onset hypertension (BP≥140/90 mmHg) after 20 weeks of gestation + Proteinuria (≥300 mg in 24-hour urine) OR signs of organ dysfunction (even without proteinuria) such as: thrombocytopenia, Impaired liver function, Renal insufficiency, Pulmonary edema, Cerebral or visual symptoms.



ECLAMPSIA: is a severe complication of pre-eclampsia, defined as occurrence of generalized tonic-clonic seizures in a woman with pre-eclampsia. Seizures cannot be attributed to other neurological causes.

2. Why do you call this severe pre-eclampsia-what exact criteria does she meet?

This patient fulfills multiple criteria for severe pre-eclampsia based on clinical and laboratory findings. She has severe hypertension with BP $\geq 160/110$ mmHg, specifically 168/112 mmHg. She also has significant proteinuria with 3+ urine albumin and >2 g/24 hours. Additionally, she has end-organ involvement including CNS symptoms like headache and visual disturbances, renal impairment with elevated creatinine, hepatic involvement with raised transaminases, and thrombocytopenia with platelet count of $90,000/\text{mm}^3$. The presence of fetal growth restriction further supports severity. Thus, this is not just pre-eclampsia but clearly severe disease with maternal and fetal complications.

3. Explain the progression from gestational hypertension to severe preeclampsia in this case.

Initially at 28 weeks, the patient had elevated BP of 140/90 mmHg without proteinuria, fitting gestational hypertension. By 32 weeks, BP increased to 150/100 mmHg and proteinuria (1+) appeared, indicating progression to pre-eclampsia. She also developed symptoms like pedal edema and headache. By 35–36 weeks, the disease progressed further with BP 168/112 mmHg, heavy proteinuria (3+), and systemic involvement including CNS, renal, and hepatic features. This stepwise worsening reflects the natural progression of hypertensive disorders of pregnancy due to endothelial dysfunction and placental pathology.

4. What RED FLAG SIGNS in this case indicate impending eclampsia?

This patient has multiple warning signs of impending eclampsia. Persistent headache and visual disturbances suggest cerebral involvement and possible vasospasm. Epigastric pain indicates hepatic involvement and possible liver capsule distension. The presence of severe hypertension itself increases the risk of seizures. Laboratory findings such as falling platelet count and rising liver enzymes further indicate worsening disease. These red flags together signify a high risk of progression to eclampsia and require

urgent attention.

5. How do you interpret the platelet count of $90,000/\text{mm}^3$ in this case?

A platelet count of $90,000/\text{mm}^3$ is significant and indicates thrombocytopenia, which is one of the severe features of pre-eclampsia. It suggests increased platelet consumption due to endothelial damage and microangiopathy. In this context, it raises concern for evolving HELLP syndrome, especially when associated with elevated liver enzymes. The trend of platelet count is important, as a falling count indicates worsening disease. It also has implications for bleeding risk and overall disease severity.

6. What is the significance of elevated liver enzymes in this patient?

Elevated AST and ALT indicate hepatic involvement in pre-eclampsia. This occurs due to hepatic endothelial dysfunction leading to ischemia and periportal necrosis. It may also cause stretching of the liver capsule, resulting in epigastric pain. When combined with thrombocytopenia, it raises suspicion for HELLP syndrome. Hepatic involvement is a marker of severe disease and increases the risk of complications like hepatic rupture, although rare. Thus, it signifies systemic disease progression beyond simple hypertension.

7. How do you differentiate HELLP syndrome from severe pre-eclampsia in this case?

Severe pre-eclampsia and HELLP syndrome overlap, but HELLP is characterized by hemolysis, elevated liver enzymes, and low platelets. In this patient, there is thrombocytopenia and elevated liver enzymes, suggesting partial features of HELLP. However, evidence of hemolysis such as elevated LDH, bilirubin, or schistocytes is not mentioned. Therefore, this case may represent severe preeclampsia with evolving HELLP rather than full-blown HELLP syndrome.

Differentiation is important as HELLP carries higher maternal risk.

8. What findings suggest fetal growth restriction in this case?

Fetal growth restriction is suggested by discrepancy between fundal height and gestational age, with fundal height corresponding to 34 weeks instead of 36+4 weeks. Ultrasound confirms estimated fetal weight below the 10th percentile. Doppler studies showing



increased resistance in the uterine artery further support placental insufficiency. These findings indicate compromised uteroplacental blood flow, which is common in severe pre-eclampsia due to abnormal placentation.

9. What risk factors for pre-eclampsia are present in this patient?

The main risk factor present in this case is primigravida status, which is a wellknown risk factor due to immunological maladaptation between maternal and fetal tissues. The patient's age of 24 years may also contribute, as younger maternal age has been associated with increased risk in some populations. Additionally, underlying endothelial dysfunction and abnormal placentation are implied in the disease progression. No history of chronic hypertension or diabetes is present, making this a case of de novo pre-eclampsia.

10. What are the goals in medical management of this patient?

- Aims to reduce maternal and fetal complications.
- **Treat hypertension:** The aim is to reduce the MAP gradually by about 15-25% initially
- Avoid reduction of MAP by > 25% in the first hour
- **Target BP:** Systolic 140-150 mmHg, Diastolic: 90-100 mmHg (MAP: 100-120)
- Bed rest with **avoidance of aorto-caval compression**
- **Prevention & Control of convulsions**
- **Fluid therapy** and treatment of oliguria
- Decision when to deliver Management of coagulation abnormalities

11. Elaborate the medical management of this patient?

- **Labetalol:** Beta blocker; used in the first line management of PIH, IV can be administered every 20 Mins. Starting with 20mg -> 40mg -> 80mg (x3)
- Max dose can be 300mg in 24 hours.
- Or oral labetalol 200mg-1600mg can be administered.
- **Hydralazine:** A Direct-acting vasodilator, 5-10mg IV increments followed by intravenous infusion at 5 - 20mg/h titrated against the blood pressure. (max 30mg in 24hrs)
- **Nifedipine:** A calcium channel blocker, start with

10mg->20mg -> 20mg, every 20 min

11. Elaborate the medical management of this patient?

• **Methyldopa:** 1-3g daily in divided doses.

• **Nitroprusside / Nitroglycerine(by continuous infusion) :** IV inf- typical range 5-100mcg/min

• **Sodium Nitroprusside:** A last resort for severe refractory hypertension. IV inf- 0.25mcg 0.5mcg/kg/min an titrate carefully, with Max dose 8-10mcg/kg/min

12. What is the role of Regional Anaesthesia in this patient?

- Spinal Anaesthesia is preferred in the absence of thrombocytopenia in Pre-eclampsia.
 - Hyperbaric Bupivacaine 0.5% of 2 ml +/- Fentanyl 15-20 mcg.
 - Advantages:
 - Avoidance of GA and airway difficulties.
 - Avoidance of intubation response with that may result in intracranial haemorrhage.
- Smaller needle and quicker procedural time compared to epidural anaesthesia.

13. What is the seizure prophylaxis in Pre-eclampsia?

- Magnesium Sulphate is the initial therapy for seizure prophylaxis.
 - Magnesium Sulphate: A potent cerebral vasodilator, as well as a powerful catecholamine antagonist. The therapeutic blood level lies between 4-6 mEq/L.
 - Monitor urine output, respiratory rate, patellar reflexes and serum magnesium levels.
- There are two commonly accepted dosage regimens: Pritchard and Zuspan.

Regimen	Loading Dose	Maintenance Dose	Monitoring / Notes
Pritchard Regimen	4 g MgSO ₄ IV slowly over 5-10 min PLUS 10 g IM (5 g in each buttock with lignocaine)	5 g IM every 4 hours in alternate buttocks	• RR >12/min • Urine output >25 - 30 ml/hr • Patellar reflex present
Zuspan Regimen	4 - 6 g MgSO ₄ IV over 15-20 min	1 - 2 g/hr IV infusion	Requires infusion pump and close monitoring of: • Reflexes • Respiratory rate • Urine output



14. Elaborate on fluid management in PIH?

- **Restrictive fluid therapy:** 60–80 mL/h of Crystalloids.
- Avoid preloading with IV Fluids preferably or preload with 100-200 ml bolus. Even in the presence of oliguria, fluid loading is avoided.
- **Target Urine output:** more than 0.5ml/kg/hour or 25-30 ml/hour.
- Presence of Renal impairment, restrict the fluid input to 50-60 ml/hour.
- The use of colloids remains controversial and is generally not routine.
- A minimal preload or co-loading approach with crystalloids is preferred, combined with readiness to use vasopressors.

15. Discuss the conduct of General Anaesthesia in PIH?

- **Aspiration prophylaxis:** Acceptable methods include 30ml of 0.3 M Sodium Citrate per oral and/or H2 blocker I.V. and Metoclopramide 10 mg I.V.
- Prepare for difficult airway.
- Pre-oxygenation & De-nitrogenating with 100% oxygen via face mask for 3 minutes with VT breaths.
- Induction via RSI with Sellick's maneuver.
- **Choice of induction agent:** Thiopentone 4 - 5 mg/kg or Etomidate 0.2mg/kg or Propofol 2mg/kg and Succinylcholine 1-1.5mg/kg.
- Attenuate intubation response with Lignocaine 1.5mg/kg or preferably B-Blocker Labetalol 5-10 mg incremental doses or Esmolol 0.5 mg/kg.
- Use a smaller endotracheal tube.
- Continuation of MgSO4 intra-operatively.
- Administer of Volatile agents up till delivery of the foetus.
- MAC of 0.5 - 1 with Isoflurane to minimize uterine atony and a suitable opiate after delivery.

16. Discuss the management of Seizures intra operatively in PIH?

- Call for help immediately.

- **Left lateral tilt 15–30°**

- Secure **Airway & Maintain Breathing** with 100% oxygen at 10–15 L/min.

- Continuous foetal assessment if time allows, but **maternal resuscitation takes priority.**

- **First-line anticonvulsant:** Magnesium sulphate
If seizure recurs/persists: Additional 2 g IV magnesium over 5–10 min.

- **Second-line** if seizures continue; **Benzodiazepine:** Lorazepam 4 mg IV slow or diazepam 5–10 mg IV slow.

- **Third-line for refractory seizure/status:** Fosphenytoin 20 mg/kg IV or Levetiracetam up to 60 mg/kg IV (max 4.5 g).

- **Last resort:** Rapid sequence intubation and general anaesthesia with seizure-suppressing agents such as propofol, followed by controlled ventilation.

Definitive treatment: Delivery of the foetus and placenta after maternal stabilization.

17. Discuss the management of cerebral haemorrhage peri-operatively in PIH?

- **Position:** Left uterine displacement 15–30° and head-up 20–30° if feasible.
- **IV access:** 2 large-bore IV lines (16–18G), arterial line & standard monitoring
- **Pre-oxygenation:** 100% O₂ before RSI; maintain SpO₂ >94–95%.
- **Choice of induction drugs:** Propofol 1.5–2 mg/kg or thiopentone 3–5 mg/kg
- **Paralysis:** Succinylcholine 1–1.5 mg/kg.
- **Ventilation goal:** Normo-ventilation, target PaCO₂ about 35–40 mmHg.
Restrictive fluid therapy: 60–80 mL/h unless bleeding/hypovolaemia
- To continue intra-op seizure prophylaxis and definitive treatment for onset of seizures intra OP as discussed earlier.



- Labs: CBC, Electrolytes, ABG, Coagulation +/- DIC profile.
- **Correct coagulopathy:** Aim INR ≤ 1.4 with blood and blood products.
- **ICP management:**
 - Avoid hypoxia, hypercarbia, coughing, bucking, neck obstruction; head elevated.
- Mannitol 25 g IV has been used before decompression.
- Emergency caesarean followed by neurosurgical decompression if mother unstable.
- **Post-op:** ICU/NCCU, invasive BP control, magnesium continuation, strict fluid balance & repeat labs.

18. Discuss the management of Pulmonary Edema in PIH?

- Airway protection, left lateral tilt 15–30°, 100% O₂ to maintain SpO₂ >95%.
- **Respiratory support:** CPAP: 8–12 cm H₂O or BiPAP: IPAP 10–14 cm H₂O/EPAP 5–8 cm H₂O if distress persists
- **Intubation if:** SpO₂ <95% despite CPAP, respiratory fatigue, GCS <8.
- Lung protective ventilation with 6–8 ml/kg of VT with PEEP 5–8 cmH₂O and maintain Plateau pressures < 30 cmH₂O
- Diagnosis: Chest X-ray (bilateral infiltrates), Echo (LV function), ABG (hypoxemia)
Fluid restriction
- First-line diuretic: Furosemide 20–40 mg IV over 1–2 minutes.
- Repeat furosemide: 40–80 mg IV if no response in 15–20 minutes.
Target urine output: 0.5–1 mL/kg/hour
- Venodilator: IV Nitroglycerin infusion 5 mcg/min, increase by 5 mcg/min every 5 min to treat pulmonary hypertension.

Avoid drug combination: No IV magnesium + calcium channel blockers due to the risk of myocardial depression.

19. Discuss the Post-operative care in PIH?

- 70% of convulsions and pulmonary complications occur in the postoperative period in preeclampsia.
- Laryngeal oedema may worsen during the operative procedure and airway embarrassment, sufficiently severe to require reintubation, may follow extubation.
- Antihypertensive therapy should be continued for as long as clinically indicated and anticonvulsant medication maintained for as long as the patient remains symptomatic.
- Invasive monitoring, if used intra-operatively, should be continued in the intensive care environment post-operatively.
- Post-operative analgesia can contribute to the ease of management of these cases.
- Fluid balance and the correction in the presence of oliguria.

20. Discuss the Post-operative analgesia in PIH?

- Adequate analgesia essential to prevent pain-induced sympathetic stimulation, hypertension, and cerebral complications.
- Epidural analgesia provides excellent pain relief and better haemodynamic stability.
- Neuraxial opioids (intrathecal/epidural morphine). Paracetamol useful as opioid-sparing analgesic (dose adjustment if hepatic dysfunction severe).
- NSAIDs used cautiously/avoided due to:
 - Renal impairment
 - Platelet dysfunction
 - Fluid retention risk
 - Systemic opioids in small titrated doses to avoid over-sedation and hypercarbia
 - Continuous monitoring for:
 - BP control
 - Respiratory depression
 - Neurological deterioration



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13th JUNE 2026

10:20AM - 10:50AM

DIABETES MELLITUS**DKA · HHS · Sepsis & Anesthetic Management****CASE SUMMARY**

A 45-year-old male patient with a history of scrotal swelling, with known case of type 2 diabetes mellitus on insulin therapy since 10 years, now presenting with systemic sepsis in septic shock, most probably due to a pyocele. He is coming in for an incision and drainage surgery.

1: DIABETES MELLITUS?**Classification · Physiology · Diagnosis · Treatment ?****1. Definition and Epidemiology**

Diabetes mellitus (DM) is primarily a disease of glucose metabolism that also significantly affects lipid and protein metabolism, impacting a wide range of endocrine functions. Its hallmark is an absolute or relative insulin deficiency. An estimated 9.4% of the adult U.S. population have DM, and approximately 40 million Americans have prediabetes. DM is the most commonly occurring endocrine disease found in surgical patients, with 25–50% of diabetics requiring surgery at some point in their lives.

DM can remain clinically unapparent until exacerbated by the stress of trauma or surgery. Observational studies report hyperglycemia present in 32–38% of patients in community hospitals, 41% of critically ill patients with acute coronary syndromes, and 80% of patients after cardiac surgery — with approximately one-third of non-ICU patients and 80% of ICU patients having had no history of diabetes before admission.

2. What is the Diagnostic Criteria?

Diagnostic Criterion	Notes
A1c $\geq 6.5\%$	Certified laboratory method required
Fasting plasma glucose ≥ 126 mg/dL (7 mmol/L)	Fasting = no caloric intake ≥ 8 hours
2-h plasma glucose ≥ 200 mg/dL during OGTT	75 g anhydrous glucose load (WHO protocol)
Random glucose ≥ 200 mg/dL + hyperglycemia symptoms	Classic symptoms must be present
HbA1c 5.7–6.4% / FPG 100–125 mg/dL	Prediabetes — requires monitoring

Table 1 — Criteria for the Diagnosis of Diabetes Mellitus (ADA)

1. Classification of DM?**Type 1 Diabetes Mellitus**

Type 1 DM is due to pancreatic β -cell destruction, usually leading to absolute insulin deficiency. It accounts for 5–10% of DM cases. Most patients experience disease onset early in life. Hyperglycemia in

Type 1 DM cannot be controlled with diet or oral hypoglycemic agents — it mandates insulin. These patients are more likely to become ketotic and sustain progressive end-organ complications. Patients with Type 1 DM are also more vulnerable to diabetic stiff joint syndrome, leading to decreased atlanto-occipital joint mobility and potentially difficult laryngoscopy (see 'Prayer Sign' below).

Type 2 Diabetes Mellitus

Type 2 DM accounts for 90–95% of DM cases and is due to a progressive loss of insulin secretion against a background of insulin resistance. Patients are often overweight and may have normal or even elevated insulin levels initially. In milder forms it can be managed with diet, lifestyle modifications, and oral agents. Because of the obesity epidemic, adolescents and teenagers are presenting more frequently with Type 2 DM. These patients are relatively resistant to ketosis; their disease may not be clinically apparent until exacerbated by surgical stress.

Gestational Diabetes

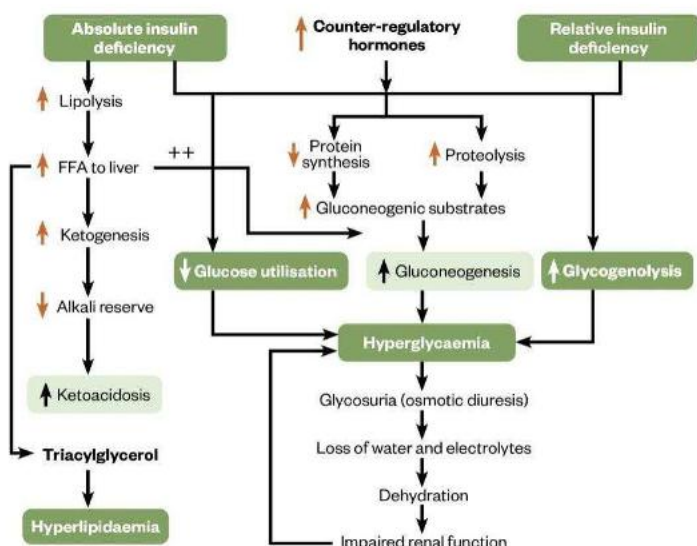
Typically diagnosed in the second or third trimester of pregnancy using the glucose challenge test or oral glucose tolerance test, and may presage future Type 2 DM. **Diabetes Due to Other Causes**

Secondary DM may result from diseases that damage the pancreas or produce counter-regulatory hormones. Causes include:

- Pancreatic surgery, chronic pancreatitis, cystic fibrosis, or hemochromatosis
- Glucagonoma, pheochromocytoma, or acromegaly (counter-regulatory hormone excess)
- Cushing disease or steroid/tacrolimus therapy (glucocorticoid excess)
- Thiazide diuretics and atypical antipsychotics (clozapine, olanzapine, risperidone, ziprasidone, quetiapine)
- HIV/AIDS treatment, genetic defects in β -cell function or insulin action (monogenic diabetes)



Pathophysiology of DM :



4. Enumerate the Physiology of Insulin and Glucose Metabolism?

Insulin is a protein produced by the β cells of the islets of Langerhans. Basal secretion is approximately 1 unit/hour, rising 5–10 fold after food ingestion. Normal daily production is 40–50 units. The half-life in circulation is only 5 minutes, though clinically it appears to have a longer duration due to delays in receptor binding and release. Once binding sites are saturated, additional insulin produces a more prolonged rather than more potent effect. Insulin is metabolized in the liver and kidneys - hepatic or renal dysfunction prolongs insulin action and increases hypoglycemia risk.

Key Metabolic Actions of Insulin

The most fundamental action of insulin is to stimulate cellular uptake of glucose in skeletal muscle, adipose tissue, and cardiac cells. The brain, liver, and immune cells are exceptions - insulin does not affect glucose transport in these tissues. Along with glucose, potassium enters cells under insulin's influence, so diabetic patients are likely to have potassium imbalance across cell membranes.

- Stimulates glycogen formation (suppresses gluconeogenesis and lipolysis)
- Increases amino acid uptake into muscle cells (insulin deficiency leads to catabolism and negative nitrogen balance)
- Blocks fatty acid liberation from adipose tissue at even low concentrations
 - Insulin deficiency: accelerated lipid catabolism $\rightarrow$ increased ketone formation (acetoacetate, β -hydroxybutyrate)

- Low insulin levels - insufficient to prevent hyperglycemia - are often sufficient to block lipolysis, explaining hyperglycemia without ketosis

Glucagon

Glucagon is a polypeptide from α -cells of the pancreas. It stimulates insulin release but also opposes some of its effects. Glucagon both directly and indirectly increases circulating glucose via stimulation of gluconeogenesis and glycogenolysis. It is stimulated by hypoglycemia, epinephrine, and cortisol, and suppressed by glucose ingestion.

After total pancreatectomy, glucose balance may be better than expected due to concomitant absence of glucagon.

Correlation of Stress Response and Diabetes?

During stress, elevated cortisol, glucagon, catecholamines, and growth hormone stimulate gluconeogenesis and glycogenolysis causing hyperglycemia. Glucagon and adrenergic stimulation also suppress insulin release. Inflammatory mediators released during stress enhance counter-regulatory hormones and directly affect intracellular insulin signaling - culminating in significant insulin resistance. Even a patient without DM may develop mild stress induced hyperglycemia; a patient with subclinical DM may become very difficult to manage.

2: PHARMACOLOGICAL TREATMENT of DM?

Non-Insulin Agents · Insulin Preparations

1. Treatment of Type 1 DM

Patients with Type 1 DM require exogenous insulin to survive. The risk of microvascular complications decreases with near-normal glycemic control (HbA1c < 7%). Patients may be on multiple-component insulin regimens, multiple daily injections (MDI), or continuous subcutaneous insulin infusion (CSII -insulin pump). Intensive insulin therapy (IIT) prior to and during pregnancy reduces the risk of fetal malformations and morbidity.

2. Treatment of Type 2 DM

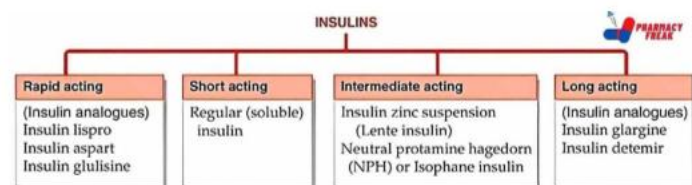
Initial management includes diet/lifestyle modification, exercise, and metformin. If glucose control is inadequate or diabetes worsens, additional oral agents and/or insulin are added. The goal is HbA1c < 7% without causing hypoglycemia. Each new class of non-insulin agents added reduces HbA1c by approximately 0.9–1.1%.



Bariatric surgery is considered for BMI > 35 kg/m² with Type 2 DM, especially with comorbidities difficult to control. The ADA recommends metabolic surgery for BMI > 30 kg/m² if hyperglycemia is inadequately controlled.

3. Non-Insulin Glucose-Lowering Drugs(OHA's):

Class	Compounds	Mechanism	Actions
Biguanides	Metformin	Activate AMP-kinase	↓ hepatic glucose production
Sulfonylureas (2nd gen) [IDE]	Glyburide, Glipizide, Glipizide, Glipizide	Close KATP channels on β cell	↑ Insulin secretion
Meglitinides[GLINIDE]	Repaglinide, Nateglinide	Close KATP channels on β cell	↑ Insulin secretion
Thiazolidinediones [GLITAZONE]	Pioglitazone, Rosiglitazone	Activate PPAR- γ	↑ Peripheral insulin sensitivity
α -Glucosidase inhibitors [BOSE]	Acarbose, Miglitol, Voglibose	Inhibit intestinal α -glucosidase	↓ Intestinal carbohydrate digestion/absorption
GLP-1 Receptor Agonists[GLUTIDE]	Exenatide, Semaglutide, Liraglutide, Dulaglutide	Activate GLP-1 receptors	↑ Insulin secretion (glucose dependent), ↓
DPP-4 Inhibitors[GLIPTIN]	Sitagliptin, Vildagliptin, Saxagliptin, Linagliptin, Alogliptin	DPP-4 → ↑ increase incretin level	Is ↑ Active GLP-1 & GIP, ↑ insulin secretion,
SGLT2 Inhibitors [GLIFLOZIN]	Canagliflozin, Dapagliflozin, Empagliflozin, Ertugliflozin	Inhibit SGLT2 in proximal nep	chronBlock renal glucose reabsorption →
Amylin Mimetics	Pramlintide	Activate amylin receptors	↓ Glucagon secretion, slow gastric emptying,
Bile Acid Sequestrants	Colesevelam	Bind bile acids in intestine	↓ Hepatic glucose production, ↑
Dopamine-2 Agonists	Bromocriptine	Activate dopaminergic receptors	rsAlter hypothalamic metabolism; ↑



Insulin Preparation	Onset	Peak	Duration
Lispro, Aspart, Glulisine (SC)	10–15 min	0.5–1.5 h	3–5 h
Regular (SC)	0.5–1 h	2–3 h	4–8 h
Regular (IV)	15 min	15–30 min	0.5–1 h
NPH (SC)	2–4 h	4–10 h	10–16 h
Glargine (SC)	2–4 h	None	20–24 h
Detemir (SC)	1–4 h	None	12–24 h
Degludec (SC)	1–9 h	None	~42 h

Table 3 -
Properties of Common Insulin Preparations

3: What are the END-ORGAN COMPLICATIONS OF DIABETES on various systems?

Cardiovascular · Renal · Neuropathy ·

Chronic complica-ons : Macrovascular

1. Cardiovascular Disease

Atherosclerosis develops earlier and more extensively in diabetic patients compared with non-diabetics. Manifestations include coronary artery disease (CAD), peripheral vascular disease, cerebrovascular disease, and renovascular disease. The incidence of postoperative myocardial infarction is increased in diabetic patients, and the perioperative complication rate is higher. The ACC/AHA guidelines recognise DM as a cardiac risk factor for noncardiac surgery evaluation. Preoperative hyperglycemia (elevated HbA1c) has consistently been associated with poor perioperative outcomes. Preoperative HbA1c < 8% is recommended for elective surgery; the ESC recommends postponing elective noncardiac surgery if HbA1c ≥ 8.5%.

- CAD can manifest at a young age or atypically in Type 1 diabetics
- Silent myocardial ischemia and infarction more common — possibly due to sensory neuropathy of visceral cardiacaferents
- DM-associated cardiomyopathy may exist despite angiographically normal coronary arteries

Central nervous system : risk of cerebrovascular accidents and strokes secondary to chronic hypertension, accelerated atherosclerosis, dyslipidemia

Peripheral vascular disease : due to accelerated atherosclerosis secondary to endothelial dysfunction due to oxidative stress ,dyslipemia and abnormal endothelial proliferation (advanced glycated end products)

2. Diabetic Nephropathy

Diabetic nephropathy occurs in 20–40% of patients with diabetes and is the leading cause of end-stage renal disease (ESRD). Albuminuria usually precedes steady decline in renal function. Microalbuminuria (30–299 mg/24h) is the earliest stage of nephropathy in Type 1 DM and a marker for nephropathy development in Type 2 DM. Renal disease prolongs insulin action - patients are more prone to hypoglycemia and exogenous insulin should be used judiciously



3. Neuropathy

Diabetic patients develop heterogeneous neuropathies with diverse clinical manifestations. The most common are, -chronic sensorimotor

- Distal symmetric polyneuropathy
- Distal sensory polyneuropathy
- Autonomic neuropathy
- Cranial neuropathies
- Limb mononeuropathy
- Compression and entrapment neuropathy - femoral, sciatic, ulnar, peroneal neuropathy -Truncal mononeuropathies

Mononeuropathy multiplex

- Asymmetric lower-limb motor neuropathy (amyotrophy)

Cardiovascular Autonomic Neuropathy

Clinically the most important neuropathy. Presents as:

- Resting tachycardia
- Exercise intolerance
- Orthostatic hypotension
- Exaggerated pressor response to tracheal intubation
- Increased risk of intraoperative hypotension requiring vasopressor support
- Increased risk of perioperative cardiorespiratory arrest
- Predisposition to intraoperative hypothermia

Testing: beat-to-beat heart rate variation during breathing, heart rate response to Valsalva maneuver, and orthostatic changes in diastolic BP and heart rate.

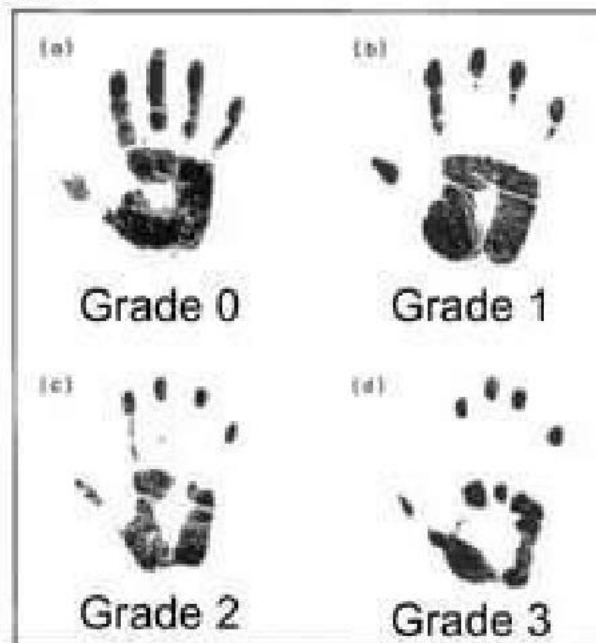
Gastrointestinal Neuropathy

GI neuropathies include esophageal enteropathy, gastroparesis, constipation, diarrhea, and fecal incontinence. Delayed gastric emptying increases the risk of pulmonary aspiration of gastric contents. Autonomic function tests can predict solid food in gastric contents but not increased gastric volume or acidity. Metoclopramide or ranitidine may be useful in emptying the stomach of solid food.

Diabetic Stiff Joint Syndrome(LJM) - Airway Implications

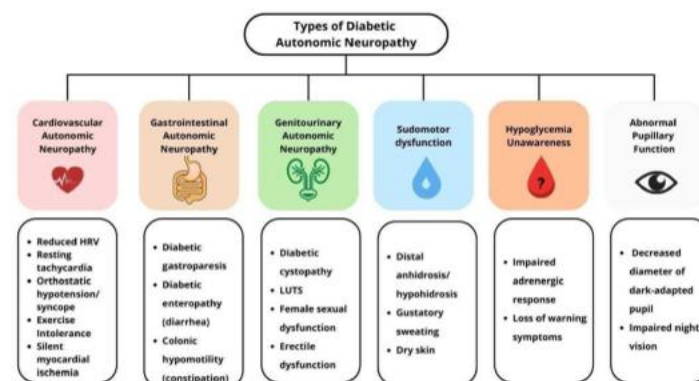
Direct laryngoscopy can be difficult due to stiff joint syndrome leading to decreased atlanto-occipital joint mobility. This is due to the **abnormal non-enzymatic**

glycosylation of proteins and abnormal cross linking of collagen leads to stiff joints. The 'Prayer Sign' - inability to approximate the palmar surfaces of the interphalangeal joints - is associated with stiff joint syndrome and may predict difficult laryngoscopy. Plan accordingly with awake intubation techniques if predicted.



Palm print sign

- Assess autonomic neuropathy :



What are the Tests for autonomic neuropathy?

Parasympathe-c tests :

HRV in response to - **deep breathing, standing , valsalva manoeuvre**

Response to deep breathing: patient is asked to take 6 deep breaths (max heart rate variability occurs at this heart rate)

Respiratory sinus arrhythmia (RSA) is a normal phenomenon due to vagal input to sinus node during expiration causing cardiodeceleration. Autonomic neuropathy decreases the HRV,



I normal variability >15 beats/min

II Abnormal variability <10 beats/min

Response to standing : (30:15) there is rapid increase in HR in response to standing that is maximal at 15th beat and decrease in

HR at 30th beat, it is measured as the longest RR interval(20-40 beats) divided by the shortest RR interval (5-25 beats)

In DAN, 30:15 >1.04 is normal

1.01-1.03 is borderline

<1.01 is abnormal

Response to valsalva : hemodynamic responses in BP:(sympathe-c tests)

Phase 1: transient rise in BP and fall in heart rate due to compression of aorta

Phase 2 : Initial fall in bp followed by recovery of blood pressure later in the phase and accompanied by increase in heart rate.

There is fall in CO due to decreased venous return

Phase 3 : drop in BP and rise in HR, expiration stops

Phase 4 : overshoot of BP and reflex bradycardia due to increase in venous return.

Valsalva ratio longest RR interval in phase 4 to the shortest RR interval, ratio of <1.2 is abnormal.

How does autonomic neuropathy affect the preoperative course ?

It affects the compensatory mechanisms (baroreceptor reflexes), increasing the risk of hemodynamic liability, with PPV, Change in poisons, sympathetic blockade.

Diabetic neuropathy: progressive endothelial damage and dysfunction leads to renal damage, renal vessels ischemia and micro aneurysms leading to staged diabetic neuropathy. Hence, fundoscopy preoperatively to be advised to grade the severity of end organ damage and neuropathy.

What are the **ANESTHETIC MANAGEMENT?**

Preoperative · Intraoperative · Glycemic Goals · Perioperative Control

1. Overview

Successful management of diabetic patients is as dependent on- or more dependent on-chronic complication management than on acute hyperglycemia. Three preoperative goals exist: (1) determine end-organ complications, (2) determine the

glucose-lowering regimen, and (3) assess current glycemic control and need for preoperative intervention.

2. Preoperative Evaluation

A thorough preoperative assessment should include:

- Detailed history and physical examination
- Recent ECG
- BUN, potassium, creatinine, glucose, and urinalysis
- HbA1c level — target < 8% for elective surgery (ESC: postpone if HbA1c ≥ 8.5%)
- Assessment for DKA if glucose > 300 mg/dL in preoperative area (some institutions use 300 mg/dL trigger; postpone elective surgery if glucose > 400 mg/dL)
- Evaluate for gastroparesis, autonomic neuropathy, difficult airway (stiff joint syndrome)

3. Preoperative Medication Management

Oral Antihyperglycemic Medications (ADA Protocol)

- Discontinue OHA's medications the night before surgery
- No oral hypoglycemic agents on the morning of surgery
- Sulfonylureas: particular risk of hypoglycemia, impairs the ischemic preconditioning of heart — withhold
- Metformin: withhold day of surgery (associated with lactic acidosis during hypotension/hypoxia) especially in renal disorders pts.
- SGLT2 inhibitors: must be discontinued 3–4 days before surgery (risk of euglycemic DKA)
- Reinstitute medications after patient resumes normal diet.

Talk about Preoperative Insulin strategies in type 1 and 2 pts?

1) Type 1 DM pts for major and minor surgeries :

Insulin Adjustments - Night Before Surgery

- Usual dose of evening short-acting insulin
- Reduce intermediate and long-acting insulin by 20%-25%
- Premixed insulin: reduce evening dose by 20%

Insulin Adjustments - Morning of Surgery

- Omit morning short-acting insulin
- Reduce intermediate-acting insulin by 50%
- Reduce long-acting insulin by 20% (only if fasting glucose > 120 mg/dL)



- Type 1 DM: always requires some basal insulin- never completely omit

Insulin Pump (CSII) Therapy

- Insulin pumps can be safely utilised during surgery
- Reduce pump basal insulin dose by 20–25%
- Check blood glucose every hour if pump is continued during surgery
- Check every 2–4 hours while fasting

2) Type 2 DM pts for minor surgeries & major surgeries:

Pts on OHA's continue on night before surgery and skip all OHA's on morning of sx, Pts on insulin - follow same as above as in for Type 1 DM pts

4. What are our Intraoperative concerns?

The anesthetic plan depends intimately on end-organ complications present: invasive monitoring for cardiac disease; awake intubation if difficult airway predicted; fluid/drug choices based on renal function; aspiration precautions if gastroparesis present.

Blood Glucose Monitoring

- Measure blood glucose before, during, and after surgery
- Monitor every 4–6 hours while nil per oral
- Hourly measurements in high-risk patients (continuous insulin pump/infusion)
- Aberrant values should be verified by central laboratory - point-of-care devices can vary by 20%
- Continuous glucose monitoring (CGM) devices now approved for inpatient use (post-COVID-19)

Glucose Administration

- Standard adult dose: 5g/hour (100–125 mL of 5% dextrose hourly)
- Intraoperative glucose guided by patient's glucose level - routine glucose-containing IV fluids not recommended
- Record dextrose administration and IV fluids separately

5. What are the Insulin Administration Strategies intraoperatively for type 1 & type 2 pts for minor and major surgeries?

In the perioperative setting, subcutaneous absorption is unreliable due to vasoconstriction from hypovolemia or hypothermia:

- Sliding scale insulin (RISS) alone — strongly discouraged in hospitalized patients; predisposes to

glucose fluctuations

- Continuous IV insulin infusion + IV boluses-most effective for acute glycaemic control; achieves targets promptly

Insulin strategies for Type 1 DM pts coming for minor surgeries: (<2hrs duration)

Check for basal glucose levels and give correctional insulin S/C and recheck BG levels after 30mins. (to avoid risk of ketosis), for every 50mg/dl rise in BG above 180mg/dl administer 2U of rapid acting insulin S/C route to maintain intraop 140-180mg/dl

For Type 1 DM coming for major surgeries :

intraoperative BG targets 140-180mg/dl, if BG >180mg/dl

Follow the VRILL (variable rate intravenous insulin infusion) regimen : insulin infusion= BG/100 U/hr

Recheck BG every hourly and for every 30mg/dl rise in BG levels ,add 1U to the infusion to maintain BG 140-180mg/dl

If BG levels <110mg/dl, stop the infusion and administer 5%Dextrose

For Type 2 DM pts coming for minor surgeries:

No insulin and no glucose, restart preoperative regimen as and when pt resumes oral feeds

For Type 2 DM pts coming for major surgeries:

Pts on insulin therapy preoperatively, follow preop insulin regimen as mentioned earlier, and intraoperatively give correctional insulin via S/C route, for every 50mg/dl rise in BG above 180mg/dl administer 2U of rapid acting insulin S/C route to maintain intraop 140-180mg/dl and don't repeat insulin S/C <4hrs of previous dose

Choice of anaesthesia :

Regional over general anaesthesia : Advantages : choosing regional over general as it has minimal effect on glucose metabolism and circulating levels of cortisol, catecholamines and glucagon and growth hormone

➤ pts are awake , hence symptoms of hypoglycaemia /hyperglycemia can be identified early which is masked under GA

➤ Regional avoids risk of difficult airway associated with GA and risk of aspiration



- Excellent analgesia post operatively, spares usage of NSAID's due to risk of nephropathy, reduces the risk of insulin resistance
- Allows early return of normal oral intake and improves glucose utilisation and ERAS , improves wound healing
- Disadvantages: severe hypotension refractory to vasopressors is a concern
- Risk of neuronal damage and ischemic nerve injury is high involved in peripheral nerve blockade
- Risk of infections in diabetics resulting in SSI & epidural abscess

5: DIABETIC KETOACIDOSIS (DKA)?

Definition • Pathophysiology • Management

• Complications?

1. Definition and Diagnostic Criteria

DKA is defined by the biochemical triad of ketonemia, hyperglycemia, and acidemia. It is a life-threatening medical emergency, more common than HHS, accounting for approximately 1% of diabetes-related hospitalizations. DKA mortality is approximately 5% (lower series report ~1%). HHS mortality is higher (10–20% or up to 15% in some series). DKA primarily occurs in Type 1 DM, but is increasingly recognised in some Type 2 diabetics. Incidence: 4.6–8 episodes per 100 patient-years of diabetes.

Parameter	Criterion / Notes
Blood glucose	> 250 mg/dL (> 14.0 mmol/L) — usually 250–600 mg/dL; euglycemic DKA possible with SGLT2 inhibitors
Ketonemia / significant ketonuria	Positive urinary or plasma ketones
Metabolic acidosis	Serum bicarbonate < 18 mmol/L and/or arterial pH < 7.3
Anion gap	Elevated: calculated as (Na + K) - (HCO ₃ + Cl); normal 8–12 mmol/L

Table 5 - Diagnostic Criteria for DKA

2. What are the Common Precipitating Events?

Precipitant	Frequency
Infections (commonly urinary tract)	~30%
Non-compliance with treatment	~15%
New diagnosis of Type 1 diabetes	5–15%
Other stresses (MI, alcohol, pancreatitis, drugs)	~5%
No identifiable cause found	~40%

3. Classify DKA

Mild DKA

Plasma glucose: > 250 mg/dL
 Arterial pH: 7.25–7.30
 Serum bicarbonate: 15–18 mEq/L
 Ketones: Positive
 Anion gap: > 10
 Mental status: Alert

Moderate DKA

Plasma glucose: > 250 mg/dL
 Arterial pH: 7.00–7.24
 Serum bicarbonate: 10–<15 mEq/L
 Ketones: Positive
 Anion gap: > 12
 Mental status: Drowsy / mild confusion

Severe DKA

Plasma glucose: > 250 mg/dL
 Arterial pH: < 7.00
 Serum bicarbonate: < 10 mEq/L
 Ketones: Positive Anion gap: > 12
 Mental status: Stupor / coma

4. Describe the Pathophysiology associated with DKA?

DKA results from insulin deficiency combined with excess counter-regulatory hormones (glucagon, growth hormone, catecholamines). Absent insulin → poor peripheral glucose utilisation; glucagon, GH, and catecholamines → triglyceride breakdown into free fatty acids (FFA) and hepatic gluconeogenesis. Beta-oxidation of FFA produces acetoacetate and β-hydroxybutyrate (ketoacids) → elevated anion gap metabolic acidosis.

- Rising blood glucose → osmotic diuresis once renal threshold (~12 mmol/L) is exceeded
- Urinary loss of water, glucose, ketones, sodium, potassium, and phosphate
- Patients present severely dehydrated with electrolyte disturbances
- Initial serum K may be normal or elevated despite total body K deficit (acidosis shifts K out of cells)
- On insulin + fluid treatment, K rapidly shifts intracellularly — risk of hypokalemia
- Leukocytosis (10–15 × 10⁹/L) is characteristic of DKA — WBC > 25 × 10⁹ suggests underlying infection
- Elevated amylase common without true pancreatitis — may mimic surgical abdomen
- Euglycemic DKA associated with SGLT2 inhibitor use — pharmacologic effect persists 10–14 days postdiscontinuation



5. Clinical Features

Symptoms

- Thirst, polyuria, polydipsia
- Weakness and lethargy
- Nausea, vomiting, abdominal pain
- Weight loss

Signs

- Evidence of dehydration
- Tachycardia and hypotension
- Kussmaul respiration (deep, rapid breathing - respiratory compensation for metabolic acidosis) • Ketotic/fruity breath (exhaled acetones)
- Temperature usually normal or low even with underlying infection
- Altered consciousness and confusion

6. What are the Initial Investigations to be evaluated?

- Blood glucose: hourly — point-of-care can be inaccurate at very high levels; send to laboratory initially
- Urine ketones: nitroprusside method does not measure β -hydroxybutyrate — direct β -hydroxybutyrate measurement preferred
- Serum U&E every 2–4 hours: sodium (correct for hyperglycemia: add 1.6 mmol/L per 5.5 mmol/L elevation in glucose above 5.5), potassium, urea, creatinine
- Serum osmolality: calculated as $2(\text{Na}) + \text{glucose} + \text{urea}$; coma with osmolality $< 330 \text{ mOsm/kg}$ → seek other causes
- Venous or arterial blood gas every 2–4 hours (venous pH is 0.02–0.03 lower than arterial)
- Anion gap: $(\text{Na} + \text{K}) - (\text{HCO}_3 + \text{Cl})$; elevated in DKA
- Full blood count, chest X-ray, blood cultures, urine cultures, ECG, cardiac enzymes
- Elevated acetoacetate may cause falsely elevated creatinine (colorimetric method)

7. How do you Manage pt of DKA in Adults?

The basic principles are: (1) rapid restoration of circulation with isotonic IV fluids; (2) gradual rehydration and electrolyte repletion; (3) insulin to reverse ketosis and hyperglycemia; (4) careful regular monitoring to detect and treat complications.

Diabetic Ketoacidosis (DKA) Patient for Anaesthesia – Emergency OT approach

DKA pt in OT = nightmare unless you fix the physiology first. Rule #1: *No elective surgery in DKA. Emergency

surgery only after resuscitation.

Why anaesthesia is risky?

1. Hypovolemia → profound hypotension on induction
2. Acidosis → ↓ myocardial contractility, arrhythmias
3. K^+ derangement → cardiac arrest
4. Cerebral edema risk if glucose corrected too fast

2. Pre-op: Resuscitate first, operate later.

Only exception: Life-threatening surgical emergency like perforation, torsion. Even then, give 1-2 hours for stabilization if BP allows.

Checklist before shifting to OT:

Parameter Target before induction Why pH > 7.2 Severe acidosis → myocardial depression

$\text{HCO}_3^- > 10 \text{ mEq/L}$ If < 10 , very high risk

$\text{K}^+ 3.3-5.5 \text{ mEq/L}$ < 3.3 = arrhythmia risk with insulin. > 5.5 = defer

Volume UO $> 0.5 \text{ mL/kg/h}$, SBP > 90 Hypovolemia = CV collapse on induction

Glucose $< 300 \text{ mg/dL}$, falling But don't normalize acutely → cerebral edema

Mental status Conscious, no coma Cerebral edema = CI for GA

If pH < 7.0 , $\text{K}^+ < 3$, SBP < 80 : Tell surgeon to wait. 1 hour of fluids + insulin in ICU changes mortality.

Essential investigations: VBG/ABG, glucose, K^+ , Na^+ , urea, creatinine, ECG, ketones. Calculate anion gap: > 12 = DKA.

3. Intra-op Management:

A. Monitoring

1. Mandatory: ECG, SpO_2 , EtCO_2 , A-line, CVP, Temp, UO.

2. Hourly: ABG, glucose, K^+ , Na^+ . DKA is dynamic.

3. Consider: TEE if shocked/cardiac disease.

B. Induction

1. Hypovolemia is the killer: Give 500mL-1L NS before induction even if you already resuscitated.

2. Drugs: Ketamine 1-2 mg/kg + fentanyl 1-2 mcg/kg. Avoid propofol/thiopentone bolus → ↓SVR → crash. Etomidate used if available.

3. RSI: DKA = full stomach risk + acidosis. Use succinylcholine only if $\text{K}^+ < 5.5$. If $\text{K}^+ > 5.5$, use rocuronium 1.2 mg/kg.

4. Airway: Hyperventilation needed to match pre-op compensation. Set RR to keep EtCO_2 2-3 mmHg below baseline. If baseline pCO_2 was 20, target EtCO_2 17-18. Don't "normalize" CO_2 → worsens acidosis.



C. Maintenance

1. Fluids: NS is fluid of choice initially. When glucose <200, add 5% dextrose to prevent hypoglycemia while continuing insulin.
2. Insulin: Continue 0.1 U/kg bolus + IV infusion 0.1 U/kg/h. Don't stop for surgery. Target glucose 150-200, not normal.
3. K⁺ replacement : DKA pts are total body K⁺ depleted despite normal/high serum K⁺. As acidosis corrects, K⁺ shifts intracellular → crash.
Add 20-40 mEq KCl/L when K⁺ <5.3 and UO present.
4. Bicarbonate : Only if pH <6.9 with hemodynamic instability. HCO₃⁻ may worsen hypokalemia, paradoxical CSF acidosis.
5. Ventilation: Low-moderate Vt 6-8 mL/kg. Avoid resp acidosis. No PEEP >5 if hypovolemic.
6. Drugs to avoid : Steroids, dextrose boluses, β-blockers.

D. Choice of anaesthesia

1. GA : Preferred. Controlled ventilation needed.
2. Regional : Avoid spinal/epidural → sympathectomy + hypovolemia = severe hypotension. If used, give lots of fluid + vasopressor ready.
3. LA : OK for minor procedures if patient stable.
4. Post-op
 1. ICU mandatory : Continue insulin drip + fluids + K⁺ monitoring till anion gap closed, HCO₃⁻ >18, pt eating.
 2. Transition : Overlap SC insulin 1-2h before stopping infusion to prevent rebound DKA.

3. Biggest risks: Hypokalemia, hypoglycemia, cerebral edema in kids, aspiration.

Fluid Therapy

- Initial: 0.9% saline 1–1.5 L (15–20 mL/kg) in first hour
- Hypovolaemic shock: continue 0.9% saline at 1 L/hour
- Cardiogenic shock: consider inotropes and haemodynamic monitoring
- Mild hypotension or normal: calculate corrected sodium — if high/normal: 0.45% saline at 4–14 mL/kg/h; if low: continue 0.9% saline
- When blood glucose < 14 mmol/L (250 mg/dL): change to 5% dextrose with 0.45% saline; continue until acidosis and ketosis resolve
- Aim: correct estimated water deficit over 24 hours at 4–14 mL/kg/hour
- Avoid Hartmann's solution: lactate load increases

glucose, potassium content risks hyperkalaemia, hypotonicity may precipitate cerebral oedema

- Avoid overly rapid or excessive fluid — risk of cerebral oedema

Insulin Therapy

- Give IV, not SC (subcutaneous absorption unreliable in shock)
- Exclude hypokalaemia before starting (K must be > 3.3 mEq/L; withhold insulin if K < 3.3 — risk of dangerous hypokalaemia)
- Do NOT use bolus insulin at diagnosis (no longer accepted practice); IV fluid alone reduces glucose by diluting blood glucose and counter-regulatory hormones
- Start infusion: 50 units actrapid in 50 mL 0.9% saline at 0.1 units/kg/hour
- Alternative: 0.1 units/kg IV bolus then 0.1 units/kg/hour infusion
- Target fall in plasma glucose: 3–4 mmol/L/hour
- If glucose fails to fall at target rate: double insulin rate hourly
- If glucose falls > 5 mmol/L/hour: temporarily reduce to 0.05 units/kg/hour
- If hypoglycaemia occurs before DKA resolved: add glucose to IV fluids — do NOT stop insulin infusion
- Continue insulin infusion until pH > 7.3 and acidosis/ketosis resolved and anion gap normalises.

Potassium Replacement

- Add 20–40 mmol/L KCl to maintain serum K around 4.5 mmol/L
- If initial K > 5.3 mmol/L: omit potassium; recheck every 2 hours
- If anuria or peaked T waves on ECG: omit potassium initially until K is known
- Continuous cardiac monitoring for K replacement > 10 mEq/hour

Sodium Bicarbonate

Rarely, if ever, necessary. Deleterious effects include hypokalaemia, cerebral oedema, and reduced tissue oxygenation (effects on O₂ dissociation curve). Acidosis improves with fluid and insulin; continuing acidosis = insufficient resuscitation.

- Consider ONLY in: profound acidosis (pH < 6.9) with circulatory failure resistant to inotropes
- Some protocols: bicarbonate if pH < 7.1 with haemodynamic instability
- Several studies show no difference in resolution of acidosis or time to discharge with bicarbonate use



Conversion to Subcutaneous Insulin

- Once plasma glucose < 10 mmol/L AND acidosis resolved (pH > 7.3): change from constant rate infusion to sliding scale
- Resume oral diet as soon as tolerated
- SC insulin can begin when patient tolerates oral diet and ketonuria fallen to 1+ or 0
- 20–30 minutes before meal: give SC insulin; continue IV insulin for another 2 hours (overlap to prevent rebound ketosis)

7. What are the Complications of DKA Treatment?

Cerebral Oedema

Monitor from admission for at least first 12 hours; typically presents 2–24 hours into treatment. Early signs: headache, confusion, irritability. Later signs: reduced consciousness, seizures.

Risk factors: severity/duration of DKA, overzealous fluid administration, sodium bicarbonate administration, too rapid glucose correction.

Management: exclude hypoglycaemia first → IV mannitol 1.0 g/kg (5 mL/kg 20% mannitol) immediately → restrict fluids to 2/3 maintenance, replace deficit over 72 hours → ICU transfer for intubation + mechanical ventilation → urgent CT head → neurosurgical liaison re: ICP monitoring.

Other Complications

- Hypoglycaemia: treat with increased dextrose; do not stop insulin before acidosis/ketosis resolved
- Hypokalaemia: standard replacement; central access allows K > 40 mmol/hour with cardiac monitoring
- Hypophosphataemia: usually resolves with oral diet; replacement in cardiac dysfunction, anaemia, or respiratory depression
- Venous thrombosis (↑ risk with hypercoagulable state of DKA)
- Thromboprophylaxis: prophylactic heparin (LMWH or UFH) until mobile, rehydrated, and osmolality normalised
- Nasogastric tube drainage: DKA causes gastric stasis; mandatory with impaired consciousness
- Urinary catheterisation: required for accurate fluid balance
- Continuous ECG monitoring: significant cardiac disease, hyper/hypokalaemia, severe DKA
- Upper GI bleeding and acute respiratory distress syndrome: occasional complications

- Lactic acidosis: metabolic acidosis without elevated ketones → suggest poor tissue perfusion or sepsis; diagnose with elevated serum lactate

8. Criteria for resolution of Diabetic Ketoacidosis

- DKA is considered resolved when all of the following are achieved: Plasma glucose < 200 mg/dL (~11.1 mmol/L) AND at least two of the following: Serum bicarbonate ≥ 15 mEq/L Venous/arterial pH > 7.3 Anion gap ≤ 12 mEq/L (normalized)

DKA vs HHS		
Diabetic ketoacidosis		Hyperosmolar hyperglycaemic state
More common in type 1 diabetes mellitus	Association	More common in type 2 diabetes mellitus
Relatively lower	Mortality rate	Relatively higher
Abdominal pain, polyuria, polydipsia, dehydration, Kussmaul breathing, acetone-smelling (sweet) breath	Presentation	Fatigue, altered level of consciousness, hyperviscosity (increases risk for MI and stroke), hypotension
Hours	Onset	Days
1. Glucose >11.1 mmol/L 2. pH <7.3 or bicarbonate <15 mmol/L 3. Ketone >3 mmol/L or urine ketone ++ on dipstick	Diagnostic criteria	1. Hypovolaemia 2. Marked hyperglycaemia (>30 mmol/L) without significant ketonuria or acidosis 3. Significantly raised serum osmolality (>320 mosmol/kg)
• Fluid replacement • IV insulin (0.1 unit/kg/h) • Potassium replacement	Management	• Normalised the osmolality gradually • Replace fluid and electrolyte • No insulin unless significant ketonuria or acidosis
Thromboembolism, gastric stasis, arrhythmias secondary to hyper/hypokalaemia, ARDS, AKI (iatrogenic due to incorrect fluid therapy), cerebral oedema, hypokalaemia, hypoglycaemia	Complication	MI, stroke, peripheral arterial thrombosis, Rare: seizure, cerebral oedema, central pontine myelinolysis

GRAM PROJECT

6: HYPERGLYCAEMIC HYPEROSMOLAR STATE (HHS)?

Diagnosis · Pathophysiology · Management?

1. Definition and Epidemiology

HHS represents one extreme in the spectrum of decompensated DM (DKA the other). It typically affects elderly patients with mild or minimal DM and presents with remarkably high blood glucose levels (> 600 mg/dL) and profound dehydration (estimated fluid deficit: 9–12 L). Mortality is substantially higher than DKA: 10–20% (some series report 15% vs DKA 1%). The main differential from DKA: HHS patients present with pH > 7.3 — sufficient residual insulin prevents lipolysis and ketosis even at blood glucose of 1,000 mg/dL (only 1/10 of the insulin required to suppress lipolysis vs stimulate glucose utilisation)

2. Enumerate the Precipitating Factors?

- Myocardial infarction
- Stroke
- Sepsis
- Pneumonia and other serious infections
- Impaired thirst response + decreased fluid intake in relative insulin deficiency

1. Pathophysiology of HHS?

Relative insulin deficiency allows hyperglycemia to develop. Hyperglycemia induces osmotic diuresis → intravascular volume depletion.



Marked hyperosmolality may lead to coma and seizures. Increased plasma viscosity predisposes to intravascular thrombosis. Unlike DKA, enough insulin remains to suppress lipolysis-no significant ketoacidosis.

4. Brief about the Management of HHS?

HHS responds quickly to rehydration and small doses of insulin.

- If no cardiovascular contraindications: infuse 1–2 L (or 15–30 mL/kg) normal saline over first hour
- CRITICAL: Administer insulin AFTER initial volume has been given-premature insulin → glucose uptake moves water out of intravascular space → severe hypovolemia and cardiovascular collapse
- After adequate hydration: administer insulin (bolus or infusion)

Slow correction of osmolality and hyperglycemia to avoid cerebral oedema-recovery of mental acuity may be delayed even after glucose and volume normalised

- Concurrent treatment of underlying precipitant (infection, MI, etc.)

7: HYPOGLYCAEMIA?

Classification · Clinical Features · Perioperative Considerations?

3. Overview

Hypoglycaemia is a major limiting factor in glycemic management of diabetic patients and the most serious acute complication of insulin therapy. The precise blood glucose level causing symptoms is variable — a normal fasted patient may have glucose no higher than 50 mg/dL without symptoms, while a chronically hyperglycaemic diabetic may be symptomatic at higher levels. Hypoglycaemia is almost impossible to diagnose clinically in the unconscious patient.

4. How do you Classify (ADA Three-Level System)?

- Level 1: glucose < 70 mg/dL but > 54 mg/dL - alert value; review medications
- Level 2: glucose < 54 mg/dL - threshold at which neuroglycopenic symptoms begin; requires immediate treatment
- Level 3: glucose: < 40 mg/dL: severe event with altered mental and/or physical functioning requiring assistance from another person for recovery
Administer 100ml of 25D or 50ml of 50D or 1mg (IV/IM) glucagon

3. List the Clinical Features?

Autonomic/Adrenergic Symptoms

- Sweating
- Palpitations
- Tremor
- Hunger

Neuroglycopenic Symptoms

- Behavioural and cognitive impairment
- Drowsiness
- Speech difficulty
- Blurred vision
- Seizures
- Coma
- Death

4. Perioperative Considerations

CRITICAL PERIOPERATIVE RISK: In the anaesthetised patient, autonomic signs of hypoglycaemia (tachycardia, hypertension, diaphoresis) can be MISINTERPRETED as inadequate or 'light' anaesthesia. β -adrenergic blocking agents or advanced diabetic autonomic neuropathy may further blunt these signs. Maintain high clinical suspicion and frequent glucose monitoring in all anaesthetised diabetic patients.

- Autonomic and early neurological signs notably absent in intubated, sedated, critically ill, or anaesthetised patients
- Signs of sympathetic hyperactivity can mimic 'light' anaesthesia
- β -blockers or advanced autonomic neuropathy blunt the sympathetic hypoglycaemia response
- Hypoglycaemia rare compared to hyperglycaemia but associated with increased mortality
- Monitor glucose at least hourly in high-risk patients; correct immediately with IV dextrose

8: PERIOPERATIVE HYPERGLYCAEMIA

Pathophysiology of Harm · Modulators · Outcome Data

1. What are the effects of Hyperglycaemia?

- Decreased immune function (impaired chemotaxis, phagocytosis, reactive oxygen species generation, intracellular bacterial killing)
- Increased oxidative stress
- Endothelial dysfunction (decreased nitric oxide production → reduced vascular reactivity)
- Increased inflammatory factors
- Procoagulant state
- Increased mitogen levels



- Fluid shifts and electrolyte fluxes
- Delayed wound healing and increased surgical site infections
- Delayed recovery and increased hospital LOS
- Poor outcomes in MI and stroke (acute hyperglycaemia)

2. Modulators of Perioperative Hyperglycaemia

Preoperative metabolic state:

- Diabetes
- Sepsis/inflammation
- Obesity

Intraoperative management factors:

- Anaesthetic agents (decrease insulin secretion)
 - Dextrose-containing IV fluids
 - Steroids
 - Adrenergic agonists
 - Alpha-2 agonists
- Hypothermia

Neuroendocrine stress response:

- Cardiac surgery
- Surgical duration
- Location of surgery
- Surgical technique

Acute insulin resistance is directly related to degree of surgical trauma. Insulin sensitivity can fall to ~45% of baseline on postoperative day 1–2, gradually recovering by day 20.

1. DKA Management Summary Table

Component	Key Action
Airway/Breathing/Circulation	ABCD assessment; 100% O ₂ via face mask + reservoir bag; IV access; blood cultures
Fluids (Adults)	0.9% saline 1–1.5 L in first hour; then based on corrected Na; 5% Dextrose + 0.45% saline when glucose < 14 mmol/L
Insulin	0.1 U/kg bolus + 0.1 units/kg/hour infusion (after excluding hypokalaemia); target glucose fall 3–4 mmol/L/hour; do NOT use bolus
Potassium	Add 20–40 mmol/L KCl; target K ~4.5 mmol/L; withhold insulin if K < 3.3 mEq/L
Bicarbonate	Only if pH < 6.9 with circulatory failure resistant to inotropes
Monitoring	Hourly glucose; U&E; and blood gas q2–4h; strict fluid balance; continuous ECG if significant cardiac/electrolyte issues
Complications to watch	Cerebral oedema (esp. children), hypoglycaemia, hypokalaemia, hypophosphataemia, VTE
Transition to SC insulin	Glucose < 10 mmol/L AND pH > 7.3; overlap IV and SC insulin for 2 hours around meals
Antibiotic therapy	Treat identified infections aggressively; prophylaxis per local guidelines
Thromboprophylaxis	LMWH or UFH until mobile and fully hydrated

Table 8 — DKA Management Summary 2.

Perioperative Diabetic Checklist?

Phase	Checklist Item
PRE-OP	Post the diabetic case as 1st case of the day
PRE-OP	HbA1c: target < 8%; check ECG, renal function, glucose, urine ketones, serum electrolytes
PRE-OP	Assess for CAD, autonomic neuropathy (orthostatic BP, HR variability), gastroparesis, stiff joint syndrome
PRE-OP	Medication adjustment: stop oral agents night before; SGLT2 inhibitors 3–4 days before; adjust insulin per protocol
INTRA-OP	Monitor glucose every 1–4 hours (hourly for high-risk/pump patients)
INTRA-OP	Target glucose 140–180 mg/dL; start insulin infusion if > 180 mg/dL
INTRA-OP	Avoid routine dextrose-containing IV fluids unless glucose trending low
INTRA-OP	Careful positioning — neuropathy/PVD increases nerve and limb injury risk
INTRA-OP	Awake intubation if "Prayer Sign" positive or other difficult airway predictors
POST-OP	Continue glucose monitoring q4–6h; transition to basal-bolus insulin (not sliding scale alone)
POST-OP	Watch for hypoglycaemia — missed in recovery; monitor glucose in PACU
POST-OP	Gastric bypass patients: significant reduction in insulin/oral agent requirements post-surgery
POST-OP	SGLT2 inhibitor patients: euglycaemic DKA risk up to 10–14 days post-op if not restarting oral intake

Table 9 — Perioperative Diabetic Patient Checklist

Enumerate the different Regimens of insulin preparations with their advantages and disadvantages?

GIK regimen: (Albertti regimen) : (modified)

Here, 5% dextrose 500ml + 10U rapid acting insulin + 15meq KCl Administered over 5 hrs (100ml/hr)

Advantage: provides potassium correction alongside glucose and insulin as infusion

- **Disadvantage:** with every time change in BG levels, new set of preparation of infusion has to be made and previous one is discarded.

Vellore regimen:

Blood glucose (mg/dL)	Treatment
< 70	Stop insulin if on infusion. Rapid infusion of 100 mL of D5W, measure blood glucose after 15 min
71–100	Stop insulin, infuse D5W at 100 mL/h
101–150	1 U of insulin + 100 mL of D5W/h
151–200	2 U of insulin + 100 mL of D5W/h
201–250	3 U of insulin + 100 mL of D5W/h
251–300	4 U of insulin + 100 mL of D5W/h
> 300	1 U of insulin for every 1–50 mg more than 100 mg/dL + 100 mL of normal saline/h

D5W = 5% dextrose in water.

Advantages: insulin infusion can be varied accordingly to the BG levels

Disadvantage: doesn't provide potassium correction



VRIII regimen: (Variable rate intravenous insulin infusion)

First, activating dose of insulin is administered to the patient based on,

(BG-100)/ISF

Where, ISF- insulin sensitivity factor
(ISF= 1800/total daily dose of insulin)

Next, Start the patient on infusion of insulin based on BG/100, Check BG levels hourly, if BG increased by 30mg/dl, add 1U of insulin to the ongoing infusion to maintain 140-180mg/dl.

Insulin Sensitivity Factor (ISF) = Correction Factor

This is how much 1 unit of rapid-acting insulin will lower your blood glucose. Very important for VRIII and diabetic patients in OT

1. What is ISF?

Definition: The drop in blood glucose mg/dL caused by 1 unit of rapid-acting insulin.

Other names: Correction factor, Insulin correction factor Used for: Calculating correction dose when glucose is high

2. How to calculate ISF? – "Rule of 1800" or "Rule of 100"

For *mg/dL* system used in India:

ISF = 1800 ÷ Total Daily Dose of insulin

Total Daily Dose (TDD) = All insulin in 24h = Basal + Bolus + Correction

Example:

Patient takes Lantus 20U morning + Actrapid 8U TID = 20 + 24 = *44U TDD*

ISF = 1800 ÷ 44 = 40 mg/dL

Meaning: 1 unit insulin will drop glucose by ~40 mg/dL

For *Type 1 DM*: Use 1800 Rule

For *Type 2 DM*: Use 1500 Rule as they're more insulin resistant ISF = 1500 ÷ TDD

3. When do we use ISF in anaesthesia/OT?

A. VRIII fine-tuning

Standard VRIII charts are fixed. But if pt is very sensitive or resistant, we use ISF to adjust.

Example: Pt on table, glucose = 280, target = 150

Excess glucose = 280 - 150 = 130 mg/dL

If ISF = 40, then correction dose = 130 ÷ 40 = 3.25 units stat bolus

You can give this as bolus + adjust VRIII rate.

B. Treating intraop hyperglycemia

Instead of blindly increasing VRIII, calculate exact correction.

Prevents "overshooting" and causing hypoglycemia.

C. Planning post-op transition from VRIII to SC

When pt starts eating, you need correction dose for meals. *Correction dose = (Current glucose - Target glucose) ÷ ISF*

4. Important ISF points for anaesthetists

Situation ISF change Why

****Sepsis/Steroids/Surgery stress**** ISF decreases Insulin resistance ↑, so 1U drops glucose less. Need more insulin

Renal failure ISF increases, Insulin clearance ↓, so 1U drops glucose more. Risk of hypoglycaemia.

****Pregnancy**** ISF decreases, esp 3rd trimester

Hormones cause resistance

****DKA resolving**** ISF increases As acidosis corrects, sensitivity returns. Risk of hypo if rate not reduced

***Thumb rule*: Sick patient in ICU/OT = assume insulin resistance. Start with ISF 25-30 even if calculated ISF is 50.**



13th JUNE 2026

11:05AM - 11:45PM

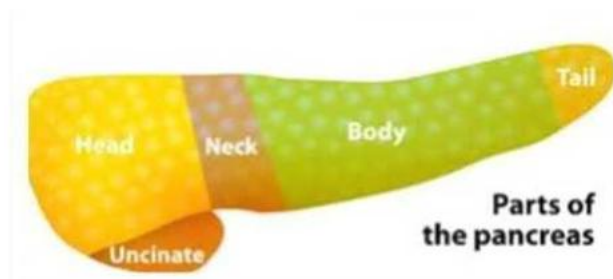
ANAESTHETIC MANAGEMENT OF A PATIENT WITH OBSTRUCTIVE JAUNDICE (CA HEAD OF PANCREAS) – POSTED FOR WHIPPLE'S PROCEDURE.

Dr Arathi B H (Prof & HOD), Department of Anaesthesiology, KMIO, Bengaluru.

Dr Amrutha. A , Dr Nikhila. G (post graduates)

ANATOMY OF PANCREAS:

- The pancreas is divided into head, neck, body, and tail anatomically.
- It is a secretory organ with both:
Exocrine function → digestion
Endocrine function → regulation of blood sugar

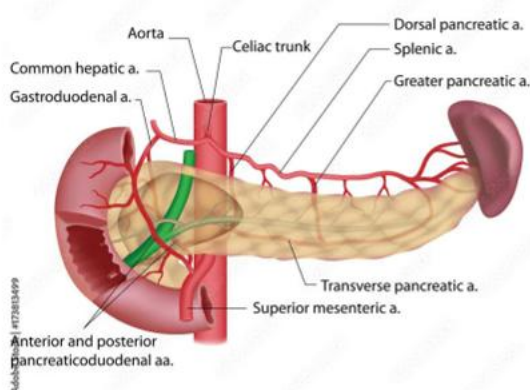


Arterial supply:

Branches of:

- o Common hepatic artery
- o Splenic artery (from celiac artery)
- o Superior mesenteric artery

Arterial Supply of the Pancreas

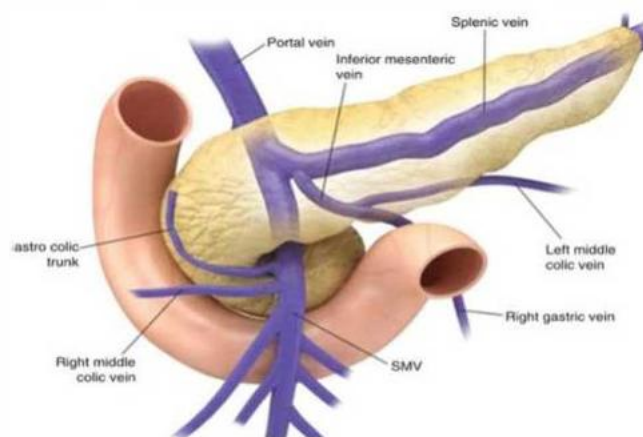


Venous drainage:

- o Superior mesenteric vein
- o Portal vein

Nerve supply:

- o Sympathetic → splanchnic nerve
- o Parasympathetic → vagus nerve



Relations of the Pancreas

- Anterior: Stomach, lesser sac, transverse colon, and transverse mesocolon.
- Posterior: Aorta, inferior vena cava (IVC), splenic vein, portal vein, superior mesenteric vessels, left kidney, left adrenal gland, and crus of the diaphragm.
- Superior: Celiac trunk, hepatic artery, and splenic artery.
- Inferior: Duodenum (3rd part), jejunum, and mesenteric vessels.

Lymphatic Drainage

- Follows the arterial supply.
- Drains into the pancreaticosplenic, celiac, pyloric, and superior mesenteric lymph nodes.

PHYSIOLOGICAL ASPECTS:

Exocrine:

- 90% of pancreas is formed by exocrine acinar cells
- These secrete digestive proteolytic enzymes into a tubular system
- This system ultimately forms the main pancreatic duct. Around 2000 mL/day of enzyme-rich alkaline fluid is produced
- Regulation:
- Neuroendocrine system



- Modulated by endocrine pancreas
- **Function:** Breakdown of nutrients for absorption

Endocrine:

- Endocrine cells form the Islets of Langerhans
- These are the functional units of the endocrine pancreas
- Adult pancreas contains ~1 million islets • Constitutes 1–2% of gland
- Cell types in islets:
 - † β (Beta) cells → secrete insulin
 - † α (Alpha) cells → secrete glucagon
 - † δ (Delta) cells and F cells → secrete pancreatic polypeptide (PP)
 - † Main role → Glucose homeostasis

WHAT IS OBSTRUCTIVE JAUNDICE?

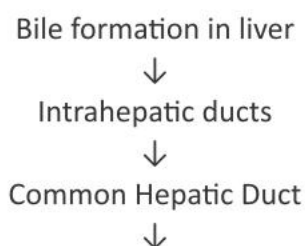
Jaundice : Yellowish discolouration of sclera, skin and mucus membrane due to increased serum bilirubin level (>2mg/dl)

Obstructive jaundice is due to mechanical obstruction to bile flow causing accumulation of conjugated bilirubin in the blood

Classification of Jaundice

- Pre-hepatic (Hemolytic): Excess production of bilirubin overwhelming hepatic conjugating capacity (e.g., malaria, sickle cell anemia, spherocytosis, mismatch blood transfusion).
- Hepatic (Hepatocellular): Damage to hepatocytes reducing uptake, conjugation, and excretion (e.g., viral hepatitis, alcoholic liver disease, cirrhosis, drugs/toxins like paracetamol).
- Post-hepatic (Obstructive/Surgical): Mechanical obstruction to the extrahepatic biliary system preventing bile flow into the duodenum (e.g., choledocholithiasis, periampullary carcinoma, CA head of pancreas, biliary stricture).

Bile Synthesis and Pathway:



Gallbladder (Storage & Concentration)



Causes of Biliary Obstruction:

- BENIGN:
 - -Choledocholithiasis (CBD stone) is the most common etiology.
- MALIGNANT:
 - Cholangiocarcinoma
 - Periampullary carcinoma
 - Pancreatic cancers
 - Benign stricture

HYPERBILIRUBINEMIA

UNCONJUGATED:

- ↑ Bilirubin Production
- ↓ Hepatic uptake
- ↓ Conjugation

CONJUGATED:

- Obstruction of bile ducts
- ↓ Canalicular transport
- Acute/chronic hepatocellular dysfunction

PATHOPHYSIOLOGY OF OBSTRUCTIVE JAUNDICE

1. Gastro Intestinal Tract:

- **Malabsorption of fats and steatorrhea**
- Poorly absorbed fat-soluble vitamins – A,D,E,K and their corresponding deficiency symptoms.
- Deficiency of vitamin K – Prolonged prothrombin time and loss of Coagulation factors.
- **Altered Drug Metabolism:** Impaired hepatic clearance and decreased cytochrome P450 enzyme activity lead to significantly prolonged drug half-lives and an increased risk of systemic toxicity.



Anesthetic implication:

- Decreased albumin—increased free drug concentration and increased sensitivity to anesthetic agents.
- Reversal of coagulopathy is important for managing intraoperative bleeding and also for epidural analgesia.

2. Bacterial translocation:

- Due to obstruction, bacteria are accumulated in bile and develop infectious complications.
- These bacteria release endotoxins into the vascular system eventually leads to systemic inflammatory response syndrome leading to sepsis.

Anesthetic implication: Bacterial translocation increases risk of sepsis and vasodilatory shock, so, invasive monitoring, and readiness for vasopressors are essential

3. Intestinal Mucosal Barrier Dysfunction:

- When obstructive jaundice occurs, the intestinal barrier function is damaged and gut permeability is increased.
- Thus enabling intestinal pathogens and endotoxin to invade into the mesenteric lymph nodes, portal vein circulation.
- Meanwhile, the accumulated intrahepatic bile acids significantly inhibit the clearance of Kupffer cells (KC) from the resident macrophages, causing endotoxin spilling into the systemic circulation from the portal vein and sustained release of proinflammatory cytokines, eventually forming gut-derived sepsis and multi-organ failure.

1. Renal Pathophysiology:

- Endotoxins absorbed from the intestine and entering the systemic circulation may cause renal vasoconstriction
- RENAL VASOCONSTRICTION—HYPOPERFUSION—RENAL FAILURE.
- Correction of volume deficits in patients with bile duct obstruction can increase renal blood flow and urine output and decrease circulating concentrations of toxic substances.
- Bilirubin Cast Nephropathy: High levels of circulating conjugated bilirubin filtration lead to the intratubular precipitation of bilirubin casts, causing

direct cellular toxicity to proximal tubular cells and acute tubular necrosis (ATN).

- **Anesthetic Implication:** The preoperative use of nephrotoxic drugs such as aminoglycoside antibiotics may exacerbate kidney damage.

5. Cardiovascular physiology

- Obstructive jaundice → bile salts (taurocholate, taurogallate) + endotoxins ↑ → nitric oxide & prostaglandins production → peripheral vasodilation → ↓ SVR → hypotension.
- **Vascular hypo responsiveness** : These mediators also cause vascular hyporesponsiveness → and the blood vessels become insensitive to endogenous and sympathomimetics → blood loss poorly tolerated.
- Additionally, **myocardial depression** → ↓ contractility → ↓ cardiac output → worsened hypotension.
- **Volume depletion** : Bile salts impair Na⁺ reabsorption in proximal convoluted tubules → further ↓ circulating volume → volume depletion
- Biliary manipulation → vagal stimulation → **bradycardia** → further compromise of cardiac output.
- Subclinical Cardiomyopathy: Chronic exposure to high concentrations of bile acids and jaundice results in subclinical myocardial remodeling, localized fibrosis, and a blunted cardiac index response under acute stress.
- Refractory Hypotension: The profound down-regulation of alpha-1 adrenergic receptors induces a state of vasoplegia, making intraoperative hypotension resistant to standard fluid boluses and low-dose sympathomimetics.

•Anesthetic implication :

- Maintain MAP → fluids + early vasopressors
- Titrate induction → avoid hypotension
- Avoid myocardial depressants & nephrotoxins
- Prepare for bradycardia & blood loss intolerance

6. Hematological System:

- Hyperbilirubinemia alters the red blood cell (RBC) membrane lipid composition, significantly reducing RBC deformability.



- This reduction in flexibility accelerates splenic clearance, inducing a subclinical hemolytic state and worsening chronic anemia.
- Anesthetic Implication: Optimizing baseline hemoglobin and ensuring strict intraoperative oxygenation parameters are vital to prevent tissue hypoxia.

CLINICAL FEATURES

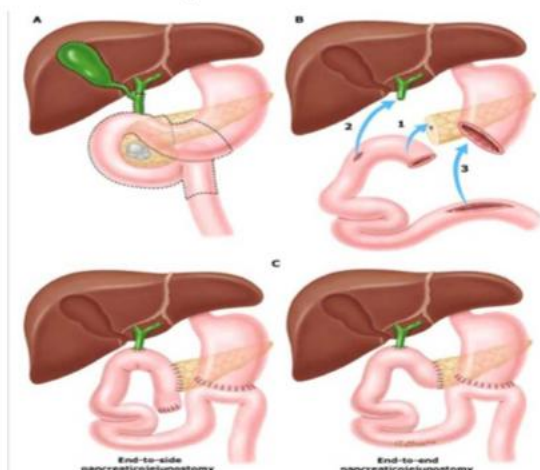
- 1) Abdominal pain
- 2) Weight loss
- 3) Generalized pruritus
- 4) Malnutrition
- 5) Fatigue
- 6) High colored urine
- 7) Clay colored stools

WHAT IS WHIPPLE'S SURGERY?

- A Whipple procedures (or Whipple's procedure) is a pancreaticoduodenectomy. It's known as a "Whipples" after Allen Oldfather Whipple, who in the 1930s modified a much older procedure originally described by Alessandro Codivilla (in 1898).
- Whipple resection consists of a pancreaticoduodenectomy followed by anastomosis of the pancreas, liver and stomach to the jejunum.
- If diffuse carcinomatosis is discovered the operation is aborted
- If the tumor is resectable then the procedure is done as follows :

Head of the pancreas is mobilized

Resection of the proximal pancreas, along with distal stomach, duodenum, distal bile duct, gall bladder and lymph nodes as an en-bloc specimen and The multiple anastomoses are performed



RISK ASSESSMENT

A. Child and Turcotte (1964)

- Originally conceptualized by Child and Turcotte .
- To guide the selection of patients who would benefit from elective surgery for portal decompression

Three categories:

A - good hepatic function

B - moderately impaired hepatic function C - advanced hepatic dysfunction.

	CLASS A	CLASS B	CLASS C
S. Bilirubin (umol/L)	<40	40-45	>50
S. Albumin (g/dL)	>3.5	3-3.5	< 3
Ascites	nil	Easily controlled	Poorly controlled
Encephalopathy	none	minimal	advanced
Nutrition	excellent	good	Poor
Operative mortality	5%	10%	50%

B. Modified Child- Pugh

In 1972, Pugh modified the score, replacing nutritional status with INR

- Predictor of postop mortality risk after major surgery.

Limitations

- a) Grading ascites and encephalopathy require a subjective assessment
- b) It does not account for renal function

	1	2	3
Encephalopathy	None	Grade 1-2	Grade 3-4
Ascites	None	Mild-Moderate	Severe
Bilirubin (mg dL)	<2	2-3	>3
Albumin (g 'dL)	>3.5	2.8-3.5	<2.8
INR	<1.7	1.7-2.3	>2.3

POINTS	CHILD-PUGH	CLINICAL SIGNIFICANCE	MORTALITY
5-6	A (Least severe liver disease)	safe candidates for elective surgery	10%
7-9	B(Moderate severe liver disease)	proceed with surgery after medical optimization	30%
10-15	C(Most severe liver disease)	Contraindicated for elective surgery	80%



C. MELD (Model for End Stage Liver Disease

Minimum for all values is 1

Maximum value for creatinine is 4

MELD score <8 predicts good outcome and score >18 predicts poor outcome

A MELD score of **at least 8** predicts an increased risk of postoperative complications MELD Score Formula:

$$\text{MELD} = (0.957 \times \ln [\text{Cr}]) + (0.378 \times \ln [\text{bilirubin}]) + (1.120 \times \ln [\text{INR}]) + 0.643$$

Bilirubin in mg/dL

INR = International Normalized Ratio Creatinine in mg/dL Special

Note:

If patient had dialysis ≥ 2 times in last 7 days $\rightarrow$ creatinine = 4 mg/dL

3-Month Mortality Based on MELD

$\leq 9 \rightarrow 1.9\%$

10–19 $\rightarrow 6.0\%$

20–29 $\rightarrow 19.6\%$

30–39 $\rightarrow 52.6\%$

$\geq 40 \rightarrow 71.3\%$

PRE-OPERATIVE ASSESSMENT

PRE HABILITATION:

1. Counselling

- Explain surgery, ICU stay, recovery course
- Reduce anxiety $\rightarrow$ improves cooperation & outcomes

2. Nutritional Optimization

- High-protein, high-calorie diet
- Correct malnutrition (common in obstructive jaundice)
- Consider enteral supplements $\pm$ pancreatic enzymes

3. Respiratory Training

- Incentive spirometry
- Deep breathing exercises
- Chest physiotherapy
- $\downarrow$ Post-op pulmonary complications

4. Early Mobilisation

- Start within 24 hours

- Prevents DVT, atelectasis, muscle wasting

5. Pain Management

- Thoracic epidural (preferred)
- Multimodal analgesia

6. Selective Biliary Drainage

- Not routine, Indicated in:
- Severe jaundice (>15 mg/dL)
- Cholangitis
- Renal dysfunction

Assess comorbidities (diabetes mellitus, hypertension, COPD, heart failure, nutritional status) and optimization
Labs: CBC, electrolytes, liver function tests, renal function tests, coagulation studies, glucose.
Cardiac Risk Markers NT-proBNP : Screening for cardiac dysfunction $\uparrow$ levels $\rightarrow$ $\uparrow$ risk of myocardial injury Indications: Age ≥ 65 years, Age 45–64 with significant CVD, Revised Cardiac Risk Index ≥ 1
Functional Capacity & Frailty : Assess METS
Risk Scoring: P-POSSUM : SORT $\rightarrow$ morbidity & mortality prediction, PREPARE score (pancreatic resection-specific)
VTE prophylaxis: compression stockings + LMWH if not contraindicated.
Antibiotic and antiemetic prophylaxis.
Explain analgesia (multimodal)

1. Hematological & Coagulation Profile:

- Complete Blood Count (CBC): To rule out anemia (due to chronic disease/malnutrition) and leukocytosis (indicative of cholangitis).
- Coagulation Studies: Prothrombin Time (PT), Activated Partial Thromboplastin Time (aPTT), and INR to quantify the baseline coagulopathy secondary to Vitamin K malabsorption.

2. Liver Function Tests (LFTs):

- Total & Fractionated Bilirubin: Confirms a predominant rise in conjugated (direct) bilirubin.
- Alkaline Phosphatase (ALP) & Gamma-Glutamyl Transferase (GGT): Elevated >3 times the upper limit of normal, confirming a canalicular/obstructive pattern.
- Transaminases (AST/ALT): Usually normal or mildly elevated; a sharp, massive spike indicates secondary hepatocellular injury or acute cholangitis.
- Serum Albumin & Total Protein: Serves as a direct indicator of hepatic synthetic function and chronic nutritional depletion.

3. Renal Function & Electrolytes:

- Serum Creatinine & Blood Urea Nitrogen (BUN): Essential to calculate baseline MELD scores and monitor for Hepatorenal Syndrome (HRS) or cast nephropathy.



- Serum Electrolytes (Na⁺, K⁺, Cl⁻, Ca²⁺): Essential to detect baseline derangements secondary to third-spacing, vomiting, or altered renal handling.

4. Tumor Markers:

- CA 19-9: Highly associated with pancreatic adenocarcinoma and periampullary tumors (used for baseline staging and monitoring response).
- CEA (Carcinoembryonic Antigen): Evaluated as a secondary non-specific marker for GI malignancies.

5. Imaging Modalities:

- Transabdominal Ultrasound: The initial screening tool to demonstrate intrahepatic biliary duct (IHBRD) dilation and gallstones.
- Triple-Phase Contrast-Enhanced CT (CECT) Abdomen: The gold standard for surgical staging, assessing tumor resectability, and evaluating major vascular invasion (SMV, Portal Vein, Celiac Axis).
- M R C P (M a g n e t i c R e s o n a n c e Cholangiopancreatography): Non-invasive mapping of the biliary tree anatomy and localized site of obstruction.
- E R C P (E n d o s c o p i c R e t r o g r a d e Cholangiopancreatography): Reserved for therapeutic interventions, such as biliary brush cytology or placing a plastic/metal stent for selective biliary drainage.

Intra operative anesthetic management

Pre-Medication & Airway Strategy

- Aspiration Prophylaxis: Due to delayed gastric emptying associated with pancreatic tumors and mechanical bile duct obstruction, administer an H₂-receptor antagonist (e.g., Ranitidine 50 mg IV) and a prokinetic agent (e.g., Metoclopramide 10 mg IV) 30–60 minutes pre-induction.
- Anxiolysis: Avoid or significantly down-titrate long-acting sedatives or benzodiazepines (like midazolam) in patients with advanced jaundice or high MELD scores to prevent prolonged emergence or worsening hepatic encephalopathy.
- Airway Control: Perform a smooth, controlled induction. If severe delayed gastric emptying or gastroparesis is suspected clinically, utilize a Rapid Sequence Induction (RSI) with cricoid pressure to definitively secure the airway and prevent pulmonary aspiration

Anaesthesia Technique General anaesthesia + epidural analgesia (standard) Thoracic epidural (T6–T9) level Bupivacaine 0.1–0.2% +/- opioid
Vascular Access 2 large-bore IV cannulas Central line if: major resection hemodynamic instability Blood arranged (=2 units crossmatched)
Monitoring Standard: ECG, NIBP, SpO ₂ , EtCO ₂ , temperature, urine output Invasive arterial line ? beat-to-beat BP + ABG CVP dynamic indices (SVV, PPV) for fluid guidance Consider TEE in high-risk cardiac patients Neuromuscular monitoring (TOF)
Induction Prefer: Propofol (titrated) to prevent hypotension Etomidate (hemodynamically stable) Avoid: thiopentone (hepatic metabolism)
Maintenance Isoflurane / Desflurane Sevoflurane (minimal hepatic metabolism) Avoid halothane (hepatotoxic) MAC ? in jaundice ? titrate carefully Maintain adequate hepatic perfusion
Muscle Relaxants Preferred: Atracurium / Cisatracurium (Hofmann elimination) Avoid/limit: vecuronium, rocuronium (hepatic dependence) Succinylcholine ? caution (bradyarrhythmia)
Hemodynamic Goals Maintain: Normovolemia, MAP adequate for hepatic & renal perfusion ○ Vasopressors (noradrenaline) if needed
Fluid Management Goal-directed therapy --The goal is to optimize oxygen delivery and tissue perfusion by improving cardiac output and stroke volume. Avoid: Overload ? edema, anastomotic leak Under-resuscitation ? renal hypoperfusion Use: SVV / PPV for fluid responsiveness Maintain urine output (>0.5 mL/kg hr)
Coagulation & Blood Correct coagulopathy: Vitamin K / FFP platelets Monitor: PT INR, Hb Replace blood loss appropriately
Analgesia Epidural = gold standard Opioids: Prefer fentanyl/remifentanyl Avoid morphine (active metabolites, prolonged action) Multimodal analgesia
Antibiotic Prophylaxis Give within 30–60 min before incision Repeat dose if prolonged surgery / major blood loss
Metabolic & Other Care Maintain: Normoglycemia, Electrolytes Avoid drugs with extensive hepatic metabolism Prevent air embolism (open abdomen handling)

INTRAOPERATIVE COMPLICATIONS:

- Massive blood loss (major vessels)
- Hemodynamic instability (long duration + fluid shifts), coagulopathy (jaundice/massive transfusion)
- Bile leak/spillage, pancreatic leak (friable gland)
- Hypothermia, electrolyte & glucose imbalance,
- Rare air embolism, and adjacent organ/vascular injury



POST OPERATIVE CARE

Airway & Ventilation

- Early extubation only if normothermic, hemodynamically stable, no acidosis
- Otherwise elective ventilation (common due to long surgery + fluid shifts)

ICU Care (48–72 h)- due to long duration surgery, major fluid shifts

- Continuous monitoring: invasive BP, urine output, $\pm$ CVP
- Watch for delayed instability (3rd space loss)

Hemodynamic Management

- Maintain MAP ≥ 65 mmHg
- Goal-directed fluids (avoid overload $\rightarrow$ anastomotic leak; avoid deficit $\rightarrow$ AKI)
- Vasopressors often required (vasodilated, jaundiced patients)

Temperature

- Correct hypothermia (common due to long duration)
- Prevent coagulopathy & delayed recovery

Analgesia : Implement multimodal

Respiratory Care

- High risk : atelectasis (long incision + pain)
- Incentive spirometry, early physiotherapy, adequate analgesia

Fluids, Electrolytes & Glucose

- Frequent monitoring (major shifts intra-op)
- Maintain urine output >0.5 mL/kg/hr
- Hyperglycemia common (pancreatic resection) $\rightarrow$ insulin

Drain Monitoring

- Pancreatic leak $\rightarrow$ high drain amylase
- Bile leak $\rightarrow$ bilious output
- Watch for hemorrhage

Nutrition

- Early enteral feeding (24–48 h)
- High risk of malnutrition $\rightarrow$ aggressive support

Post operative complications

- Pancreatic fistula
- Delayed gastric emptying
- Sepsis
- Bleeding
- AKI

VTE Prophylaxis

- LMWH + mechanical (high risk due to malignancy + long surgery)

Mobilization

- Early (<24 h) to reduce pulmonary & thrombotic complications

WHY IS WHIPPLE'S SURGERY CONSIDERED HIGH-RISK FOR ANESTHESIA?

- Both the disease physiology and surgical stress synergistically increase anesthetic risk
- Multisystem involvement—
- Hepatic dysfunction alters drug metabolism, coagulopathy increases bleeding risk
- Cardiovascular instability predisposes to hypotension, with added renal vulnerability.
- Superimposed on this, Whipple's procedure is a prolonged major surgery associated with significant blood loss, fluid shifts, hypothermia, and metabolic disturbances, further compounding perioperative risk

"Knowledge of pancreatic anatomy and physiology is essential in anesthesia for Whipple surgery to anticipate hemorrhage, manage metabolic derangements, and respond to hemodynamic instability."

CASE HISTORY

A 61 year old male, agricultural worker from chikkaballapur, presented with complaints of :

- Loss of appetite and weight $\times 3$ months
- Yellowish discoloration of eyes $\times 20$ days
- High colored urine $\times 20$ days

Patient was apparently normal 3 months back, had loss of weight upto 6 kg associated with loss of appetite , developed yellowish discoloration of eyes and high coloured urine over the last 2 weeks, not



associated with waxing and waning.

K/C/O systemic hypertensive from 6 months on Tablet Amlodipine 5mg OD Not a known case of DM/TB/BA/COPD/CAD/thyroid disorder/CKD

Underwent hernioplasty and appendicectomy under regional anesthesia – 5years ago, uneventful Patient is Moderately built and nourished, weight 56kg, height 164 cm, BMI 20.8 kg/m²

Icterus present, no cyanosis/ clubbing /pallor/ generalised lymphadenopathy / pedal edema Lab findings: Total bilirubin – 7.8 mg/dl

SUMMARY :

67-year-old male, known hypertensive on treatment, presented with painless progressive jaundice associated with loss of weight and appetite-probably of obstructive jaundice-came for further evaluation and management.”



13th JUNE 2026

11:45PM - 12:15PM

Anaesthetic Management of a Patient with Cardiac Pacemaker posted for ORIF of Shaft of Humerus Fracture

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Introduction:

The operating theatre has always been a place of controlled complexity, but the rising prevalence of patients carrying Cardiac Implantable Electronic Devices (CIEDs) — permanent pacemakers (PPMs), implantable cardioverter-defibrillators (ICDs), and cardiac resynchronisation therapy (CRT) devices — has added a new dimension to perioperative risk. The anaesthesiologist today must be conversant not merely with the physiology of the diseased heart, but with the physics of electromagnetic fields and the engineering of sophisticated battery-powered miniature computers sutured beneath a patient's clavicle.

In the United Kingdom, 58% of permanent pacemakers implanted in 2010 were inserted for symptomatic atrioventricular (AV) block. Sick sinus syndrome accounted for a further 23.6%. Globally, implantation rates continue to climb, driven by an ageing population and expanding indications for ICD therapy. Every anaesthesiologist, from trainee to consultant, will encounter at least one patient per week carrying one of these devices, often presenting for surgery entirely unrelated to their cardiac condition — a hip replacement, a bowel resection, an ophthalmic procedure.

2. Understanding the Device: Anatomy, Physiology & Nomenclature

2.1 What Is a Cardiac Implantable Electronic Device?

A permanently implanted cardiac pacemaker is a CIED composed of a pulse generator, one or more sensing and pacing electrodes (usually positioned in the right

atrium and right ventricle), and a lithium-iodide battery. Electrical impulses originating in the pulse generator are transmitted through specialised leads to excite endocardial cells and produce a propagating wave of depolarisation. The lithium battery may last up to 10 years, but depletion requires surgical replacement of the entire pulse generator — a fact that informs device surveillance intervals.

ICDs share the same basic architecture but add high-voltage coils within the ventricular lead, capable of delivering up to 35 joules of internal defibrillatory shock within 15 seconds of detecting a shockable rhythm. Modern ICDs are therefore truly multi-functional devices: they can pace for bradycardia, deliver anti-tachycardia pacing bursts, perform synchronised cardioversion, and internally defibrillate. CRT devices (biventricular pacemakers) add a third lead into a lateral branch of the coronary sinus to re-synchronise left and right ventricular contraction in patients with dilated cardiomyopathy and widened QRS, improving ejection fraction.

2.2 The NBG Pacemaker Code

The North American Society of Pacing and Electrophysiology / British Pacing and Electrophysiology Group (NASPE/BPEG) Generic Code — universally referred to as the NBG code — provides a systematic language for describing pacemaker function. Although first proposed in 1974 as a three-letter code, the current standard uses five positions. Most clinicians continue to use the shorthand three-letter version in everyday practice.



Position	Parameter Described	Options	Clinical Significance
I	Chamber(s) paced	O, A, V, D (Dual)	O = no pacing; D = atrium + ventricle
II	Chamber(s) sensed	O, A, V, D	O = paces at fixed rate, ignores intrinsic rhythm
III	Response to sensing	O, I, T, D	I = inhibited; T = triggered; D = dual (inhibit + trigger)
IV	Rate modulation	O, R	R = rate-responsive sensor active; O = disabled
V	Multisite pacing	O, A, V, D	Most commonly biventricular (CRT)

The most common clinical configuration is VVI - ventricular pacing, ventricular sensing, inhibited response. This mode is encountered typically with a transvenous catheter in the right ventricle. Anaesthesiologists must recognise the pacing spikes on ECG that precede each QRS in a paced patient, and appreciate that artefact from electrocautery may masquerade as QRS complexes, causing the monitor to display a falsely normal heart rate whilst the patient is in fact in asystole.

2.3 Indications for Permanent Pacing

Indication Category	Specific Conditions	Notes
AV Block (Class I)	Third-degree, persistent second-degree AV block; post-MI infranodal block + LBBB	Most common indication globally
Sinus Node Dysfunction	Symptomatic sick sinus syndrome; chronotropic incompetence; drug-induced bradycardia	Second most frequent PPM indication (23.6% in UK 2010)
Post-Myocardial Infarction	Persistent complete heart block; persistent Type II second-degree AV block	Ventricular escape rhythms in MI setting are unstable
Bifascicular Trifascicular Block	Intermittent complete block with symptoms; Type II second-degree AV block; alternating BBB	Risk of progression to complete block mandates PPM
Carotid Sinus Neurocardiogenic	Recurrent syncope with asystole >3 s on carotid sinus stimulation	Hypertensive carotid sinus syndrome
ICD Indications	Cardiac arrest from VT VF; ischaemic cardiomyopathy EF <30%; Brugada syndrome; HOCM	Expanding indications; primary prevention increasingly common

3. Electromagnetic Interference: The Principal Perioperative Hazard

3.1 Mechanisms and Sources

Electromagnetic interference (EMI) is the potential disruption of an electronic device when it enters the vicinity of an electromagnetic field generated by an external source. In surgical practice, the most significant source of EMI is the electrosurgical unit (ESU), otherwise known as diathermy or electrocautery. The ESU uses radiofrequency current — typically between 300 kHz and 3 MHz — to cut or coagulate tissue. This radiofrequency energy can be sensed by the CIED's leads and misinterpreted as intrinsic cardiac activity.

Radio frequency waves with frequencies between 0 and 10^9 Hz — including AC power supplies and electrocautery — and microwaves between 10^9 and 10^{11} Hz are the principal culprits for device interference. Higher-frequency waves such as X-rays, gamma rays, and infrared light do not cause device interference and are therefore safe from an EMI perspective.

The consequences of EMI for CIEDs depend on the type of device and the programming mode. The three key effects on pacemakers are: (1) inhibition of pacing — the device interprets EMI as intrinsic ventricular activity and withholds its pacing output, potentially causing bradycardia or asystole in a pacing-dependent patient; (2) asynchronous pacing — the pacemaker reverts to a fixed-rate mode, risking R-on-T phenomenon; and (3) reset to back-up mode — the device reverts to factory default settings. For ICDs, the additional risk is the delivery of inappropriate shocks triggered by EMI that is misclassified as ventricular fibrillation.

3.2 Unipolar vs Bipolar Lead Configurations

The susceptibility of a CIED to EMI is profoundly influenced by its lead configuration. In a unipolar system, one electrode at the lead tip serves as the cathode while the pulse generator casing serves as the anode — creating a large electrical circuit traversing most of the thorax. Extraneous electromagnetic signals easily intrude into this large circuit. In a bipolar configuration, two closely spaced electrodes near the distal tip form the circuit; the small inter-electrode distance markedly reduces the antenna effect, conferring substantially greater resistance to EMI. Modern CIEDs are predominantly programmed to bipolar sensing, but older devices and some specific programming scenarios may retain unipolar sensing — a detail the preoperative assessment must establish.

Clinical Anecdote 1: The Silenced Pacemaker

A 74-year-old man with complete heart block and a VVI pacemaker was admitted for open cholecystectomy. During dissection, the surgeon began using monopolar diathermy close to the epigastrium. The ECG monitor continued to display a rate of 72 bpm — but the pulse oximeter waveform had flatlined. The anaesthesiologist, alert to the fact that electrocautery artefact can mimic QRS complexes, immediately palpated the radial pulse and found none. Diathermy was halted; the pacemaker, which had been inhibited by the ESU signal mistaken as intrinsic R waves, resumed pacing within seconds. This near-miss highlighted two lessons: always monitor mechanical cardiac output (pulse oximetry waveform or arterial line), and always establish pacing dependency before surgery.



3.3 Mind Map: Sources & Consequences of EMI in the Theatre

Electromagnetic Interference & CIEDs	
Surgical Sources	- Monopolar electrocautery (most common) - Bipolar diathermy (safer, rarely problematic) - Radiofrequency ablation (high prolonged exposure risk) - Nerve stimulators / peripheral stimulators
Non-surgical Sources	- MRI (magnetic field heating of leads) - Radiation therapy (ionising damage to circuitry) - ESWL shock waves (older piezoelectric sensors) - ECT electrical stimulus + subsequent seizure
Effects on Pacemaker	- Pacing inhibition ? bradycardia / asystole - Asynchronous pacing ? R-on-T risk - Reset to factory / back-up mode 'Runaway' pacing (rare; 180–200 bpm)
Effects on ICD	- Inappropriate shock delivery - Inhibition of tachyarrhythmia therapy - Antitachycardia pacing triggered falsely - Permanent device damage (rare)
High-Risk Patients	- Pacing-dependent (complete AV block) - ICD carriers undergoing surgery above umbilicus - Rate-responsive pacemaker with respiratory sensor - Recently implanted (<3 months) — lead dislodgment risk

4. Preoperative Assessment: The Cornerstone of Safe Management

4.1 Information Gathering

A structured preoperative assessment of any patient with a CIED is mandatory before elective surgery. The patient's device identification card — the European Pacemaker Patient Identification Card in the UK — contains the device model, serial number, implanting centre, leads, aetiology, current programmed mode, sensing thresholds, and battery status. This card is the starting point for preoperative planning. In the absence of the card, chest radiography can reliably differentiate a PPM from an ICD (the ICD's ventricular lead shows two thick radiopaque sections representing high-voltage coils), determine lead position, and identify fractured leads.

4.2 Device Interrogation

The Heart Rhythm Society recommends formal device interrogation: within 6 months for an ICD, within 12 months for a conventional PPM, and within 3–6 months for any CRT device, before elective surgery. This interrogation, performed by trained technicians or device nurses, provides the current battery voltage, lead impedances, sensing thresholds, pacing thresholds, and — crucially — the percentage pacing burden.

Patients who have a pacing burden of $\geq 40\%$ are deemed pacing-dependent. An ECG showing predominantly paced morphology, with a pacing spike before every QRS, is a strong bedside indicator of pacing dependence. A history of atrioventricular nodal ablation, prior syncope due to bradyarrhythmia, or absence of spontaneous ventricular activity at VVI 40 bpm on interrogation all confirm true pacing dependence — a status that significantly raises perioperative risk from EMI-induced pacing inhibition.

4.3 The Four Key Preoperative Questions

Question	Why It Matters	Action If Yes
Is the patient pacing-dependent?	EMI-induced pacing inhibition may cause asystole	Programme to asynchronous mode (AOO VOO/DOO) or apply magnet
Is there an ICD?	ICD may deliver inappropriate shocks if EMI mimics VF	Suspend antitachyarrhythmia therapy preoperatively
Is the surgery above the umbilicus?	ESU above umbilicus greatly increases EMI exposure to generator in chest	Bipolar diathermy preferred; reprogram or magnet mandatory
Is rate-responsive pacing active?	Respiratory-triggered rate sensors may accelerate HR from ventilator movements	Disable rate-adaptive function preoperatively

4.4 Magnet vs Reprogramming: A Critical Distinction

For pacemakers, application of a medical-grade magnet over the pulse generator activates a reed switch within the device, triggering asynchronous pacing at a manufacturer-specific 'magnet rate' (typically 85 bpm for Medtronic, 96.4 bpm for Boston Scientific), regardless of intrinsic cardiac activity. This protects the pacing-dependent patient from EMI-induced pacing inhibition. The magnet's effect is immediately reversible upon removal — its main operational advantage.

However, the magnet effect is variable and must be verified preoperatively by the CIED care team. Certain devices (Boston Scientific, Abbott/St Jude Medical, Biotronik) can have their magnet response disabled via programming; in such cases, magnet application would have no effect — a potentially catastrophic misapprehension during surgery. Furthermore, some leadless pacemakers do not initiate asynchronous pacing in response to magnet application. Magnet placement does not disable rate-adaptive functions. For these reasons, formal reprogramming by a pacing technician is preferred whenever feasible.



For ICDs, the magnet response is different: magnets suspend tachyarrhythmia detection and therapy (preventing inappropriate shocks) but do NOT alter the pacing mode. An ICD patient who is also pacing-dependent may therefore still experience pacing inhibition from EMI even with a magnet in place—requiring formal asynchronous reprogramming, not magnet alone. The ASA Task Force (2011) explicitly cautions against routine magnet use as a substitute for proper preoperative CIED consultation.

Clinical Anecdote 2: The Magnet That Did Nothing

A 67-year-old woman with an ICD for non-ischaemic dilated cardiomyopathy was scheduled for laparoscopic cholecystectomy. The anaesthesiologist, aware that carbon dioxide insufflation creates impedance changes that can trigger ICD sensing, applied a magnet over the device preoperatively, assuming it would place the device in asynchronous pacing mode. Intraoperatively, monopolar diathermy triggered an inappropriate 35-joule shock — the patient, though sedated, lurched violently, and arterial line waveforms confirmed sinus tachycardia, not VF. Post-operatively, interrogation revealed the ICD had its magnet response disabled during a prior clinic visit to prevent accidental deactivation by household magnets. The lesson: always confirm actual magnet response with the device care team; never assume.

5. Intraoperative Management

5.1 Monitoring

Intraoperative monitoring must include both continuous ECG and peripheral pulse monitoring. The critical point—one that has saved lives—is that ECG monitoring alone is insufficient in patients with CIEDs. Electrocautery artefact may be misinterpreted as QRS complexes by the monitor's rate algorithm, displaying a falsely reassuring heart rate on the screen while the patient is in pacing-induced asystole. The 'artifact filter' on the ECG monitor should be disabled to detect pacing spikes, and the ECG monitor should be kept in diagnostic mode (frequency response 0.05–100 Hz) rather than monitoring mode, to enable accurate QRS morphology assessment.

The pulse oximeter plethysmographic waveform and, where indicated, an invasive arterial line waveform, display mechanical cardiac activity in real time and are therefore the gold-standard monitors for true cardiac

output during surgery. Capillary refill, precordial or oesophageal stethoscope, and urine output are additional perfusion parameters that must not be neglected in patients with CIEDs and underlying cardiac dysfunction.

5.2 Electrocautery Precautions

Precaution	Rationale	Evidence Level
Prefer bipolar over monopolar ESU	Current confined between instrument poles; minimal EMI to CIED	Strong consensus; guideline-recommended
If monopolar, use 1–2 s bursts with 10 s pauses	Short intermittent bursts reduce cumulative EMI exposure	Expert consensus
Use cutting current rather than coagulation	Lower peak radiofrequency energy than coagulation mode	Expert opinion
Position return electrode so current path avoids CIED and leads	Minimises radiofrequency current traversing the device circuit	Strong consensus
Consider harmonic (ultrasonic) scalpel	Operates at ultrasonic frequency; does not generate radiofrequency EMI	Recommended alternative when feasible
Keep diathermy cables away from implant site	Reduces proximity-related EMI coupling into leads	Manufacturer and guideline recommendation

5.3 Anaesthetic Agent Considerations

Anaesthetic agents per se do not directly affect CIED function — the devices are sealed and electronically isolated. However, their haemodynamic and neurohumoral effects have indirect implications. In patients who are already bradycardic or pacing-dependent, high doses of fentanyl or dexmedetomidine — which depress sinus node automaticity and AV conduction — may increase pacing burden and PPM dependence, potentially exposing the patient to greater risk from any subsequent pacing failure.

In patients with Long QT syndrome (a common reason for ICD implantation), drugs that further prolong the QT interval pose a risk of precipitating polymorphic ventricular tachycardia (torsade de pointes). Agents to avoid or use cautiously include methadone, haloperidol, ondansetron, and high doses of volatile anaesthetic agents. Propofol is the induction agent of choice in such patients. Etomidate and ketamine are



best avoided as these can cause myoclonic movements; succinylcholine and direct muscle stimulation may also inhibit or trigger pacemaker firing through myopotential generation.

Skeletal myopotentials - generated by fasciculations from succinylcholine, myoclonic movements, or direct muscle stimulation - can be sensed by a CIED in unipolar pacing mode as intrinsic cardiac activity, leading to pacing inhibition. Non-depolarising muscle relaxants are safe. Perioperative hypothermia must also be avoided, as shivering-generated myopotentials may inhibit pacemaker output.

5.4 Haemodynamic Management

A subtlety that catches junior anaesthesiologists off guard: if a patient is pacemaker-dependent and programmed to a fixed rate (asynchronous mode for the operation), tachycardia will NOT occur in response to hypovolaemia. The physiological compensatory mechanism of increasing heart rate is abolished. The anaesthesiologist must therefore maintain meticulous attention to fluid balance, use more liberal filling targets, and vigilantly monitor urine output and direct perfusion markers. Hypotension in such patients demands fluid resuscitation and vasopressors rather than the expectation of compensatory tachycardia.

5.5 Central Venous Catheter Insertion

During insertion of a central venous line in a patient with a CIED, the guide wire passage through the right atrium and right ventricle can dislodge pacing leads, trigger arrhythmias, and irritate the myocardium. The anti-tachyarrhythmia function of any ICD must be suspended before CVL insertion, and asynchronous pacing must be initiated. The chance of lead dislodgment is maximal within the first three months of CIED implantation. A pre-procedure chest X-ray documents lead position; a post-procedure film identifies dislodgment. Pulmonary artery catheter insertion carries similar risks and is best avoided entirely in patients with CIEDs.

Clinical Anecdote 3: The Knee Replacement That Wasn't Straightforward

A 65-year-old man with a VVI pacemaker inserted four years prior for a myocardial infarction-associated complete heart block presented for total knee replacement (TKR). The patient, now asymptomatic but restricted by his arthritis, had no pacemaker card. Preoperative assessment: ECG showed pacing spikes preceding every QRS — pacing-dependent. Device interrogation (Medtronic system) confirmed 100% pacing burden at VVI 70, battery at beginning of life. Decision: spinal anaesthesia was planned (T12 level for TKR), bipolar diathermy requested from the surgical team, and the pacemaker was reprogrammed to VOO 80 (asynchronous) by the technician. Intraoperatively, a pulse oximeter waveform confirmed mechanical cardiac output throughout. The procedure was uneventful. Postoperative device interrogation confirmed no threshold changes. Key learning: even sub-umbilical surgery can generate EMI if monopolar diathermy is used, and the anaesthesiologist must negotiate the choice of diathermy type with the surgeon proactively.

6. Procedure - Specific Considerations

6.1 Mind Map: Perioperative CIED Management Decision Framework

Perioperative CIED Management	
Preoperative	- Obtain device ID card / interrogation report - Establish pacing dependence (pacing burden =40%) - Confirm magnet response with care team - Plan: reprogram vs magnet application - Suspend ICD antitachyarrhythmia if EMI likely - Disable rate-adaptive function if present
Intraoperative	- ECG in diagnostic mode; artifact filter OFF - Pulse oximetry waveform as primary monitor - Bipolar or harmonic scalpel preferred - Cautery bursts =2 s with 10 s pauses - Return electrode positioned away from device - Transcutaneous pacing / defibrillator pads applied - Avoid succinylcholine, etomidate, ketamine
Emergency Scenarios	- VT/VF: stop all EMI, remove magnet, allow ICD to fire - If ICD fails: external defibrillation (pads =15 cm from device) - Anteroposterior pad position; lowest clinically appropriate energy - Asystole in PPM patient: transcutaneous pacing - Runaway pacing (180–200 bpm): magnet removal, interrogate
Postoperative	- Restore original device settings immediately - Interrogate CIED before patient leaves monitored area - Continue ECG + pulse oximetry until reprogramming confirmed - Document intraoperative events for CIED records



6.2 Specific Procedures and Their Nuances

Radiofrequency Ablation

Radiofrequency ablation involves continuous application of radiofrequency energy for several minutes - a prolonged EMI exposure that far exceeds the brief bursts of surgical diathermy. This technique is especially hazardous for ICD patients; defibrillation function must be disabled. Direct contact between the ablation catheter and any CIED component must be avoided, and the radiofrequency current path should be as far from the CIED and leads as technically feasible.

Electroconvulsive Therapy (ECT)

The brief (1–2 second) electrical stimulus of ECT may cause pacemaker inhibition, though this is usually clinically insignificant due to its brevity. The subsequent tonic-clonic seizure, however, generates sustained myopotentials that may cause prolonged pacemaker oversensing and pacing inhibition. For ICD patients, the reactive sinus tachycardia following a seizure may encroach on the programmed ICD detection rate, triggering an inappropriate shock. Reprogramming to asynchronous mode before ECT is advisable, and ICD antitachycardia therapy should be suspended. The isolated environment of many ECT suites adds logistical complexity to planning.

MRI

The intense magnetic field of MRI creates multiple hazards for CIED patients: force and torque on the ferromagnetic components of the generator, magnetic field-induced lead heating with myocardial thermal injury, induced voltages causing pacing inhibition or triggering, and reed switch activation causing asynchronous pacing. No CIED carries an 'MR-safe' designation. The designation 'MR-conditional'-applicable to specific modern devices under specified field strengths and scan conditions-has expanded access to MRI for CIED patients, but requires formal institutional protocols and device-specific manufacturer guidance. All other legacy devices are 'MR non-conditional' and traditionally contraindicated.

Radiation Therapy

High-energy ionising radiation used in cancer treatment can damage CIED electronics even at small cumulative doses. The possible effects include

temporary oversensing, reset to factory settings, and complete device failure. If the radiation field overlaps with the CIED's location, device relocation may be required. Patients should be assessed for pacing dependence; if pacing dependent, weekly CIED evaluation during radiotherapy is recommended.

Extracorporeal Shock Wave Lithotripsy (ESWL)

Modern ESWL devices do not incorporate the piezoelectric crystals that previously posed a risk to CIED rate sensors. However, for patients with any significant pacing dependence, ESWL shock waves can potentially inhibit ventricular pacing. Reprogramming to VOO or DOO (asynchronous mode) before ESWL is recommended.

TENS and Nerve Stimulators

Transcutaneous electrical nerve stimulation (TENS) is considered contraindicated in patients with CIEDs because transcutaneous impulses may cause inappropriate sensing and pacing inhibition or, in ICD patients, inappropriate shock delivery. Peripheral nerve stimulators used for neuromuscular blockade monitoring have been reported to inhibit pacemakers when electrode placement creates a current pathway across the device. The current path for any electrical stimulator must be arranged so that it does not traverse the CIED or its leads.

7. Emergency Surgery: When Time is Not a Luxury

Emergency surgery with a CIED patient presents the anaesthesiologist with a compressed decision window. The ideal preoperative CIED assessment and reprogramming by a pacing technician may be impossible. In this scenario, pragmatism governs:

- Apply a magnet over a pacemaker in a pacing-dependent patient to initiate asynchronous pacing. Record an ECG before and after magnet application to confirm the desired response.
- Apply a magnet over an ICD to disable antitachyarrhythmia therapy. Remember: this does NOT convert ICD to asynchronous pacing mode. A pacing-dependent ICD patient still requires bipolar diathermy and careful EMI minimisation.
- Use bipolar diathermy as the surgical default. If monopolar is unavoidable, use 1–2 s bursts, lowest feasible energy, and position the return electrode to



minimise current through the CIED.

- Transcutaneous pacing/defibrillator pads must be applied and connected to an operational external defibrillator with pacing capability before skin incision. Pads in anteroposterior position, no closer than 15 cm to the CIED.

- Continuous ECG monitoring plus pulse oximetry waveform or arterial line is mandatory throughout.

Total CIED failure during surgery is rare but catastrophic. It may manifest as absent pacing in a pacing-dependent patient, inappropriate repeated shocks, or 'runaway' pacing at 180–200 bpm (an older device failure mode rendered rare by modern design). Should runaway pacing occur, magnet removal, cessation of any EMI source, and urgent device interrogation are required. If inappropriate shocks recur, the magnet must remain in place to keep ICD therapy suspended whilst surgery is completed, and the device interrogated immediately postoperatively.

8. Postoperative Management & Special Scenarios

8.1 Restoration of Device Settings

If the CIED was reprogrammed preoperatively, the original settings must be restored in the immediate postoperative period-before the patient leaves the monitored recovery area. The device should be re-interrogated to confirm proper function, establish that pacing and sensing thresholds remain within acceptable limits, and check for any intraoperative events stored in the device memory. Until reprogramming is confirmed, the patient must remain on continuous ECG and pulse oximetry monitoring with transcutaneous pacing equipment immediately available.

8.2 DC Cardioversion and Defibrillation

If external cardioversion or defibrillation is required in a patient with a CIED, specific precautions apply: use the lowest clinically appropriate energy; position defibrillation pads no closer than 15 cm to the CIED; prefer the anteroposterior pad position; avoid placing pads directly over the device. After defibrillation, the CIED must be interrogated as soon as possible. The current from external defibrillation may cause changes in pacing threshold, sensing threshold, or even cause device reset, each of which demands identification and management.

8.3 The Future: Leadless Pacemakers and Subcutaneous ICDs

The landscape of CIEDs continues to evolve. Leadless pacemakers - entirely intracardiac devices deployed via femoral vein catheter, without a subcutaneous generator or transvenous leads-are increasingly implanted for single-chamber ventricular pacing. These devices eliminate lead-related complications but behave differently in response to magnets: some leadless PPMs do not initiate asynchronous pacing upon magnet application, making preoperative interrogation and clear communication with the CIED team even more critical. Subcutaneous ICDs (S-ICDs), which have the generator implanted lateral to the left chest wall with a subcutaneous (not transvenous) lead, use wider electrode spacing and are more susceptible to EMI than transvenous bipolar ICDs. The anaesthesiologist must be aware of these newer device architectures.

9. Summary: At-a-Glance Reference

Phase	Key Actions
Pre-op Assessment	Obtain device ID card; recent interrogation report (PPM =12 months, ICD =6 months, CRT =6 months); establish pacing dependence; confirm magnet response; plan reprogramming vs magnet; disable ICD antitachy therapy if EMI likely; disable rate-adaptive function
Pre-op Planning	Communicate with surgeon: bipolar diathermy preferred; advise on return electrode positioning; arrange pacing technician presence or on-call; plan anaesthesia technique based on haemodynamic impact (spinal caution in pacing-dependent patients)
Intraoperative Monitoring	ECG in diagnostic mode (artifact filter OFF); pulse oximetry waveform for mechanical output; arterial line if haemodynamically complex; apply transcutaneous pacing/defibrillator pads anteroposteriorly, =15 cm from device
Drug Choices	Avoid succinylcholine, etomidate, ketamine (myoclonus); avoid QT-prolonging agents in long QT syndrome; use non-depolarising relaxants; limit fentanyl/dexmedetomidine in bradycardic patients
Diathermy Protocol	Bipolar preferred; monopolar in 1–2 s bursts, 10 s pauses, cutting current, lowest energy; return electrode positioned distally; keep cables away from device site; consider harmonic scalpel
Haemodynamic Management	Fixed-rate pacing = no tachycardia response to hypovolaemia; maintain euvoaemia; monitor perfusion directly; vasopressors for hypotension
Intraoperative Emergency	VT:VF: stop EMI, remove magnet (restores ICD); if ICD fails — external defibrillate (=15 cm from device, AP pads, lowest energy); interrogate CIED post-emergency
Post-op	Restore original settings immediately; interrogate CIED; continuous monitoring until settings confirmed; document intraoperative events



10. Recent Advances in CIED Technology and Perioperative Management

10.1 Leadless Pacemakers

The Micra Transcatheter Pacing System (Medtronic) and the AVEIR device (Abbott) represent the current generation of leadless intracardiac pacemakers, implanted via the femoral vein and anchored directly within the right ventricular apex or septum. These self-contained capsule devices eliminate all conventional leads and the subcutaneous pulse generator pocket, thereby removing the risk of lead fracture, lead dislodgement, and pocket infection that collectively account for a significant proportion of conventional CIED complications. The LEADLESS II trial demonstrated a major complication rate of 2.7% at six months, substantially lower than historical data for transvenous single-chamber devices. The newer Micra AV system provides atrioventricular synchrony without an atrial lead, using an accelerometer to detect atrial mechanical activity through passive haemodynamic sensing.

The perioperative implications are significant and distinct. Leadless devices do not respond predictably to magnet application — many models do not initiate asynchronous pacing in response to the reed switch, making the standard “magnet for pacing protection” strategy unreliable. Device interrogation by a trained technician with the specific programming wand is therefore non-negotiable before any surgical procedure. Furthermore, the lack of a subcutaneous generator means the return electrode positioning rules change — the electrosurgical current path must be planned recognising that the device is entirely intracardiac.

10.2 Subcutaneous ICDs and Extravascular Defibrillators

The subcutaneous ICD (S-ICD, Boston Scientific) employs a generator implanted in the lateral chest wall at the left mid-axillary line with a subcutaneous (not transvenous) sensing and shock lead. It delivers 80-joule defibrillation shocks and does not contact the heart or venous system, virtually eliminating lead-related intravascular complications and infection risk. However, it cannot provide antibradycardia pacing (other than brief post-shock pacing) or anti-tachycardia pacing (ATP), and its wide-field sensing is considerably

more susceptible to T-wave oversensing and myopotential artefact. For the anaesthesiologist, the S-ICD generator lies laterally in the thorax, altering the optimal defibrillation pad placement strategy: standard anteroposterior positioning must avoid direct pad placement over the lateral generator.

The extravascular ICD (EV-ICD, Medtronic Empower/Aurora system) is the newest iteration—the lead is tunnelled substernally, providing a cardiac-adjacent defibrillation vector without entering the vasculature, while retaining the capacity for antibradycardia pacing. Regulatory approval in the United States was granted in 2022, and familiarity among anaesthesiologists in India remains very limited. The perioperative team must appreciate that device identification from a chest X-ray alone may be non-trivial with these novel architectures.

10.3 Remote Monitoring and Wireless Interrogation

All major CIED manufacturers now offer wireless remote monitoring platforms — Medtronic CareLink, Abbott Merlin.net, Boston Scientific LATITUDE, and Biotronik Home Monitoring — that transmit stored electrograms, battery status, lead parameters, and detected arrhythmia episodes nightly to a secure server accessible by the implanting centre. This represents a paradigm shift: a patient presenting for surgery at a district hospital can, in principle, have their CIED data reviewed by an electrophysiologist at the implanting tertiary centre in real time, without physical device interrogation. The 2024 AHA Scientific Statement (Wan EY et al.) specifically recommends leveraging remote monitoring data for preoperative risk stratification when in-person interrogation is logistically unavailable. In the Indian context, this holds transformative promise for centres distant from metropolitan electrophysiology services, though it requires stable internet infrastructure and institutional subscription to the monitoring platform.

10.4 MR-Conditional Devices and Evolving MRI Access Protocols

The proportion of newly implanted devices with MR-conditional designation now exceeds 75% globally. The MagnaSafe Registry (Russo RJ et al., NEJM 2017) demonstrated that non-conditional legacy devices



could also undergo clinically indicated MRI at 1.5 Tesla with acceptable safety under a formal institutional protocol, effectively challenging the traditional absolute contraindication. The 2017 HRS Expert Consensus Statement (Indik JH et al.) provides a structured framework for safe MRI in CIED patients, requiring: device reprogramming to asynchronous pacing (for pacing-dependent patients) with ICD therapy suspended, physiological monitoring throughout the scan, and post-MRI interrogation. For the anaesthesiologist called to provide monitored anaesthesia care or sedation for MRI in a CIED patient, awareness of these protocols is essential. The anaesthesia machine and monitoring equipment must themselves be MRI-compatible, and resuscitation capability must be immediately available outside the scanner bore.

10.5 Conduction System Pacing: His Bundle and Left Bundle Branch Area Pacing

Physiological pacing via His bundle pacing (HBP) and left bundle branch area pacing (LBBAP) represents a significant conceptual advance over conventional right ventricular apical pacing. By stimulating the native conduction system directly or proximally, these techniques produce a narrow, coordinated QRS complex and preserve interventricular synchrony, avoiding the pacing-induced cardiomyopathy that can develop with long-term right ventricular apical pacing. LBBAP, pioneered substantially in China (Huang et al., 2017) and now gaining rapid global adoption, involves deep septal lead implantation to capture the left bundle branch directly. The 2021 ESC Pacing Guidelines now list HBP as a recommended alternative (Class IIb) to conventional right ventricular pacing. Anaesthesiologists should be aware that these leads are deeply embedded in the interventricular septum, carrying a specific risk profile for septal perforation if manipulated, and that the ECG morphology in paced beats differs from conventional RV pacing — the QRS is narrow rather than LBBB-pattern, which may alter the interpretation of intraoperative ECG changes.

11. Challenges and Limitations in Perioperative CIED Management

11.1 Absence of Standardised Preoperative Pathways and the Evidence Gap

Despite the publication of multiple society guidelines, a

critical limitation acknowledged even in the most authoritative documents is the low quality of the underlying evidence. The ASA Practice Advisory (2011) and the HRS/ASA Expert Consensus Statement (2011) are largely built on expert opinion, case series, and bench-top electromagnetic testing rather than prospective randomised controlled trials. Many recommendations — such as the precise “10-second burst” interval for monopolar diathermy, or the “15 cm from device” rule for defibrillator pad placement — originate from manufacturer recommendations and expert consensus rather than formal clinical outcome data. Prospective perioperative CIED registries remain scarce globally, and the few available observational datasets are dominated by Western cohorts that may not reflect the device profiles, surgical case mix, or resource environments typical of Indian practice.

11.2 Limitations of the Magnet Strategy

The magnet remains the most accessible emergency tool for CIED management in theatres lacking pacing technician support, yet its limitations are profound. Magnet response is manufacturer- and model-specific and may be programmed “off” without the anaesthesiologist’s knowledge. Leadless pacemakers do not reliably respond to magnets. Magnets do not disable rate-adaptive functions in most devices, meaning a respiratory-triggered accelerometer may still drive inappropriate tachycardia during positive-pressure ventilation. For ICD patients, the magnet suspends tachyarrhythmia detection but does not convert the pacing mode to asynchronous — a distinction that is life-critical for the pacing-dependent ICD patient. The anaesthesiologist who treats magnet application as a universal solution for all CIED scenarios is placing the patient at risk. The 2024 AHA Scientific Statement explicitly states that a magnet strategy, where used, must be pre-confirmed with the CIED care team.

11.3 Interdisciplinary Communication Gaps and Human Factors

Multiple studies of adverse perioperative CIED events have identified failure of interdisciplinary communication as a root cause. The surgeon's choice of monopolar vs bipolar diathermy, the cardiologist's decision whether to reprogram the device, and the anaesthesiologist's monitoring strategy are each



individually incomplete — they are only safe when integrated. Structured preoperative checklists specifically designed for CIED patients, analogous to the WHO Surgical Safety Checklist, have been proposed but are not yet universally adopted. Survey studies of anaesthesiologists in the United Kingdom and the United States have consistently found knowledge gaps in device identification, correct magnet response, and the distinction between pacemaker and ICD management - deficiencies that are amplified in emergency surgical settings where preparation time is compressed.

11.4 The Rapidly Evolving Device Landscape and Guideline Lag

The pace of CIED innovation - leadless pacemakers, S-ICDs, EV-ICDs, conduction system pacing, closed-loop stimulation algorithms, biosensor-integrated devices - consistently outpaces the publication cycle of clinical guidelines. The most recent comprehensive guidance (2024 AHA Scientific Statement) already acknowledges that several of its recommendations predate widespread clinical adoption of leadless and EV-ICD platforms. For the practising anaesthesiologist, this creates an obligation for continuous self-education beyond formal guideline documents. Device manufacturer websites, product-specific perioperative guides (available from Medtronic, Abbott, Boston Scientific, and Biotronik), and institutional CIED teams are essential resources that must supplement guideline knowledge.

12. The Indian Perspective: CIED Anaesthesia in the Indian Healthcare Ecosystem

12.1 Epidemiology of CIEDs in India: A Growing but Undercharted Burden

India implants approximately 70,000–80,000 permanent pacemakers annually, representing one of the fastest-growing CIED markets in Asia. However, the implantation rate of approximately 6 devices per million population per year remains far below the European average of 800–1,000 per million, reflecting a substantial unmet burden of untreated symptomatic bradyarrhythmias across the country. The Indian Heart Rhythm Society (IHRS) estimates that fewer than 10% of patients who meet current guideline criteria for pacing actually receive a device, primarily because of cost, limited access to electrophysiology services

outside metropolitan centres, and low physician and patient awareness of indications.

The epidemiological profile of Indian CIED recipients also differs from Western cohorts. Rheumatic heart disease with associated complete heart block remains a significant indication in younger patients from lower socioeconomic backgrounds, unlike the predominantly degenerative conduction disease and cardiomyopathy-related ICD indications seen in high-income countries. Infective endocarditis involving CIED leads - a catastrophic complication - carries a higher risk in populations with endemic dental sepsis, untreated genitourinary infections, and limited antibiotic stewardship. These epidemiological nuances mean that the Indian anaesthesiologist frequently encounters a different “CIED patient phenotype” from that described in Western literature.

12.2 Infrastructure, Resource, and Access Challenges

The foremost infrastructure challenge is the scarcity of device-specific interrogation technology outside tertiary cardiac centres. A pacing technician with the appropriate programmer wand - essential for preoperative CIED interrogation and reprogramming - is unavailable in most district hospitals, taluk-level government facilities, and smaller private institutions. In practice, many CIED patients presenting for non-cardiac surgery in tier-2 and tier-3 cities arrive without their device identification card, without recent interrogation records, and without any facility for intraoperative reprogramming. The anaesthesiologist is frequently the only qualified clinician in the theatre who is even aware that the patient has a CIED.

Cost constraints impose additional limitations. The bipolar electrosurgical unit, the harmonic (ultrasonic) scalpel, and the arterial line are guideline-recommended but may not be available or affordable in resource-limited settings. The transcutaneous external pacing-defibrillator, mandatory for intraoperative standby, is not universally present in Indian operation theatres outside major hospitals. Imported CIEDs carry high costs exacerbated by import duties; domestic manufacture remains limited to a small number of single-chamber devices, constraining access for patients under government insurance schemes (PMJAY, ESI, CGHS).



12.3 The Device Documentation Problem

A recurring clinical scenario in Indian hospitals is the patient presenting with a pacemaker but without any documentation — no device card, no discharge summary from the implanting centre, and no accompanying relative who can provide history. Chest radiography remains the practical first line of assessment in this situation: the pacemaker vs ICD distinction (high-voltage coils in the ventricular lead of an ICD), approximate lead position, and lead integrity can all be partially assessed radiologically. The IHRS and several major cardiac centres have advocated for a nationally standardised CIED patient registry — analogous to the UK's NICOR National Audit for CIEDs — but such a registry does not yet exist at the national level in India. In its absence, smartphone-based CIED identification applications and manufacturer-maintained device identification portals (e.g., the Medtronic Product Performance Report) offer partial solutions.

12.4 Training, Curriculum Gaps, and the Role of Postgraduate Education

The MD Anaesthesia curriculum prescribed by the National Medical Commission (NMC) of India includes cardiac anaesthesia as a competency domain, but specific CIED management is variably taught across institutions. The majority of MD trainees complete their programme without having performed a single preoperative CIED interrogation or witnessed formal device reprogramming. This practical deficit is compounded by an uneven distribution of electrophysiology services: trainees at AIIMS, PGI Chandigarh, JIPMER, and major private medical colleges may have regular exposure, while those at peripheral government medical colleges often do not. National organisations including the Indian Society of Anaesthesiologists (ISA) and the Association of Cardiac Anaesthesiologists of India (ACAI) have increasingly incorporated CIED workshops, simulation sessions, and focused lectures — including through platforms such as AOTTA — into their national conference programmes, recognising this as a priority educational need for Indian postgraduates.

12.5 Practical Recommendations for the Indian Resource-Stratified Context

In the absence of on-site pacing technician services and

formal reprogramming, the following pragmatic framework is recommended for Indian anaesthesiologists operating in resource-limited settings. First, establish pacing dependence clinically from the ECG (every QRS preceded by a pacing spike = likely pacing-dependent) and history (complete heart block, post-AV nodal ablation). Second, contact the implanting cardiologist by telephone to obtain device details and confirm safe magnet response — most major device manufacturers also maintain 24-hour physician helplines in India. Third, confirm actual magnet response on the ECG before incision by applying the magnet and observing for rate change to asynchronous mode. Fourth, default to bipolar diathermy and request the surgeon use brief, intermittent bursts if monopolar is unavoidable. Fifth, ensure a functional external pacing-defibrillator with transcutaneous pacing capability is in the theatre and tested before incision. Sixth, use the pulse oximeter waveform as the primary monitor of mechanical cardiac output throughout the case. Finally, document all intraoperative CIED-related decisions and refer the patient back to their implanting cardiologist for post-operative device interrogation before discharge.

12.6 Indian Contributions to Anaesthesia and CIED Research

Indian anaesthesiologists have made meaningful contributions to the published literature on CIED perioperative management. Chakravarthy M and colleagues from Bangalore have published extensively on anaesthetic management of patients with electronic cardiac devices in the Indian Journal of Anaesthesia. The IHRS national guidelines on cardiac pacing (2019) provide an India-specific framework adapted to local device availability, cost, and infrastructure. Increasingly, Indian centres are contributing case series and audit data that reflect the distinctive challenges of managing CIED patients in a mixed public-private healthcare system. For postgraduate trainees, engaging with this Indian literature-published in the Indian Journal of Anaesthesia, Annals of Cardiac Anaesthesia, Journal of the Indian Medical Association, and Indian Heart Journal-is essential for contextualising international guidelines to local realities.



13. Conclusion

Managing a patient with a cardiac implantable electronic device in the perioperative period demands a synthesis of cardiology, physics, pharmacology, and clinical acumen that few other anaesthetic challenges match. The devices themselves are marvels of engineering - capable of sustaining life beat-by-beat in patients whose intrinsic conduction would otherwise be incompatible with survival. But they are also electronic systems susceptible to the electromagnetic environment of the modern operating theatre.

The safe conduct of anaesthesia in these patients rests on three pillars: thorough preoperative knowledge of the device and its dependence; proactive intraoperative strategies to minimise electromagnetic interference; and vigilant, multimodal monitoring that recognises the limitations of ECG alone. The anecdotes woven through this guide illustrate what happens when any one of these pillars is missing - and, equally, what good outcomes look like when the team works together with shared awareness.

As CIEDs become more sophisticated-leadless pacemakers, subcutaneous ICDs, extravascular defibrillators, remote monitoring systems-the anaesthesiologist's knowledge must keep pace. The fundamental question remains unchanged: in this patient, with this device, for this surgery - what could go wrong, and what have I done to prevent it?

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13th JUNE 2026
12:15PM - 12:55PM

Anaesthetic management of a patient with RHD (severe MS) with Hollow viscous perforation posted for Emergency Laparotomy

MITRAL STENOSIS

CASE SCENARIO :1

A 48-year-old man presents to the emergency department with acute onset severe abdominal pain for 1 day, associated with vomiting and abdominal distension.

He is a known case of rheumatic heart disease with severe mitral stenosis diagnosed 5 years ago.

Patient gives history of progressive breathlessness (NYHA Class III), syncope, orthopnea and occasional palpitations.

The patient is on warfarin and metoprolol, poor compliance.

No history of previous surgery.

On examination:

Patient is tachypnoea, anxious, and in severe abdominal pain

Pulse: 110/min, irregularly irregular

BP: 100/60 mmHg

RR: 28/min

SpO₂: 92% on room air

JVP is raised

Cardiovascular examination:

Loud S1, opening snap, Mid diastolic murmur rumbling in character, best heard in left lateral position

Loud P2

Abdomen:

Board-like rigidity, guarding, absent bowel sounds

Investigations:

ECG: Atrial fibrillation

ECHO: Severe mitral stenosis (MVA = 1 cm²), pulmonary hypertension, EF 55%

X-ray abdomen: Free air under diaphragm

DEFINITION

Mitral stenosis is a pathological narrowing of the mitral valve orifice that obstructs blood flow from the left atrium to the left ventricle during diastole.

It is a progressive, lifelong disease that remains slow and stable in the early years but accelerates later, with a latency of 20–40 years after rheumatic fever before symptom onset.

Once symptoms begin, the disease typically progresses over about 10 years to become disabling.

ETIOLOGY

- **Rheumatic heart disease (mc)** : Follows acute rheumatic fever, which is an inflammatory, autoimmune, multisystem disorder. Occurs as a delayed sequel of Group A β -haemolytic streptococcal pharyngitis. Bacteraemia sensitizes the individual, leading to antibody cross-reactivity between the streptococcal cell wall M protein and myofibrils in the heart. Antibodies attack: Cardiac myofibrils (valves) → fibrosis → mitral stenosis, Joints, Central nervous system, Skin, Connective tissue
- SLE
- Carcinoid syndrome
- Active infective endocarditis
- Left atrial myxoma
- Congenital MS
- Massive annular calcification
- Hunter syndrome
- Hurler's syndrome
- Amyloidosis
- Rheumatoid arthritis
- Lautenbacher's syndrome (ASD + MS)

SYMPTOMS

The patient will present with a history of

- Easy fatiguability, syncope and low effort tolerance- due to decreased cardiac output.



• **Dyspnoea on exertion** - Dyspnea is the subjective experience of breathing discomfort that consists of qualitatively distinct sensations that vary in intensity

In cardiac patients, we use the NYHA (New York Heart Association) classification

Class	Description
Class I	No limitation of physical activity; ordinary activity does not cause dyspnea
Class II	Mild limitation; dyspnea on ordinary activity
Class III	Marked limitation; dyspnea on less than ordinary activity
Class IV	Dyspnea at rest or with minimal activity.

- **Orthopnoea**- In the supine position, there is an increase in venous return, leading to increased blood flow to the left atrium. Due to mitral valve obstruction, the left ventricle is unable to accommodate the increased preload, resulting in a rise in left atrial pressure. This elevated pressure is transmitted backward to the pulmonary veins, causing an increase in pulmonary venous and capillary hydrostatic pressures. When the hydrostatic pressure exceeds the plasma oncotic pressure, transudation of fluid into the alveoli occurs. This leads to decreased lung compliance, increased airway resistance, and ventilation-perfusion mismatch, ultimately resulting in dyspnoea in the lying position, known as orthopnoea.
- **PND**- The mechanism of paroxysmal nocturnal dyspnoea is similar to that of orthopnoea, as pulmonary congestion occurs due to increased venous return in the supine position. During sleep, additional factors contribute to worsening symptoms, including reduced sympathetic activity leading to decreased left ventricular contractility, and a fall in PaO₂. There is also gradual reabsorption of interstitial fluid, which increases intravascular volume. These changes result in a further rise in pulmonary venous pressure and lead to acute pulmonary congestion. Consequently, the patient experiences sudden episodes of dyspnoea typically occurring 2–5 hours after the onset of sleep, and relief is obtained upon

sitting upright.

- **Haemoptysis**- due to dilatation and rupture of pulmonary/bronchial veins. Blood leaks into alveoli causing haemoptysis
- **Palpitations** – due to atrial fibrillation
- **Hoarseness of voice** – ORTNER'S SYNDROME

CLINICAL SIGNS

General Appearance

- **Mitral facies** -malar flush with cyanosis
- **Cold extremities**, peripheral cyanosis (severe MS)

Pulse

- **Low volume pulse**
- **Irregularly irregular pulse** (delirium cordis) → Atrial Fibrillation
- **Pulse deficit** may be present

JVP is raised

- **Prominent a wave** → ↑ RA pressure
- **Prominent v wave** → tricuspid regurgitation

CARDIOVASCULAR SYSTEM EXAMINATION

Palpatory findings of MS

- **Tapping apex beat** due to palpable loud S1
- **Parasternal heave** Indicates right ventricular hypertrophy
- **Mid-diastolic thrill** felt at apex in severe MS
- **Palpable P2** (in pulmonary area) due to pulmonary hypertension

Auscultatory findings of MS

- **S1 – S2 – Opening Snap – Mid-diastolic murmur**(sequence)
- **Loud S1**: the transmitral gradient holds the mitral valve open almost to the end of the diastole, so that at the beginning of ventricular systole, the mitral valve leaflets move a long a long distance for closure.
- **Opening Snap**: After A2 due to sudden tensing of stenosed valve

Murmur

- **Low-pitched, rumbling mid-diastolic murmur** best heard at the apex with bell in left lateral position during expiration with presystolic accentuation (only in sinus rhythm), radiates to axilla, Increases with exercise (handgrip/squatting)



- Differential diagnosis of mid-diastolic murmur
 1. Mitral stenosis
 2. Tricuspid stenosis
 3. Atrial myxoma
 4. Ball – valve thrombus in LA
 5. ASD (increased flow across the tricuspid valve)
 6. Austin Flint murmur (in severe AR)
 7. MR with increased flow through the MV during diastole

- The opening snap (OS) - OS in Mitral stenosis is a classic auscultatory finding, and it comes down to sudden tensing of the stenotic mitral valve leaflets. This indicates valves are pliable.
- Shorter A2–OS interval indicates severe MS
- Pulmonary Hypertension Signs**
- Loud P2 ± pulmonary ejection click

Right Heart Failure Features

- Ascites precox (ascites before pedal oedema)
- Tender hepatomegaly
- Parasternal heave (RV hypertrophy)
- Pleural effusion, anasarca

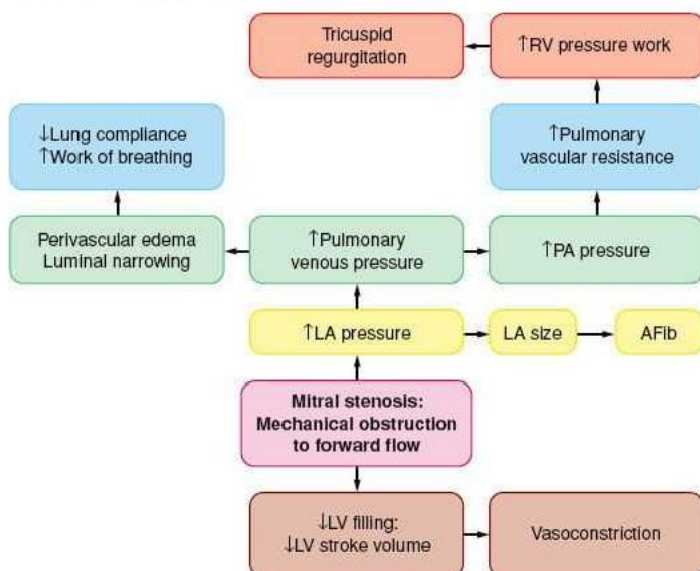
Respiratory system

- Tachypnoea
- Basal crepitations in pulmonary congestion

Compression Features

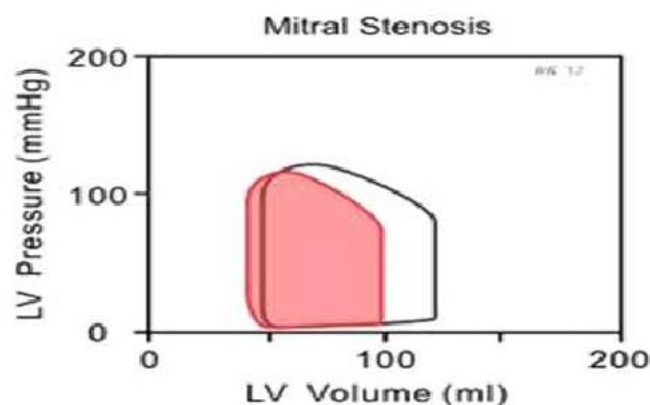
- Ortner's syndrome :hoarseness due to enlarged LA
- Dysphagia / cough :oesophageal or bronchial compression

PATHOPHYSIOLOGY



PRESSURE – VOLUME LOOP IN MS

Mitral stenosis reduces left ventricular filling, leading to decreased LV end-diastolic volume and reduced stroke volume, producing a smaller leftward pressure-volume loop.



Normal mitral valve area 4-6 cm²

Normal diastolic flow across mitral valve 150 – 200 ml/sec/diastole

Normal diastolic transvalvular pressure gradient < 2 mm Hg

GRADING OF MS

SEVERITY	MITRAL VALVE AREA (Cm ²)	END DIASTOLIC PRESSURE GRADIENT (mmHg)	PULMONARY ARTERY PRESSURE (mmHg)	SYMPTOMS
Mild	>1.5	<5	Normal <30	Usually absent
Moderate	1.0-1.5	5-10	30-50	NYHA class II
Severe	<1.0	>10	>50	NYHA class III and IV

CRITICAL MS - <0.6

NEW REVISED JONES CRITERIA

Major Criteria

- Carditis (clinical or subclinical)
- Arthritis (polyarthritis / monoarthritis in high-risk)
- Chorea
- Erythema marginatum
- Subcutaneous nodules

Minor Criteria

- Clinical:
 - Fever
 - Arthralgia
- Laboratory:
 - Raised ESR
 - Raised CRP
- ECG: Prolonged PR interval



Diagnostic Requirement

(Evidence of preceding Group A streptococcal infection is essential)

Initial Episode: 2 Major OR 1 Major + 2 Minor

Recurrent Episode: 2 Major OR 1 Major + 2 Minor
OR 3 Minor

ANATOMY OF MITRAL VALVE

- The mitral valve is a bicuspid atrioventricular valve located between the left atrium and left ventricle.

- It has two leaflets:
- Anterior leaflet (larger, semi-circular)
- Posterior leaflet (smaller, scalloped)

The mitral valve functions as a complex apparatus, consisting of:

- Leaflets (anterior and posterior)
- Chordae tendineae
- Papillary muscles
- Mitral annulus
- Left ventricular wall

PATHOLOGICAL VARIANTS OF MS

1. Fish Mouth (Most Common)

- Classical rheumatic mitral stenosis
- Due to commissural fusion
- Valve orifice appears slit-like / fish mouth shaped
- Often associated with leaflet thickening and calcification

2. Button Hole Type

- More rounded, small central opening
- Severe narrowing with fibrosis

3. Funnel-Shaped Type

- Due to marked sub valvular involvement
- Chordae tendineae thickened, shortened, fused

MORPHOLOGICAL VARIANTS OF MS

- Pure mitral stenosis**-valve leaflets become stiff, thickened, and fused, making it hard for the valve to open. It restricts forward flow. (25%)
- Mixed mitral valve disease** – this occurs when a patient has stenosis and regurgitation at the same time. The valve cannot open properly and the seal is broken so it leaks backward during contractions.(40%)

WHY MITRAL VALVE -IS MORE VULNERABLE ?

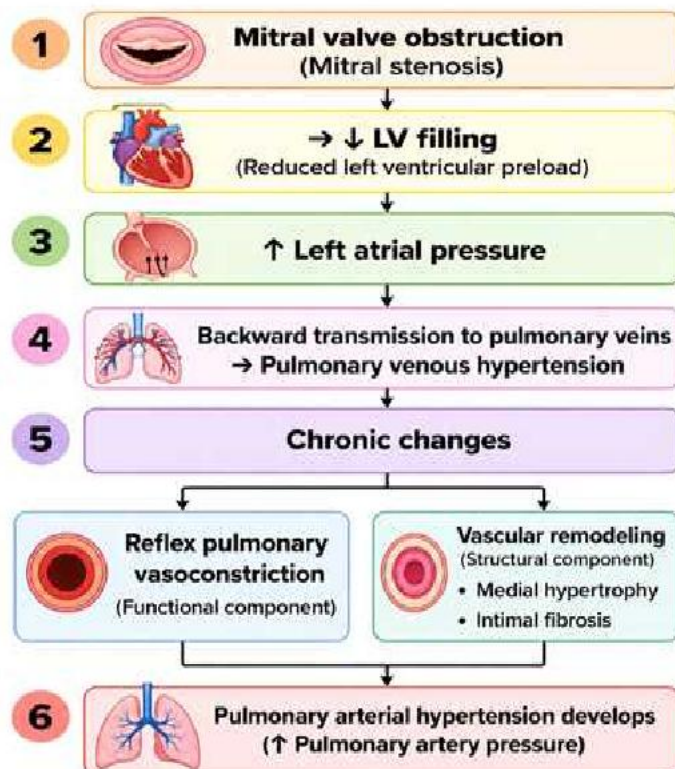
- DEPENDS ON VASCULARITY OF THE VALVE
 - DEGREE OF NATURAL TRAUMA
 - LARGE AREA OF MITRAL LEAFLETS
 - HIGHER PRESSURE GRADIENT
- Hence mitral valve is most commonly effected.

Pulmonary Hypertension

- Pulmonary hypertension is defined as a mean pulmonary artery pressure ≥ 25 mmHg at rest.

Classification (based on pulmonary artery pressure)

- Mild < 30 mmHg
- Moderate $30-50$ mmHg
- Severe > 50 mmHg



INVESTIGATIONS

- CBC, coagulation profile, LFT, RFT
- ABG and electrolytes when indicated
- Specific investigations like – CXR, ECG, 2D ECHO
- Exercise tolerance testing.
- Angiography in patients with co-existing coronary artery disease

ECG CHANGES

ECG may be normal or the patient may be in Atrial fibrillation- Chaotic atrial activity,
No discernible or absent P waves, Irregular R-R intervals

- If patient is in sinus rhythm :P mitrale -Classic broad, M-shaped P wave in Lead II, P wave duration > 120 ms
- Biphasic P wave in V1



broad, M-shaped P wave in Lead II, P wave duration > 120 ms

- Biphase P wave in V1
- Prominent negative component (P negative in V1)-Suggests increased left atrial pressure
- Right Ventricular Hypertrophy (RVH)-Tall R wave, Small S wave, Increased R/S ratio in V1
- Right Axis Deviation (RAD)
- Associated repolarization changes-ST depression and T wave inversion in V1, V2
- Digoxin (Digitalis) effect on ECG: ST segment depression with characteristic "scooped / sagging" appearance
- Decreased T wave amplitude / flattened T waves, Shortened QT interval, Prominent U waves, Prolonged PR interval (may be seen)

CHEST XRAY CHANGES

1. May be normal in mild disease or show slightly increased cardiothoracic ratio
2. Left atrial enlargement: Double density sign (right heart border)
3. Straightening of left heart border
4. Elevation of left main bronchus
5. Indentation of oesophagus (on barium swallow)
6. Pulmonary changes: Pulmonary venous hypertension
7. Moustache / antler sign (prominent upper lobe veins)
8. Miliary mottling due to pulmonary hemosiderosis
9. Pulmonary hypertension features: Prominent pulmonary conus
10. May have right ventricular enlargement (late)
11. Other findings: Small aortic knuckle (decreased cardiac output)
12. Calcification of mitral valve
13. Left lower lobe collapse (due to bronchial compression)
14. Left ventricle: Typically, not enlarged

ECHOCARDIOGRAPHY

- Mitral valve morphology
- Leaflet mobility & flexibility
- Leaflet thickness
- Calcification
- Subvalvular fusion
- Commissural involvement
- Cardiac structure & function
- Left atrial size and LA thrombus
- Ventricular function

- Other valves
- Chamber size
- Pulmonary artery pressure

Scoring Systems (for intervention planning)

- Wilkins score (grades 1–4 each): Mobility, thickness, Calcification, Subvalvular involvement
- Favourable for balloon valvotomy: Wilkins score < 8, Padial score < 10, Less than moderate MR
- Padial score: Leaflet thickening, Commissural calcification, Subvalvular disease

TEE

- Performed before percutaneous valvotomy to rule out left atrial (LA) thrombus
- Used intraoperatively and postoperatively to reassess the mitral valve and evaluate outcomes

Exercise Echocardiography in Mitral Stenosis

- Performed using: Upright treadmill OR Supine bicycle
- Uses Doppler echocardiography
- Measures: Trans mitral valve gradient, Tricuspid valve velocities

Cardiac Catheterization in Mitral Stenosis

- Invasive investigation
- Indications :When non-invasive tests are inconclusive
- When there is discrepancy between non-invasive findings and clinical findings
- Purpose: To assess severity of mitral stenosis
- Mitral valve area can be calculated based on Gorlin formula

$$\text{Formula: MVA} = \frac{\text{CO (mL/min)}}{37.7 \times \text{HR} \times \text{DFP} \times \sqrt{\text{mean gradient (mmHg)}} \cdot$$

CO (mL/min): Cardiac output.

HR (beats/min): Heart rate.

DFP (sec/beat): Diastolic filling period (time the mitral valve is open).

37.7: A constant derived from the discharge coefficient (Cd = 0.85 for mitral) and unit conversions.

COMPLICATIONS OF MITRAL STENOSIS

Cardiac & Hemodynamic Complications

- Atrial fibrillation, leading to: Loss of atrial contribution to ventricular filling-Sudden fall in cardiac output. Thromboembolic risk markedly increases in Atrial fibrillation



- Pulmonary hypertension
- Right ventricular failure and functional tricuspid regurgitation

Pulmonary Complications

- Pulmonary oedema
- Recurrent infections

Thromboembolic Complications

- Thrombus formation
- Systemic embolization, especially: Ischemic stroke, Peripheral embolism

ATRIAL FIBRILLATION

Occurs due to structural heart changes, especially:

- Left atrial dilatation
- Electrophysiologic abnormalities

These lead to: Abnormal impulse generation and propagation

In MS: chronic pressure overload leads to LA dilatation this predisposes to AF

Signs and Symptoms of Atrial Fibrillation

Fatigue
Generalized weakness
Palpitations
Hypotension
Syncope
Angina pectoris
Shortness of breath
Orthopnoea
Increased JVP, Absent a wave

ECG Findings

Chaotic atrial activity
No discernible or absent P waves
Irregular R-R intervals

Echocardiographic Findings

- Reflects consequences of AF and MS:
- Left atrial dilatation >50%
- Absence of coordinated atrial contraction
- Blood stasis in LA
- Atrial thrombus formation (due to stasis)

Treatment Strategies for Mitral Stenosis (MS)

1. Medical Management

Medical therapy is mainly aimed at symptom control and prevention of complications:

• Diuretics are used to relieve pulmonary congestion and reduce symptoms of fluid overload.

- In patients with atrial fibrillation (AF):
- Anticoagulation (e.g., warfarin) is indicated to prevent thromboembolism.
- Rate control is achieved using beta-blockers or calcium channel blockers.

Indications for anticoagulation therapy in MS

- Patients with AF, prior embolic event, thrombus in LA, severe MS, LA dimension 55mm in 2D ECHO

2. Interventional and Surgical Management

These are definitive treatment options, chosen based

- on severity and valve morphology:
 - Percutaneous Balloon Mitral Valvuloplasty (PBMV)
 - Preferred in patients with moderate to severe MS (mitral valve area < 1.5 cm²)
 - Best suited for patients with favourable valve anatomy
- Contraindications to PBMV include:
 - Left atrial or left atrial appendage thrombus
 - Moderate to severe mitral regurgitation
 - Unfavourable valve morphology (e.g., heavily calcified or fibrotic valves)
- Surgical Options
 - Open mitral valvotomy (commissurotomy)
 - Mitral valve repair or replacement (used when the valve is not suitable for repair)
 - Closed mitral valvotomy – not doing now a days

How does this surgical condition affect symptoms of MS?

MS is a fixed cardiac output state. Patients need normal heart rates for adequate diastolic filling. Pain, sepsis, hypovolemia can cause sympathetic surge leading to tachycardia which causes decreased, decreased LV filling, decreased cardiac output, increased left atrium pressure causing pulmonary congestion which can result in worsening of dyspnoea, orthopnoea and pulmonary oedema. Drop in systemic vascular resistance (SVR) is detrimental leading to drop in CO. Sepsis causes decrease in SVR causing severe hypotension resulting in tissue hypoperfusion and shock. Hypoxia, acidosis, hypercarbia seen in sepsis can increase PVR, worsen pulmonary hypertension and right heart failure. Surgical stress results in tachycardia and worsening of symptoms.



What are the principles and goals of anaesthetic management in this case?

- 1) Avoid tachycardia, maintain sinus rhythm.
- 2) Avoid increase in PVR.
- 3) Maintain SVR.
- 4) Avoid hypoxia, hypercarbia and acidosis.
- 5) maintain myocardial contractility.
- 6) Judicious fluid management.
- 7) Adequate depth of Anaesthesia.

How do you optimize patient preoperatively?

Pre operative optimization is challenging since this is an emergency case scenario. Aim is rapid stabilization while preventing decompensation of severe MS.

- Provide analgesics for pain and antipyretics for fever to avoid tachycardia.
- Ryles tube insertion and suctioning
- Optimize volume status carefully with small fluid boluses with frequent reassessment (bedside ECHO if available) avoiding aggressive fluid loading
- If patient is hypoxic oxygen supplementation done.
- Treat pulmonary oedema with diuretics cautiously.
- If atrial fibrillation present

1) Treat with beta blockers/amiodarone/calcium channel blockers for rate control/reverting to sinus rhythm

2) Cardioversion if unstable

- Aggressive sepsis management with IV antibiotics.
- Correct metabolic acidosis and electrolyte imbalance.
- To use vasopressors if indicated to maintain good SVR since hypotension is poorly tolerated in MS patients.
- Oral anticoagulants like warfarin, newer oral anticoagulants have to be stopped. Injection heparin may have to be started before surgery if there is LA thrombus
- If coagulation is deranged either because of sepsis or if patient is on anticoagulants for LA thrombus/AF then FFP and injection vitamin K may have to be given before surgery.
- Drugs like beta blockers, digoxin, diuretics to be continued preoperatively. As patient will be kept Nil per orally alternative IV drugs to be given.

What are the indications of balloon mitral valvotomy?

Do you consider BMV in this patient?

Indications-

- Class I recommendation – symptomatic patients with mod- severe MS
With: favourable valve morphology
No left atrial thrombus
Mild MR
- Class IIB recommendation – asymptomatic patients with mod- severe MS
With: PASP >50mmhg
New onset AF
- BMV can be considered before elective surgeries. Since this is an emergency case BMV is not considered in this patient.
- BMV can be done antenatally in mid trimester in case of severe MS
- It can be considered done in sick patients with severe MS with pulmonary oedema on emergency basis

Which is the ideal induction agent of choice?

Etomidate is the ideal induction agent of choice as it maintains hemodynamic stability with minimal cardiovascular depression, no tachycardia and maintains SVR. Thiopentone can also be used .

What are the basic monitors used?

- Standard ASA monitors – ECG, Pulse oximeter, NIBP, ETCO₂, Temperature
- IBP (PPV, SPV), CVP monitoring, urine output, airway pressures.
- Point of care ultrasound- TTE and Lung Ultrasound.

Discuss induction of anaesthesia in this case

General anaesthesia with endotracheal intubation (RSI) with controlled ventilation is the anaesthesia plan. Standard ASA monitors attached. Wide bore iv cannula taken. Awake arterial line secured. Preoxygenated with 100% fio₂ for 3min. premedicated with inj Midazolam 0.03 – 0.05mg/kg iv , inj Fentanyl 3-5mcg/kg. induced with inj Etomidate 0.2-0.5mg/kg iv and muscle relaxation with inj Succinylcholine 2mg/kg or inj Rocuronium 1.2mg/kg iv. Cricoid pressure to be applied. Glycopyrrolate avoided. Endotracheal intubation done after gentle laryngoscopy.

Discuss maintenance of anaesthesia

Adequate depth of anaesthesia is maintained with isoflurane and intermittent inj Atracurium iv for muscle



peripheral perfusion emergency cardioversion has to be done irrespective of status of LA thrombus.

What is the role of infective endocarditis (IE) prophylaxis in this case?

IE prophylaxis is not indicated in a patient with severe MS undergoing surgery.

Indications- 1. Patients with prosthetic cardiac valve.

2. History of previous IE.

3. CHD patients.

4. Cardiac transplant recipients.

(IE prophylaxis only in patients Undergoing dental procedures involving gingival manipulation and perforation of oral mucosa and not for GI/GU procedures)

VIVA QUESTIONS

1) WHAT IS MITRAL STENOSIS ?

2) WHAT ARE THE CAUSES MS?

3) WHAT IS DYSPNEA ?

HOW DO YOU GRADE DYSPNEA ?

4) WHAT ARE THE SYMPTOMS OF MS?

5) WHAT ARE THE CLINICAL FINDINGS?

6) WHAT ARE THE PALPATORY FINDINGS OF MS?

7) WHAT ARE THE OTHER CAUSES OF MID DIASTOLIC MURMUR?

8) IN WHICH CONDITIONS DO YOU SEE IRREGULARLY IRREGULAR PULSE?

9) WHAT CAUSES OPENING SNAP WHAT DOES IT INDICATE ?

10) WHAT IS THE PATHOPHYSIOLOGY?

11) HOW DO YOU GRADE MITRAL STENOSIS ?

12) WHAT IS NEW REVISED JONES CRITERIA ?

13) DESCRIBE ANATOMY OF MITRAL VALVE

14) WHAT ARE THE PATHOLOGICAL VARIANTS OF MS?

15) WHY MITRAL VALVE COMMONLY INVOLVED IN MS?

16) WHY DOES MITRAL STENOSIS CAUSE PULMONARY HYPERTENSION?

17) WHAT ARE THE TYPES OF PULMONARY HYPERTENSION?

18) WHY DOES HEMOPTYSIS OCCUR IN MS?

19) WHAT IS THE MECHANISM OF ORTHOPNEA AND PND?

20) WHAT INVESTIGATIONS WOULD YOU ORDER FOR A CASE OF MS ?

21) WHAT ARE THE ECG FINDINGS ?

22) WHAT ARE THE CHEST XRAY CHANGES?

23) MITRAL VALVE EVALUATION ON ECHOCARDIOGRAPHY: WHAT TO ASSESS?

24) HOW DO YOU CALCULATE MITRAL VALVE AREA?

25) ENUMERATE THE COMPLICATIONS OF MS

26) WHAT ARE THE INDICATIONS OF ANTICOAGULATION THERAPY IN MS?



13th JUNE 2026

02:05PM - 02:35PM

Anaesthetic Management Of A Three Year Old With Foreign Body In Bronchus

CASE SUMMARY:

A 3-year-old male child, weighing 14 kg, was brought to the emergency department by his parents with complaints of sudden onset coughing & difficulty in breathing, and noisy breathing since 2 hours, following a witnessed episode of playing with peanuts.

1) What is the importance of foreign body aspiration in children and why children aged between 1-3 years are more prone?



FB aspiration is the leading cause of death in children between 1-3 years of age (Toddler).

1. Explorative nature
2. Mouthing
3. Inadequate dentition absence of molars and premolars leads to improper grinding of food)
4. Immature swallowing coordination reflex
5. Lack of parental supervision

Newborn	0 d to 1 wk
Neonate	1 wk to 1 mo
Infant	1 mo to 1 y
Toddler and preschool	2-5 y
School age child	6-12 y
Adolescent and young adult	13 to <18 y



2) What are the locations of foreign body aspiration, which is the most common site and why?

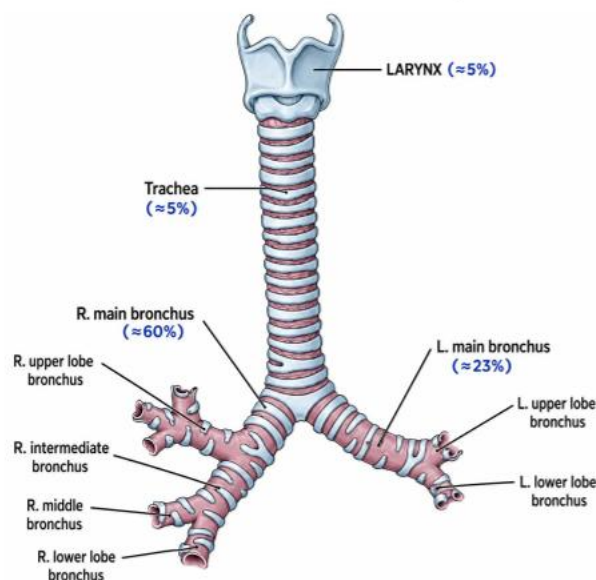
Location of foreign body depends on:

- size of FB
- shape of FB
- nature of FB

Most common site is RIGHT bronchus.

Symmetrical bronchial angles till 15 years of age (aortic arch development makes left main stem bronchus more horizontal)

3)What are the locations of foreign body obstruction , which is the most common site and why?



Right bronchus – 60 percent

Left bronchus – 23 percent

Trachea/carina – 13 percent

Larynx – 3 percent

4) What are the different types of foreign body aspirated, the implications and complications of the various types?

Organic (96%)

Seeds (Groundnuts, Custard apple and tamarind most common), legumes
Bones, popcorns

More irritant and swells up(Hygrosopic) causing progressive worsening of obstruction (partial to complete obstruction)

More chances of fragmentation causing multiple distal obstructions

More inflammatory and granulomatous reaction



Inorganic (4%)

Metallic: coins, screw pins, button batteries

Non metallic: Toy parts, pen caps, pebbles, wood pieces

Sharps: safety pins are lodged predominantly supraglottic,



Less irritant, Less chances of fragmentation, Less inflammatory

5) What is the clinical presentation of a child with foreign body aspiration?



Sudden onset coughing, choking, wheezing

Upper airway:

stridor, **cyanosis**, aphonia and hoarseness of voice

Severity depends on

1. Early versus late presentation
2. Size and shape of inhaled object
3. Location of obstruction (trachea/ main bronchus/distal airways)

6) What are the different stages of foreign body aspiration?

May present in 3 phases or may progress

Stage 1: immediate phase: choking, gagging, coughing, wheezing, stridor or cyanosis. (High Mortality)

Stage 2: Asymptomatic phase: (minutes - months)

Stage 3: Clinical Phase: renewed symptomatic period airway inflammation, repeated cough, sputum production, fever, wheezing and hemoptysis

Chronic long standing FB: misdiagnosed as LRTI, Asthma

HIGH DEGREE OF SUSPICION REQUIRED

7) What are the clinical signs on examination of a child with foreign body aspiration?

NORMAL VITALS IN 25-40%

Pulse: tachycardia followed by bradycardia (hypoxia)

Cyanosis in laryngeal and complete main stem

bronchus obstruction

Persistent cough (78% sensitivity and 37%specific)

Barking cough (laryngeal edema)

8) What are the clinical signs on examination of a child with foreign body aspiration?

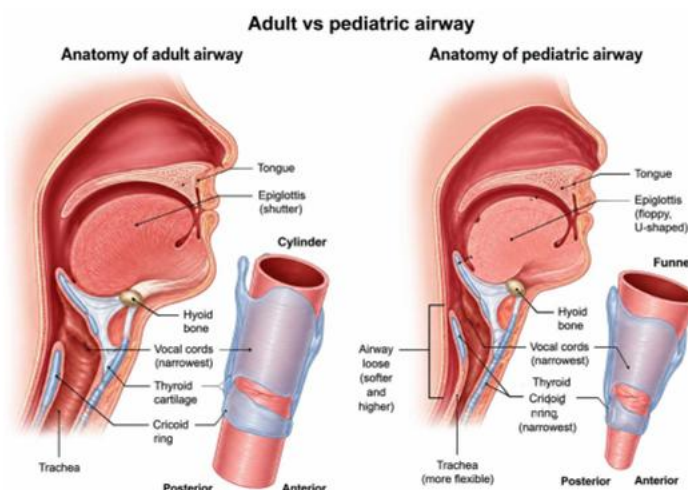
If complete airway obstruction-Inability to cry/speak

,Cyanosis ,Severe respiratory distress

,Collapse/unconsciousness

Respiratory system examination:

- Inspection: Stridor, Intercostal/ sternal retractions, unilateral reduced chest movements (atelectasis/post obstructive hyperinflation)
- Auscultation: U/L reduced breath sounds, with Ronchi(monophonic/polyphonic)
- Alternating loss of breath sounds indicate shifting FB



10) What are the physiological changes in Respiratory System in Paediatric age group?

1. Lung Development & Growth - At birth, ~50 million alveoli are present; this increases to ~300–500 million by age 8. Alveolar surface area grows from ~3–4 m² at birth to ~70–80 m² in adulthood. Airway branching is complete by birth, but airway diameter increases with age.

2. Mechanics of Breathing - Paediatric chest wall is highly compliant (cartilaginous ribs, immature musculature), offering little outward recoil. This reduces functional residual capacity (FRC) and predisposes to airway collapse, atelectasis, and dynamic airway obstruction.



3. Diaphragm Dependence - Infants are almost entirely diaphragm-dependent for ventilation. The diaphragm has fewer Type I (fatigue-resistant) fibers at birth (~25% vs. ~55% in adults), making it prone to fatigue during increased work of breathing.

4. Intercostal Muscles - Poorly developed; contribute minimally to tidal breathing.

5. Lung Volumes & Capacities

Parameter	Neonate/Infant	Adult
Tidal Volume (mL/kg)	6–8	6–8
RR (breaths/min)	40–60	12–20
FRC (mL/kg)	~27	~35
Closing Capacity	> FRC	< FRC

6. Oxygen Consumption & Delivery - Metabolic rate is 2–3× higher per kg in infants vs. adults. Oxygen consumption is ~6–8 mL/kg/min. This means apnea leads to rapid desaturation-preoxygenation is critical. Hemoglobin transitions from fetal (HbF, high O₂ affinity, left-shifted ODC) to adult form (HbA) by ~6 months, which initially impairs O₂ offloading to tissues.

7. Respiratory Rate & Control - Respiratory rate is inversely proportional to age. Tidal volume per kg remains relatively constant across ages.

Age	Normal RR
Neonate	40 – 60
1 year	24 – 40
5 years	20 – 30
10 years	15 – 25
Adult	12 – 20

Central respiratory control is immature in neonates — periodic breathing and apnea of prematurity reflect this.

8. Airway Resistance - A small reduction in airway radius (e.g., 1 mm edema) dramatically increases resistance in infants. A 1 mm reduction in a 4 mm infant airway increases resistance by ~75%. This explains why minor airway inflammation causes severe respiratory distress in infants.

9. Surfactant & Lung Compliance - Surfactant production begins ~24 weeks; mature levels by ~35 weeks. Surfactant reduces surface tension, prevents alveolar collapse, and maintains FRC.

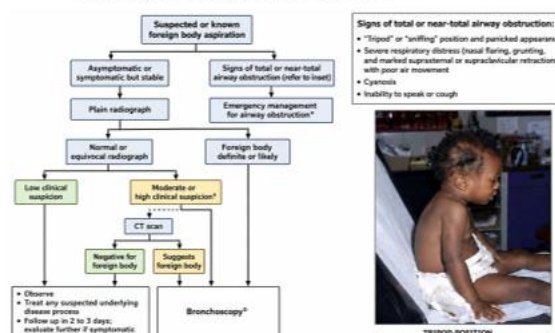
Deficiency → Respiratory Distress Syndrome (RDS/HMD) in premature infants.

10. Dead Space & Gas Exchange - Anatomical dead space (~2 mL/kg) is proportionally similar to adults, but because RR is high, minute ventilation is relatively higher per kg. Neonates have higher physiologic shunt (~15–20%) due to persistence of fetal channels (PFO, PDA) and V/Q mismatch, normalizing over weeks to months.

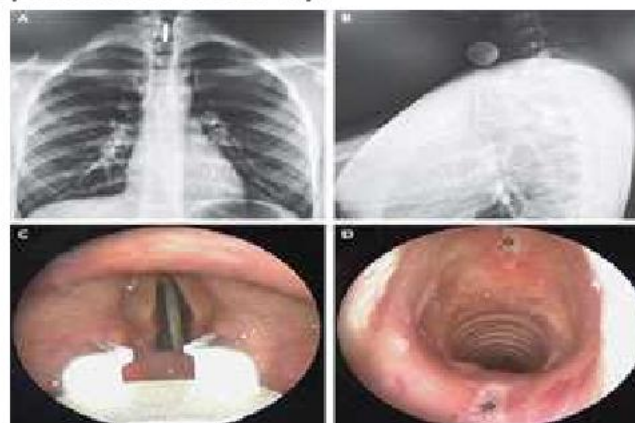
Clinical Implications

- Rapid desaturation during apnea → aggressive preoxygenation
- Easy fatigue → early intubation in distress
- Closing capacity > FRC → PEEP is beneficial
- High resistance to minor obstruction → humidify, avoid mucosal trauma
- Immature respiratory drive → monitor for apnea in ex-premature infants up to 60 weeks post-conceptual age

11) How do you evaluate a child with a suspected foreign body aspiration?



12) What is the role of imaging in diagnosing foreign body aspiration and how do you pick foreign body aspiration on a chest x-ray?



Chest X-ray:

1. Anteroposterior
 2. Lateral views
 3. Inspiratory and expiratory films
- Most FB NOT radio-opaque (10 percent of FB)
Indirect findings: Mediastinal shift, Atelectasis, Hyperinflation, Pneumonia.



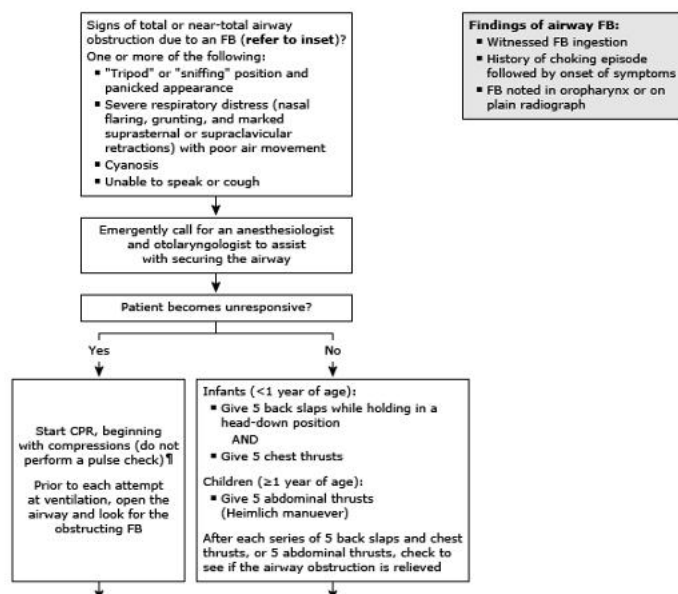
13) What is the role of CT scan in foreign body aspiration and when is it required?

Computed tomography

Airway CT is appropriate for children with all of the following:

- Moderate clinical suspicion of FBA
- Inconclusive results of conventional radiographs (three views)
- CT is available with a low-dose, high-speed protocol, so that sedation is not required

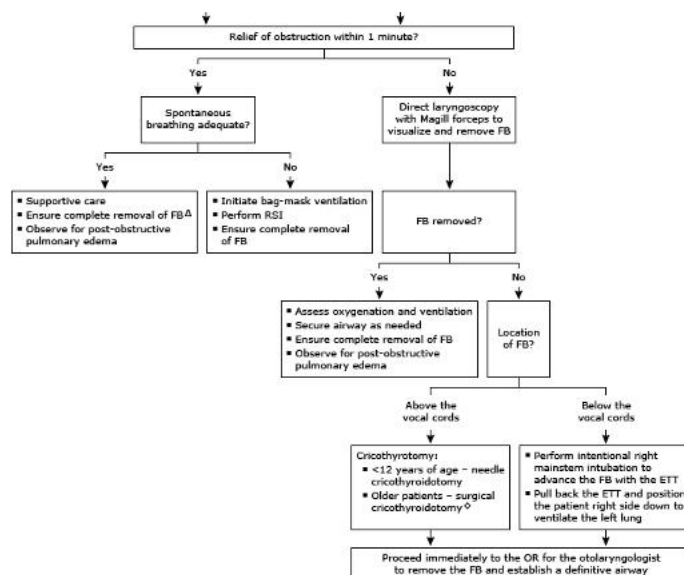
14) How do you manage foreign body aspiration in a three year old child?



Do not thrust hard enough to lift the child off his feet



Chest Thrusts
Rescuer holds infant on thigh in head-down position and delivers up to 4 chest thrusts in same manner as chest compressions (see below)



15) How do you manage a case of foreign body bronchus removal?

- The definitive diagnosis and management of foreign body typically involves bronchoscopy to remove the impending object.
- If patient is in distress-emergency bronchoscopy
- If patient is stable-planned bronchoscopy
- Both rigid bronchoscopy and fiberoptic bronchoscopy can be done.
- Rigid bronchoscope is the gold standard for removing foreign body.

Measures before bronchoscopy - A team effort.

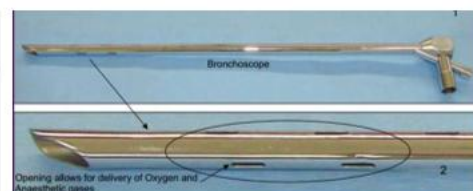
- Good communication between surgeon and an Anaesthesiologist.
- Expert ENT surgeon and Anaesthesiologist.
- Light and suction checked before procedure.
- Proper size bronchoscope, ventilatory circuits and connectors checked
- **Rigid Bronchoscope** : Standard of care in most centers for evaluation.

Allows visualization, ventilation, removal with multiple forceps and ready management of mucosal haemorrhage.

Ventilating bronchoscope is preferred for rigid bronchoscopy.

There are various sizes ranging from 2.5- 6.0mm internal diameter.

Concerns



Concerns during Bronchoscopy



- Lumen of bronchoscope is narrowed when telescope, forceps suction catheter are inserted.
- Rate of rise PCO₂ in apneic infants and children is extremely high (9mmHg/min)
- Ventilation has to be interrupted during suctioning and removal of foreign body.
- After each period of apnea, telescope and endo bronchial suction are removed through bronchoscope sidearm.
- Distal end of bronchoscope pulled above carina and proximal end of bronchoscope is occluded with endoscopists thumb to facilitate ventilation.

Anesthetic concerns of foreign body removal :

- Handling of irritable child
- Full stomach
- Difficulty pertaining to paediatric airway
- Sharing airway with the surgeon
- Difficulty to maintain oxygenation and ventilation

Flexible bronchoscopy:

- 3 main parts
1. Handle
 2. Insertion tube
 3. Flexible tip

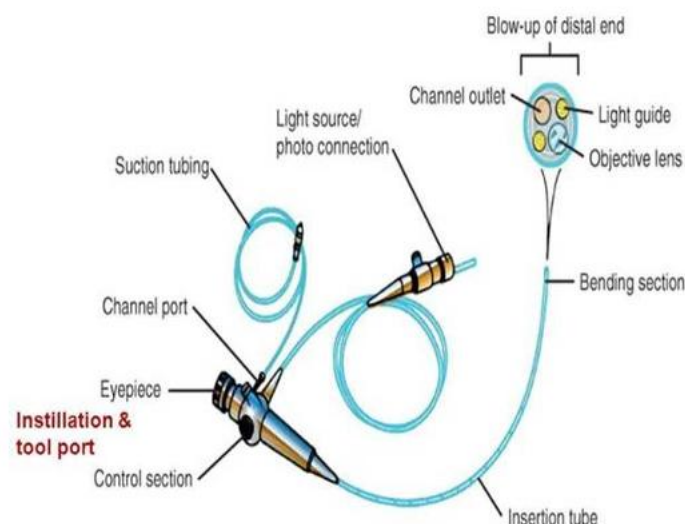


FIGURE 33-43 Flexible fiberoptic bronchoscope.



16) Explain steps of providing General Anesthesia

Premedication

- Use of sedatives depend on the status of the child.
- It is questionable because of risk of airway obstruction and worsening the condition of the child.
- If at all sedatives used, use with caution with supplemental oxygen.
- Dexamethasone given (0.4-1mg max of 20mg) prophylactically to minimize postoperative stridor and laryngeal edema
- Anticholinergics (atropine, glycopyrrolate) to reduce secretions and reflex bradycardia associated with airway instrumentation

Induction – Inhalation Induction & IV Induction

Inhalational induction with sevoflurane 8% with 100% oxygen.

Advantages:

- Makes the induction smooth especially when iv access is not available.

Disadvantages:

- Hypoventilation and leaks around rigid bronchoscope may produce inadequate depth of inhaled anaesthesia.
- Pollution of the operating room and high gas flows needed for ventilation.

IV Induction

- Propofol is used at a dose of 2.5-3mg/kg Advantage
- It provides constant level of anaesthesia irrespective of ventilation
- Propofol is used because of its good reflex suppression and rapid recovery.

Maintenance:

- Volatile anaesthetic agents through bronchoscope or through propofol infusion
- Topical anaesthesia spray with lignocaine 2-4% (up to 3mg/kg) useful in preventing laryngospasm and reaction to surgical manipulation
- Once the rigid bronchoscope is inserted through the glottic opening, anaesthesia circuit is connected to side arm of rigid bronchoscope, allowing maintenance with sevoflurane with oxygen and use spontaneous ventilation or paralyze the patient and use controlled ventilation.



Ventilation : SPONTANEOUS/ CONTROLLED VENTILATION/ JET VENTILATION/ THRIVE(trans nasal humidified rapid-insufflation ventilatory exchange)/ APNOEIC OXYGENATION

1) SPONTANEOUS VENTILATION

Advantages of spontaneous ventilation

- Lower risk compared with positive pressure ventilation that foreign body may move distally
- Allows for continued ventilation during removal of foreign body
- Rapid assessment of adequacy of airway after removal of foreign body.

Disadvantages of spontaneous ventilation

- Depth of anaesthesia required to permit insertion of instruments in to the airway decreases both cardiac output and ventilation
- Increased resistance to ventilation
- Hard to guarantee no movement, which leads to airway trauma.

2) CONTROLLED VENTILATION

Advantages of controlled ventilation

- Usage of muscle relaxant leads airway immobilization
- PPV avoids hypoxemia and improves oxygenation
- Decreased airway resistance

Disadvantages of controlled ventilation

- May cause distal migration of foreign body and may cause difficulty in removing
- Leads to over distension of obstructed airway > may cause rupture of alveoli > pneumothorax

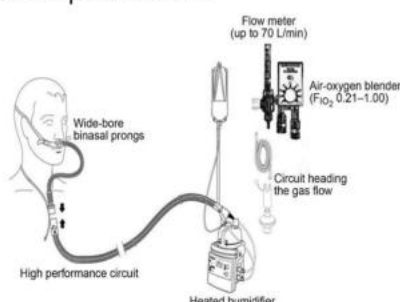
How is adequacy of ventilation checked when ventilating through a bronchoscope-

Bilateral breath sounds

- Bilateral chest rise
- SpO₂ level can confirm
- EtCO₂ level

3) THRIVE (Trans nasal humidified rapid-insufflation ventilatory exchange)

- High flow oxygen at the rate of 20 L/min can be given.
- Increases safe apneic time.



4) MANUAL JET VENTILATION



- It uses high pressure gas source via narrow cannula attached to a suspension laryngoscope or bronchoscope.

- Gas is delivered by operation of a hand operated at a switch at a rate of 10-20bpm.

Advantage: fewer episodes of intraoperative hypoxemia, provides continuous ventilation

Disadvantage: barotrauma

5) APNEIC VENTILATION

- It provides oxygen rich mixture in the pharynx and due to bulk flow of gases down a pressure gradient there is an improvement in arterial oxygen saturation and safe apnea time.



Techniques:

1. Nasal prongs
2. THRIVE
3. Nasopharyngeal catheter
4. Tracheal catheter
5. Endobronchial catheter

17) Why Inhalational Induction is preferred in children & what is Step-Up & Step-Down Inhalational Induction in Pediatrics?

Children fear needles. Inhalational induction allows anaesthesia to be established before IV access, avoiding the distress of awake cannulation. Sevoflurane is the agent of choice - non-pungent, sweet-smelling, rapid onset, and hemodynamically stable.

Step-Up Technique - Start with low concentrations of sevoflurane and gradually increase in incremental steps until loss of consciousness.

Technique

- Start with 100% O₂ for a few breaths to familiarize
- Begin sevoflurane at 0.5–1%
- Increase by 0.5–1% every 3–5 breaths
- Progress through 2% → 4% → 6% → 8%
- Loss of consciousness typically occurs at 4–6%
- Once unconscious - establish IV access

Advantages

- Better tolerated in anxious or sensitive children
- Less breath-holding and excitement phase
- Smoother induction with less airway irritability
- Child feels gradual, gentle transition
- Reduces laryngospasm risk

Disadvantages

- Slower - takes longer to reach surgical depth

Step-Down Technique

Start immediately with high concentration of sevoflurane (7–8%) and reduce once unconsciousness is achieved. Rapid induction first, then titrate down. mainly used for an uncooperative child.

Technique

- Pre-fill circuit with 8% sevoflurane + 100% O₂
- Apply mask with high flow (6–8 L/min)
- Child takes 3–5 breaths of high concentration
- Loss of consciousness within 30–60 seconds
- Immediately reduce to maintenance level (2–3%)
- Establish IV access once unconscious

Advantages

- Rapid loss of consciousness-minimizes time in excitement phase
- Ideal for uncooperative or very anxious children
- Reduces struggling and breath-holding duration

18) What are the numerous intra-operative complications anticipated?

1. Trauma to the lips, teeth, base of tongue, larynx
2. laryngospasm/bronchospasm- Prevented by maintaining adequate depth of anaesthesia and muscle relaxation
3. Hypoxia
4. Arrhythmia
5. Pneumothorax

19) what is the next step after foreign body removal?

- Secure the airway.
- It allows for tracheobronchial suction, lung expansion, oxygenation and ventilation until adequate reversal of muscle relaxation and return to spontaneous breathing

20) what are the indications of endotracheal intubation after foreign body removal ?

Airway edema

- Significant laryngeal or subglottic edema
- Persistent stridor after removal
- Airway trauma during bronchoscopy
- Suspected airway swelling likely to worsen

Respiratory failure

- Persistent hypoxia despite oxygen
- Hypercapnia
- Apnea or poor respiratory effort
- Severe work of breathing/exhaustion

Hemodynamic Instability

- Shock
- Cardiorespiratory arrest

Inadequate Airway protection

- Depressed consciousness
- Poor gag/cough reflex
- Excessive secretions/blood with aspiration risk



21) what are the anticipated post-operative complications?

- Hypoxia
- Bleeding
- Laryngospasm
- Retained fragments of foreign body.
- Laryngeal edema:

Treated by intravenous corticosteroids, nebulization with steroids and racemic epinephrine nebulisation(0.5ml of 2.25% solution diluted in 3ml of saline solution)

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13th JUNE 2026

04:20PM - 04:55PM

ANAESTHESIA EQUIPMENT

Dr. Madhuri S. Kurdi

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AIRWAY DEVICES

FACE MASKS



It is an anatomical face mask.

It consists of 3 parts:

- a) The body
- b) The edge
- c) The mount

2. What are the different types of anatomical face masks?

There are 2 types of anatomical face masks:

- a) Reusable masks
- b) Disposable masks

3. What are the commonly used masks?

- a) Rendell-Baker Soucek mask
- b) Endoscopic mask
- c) Scented mask
- d) Anatomic mask
- e) Ambu transparent mask
- f) King mask

4. What are the different techniques of face mask application?

- a) One hand technique
- b) Two hands technique
- c) Two-handed jaw thrust
- d) Claw hand technique
- e) Mask ventilation of tracheostomy stoma

AIRWAYS

1. What are the different types of airways?

- a) Oropharyngeal airway
- b) Nasopharyngeal airway
- c) Modified laryngeal mask airway
- d) Cuffed oropharyngeal airway

2. What are the parts of an oropharyngeal airway?

- a) Bite block
- b) Body
- c) Central lumen

3. How to estimate the appropriate size of an oropharyngeal airway?

The appropriate size is estimated by selecting the airway whose length corresponds to the vertical distance between the patient's incisors and the angle of the jaw.

4. What are the different types of oral airway?

- a) Guedel airway
- b) Waters' airway
- c) Safar's airway
- d) Connell airway
- e) Berman intubating pharyngeal airway
- f) Ovassapian fiberoptic intubating airway
- g) Patil endoscopic airway
- h) Williams airway intubator

5. What is the nasopharyngeal airway made of?

Nasopharyngeal airways are made of soft plastic, polyurethane, or latex rubber.

6. What are the parts of nasopharyngeal airway?

- a) Beveled tip
- b) Body/tube
- c) Flange

7. How to estimate the appropriate size of nasopharyngeal airway?

The length of the airway needed is calculated as the distance from the tragus of the ear to



the tip of the nose plus 1 inch, or the distance from the tip of the nose to the meatus of the ear.

8. What are the different types of nasal airway?

- Bardex airway
- Binasal airway
- Cuffed nasopharyngeal airway
- Epistaxis airway
- Linder nasopharyngeal airway
- Rusch airway

ENDOTRACHEAL TUBES (ETTs)

1. What are the two conventional types of ETT cuffs?

- High pressure low volume cuff
- Low pressure high volume cuff

2. Describe the parts of endotracheal tube.

- Patient/distal end
Bevel
Murphy eye
Cuff
- Tube body/shaft
Radiopaque line
Depth marker
Internal & outer diameter
Pilot balloon
- Machine end
Adapter

3. What are the special varieties of ET tubes?

- Armoured/Anode/Spiral embedded/Reinforced/Flexometallic ETT
- Ring, Adair & Elwyn ETT
- Cole's ETT
- Microcuff endotracheal ETT
- Oxford tube
- Micro-laryngeal tracheal surgery tube
- Tehran tube
- Carden tube
- Hunsaker Mon-jet ventilation tube
- Laryngectomy tube
- Endotrol tube
- Endoflex tip tube
- Parker Flex tip tube
- Electromyogram reinforced tracheal tube
- Intubating laryngeal mask airway tracheal tube
- Double lumen tubes
- Laser resistant tubes

4. What are the indications for endotracheal intubation?

Surgical indications

- Patients for surgery who have full stomach
- Patients requiring IPPV
- All head & neck surgeries with compromised airway
- Surgery for a long time
- Abdominal, thoracic, neurosurgical procedures
- Double lumen tubes for intrathoracic surgery

Non-surgical indications

- Cardiopulmonary-cerebral resuscitation
- Conscious or semiconscious patients unable to protect the airway
- Inadequate/gasping respiration
- Tracheobronchial toilet for retained secretions

5. What are the parts of ETT cuff system?

- Inflatable cuff
- Cuff inflation tubing
- Pilot balloon
- Self-sealing valve to inflate/deflate the cuff

6. How do you confirm the correct placement of tube?

Clinical confirmation

- Direct visualization of tube passing through vocal cords
- Moisture of exhaled gases in the lumen of tube
- Palpation of tube over trachea
- Bilateral chest movement
- Bilateral air entry on auscultation of chest

Confirmation with equipment

- Capnometer – ET_{CO}₂
- Esophageal detector device
- Calorimetric end-tidal CO₂
- Chest X-ray
- Sonomatic confirmation of the tracheal intubation
- Fiberscopic view
- Tracheal illumination
- Pressure & flow-volume loops



MAGILL FORCEPS, BOUGIE AND STYLET

1) What are Magill forceps and what is their role in intubation?

Magill forceps are twin-bladed tong-like curved metallic forceps used mainly during nasotracheal intubation. Magill developed this forceps while working at the Queen's Hospital for Facial and Jaw injuries in Kent to aid in lifting and guiding the nasotracheal tube into the larynx.

After the endotracheal tube passes through the nose into the oropharynx, the forceps help guide the tube toward the glottic opening under direct laryngoscope vision.

Their angled design allows easy manipulation inside the airway without obstructing the view.

Magill forceps are also useful for removing foreign bodies from the airway.

They should be used carefully to avoid cuff damage or trauma to surrounding tissues.

2) What precautions should be taken while using Magill forceps?

While using Magill forceps, the operator must avoid grasping the cuff of the endotracheal tube because it can cause cuff rupture.

The forceps should hold only the proximal part of the tube.

Excessive force may injure the pharynx, vocal cords, or nasal mucosa.

Sterility and gentle handling reduce complications such as bleeding and edema.

3) What is bougie and why is it important in difficult intubation?

A bougie is a flexible tracheal tube introducer used during difficult intubation.

It is useful when only a partial view of the glottis is obtained during laryngoscopy.

4) What are the advantages of a Bougie?

It helps secure the airway even when visualization is poor.

It is simple to use and increases the success rate of difficult intubation.

These are available in operation theatres and ICUs.

5) What are the disadvantages of Bougie?

Advancing the bougie forcefully may injure the trachea or bronchi.

Sometimes the ET tube may get stuck while railroading over the bougie.

6) What is a stylet?

A stylet is a malleable metal and plastic rod inserted inside the ET tube to provide shape and rigidity.

7) How does stylet help during intubation?

It helps direct the tube toward the vocal cords during intubation.

The tube is commonly shaped into a hockey-stick curve for better navigation in difficult airways.

Stylets are especially useful during video laryngoscopy where greater angulation is needed.

8) What precautions should be taken while using a stylet?

The distal end should always remain within the ET tube to prevent mucosal injury.

The tube should be shaped gently without creating very sharp angles.

Excessive force should not be used during advancement because it can traumatize the airway. Lubrication helps easy removal of the stylet after intubation.

9) What are the contributions of Magill?

He contributed a lot to the development of endotracheal intubation.

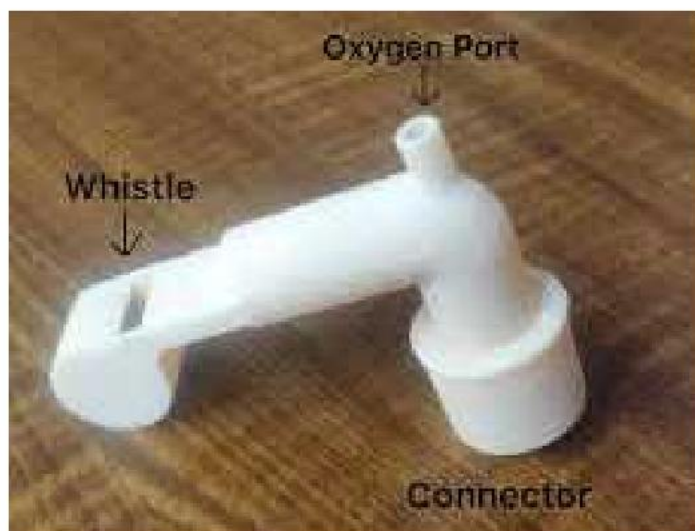
- Modern Single tube technique for endotracheal anaesthesia; 'Magill curve': natural curve of ETT to avoid use of stylet (140+-20mm)
- Blind endotracheal intubation
- Anaesthetic breathing system
- Laryngoscope blade (1926)
- Catheter mount
- Magill's tracheal spray
- Single lung anaesthesia
- Bobbin flowmeters
- Bronchial blockers
- Magill Intubating forceps (1921)
- Magill anaesthetic apparatus (a composite unit of an endotracheal ether insufflation apparatus and a nitrous-oxide and oxygen apparatus)(1927)



10) What are the commonly available introducers, guides and bougies?

- Portex gum elastic bougie
- Frova intubating catheter
- Aintree intubating catheter
- Arndt airway exchanger catheter set
- Sun Med tracheal tube introducer
- Radlyn R – vue
- The flex – guide endotracheal tube introducer
- The Sheridan tube exchanger
- S – guide malleable intubating guide
- Pocket introducer
- Muallem ETT stylets
- The Schroeder oral/nasal directional stylet
- Truflex articulating stylet

11) Identify the device and describe in brief.



Answer- It is Dambale's whistle. Dr. Dambale's Whistle is an innovative, low-cost airway aid developed by Dr. Hemant Dambale for difficult intubation, especially awake nasal intubation. It produces a whistle sound when airflow is aligned with the glottic opening, helping guide the endotracheal tube toward the trachea.

VIDEO LARYNGOSCOPE

1) What is a video laryngoscope? What are the parts of VLS? Answer- Video laryngoscope is an indirect laryngoscope that work on the principle of "looking around the corner" which is made possible by placing the camera at the distal tip of laryngoscope blade

Parts of VLS -

- a) Handle
- b) Laryngoscope blade

- c) Micro video chip camera (embedded at end of blade)
- d) External monitor (LCD)

2) What are the advantages of VLS? Answer- These are the advantages of VLS -

1. Magnified airway view
2. Wider viewing angle
3. Shared visualization for team coordination
4. Excellent teaching tool
5. Improved ergonomics
6. Reduced neck movement
7. Increased distance from patient
8. Documentation and medicolegal utility
9. Visualisation of bougie placement
10. Assessment of vocal cord movement
11. Verification of tracheal tube position
12. Aid in endotracheal tube exchange

3) What are the limitations of VLS?

Answer- These are the limitations of VLS -

1. Needs special training
2. Requires hand-eye coordination
3. Good view doesn't mean easy intubation
4. Often needs stylet-shaped ETT
5. Different VLS devices need separate skills
6. Cannot be used nasally/tracheostomy
7. Difficult in limited mouth opening/trismus
8. Not useful for DLT confirmation/pulmonary toilet
9. Risk of soft palate/oropharyngeal injury
10. One VLS does not fit all situations

4) How do you classify VLS?

Answer - VLS are classified depending on several features

1) Method of advancement:

a) VLS with integrated tube channel Eg- Airtraq, Pentax airway scope , King Vision

b) VLS without integrated tube channel

Eg- GlideScope, McGrath, C-Mac

2) Depending on curvature of blade :a) VLS with standard Macintosh blade

Eg- V-Mac, C- Macb) VLS with angulated blades Eg- GlideScope, C-Mac with D blade

3) Optical laryngoscopes :

Eg- Truview, Bullard laryngoscope, Airtraq optical laryngoscope



5) Identify this device and what are the various types



Answer - This is GlideScope video laryngoscope It has three types -

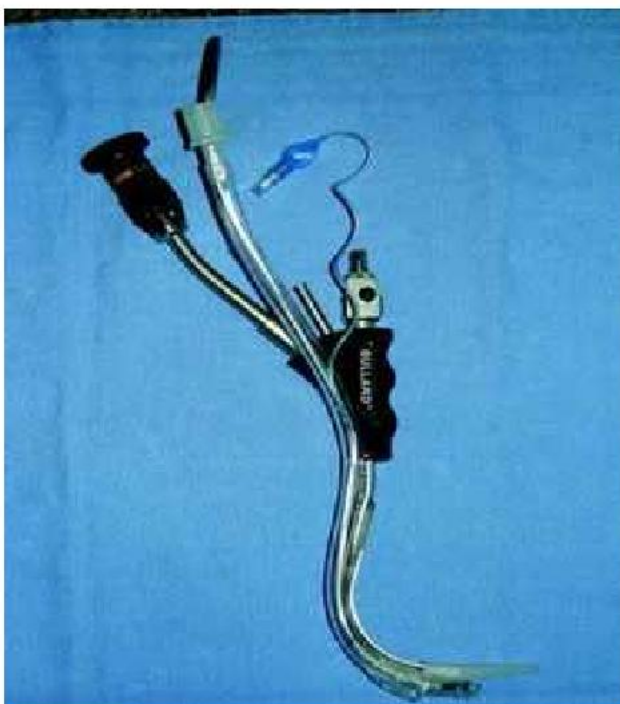
- 1) GlideScope original
- 2) GlideScope Cobalt
- 3) GlideScope Ranger

6) What are limitations of VLS over Fibreoptic bronchoscope?

Answer-

- 1) VLS cannot be introduced through nose / tracheostomy
- 2) Some space is required to introduce VLS, so cannot be used in patients with reduced mouth opening
- 3) Not helpful in confirming the placement of DLT, bronchial blocker, performing pulmonary toilet

7) Identify the device and mention its advantages



Answer - This is Bullard rigid fibreoptic laryngoscope, it is S-shaped rigid fibreoptic laryngoscope with anatomically curved blade.

Advantages-

- a) Can be used in patients with mouth opening as small as 6mm
- b) Allows jet ventilation or oxygen insufflation through working channel
- c) Can be converted to video laryngoscope

8) Identify the device and mention its advantages



Answer- This is Airtraq optical Laryngoscope, developed by Dr. Acha, it is a disposable, optical laryngoscope Advantages -

- a) It has an antifog system built into optical channel which requires 30-45 sec warm up time prior to use
- b) Can be converted to VLS by attaching wireless or wired video system

9) What is Kepler intubation system (KIS)?

Answer - It is a robotic intubation system, remotely controlling a standard video laryngoscope with option of semiautomated intubation or Pentax channelled scope mounted over robotic arm named after astronomer - Johannes Kepler.

10) What is the role of VLS in difficult airway?

Answer- "Video laryngoscope should be used whenever possible."

The DAS (Difficult Airway Society) 2025 guideline strongly supports:

- Early VLS use
- First-pass success
- Fewer attempts
- Continuous oxygenation
- Improved difficult airway safety

Rather than repeated failed direct laryngoscopy attempts.



11) Identify the device and describe in brief



Answer- It is King vision Video laryngoscope.

- Portable, battery-operated video laryngoscope with integrated reusable display and disposable blades.

- Provides indirect glottic visualization with improved view during difficult intubation.

Available with:

- Standard blade
- Channelled blade (guides ETT without stylet)

Useful in:

- Difficult airway
- Cervical spine immobilization
- Emergency/ICU intubation
- Teaching and training

12) Identify the device and describe in brief.



Answer- It is a USB Borescope, it is a low cost device used for intubation, particularly beneficial for patients with potentially difficult airways.

- It can be mounted over a direct laryngoscope blade, providing a clear view and reducing fogging during intubation. Studies have shown that intubation times are comparable between USB borescopes and video laryngoscopes, with the borescope being more satisfying for anesthetists.

- USB borescopes are compatible with various devices, allowing for easy use on PCs, Macs, or Android devices.

- Overall, the USB borescope is a practical and cost-effective option for intubation in challenging airway situations.

DIRECT LARYNGOSCOPE

1) Identify the device and mention its parts



Answer - It is a Macintosh blade Its parts consist of following - Base, Heel, Spatula/tongue, Flange , Web, Tip/beak, Light.

2) Identify the device



Answer - It is Cavallari Jackson Laryngoscope It has a U-shaped handle with straight blade and "O" shaped flange, it has battery based external light source



3) What are types of laryngoscopes?

Answer -

A) Rigid- direct and indirect

i) Direct – classified on the basis of blade type

-Laryngoscopes with straight blade: Guedel, Wisconsin, Miller, etc. -Laryngoscopes with curved blade: Macintosh, Bizzarri-Giuffrida, etc. -Laryngoscopes with features of both blades: Cardiff, Dorges -Laryngoscopes for special purpose: Flexitip, Polio, Oxiport, Tull, etc.

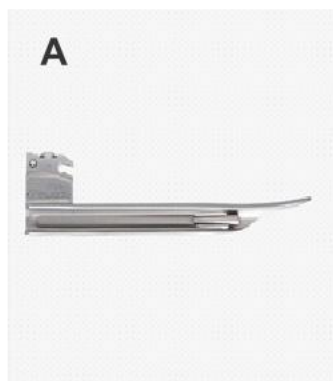
ii) Indirect

Prisms: Belscope, Trueview, Huffman prism -Optical: Bullards, UpsherScope, Augustine Scope -Optical stylet type: Bonfil's retromolar, Shikani's intubating stylet - Video laryngoscopes: Berci DCI, C-Mac, Glidescope, etc

B) Flexible- Fiberoptic bronchoscope

4) Identify the laryngoscope blades in image

A, B and C



Answer -

A - Miller's blade

B - McCoy blade

C - Oxiport blade

5) Identify the laryngoscope blade and mention its features



Answer- It is Polio blade A modification of Macintosh curved blade, it makes an obtuse angle with the handle thus does not impinge on the chest plate of respirator during intubation. Use - Obese patients with large breast, kyphoscoliosis with barrel chest, patients with restricted neck movements due to cervical collar

6) Mention few rigid fibreoptic indirect laryngoscopes

Answer-

a) Bullard laryngoscope

b) UpsherScope laryngoscope

c) Augustine Scope

d) WuScope

7) Identify the device and describe



Answer - It is Choi double angle laryngoscope It was designed to eliminate the drawbacks of Macintosh and miller blade; the straight tongue has two incremental angles -20(proximal) and 30 degrees (distal) The double angled blade can be used to lift epiglottis directly or indirectly, commercially available in adult and paediatric sizes.

8) Mention few laryngoscope blades with special functions

Answer -

- a) Oxiport blade - has channel for oxygen insufflation
- b) Tull(suction) blade - has suction port which extends down to handle and has finger-controlled valve to operate
- c) Polio blade - has obtuse angle with handle, eases use in obese patients with large breasts, barrel chest, patients with restricted neck movements.

9) What are the uses of left-handed Macintosh blade?

Answer -

It has flange on opposite side from the usual Macintosh blade useful for abnormalities of right side of face (e.g.- cleft palate) or oropharynx, left-handed persons, intubation in right lateral position and positioning a tracheal tube on left side of mouth.

10) How to sterilize laryngoscopes?

Answer -

Blades are cleaned with soap and water followed by spirit or chlorhexidine swabbing to disinfect it. Blades can be autoclaved without the light carrier.

SUPRAGLOTTIC AIRWAY DEVICES

1) Who invented Laryngeal mask airway?

Answer- Sir Archie Brain was a British anaesthetist who invented the Laryngeal Mask Airway in the early 1980s. His aim was to create an airway device intermediate between a face mask and an endotracheal tube, allowing easier and less traumatic airway management during anaesthesia.

2) Identify the device and describe in brief.



Answer- Classic Laryngeal Mask Airway is a first-generation supraglottic airway

device used to maintain airway without tracheal intubation.

- Parts: airway tube, inflatable elliptical mask/cuff, aperture bars, pilot balloon, and connector.
- Provides easy insertion with minimal haemodynamic response and less airway trauma compared to endotracheal intubation.
- Commonly used for elective short procedures but does not protect completely against aspiration.

3) What are the features of an ideal supraglottic airway device (SAD)?

Answer- Some of the features are-

- a) Must efficiently seal the upper airway during spontaneous and positive pressure ventilation
- b) Low resistance to respiratory gas flow
- c) should protect the supraglottic airway from upper airway secretions and gastric contents
- d) low incidence of airway morbidity and adverse events
- e) Should be easily accepted by the oropharynx

4) What are the contraindications for SAD use?

Answer -

- a) Aspiration risk – inadequate fasting, intestinal obstruction, emergency surgery
- b) Upper airway obstruction – tumor, abscess, edema, hematoma
- c) Restricted mouth opening – trismus
- d) Disrupted upper airway – facial/airway trauma, caustic burns
- e) Delayed gastric emptying conditions – morbid obesity, pregnancy >14 weeks, opioid use
- f) Stiff lungs/poor compliance – pulmonary fibrosis, status asthmaticus
- g) High airway pressure requirement – leak, gastric insufflation, inadequate ventilation

5) Identify the airway device and describe



Answer - It is LMA Fastrach or Intubating LMA(ILMA) It is designed to act as a conduit for endotracheal intubation . It consists of cuff, airway tube, epiglottic elevating bar, pilot inflation balloon, inflation line, valve, 15mm connector, handle.

6) What is Chandy's manoeuvre?

Answer- It consists of two steps First step- rotating LMA in coronal and sagittal plane in an attempt to find a position that offers least resistance to ventilation. Second step- Grasping the handle firmly and use it to draw LMA forward 2-5 mm in a lifting action without levering on teeth. This manoeuvre increases the seal pressure and aligns the axes of trachea and FETT and facilitates blind intubation.

7) Identify the SAD and mention its parts



Answer - It is I gel supraglottic airway device; Invented by Mohammed Nasir, a Pakistani anaesthetist, now in England. Parts include - Soft non inflatable cuff, epiglottic rest, buccal cavity stabilizer, integral bite block, 15 mm connector, proximal and distal end of gastric channel

8) Identify the SAD and describe its advantages and disadvantages



Answer - It is SLIPA (Streamlined liner of the pharynx airway). It is a latex-free device, shaped like a slipper. It is made of medical grade thermoplastic material stiff enough to facilitate easy insertion.

Advantages -

- Cheap, uncomplicated, disposable device with minimal risk of aspiration.
- It has large empty compartment which has space for collection of almost 50 ml of oropharyngeal secretions , hence protects against aspiration risk

Disadvantages -

- Selecting the correct size is difficult
- First attempt insertion are successful in 75-85% patients

9) Identify this SAD and mention its special features



Answer - **Baska Mask SAD**; Invented by Kanaga and Meena Baska of Australia. Special features include-

1. Non-inflatable self-sealing cuff
2. Seal improves with positive pressure ventilation
3. Very high oropharyngeal leak pressure
4. Dual gastric drainage channels
5. Repositioning tab for airway adjustment

10) Which are the first, second and third generation SADs?

Answer - A) First Generation SADs

(Simple airway tube without gastric drainage channel)

- Classic Laryngeal Mask Airway
- Flexible Laryngeal Mask Airway
- Intubating Laryngeal Mask Airway
- Cobra Perilaryngeal Airway
- Laryngeal Tube

B) Second Generation SADs(With gastric drainage channel and better airway seal)

- ProSeal Laryngeal Mask Airway
- Supreme Laryngeal Mask Airway
- i-gel
- Baska Mask
- Laryngeal Tube Suction II
- Ambu AuraGain

C) Third Generation SADs(Video-assisted / self-guiding intubating SADs)

- SaCo Video Laryngeal Mask
- Video laryngeal masks with integrated visualization systems



11) Identify this device and describe it.



Answer- It is Ambu AuraGain LMA. It is a single use second generation SAD with gastric channel. It is designed with an anatomical curve to facilitate rapid placement which makes it differ from Proseal LMA and provides a high seal pressure to ensure patient safety during ventilation. It usually comes in green colour. It can serve as a conduit for endotracheal intubation. Offers high pressure seal upto 40 cms H₂O.



12) Identify these SADs



Answer -

- A- LMA Supreme
- B- LMA gastro
- C- LMA Protector
- D- Cobra PLA(Peri laryngeal airway)

13) Describe LMA Protector

Answer: It is a second generation, anatomically curved, single use perilaryngeal – sealer type of SGD. It has a large volume pharyngeal chamber with dual gastric drainage channels. It is designed to functionally separate respiratory and gastrointestinal systems. It is made of 100% silicone. The elongated cuff of this device helps in achieving a laryngeal seal due to elliptical airway tube and second seal is formed at the upper esophageal sphincter. The cuff has a built-in pressure indicator which allows for continuous monitoring of cuff pressure. The integrated cuff pressure indicator reduces risks of over inflation and under inflation.

14) Describe Blockbuster LMA

Answer: It is a second- generation SGD with intubation capability and access for gastric drainage. It is made up of silicone and the airway tube is angulated more than >95 degree which gives it a shape similar to oropharyngeal curve. This makes intubation easier through this device. It has an integrated bite block and a four- way connector for fixing the LMA after insertion. The gastric outlet of Blockbuster LMA is bevel shaped allowing easier insertion of gastric tube. The mask portion of the LMA has a built – in guidance device which directs the ETT toward larynx at an angle of 30 degree . It has a reinforced ETT with a soft angulated tip flexible tip, which faces posteriorly.

FIBROPTIC INTUBATION

1) Give a brief history of use of fibrescope for tracheal intubation

Answer: Dr Peter Murphy was the first to use a flexible fibrescope to perform tracheal intubation. He performed the intubation as a registrar at the National Hospital, Queen Square in 1967.

- Eyepiece
- Control section
- Insertion cord
- Universal cord for light transmission
- Light source

3) What is SAGO technique and how it is done?

Answer: Spray-as-you-go (SAGO) technique is a method of topical airway anesthesia used during awake fiberoptic intubation to numb the airway while advancing the bronchoscope.

A fiberoptic bronchoscope has a working channel. Through this channel, local anesthetic (usually Lidocaine 2–4%) is sprayed directly onto airway structures as the scope advances.

The anesthetic is sprayed step-by-step on:

Nasal/oral mucosa (entry passage)

Oropharynx

Epiglottis

Vocal cords

Trachea and carina

Each spray is usually followed by waiting 30–60 seconds for anesthesia to work before advancing further.

It helps to:

- Suppress gag reflex
- Reduce coughing
- Minimize laryngospasm
- Improve patient comfort
- Allow smoother passage of the bronchoscope and endotracheal tube



13th JUNE 2026

04:20PM - 04:55PM

ANAESTHESIA EQUIPMENT

Dr. Prasad K Kulkarni

Professor

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SPINAL NEEDLE

1. Who introduced spinal anesthesia?

A. Leonard Corning (1885).

2. Who introduced the lumbar puncture technique for CSF drainage?

A. Heinrich Irenaeus Quincke (1891).

3. Who performed the first spinal anesthesia?

A. August Bier (1898), on himself was accidental.

4. Who introduced spinal analgesia in children?

A. Bainbridge (1900).

5. Who described the atraumatic spinal needle?

A. Greene (1926).

6. Who developed the pencil-point spinal needle?

A. Whitacre (1951).

7. Who modified the Whitacre needle into Sprotte needle and what modification?

A. Sprotte(1987) modified the Whitacre needle by giving it a more rounded (ogival) tip and a larger, positioned lateral opening behind the Tip to improve flow while preserving atraumatic properties.

8. What are the parts of a spinal needle?

A.

- Hub
- Cannula (shaft with tip)
- Stylet

9. What is the function of the stylet?

A. It occludes the lumen to prevent tissue coring.

10. What material is used for spinal needles?

A. Stainless steel

11. What is the standard spinal needle?

A. Quincke-Babcock needle.

12. What are the main types of spinal needles?

A.

- Quincke (cutting)
- Whitacre (pencil point)
- Sprotte (modified pencil point)
- Greene
- Pitkin

13. What are cutting needles? Give example.

A. Needles that cut dura → Example: Quincke needle.

14. What are atraumatic needles? Give examples.

A. Needles that separate dural fibers →

- Whitacre
- Sprotte
- Greene

15. Why are pencil-point needles preferred?

A.

- Separate dural fibers
- Smaller puncture hole
- Reduced CSF leak
- Lower incidence of PDPH (Post-dural puncture headache)

16. What is PDPH?

A. PDPH is a bilateral frontal, retro-orbital, or occipital headache radiating to the neck, which may be throbbing or constant, associated with photophobia and nausea, and is postural-worse on sitting/standing and relieved on lying flat, typically occurring 12–72 hours after dural puncture.

PDPH, results from CSF leakage through a dural defect resulting intracranial hypotension causing traction on meninges, dura, tentorium, blood vessels, and cranial nerves, causing headache; cranial nerve traction may lead to diplopia (VI nerve) and tinnitus.

17. What is the range of spinal needle sizes?

A. 16G to 30G.



18: What is the “needle-through-needle” technique?

A: It is a combined spinal–epidural (CSE) technique in which a fine spinal needle is passed through a Tuohy epidural needle into the subarachnoid space to administer spinal anesthesia

19. What are the common lengths of spinal needles?

- A.
- 8–10 cm (normal)
 - 15 cm (obese patients)

20. What is the purpose of a spinal introducer?

- A.
- Prevent bending of fine needles
 - Reduce infection
 - Facilitate insertion

21. Name types of introducers.

- A.
- Sise introducer
 - Corning introducer
 - Lundy introducer



22: Why are spinal introducers required?

A: To support and guide fine spinal needles, preventing bending and aiding passage through tough tissues.

23. Who first performed continuous spinal anesthesia?

A. Dean (1907).

24. Who introduced ureteral catheter for spinal use?

A. Edward Tuohy (1944).

25. What is the advantage of continuous spinal anesthesia?

A. Allows controlled and repeated dosing.

26. Which needle is most commonly used today?

A. Whitacre needle.

27. Which needle is preferred in obstetrics?

A. Whitacre needle.

28. Why were disposable needles introduced?

A. To reduce contamination and neurological complications.

29. Hub of a spinal needle is called as?

A. The hub of a spinal needle is commonly referred to as the Luer-lock hub (connector).

30. CSFID (cerebral spinal fluid identification) system helps in improving:



- A. Epidural catheter size
B. CSF flashback visibility
C. Drug viscosity
D. Needle length

Ans: B



31



A. What type of needle is shown?

Atraumatic spinal needle (Atrauncan)

B. What is the special feature of this needle?

- o Two-zone bevel design
- o Combines cutting + non-cutting tip → reduces tissue trauma

C. What is the main advantage?

Reduced risk of Post Dural Puncture Headache

D. Why does it reduce PDPH?

Causes less dural fiber damage ,smaller hole ,less CSF leakage

32. MATCH THE FOLLOWING

Gauge

Hub Color

A. 16G

1. Pink



B. 17G

2. Black



C. 18G

3. White



D. 19G

4. Orange



E. 20G

5. Purple



F. 21G

6. Deep Green



G. 22G

7. Yellow



H. 23G

8. Cream



I. 24G

9. Violet



J. 25G

10. Deep Blue



K. 26G

11. Brown



L. 27G

12. Grey



A-3,B-5,C-1,D-8,E-7,F-6,G-2,H-10,I-9,J-4,
K-11,L-12

33. MATCH THE FOLLOWING

Needle Type

Tip Characteristic

1. Pencil-point tip



A. Quincke needle



2. Cutting bevel tip



B. Whitacre needle



3. Modified pencil-point
with side opening

C. Sprotte needle



Needle Type

Tip Characteristic



A - 2,B - 1,C - 3



EPIDURAL NEEDLES

1. MATCH THE FOLLOWING

Epidural Needle	Characteristic
A. Tuohy needle	1. Straight tip
B. Crawford needle	2. Standard curved (Huber) tip
C. Coudé needle	3. Slightly less curved tip
D. Hustead needle	4. More pronounced curved tip



A – 2, B – 1, C – 4, D – 3

2. IDENTIFY PARTS



1. Epidural Catheter
2. Luer Lock Syringe Or LOR Syringe
3. Epidural Needle
4. "Snap" Catheter Connector
5. Epidural-Flat Filter 0.2 µm MCQs

1. Who first described the epidural space?

- A. Pagés
- B. Tuohy
- C. Corning
- D. Bromage

Ans: C – James Leonard Corning

2. First use of epidural anesthesia in humans?

- A. Tuohy
- B. Pagés
- C. Curbelo
- D. Sprotte

Ans: B – Fidel Pagés

3. Most commonly used epidural needle?

- A. Sprotte
- B. Quincke
- C. Tuohy
- D. Weiss

Ans: C – Tuohy needle

4. Angle of Tuohy needle tip?

- A. 5–10°
- B. 10–15°
- C. 15–30°
- D. 30–45°

Ans: C

5. Main purpose of curved bevel in Tuohy needle?

- A. Increase sharpness
- B. Reduce pain
- C. Direct catheter away from dura
- D. Increase flow

Ans: C

6. Who introduced epidural catheter?

- A. Bromage
- B. Curbelo
- C. Tuohy
- D. Pagés

Ans: B – Manuel Curbelo



7. Most common complication of epidural needle?

- A. Hemorrhage
- B. Infection
- C. Dural puncture
- D. Nerve injury

Ans: C

8. Preferred LOR medium?

- A. Air
- B. Saline
- C. Blood
- D. CSF

Ans: B

9. Pore size of epidural filter?

- A. 0.1 μ
- B. 0.22 μ
- C. 1 μ
- D. 5 μ

Ans: B

10. Filling volume of epidural-flat filter is:

- A. 0.2 ml
- B. 0.3 ml
- C. 0.45 ml
- D. 1 ml

Ans: C (0.45 ml)

Maximum pressure resistance of epidural filter:

- A. 3 bar
- B. 5 bar
- C. 7 bar

11. Maximum pressure resistance of epidural filter:

- A. 3 bar
- B. 5 bar
- C. 7 bar
- D. 10 bar

Ans: C (7 bar)

12. Length of epidural catheter is:

- A. 50 cm
- B. 75 cm
- C. 100 cm

D. 120 cm

Ans: C (100 cm / 1000 mm)

13. Threading assist guide in catheter helps in:

- A. Drug injection
- B. Sterilization
- C. Easy catheter insertion
- D. Measuring depth

Ans: C

14. Available sizes of Paed epidural needles include:

- A. 14G, 16G
- B. 16G, 18G, 20G
- C. 18G, 20G, 22G
- D. 20G, 22G, 24G

Ans: C

15. Markings on PEADIATRIC epidural needles are present at:

- A. 1 mm intervals
- B. 5 mm intervals
- C. 1 cm intervals
- D. 2 cm intervals

Ans: B

16. Epidural catheter markings are typically present at:

- A. Every 1 cm only
- B. Specific intervals such as 5, 6, 7, 8, 9, 10, 15, 20 cm
- C. Every 2 cm only
- D. Only at the tip

Ans: B

17. During epidural placement, ideal catheter length inside epidural space is:

- A. 1–2 cm
- B. 2–3 cm
- C. 3–5 cm
- D. 6–8 cm

Ans: C, This provides secure fixation and reliable analgesia while reducing complications such as catheter migration, vascular/foraminal placement that may occur with excessive advancement.



18. General rule for epidural catheter passage is:

- A. Same size only
- B. Larger catheter through smaller needle
- C. Smaller or equal size catheter through larger needle
- D. Any size fits

Ans: C

19. Which combination is correct?

- A. 16G catheter → 18G needle
- B. 20G catheter → 18G needle
- C. 18G catheter → 20G needle
- D. 19G catheter → 20G needle

Ans: B

20. The tip of a standard epidural catheter is usually:

- A. Open with no holes
- B. Closed with a single hole
- C. Closed with multiple lateral holes
- D. Sharp with cutting edge

Ans: C

21. Common number of lateral holes in a multi-orifice epidural catheter:

- A. 1
- B. 2
- C. 3
- D. 8

Ans: C

22. Advantage of multi-orifice epidural catheter:

- A. Less drug spread
- B. Uniform drug distribution
- C. Increased stiffness
- D. More trauma

Ans: B

23. Closed tip with lateral holes is designed to:

- A. Prevent drug leakage
- B. Reduce trauma and allow lateral spread
- C. Increase resistance
- D. Improve rigidity

Ans: B

24. Which catheter type provides more uniform analgesia?

- A. Single end-hole
- B. Multi-orifice
- C. Metal catheter
- D. Rigid catheter

Ans: B

COMBINED SPINAL NEEDLE

1: Who first described the combined spinal–epidural technique? What was the method?

A: Soresi (1937), who described it as “episubdural.” He first injected local anesthetic epidurally, then advanced the same needle intradurally to give spinal dose.

2: Who introduced the double-segment (double-space) technique? What was the method?

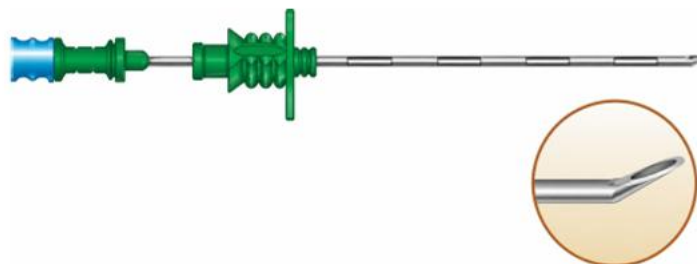
A: Curelaru (1979). He used two separate punctures—epidural catheter placed first, then spinal injection at a different interspace.

3: Who described the use of double-space CSE for cesarean section?

A: Bainbridge (1981).

4: Who described the needle-through-needle technique? What is needle-through-needle technique?

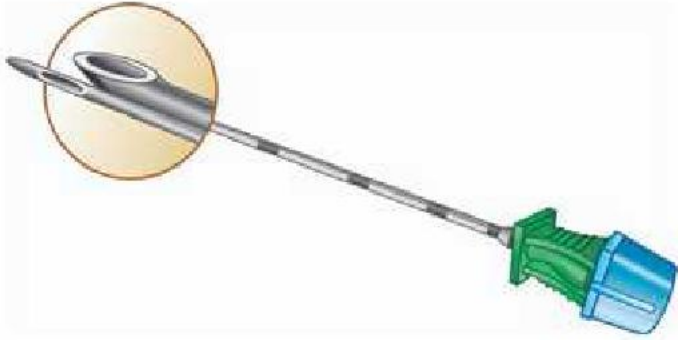
A: Coates and Mumtaz (1982). A spinal needle is passed through a Tuohy epidural needle into the subarachnoid space after epidural needle placement.



5: Who developed a special CSE needle design? What is special about CSE needle?

A: Hanaoka (1986). It has a “back hole” allowing spinal needle passage through the epidural needle.





6: What is the standard needle used in needle-through-needle technique?

A: Tuohy epidural needle with a fine spinal needle (e.g., 25G–27G).

7: What is advantage of needle-through-needle technique?

A: Single-site puncture, faster onset, and ability to extend analgesia via epidural catheter.

8: Name types of combined spinal–epidural needles.

A:

- Tuohy with additional aperture (back eye)
- Double-lumen Tuohy needle
- Portex “Secure” CSE set
- B Braun CSE set
- Pajunk EpiSpin Lock system

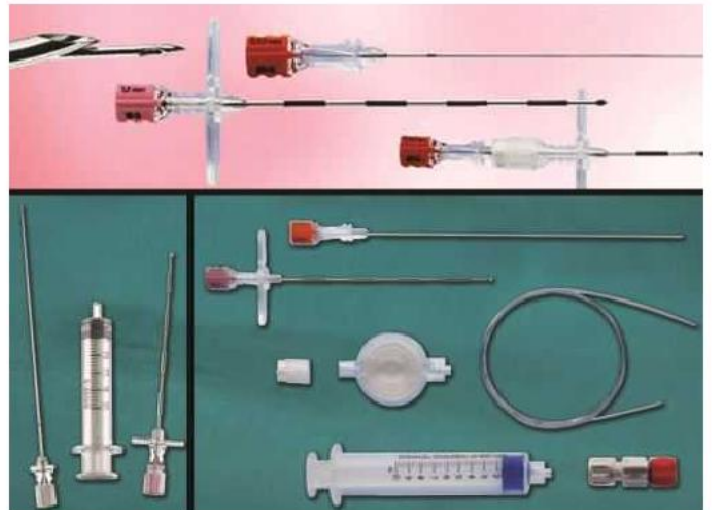
9: What is special about Portex “Secure” CSE needle?

A: It has graduated markings and locking mechanism to prevent spinal needle displacement and improve accuracy.



10: What is EpiSpin Lock system?

A: A modern CSE set with fixation system, depth indication, and lockable spinal needle position for precision puncture.



SIMPLE FACE MASK

1. Identify this device and describe its components.



A. This is a Simple Face Mask (Hudson mask). It is a low-flow, variable-performance oxygen delivery device. It consists of:

- A clear plastic body (for visual monitoring of vomitus/condensation).
- A metal nose clip for anatomical fit.
- Lateral exhalation ports (holes) on both sides.
- Inlet port at the base for oxygen tubing.

2. Classify oxygen therapy devices

A. There are number of methods of classifying oxygen therapy devices. They can be classified as follows-



- Performance of device: Fixed (Venturi) or Variable (nasal prongs, face mask) performance devices
- Flow delivered by the device: Low flow (nasal prongs, face mask) or High flow (Venturi) devices
- Degree of dependency: Low dependency (face mask, nasal prongs), medium dependency (CPAP mask and equipment), high dependency (noninvasive or invasive positive pressure ventilation).

3. Why is it called a Variable Performance device?

A. Because the actual FiO_2 delivered to the patient depends on the patient's ventilatory parameters (Inspiratory Flow Rate, Tidal Volume, and Respiratory Rate) rather than just the flow set on the flowmeter.

4. What is the FiO_2 range provided by this mask, and at what flow rates?

A. It provides an FiO_2 of approximately 35% to 55% (0.35–0.55). The flow rate must be set between 5–10 L/min.

5. Why is it contraindicated to use a flow rate of less than 5 L/min with this mask?

A. At flows < 5 L/min, the mask's volume acts as additional dead space. The flow is insufficient to flush out exhaled CO_2 from the mask, leading to CO_2 rebreathing.

6. How do the lateral ports on the mask function during the respiratory cycle?

A. During inspiration, the patient draws oxygen from the inlet and entrains room air through the side ports and during expiration, exhaled gases are vented out through these ports.

7. Identify the dead space in this device. How much is it?

A. The internal volume of the mask serves as the dead space. In an adult simple face mask, this is approximately 100–200 mL.

8. What are the disadvantages or complications of using an simple face mask?

A.

- Discomfort: Pressure effects on the nose bridge and ears.
- Aspiration risk: If the patient vomits, the mask can trap it.
- Interference: Difficulty in talking or eating.
- Eye irritation: Oxygen flow can leak from the top and dry the corneas if not fitted properly.

9. How does the patient's Peak Inspiratory Flow Rate (PIFR) affect the FiO_2 in an SFM?

A. There is an inverse relationship. If a patient is in respiratory distress and has a high PIFR (e.g., 30–40 L/min), they will entrain more room air through the side ports to meet that demand. This dilutes the 5–10 L/min of oxygen coming from the flowmeter, thereby decreasing the delivered FiO_2 .

10. What is the significance of the Metal Clip on the bridge of the nose?



A. It serves two purposes:

- Fit: To prevent oxygen from leaking into the patient's eyes (which can cause drying and corneal irritation).
- Dilution: To ensure that the oxygen flow is directed toward the nose and mouth rather than escaping out the top, which would further lower the FiO_2 .



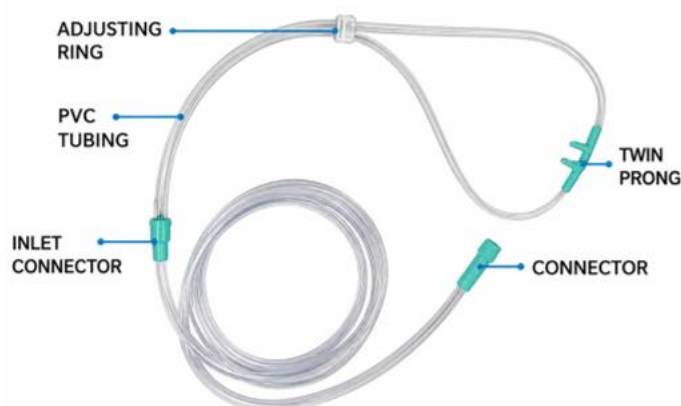
.11. Mention two specific clinical situations where an SFM is the preferred choice.

A.

- Post-operative recovery (PACU): For patients stable and recovering from anesthesia who need mild supplemental oxygen.
- Transporting a stable patient: When the patient requires more than what prongs can give but is not so critical as to need a reservoir bag or Venturi.

NASAL PRONGS

1. Identify this device and describe its classification.



A. This is a Nasal Cannula (Nasal Prongs). It is classified as a Low-Flow, Variable-Performance oxygen delivery device.

2. Explain the Variable-Performance nature of this device.

A. The FiO_2 delivered is not fixed. It depends on the patient's Inspiratory Flow Rate and Minute Ventilation. When the patient's inspiratory flow exceeds the flow from the cannula, they must entrain room air (21% O_2) to make up the difference, thus diluting the oxygen concentration.

3. How do you calculate the estimated FiO_2 for a Nasal Cannula?

A. We use the "Rule of Fours." Starting with room air (20-21%), every 1 L/min increase in oxygen flow adds approximately 4% to the FiO_2 .

- 1 L/min approx 24%
- 2 L/min approx 28%
- 3 L/min approx 32%
- 4 L/min approx 36%
- 5 L/min approx 40%
- 6 L/min approx 44%

4. Why is the maximum recommended flow rate for a standard nasal cannula capped at 6 L/min?

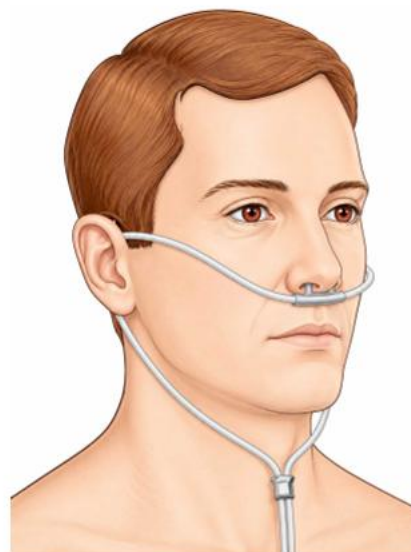
A. There are two main reasons:

- Anatomical Reservoir: The nasopharynx (which acts as the oxygen reservoir) has a limited capacity. Flows above 6 L/min do not significantly increase the FiO_2 because the reservoir is already saturated.
- Patient Comfort: High flows cause significant mucosal drying, irritation, and potential epistaxis.

5. Is a humidifier necessary for a patient on nasal prongs at 2 L/min?

A. Generally, no. For low flows (< 4 L/min), the patient's own upper airway (nasopharynx) provides sufficient humidification. Humidification is typically recommended only when flows exceed 4 L/min or for long-term use.

6. How do you properly measure and fit the nasal prongs?



A. The prongs should be inserted into the nares with the curved tips pointing downward, following the



natural anatomy of the nasal floor. The tubing should be looped over the ears and secured under the chin.

7. Compare Nasal Prongs to a Simple Face Mask (SFM). What are the advantages of prongs?

A.

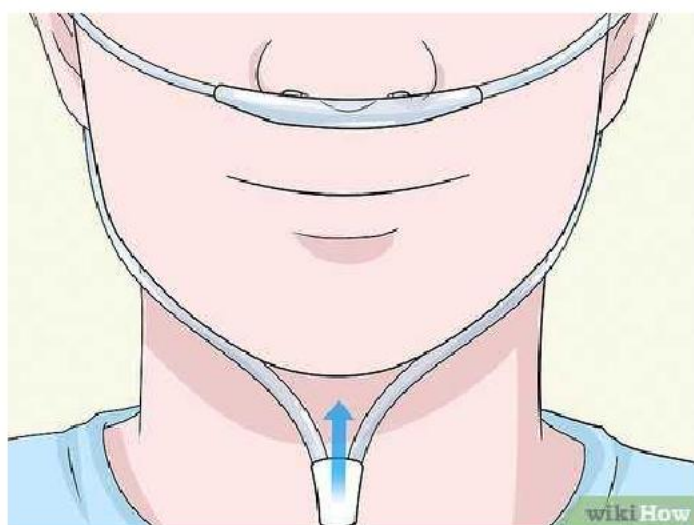
- Patient Compliance: They are better tolerated for long periods.
- Functionality: The patient can eat, drink, and speak without interrupting oxygen therapy.
- Dead Space: There is no risk of CO₂ rebreathing because there is no mask body to act as dead space.

8. What materials are typically used to manufacture nasal prongs, and why?

A. They are usually made of Medical Grade Polyvinyl Chloride (PVC). PVC is used because it is:

- Thermoplastic: It softens at body temperature, making it more comfortable for the patient.
- Clear: Allows for the detection of blockages (e.g., mucus/blood).
- Latex-free: To prevent allergic reactions.

9. Identify the slide adjustment or toggle on the tubing. What is its purpose?



A. The slide adjustment is used to tighten the tubing under the chin. This ensures the prongs remain correctly oriented within the nares and prevents the

tubing from slipping off the ears, which is a common cause of equipment failure in restless patients.

10. How do you verify the flow of oxygen at the bedside if you suspect the tubing is kinked?

A.

- Acoustic: Listen for the "hiss" near the prongs.
- Tactile: Direct the prongs toward your own hand or cheek to feel the air.
- Visual (Water Test): Submerge the tips in a cup of sterile water; bubbling confirms flow.

VENTURI MASK

1. Identify this device and explain the physics of its function.



A. This is a Venturi Mask, a high-flow, fixed-performance oxygen delivery system. It works based on the Bernoulli Principle:

- Oxygen is forced through a narrow orifice, which increases its velocity.
- This creates a drop in lateral pressure (the Venturi Effect).
- This low pressure "entrains" or sucks in room air through side ports.
- The ratio of oxygen to entrained air remains constant, ensuring a fixed FiO₂.



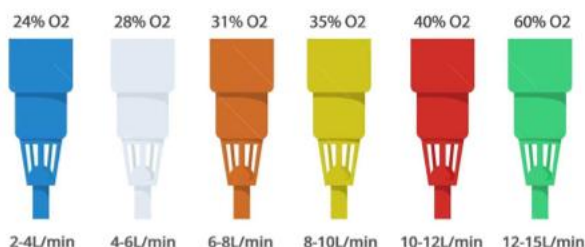
2. Who is credited with the clinical invention of the Venturi mask?



A. The mask was created by the British Physician Earl James Moran Campbell. It was named after the Italian physicist Giovanni Battista Venturi who described the principal of increased velocity of a gas resulting in lower pressures, Campbell incorporated Venturi's principle into the oxygen delivery facemask.

3. Identify the color-coded valves and their significance.

Venturi Mask Valves



A. Each color corresponds to a specific FiO₂ and requires a specific minimum flow rate from the flowmeter to ensure the total gas flow meets or exceeds the patient's peak inspiratory flow.

- Blue: 24% (2 L/min)
- White: 28% (4 L/min)

- Yellow: 35% (8 L/min)
- Red: 40% (10 L/min)
- Green: 60% (15 L/min)

4. Why is there a minimum flow written on each valve?

A. To ensure that the Total Gas Flow (Oxygen + Entrained Air) is higher than the patient's Peak Inspiratory Flow Rate (PIFR), which is usually 25–30 L/min. If the total flow is less than the PIFR, the patient will draw in extra room air, and the FiO₂ will no longer be fixed.

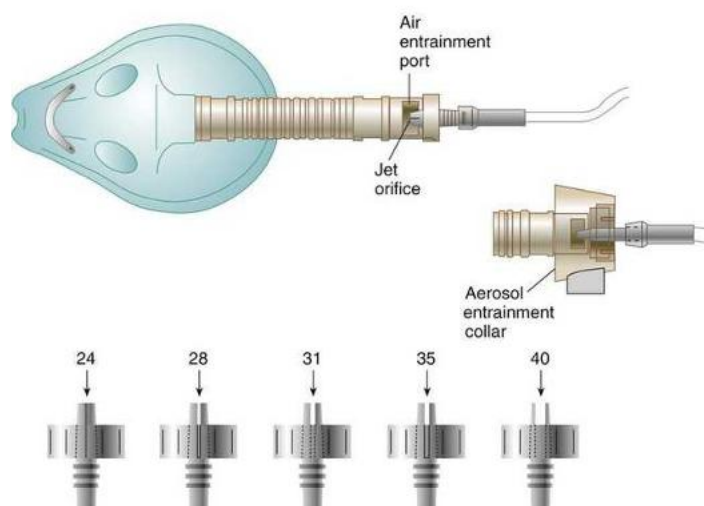
5. What happens to the FiO₂ if the entrainment ports are covered by a bedsheet?

A. If the ports are blocked, less room air is entrained. Therefore, the FiO₂ increases, but the total gas flow decreases. The device essentially reverts to behaving like a simple face mask.

6. Why is this the Gold Standard for COPD patients?

A. COPD patients with chronic hypercapnia rely on their "hypoxic drive" (low PaO₂) to breathe. If we give them too much oxygen (variable FiO₂ from a simple mask), we may abolish this drive, leading to respiratory depression -> hypercarbia -> respiratory arrest. The Venturi allows us to titrate oxygen precisely (e.g., exactly 24% or 28%).

7. How does the size of the Jet Orifice inside the Venturi valve relate to the FiO₂?



A. There is an inverse relationship.

- A smaller orifice creates a higher-velocity jet, which creates a greater pressure drop and entrains more room air, resulting in a lower FiO_2 (e.g., 24%).

- A larger orifice creates a lower-velocity jet, entraining less room air, resulting in a higher FiO_2 (e.g., 60%).

8. What is the Air Entrainment Ratio? Calculate the ratio for 24% and 40%.

A.

The ratio determines how many parts of air are mixed with one part of oxygen.

$$\text{Ratio} = \frac{100 - FiO_2}{FiO_2 - 21}$$

- For 24%: $\frac{100 - 24}{24 - 21} = \frac{76}{3} =$ approx 25:1 (Total flow is very high).

- For 40%: $\frac{100 - 40}{40 - 21} = \frac{60}{19} =$ approx 3:1 (Total flow is lower).

9 What is the Total Flow provided by a 24% Venturi valve set at 2 L/min? Why is this number significant?

A. For 24%, the air-to-oxygen ratio is 25:1.

- If the O_2 flow is 2 L/min, the entrained air is 2 times 25 = 50 L/min.

- Total Flow = 50 + 2 = 52 L/min.

- Significance: This flow is much higher than a normal person's peak inspiratory flow rate (approx. 30 L/min), ensuring the patient only breathes the gas delivered by the mask.

NON REBREATHING MASK

1. What is this device?



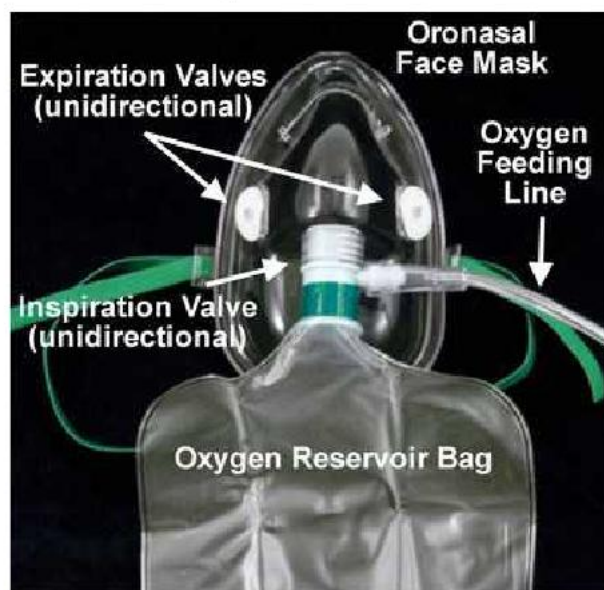
A. This is a Non-Rebreathing Mask (NRBM) used for delivering high concentration oxygen to spontaneously breathing patients.

2. Why is it called a non-rebreathing mask?

A. Because the system is designed to minimize rebreathing of expired gases using One-way valves, Reservoir bag and High oxygen flow.

Expired air escapes through side ports and does not significantly enter the reservoir bag.

3. What are the components of an NRBM?



ANSWER –

- Face mask
- Reservoir bag
- Oxygen inlet tubing
- One-way inspiratory valve between mask and bag
- Expiratory one-way valves/side ports
- Elastic head strap
- Metal nose clip

4. Explain the working principle of NRBM.

A.

- Oxygen flows continuously into reservoir bag.
- During inspiration, patient inhales oxygen from Reservoir bag and Fresh gas inflow
- One-way valve prevents exhaled gas entering reservoir bag.
- Expired gas exits through side ports.
- High flow minimizes entrainment of room air.



5. What oxygen concentration can NRBM deliver?

A. Approximately 60–90% FiO_2 , depending on Oxygen flow rate, Mask fit and Patient's minute ventilation.

6. What is the minimum oxygen flow required?

A. Usually 10–15 L/min to keep the reservoir bag partially inflated during inspiration.

7. Why should reservoir bag not collapse completely?

A. Collapse indicates inadequate flow and may lead to Room air entrainment, Reduced FiO_2 and Rebreathing.

8. What happens if oxygen flow is too low?

ANSWER – It will cause

- Bag collapse
- Rebreathing
- Lower FiO_2
- Increased work of breathing

9. What are disadvantages of NRBM?

A. Few disadvantages are -

- Claustrophobia
- Poor tolerance
- Dryness of airway
- Difficulty eating/speaking
- Pressure sores
- Variable FiO_2 if poor fit

10. What are the contraindications or limitations?

A.

- Apnea
- Severe respiratory fatigue
- Need for ventilatory support
- Uncooperative patient

11. Why is preinflation of reservoir bag important?

A. Preinflation ensures:

- Immediate availability of high concentration oxygen
- Prevention of room air entrainment during first breath
- Avoidance of hypoxia at initiation

12. How do you preinflate the reservoir bag?

A.

- Connect mask to oxygen source
- Start oxygen flow at 10–15 L/min
- occlude mask opening lightly if required
- Allow reservoir bag to fill fully before applying to patient

13. What will happen if NRBM is applied without preinflation?

A. Patient initially breathes room air which leads to Reduced FiO_2 delivery, possible hypoxemia and delayed oxygenation

4. How much should the reservoir bag remain inflated during inspiration?

A. It should remain at least One-third to one-half inflated

PEDIATRIC OXYGEN DELIVERY DEVICES

1. What are the commonly used pediatric oxygen delivery devices?

A. Nasal prongs/cannula, Simple face mask, Non-rebreathing mask, Venturi mask, Oxygen hood (head box), High-flow nasal cannula (HFNC), CPAP devices

2. What is an oxygen hood?



A. An oxygen hood (head box) is a transparent enclosure placed around the infant's head to deliver humidified oxygen at controlled concentrations.

3. What age group commonly uses oxygen hood?

A. Mainly in Neonates, Infants and Small children who cannot tolerate masks or nasal devices

4. What is the minimum flow rate required in an oxygen hood?

A. Usually Minimum 7–10 L/min. This prevents:

- Carbon dioxide accumulation
- Rebreathing

5. Why is high flow important in oxygen hood?

A. To Wash out exhaled CO_2 , maintain stable FiO_2 and Prevent rebreathing.

6. Why should humidification be used?

A. Because prolonged oxygen therapy may cause Drying of airway mucosa and Heat loss in neonates. Humidification maintains airway moisture and temperature.

7. What are indications for oxygen hood?

A.

- Neonatal hypoxemia
- Respiratory distress syndrome
- Transient tachypnea of newborn
- Post-extubation oxygen therapy
- Pneumonia
- Congenital heart disease support

8. What are advantages of oxygen hood?

A.

- Better tolerated by infants
- Delivers high FiO_2
- Noninvasive
- Minimal facial irritation
- Allows easy observation of child

9. What are disadvantages of oxygen hood?

A.

- Difficult nursing access
- Noise inside hood
- Oxygen consumption is high
- Difficulty maintaining constant temperature
- CO_2 accumulation if flow inadequate

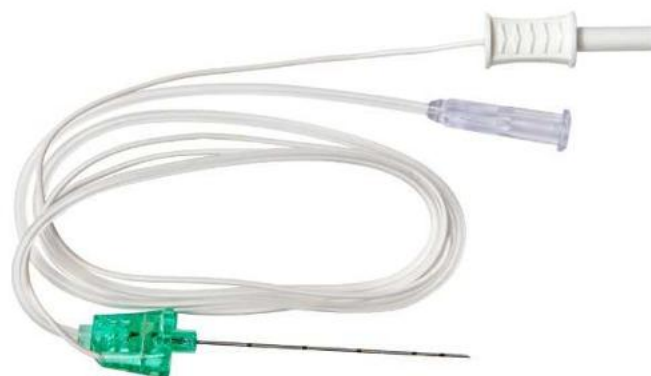
10. What are hazards of excessive oxygen administration in neonates?

A.

- Retinopathy of prematurity (ROP)
- Bronchopulmonary dysplasia
- Oxygen toxicity
- Absorption atelectasis

STIMULATOR NEEDLE

1. Identify this needle and describe its unique features.



A. This is an insulated nerve stimulator needle. Its key features include:

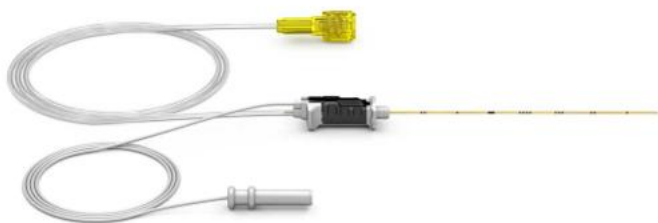
- Insulation: The entire shaft is coated with a non-conductive material (usually Teflon or technical polymers).
- Conductive Tip: Only the very tip of the needle is uninsulated, acting as a point source for the electrical current.
- Electrical Extension: A side lead (wire) with a female connector to attach to the Peripheral Nerve Stimulator (PNS).
- Injection Tubing: A clear side-port tubing to allow for aspiration and injection without moving the needle (immobile needle technique).

2. What is the purpose of the insulation?

A. Insulation ensures that the electrical current is concentrated at the distal tip only. This allows for precise localization of the nerve. If the whole shaft were conductive, stimulation could occur anywhere along the needle, leading to inaccurate needle-to-nerve distance estimation.



3. Which electrode (Positive or Negative) should be connected to the needle? Why?



ANSWER – The Negative electrode (Cathode/Black) should always be connected to the needle.

- Reason: The cathode is more effective at depolarizing the nerve membrane because it decreases the threshold required for an action potential.
- Connecting the positive lead (Anode) to the needle would require a much higher current (3–4 times higher) to get the same motor response, making the block less precise and potentially more painful.

4. Explain the relationship between current (mA) and needle-to-nerve distance.

A. This is governed by Coulomb's Law $E = K * Q / r^2$.

- As the needle tip (r) gets closer to the nerve, the current (Q) required to elicit a motor response decreases.
- A motor response at 0.3 to 0.5 mA generally indicates the needle tip is in close proximity to the nerve, but not intraneural.

5. Differentiate between a Short Bevel and Long Bevel needle in the context of nerve blocks.



ANSWER –

- Short Bevel (usually 30–45°): Preferred for nerve blocks. It provides better tactile feel as it passes through fascial planes (the pop) and is theoretically less likely to penetrate the perineurium or cause nerve fascicle laceration compared to a sharp, long-bevel needle.
- Long Bevel (usually 14–18°): Standard IV needles. These are sharper but have a higher risk of transfixing a nerve.

6. What is Injection Pressure, and why is it monitored during needle use?

A. Injection pressure is the resistance felt during the administration of local anesthetic.

- High pressure (>15–20 psi) suggests intraneural or intrafascicular needle placement.
- Modern needles are often used with Pressure Monitoring devices (like the B-Smart) to prevent nerve injury caused by high-pressure injections.

7. Why do we use a Clear extension tubing attached to the needle?



A. To visualize blood (inadvertent vascular puncture).

8. What are the advantages of a nerve locator?

A. A nerve locator is helpful because:

- It has a higher success rate.
- Can be used along with Ultrasound guidance



- There is better localization of nerve trunks in difficult situations.
- There are less chances of nerve damage.
- Toxic dose of local anesthetic can be avoided by giving titrated volume. • Blocks can be performed in pain clinics and rehabilitation centers.

9. What are the limitations of a nerve locator?

A. A nerve locator has the following limitations:

- It is only applicable to peripheral nerves (not relevant to central axis blockade).
- The aim is to stimulate motor nerves; hence, its use is limited to mixed peripheral nerves.
- Equipment costs.
- It cannot be used after paralysis with neuromuscular blocking drugs.
- PNS should not be used as a substitute for the lack of anatomical knowledge of the nerves that are being blocked.

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13th JUNE 2026

02:35PM - 03:05PM

Burns patient with neck contracture posted for contracture release**Dr Sripada G Mehandale, Dr Keerthy Manoj**

K S Hegde Medical Academy (KSHEMA), Nitte DU Deralakatte, Mangaluru

1. INTRODUCTION

Definition: Contractures are the most prevalent postburn issue managed by reconstructive surgeons. They consist of tight, shortened scar tissue resulting from a tissue deficit.

Impact on Mobility: These contractures typically form over joints, restricting movement, or cause physical deformities by pulling on mobile anatomic structures.

Depth of Involvement: While often associated with the skin, contractures can extend deeper to involve **muscle, fascia, and joint structures**, which may also undergo shortening or fibrosis.

Anatomical Prevalence: They occur most frequently on **flexor surfaces**. This is attributed to the fact that flexor muscles (anti-gravity) are generally stronger, and the flexed position is frequently the "position of comfort" for patients.

1. CHALLENGES

Involvement of the face and neck results in scar contracture in a post burn patients which present a distinctive challenge when it comes to airway management

- Underlying thick fibrous scar can deform the larynx and the mandible.
- Severe mento-sternal contracture can restrict the cervical motion in all directions, making the sniffing position not feasible (figure 1)



Figure 1. Severe contracture neck resulting in a fixed flexion of the neck leading to restriction of movements in all directions. A. Fixed flexion, almost mentum to sternum position. B. The x-ray image showing the position of head and neck in the same position. C. Frontal view highlights the fibrous band in the midline. The stoma of the mouth is difficult to identify. Also, note the atresia of the nostrils due to fibrosis

- Posterior displacement of the mandible maybe present.
- Facial burns during infancy can lead to underdevelopment of jaw- micrognathia.
- Obliterated nasal passages and decreased oropharyngeal space (figure 1C).
- Traction forces cause by scar contracture may pull the laryngeal structures anteriorly or laterally.
- History of inhalation burns could indicate stenosis of the trachea.

3. PREOPERATIVE EVALUATION

History of the cause of burns, duration and previous surgery under anaesthesia are important. Cause of burns may point to internal burns: acid ingestion – pharynx and hypopharynx may be involved. It may cause severe distortion of airway.

History of snoring – difficult mask ventilation following induction anaesthesia.

History of inhalational injury – tracheal stenosis and may hamper tracheal tube placement.



Previous anaesthesia records if available may yield information about airway management. However, the disability may progress with time: previous successful management may not guarantee success this time! Airway examination assesses anticipated difficulty with ventilation, use of supraglottic device, intubation and surgical airway (figure 1C). There may be involvement of nostrils and lips leading to narrowed passages – nostril stenosis and microstomia (figure 1C, 2).



Figure 2. 2A. Facial burns leading to scarring affecting the nostrils and the stoma of the mouth. 2B. Note the fixed position of the head due severe contracture of the bands in the anterior neck (Modified Onah 3).

In post burn contracture neck, assessment of Mallampati is possible only in the flexed neck position. Also, flexed neck position may prevent adequate mouth opening, even if the oral cavity is not affected (figure 2C).

Thyromental distance and ability to protrude mandible are impossible to precisely measure.

1.CLASSIFICATION OF POST BURN CONTRACTURE NECK

The classification is based on the location of the contracting band and extent of flexion or extension away from the anatomical position of the neck and jaws. Each group is further sub classified based on the contracting segment width.

Table 1. The Modified Onah Classification of Post Burn Contracture Neck (Jeong et al. 2009)

Sl. No.	Type	Degree of Contracture
1	1. Mild anterior contracture: The patient is able to flex the neck and bring the neck and jaws to the anatomical position while erect.	a. Narrow, <2finger breadths
2		b. Broader, >2finger breadths
3	2. Moderate anterior contracture: attempts at extension away from the anatomical position results in a significant pull at the lower lip	a. Narrow, <2finger breadths
4		b. Broader, >2finger breadths
5	3. Severe anterior contracture: The patient's neck is contracted in the flexed position and the chin is occasionally restrained down to the anterior trunk. The patient is unable to reach the anatomical position of the neck and jaws.	

Significant correlation between Class 2b and 3 and Cormack Grade 3 and 4 laryngoscopic views is seen.

Application of the modified Onah class can reduce the frequency of an unanticipated failure to visualise laryngeal structures.

5. AIRWAY MANAGEMENT STRATEGY

Strategy could include intubating the trachea with the patient awake or after induction of general anaesthesia, with or without neuromuscular blocking agents.

Awake intubation has been advocated as the safest technique to secure airway.

Induction of general anaesthesia with or without neuromuscular blocking agents causes the soft palate, tongue, epiglottis to approximate to the posterior pharyngeal wall thus decreasing the oropharyngeal airspace available for manoeuvring the tip of flexible bronchoscope to locate the glottis.

It is essential to have a back up plan in the event of failure of the primary plan.

2. PREPERATION

Proposed airway management technique should be explained during preoperative evaluation.

Reassure that the discomfort will be minimised by administration of sedatives.

Risks associated with the anaesthetic management of the difficult airway should be informed.

Have one additional individual as an assistant in difficult airway management. Preferable to have a second member of the anaesthesia care team who can assist in the monitoring, ventilation and pharmacotherapy of the patient as well as providing an extra set of hands during fibreoptic intubation.

For patients who refuse awake fibreoptic intubation, a surgeon trained in performing a surgical airway should be available with a tracheostomy/cricothyrotomy tray. However, it is noteworthy that on occasions surgical airway may not be feasible as described earlier due to the scar tissue.

In desperate cases, where the fiberoptic/blind intubation is not possible for any reason and awake intubation is not feasible, division of scar tissue under tumescent local anaesthesia or in combination with intravenous ketamine may tried, following securing airway for further procedure (Agarwal 2004).

1. ANAESTHETIC MANAGEMENT

Premedication

- Antisialagogues

Glycopyrrolate 0.2 to 0.3 mg IV or IM

Scopolamine 0.4 mg IV or IM

Atropine 0.4 to 0.6 mg IV or IM

- Nasal mucosal vasoconstrictors

A mixture of 4 % lignocaine and 1 percent phenylephrine (3:1) – applied via cotton swabs or pledgets to the nasal mucosa, can produce adequate anaesthesia and vasoconstriction in 10 – 15 minutes. However, it has to be done with a cotton swab/pledget place inside the nasal cavity under vision with the help of a nasal speculum. The placement has to be at the back of the middle turbinate, producing blockade of sphenopalatine ganglion.

Nasal decongestants like phenylephrine 0.5% and oxymetazoline 0.05 % may be helpful if applied 15 min prior to attempted intubation.

- Sedatives and hypnotics

Sedation may be provided by judicious use of small doses of benzodiazepine (midazolam 0.03 mg/kg) and opioid (fentanyl 2 g/kg) that results in a calm, comfortable and cooperative patient.

Dexmedetomidine, a highly selective alpha-2 adrenergic receptor agonist, is a useful adjunct for awake intubation (1g/kg) because of its sedative, analgesic, and anxiolytic properties without respiratory depression.

A loading dose (1 g/kg over 10 min) provides procedural sedation for fiberoptic nasotracheal intubation.

- Aspiration prophylaxis

Ranitidine (50 mg IV) and metoclopramide (10 mg IV), if there is a risk regurgitation and aspiration of gastric contents. However, in the face of risk of aspiration of gastric content, the awake intubation under local anaesthesia is not advised.

Monitors

Routine use of ECG, pulse oximetry, non-invasive blood pressure monitoring and capnography is required. Depending on the complexity of the surgery and the patient condition, additional monitoring including invasive haemodynamic monitoring may be decided.

Supplemental Oxygen

Oxygen should be given throughout airway

management- sedation, intubation, extubation. To start with, 100% oxygen (at 10 l/min) on a well-fitting face mask to be provided to achieve an end tidal oxygen of 95%. Once the procedure is about to start, it can be quickly changed to nasal oxygenation, with a gas flow to 15 l/min. If available, high flow nasal oxygenation (30 to 60 l/min) is strongly advised in recent airway management guidelines. It has to be continued if the patient becomes apnoeic.

Apnoeic oxygenation – continuous oxygen insufflation (10-15 L/min) via nasal cannula/airway/catheter.

Topicalization

Topical anaesthesia is the mainstay of airway preparation in PBC neck and can be achieved by use of aerosol sprays, nebulization, gargling or spray-as-you-go technique (SAYGO) or applying local anaesthetic with atomizer.

Nerve Blocks

Superior laryngeal and translaryngeal nerve blocks are usually not possible in PBC neck due to lack of access to the anterior and lateral neck, hyoid bone and limited or no neck extension.

Table 2 : Overview of difficult airway trolley

Part of the DAT	Contents	Monitor(s) for videolaryngoscope/ videobronchoscope alt.
On top	Difficult airway algorithm flowchart Cognitive aids Direct access phone numbers to ENT and anesthesiology/intensive care physician resources Stopwatch	Clear directions to bring videolaryngoscope and videobronchoscope
On the side	Introducers Airway exchange catheter Videobronchoscope (regular size)	
Drawer 1	Laryngoscope handles: standard, short Laryngoscope blades: Macintosh 3 and 4; Miller 2 and 3 Videolaryngoscope blades (if monitor attached to the DAT) Endotracheal tubes 5.0, 6.0, 7.0, and 8.0 Extralingual endotracheal tubes 4.0, 5.0, and 6.0 Nasal endotracheal tubes 6.0 and 7.0 Stylet Lubrication gel	Syringe 5, 10 ml Magill forceps Adhesive tape, wide and narrow Bite block Aspiration cannula Rocuronium 10 mg/ml Preprinted labels "Rocuronium"
Drawer 2	Two different types of 2nd generation SADs size 3, 4, and 5 Lubrication gel Syringe 20 ml Orogastric tube size 12 and 14	Cognitive aid (continuous waveform capnography) Adjuncts for flexible videobronchoscopic-guided intubation: endoscopy mask, breakaway oropharyngeal airway, swivel connector, spray solution lidocaine 40 mg/ml and 100 mg/ml, antifog solution, and tongue depressor
Drawer 3	Facemask size 3 and 4 Neonatal facemask size 0 Oropharyngeal airways (e.g., 7, 9, 10, and 11 cm) Nasopharyngeal airway 6.0, 7.0, and 8.0	Sugammadex 100 mg/ml, 2 ml/vial, 8 vials Syringe 10 ml Aspiration cannula Preprinted labels "sugammadex"
Drawer 4	Emergency cricthyrotomy catheter set Endotracheal tube 6.0	Scalpel blade 10 Prova introducer
Drawer 5	Optional, customized equipment (e.g., left-hand laryngoscope blades, equipment for management of tracheostomies, and Ventrain device)	

DAT = difficult airway trolley; ENT = ear, nose, and throat; SAD = supraglottic airway device.

Figure 3: Drawer 1 (PLAN A – INTUBATION)



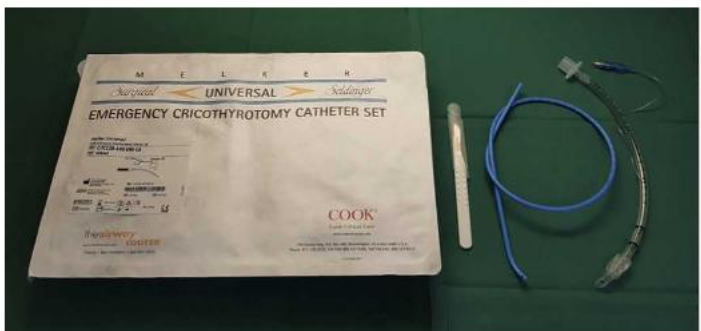
Figure 4: Drawer 2 (PLAN B – OXYGENATION VIA SUPRAGLOTTIC AIRWAY DEVICE)



Figure 5: Drawer 3 (PLAN C – MASK VENTILATION)
Figure



Figure 6 : Drawer 4 (PLAN D FRONT OF NECK ACCESS)



There are many techniques that can be used to secure airway in the awake patient.

Direct laryngoscopy, intubating laryngeal mask airways, FFB, rigid fibreoptic laryngoscopy, retrograde intubation, lighted stylets and blind nasal intubation.

In post burn contracture neck patient, location of the glottis and insertion of the fiberscope into the trachea can be difficult because of the fixed flexion deformity of the neck and distortion of upper airway structures by the fibrotic scar.

Manoeuvres that facilitate glottis visualization, such as jaw thrust and head extension may not be possible in

such patients. Sometimes, because of extreme flexion deformity of the neck, it may be very difficult or even impossible to advance the tracheal tube over the scope into the trachea when the fiberscope has been successfully placed in the trachea. An important reason for this difficulty is deviation of the course of the tube from that of the fiberscope toward the epiglottis, arytenoids cartilage, pyriform fossa or oesophagus. Oesophageal intubation may occur despite correct placement of the fiberscope into the trachea.

Backup plan for airway management

There are occasions when a planned gold-standard awake fibreoptic-guided intubation may fail. Back-up plans include ILMA-guided tracheal intubation or use of LMA. Surgical release of the neck contracture (and/or microstomia) using ketamine, with or without local anaesthetic infiltration, followed by direct laryngoscopic intubation, ILMA-guided intubation or LMA is an option after a failed fibreoptic intubation attempt.

Use of muscle relaxant

If traditional approach with general anaesthesia is used for the procedure, question may arise regarding suitability of the relaxant with the quickest onset. However, succinyl choline, being a depolarising agent, is known to cause transient raise in plasma potassium (on an average 0.5 mEq/l), and acute burns patients are likely to have pre-existing hyperkalaemia,

Tumescent Anaesthesia

Tumescent anaesthesia (TA) involves subcutaneous infiltration of a large volume of very dilute lignocaine (as low as 0.05% or 0.1%) and adrenaline (1:200,000). This allows administration of up to 55 mg/kg of lignocaine.

8. EXTUBATION

The trachea should be extubated only once the patient is awake. An airway management plan must be in place in case the patient cannot maintain adequate ventilation after extubation. In situations where intubation is still difficult, consider briefly using a tube exchanger to facilitate rapid re-intubation. An airway exchange catheter can also be used to deliver oxygen (by insufflation, manual, or jet ventilation), buying time to attempt other airway techniques such as fiberoptic bronchoscopy or a surgical airway.



9. BLOOD LOSS

Blood loss may be a significant concern during the contracture release as extensive tissue dissection and reconstruction with skin grafting is involved. An average estimated blood loss reported is between 15 to 400 ml. Also, the loss of 0.5 to 0.75 ml per square centimetre area may be a quick guide to be prepared and to estimate the loss at the earliest.

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Anaesthesia for Release of Post-Burn Neck Contracture Q&A

1. What are the anaesthetic concerns in post-burn neck contracture?

Major concerns include:

- Difficult mask ventilation
- Difficult laryngoscopy and intubation
- Distorted airway anatomy
- Restricted neck extension
- Limited mouth opening (microstomia)
- Poor venous access
- Difficult patient positioning
- Blood loss during contracture release
- Hypothermia during reconstructive surgery
- Postoperative airway edema and extubation

difficulty

Airway management remains the most important anaesthetic concern.

2. How does neck contracture affect airway assessment?

Neck contracture causes:

- Fixed flexion deformity
- Reduced atlanto-occipital extension
- Reduced submandibular space compliance

• Distortion of oral, pharyngeal and laryngeal axes

• Difficulty in assessing thyromental and sternomental distance

These changes predict difficult laryngoscopy and intubation

3. Which airway assessment tools should be used?

- Mouth opening (inter-incisor distance)
- Mallampati classification
- Thyromental distance
- Sternomental distance
- Upper lip bite test
- Neck movement assessment
- Previous anaesthetic records
- Evaluation for nasal patency if nasal intubation planned

No single test predicts difficulty; combined assessment is recommended.

4. Why is Mallampati grading unreliable?

Mallampati grading assesses only volume oropharyngeal space or how much is occupied by base



tongue.

In severe neck contracture:

- Mouth opening may be restricted.
- Neck extension
- is absent.
- Airway difficulty is mainly due to cervical deformity.
Therefore Mallampati alone underestimates airway difficulty.

5. Why may mask ventilation be difficult?

- Facial scarring and difficult mask grip
- Microstomia
- Distorted facial contours leading to Poor mask seal
- Reduced mandibular mobility

6. Why may laryngoscopy be difficult?

Because of:

- Limited neck extension and inability to align airway axes
- Reduced mouth opening
- Reduced mandibular space
- Altered tracheal position

7. What burn history is important?

- Cause and duration of burn, associated inhalational injury
- Site and extent of burns
- Previous surgeries and anaesthetics, previous airway problems, if any
- History of tracheostomy
- Current functional limitation

Previous anaesthetic records are particularly valuable. However, in case of caustic injury, the finding may change for bad with time.

8. Why assess for other contractures?

Associated contractures may:

- Limit positioning
- Cause difficulty obtaining IV access
- Interfere with monitor placement
- Complicate regional anaesthesia

9. What investigations are required?

- CBC
- Renal function tests
- Electrolytes
- Blood sugar
- Coagulation profile (if indicated)
- X-ray and other imaging of neck

- ECG (elderly/comorbid patients)

10. What nutritional issues occur in burn patients?

Chronic burn patients may have:

- Malnutrition
- Anaemia
- Hypoproteinaemia
- Delayed wound healing
- Altered drug pharmacokinetics
Optimization before surgery improves outcomes.

11. Why is airway management the primary concern?

Airway distortion from scar contracture may lead to:

- Difficult ventilation
- Difficult intubation
- Cannot-intubate/cannot-oxygenate (CICO)

situation

And therefore failure to secure the airway is the leading perioperative risk.

12. What are the predictors of difficult airway?

- History of inhalation burns and tracheostomy
- Severe neck contracture
- Fixed flexion deformity
- Microstomia
- Limited neck movement
- Mallampati III/IV
- Previous difficult intubation
- Scar involving face and chin

13. Which is the safest airway plan in severe mentosternal contracture?

Awake fiberoptic intubation while maintaining spontaneous ventilation is considered the safest and most established approach.

14. When is awake fiberoptic intubation indicated?

When difficult:

- Mask ventilation
- Supraglottic airway insertion
- Laryngoscopy

is anticipated and spontaneous ventilation should be preserved. ([Difficult Airway Society](#))

15. List advantages and disadvantages of awake fiberoptic intubation.

Advantages

- Maintains spontaneous breathing
- Allows unhurried approach



- Allows neurological examination
- Avoids airway collapse which may follow induction of anaesthesia
- Possible to interrupt the procedure and postpone

Disadvantages

- Requires expertise and equipment
- Needs patient cooperation
- Secretions and blood impairs view, hence cannot be a rescue technique

16. How is the airway prepared for awake intubation?

- Counselling and consent
- Antisialagogue (glycopyrrolate IM)
- Topical lidocaine spray/nebulization
- Nasal vasoconstrictor (if nasal route)
- Sedation with preserved respiration (Dexmedetomidine, Midazolam, Fentanyl)
- Oxygen supplementation

17. Describe topical anaesthesia technique.

- Lidocaine nebulization (2 ml of 4% topical; preparation)
- Lidocaine spray (10%, each puff delivers 10 mg)
- Placing cotton pledgets impregnated with LA in the middle turbinate
- Local anaesthetic gargle (Viscous preparation is not available currently)
- Superior laryngeal nerve block and transtracheal block (may not be feasible here)
- Spray-as-you-go (SAYGO)

A combination of these techniques may be used effectively depending on the condition and cooperation of the patient. However, it is important to remember not to cross the safe upper limit of lignocaine total dose (7 mg/kg).

18. Which sedative agents are useful for awake intubation?

Following agents in titrated doses can significantly reduce anxiety and improve cooperation of the patient:

- Dexmedetomidine
- Remifentanyl
- Fentanyl (small doses)
- Midazolam (carefully titrated)

May be used in combination with close observation to avoid oversedation as well as respiratory depression.

19. Why dexmedetomidine is preferred?

Dexmedetomidine provides following without causing significant effect on respiratory drive or the respiratory muscle tone:

- Sedation
- Anxiolysis
- Cooperative patient state
- Also, obtunds the response to airway manipulation

20. What are the alternatives if fiberoptic bronchoscope is unavailable?

- If mouth opening is reasonably adequate, securing airway with
- Videolaryngoscope
- Intubating LMA
- Airtraq
- Scar release under local anaesthesia and sedation and then general anaesthesia
- Elective tracheostomy (may be difficult in few cases deserving fiberoptic intubation)

21. Why avoid muscle relaxants before securing airway?

Loss of spontaneous breathing may produce:

- Impossible mask ventilation
- Airway obstruction
- CICO scenario

This may get worse with impossible surgical airway due to lack of anatomical landmarks in the anterior neck. Therefore airway should preferably be secured first.

22. Is inhalational induction useful?

Yes. When awake intubation is not feasible:

- Sevoflurane induction
- Maintain spontaneous ventilation may be considered.

23. Why preserve spontaneous ventilation?

- Preserved spontaneous breathing prevents:
- loss of muscle tone and maintains airway patency
- Facilitates oxygenation during the process of intubation
- Aids in maintaining suitable depth of anaesthesia

Thus provides safety during difficult airway management, by ensuring spontaneous breathing, if intubation fails. If intubation fails, patient continues breathing spontaneously.



24. Which are the preferred induction agents?

The induction agents can be:

- Sevoflurane
- Ketamine
- Propofol (carefully titrated)

Where choice depends on airway plan and patient condition. Preservation of breathing and airway patency takes precedence.

25. How will you manage if mask ventilation becomes impossible?

Follow difficult airway algorithm:

1. Call for help
2. Continue high flow nasal oxygen with nasal prongs/catheter or HFNO
3. Monitor SpO₂
4. Attempt SGA placement
5. Attempt alternate intubation technique
6. Awaken the patient
7. If paralysed, reverse the paralysis
8. If above is not helping plan emergency surgical airway

26. Which difficult airway guideline are handy?

Several airway guidelines are available. However, the AIDAA guideline for unanticipated difficult airway prepares the operator better.

27. What are the contents of difficult airway cart?

The difficult airway cart must contain:

- Fiberoptic bronchoscope
- Videolaryngoscope
- Various LMAs
- Bougie
- Stylets
- Cricothyrotomy set
- Tracheostomy set
- Oxygen delivery devices

Please refer to the section on airway cart in the write up.

28. Describe management of total ventilation failure.

Ensure continuous oxygenation. Call for expert help, if not already requested. Continue mask ventilation/device ventilation while planning the surgical airway. The surgical airway recommended is scalpel bougie technique. Do not go for surgical tracheostomy. If there is an option to quickly wake up

the patient that may be the first choice. Do not persist with repeated intubation attempts.

29. Why is front-of-neck access difficult?

Scar tissue:

- Distorts landmarks
- Reduces neck mobility
- Makes identification of cricothyroid membrane difficult
- Dissection of the tissues is tough

30. When should elective postoperative ventilation be considered?

If there is:

- Airway oedema
- Prolonged surgery
- Difficult intubation
- Massive fluid administration
- Doubtful airway patency

31. Outline the extubation strategy.

Extubation is an elective procedure. Therefore, it has to be well planned. Following are the pre requisites for planned extubation:

- Awake patient, obeying
- Full muscle power/complete reversal of the neuromuscular block
- Adequate assistance and equipment to re-intubate
- Airway exchange catheter at hand
- Positive leak test

32. How postoperative analgesia is provided?

Multimodal analgesia:

- Paracetamol
- NSAIDs (if not contraindicated)
- Opioids, with due caution: now agonist-antagonist/atypical opioid are popular for the lack of respiratory depressant effect
- Regional techniques where feasible

33. What is the most important take-home message?

Answer

In post-burn neck contracture surgery, airway management should be planned meticulously. Awake fiberoptic intubation with preservation of spontaneous ventilation remains the gold standard for severe contractures, and multiple backup airway strategies must always be available.



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Scientific Schedule

DAY -2

14th JUNE 2026

Time	Topic	Speakers
07:30AM - 08:30AM	Poster presentation and Breakfast	
08:30AM - 09:10AM	Anaesthetic management of patient with IHD posted for THR	Dr Balasubramanya BMCRC, Ballari Dr VY Srinivas MMCRI, Mysore
09:10AM - 09:50AM	Anaesthetic management of a hyperthyroid patient with posted for total Thyroidectomy	Dr Karthik GS Rajarajeshwari Medical, Bangalore Dr Jyothi KIMCRI, Hubli
09:50AM - 10:20AM	Open Eye Globe injury	Dr Darshan CIMS, Chamarajanagar Dr Rajendra Kumar Al Ameen Medical College, Bijapur
10:20AM - 10:40AM	Tea Break	
10:40AM - 11:20AM	Anaesthetic management of 32 years old female with posterior cranial fossa tumour posted for surgery	Dr Sriganesh NIMHANS, Bangalore Dr Ramesh Koppal SNMC, Bagalkot
11:20AM - 12:00Noon	Anaesthetic management of morbidly obese patient with OSA posted for bariatric surgery	Dr C L Gurudutt Mysuru Dr Prabhu BG JJMMC, Davangere
12:00Noon - 12:30PM	3 months baby with cleft lip and palate posted for surgery	Dr Deepak ESI Medical College, Rajajinagar, Bangalore Dr Vinay Marulasiddappa BMCRI, Bengaluru
12:30PM - 01:30PM	Vijayanagara Quiz - Finals	Dr Rashmi N R KIDWAI, Bangalore Dr Megha Chitradurga Medical College, Chitradurga Dr Krishna Rashad GIMS, Kalburgi
01:30PM - 02:10PM	Lunch	



Scientific Schedule

Time	Topic	Speakers
02:10PM - 02:40PM	Twenty weeks pregnant lady with past history of peripartum cardiomyopathy with acute appendicitis posted for appendicectomy	Dr Shivanand L K BLDE, Vijayapura Dr Sumesh Rao KMC, Mangalore
02:40PM - 03:10PM	A geriatric patient with BPH posted for TURP	Dr Narendra Babu Sapthagiri institute of Medical Sciences and Research Centre, Bangalore Dr Vandana Kulkarni East Point Medical College Bangalore
03:10PM - 03:40PM	Anaesthetic management of patient with CKD on dialysis posted for renal transplantation	Dr Geetha MS Ramaiah MC, Bangalore Dr Thippeswamy Yenepoya Medical College, Mangalore
03:40PM - 04:10PM	OSCE	Dr Yoganarasimha BGS Global, Bengaluru Dr Ashwini Basweshwara Medical College, Chithradurga
04:10PM - 04:40PM	Panel:"How to write Theory exam paper"	Moderator: Dr Harsoor, Dr B R Ambedkar Medical College, Bangalore Panelists Dr H S Murthy Manipal Hospital, Old Airport Road, Bangalore Dr S Bala Bhaskar BMCRC, Ballari Dr Prashant Sajjan East point MC, Bangalore
04:40PM - 04:50PM	Tea Break	
04:50PM - 06:10PM Viva-voce		
04:50PM - 05:25PM	Anaesthesia Machine	Dr Selvakumar Madurai
05:25PM - 05:40PM	X-Rays	Dr Saraswathi BMCRI, Bangalore
05:40PM - 05:55PM	PFT and Capnography	Dr Netra HN BMCRI, Bangalore
05:55PM - 06:10PM	ABG	Dr Ravibhat SDM Dharwad



14th JUNE 2026
07:30AM - 08:30AM

ANAESTHETIC MANAGEMENT OF PATIENT WITH IHD POSTED FOR TOTAL HIP REPLACEMENT: PART 1: PREOPERATIVE ASSESSMENT, RISK STRATIFICATION, OPTIMIZATION)

Authors: Dr Bala Subramanya H, Dr Shravanthi and Dr Kavya,
Ballari Medical College & Research, Centre, Ballari

Introduction: Coronary artery disease (CAD) is prevalent in approximately 18% of patients undergoing major non cardiac surgery. It is associated with 3.5 fold increased risk of perioperative major adverse cardiac events(MACE). Less than 3%of patients who undergo elective total hip replacement have a perioperative MI.

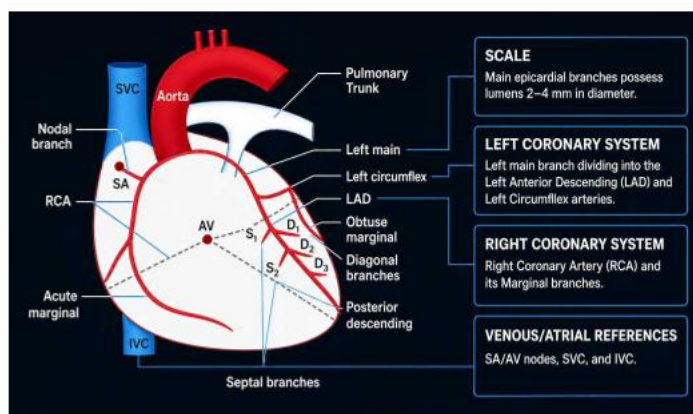
Q1. Describe the coronary circulation

Cardia is supplied by Left and right coronary arteries

A. Right Coronary Artery (RCA) -Arises from right aortic sinus It Supplies -Right atrium, Right ventricle, Inferior wall of left ventricle, SA node, and AV node.

B. Left Coronary Artery (LCA) - Arises from left aortic sinus It Supplies: Anterior wall of left ventricle, Anterior 2/3 of interventricular septum, Bundle branches

2. Left Circumflex (LCX)- Runs in left AV groove, it



Supplies: Lateral wall of left ventricle, SA node (~40% cases)

Q2.What is the pathophysiology of ischemic heart disease?

Atherosclerosis of the coronary arteries is the most common cause for ischemic heart disease which leads to the narrowing the these vessels.

The main coronary artery epicardial branches have lumens that are 2–4 mm in diameter. In the absence of collaterals, exertional angina occurs when the lumen area is reduced to 1 mm (50–60% reduction in diameter or 75% reduction in the cross-sectional area).

In a patient with coronary stenosis initially angina occurs at the physical activity which causes imbalance of myocardial oxygen supply and demand leading to ischemic chest pain.

Angina at rest occurs when the luminal area is reduced to 0.65 mm (75% reduction in diameter or 90% reduction in cross-sectional area).

STEMI occurs when coronary blood flow decreases abruptly. This acute decrease is attributable to thrombus formation at a site when an atherosclerotic plaque fissures, ruptures or ulcerates creating local environment that favors thrombogenesis. Vulnerable plaques are those with rich lipid core and thin fibrous cap.

Unstable angina/non-STEMI – results from a reduction in myocardial oxygen supply. Typically, rupture of coronary plaque leads to thrombosis, inflammation, and vasoconstriction. In most culprit arteries stenosis is less than 50%.

Non stenotic causes of MI are Aortic valve disease, LVH, Ostial occlusion, Coronary embolism Coronary arteritis and Vasospasm.

Balancing oxygen supply and demand: Is important in order to reduce the workload on cardia

Q3.Name the factors affecting myocardial oxygen supply?

- ◆ Coronary blood vessel diameter
- ◆ Autonomic innervation
- ◆ Autoregulation
- ◆ Coronary steel
- ◆ Coronary perfusion pressure
- ◆ Arterial oxygen content
- ◆ Heart rate, Diastolic time
- ◆ Exogenous substances – O₂, CO₂, nitric oxide



Q4. What are the factors affecting myocardial oxygen demand?

- ♦ Basal requirement
- ♦ Heart rate
- ♦ Wall tension – preload and afterload
- ♦ Contractility

Q5. Enumerate the risk factors for IHD

Risk factors for IHD may be modifiable and non-modifiable

1. Non-modifiable Risk Factors: male gender, increasing age, genetic factors, family History, and ethnicity

2. Modifiable Risk Factors: Hypercholesterolemia, hypertension, diabetes Mellitus, cigarette smoking, obesity, sedentary life style, diet, psychosocial stress and alcohol

3. Others: Metabolic syndrome, chronic kidney disease, inflammatory markers (e.g., CRP), hypercoagulable status

Q6. What are the goals of preoperative assessment of these patients?

The goals of preoperative evaluation are to

- 1) Ascertain whether the patient has previously undiagnosed significant IHD
- (2) Characterize known IHD with respect to severity, functional limitations, therapy, and prior investigations
- (3) Determine whether additional specialized testing or consultations are warranted
- (4) Identify opportunities for reducing peri-operative risk related to IHD

Q7. How do you perform the preoperative assessment of this patient?

Preoperative assessment includes history taking, general physical examination, systemic examination and investigations (both routine and special)

History – In patients without known IHD, evaluation of traditional risk factors (as mentioned above) for IHD should be done. If patient is a known case of IHD then severity, progression, stability of disease, revascularisation intervention undertaken and review of medication currently patient is on should be done.

Following are the common symptoms present in a patient with IHD.

Stable angina- characterised by

- ♦ Retrosternal pain/ discomfort/ heaviness
- ♦ Which may radiate to neck, jaw, back or shoulder
- ♦ Crescendo decrescendo in intensity
- ♦ Lasting several minutes induced by physical or emotional stress

Unstable angina:

Defined as angina at rest, angina of new onset, or an increase in the severity or frequency of previously stable angina without an increase in levels of cardiac biomarkers.

Prinz metal's angina: It is a form of unstable angina caused by transient coronary artery vasospasm, leading to temporary myocardial ischemia. It typically occurs at rest.

Characterise history of angina with respect to its duration, precipitating factors, associated symptoms, and relieving factors.

Angina may be classified as per Canadian cardiovascular Society grading scale.

Dyspnoea on exertion may be an angina equivalent but may be seen in a patient with respiratory diseases.

One should know the severity, progression and functional limitations imposed by ischemic heart disease.

Evaluate patient's response to various physical activities - quantify in terms of METS

Ask for associated co-morbidities such as hypertension, diabetes, hypercholesterolemia, cerebrovascular disease, peripheral vascular disease, COPD and renal insufficiency.

Past history of MI: Acute MI: 1- 7 days old; Recent MI: 8 - 30 days; Old MI - more than 30 days.

Complications of prior MI include arrhythmias (Atrial fibrillation, bradyarrhythmias) conduction blocks, pericarditis, mitral regurgitation, ventricular septal rupture, congestive heart failure and cardiogenic shock, cerebro-vascular complications



ECG Lead	Coronary artery responsible for ischemia	Area of myocardium that may be involved
II, III, aVF (Inferior wall)	Right coronary artery	Right atrium, right ventricle, sinoatrial node, inferior aspect of left ventricle, atrioventricular node
I, aVL (Lateral wall)	Circumflex coronary artery	Lateral aspect of left ventricle
V2-V4 (Antero septal)	Left anterior descending artery	Anterolateral aspect of left ventricle

Symptoms due to reduced functioning of cardia(failure) include - dyspnoea on exertion, Orthopnoea / PND

Medication history: medications are designed to decrease myocardial oxygen requirements, improve coronary blood flow, prevent thrombosis and remodel injured myocardium. These goals are achieved by – statins, anti-platelets drugs, beta blockers, ACE inhibitors etc.,

Physical Examination – look for signs of left and right ventricular dysfunction (Raised JVP, pedal edema, S3 gallop/ rales on auscultation), cerebrovascular disease (carotid bruit), and autonomic dysfunction (Orthostatic hypotension).

Q8. How do you screen a patient with known or suspected IHD coming for surgery?

Other than routine investigations such as HB%, TC/DC, Urine routine, RBS, Blood grouping & RH typing, blood urea and serum creatinine following are special preoperative screening tests done in patient with known IHD.

Resting ECG: ECG changes commonly seen in patients with IHD are ST elevation or depression, T wave inversion, Old MI (Q waves), disturbance in conduction and rhythm, and LVH.

Exercise ECG – is useful for detecting myocardial ischemia and establishing their relationship to chest pain. It is less accurate but cost effective. Myocardial ischemia most likely when there is at least 1 mm horizontal or down sloping ST segment depression

during or immediately after exercise. A negative test does not exclude presence of coronary artery disease but chances of left main coronary artery disease or 3 vessel disease unlikely.

Echocardiogram done at rest or after exercise – regional wall motion(RWMA) analysis may indicate CAD.RWMA may be in the form of hypokinesia(reduced movement), dyskinesia (abnormal or paradoxical movement of the heart wall). (moves in the opposite direction of normal contraction) and akinesia (complete lack of movement) It also provides information about the condition of valves, cardiac chambers, wall thickness, systolic and diastolic function of LV and presence any intracardiac thrombus if any.

Pharmacological stress test: may be done using dobutamine, adenosine; useful in a patient with physical deconditioning or in patients who are unable to exercise.

Nuclear stress imaging using thallium or technetium tracers – it can define vascular regions where stress induced coronary blood flow is limited.

Biomarkers: to know the basal levels (trop-I, T, BNP) are part of assessment in recent ACC/AHA guidelines. They have a prognostic value

Coronary angiography – provide best information about the condition of coronary arteries. It is indicated in cases of unstable coronary syndrome, in patients with angina despite maximum medical therapy, equivocal non-invasive test in a patient undergoing



high risk surgery or before coronary revascularisation. It is also useful in establishing coronary artery spasm.

CT angiogram is less invasive alternative to traditional catheter angiogram which provides high resolution 3D models. It is not useful in elderly patients or in patients severe coronary artery calcifications.

MRI – provide greater image clarity and can delineate the proximal portions of coronary arterial circulation.

Q8. How do you optimise this patient preoperatively?

Physical optimisation: includes lifestyle modification, cessation of smoking, regular aerobic exercise, low-fat and low cholesterol diet, reduction of alcohol consumption and treatment of hypertension.

Patient coming for elective procedure following therapeutic options are available. They are

- ♦ Revascularisation by surgery
- ♦ Revascularisation by PCI
- ♦ Optimal medical management

Revascularisation: is indicated when optimal medical therapy fails to control angina. Revascularization is achieved either by CABG or percutaneous coronary intervention (PCI).

PCI includes coronary balloon angioplasty and stenting of effected coronary artery with either drug eluding or bare metallic stents. Reserved for patient with uncomplicated single vessel disease with good LV function.

CABG – Preop CABG or revascularization should be reserved for patients when coronary revascularization risk is acceptable such as patients with Left main coronary artery stenosis, three vessel disease with left ventricular dysfunction, intractable coronary ischemia despite maximal medical therapy.

Major non cardiac surgeries should wait at least 4 – 6 weeks (Possibly 6 months) following CABG.

Pharmacological optimisation:

May be achieved with following medications - Antiplatelet drugs, nitrates, beta blockers, CCB, and ACE inhibitors.

Q9. How do you perform risk stratification in a patient

with IHD?

ACC/AHA guidelines integrate risk stratification according to clinical risk factors, functional capacity and surgery specific risk factors.

Functional status (METs): may be assessed as follows

Excellent - > 10 (ex – jogging, swimming, tennis)

Good – 7 – 10 (walking uphill, dancing)

Moderate – 4 – 6 (climbing stairs)

Poor - < 4 (eating, dressing, bathing)

Surgery specific risk factors:

1. **High risk surgeries**- emergency surgeries, aortic and other major vascular surgery, anticipated prolonged surgical procedures associated with large fluid shifts or anticipated blood loss) are often reported to have a cardiac risk of greater than 5%.

2. **Intermediate risk surgeries**- (carotid endarterectomy, head and neck surgery, intraperitoneal and intrathoracic surgery, prostate surgery) are reported generally to have cardiac risk of less than 5%.

3. **Low risk procedures**: - endoscopic procedures, superficial procedures, cataract surgeries, breast surgery are reported to have less than 1% risk of cardiac events.

As per this risk stratification THR is an intermediate risk surgery.

Clinical risk predictors in a patient with IHD undergoing surgery:

Obtained by history, physical examination & review of ECG, the clinical risk factors are grouped into 3 categories...

1. **Major clinical predictors** are unstable coronary syndrome, decompensated heart failure, significant dysrhythmia and severe valvular disease. They mandate intensive management even if that leads to delay or cancellation except emergency surgery.

2. **Intermediate clinical predictors** are mild angina pectoris, previous MI by history or pathological Q waves, compensated or prior heart failure, insulin dependent diabetes mellitus, and renal insufficiency. These are markers of enhanced risk of peri-operative cardiac complications.

3. **Minor clinical predictors** are hypertension, LBBB, nonspecific ST-T wave changes and history of stroke. They have not proved to increase risk independently.



Q10.What is Lee's Revised Cardiac Risk Index (RCRI)?How do you interpret it?

Lee's RCRI describes six independent predictors of major cardiac complications. They are

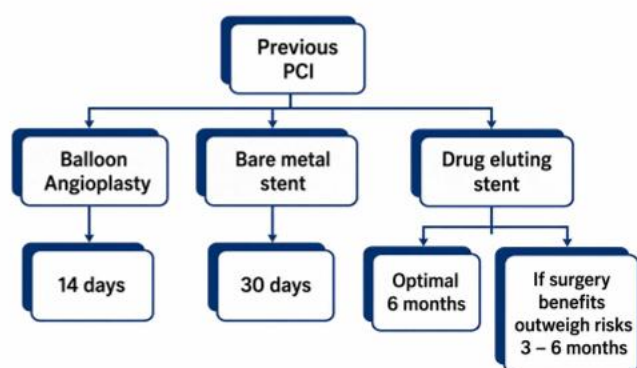
- ♦ High-risk surgical procedures such as intrathoracic, intraperitoneal or supra-inguinal vascular reconstruction
 - ♦ History of IHD
 - ♦ History of congestive cardiac failure
 - ♦ History of a stroke or transient ischemic attack (TIA)
 - ♦ Preoperative insulin therapy
 - ♦ Serum creatinine > 2 mg%

No. of risk factors	Event rate
0	0.4%
1	0.9%
2	6.6%
>3	11%

Q11.Name the other Cardiac Risk Indices?

Goldman cardiac risk index, Detsky modified multifactorial index, Eagle criteria for cardiac risk assessment

Q12.What should be the minimum duration of delay for non-cardiac surgery in a patient with previous PCI?



Q13.Describe the preoperative medication management in a patient with IHD

- ♦ Aspirin – to be continued
- ♦ Clopidogrel - should be stopped 5 - 7 days prior to surgery
- ♦ Anticoagulants – should be stopped prior to surgery. Duration of interruption depends on the surgical/procedural risk of bleeding. If

peri-operative risk of thromboembolism is high then bridging with short acting anticoagulants should be done.

- ♦ Patients already on Beta blockers or CCBs should continue to taking them to prevent rebound hypertension. Beta blockers should not be started acutely but started at least 24 hours before elective surgery.
- ♦ ACE and ARBs – withhold 24 hrs before non-cardiac surgery.
- ♦ Statins – may be continued during perioperative period.

Q14.What is the multistep algorithm for patient with

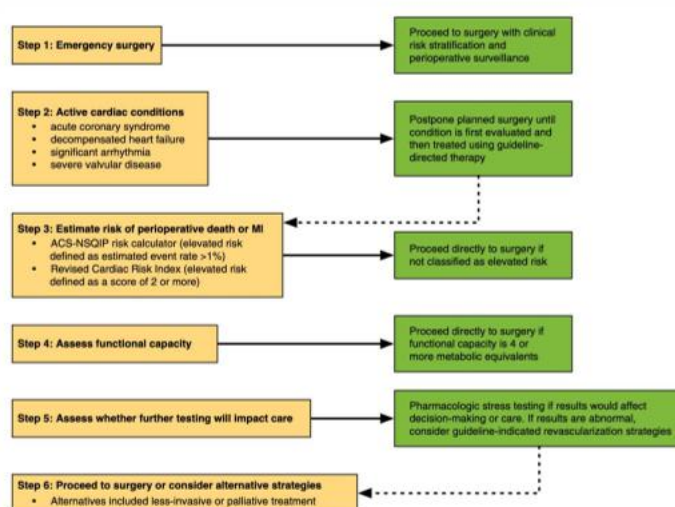


Fig. 28.5 Simplified cardiac evaluation algorithm for noncardiac surgery proposed by the 2014 American Heart Association and American College of Cardiology guidelines. ACS-NSQIP, American College of Surgeons National Surgical Quality Improvement Program; MI, myocardial infarction. (from Fleisher LA, Fishman RJ, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;130:e278-e333.)

IHD undergoing surgery?

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ANAESTHETIC MANAGEMENT OF PATIENT WITH IHD POSTED FOR TOTAL HIP REPLACEMENT: PART 2: ANAESTHETIC MANAGEMENT AND POST OPERATIVE CARE.

AUTHORS: Dr. V.Y.Srinivas, Dr. Ananya .A. Potdar, Dr. Anjali Ranjan.

DEFINITION:

Ischemic heart disease (IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium.

It typically occurs when there is an imbalance between myocardial oxygen supply and demand.

ACUTE CORONARY SYNDROME

Acute Coronary Syndrome (ACS) refers to a spectrum of clinical conditions caused by acute myocardial ischemia and/or infarction, resulting from a sudden reduction in coronary blood flow, usually due to rupture of an atherosclerotic plaque with superimposed thrombus formation.

ACS includes three main entities:

♦ Unstable Angina (UA)

Ischemia without myocardial necrosis
No rise in cardiac biomarkers

♦ Non-ST Elevation Myocardial Infarction (NSTEMI)

Ischemia with myocardial necrosis
Elevated cardiac biomarkers, no ST elevation

♦ ST Elevation Myocardial Infarction (STEMI)

Complete coronary occlusion
ST elevation on ECG + positive biomarkers.

Components of ACS

CLASSIFICATION BASED ON NYHA AND CCS

TABLE 273-1 Cardiovascular Disease Classification Chart		
CLASS	NEW YORK HEART ASSOCIATION FUNCTIONAL CLASSIFICATION	CANADIAN CARDIOVASCULAR SOCIETY FUNCTIONAL CLASSIFICATION
I	Patients have cardiac disease but <i>without</i> the resulting <i>limitations</i> of physical activity. Ordinary physical activity does not cause undue fatigue, palpitations, dyspnea, or anginal pain.	Ordinary physical activity, such as walking and climbing stairs, <i>does not cause angina</i> . Angina present with strenuous or rapid or prolonged exertion at work or recreation.
II	Patients have cardiac disease resulting in <i>slight limitation</i> of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.	<i>Slight limitation</i> of ordinary activity. Walking or climbing stairs rapidly, walking uphill, walking or stair climbing after meals, in cold, or when under emotional stress or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of stairs at a normal pace and in normal conditions.
III	Patients have cardiac disease resulting in <i>marked limitation</i> of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.	<i>Marked limitation</i> of ordinary physical activity. Walking one to two blocks on the level and climbing one flight of stairs at normal pace.
IV	Patients have cardiac disease resulting in <i>inability</i> to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.	<i>Inability</i> to carry on any physical activity without discomfort—anginal syndrome <i>may</i> be present at rest.



CLASSIFICATION BASED ON EF

Categories of left ventricular ejection fraction

LVEF (%)	Qualitative assessment
>70	Hyperdynamic
50-70	Normal
40-49	Mild dysfunction
30-39	Moderate dysfunction
<30	Severe dysfunction

LVEF: left ventricular ejection fraction.

CLASSIFICATION BASED ON CORONARY ANGIOGRAPHY

Category	Description	Significance
Single vessel disease (SVD)	One major coronary artery involved	Lower risk
Double vessel disease (DVD)	Two vessels involved	Moderate risk
Triple vessel disease (TVD)	All three major vessels involved	High risk
Left Main Coronary Artery Disease (LMCA)		Very high risk (critical)

BASED ON DEGREE OF STENOSIS

Severity Stenosis

Mild	<50%
Moderate	50–70%
Severe	>70%
Critical	>90%

The 2014 ACC/AHA guidelines categorises patients into only 2 groups – low risk and elevated risk based on the presence of clinical risk factors

- **LOW RISK** – one in which the combined surgical and patient characteristics predict a risk of a major adverse cardiac event (MACE) of death or MI of <1%. eg:- cataract and plastic surgery

- **ELEVATED RISK** – procedures with a risk of MACE >or=1%

• MACE

1. Myocardial infarction
2. Pulmonary edema
3. Ventricular fibrillation
4. Primary cardiac arrest
5. Complete heart block

Ans. The Canadian Cardiovascular Society introduced a grading system for angina:

- **Class I:** Angina with strenuous or rapid prolonged exertion at work or recreation
- **Class II:** Angina with walking or climbing stairs rapidly, walking uphill or walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace
- **Class III:** Angina with walking one to two blocks on the level and climbing one flight of stairs at a normal pace
- **Class IV:** Angina may be present at a very low level of physical activity or at rest.

Table 8. Association of Heart Failure and Left Ventricular Ejection Fraction With 90-day Mortality in Patients Undergoing Noncardiac Surgery

	N	Crude Mortality	Crude OR	Adjusted OR
No heart failure	561 738	1.22%	Reference	Reference
HFpEF, LVEF ≥50%	28 742	4.88%	4.14 (3.90-4.39)	1.51 (1.40-1.62)
LVEF 40%-49%	7612	5.11%	4.34 (3.91-4.82)	1.53 (1.38-1.71)
LVEF 30%-39%	6048	6.58%	5.68 (5.12-6.31)	1.85 (1.68-2.05)
LVEF <30%	4185	8.34%	7.34 (6.56-8.21)	2.35 (2.09-2.63)

Adapted with permission from Lerman et al.¹⁰ Copyright 2019 American Medical Association. All rights reserved.

HFpEF indicates heart failure with preserved ejection fraction; LVEF, left ventricular ejection fraction; and OR, odds ratio (with 95% CI).

Perioperative risk stratification:

- Acute MI (<7 days)
- Recent MI (7-30 days)
- considered major clinical predictors of perioperative risk.
- Healed MI/ Prior MI(>30 days)



Class of Recommendations and Levels of evidence

Table 3. Applying the American College of Cardiology/American Heart Association Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care (Updated May 2019)

CLASS (STRENGTH) OF RECOMMENDATION	LEVEL (QUALITY) OF EVIDENCE‡
CLASS 1 (STRONG) Benefit >>> Risk Suggested phrases for writing recommendations: - Is recommended - Is indicated/useful/effective/beneficial - Should be performed/administered/other - Comparative-Effectiveness Phrases†: - Treatment/strategy A is recommended/indicated in preference to treatment B - Treatment A should be chosen over treatment B	LEVEL A - High-quality evidence‡ from more than 1 RCT - Meta-analyses of high-quality RCTs - One or more RCTs corroborated by high-quality registry studies
CLASS 2a (MODERATE) Benefit >> Risk Suggested phrases for writing recommendations: - Is reasonable - Can be useful/effective/beneficial - Comparative-Effectiveness Phrases†: - Treatment/strategy A is probably recommended/indicated in preference to treatment B - It is reasonable to choose treatment A over treatment B	LEVEL B-R (Randomized) - Moderate-quality evidence‡ from 1 or more RCTs - Meta-analyses of moderate-quality RCTs
CLASS 2b (WEAK) Benefit ≥ Risk Suggested phrases for writing recommendations: - May/might be reasonable - May/might be considered - Usefulness/effectiveness is unknown/unclear/uncertain or not well-established	LEVEL B-NR (Nonrandomized) - Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies - Meta-analyses of such studies
CLASS 3: No Benefit (MODERATE) Benefit = Risk (Generally, LOE A or B use only) Suggested phrases for writing recommendations: - Is not recommended - Is not indicated/useful/effective/beneficial - Should not be performed/administered/other	LEVEL C-LD (Limited Data) - Randomized or nonrandomized observational or registry studies with limitations of design or execution - Meta-analyses of such studies - Physiological or mechanistic studies in human subjects
Class 3: Harm (STRONG) Risk > Benefit Suggested phrases for writing recommendations: - Potentially harmful - Causes harm - Associated with excess morbidity/mortality - Should not be performed/administered/other	LEVEL C-EO (Expert Opinion) Consensus of expert opinion based on clinical experience

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR 1 and 2a; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely-used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.



PERIOPERATIVE MEDICATIONS

STATINS:

Recommendations for Statins Referenced studies that support the recommendations are summarized in the Online Data Supplement .		
COR	LOE	Recommendations
1	B-NR	1. In patients currently on statins and scheduled for NCS, continuation of statin therapy is recommended to reduce the risk of MACE. ¹⁻³
1	B-R	2. In statin-naïve adult patients who meet criteria for statin use based on ASCVD history or 10-year risk assessment and are scheduled for NCS, perioperative initiation of statin is recommended with intention of long-term use. ^{4,5}

RAAS INHIBITORS:

Recommendations for Perioperative Renin-Angiotensin-Aldosterone System Inhibitors Referenced studies that support the recommendations are summarized in the Online Data Supplement .		
COR	LOE	Recommendations
2b	B-R	1. In select* patients on chronic renin-angiotensin-aldosterone system inhibitors (RAASi) for hypertension undergoing elevated-risk NCS, omission 24 hours before surgery may be beneficial to limit intraoperative hypotension. ¹⁻⁶
2a	C-EO	2. In patients on chronic RAASi for HFrEF, perioperative continuation is reasonable. ^{†1,2}

*Patients with controlled BP and undergoing elevated-risk surgical procedures.

†Modified from the “2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure.”⁷

CALCIUM CHANNEL INHIBITORS

Continuation may be reasonable with recognition of the potential for intraoperative hypotension

(for dihydropyridines) and/or bradycardia
(for nondihydropyridines).



ALPHA-2 RECEPTOR AGONIST

Recommendation for Perioperative Alpha-2 Receptor Agonists Management Referenced studies that support the recommendation are summarized in the Online Data Supplement.		
COR	LOE	Recommendation
3: No benefit	B-R	In patients undergoing NCS, initiation of low-dose clonidine perioperatively is not recommended to reduce cardiovascular risk.¹

ANTIPLATELET THERAPY

Recommendations for Antiplatelet Therapy and Timing of Noncardiac Surgery in Patients With Coronary Artery Disease Referenced studies that support the recommendations are summarized in the Online Data Supplement.		
COR	LOE	Recommendations
1	B-NR	For patients with CAD undergoing elective NCS, management of perioperative antiplatelet therapy and timing of surgery should be determined by a multidisciplinary team with shared decision-making to weigh the risks of bleeding, thrombosis, and consequences of delayed surgery.¹⁻⁷
Timing of NCS After PCI		
1	C-LD	In patients with recent coronary artery balloon angioplasty without stent placement, elective NCS should be delayed for a minimum of 14 days to minimize perioperative MACE.^{8,9}
1	B-NR	In patients with DES-PCI placed for ACS who require elective NCS with interruption of ≥ 1 antiplatelet agents, surgery should ideally be delayed ≥ 12 months to minimize perioperative MACE.^{5,10-15}
2a	B-NR	In patients with DES-PCI placed for CCD who require elective NCS with interruption of ≥ 1 antiplatelet agents, it is reasonable to delay surgery for ≥ 6 months after PCI to minimize perioperative MACE.¹⁶⁻²⁴
2b	B-NR	In patients with DES-PCI who require time-sensitive NCS with interruption of ≥ 1 antiplatelet agents, NCS may be considered ≥ 3 months after PCI if the risk of delaying surgery outweighs the risk of MACE.^{5,23,24}
3: Harm	B-NR	In patients with a recent (≤ 30 days) bare-metal stent (BMS) or DES-PCI, elective NCS requiring interruption of ≥ 1 antiplatelet agents is potentially harmful due to a high risk of stent thrombosis and ischemic complications.^{6,9,22,25,26}

Recommendations for Antiplatelet Therapy and Timing of Noncardiac Surgery in Patients With Coronary Artery Disease (Continued)		
COR	LOE	Recommendations
3: No benefit	B-R	In patients with CAD but without prior PCI who are undergoing elective noncarotid NCS, routine initiation of aspirin is not beneficial.^{27,36}

*Platelet adenosine diphosphate receptor (P2Y₁₂) monotherapy may be considered if surgical bleeding risks are acceptable or if aspirin is not tolerated.



DURATION OF ANTIPLATELET THERAPY

Table 12. Duration of Antiplatelet Therapy Effect

Antiplatelet Agent	Minimum Time From Drug Interruption to Restoration of Platelet Function
Aspirin	4 d
Clopidogrel	5–7 d
Prasugrel	7–10 d
Ticagrelor	3–5 d

Minimum times from drug interruption to noncardiac surgery should be guided by pharmacokinetic data, restoration of platelet function after drug withdrawal, and drug-specific FDA-prescribing information.^{67–71}

ANESTHETIC GOALS:

- To prevent myocardial ischemia by optimizing myocardial oxygen supply and reducing myocardial oxygen demand.
- To monitor for ischemia
- To treat ischemia if it develops
- To maintain hemodynamic stability.

ANAESTHESIA MANAGEMENT

- In patients undergoing arthroplasty, the first anaesthesia method to choose is the central neuraxial block depending on cardiac status
- In arthroplasties, it is known that regional anaesthesia decreases the incidence of deep vein thrombosis and pulmonary embolism, shortens the length of hospital stay and reduces intraoperative blood loss and consequently, the need for transfusion.
- However, despite these advantages, it is sometimes an evaded anaesthetic method because it may cause unwanted side-effects, such as severe hypotension, motor block, urinary retention and pruritus and pose unwanted dural puncture and neurological damage risks.

The advantages of regional techniques include:

- Reduced blood loss, reducing the need for transfusion
- Avoids effects of general anaesthesia on pulmonary function
- May avoid intubation
- Good early postoperative analgesia

- Reduced incidence of postoperative venous thrombosis and
 - pulmonary embolism (sympathectomy-mediated increase in blood flow, and amelioration of the hypercoagulable state associated with surgery)
 - Lower cost
- Simple technique in rural set-up.

Management for hyperdynamic, normal and mild ejection fraction posted for THR: 40-49%

The use of regional anaesthetic techniques has theoretical and practical advantages: epidural anaesthesia reduces preload and afterload, coagulation responses, and in the case of thoracic

epidurals, causes coronary vasodilation. These effects should reduce perioperative myocardial ischemia, but this is not supported by research. However, good epidural analgesia may reduce the incidence of tachycardia arising due to postoperative pain.

Procedure of combined spinal epidural

Patient in sitting position. Under aseptic precautions, Parts painted and draped. Skin infiltrated with 2 % plain lignocaine and 18G Tuohy needle introduced at L2 - L3 intervertebral space. Epidural space confirmed by loss of resistance technique; 20G epidural catheter was threaded up to T10 - T11 level and fixed to skin. 3 ml of 2 % lignocaine test dose {or Fentanyl may be used} was given after negative aspiration. Spinal anesthesia was performed using 23 G



spinal needle in L3 - L4 intervertebral space and clear free flow of CSF noted and Inj. Bupivacaine 0.5 % (H) 2 ml and Fentanyl 25 mcg injected into subarachnoid space. Adequate block was obtained till T 10 level. Inj. Midazolam 1 mg and Oxygen through face mask was given. We preferred combined spinal-epidural (CSE) technique as it permits advantage in early onset of effect as well as the flexibility of graded doses of local anaesthetics through epidural catheter and in maintaining hemodynamic stability.

Management for moderate ejection fraction: 30%-39%

Regional anaesthesia can be used as a choice of anaesthesia with combined spinal epidural with low dose bupivacaine 0.5% heavy and additive fentanyl, epidural volume expansion can be done by injecting saline into the epidural space.

Advantages of EVE in IHD patients

- Lower dose of intrathecal local anaesthetic need – with less sympathetic blockade – reduced risk of sudden hypotension.
- Better control over block height – gradual titration via epidural catheter.
- Improved hemodynamic stability compared to conventional spinal anaesthesia.

Management for low ejection fraction : <30%

The most commonly used and preferred mode of anaesthesia in patients with severely low ejection fraction is usually General Anaesthesia

The essential requirements of general anaesthesia for IHD are avoiding tachycardia and extremes of blood pressure, both of which adversely affect the balance between oxygen supply and demand. These are discussed in detail below during each phase of an operation.

Pre-medication:

- Patient may be tachycardic and require an anxiolytic premedication such as midazolam.
- Beta-blockers also reduce tachycardia, and prevent perioperative myocardial ischemia. A regime of

intravenous Metoprolol followed by postoperative oral treatment.

Induction: All intravenous anaesthetic agents have a direct depressant action on the myocardium, and may also reduce vascular tone. Hence, Etomidate can be used to induce the patient, as it is said to be more cardiostable. In general all agents can be used safely if given slowly in small increments. However, ketamine is unique in causing indirect stimulation of the sympathetic nervous system, leading to both hypertension (increased afterload) and tachycardia. This will be dangerous for a patient with IHD and should be avoided.

high opioid induction

using **large doses of potent opioids** as the primary induction technique to:

- Blunt sympathetic response to laryngoscopy
- Prevent tachycardia & hypertension
- Maintain myocardial oxygen balance

1. Fentanyl

- Most commonly used
- Dose: 5–10 µg/kg IV (high-dose technique)
- Advantages:
- Hemodynamic stability
- Blunts intubation response

Limitation:

- Chest wall rigidity at very high doses

2. Sufentanil

- More potent than fentanyl (~5–10×)
- Dose: 0.5–2 µg/kg IV

Advantages:

- Very stable hemodynamics
- Less histamine release
- Widely used in cardiac anaesthesia

3. Alfentanil

- Dose: 20–50 µg/kg IV
- Rapid onset (good for intubation response control)
- Shorter duration than fentanyl



4. Remifentanyl

- Ultra–short acting (ester metabolism)
- Given as infusion: 0.5–1 µg/kg bolus + infusion
- Excellent control of hemodynamic response

Intubation: Laryngoscopy is a powerful stressor, causing hypertension and tachycardia. This can be avoided with a supplemental dose of intravenous induction agent or opioid e.g., alfentanil, just prior to laryngoscopy.

Drugs to attenuate intubation response

1. Lignocaine IV – Afferent blockade
2. Central suppression – opioids
3. Efferent blockade – beta blockers, calcium channel blockers
4. Direct vasodilator- nitroglycerin
5. Depth of anaesthesia- propofol and volatile agents

Maintenance: Volatile agents have minimal effects on cardiac output, although they do reduce myocardial contractility, especially halothane. They cause vasodilation, and isoflurane has been implicated in the 'coronary steal' syndrome. The theory is that pre-stenotic vasodilation diverts blood away from already ischaemic areas of the myocardium. However, there is doubt as to the clinical significance of this phenomenon. Vagal stimulation due to halothane can cause bradycardias and nodal rhythms. Bradycardias can be beneficial by allowing greater coronary diastolic filling, providing blood pressure is maintained.

Table 3. Factors affecting myocardial oxygen supply and demand	
Oxygen supply	Oxygen demand
Heart rate – diastolic time	Heart rate
Coronary perfusion pressure – aortic diastolic blood pressure – ventricular end-diastolic blood pressure	Ventricular wall tension – preload – afterload
Arterial oxygen content – arterial oxygen partial pressure – haemoglobin concentration	Contractility
Coronary artery diameter	

Analgesia: High doses of opioids reduce the stressor response to surgery. Theoretically, non-steroidal anti-inflammatory drugs (NSAIDs) may have both a useful postoperative analgesic action and an anti-platelet effect which may reduce coronary thrombosis.

Analgesia can be supplemented by peripheral nerve

blocks.

This can be useful in maintaining intraoperative hemodynamics and reducing stressor response and also helpful post operative analgesia.

A femoral 3:1 block or a psoas lumbar plexus block plus lateral cutaneous nerve of thigh block can be used to supplement GA if central neuraxial blocks are contraindicated.

Landmark and Nerve-Stimulation Technique The inguinal ligament is identified between the anterior superior iliac spine and pubic tubercle. The femoral artery can be palpated just below the femoral crease. The needle is inserted approximately 1 cm lateral to the femoral artery pulse in a cephalad direction and local anaesthetic is injected.

Lumbar plexus block has been widely used for hip surgery over the past decades. The clinical efficacy of LBP in hip surgeries has been widely demonstrated as it reduces analgesic use during hip arthroplasty both intra- and postoperatively

The patient is in the lateral decubitus position with a slight forward tilt. The foot on the side to be blocked should be positioned over the dependent leg so that twitches of the quadriceps muscle and/or patella can be seen easily.

Landmarks for the lumbar plexus block include :

1. Midline (spinous processes)
2. – iliac crest
3. Needle insertion labeled 4-cm lateral to the intersection of landmarks 1 and 2

Fascia iliaca block (FIB)

FIB is a fascial block involving the injection of a large volume of local anesthetic below the fascia iliaca to achieve concurrent analgesia of the femoral, lateral femoral cutaneous, and obturator nerves.

The quadratus lumborum is a dorsal muscle that assists in lateral flexion of the trunk on the pelvis and extension movements.

- **Anterior approach:** The needle is advanced medio-laterally until its tip is located between the QL and the psoas major muscle.
- **Lateral approach:** The needle is inserted latero-medially, targeting the lateral side of the QL, where the abdominal wall muscles converge.
- **Posterior approach:** A procedure similar to the lateral approach, but the anesthetic target is posterior to the



QL muscle.

the anterior approach appears most suitable for lower limb analgesia due to its consistent and reproducible anesthetic spread to the lumbar plexus. Some studies show that this approach provides superior analgesia compared to placebo in hip surgery.

PENG (PEricapsular nerve group) BLOCK

This block involves the injection of a local anesthetic in a location proximal to the joint capsule with the rationale of anesthetizing the sensory articular branches that originate from the femoral nerve, obturator nerve, and accessory obturator nerve without involving the motor branches of the thigh muscles

Lumbar Erector Spinae Block (ESPB)

Anatomically, the term *erector spinae* refers to a group of dorsal muscles, also known as “intrinsic dorsal muscles,” that extend from the cervical to the sacral regions. At the lumbar level, the erector spinae group includes the *longissimus dorsi*, *iliocostalis*, and *multifidus* muscles. This group of muscles is located postero-laterally to the spinous processes, adhering ventrally to the transverse processes.

The spinal nerves in the lumbar region, as they exit the intervertebral foramina, divide into anterior and posterior nerves. The anterior nerves form the lumbar and sacral plexuses, while the posterior nerves further divide into medial, intermediate, and lateral branches, which move posteriorly through the fascial layers separating the muscles of this compartment.

The technique involves injecting a local anesthetic close to the vertebral transverse process and utilizing its spread among the fascial planes. In this location, the local anesthetic may spread:

- posteriorly, among the fascial planes of different muscles, reaching the dorsal branches;
- anteriorly, crossing the transverse process to involve the anterior spinal branches;
- cranio-caudally, thus affecting multiple spinal levels.

Reversal and recovery. Reversal of muscle relaxation is best obtained with **Sugammadex**

Also combined anti-cholinesterase/anti-muscarinic agents can be used, these causes tachycardias, and extubation in itself is a stressor. Problems in the

recovery phase which can cause ischaemia include; tachycardia, pain, hypothermia, shivering, hypoxia, and anemia. These should be treated not just in the immediate postoperative period, but throughout the hospital admission. The use of supplemental oxygen in the postoperative period is one of the simplest, yet most effective measures in preventing myocardial ischemia.

INTRA OPERATIVE MONITORING:

Pulse oximetry

ETCO₂

NIBP

IBP

Electrocardiogram (ECG)

- Most commonly used monitor
- Detects ST-segment depression (stress-induced ischemia) **st depression and elevation**
- Not all 12 leads can be monitored simultaneously

♦ Lead sensitivity:

- V₅ → most sensitive single lead (~75%)
- V₄ → ~61%

♦ Lead combinations:

- V₄ + V₅ → ~90% sensitivity
- II + V₅ → ~80%
- II + V₄ + V₅ → ~98% (best detection)

Transesophageal Echocardiography (TEE)

- Most sensitive monitor for ischemia
- Detects regional wall motion abnormalities (RWMA) early
- Provides continuous assessment of LV function

AHA 2024 guidelines:

- Useful in unexplained hemodynamic instability (TEE/FOCUS)
- Not recommended routinely in low-risk patients

Pulmonary Artery Catheter (PAC)

- Measures pulmonary capillary wedge pressure (PCWP)
- Indirect indicator of LV filling and function
- Sudden rise in PCWP → suggests acute LV dysfunction/ischemia

AHA 2024 guidelines:

- Consider in high-risk patients with major hemodynamic compromise
- Routine use not recommended (no mortality benefit).



POSTOPERATIVE:

- The surgeon usually prefers the patients to be placed on their bed in the supine position with the legs abducted using a pillow to prevent dislocation of the prosthesis.
- Anaesthesia techniques which lead to rapid recovery of airway control and patient cooperation is therefore an advantage. Patients are usually mobilized at 24-48 hours and simple IM/ subcutaneous opioids with regular paracetamol or NSAIDs are usually sufficient for postoperative analgesia in a simple THR.
- If an epidural has been inserted, a postoperative infusion can be used but needs to cease prior to mobilization. PCA is a suitable alternative if pain relief is needed for an extended period.
- As described above, peripheral nerve blocks can be supplemented in post operative analgesia as well. Fentanyl patch or iv infusion of fentanyl can be used.

COMPLICATIONS:

1. ARRHYTHMIAS:

Myocardial ischemia and conduction abnormalities can all contribute to the development of perioperative arrhythmias and heart block. Consequently, anesthesia providers must be prepared to treat both chronic and new-onset cardiac rhythm abnormalities.

• SUPRAVENTRICULAR TACHYCARDIA:

SVT presents with loss of the "P" wave on the ECG with a fast ventricular response. It can have hemodynamic consequences secondary to loss of AV synchrony and decreased diastolic filling time. Most SVTs occur secondary to a reentrant mechanism.

Management:

If patient is hemodynamically stable:

- ▶ Continuous ECG monitoring
- ▶ Perform vagal manoeuvre such as Valsalva, carotid sinus massage.
- ▶ Drugs such as adenosine can be used- 6mg iv bolus followed by 0.9% NS iv flush. If it is unsuccessful after 2

mins repeat 12mg, can repeat upto 30mg.

If patient is hemodynamically unstable:

- ▶ Can be treated with three synchronized DC cardioversions. Cardioversion is started with 70-120 joules which can be increased upto 120-360 joules until rhythm gets reverted.
- ▶ Check for electrolyte abnormalities and correct the same.
- ▶ If rhythm not reverted, then give iv amiodarone 300mg iv over 20mins followed by shock.

ATRIAL FIBRILLATION:

35% of cardiac surgery patients develop postoperative AF.

When AF develops perioperatively, rate control with β -blockers can often be instituted. Chemical cardioversion can be attempted with amiodarone or procainamide.

If the duration of AF is greater than 48 hours, or unknown, ACC/AHA guidelines recommend anticoagulation for 3 weeks prior to, and 4 weeks following, either electrical or chemical cardioversion.

TEE can be performed to rule out the presence of left atrial or left atrial appendage thrombus.

If AF develop postoperatively, ventricular rate response can be controlled with AV nodal blocking agents unless contraindicated.

If AF result in hemodynamic instability, synchronized cardioversion should be attempted.

Patients at high risk of AF following cardiac surgery can be treated with prophylactic amiodarone.

VENTRICULAR ARRHYTHMIA:

Ventricular premature contractions (VPCs) can appear perioperatively secondary to electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia), acidosis, ischemia, embolic phenomenon, mechanical irritation of the heart from central lines, cardiac manipulation, and drug effects.



Classification by Clinical Presentation		
Hemodynamically stable	Asymptomatic Minimal symptoms, e.g., palpitations	The absence of symptoms that could result from an arrhythmia. Patient reports palpitations felt in either the chest, throat, or neck as described by the following: • Heartbeat sensations that feel like pounding or racing • An unpleasant awareness of heartbeat • Feeling skipped beats or a pause
Hemodynamically unstable	Presyncope	Patient reports presyncope as described by the following: • Dizziness • Lightheadedness • Feeling faint • "Graying out"
	Syncope	Sudden loss of consciousness with loss of postural tone, not related to anesthesia, with spontaneous recovery as reported by the patient or observer. Patient may experience syncope when supine.
	Sudden cardiac death	Death from an unexpected circulatory arrest, usually due to a cardiac arrhythmia occurring within an hour of the onset of symptoms.
	Sudden cardiac arrest	Death from an unexpected circulatory arrest, usually due to a cardiac arrhythmia occurring within an hour of the onset of symptoms, in whom medical intervention (e.g., defibrillation) reverses the event.
Classification by Electrocardiography		
Nonsustained VT	Monomorphic Polymorphic	Three or more beats in duration, terminating spontaneously in less than 30 s. VT is a cardiac arrhythmia of three or more consecutive complexes in duration emanating from the ventricles at a rate of greater than 100 bpm (cycle length less than 600 ms) Nonsustained VT with a single QRS morphology. Nonsustained VT with a changing QRS morphology at cycle length between 600 and 180 ms.
Sustained VT	Monomorphic Polymorphic	VT greater than 30 s in duration and/or requiring termination due to hemodynamic compromise in less than 30 s. Sustained VT with a stable single QRS morphology. Sustained VT with a changing or multiform QRS morphology at cycle length between 600 and 180 ms.
Bundle-branch reentrant tachycardia		VT due to reentry involving the His-Purkinje system, usually with LBBB morphology; this usually occurs in the setting of cardiomyopathy.
Bidirectional VT		VT with a beat-to-beat alternans in the QRS frontal plane axis, often associated with digitalis toxicity.
Torsades de pointes		Characterized by VT associated with a long QT or QTc, and electrocardiographically characterized by twisting of the peaks of the QRS complexes around the isoelectric line during the arrhythmia: • "Typical," initiated following "short-long-short" coupling intervals. • Short coupled variant initiated by normal-short coupling.
Ventricular flutter		A regular (cycle length variability 30 ms or less) ventricular arrhythmia approximately 300 bpm (cycle length—200 ms) with a monomorphic appearance; no isoelectric interval between successive QRS complexes.
Ventricular fibrillation		Rapid, usually more than 300 bpm/200 ms (cycle length 180 ms or less), grossly irregular ventricular rhythm with marked variability in QRS cycle length, morphology, and amplitude.



Ventricular tachycardia:

Nonsustained VT lasts less than 30 s and spontaneously terminates, whereas sustained VT persists longer than 30secs. VT is either monomorphic or polymorphic, depending on the QRS complex. If the QRS complex morphology changes, it is designated as polymorphic VT. Torsades des pointes is a form of VT associated with a prolonged QT interval.

Management of monomorphic VT:

If hemodynamically unstable:

▶ DC cardioversion or start CPR till it is available. Follow ACLS protocol.

If hemodynamically stable:

▶ Pharmacological cardioversion with iv amiodarone 150mg over 10 mins followed by 1mg/min for next 6 hrs or

▶ Iv lignocaine 1-1.5mg/kg bolus followed by 1mg/kg/min. or

▶ Iv procainamide 20-50mg/min until arrhythmia terminates. Maximum dose is 15-17mg/kg.

Management of polymorphic VT:

If hemodynamically unstable:

▶ DC cardioversion. Follow ACLS protocol.

If hemodynamically stable:

▶ IV MgSO₄ 8mmol over 15mins then 72mmol over 24hrs.

▶ Avoid amiodarone, procainamide and sotalol. They can worsen it.

VENTRICULAR FIBRILLATION:

Perioperative ventricular fibrillation requires defibrillation and the use of resuscitation algorithms. Amiodarone can be used to stabilize the rhythm following successful defibrillation.

2. PERIOPERATIVE MYOCARDIAL INFARCTION:

Myocardial injury is defined as elevated troponin levels above the 99th percentile, occurs in upto 20% of patients after non cardiac surgery. Patients who undergo urgent hip surgery have an incidence of PMI of 5%–7%, whereas

fewer than 3% of patients who undergo elective total hip or knee arthroplasty have a PMI.

Most PMIs occur soon after surgery (1–4 days), and 90% occur within 7 days. Most are asymptomatic, of the non-Q-wave type (60%–100%), and are commonly preceded by ST-segment depression rather than ST-segment elevation. Long-duration ST-segment changes (a single episode lasting >20–30 minutes or a cumulative duration >1–2 hours, either

intraoperatively or postoperatively) seem to be more important than just the presence of postoperative ST-segment depression in the evolution of adverse cardiac outcomes.

DIAGNOSIS:

According to the definition of the World Health Organization (WHO), at least two of three criteria must be fulfilled to diagnose MI:

(I) typical ischemic chest pain

(ii) (ii) increased serum concentration of creatine kinase (CK)-MB isoenzyme

(iii) typical electrocardiographic findings, including development of pathological Q-waves.

The recent AHA guidelines recommendation for MINS surveillance:

In patients with known CVD, symptoms of CVD or age >65 yrs with cardiovascular risk factors undergoing elevated risk non cardiac surgery, it may be reasonable to measure cTn at 24 and 48 hrs after surgery to identify myocardial injury.

In patients undergoing low risk non cardiac surgery routine post-operative screening with cTn levels is not indicated without signs or symptoms suggestive of myocardial ischemia or MI.

MANAGEMENT:

In the perioperative setting, Urgent angiography and percutaneous coronary interventions are reserved for patients with STEMI or those with NSTEMI who are hemodynamically unstable.

The main stay of management is medical line of treatment consisting of good pain control, beta blockade, antiplatelet agents and anticoagulants.

▶ Oxygen supplementation

▶ Pain relief

▶ Nitrates

▶ Anti platelet agents

▶ Anti coagulants

▶ Beta blockers

▶ Statins

3. HEART FAILURE:

Heart failure is a clinical syndrome caused by structural or functional cardiac abnormalities that impair ventricular filling or ejection. It is classified into:

• HFrEF (EF <40%)

• HFmrEF (EF 41–49%)



- HFrEF (EF $\geq$ 50%)

As per American College of Cardiology / American Heart Association 2024 guidelines, HF is a major independent risk factor for perioperative complications in noncardiac surgery.

Patients with HF have increased risk of:

- Acute HF exacerbation
- Myocardial infarction
- Increased short- and long-term mortality

Importantly, this risk persists even in asymptomatic patients.

The severity of risk correlates with LV function, with the highest risk in EF <30%.

Term	Definition	Recommended action
Heart failure	HFrEF or HFpEF with current or prior symptoms or structural/functional abnormalities	Risk stratify, continue GDMT (hold SGLT2i)
Decompensated HF	Recent/worsening symptoms, fluid overload, hypotension	Defer elective NCS until optimized
Compensated HF	Stable, no congestion, on GDMT	Proceed with NCS; continue GDMT, hold SGLT2i 3 days prior

(GDMT: guideline-directed medical therapy; HF: heart failure; HFpEF: heart failure with preserved ejection fraction; HFrEF: heart failure with reduced ejection fraction; NCS: noncardiac surgery)

Compensated Heart Failure

- Stable clinical state with no signs of volume overload or hypoperfusion
- Patients may be asymptomatic or mildly symptomatic
- Maintained on GDMT (β -blockers, ACEI/ARNI, MRA, SGLT2 inhibitors)
- SGLT2 inhibitors should be withheld perioperatively (risk of hypotension, volume depletion, euglycemic DKA)
- Elective noncardiac surgery can proceed with proper monitoring and risk stratification

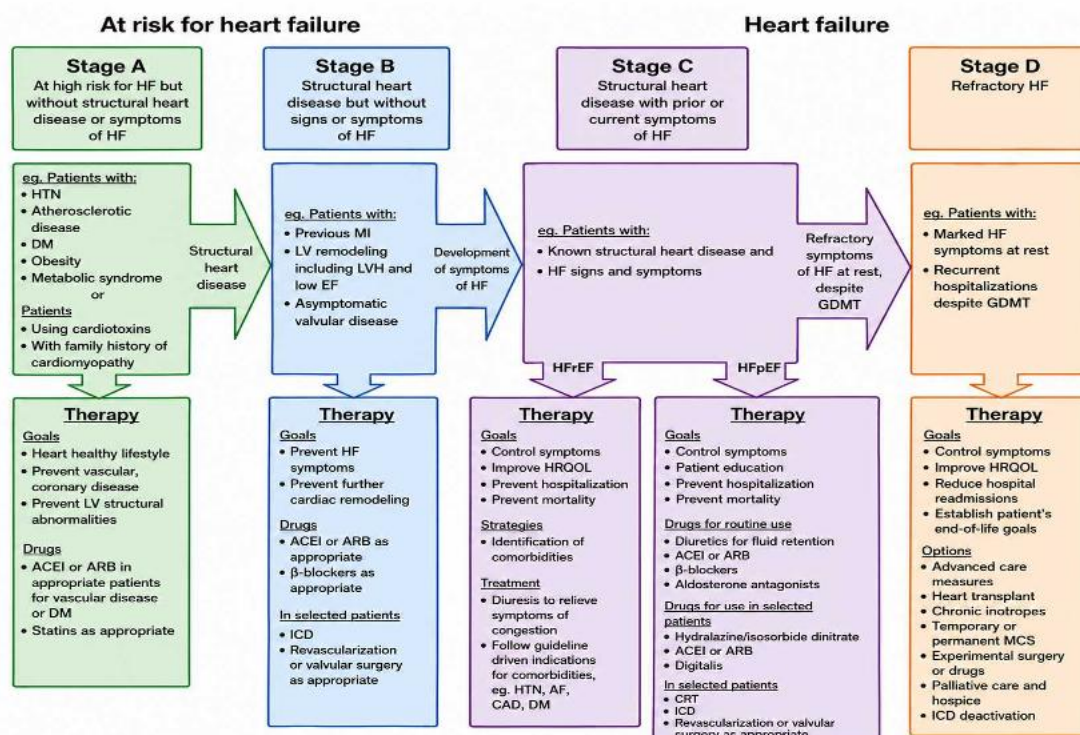
Decompensated Heart Failure

- Clinically unstable state with worsening/new symptoms: dyspnea, orthopnea, $\uparrow$ JVP, pulmonary/peripheral edema, hypotension
- May include recent hospitalization, low cardiac output, organ hypoperfusion
- Classified as an “active cardiac condition”
- Elective surgery should be deferred until stabilization
- Requires optimization: volume control, adjustment/initiation of GDMT, close monitoring

2024 ACC/AHA GUIDELINES:

- HF significantly increases perioperative risk
- Compensated HF $\rightarrow$ surgery possible with precautions
- Decompensated HF $\rightarrow$ postpone surgery until optimized.

MANAGEMENT OF HEART FAILURE



Cardiogenic Shock And Management

Cardiogenic shock is defined as a primary cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion.

Clinical criteria:

Systolic blood pressure of less than or equal to 90 mm Hg for greater than or equal to 30 minutes or support to maintain systolic blood pressure less than or equal to 90 mm Hg and urine output less than or equal to 30 mL/hr or cool extremities.

Hemodynamic criteria:

Depressed cardiac index (less than or equal to 2.2 liters per minute per square meter of body surface area) and an elevated pulmonary-capillary wedge pressure greater than 15 mm Hg.

The most common cause of cardiogenic shock is acute myocardial infarction.

Pathophysiology:

Cardiogenic shock results from severe myocardial ischemia causing both systolic and diastolic left ventricular dysfunction, leading to markedly reduced contractility and cardiac output. This produces hypotension, which further worsens coronary perfusion and perpetuates myocardial ischemia - creating a vicious cycle. Compensatory mechanisms include sympathetic activation causing peripheral vasoconstriction (improving coronary perfusion but increasing afterload) and tachycardia (which raises myocardial oxygen demand and aggravates ischemia). Subsequently, inflammatory mediators such as interleukin-1, tumor necrosis factor- α , and interleukin-6, along with nitric oxide and peroxynitrite, cause pathological vasodilation and direct myocardial depression. If untreated, this cycle leads to systemic hypoperfusion, tissue hypoxia, multiorgan failure, and death.

MANAGEMENT:

Medical Management

The goal of medical management is to restore cardiac output and prevent irreversible end-organ damage rapidly.

- The optimal choice of a vasoactive agent in cardiogenic shock is unclear.
- Norepinephrine is preferred over dopamine in

patients with severe hypotension (systolic blood pressure less than 70 mm Hg)

- Dobutamine is widely used, has beta-1 and beta-2 agonist properties, which can improve myocardial contractility.
- Milrinone can also be used.
- Saline or lactated ringer solution greater than 200 ml per 15 to 30 minutes is indicated in patients with no signs of fluid overload.
- Fibrinolytic therapy should be administered to patients who are unsuitable candidates for either percutaneous coronary intervention or coronary artery bypass graft if there are no contraindications.
- Patients with myocardial infarction or acute coronary syndrome are given aspirin and heparin. They have been shown to be effective in reducing mortality.
- Diuretics such as furosemide play a role in decreasing plasma volume and edema
- Therapeutic hypothermia is established for out-of-hospital cardiac arrest patients with shockable rhythm to prevent brain injury and improve survival.
- Primary percutaneous coronary intervention (PCI) should be performed, irrespective of time delay from the onset of myocardial infarction

Mechanical Circulatory Support

mechanical circulatory support (MCS) therapy to improve end-organ perfusion may be required. An experienced interprofessional team should evaluate MCS.

- Percutaneous circulatory assist devices provide superior hemodynamic support compared with pharmacologic therapy.
- Intra-aortic balloon pumps can be considered but are less likely to provide a benefit compared to other MCS devices and should not be routinely used but may still play an important role in patients with acute severe mitral regurgitation, ventricular septal defects, or when other MCS devices cannot be placed.
- Extracorporeal membrane oxygenation (ECMO) is indicated in patients with poor



oxygenation not expected to improve with alternative temporary mechanical support devices rapidly.

- In properly selected patients not likely to recover from cardiogenic shock without long-term MCS support, a ventricular assist device can be implanted as a bridge to recovery, bridge to bridge, bridge to transplant, or destination therapy.
- Cardiac transplantation may be performed in suitable candidates not expected to recover after MCS device implantation may be the only hope for meaningful long-term recovery. However, it remains a very limited option owing to the low number of available hearts.

Short-term mechanical circulatory assist

- IABP (Intra-aortic balloon pump)
- Non-IABP percutaneous mechanical devices
- ECMO

Strategy for managing severe, refractory heart failure when conventional measures (inotropes, vasopressors, IABP) fail- **Left Ventricular Assist Devices (LVAD)**

Classification of LVADs

- A. Pulsatile-flow LVADs (1st generation)
- B. Continuous-flow LVADs (current standard)

Temporary LVAD:

Used in ICU/OT for acute cardiogenic shock

Examples:

- Impella
- TandemHeart

1. FLASH PULMONARY EDEMA:

Pulmonary venous hypertension may result from left ventricular failure, mitral stenosis, or left atrial obstruction. Increases in pulmonary blood flow that exceed the capacity of the pulmonary vasculature will also raise capillary hydrostatic pressure. This leads to pulmonary edema.

MANAGEMENT:

Management of cardiogenic pulmonary edema involves decreasing the pressure in the pulmonary capillaries. Generally, this includes measures to improve left ventricular function, correct fluid

overload with diuretics, or reduce pulmonary blood flow. Pharmacological treatment of acute cardiogenic pulmonary edema has included oxygen, morphine, diuretics (especially loop diuretics), vasodilators such as nitrates, and inotropes such as dobutamine or milrinone. By reducing left atrial pressure, pulmonary congestion is relieved; by reducing systemic vascular resistance, cardiac output may be improved. Positive airway pressure therapy is also a useful adjunct for improving oxygenation. When pulmonary edema is a consequence of left ventricular failure from acute coronary ischemia, intraaortic balloon counterpulsation or other cardiac assist devices may be needed in addition to thrombolysis and revascularization.

UNSTABLE ANGINA

Unstable angina is a component of Acute Coronary Syndrome (ACS), typically caused by the rupture of a coronary plaque and the formation of a nonocclusive thrombus, reducing blood flow to the heart. Intraoperatively, this is often triggered by sudden increases in myocardial oxygen demand (tachycardia, hypertension) or decreases in supply.

MANAGEMENT:

1. Increase oxygen supply

Give 100% O₂, ensure adequate ventilation, correct hypoxia, anemia, hypotension, acidosis, hypothermia.

2. Reduce myocardial oxygen demand

Deepen anesthesia/analgesia, treat pain and sympathetic response.

3. Analgesia in unstable angina is primarily achieved with **opioids (morphine/fentanyl)** to reduce pain and sympathetic drive, supplemented by nitrates and **anxiolytics**, while maintaining hemodynamic stability. Maintain :

- HR around **60–80/min**
- Avoid tachycardia
- Maintain BP near baseline
- Avoid both hypotension and severe hypertension

4. Treat hemodynamics

5. Anti-ischemic therapy

- **Nitroglycerin IV** if BP allows: reduces preload and coronary spasm.
- Consider **beta blocker** if tachycardic and no contraindication.



- Continue/consider **statin** perioperatively.

7. Monitoring and investigations

- Continuous ECG with ST analysis, leads II and V5 useful.
- Arterial line if not already placed.
- ABG, Hb, electrolytes.
- 12-lead ECG as soon as possible.
- Troponin serially postoperatively.
- Consider TEE if available for new regional wall motion abnormality.

8. Antiplatelet/anticoagulation

Discuss urgently with surgeon + cardiologist because bleeding risk matters. Aspirin/heparin may be needed if ACS strongly suspected, but depends on surgical field and bleeding risk.

9. Postoperative care

SURGICAL COMPLICATIONS:

1. bone Cement Implantation Syndrome:

BCIS is a cardiovascular collapse syndrome seen during cemented orthopedic procedures (e.g., fracture neck femur, total hip replacement), especially in elderly patients.

Pathophysiology

- Methylmethacrylate (MMA) cement polymerization → exothermic expansion
- ↑ Intramedullary pressure → embolization of:
 - Fat, marrow, cement particles, air → pulmonary circulation
 - Residual monomer + mediators cause:
 - Vasodilation → ↓ SVR → hypotension
 - Histamine release → cardiovascular instability
 - Thromboplastin release → platelet aggregation & microthrombi
 - Endocannabinoids → further vasodilation
- Result → Pulmonary hypertension + RV dysfunction

MANAGEMENT:

BCIS may be a reversible complication, treatment is to maintain both coronary perfusion pressure and right heart function. We have to support the cardiovascular system by treating right heart failure, administering 100% oxygen, and maintaining aggressive volume

support. When CVP is high, fluid infusion should be stopped. Vasopressors, such as phenylephrine or norepinephrine, can be titrated to restore adequate aortic perfusion. To improve contractility and ventricular function, inotropes, such as dobutamine, can be started provided there is adequate right ventricular perfusion pressure to meet the increased oxygen demand caused by these agents. BCIS is a time-limited process.

Strategies to minimize the effects of this complication include:—

- ▶ Good anesthesia technique:-
 - Increase inspired oxygen concentration prior to cementing
 - Maintaining normovolemia, monitor blood loss carefully
- ▶ Good surgical technique-
 - Good hemostasis before cementing
 - Good intramedullary lavage
 - Minimising length of prosthesis
 - Minimising the pressure applied during insertion
 - Venting the distal femur to relieve intramedullary pressure
 - Use uncemented femoral component.

2.DEEP VEIN THROMBOSIS AND THROMBOEMBOLISM

Deep vein thrombosis (DVT) and pulmonary embolism (PE) are common complications in elderly patients undergoing orthopedic or pelvic surgery, primarily due to prolonged pre- and postoperative immobilization, reduced limb blood flow (e.g., kinking of femoral vessels), and a postoperative hypercoagulable state. Therefore, routine prophylaxis with either low-dose unfractionated heparin (5000 IU SC BD/TDS) or low molecular weight heparin is recommended in all high-risk patients, especially those with malignancy or expected bed rest >1 day. PE should be suspected in patients developing sudden respiratory

distress, hypoxia, hypotension, bronchospasm, tachycardia, or unexplained cough, with the classic triad being dyspnea, hemoptysis, and pleuritic chest pain. Investigations may show hypoxemia with hypocapnia on ABG, while ECG (occasionally S1Q3T3) and chest X-ray are often normal; confirmation is by CT



pulmonary angiography, V/Q scan, or echocardiography. Immediate intravenous heparin should be started when PE is suspected, even before confirmation, followed by heparin infusion; thrombolysis is reserved for hemodynamically unstable patients (though limited in the immediate postoperative period), and surgical embolectomy may be considered in severe cases.

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AHA/ACC/ACS/ASNC/HRS/SCA/SCCT/SCMR/SVM Guideline for Perioperative Cardiovascular Management for Noncardiac Surgery

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14th JUNE 2026
09:10AM - 09:50AM

CASE PRESENTATION ON THYROTOXICOSIS (PART-1)

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Case scenario:

A 35 years old female patient, presents with complaints of diffuse neck swelling since 6 months, which was insidious in onset, gradually progressive. Patient gives history of weight loss, palpitation, loss of sleep, anxiety and menstrual disturbances. The patient has been started on oral anti-thyroid medications and propranolol. The diagnosis of hyperthyroidism has been established.

1. What is the definition of hyperthyroidism?

Hyperthyroidism is a clinical state resulting from excess synthesis and secretion of thyroid hormones—thyroxine (T4) and triiodothyronine (T3)—by the thyroid gland, leading to a hypermetabolic state in the body.

2. What is the classification of hyperthyroidism?

A. Primary hyperthyroidism

Due to intrinsic disease of the thyroid → ↑ T3, T4 with ↓ Thyroid Stimulating Hormone

- Graves' disease
- Toxic multinodular goiter
- Toxic adenoma
- Subacute thyroiditis
- Postpartum thyroiditis

- Drug induced thyroiditis

- Functioning thyroid carcinoma
- B. Secondary hyperthyroidism

Due to excess Thyroid Stimulating Hormone from pituitary

- Thyroid Stimulating Hormone—secreting pituitary adenoma
- Thyroid hormone resistant syndrome
- Chorionic gonadotropin secreting tumors

3. What are the symptoms and signs of primary and secondary hyperthyroidism?

Primary hyperthyroidism

Symptoms:

- Weight loss despite increased appetite
- Heat intolerance
- Excessive sweating
- Palpitations
- Nervousness, irritability, anxiety
- Fatigue, proximal muscle weakness
- Diarrhea
- Menstrual irregularities (oligomenorrhea / amenorrhea)
- Sleep disturbance (insomnia) Eye discomfort

Signs:

- Thin built, restless, hyperactive
- Warm, moist skin
- Tachycardia



- Wide pulse pressure
- Possible atrial fibrillation
- Fine tremors
- Warm palms
- Exophthalmos (proptosis)
- Lid lag
- Lid retraction
- Periorbital edema
- Proximal myopathy
- Hyperreflexia

Secondary hyperthyroidism

Symptoms:

- Weight loss
- Heat intolerance
- Palpitations
- Ectopic beats
- Arrhythmias
- Chest pain
- Dyspnea on exertion

Central nervous system features may or may not be affected, which may include:

- Nervousness, tremulousness
- Increased appetite
- Headache
- Visual disturbances (blurring, field defects)

Signs

- Tachycardia
- Tremors
- Warm skin / palms
- Visual field defects (classically bitemporal hemianopia)
- Possible cranial nerve involvement

Difference between Primary and Secondary Thyrotoxicosis

PRIMARY	SECONDARY
Goitre appears along with toxic symptoms	Goiter appears first and toxic symptom appear later.
Goitre is diffuse, vascular bruit may be present	Goiter is nodular
Symptoms appear suddenly and are severe.	Symptoms appear gradually and are mild.
CNS manifestation may dominate. (Tremors, increased tendon reflexes)	CVS manifestations dominate
Exophthalmos and eye signs are common	Exophthalmos and eye signs are rare.

4. what are the eye signs in hyperthyroidism?

- Dalrymple sign (lid retraction)

- Abnormal elevation of upper eyelid causing a “staring look” with sclera visible above the cornea
- Von Graefe sign (lid lag)→ Upper eyelid lags behind the eyeball when the patient looks downward
- Stellwag sign
→ Infrequent or incomplete blinking
- Möbius sign
→ Inability to maintain convergence of eyes on a near object
- Joffroy sign
→ Absence of forehead wrinkling on upward gaze
- Kocher sign
→ Sudden jerky retraction of upper eyelid on upward gaze
- Exophthalmos (proptosis)
→ Forward protrusion of the eyeball due to increased orbital contents

5. How does thyroid affect vital organ function?

- Function of thyroid hormone is to regulate cellular metabolism throughout the body
- They lead to increase in basal metabolic rate, increased oxygen consumption and increased body heat production
- On lipid metabolism:
 - ▶ Increased thyroid hormone level increases fat metabolism.
 - ▶ Increased thyroid hormone level increases fat mobilisation leading to increased concentration of fatty acids.
 - ▶ Also enhance oxidation of fatty acids in many tissues.
- On carbohydrate metabolism:
 - ▶ Thyroid hormone stimulates almost all aspects of carbohydrate metabolism including enhancement of insulin dependent entry of glucose
 - ▶ Increase gluconeogenesis and glycogenolysis to generate free glucose.
- On cardiovascular system:
 - ▶ Thyroid hormones act directly on the cardiac myocytes and vascular smooth muscle cells in the heart.
- They bind to the nuclear receptors and enhance systolic contractile function and diastolic relaxation.
 - ▶ They increase myocardial contractility directly

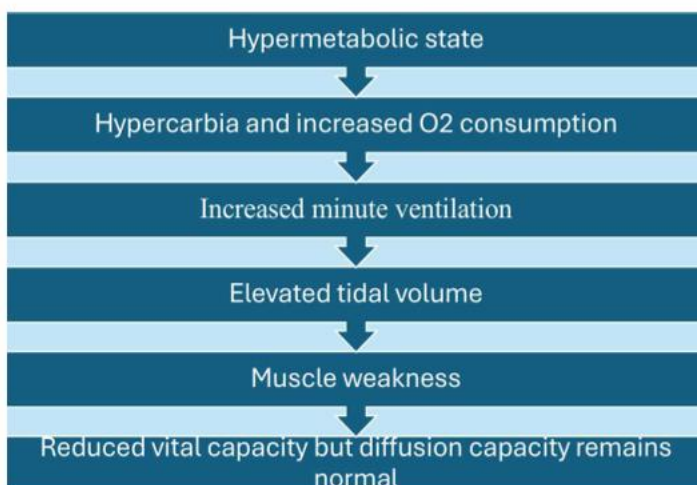


- ▶ Decreases SVR via direct vasodilatation and increase intravascular volume leading to enhanced blood flow to multiple organs.
- On Central nervous system:
 - ▶ Both increase and decrease concentration of thyroid hormone leads to alteration in mental state.
 - ▶ Hypothyroidism leads to mental sluggishness while too much induces anxiety and nervousness.
 - ▶ They play a role in brain development

6. what are the systemic effects of thyrotoxicosis on various systems?

- Cardiovascular effects
- Increased stroke volume
- Increased cardiac output
- Increased oxygen consumption
- Reduced systemic vascular resistance
- Reduced peripheral vascular resistance
- Increased myocardial contractility
- Increased automaticity and excitability
- Widened pulse pressure
- Atrial fibrillation/ sinus tachycardia
- Complete heart block
- Ventricular arrhythmias
- High output cardiac failure/ Thyrotoxic cardio myopathy

▶ Respiratory effects



▶ Neurological manifestations

- Stupor
- Obtundation
- Seizures
- Myopathy
- Coma

▶ Hematological

- Anemia
- Neutropenia
- Thrombocytopenia

▶ Gastrointestinal effects

- Diarrhea
- Vomiting

▶ Musculoskeletal effects

- Muscle weakness
- Osteoporosis
- Bone resorption

7. Explain the steps of thyroid hormone synthesis?

A. Iodide Trapping (Basolateral Uptake):

- Iodide (I^-) is actively transported from blood → follicular cell
- Via Na^+/I^- symporter (NIS) on basolateral membrane
- Driven by Na^+ gradient (Na^+/K^+ ATPase)
- Regulation: Stimulated by TSH

B. Oxidation of Iodide → Iodine:

- Occurs at apical membrane (colloid interface)
- $I^- \rightarrow I^0$ (active iodine)
- Enzyme: Thyroid peroxidase (TPO)
- Requires H_2O_2

C. Organification (Iodination of Tyrosine):

- Iodine binds to tyrosine residues on thyroglobulin
- Forms:
 - MIT (monoiodotyrosine)
 - DIT (diiodotyrosine)
- Also catalyzed by TPO

D. Coupling Reaction (Hormone Formation):

- Coupling of iodotyrosines (within thyroglobulin):
 - MIT + DIT → T3
 - DIT + DIT → T4
- Also catalyzed by TPO

E. Storage in Colloid:

- Thyroid is unique → stores large amounts of hormone extracellularly
- Stored as iodinated thyroglobulin in colloid
- Can supply hormones for 2–3 months

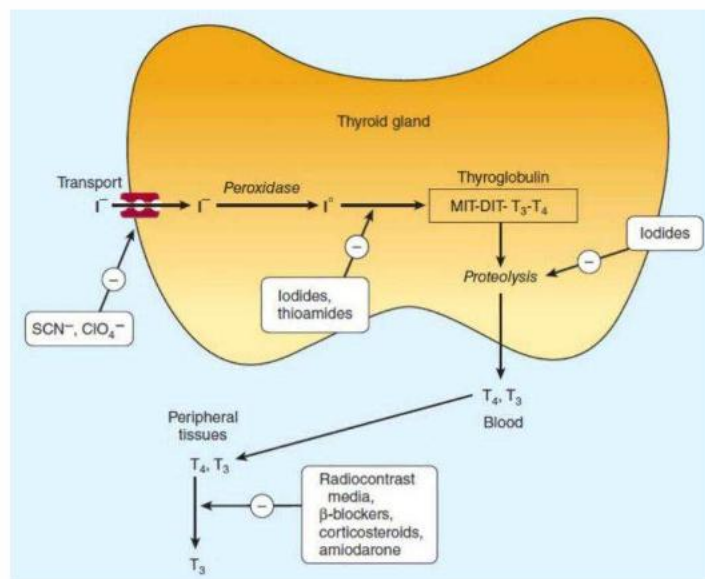


F. Endocytosis & Proteolysis (Release):

- Colloid taken back into follicular cells (endocytosis)
- Lysosomes cleave Tg → release:
- T₃ & T₄ → blood
- MIT & DIT (recycled)
- Stimulated by TSH

G. Peripheral Conversion:

- Most T₃ is formed outside thyroid
- T₄ → T₃ via 5'-deiodinase
- Types:
- Type 1: liver, kidney
- Type 2: brain, pituitary
- Type 3: forms reverse T₃ (inactive)



8. How is thyroid hormone regulated?

Regulation of thyroid function is controlled by hypothalamus, pituitary and thyroid glands which participate in a classic feedback control system. TRH is secreted in the hypothalamus which regulates the secretion of TSH from the anterior pituitary. TSH binds to specific receptors on thyroid cell membrane and enhances all processes of synthesis and secretion of T₃ and T₄.

9. What are the problems in patients with large thyroid mass?

A. Airway-related problems

- Tracheal deviation → difficult laryngoscopy
- Tracheal compression → dyspnea, stridor
- Tracheomalacia → airway collapse after extubation
- Distorted airway anatomy → difficult intubation and difficulty in airway blocks

B. Respiratory symptoms

- Dyspnea
- Orthopnea
- Stridor

C. Esophageal compression

- Dysphagia

D. Vascular compression

- Superior vena cava obstruction
- facial congestion, edema, dilated neck veins

E. Nerve involvement

- Recurrent laryngeal nerve compression
- hoarseness of voice
- Rarely superior laryngeal nerve involvement
- voice fatigue, loss of pitch

10. What is the importance of knowing functional capacity in a thyroid patient?

Hyperthyroidism causes hyper metabolism resulting in the resting energy expenditure. Patient with METs < 4 may experience severe fatigue and reduced exercise tolerance. Knowing METs score allows us to determine if heart can handle metabolic demands. Low METs score indicate high risk for heart failure. Improvement in METs score following restoration of euthyroid state after antithyroid therapy serves as a marker for efficient recovery. Overall low METs (less than 4) in a hyperthyroid patient signifies poor functional status and requires vigilant monitoring.

11. Classify antithyroid drugs

► Hormone synthesis inhibitors:

- Propylthiouracil (PTU)
- Carbimazole
- Methimazole

► Hormone release inhibitors

- Iodine
- Iodides of sodium and potassium
- Organic iodide

► Iodide trapping inhibitors

- Thiocyanates
- Perchlorates
- Nitrates

► Thyroid tissue destroyers

- Radioactive iodine (I¹³¹)



DRUG	DOSE	MECHANISM OF ACTION
Carbimazole	Initial: 15-40mg daily Maintenance: 5-15mg daily Takes 6-8 weeks to work	Prodrug rapidly converted to methimazole. Prevents synthesis of T3 and T4 by blocking oxidation of iodide to iodine and inhibiting thyroid peroxidase
Propylthiouracil	Initial: 200-400mg daily Maintenance: 50-150mg daily Takes 6-8 weeks	Blocks iodination of tyrosine residues present in thyroglobulin. Inhibits conversion of T4 – T3
Iodide/Iodine	Lugol's solution: 5g Iodine solution in 10g Potassium iodide: 0.1-0.3ml TDS	Large doses of Iodide inhibit hormone production. Reduced the effect of TSH. Marked reduction in thyroid vascularity over 10-14days
Propanolol	Oral: 40-80mg TDS (May need higher dose as metabolism increased) IV: 0.5mg titrated to effect	Controls sympathetic effects of thyrotoxic crisis. Blocks peripheral conversion of T4 to T3

12. Why is the diagnosis of toxic multinodular goitre important, and how are they different from Graves disease?

- Toxic multinodular goitre usually occurs above 50 years of age.
- They produce the most extreme thyroid enlargement
- They cause compressive symptoms like dysphagia, difficulty in breathing and Superior vena cava obstruction
- Hypermetabolism is usually less severe when compared to graves disease
- No associated ophthalmopathy / dermopathy as in graves disease

13. What is Wolff-Chaikoff effect and Jod-Basedow's phenomenon?

A. Wolff–Chaikoff effect

An autoregulatory phenomenon in which excess iodine intake acutely inhibits thyroid hormone synthesis and release.

Mechanism:

- High iodide → inhibits thyroid peroxidase-mediated organification
- Decreases formation of T3 and T4



Clinical significance:

- Used preoperatively in hyperthyroid patients
- Decreases thyroid gland vascularity and hormone release
- Effect is temporary

B. Jod-Basedow phenomenon

Development of hyperthyroidism following iodine administration, especially in patients with underlying autonomous thyroid tissue.

Mechanism:

- Autonomous thyroid nodules do not autoregulate
- Excess iodine → increased hormone synthesis

Seen in:

- Multinodular goiter
- Iodine-deficient regions after iodine exposure

Clinical significance:

- Can occur after:
 - ♦ Iodinated contrast
 - ♦ Amiodarone
- Important perioperative risk → precipitates thyrotoxicosis

14. Mention the grades of goitre.

Grade	Description
0	No palpable or visible goitre
1	Palpable goitre, not visible when the neck is in normal position
2	Visible swelling in the neck when neck is in normal position
3	Very pronounced swelling, visible from the distance

15. How do you grade retrosternal goitre and how it is important?

Grade	Anatomical location	Approach
1	Above aortic arch (above T4)	Cervical
2	Aortic arch to pericardium	Manubriectomy
3	Below right atrium	Full sternotomy

16. What are the problems of retrosternal extension of thyroid mass?

1. Airway complications (most important)

- Tracheal compression → dyspnea, stridor
- Tracheal deviation

- Tracheomalacia (long-standing compression → airway collapse after extubation)
- Difficult intubation and ventilation

2. Vascular compression

- Superior vena cava obstruction
→ facial congestion, edema, dilated neck veins
- Venous engorgement → increased bleeding during surgery

3. Esophageal compression

- Dysphagia

4. Nerve involvement

- Recurrent laryngeal nerve compression
→ hoarseness of voice

5. Intrathoracic complications

- Extension into mediastinum → compression of great vessels and lungs
- Reduced lung expansion

6. Positional symptoms

- Dyspnea worsens in supine position
- Orthopnea

7. Surgical and anaesthetic problems

- Difficult surgical access
- Pneumothorax
- Risk of major vessel injury
- Challenging airway management
- Risk of postoperative airway obstruction

17. When is the hyperthyroid patient considered optimised?

- Return of normal heart rate, pulse pressure, sinus rhythm.
- Relief of tremors, anxiety, palpitation, dyspnea, heat intolerance.

18. How do you investigate the patient with hyperthyroidism?

Non-specific investigations

I. Complete blood count: to look for presence of:-

- Anaemia
- Neutropenia / Neutrophilia
- Leucocytosis
- Thrombocytopenia



- e. Haematocrit
- f. ESR
- g. Eosinophilia

II. Serum electrolyte: to look for disturbances in sodium and potassium

III. Liver function test: altered in patients with existing right heart failure

IV. Renal function test to look for serum creatinine, blood urea nitrogen

V. ECG: to look for-

- a. Rhythm disturbances like atrial fibrillation
- b. Right axis deviation
- c. Strain pattern
- d. Bundle branch block
- e. Ischemic changes
- f. Low voltage complexes
- g. Features of right ventricular hypertrophy

VI. 2-D echo: to look for-

- a. Chambers
- b. Presence of RWMA
- c. Any associated valvular lesions
- d. Pulmonary artery pressures
- e. IVC collapsibility
- f. Fluid status
- g. Presence of thrombus

VII. X-ray neck:

- a. Lateral view to detect antero-posterior tracheal compression
- b. AP view to look for tracheal deviation

VIII. Indirect Laryngoscopy to rule out any vocal cord palsies for medicolegal purposes

IX. Chest X-ray: PA view to look for any mediastinal extension, presence of plural effusion

X. Bedside ultrasound neck :

- a. USG guided airway assessment
- b. Dynamic measurement
- c. Tracheal dimensions
- d. Subglottic airway diameter to note appropriate size of endotracheal tube
- e. Establishes presence of vocal cord palsies

- f. Reveals intrathoracic extension of goitre
- g. Tracheal invasion by thyroid malignancy
- h. Tracheal wall thickening
- i. Suboptimal positioning of LMA
- j. Airway blocks

XI. CT neck and thorax to look for retrosternal extension, tracheal compression and airway obstruction

XII. Thyroid antibody screening

Specific investigations

I. Direct test of thyroid function

Administration of radioactive iodine and measurement of its uptake by the thyroid. Radioactive iodine uptake is measured 24 hours after administration of the isotope.

II. Tests related to concentration and binding of thyroid hormones

These include measurement of T4 and T3. Radioimmunoassay of both free and total T3 and T4 are available.

Free T3: 0.23 - 0.42ng/dL

Free T4: 0.8 - 1.8ng/dL

Total T3: 80 - 200ng/dL

Total T4: 5000-12000ng/dL

Free T3,T4 - better indicator of functional thyroid state as they are metabolically active, not affected by thyroid binding factors

III. Test of homoeostatic control of thyroid function

This includes measurement of serum TSH, thyrotropin releasing hormone stimulating test. TSH values will vary inversely with thyroid hormone levels. The thyrotropin releasing hormone stimulating test will assess the hypothalamic pituitary thyroid axis.

TSH: 0.4 - 4.0mIU/L

TSH- early indicator of hypothyroidism and hyperthyroidism in presence of normal pituitary thyroid axis

IV. Thyroid scan - presence of functioning thyroid tissue

V. Radioisotope study- TC99 or I123 scan to know the activity.

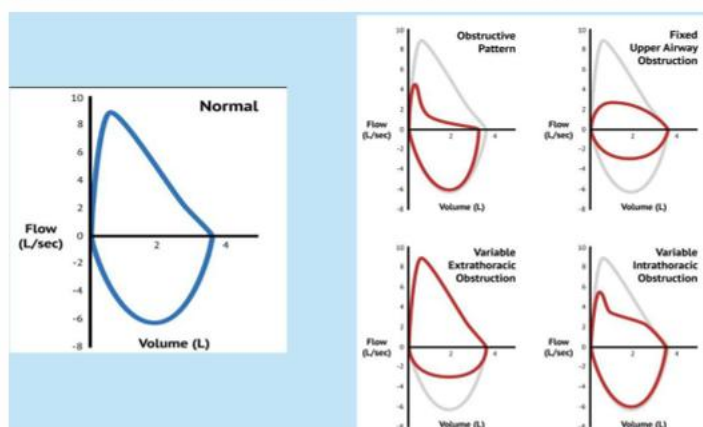
VI. FNAC to know the type of nature of thyroid mass to know the presence of malignancy.



19. what is the importance of pulmonary function test?

Pulmonary function test is a non-invasive method used to evaluate patients with airway obstruction. This gives us an idea about retrosternal extension and breath holding and breath count can be assessed.

Flow volume loop analysis, determines the extent and location of airway obstruction. Flow volume loops are generated by inhaling to total lung capacity, exhaling to residual volume and inhaling back to total lung capacity. Fixed obstruction includes tracheal tumours, subglottic stenosis and goitres which produces plateaus in both inspiratory and expiratory cycle of flow volume. Variable extrathoracic lesions are often caused



by vocal cord paralysis, vocal cord neoplasm and neoplasms in the neck. The inspiratory limb of the flow volume plateaus- during inhalation generation of negative intrathoracic pressure pulls the extrathoracic airway close. During exhalation airflow maintains the patency of airway.

Variable intrathoracic lesions include endobronchial tumors, tracheal tumors and tracheomalacia. They produce plateau of the expiratory cycle only. Inhalation is unimpaired because negative intrathoracic pressure will stent the airway. During exhalation positive intrathoracic pressure narrows the airway and results in plateau of the expiratory limb loop.

20. Why do you want to optimise the patient before taking for surgery?

Patient needs to be optimised before surgery for the following reasons:

- Thyroid hormone increases the oxygen consumption
- Alters the response to most of the anaesthetic drugs
- Hyperthyroid patients are susceptible for dysrhythmias, haemodynamic instability and circulatory collapse.
- Manipulation of gland will release thyroid hormone into circulation, precipitating a thyroid storm
- Centrally acting adrenergic antagonist such as Reserpine can be administered to patients who cannot take either beta blockers or calcium channel blockers

21. Are there any contraindications for the use of iodine preparations?

Iodine is contraindicated in :

- Children
- Pregnancy
- Breastfeeding

22.What is the role of other drugs in thyrotoxic patients?

Cholestyramine will bind T4 in the gut during enterohepatic circulation to bring down the T4 levels.

Lithium carbonate 300mg orally every 6th hourly can be given if patient is allergic to iodine.

23.What are the considerations for taking consent in this patient ?

It should be a high risk written informed consent incorporating the following aspects :

- Increased blood loss
- Possible airway obstruction and possible post op mechanical ventilation
- Possible requirement of tracheostomy
- Occurrence of post operative thyroid storm and their management.
- Development of congestive cardiac failure and rhythm disturbances and their management.



24. What scoring systems are available for diagnosing thyroid storm ?

Burch-Wartofsky Scoring system

Thermoregulatory dysfunction	points	Gastrointestinal-hepatic dysfunction	points
Temperature °C		Manifestation	
37.2-37.7	5	Absent	0
37.8-38.2	10	Moderate (diarrhea, abdominal pain, N&V)	10
38.2-38.8	15	Severe (jaundice)	15
38.9-39.4	20		
39.5-39.9	25		
≥ 40	30		
Cardiovascular	points	CNS disturbance	points
Tachycardia (bpm)		Manifestation	
100-109	5	Absent	0
110-119	10	Moderate (delirium, psychosis, extreme lethargy)	20
120-129	15		
130-139	20	Severe (seizure, coma)	30
≥ 140	25		
Atrial Fibrillation		Precipitating event	points
Absent	0	Status	
Present	10	Absent	10
Congestive cardiac failure		Present	0
Absent	0		
Mild	5		
Moderate	10		
Severe	20		

Total score
>45 Thyroid storm
25-45 Impending storm
<25 Storm unlikely

25. What are the important goals of preoperative preparation in hyperthyroid patients?

Preoperative preparation for elective surgery is necessary to make the patient euthyroid (which may take 6 weeks to 18 months) in order to:

- ▶ Prevent thyroid storm
- ▶ Control sympathetic overactivity
- ▶ Optimize cardiovascular status

This can be achieved with:

1. Anti-thyroid drugs for 6-8 weeks (Tab. Propylthiouracil 200mg – 400 mg)
2. Beta blockers (Tab. Propranolol 40mg-120mg)
3. Lugol's iodine (5- 10 drops 10 to 15 days preoperatively) or Potassium iodide (8-10 days prior) to reduce the vascularity. Antithyroid drugs must precede iodide therapy, since iodide alone may increase thyroid hormone synthesis and exacerbate thyrotoxicosis
4. Corticosteroids (Inj. Hydrocortisone 100 mg IV or Dexamethasone 2 mg q6hrly) if adrenal insufficiency is suspected

According to recent evidence, thyroidectomy can be proceeded even in hyperthyroid state (confirmed by biochemical report) if cardiovascular stability is achieved.

Pre-anesthetic evaluation also should focus on airway assessment with the goal of predicting any potential difficult airway and management of the same and to ensure vocal cord function.

In case of emergency, perioperative thyroid storm should be anticipated and adequately prepared. Rapid preparation of the patient should be done with:

▶ Beta blockers: Propranolol (0.5–1 mg/kg IV bolus PO/RT: To reduce hyperadrenergic state and to decrease peripheral conversion of T₄ to T₃ or Esmolol (0.5 mg per kg followed by 0.03 – 0.3 mg/kg/min): better alternative because of its beta-1 specificity and shorter half life.

▶ Antithyroid drugs: Propylthiouracil (200 to 400mg Q6H PO/RT) or Methimazole 20 to 40mg Q6H PO/RT) should be administered as soon as possible.



- ▶ Propylthiouracil and intravenous dexamethasone 2mg Q6H will block peripheral conversion of T4 to T3.
- ▶ Saturated solution of potassium iodide (five drops PO Q6H) or Lugol's iodine (30 drops PO Q6H or Q8H).
- ▶ Parasympatholytic such as atropine should be avoided because of unopposed sympathetic activity.
- ▶ Fluids and electrolytes must be replaced.
- ▶ Invasive monitoring maybe needed to guide the administration of inotropes and vasopressors when hypotension is unresponsive to fluids

26. How will you assess the airway?

Assessment of Airway:

- ▶ History should focus on stridor, dyspnea, dysphagia and change in voice.

On examination:

- ▶ Kocher's test: If the patient develops stridor on pushing lateral lobes postero-medially with fingers the test is considered positive.
- ▶ Routine airway assessment including assessment of neck movement in all planes (atlanto-axial and extension) and mallampatti grading.
- ▶ Preoperative neck x-ray AP and lateral view: to assess tracheal deviation, if >1cm from midline causes respiratory problems.
- ▶ Tracheal compression or stenosis is significant if there is > 30 % of tracheal lumen narrowing which is confirmed by 3DCT scan (better visualization of tracheal deviation/compression).
- ▶ CT scan of neck and upper thorax or MRI is beneficial to know the exact location and extension of large thyroid gland with retrosternal extension.
- ▶ Indirect laryngoscopy to assess vocal cord status.
- ▶ Pulmonary function test: Flow-volume loop helps in diagnosing subclinical airway obstruction, type and site of obstruction.
- ▶ If suprasternal compression shows extrathoracic obstruction pattern- inspiratory flow is decreased. Causes are: Vocal cord paralysis, vocal cord neoplasm).
- ▶ If infrasternal compression: intrathoracic pattern – expiratory flow is decreased. Causes: bronchial and tracheal tumors, tracheomalacia)
- ▶ If large mass and intrathoracic extension causes fixed obstruction where both inspiratory expiratory flow is limited. Causes: Tracheal tumors, subglottic stenosis and goitre.

27. How do you assess the patient with retrosternal goiter?

- ▶ Patients present with dyspnoea, orthopnoea, stridor hoarseness of voice, dysphagia which worsens in the supine position.
- ▶ On examination: lower border of the swelling is not palpable, tracheal deviation will be present. If there is a superior vena-caval obstruction, dilated veins of head,neck and upper part of the chest. There may be respiratory distress due to airway edema.
- ▶ Pemberton's sign: Flushing of the face, dilated veins of head,neck and upper thorax, inspiratory stridor and raised JVP occurs with raising of both arms simultaneously which is not done routinely but can be confirmed by CT scan.
- ▶ Intravenous cannulation /CVC preferably put on lower limbs like foot, ankle, femoral vein because venous drainage from upper body is impaired due to obstruction.
- ▶ Lower limb access for reliable venous return and effective drug administration.

28. What are the cardiac complications during thyroid surgery in hyperthyroid patient in perioperative period and how are they managed?

Hyperthyroid patients are more prone to cardiac complications due to sympathetic overactivity and increased circulating thyroid hormones.

Cardiac complications include:

- ▶ Tachycardia
- ▶ Arrhythmias like atrial fibrillation
- ▶ Hypertension
- ▶ Myocardial ischemia
- ▶ High output cardiac failure
- ▶ Thyroid storm
- ▶ Cardiac failure with pulmonary edema.

Management:

- ▶ **For tachycardia:** deepen the plane of anaesthesia.
- ▶ **Arrhythmias:** Beta blockers (Propranolol and esmolol), acute onset of AF : Digoxin (8-12 mcg/kg IV), Amiodarone 150mg IV over 10 minutes followed by 1 mg/ min IV for 6 hours then 0.5 mg/min), if unstable AF - electrical cardioversion with 120-200 J.



► **Congestive cardiac failure and pulmonary edema:**

- Positive pressure ventilation
- Furosemide (0.5 -1 mg/kg IV over 1 -2 minutes)
- Dobutamine (2.5-10 mcg/kg/min IV)
- Noradrenaline (0.08-0.3 mcg/kg/min IV)

► **Hypertension:** adequate depth of anaesthesia and short acting vasodilators like NTG or sodium nitroprusside.

► **Myocardial Ischemia:** 100 % oxygen and nitrates.

29. What are the anaesthetic concerns in a hyperthyroid patient undergoing surgery especially with airway difficulty due to huge thyroid swelling?

In patients with huge long standing thyroid swelling: distorted anatomy (deviation/compression) and limited neck movements leads to difficult mask ventilation, visualization, laryngoscopy and intubation. Patient on lying position, on induction and with muscle relaxation can develop stridor because of loss of muscle tone and large mass obstructing the airway leading to difficult mask ventilation.

Intraoperative concerns include:

- Hemodynamic fluctuation including tachycardia
- Hypertension
- Arrhythmias
- Bleeding

Due to tracheomalacia, there is a risk of airway collapse after extubation. Nerve injury if occurred will lead to vocal cord palsy causing stridor and aspiration.

30. What is the position given during thyroid surgery and what are the concerns associated with positioning?

► **Rose position:** The neck is fully extended with 15–20° head-up tilt to reduce venous bleeding and head rested on padded ring with sandbag kept between scapulae. But this position carries a risk of venous air embolism.

► Preoperatively, neck mobility must be assessed, especially in patients with cervical spine disease.

► Neck extension can alter endotracheal tube position (around 2.5cms), so it must be rechecked after final positioning to prevent accidental extubation.

► Increased airway resistance can be due to kinking of

tube, secretions, bronchospasm, due to surgeons manipulating trachea or compression by large retrosternal thyroid.

► There is increased risk of accidental disconnection of circuit and airway accidents because of surgeons operating near the airway.

► These patients are also prone to corneal abrasion due to exophthalmos and eyelid retraction, hence adequate eye protection is essential.

► The arms should preferably be adducted by the patient's sides. If placed abducted at right angles, care must be taken to avoid hyperextension at the shoulder, as this can stretch and injure the brachial plexus.

IV line extensions to be secured.

31. What will be your choice of anaesthetic technique?

General anaesthesia is most commonly preferred as it provides a secured airway and immobile field.

It is indicated in prolonged procedures, difficult airway, obesity/OSA, severe anxiety, uncooperative patients, or extensive surgeries (e.g., sternotomy, neck dissection).

Regional/local anaesthesia allows intraoperative voice monitoring, avoids intubation, and may reduce hospital stay and cost, with similar outcomes.

However, due to complications associated with regional block and medico legal restrictions GA is preferred.

Regional anaesthesia is contraindicated in invasive cancer, cervical lymphadenopathy, LA allergy, or patient refusal, and in some cases (about 2–12%) may need conversion to GA due to anxiety, inadequate block, or surgical difficult.

32. How will you monitor a patient intraoperatively for thyroidectomy?

Standard monitoring with ECG, NIBP, pulse oximetry, capnography and temperature monitoring (as there is a potential risk of developing hyperthermia during perioperative period) is done for all patients.

Invasive arterial BP monitoring, CVP and pulmonary artery catheterization may be required based on comorbidities and hemodynamic stability.



Others include esophageal stethoscope, neuromuscular monitoring and urine output monitoring.

Specialized monitoring: Recurrent laryngeal nerve (RLN) monitoring may be used with a specialized ETT or surface electrodes placed at the level of the vocal cords, requiring correct positioning during intubation. For effective monitoring, neuromuscular blockade should be avoided during maintenance of anaesthesia.

33. What preoperative anaesthetic preparation to be done in a patient with huge thyroid swelling especially with anticipated difficult airway?

Perform a thorough airway assessment (large goiter, tracheal deviation/compression, stridor, malignancy, neck extension, mouth opening and Mallampati grading).

Keep a fully equipped difficult airway cart ready: fiberoptic bronchoscope, rigid bronchoscope (in severe obstruction, it is difficult to pass the fiberoptic scope because of narrow passage), video laryngoscope, bougie, stylets, LMA/i-gel/ILMA (may require during extubation), and emergency cricothyrotomy set.

Ensure availability of two working laryngoscopes and ETTs (including 1–2 sizes smaller, preferably reinforced/armoured or RAE tube – north pole).

Ensure experts (ENT surgeon) available.

Keep all drugs prepared (induction agents, minimal/short-acting relaxants if needed).

Preparation to manage thyroid storm - additionally Cooling measures (cold saline, ice packs), β -blockers (esmolol, propranolol), steroids (hydrocortisone), antithyroid drugs (propylthiouracil / carbimazole) + Ryle's tube for administration and adequate IV fluids (glucose-containing) to be kept.

34. What are the Premedications used in a patient with huge thyroid swelling especially with anticipated difficult airway?

► Use minimal sedation, preferably benzodiazepines (Midazolam 0.5 mg/kg) to reduce anxiety and catecholamine surge, and only if the airway is not compromised.

► Avoid atropine/scopolamine as they cause tachycardia and impair thermoregulation; if needed, glycopyrrolate 0.02 mg/kg IV can be used as an antisialagogue.

► Short acting opioids like fentanyl (1–2 mcg/kg) can be used.

► Continue antithyroid drugs and β -blockers on the morning of surgery, provide PONV and antibiotic prophylaxis.

35. Discuss the anaesthetic management of a patient with a large thyroid swelling with emphasis on airway management in an anticipated difficult airway?

► Preoxygenation with 100 % oxygenation should be done for 3–5 minutes to enhance FRC and provide enough time to secure difficult airway.

► To prevent intubation response: Inj. Lignocaine IV 1–1.5 mg per kg IV, esmolol 200–300 mcg/kg IV bolus, Dexmedetomidine 0.5 – 1 mcg/kg and ensure adequate depth of anaesthesia before laryngoscopy.

► **If airway obstruction or difficult airway is anticipated**, prefer awake fiberoptic intubation or inhalational induction; awake technique is especially indicated in patients with obstructive symptoms/snoring. During induction, lift the thyroid mass upward or turn the patient laterally to relieve tracheal compression.

► **In cannot intubate cannot oxygenate situation**, front of neck access is required. Emergency technique used is cricothyrotomy with scalpel – bougie- tube (as cricothyroid membrane is above thyroid mass) and not tracheostomy due to distorted anatomy, tracheal displacement, retrosternal extension, highly vascular field and it is time consuming in emergency.

► If no airway obstruction is anticipated, induction can be done with thiopentone (3–4 mg/kg) + vecuronium (0.1 mg/kg) + fentanyl (1–2 μ g/kg). Thiopentone is preferred due to its antithyroid effect ($\downarrow$ T4 $\rightarrow$ T3 conversion), though propofol is an alternative with advantages like blunted sympathetic response, antiarrhythmic and antiemetic effects . Ketamine is avoided as it increases HR and BP. Loss of muscle tone after relaxant may precipitate airway obstruction.

► Intubation done with a reinforced (armoured) ETT (<15 secs), advance the tip beyond the compressed segment, and secure it firmly since access is limited. If difficulty is expected, succinylcholine may be preferred after confirming the ability to ventilate; otherwise, non-depolarizing agents (Vecuronium) can be used.



► Intraoperative monitoring done with standard ASA monitoring and continuous IABP monitoring.

► Maintenance: Maintain the depth of anaesthesia O₂ + N₂O + Isoflurane/ Sevoflurane or TIVA (with propofol infusion 50-200 mcg/kg/min, Fentanyl 1-2 mcg/kg/hr) along with muscle relaxants. But if RLN monitoring is done, avoid neuromuscular blockade for maintenance.

► Intraop hypotension is managed with directly acting alpha agonist (Inj. Phenylephrine 50- 100 mcg IV bolus). Avoid adrenaline containing solutions, ephedrine, noradrenaline and dopamine as these increases heart rate.

36. What is thyroid storm. How do you diagnose it intraoperatively?

Thyroid storm is a life-threatening complication in a patient whose hyperthyroidism has been severely exacerbated by illness, trauma, infection or surgery.

Incidence ranges from 0.20 to 0.76 per 100 000 people per year. Mortality rates of thyroid storm range between 10% and 30% despite early diagnosis.

Thyroid Storm Triggers:

General Triggers: Nonthyroid surgery, trauma, Infection, pulmonary embolism, myocardial infarction and diabetic ketoacidosis.

Thyroid-Related Triggers

- Abrupt discontinuation of antithyroid medication
- Thyroid surgery
- Overdose of thyroid hormone
- Acute iodine load:
- Radioactive iodine treatment
- Contrast dye
- Oral iodine

Clinical Features of Perioperative Thyroid Storm

Organ System	Awake Patient	Anaesthetised Patient
Central nervous system	Confusion, agitation, delirium, psychosis, seizure, stupor, coma	Seizure, delayed emergence
Cardiovascular system	Sinus tachycardia, hypotension, dysrhythmias (especially atrial fibrillation), congestive cardiac failure	Same features
Thermoregulation	Pyrexia, dehydration	Hyperthermia (often prominent sign)
Gastrointestinal system	Nausea, vomiting, abdominal pain, diarrhea, hepatocellular dysfunction (jaundice)	Hepatocellular dysfunction (jaundice)

Under anaesthesia, thyroid storm is suspected by unexplained tachycardia, hypertension, hyperthermia, and arrhythmias (AF). Other findings include increased ETCO₂, excessive sweating, flushing, increased bleeding, rapid soda lime exhaustion, and signs of cardiac failure or shock.

37. What are the differential diagnosis of thyroid storm?

► Malignant hyperthermia - temperature increase is much faster, more severe metabolic acidosis and very

high creatinine kinase, muscle rigidity. Triggering agents: Succinylcholine, halothane, increased temperature, tachycardia, HTN, increased potassium. Respond to Dantrolene.

- Pheochromocytoma - predominant feature will be cardiovascular – tachycardia, dysrhythmias and hypertension. No muscle rigidity
- Neuroleptic malignant syndrome
- Drug overdose (cocaine, amphetamine)
- Sepsis
- Anaphylactic reaction



38. How will you manage thyroid storm intraoperatively?

- ▶ Treat the precipitating cause
- ▶ Supportive care which includes administration of high-flow oxygen with increased FiO₂, change soda lime if required, and initiate aggressive cooling using cooling blankets, ice packs over major vessels, lowering OT temperature, cold IV fluids, cold saline through RT and bladder irrigation with cold saline
- ▶ Adequate IV fluid resuscitation with glucose-containing solutions should be given. Aspirin is avoided as it increases free thyroid hormone levels.
- ▶ Thyroid hormone activity is suppressed by inhibiting synthesis using propylthiouracil (200–400 mg via RT every 8 hours) or methimazole (40mg via RT followed by 25 mg Q6H) and by blocking hormone release with iodine preparations (e.g., sodium iodide (500-1000 mg IV Q8H)/ KI 5 drops TID via NGT or Lugol's iodine 5–10 drops TID via RT).
- ▶ Sympathetic overactivity is controlled with β -blockers such as propranolol (1-2 mg/min IV every 15 min upto max 10 mg or 40-80 mg Q4 hrly via RT) or esmolol (250- 500 mcg/kg IV bolus followed by 50–100 μ g/kg/min infusion) to reduce heart rate and arrhythmias (AF), along with treatment of shock or cardiac failure if present.
- ▶ Corticosteroids (Hydrocortisone 100–200 mg IV Q8H or Dexamethasone 2 mg IV Q6H) are administered to reduce peripheral conversion of T₄ to T₃ and provide additional supportive benefit.
- ▶ Hemodialysis and plasmapheresis used if refractory.

39. How will you Extubate the patient with huge thyroid swelling especially with anticipated difficult airway?

Before extubation, Valsalva manoeuvre done in trendelenberg position to check hemostasis.

Ensure full recovery of airway reflexes and complete recovery from muscle relaxation.

Check for tracheomalacia:

-Before extubation, do cuff leak test in long standing goitre to rule out tracheomalacia.

- Using direct visualization of airway patency is suggested to rule out tracheomalacia. FOB passed into

ETT and assess the airway collapse and vocal cord movement by withdrawing the ETT and bronchoscope together. If airway collapse is noted, both bronchoscope and ETT is readvanced and extubation is deferred. Or pass the modified ventilating tube exchanger (Cook airway exchange catheter – designed for exchange of ETTs) as a guide for re intubation.

To rule out RLN injury: Check for vocal cord movement using FOB through LMA or video-laryngoscopy.

Avoid stress response during extubation to prevent accidental haemorrhage from wound site during reversal and bucking movement. Therefore, extubation should be smooth.

To decrease stress response : Inj. Lignocaine IV 1-1.5 mg per kg IV, Inj. esmolol 200-300 mcg/kg IV bolus and Inj. Dexmedetomidine 0.5 – 1 mcg/kg can be used.

If extubation done in a deeper plan of anaesthesia is the possible failure of elicitation of vocal cord movements. To detect vocal cord palsy, ask the patient to speak letter “E” or the word “moon”.

After total thyroidectomy, antithyroid drugs to be stopped as entire gland is removed. And replace with thyroxine from day 1 post op.

Removal of thyroid gland doesn't immediately relieve thyrotoxicosis since half life of circulating thyroid hormone is 7-8 days. Propranolol 160mg/day continued for 1 week.

As thyroid hormone levels decreases, symptoms resolves. Medications weaned over weeks after surgery.

40. How will you provide postoperative analgesia?

- ▶ NSAIDS (if no contraindication)
- ▶ IV or oral paracetamol
- ▶ Superficial cervical plexus blockade
- ▶ Post op surgical site infiltration with LA
- ▶ IV short acting opioids (fentanyl/tramadol)

41. What is the role of Superficial and deep cervical plexus blockade in thyroidectomy?

▶ Unilateral Superficial and deep cervical plexus blockade for unilateral surgery.



▶ Bilateral blockade is contraindicated due to bilateral phrenic nerve palsy.

▶ The practice of superficial and deep cervical plexus blockade as well as cervical epidural anaesthesia are not recommended anymore as these techniques are associated with potential risk of complications such as inadequate anaesthesia or wearing of the effect of local anaesthetics and cardio respiratory arrest.

▶ Complications associated with deep cervical plexus blockade include vertebral artery injury, systemic toxicity and subdural block.

▶ Because of medico-legal restrictions, GA with ETT is the safest approach.

42. How do you approach to tackle airway obstruction after extubation?

▶ Post extubation airway block can be partial and the causes are mucus or blood in upper airway, incomplete recovery from anaesthesia, hematoma or reactionary hemorrhage with sudden increase in neck swelling, laryngeal edema and tracheal collapse.

▶ In case of tracheal collapse airway patency can be maintained with slow and prolonged inspiration.

▶ An accessible back up airway control plan is important.

▶ Ensure oxygenation and ventilation with O₂ supplementation using well fit simple face mask.

▶ Assess adequate recovery from the muscle relaxant effect.

▶ Small dose of succinylcholine 25 – 50 mg may help in case of stridor. Reintubation with smaller size ETT or LMA.

▶ IV steroids for laryngeal edema.

43. What are the postoperative complications anticipated in the postoperative period after total thyroidectomy?

- I. Respiratory distress due to Hematoma
- II. Laryngeal edema
- III. Nerve injuries
- IV. Tracheomalacia
- V. Hypoparathyroidism
- VI. Hypocalcemia
- VII. Pneumothorax
- VIII. Thyroid storm

I. Respiratory distress due to Hematoma: Rare but life-threatening. Bleeding is most commonly of venous

origin, resulting in hematoma formation and compression of vital airway structures. Patients with large or highly vascular thyroid glands are at increased risk. It is a surgical emergency. If airway compromise is suspected, immediately open the wound and release sutures at the bedside to decompress the hematoma. Simultaneously, secure the airway at the earliest (preferably by endotracheal intubation; LMA can be a temporary option). Once stabilized, the patient should undergo urgent surgical exploration under general anesthesia for hematoma evacuation and definitive hemostasis.

II. Laryngeal edema: Occurs due to venous obstruction from an enlarging neck hematoma or trauma from difficult/repeated laryngoscopy, and rarely from myxedema. It can lead to progressive airway compromise with stridor. Immediate endotracheal intubation is mandatory to secure the airway.

III. Recurrent laryngeal nerve (RLN) injury: Incidence: 0 - 4.8 %. Occurs due to traction, transection, entrapment, or ischemia, and can be temporary or permanent; nerve monitoring helps to reduce the risk to 0.4 % . Unilateral palsy causes hoarseness, weak voice, aspiration, and poor cough, while bilateral palsy can lead to severe stridor and airway obstruction. Most injuries are transient, but persistent symptoms need laryngoscopy, and severe cases may require intubation, tracheostomy, or vocal cord medialization.

IV. Superior laryngeal nerve (SLN) injury: Most commonly injured during superior pole dissection, affecting the external branch that supplies the cricothyroid muscle. This leads to impaired vocal cord tension and subtle voice changes (especially loss of pitch). Injury to the internal branch causes loss of supraglottic sensation, resulting in impaired swallowing reflex and dysphagia.

V. Tracheomalacia: Long-standing compression by a large goiter can cause atrophy and erosion of tracheal cartilaginous rings, leading to tracheomalacia. After thyroid removal, loss of external support may result in anteroposterior tracheal collapse and dynamic airway obstruction. Management includes urgent reintubation, possible ventilatory support or tracheostomy, and temporary airway support until recovery, as the condition is often self-limiting.

VI. Hypoparathyroidism: Occurs due to injury to parathyroid gland or accidental removal of the gland during thyroidectomy, which presents with hypocalcemia in 20 % patients.



VII. Hypocalcemia: The symptoms of hypocalcemia develops within 24 to 96 hours after surgery. Clinical features include perioral tingling, confusion, muscle twitching, tetany, seizures, with signs like Chvostek's and Trousseau's, and serious effects such as laryngospasm, QT prolongation, and arrhythmias. Hypocalcemia can be treated with oral calcium supplementations if Calcium level is > 2 mmol/L and with IV calcium gluconate or calcium chloride if Calcium level is < 2 mmol/L. **Calcium chloride is effective** as it has 3 times more elemental calcium (1 gm contains 270 mg of elemental calcium) than calcium gluconate. However, calcium chloride causes more tissue necrosis when extravasated, hence administer via central line. Calcium gluconate causes less tissue necrosis when extravasated, hence can administer through peripheral line. Dose: 10 ml of 10 % IV over 10 minutes followed by infusion 0.5-1.5 mg/kg/hr.

VIII. Pneumothorax: Though rare, it can occur during resection of retrosternal goiter. Intraoperative findings include sudden hypoxemia, $\downarrow$ SpO₂, hypotension, tachycardia, increased airway pressure, difficult ventilation, and absent breath sounds. Management is immediate decompression (needle in 2nd intercostal space) and, if tension pneumothorax, chest tube insertion.

IX. Thyroid storm: is a rare but life-threatening complication, usually due to poor preoperative control or severe stress, and may present intraoperatively with unexplained tachycardia, hyperthermia, and arrhythmias (classical symptoms may be masked under anaesthesia). It can result from hyperactive residual thyroid tissue or hormone release during surgery. Management is urgent and aggressive: β -blockers for tachycardia, active cooling, steroids, and high-dose antithyroid drugs (propylthiouracil or methimazole).

44. What are the nerves injured during thyroid surgery, what are their clinical consequences and their management?

External branch of SLN: Injury causes change of voice quality, voice fatigue and loss of vocal stamina. Treatment: conservative management with speech therapy.

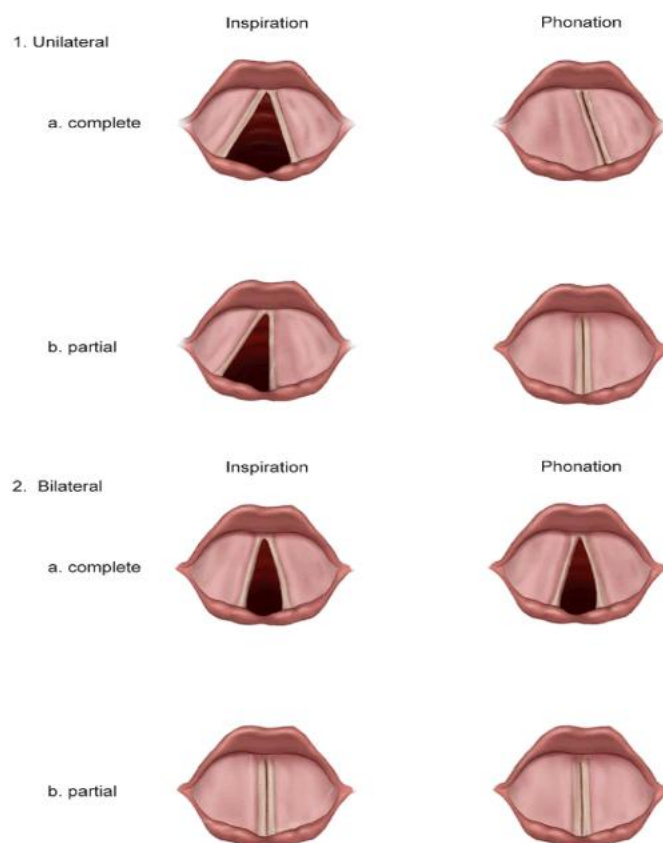
Internal branch of SLN injury: lead to loss sensation above vocal cords and risk of aspiration. Treatment: conservative management with speech therapy and swallowing therapy.

Unilateral Recurrent Laryngeal nerve injury:

Incidence: 3-4% causes hoarseness and weak cough. No significant airway obstruction, risk of aspiration and difficulty in phonation because of compensatory hyper adduction of opposite cord allowing vocal cord function. Treatment: speech therapy and voice rest. It improves over weeks to months.

Bilateral Partial Recurrent Laryngeal Nerve injury: Unopposed adduction of cords leads to glottic closure needing tracheostomy.

Bilateral Complete Recurrent Laryngeal Nerve injury: Incidence: < 1 % Vocal cords are in cadaveric position causing stridor and aspiration. Treatment: Intubation, IV steroids and trial extubation after several days. If nerve function is not reversed plan for tracheostomy. Corrective procedure is done after 6 months.

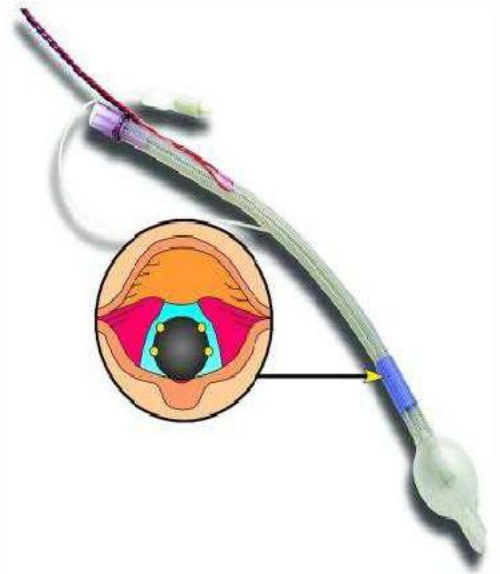


Vocal cord position in different types of Recurrent Laryngeal Nerve injury

45. How to prevent nerve injuries during thyroid surgery?

Electro physiological nerve monitoring decreases the incidence by 0.4%. RLN integrity monitor with electromyography ETT. ETT contains electrodes along its wall placed against vocal cords. Nerve stimulation produces characteristic beep which confirms nerve.





46. What is tracheomalacia, how will you diagnose and manage it?

Tracheomalacia is a rare complication (~1.4%) after thyroidectomy.

Tracheomalacia should be suspected in:

- ▶ Long-standing goiters
- ▶ Substernal goiter causing compression for > five years
- ▶ Elderly patients
- ▶ Floppy tracheal cartilage on palpation intraoperatively by the surgeon

It is due to long-standing tracheal compression causing atrophy and erosion of tracheal rings. It is defined as >50% reduction in tracheal lumen due to dynamic airway collapse. This results in extrathoracic tracheal collapse during inspiration and intrathoracic collapse during expiration.

After thyroid removal, loss of external support can cause anteroposterior tracheal collapse and postoperative airway obstruction, sometimes delaying extubation (~10% require temporary ventilatory support, though most recover within ~10 days).

Diagnosis is done by CT or MRI and Flow – volume loop showing variable intrathoracic obstruction.

Confirmation of Tracheomalacia is made by one or more of the following: Normal vocal cord mobility, absence of glottic/subglottic edema or hematoma, soft and floppy trachea on palpation, difficulty in negotiating suction catheter beyond the ETT after gradual withdrawal, absence of peritubal leak on deflation of ETT cuff (Cuff leak test) and/or obstruction

to spontaneous respiration on gradual withdrawal of ETT.

Diagnosis of Tracheomalacia can also be done: by measuring the volume of air required to inflate cuff of ETT to produce an airtight seal before surgery and same test is performed at the end of the surgery – if any decrease in volume required to inflate the cuff to produce airtight seal reflects tracheomalacia.

Cuff leak test: With the ETT in place, the cuff is deflated and a leak is assessed during IPPV by palpating the trachea or noting a difference between set and expired tidal volume. During spontaneous breathing, the ability to breathe around a plugged tube suggests adequate peritubal space. Although useful for detecting airway edema, its role in tracheomalacia is controversial and not reliable.

Management: Airway obstruction due to tracheomalacia requires immediate endotracheal intubation; mild cases may be supported with CPAP. The condition is often self-limiting, with fibrosis stabilizing the trachea over a few days. Tracheostomy may be needed in severe cases, while tracheal stenting or surgical repair is reserved for persistent airway collapse.

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ANAESTHESIA FOR OPEN GLOBE INJURY

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Introduction:

General Considerations. Open-globe injuries are rare and account for only 2% of all ocular trauma cases with an incidence of 3-6 per 100,000. This can lead to severe visual impairment, blindness, and lifelong sequelae. Ocular injuries are classified by mechanism, extent of injury, and location. Open globes injury involves full-thickness injury to the cornea or sclera. Cutting or piercing is the most common mechanism of injury in young children while blunt trauma is more common in teenagers. Patients will sometimes present with concomitant injuries such as facial or orbital fractures or traumatic injuries to other areas of the body o Intraocular pressure (IOP). Normal pressure 10-22 mmHg. < 3 months: < 8 mmHg. 6 – 9 months: < 12 mmHg. Thereafter: + 1 mmHg /year up to 10-12 years when it becomes similar to adults. Varies over the course of the day highest in the morning. (Ref1) RUPTURED GLOBE REPAIR Surgical Specialty: Ophthalmology Authors: Mingfei Wang, M.D. and Angela Lee, M.D. Children's National Medical Center, Washington, DC SPA CASE GUIDES, [Society for Pediatric anaesthesia].

An open globe injury (OGI) refers to a traumatic ocular incident characterized by a breach in the structural integrity of the eye's outer layers, typically involving the cornea and sclera. These injuries can arise from various mechanisms such as globe rupture, laceration, perforation, or penetration, and are deemed urgent in the field of ophthalmology due to their potential to cause vision impairment and intraocular infections. In eyes without prior surgical interventions, ruptures commonly occur posterior to the extraocular muscles where the sclera is thinnest. However, in cases with previous intraocular surgeries, ruptures may manifest at the prior incision site. Additionally, blunt trauma can cause globe rupture at the limbus. Notably extremely high pressures exceeding 7000 mm Hg may be necessary to rupture the globe.

Epidemiology of OGI. In a large study from north India done over 4 years, it was found that out of 402 patients who suffered OGI, 72.9% were male and 27.1% were female, ranged from 2 to 70 years old, most vulnerable age group was 6–15 years (24.38%), followed by 16–25 years (23.88%). Most patients came from rural backgrounds (61.94%), and the extent of ocular trauma was 3.93% (95% CI: 3.23–4.63). The most common eye injuries were nonoccupational (82.3%), with sportsrelated incidents (23.9%) and road traffic accidents (23.6%) being the primary causes. Assault accounted for 17.2% of injuries, and domestic accidents made up 15.2%. Mechanical injuries comprised 89.3% of all cases, with wooden objects being the most frequent source (24.9%), followed by metallic objects (20.9%), stones (16.7%), balls (6.0%), and glass (3.7%). The majority of traumatic agents were solid (82.1%), with blunt and sharp objects causing 56% and 17% of cases, respectively. About 7% of victims were drowsy during the trauma, and 98.4% were not using any protective devices at the time of injury. Among the 10.7% with nonmechanical injuries, thermal burns, cracker injuries, and alkali burns were present in 25.58%, 39.53%, and 18.6% of participants, respectively. (Ref 2 Anaesthesia for Open Globe Injury: Indian J Ophthal Anaesth | Volume 04 | Issue 2 | Jun 2024 Revisited Bhavna Gupta¹, Kunal Bishwakarma¹, Anish Gupta², Jaichandran V V³ ¹Department Of Anaesthesiology, AIIMS, Rishikesh ²Department of CTVS, AIIMS Rishikesh ³Department of Anaesthesia, Sankara Nethralaya, Chennai)

Clinical history The clinical history of patients presenting with globe rupture should focus on identifying the aetiology of the ocular or periocular trauma. Symptoms may include abrupt eye pain and visual impairment following a potential penetrating injury. Common causes of penetrating globe ruptures include glass or metal fragments, shotgun or BB pellets,



and wood shavings from wood grinding. In children, sharp objects like scissors are frequent culprits, often encountered at home. Conversely, adults may experience OGI in workplace accidents, assaults, or motor vehicle collisions. Elderly individuals are particularly susceptible to injuries from ground-level falls. In some instances, OGI may result from explosive materials, gunshot wounds, sporting accidents, stab wounds, or blast injuries. Understanding the underlying causes is vital for both prevention and effective management strategies.

Physiologic factors that increase IOP:

Obstruction of aqueous humor flow, Intraocular hemorrhage, Rise in central venous pressure, Rise in arterial blood pressure hypercarbia or hypoxia, Valsalva maneuver, Pain, Coughing and bucking.

Disease Entity:

In ocular trauma, injuries can be classified according to the Birmingham Eye Trauma Terminology (BETT) criteria as either closed globe or open globe where an open globe injury is defined as a full-thickness wound. From there, an open globe injury can be described as secondary to blunt trauma or due to a laceration. A ruptured globe is classified as secondary to blunt trauma when an impact from a blunt object results in a momentary increase of the intraocular pressure resulting in an inside-out injury mechanism (i.e. the blunt trauma causes a temporary increase in intraocular pressure, and intraocular pressure forces contents out of the eye with enough force to rupture the eye wall).[1]

By contrast, a laceration is classified as an open-globe injury that is full-thickness usually caused by a sharp object with the wound occurring at the impact site by an outside-in mechanism. Furthermore, lacerations are defined as either a penetrating injury, intraocular foreign body, or a perforating injury. A penetrating injury is defined as a single laceration with no exit wound and if there is more than one entrance wound, each must be caused by a different agent. An intraocular foreign body is defined as a retained foreign object causing an entrance laceration; of note, an intraocular foreign body is technically a penetrating

injury but is grouped separately since there are different clinical implications due to the object. A perforating injury is defined as two full-thickness lacerations both entrance and exit usually caused by a sharp object or projectile where both wounds are caused by the same agent.

(Ref 3 American academy of ophthalmology, All contributors: Anna Murchison, MD, MPH, Zeba A. Syed, MD, Grant A. Justin, MD, Alexander A. Foster, Bharat Gurnani MBBS, DNB, FCRS, FICO(UK), FAICO (Cornea, AIIMS, AIOS), FAICO (Refractive Surgery, AIIMS, AIOS), MRCS (Ed), MNAMS, Boonkit Purt, MD, Grayson W Armstrong MD MPH by Grayson W Armstrong MD MPH on January 29, 2026.)

Pathophysiology :

The eye is filled with incompressible liquid and when a blunt object with sufficient momentum creates energy transfer over a large surface area, this greatly increases the intraocular pressure causing the eyewall to give way at its weakest point, which may or may not be at the impact site. The actual wound is thus produced by an inside-out force and tissue herniation is very frequent and can be substantial.[1] The site of rupture commonly occurs where the sclera is thinnest and weakest near the equator directly behind the insertion of the rectus muscles.[2] Other regions prone to rupture include the limbus, insertion of the optic nerve, and sites of prior eye surgery.[3][4] Once a ruptured globe occurs, it may result in diffuse injury, hemorrhagic choroidal and retinal detachment leading to a vigorous healing response that can lead to additional injury.[5] Rupture of the globe posterior to the spiral of Tillaux (surgical landmark delineated by connecting the rectus muscle insertions which approximate the underlying ora serrata) is normally accompanied by choroidal rupture and hemorrhage which may percolate through the retina to present as a vitreous hemorrhage.(Ref 3)

Objectives of management The objectives of anaesthesia for OGI are centred around ensuring adequate analgesia, maintaining optimal operating parameters, and preventing rises in IOP to minimize the risk of ocular content expulsion. The physical examination of patients with potential globe rupture necessitates a detailed ophthalmologic assessment.



Key aspects include evaluating the patient's baseline visual acuity, conducting a slit-lamp examination to assess the anterior segment of the globe and anterior chamber, and inspecting the conjunctiva for defects, foreign bodies, or lacerations. Special attention should be given to the equatorial sclera immediately posterior to the insertion of the rectus muscles, as this region is a common site for globe rupture due to its thin scleral composition. Additionally, pupil examination for reactivity and shape irregularities is crucial. It's important to note that during the evaluation, no pressure should be applied to the globe, which excludes tonometry and eversion of the lids from the initial assessment. Attention to the airway is very essential to assess the potential for a difficult airway.

Initial assessment and evaluation of ocular trauma

Systemic triaging Teamwork (neurosurgeons, trauma specialists, ICU Physicians) intervention decisions. Mechanism of trauma, associated injuries to cervical spine, maxillofacial structures and intracranial trauma.

Ocular triaging This includes the determination of the sequence of interventions as well as the nature and timing of the intervention.

Incident Date, Time, Place, Witness, Mechanism, Velocity of impact. Immediate visual symptoms. Associated head injury, other injuries, last oral intake; quick review of other systems

Symptoms Decreased visual acuity (sudden/ gradual), floaters, flashes, field defects, diplopia, pain.

Ophthalmic H/O Previous ocular disease or surgery.

Medical H/O Any systemic diseases, Tetanus Status, sickle cell anemia

Personal H/O Family support, Alcohol/Drug abuse

Drug & Allergy H/O Medications for systemic illness; allergy to any drugs. On any Anti coagulants

Anxiolysis during eye examination

Anxiolysis, if desired, can be achieved using benzodiazepines such as intravenous (IV) midazolam (1 – 2 mg) as it does not alter IOP significantly. However, caution is advised when administering midazolam to older adults, with consideration for dose titration in 0.5 mg increments. An alternative premedication option is

dexmedetomidine IV (0.2 to 0.8 mcg/kg) administered over 10 minutes, although this may induce hypotension and bradycardia and should be used cautiously or at lower doses in patients with hypovolemia, cardiovascular disease, or advanced age.

Other diagnostic modalities

Diagnostic modalities play a critical role in evaluating open globe injuries. Plain X-ray, including AP and lateral views, are essential for assessing intraocular and extraocular injuries as well as fractures, and may also aid in detecting foreign bodies. Ultrasonography using B-scan is valuable for visualizing intraocular and intra-orbital damage, particularly when ocular media clarity is compromised. B-scan ultrasound can detect vitreous haemorrhage, retinal detachment, intraocular foreign bodies, extraocular muscle damage, and scleral rupture, providing valuable insights into the extent of ocular trauma with concerns of iatrogenic expulsion of globe contents. CT scans, especially with 1.5 to 2 mm axial and coronal cuts, are superior to ultrasound in locating foreign bodies and assessing damaged eye structures without direct eye contact, making them a non-invasive and valuable tool in diagnosing ocular injuries. MRI may be beneficial for patients with non-magnetic foreign bodies, although caution is advised in cases of suspected ruptured globe, particularly with potential metallic foreign bodies.

Importance of ocular trauma score

The Ocular Trauma Score (OTS) incorporates functional and anatomical characteristics to assess the severity and prognostication of open globe injuries. Functional characteristics include initial visual acuity (VA), with lower VA indicating poorer outcomes (e.g., counting fingers, hand motion, light perception). Anatomical characteristics such as rupture, endophthalmitis, perforating injury, retinal detachment, and afferent pupillary defect are considered negative prognostic indicators, raising suspicion for optic nerve damage or avulsion. Higher OTS values generally suggest a better prognosis. For patients, higher scores can enhance quality of life, alleviate anxiety, and inform economic decisions. For ophthalmologists, OTS scores assist in providing counselling, triaging patients, managing cases, and conducting research.



Computational method for deriving the OTS score

Initial visual factor	Raw points	
A. Initial raw score (based on initial visual acuity)	NPL =	60
	PL or HM =	70
	1/200 to 19/200 =	80
	20/200 to 20/50 =	90
	$\geq 20/40 =$	100
B. Globe rupture		-23
C. Endophthalmitis		-17
D. Perforating injury		-14
E. Retinal detachment		-11
F. Relative afferent pupillary defect (RAPD)		-10
Raw score sum = sum of raw points		

Intraocular pressure:

IOP dynamics in OGI may exhibit increases, decreases, or remain within normal ranges following injury. Elevated IOP may result from leakage of lens protein or intraocular foreign bodies, often associated with inflammation. Presence of blood in the anterior chamber (hyphema) can decrease outflow through the trabecular meshwork, contributing to increased IOP. Occult bleeding within the orbit (orbital compartment syndrome) can manifest as tight eyelids, difficulty in lid opening, proptosis, periorbital oedema, and a relative afferent pupillary defect, indicating a true ocular emergency that requires immediate attention.

Surgical timing of repair:

The timing of surgical repair is a critical consideration in open globe injuries, especially concerning anaesthesia administration. Penetrating injuries pose a heightened risk of infection, including endophthalmitis, necessitating swift intervention. Moreover, OGI carry the potential for vitreous loss and retinal detachment. However, not all cases of OGI require immediate surgical intervention. Some ophthalmic surgeons may opt to delay surgery until the patient has undergone adequate fasting before anaesthesia. This decision is often influenced by the extent of eye damage, where surgical intervention may not significantly enhance vision. In such instances, patients are advised for bed rest, and their injured eye is shielded until they are deemed ready for primary wound closure. For cases

where the eye remains substantially intact, and there is a promising visual prognosis, a more urgent surgical approach is warranted. The urgency of intervention hinges on various factors such as the size of the laceration, contamination of the wound, and the risk of infection. Typically, a fasting period of six hours is recommended for uncomplicated patients undergoing surgery.

INTRODUCTION

An open globe injury refers to a traumatic ocular incident characterized by a breach in the structural integrity of the eye's outer layers typically involving cornea and sclera. These injuries arise from various mechanisms such as globe rupture, laceration, perforation, blunt injury or penetration, these injuries considered emergency as they have potential to cause vision impairment and intraocular infections also note has to be taken on concurrent injuries and co-existing medical disorders. The anaesthesiologist plays a crucial role in preventing further extrusion of intraocular contents, minimizing increases in intraocular pressure (IOP), ensuring haemodynamic stability, and providing optimal operating conditions. Management of open globe injury comes as emergent surgery so life-threatening coexisting disorders can be optimised for up to 24 hours. Management is challenging because these patients often present as trauma emergencies with full stomach status, associated systemic injuries, pain, anxiety, and risk of aspiration.



CLASSIFICATION OF OCULAR TRAUMA

According to Birmingham Eye Trauma Terminology classification

- Closed globe injury : no full thickness wound (contusion, lamellar laceration)
- Open globe injury : full thickness wound present – subdivided into rupture, laceration, penetrating injury, intraocular foreign body and perforating injury.

Zones of OGI (Birmingham classification) guides prognosis and choice of anaesthesia

- o ZONE I : Isolated to cornea and limbus (regional anaesthesia is viable)
- o ZONE II : Involves anterior 5 mm of sclera (regional anaesthesia can be considered)
- o ZONE III : Involves sclera posterior to anterior 5mm and is associated with vitreous hemorrhage, choroidal rupture. (General anaesthesia is preferred)

CONCERNS

- IOP and management of raised IOP if present
- Full stomach and risk of aspiration
- Prevention of OCR
- Prevention of ocular sepsis

GOALS OF ANAESTHESIA

- ▶ Prevention of any increase in IOP
- ▶ Restoration of vision
- ▶ Prevention of coughing, bucking, vomiting, and straining.
- ▶ Provision of adequate analgesia and akinesia
- ▶ Smooth induction and emergence
- ▶ Maintenance of adequate ocular perfusion pressure and minimal bleeding
- ▶ Obtundation of oculocardiac reflex
- ▶ Prevention of raise in IOP by cry , anxiety in paediatric patients
- ▶ Maintenance of normocarbida, normoxia
- ▶ A delicate balance is required between aspiration prevention and avoidance of sudden rises in IOP

PREOPERATIVE ASSESSMENT AND PREPARATION

1. Initial assessment and resuscitation: open globe injury should be approached as trauma emergency

- ❖ Evaluate for associated head injury, facial trauma , cervical spine injury and any intracavitary injuries
- ❖ Note the mechanism of injury
- ❖ Presence of foreign body
- ❖ Check for symptoms like decreased visual activity, diplopia, pain
- ❖ Previous history of ocular surgery
- ❖ Optimisation of underlying co-existing medical disorders
- ❖ Drug history
- ❖ NPO status of patient
- ❖ Alcohol consumption

1. Ocular protection : manipulation of injured eye must be avoided. Pressure on globe can cause extrusion of vitreous or retinal contents. Patient is instructed to avoid coughing. Patient is put in head up position which reduces venous congestion and IOP. Avoid tonometry or ocular ultrasound as they increase IOP.

2. Aspiration prophylaxis : most patients present with full stomach, consider prophylaxis with H2 receptor blockers, proton pump inhibitors, metoclopramide, sodium citrate.

3. Premedication : Pain, agitation, crying and vomiting markedly increase IOP . Excessive sedation avoided in patients with head injury or compromised airway. Intravenous opioids, Midazolam in titrated doses, atomized intranasal dexmedetomidine can be considered. Ondansetron or dexamethasone for antiemesis. Consider tetanus vaccination. Topical antibiotics before surgery can reduce endophthalmitis risk .

PHYSIOLOGY AND PHARMACOLOGICAL MAINTAINENCE OF IOP

Normal IOP ranges from 10-21 mmHg . IOP is determined by balance between aqueous humour production, outflow and extra ocular pressures. Maintenance of normal intraocular pressure (IOP) is essential for preserving ocular perfusion and visual function. Both increased and decreased IOP can have



detrimental effects on the eye. Increased IOP may reduce optic nerve and retinal perfusion, leading to optic nerve damage, visual field defects, potential blindness and extrusion of ocular contents in OGI. Conversely, decreased IOP can result in choroidal effusion, retinal folds, shallow anterior chamber, impaired vision and in severe cases retinal detachment occurs.

Factors INCREASING IOP	Factors DECREASING IOP
Hypoventilation, hypoxia, venous obstruction, uncontrolled hypertension	Most anaesthetic induction agents (dose-dependent reduction)
External pressure from face masks, fingers, orbital tumours	Majority of sedatives and hypnotics
Supine and Trendelenburg positioning	Opioids (small reduction)
Laryngoscopy and intubation (most significant spike)	Dexmedetomidine (also attenuates sympathomimetic response)
Repeated/failed laryngoscopy attempts	Hypotension, hyperventilation, hypothermia, hypocarbia
Coughing, straining, vomiting, Valsalva	Acetazolamide (? aqueous humour); Mannitol (? vitreous volume)
Succinylcholine (8–10 mmHg, resolves in 7–10 min)	Volatile inhalational agents (all reduce IOP)
Peribulbar block (transient spike, resolves in 5 min)	Propofol TIVA (sustained IOP reduction)
Ketamine (controversial; avoid as sole agent)	LMA insertion (minimal IOP change vs. laryngoscopy)
Desflurane (airway irritation ? coughing ? ? IOP)	Video laryngoscopy (attenuated pressor response vs. direct laryngoscopy)
Hypoventilation, hypoxia, venous obstruction, uncontrolled hypertension	

OCULOCARDIAC REFLEX

Also known as the Oculovagal reflex or Aschner reflex or trigeminovagal reflex — is a sudden decrease in heart rate of more than 20% from baseline, triggered by traction on the extraocular muscles, pressure on the globe, or stimulation of orbital tissues. First described by Bernard Aschner and Giuseppe Dagnini in 1908, it remains one of the most clinically important reflexes in ophthalmic anaesthesia and a recognised cause of intraoperative bradycardia, AV blocks, asystole, and cardiac arrest.

AFFERENT

Stretch receptors → Ciliary nerves → Ciliary ganglion → V₁ (Ophthalmic) → Gasserian ganglion → Trigeminal nucleus (pons)

EFFERENT

Vagal motor nucleus (medulla) → Vagus nerve (CN X) → Cardiac plexus → SA node → Bradycardia



Although the most common manifestation of the oculocardiac reflex is sinus bradycardia, a wide spectrum of cardiac dysrhythmias may occur, including junctional rhythm, ectopic atrial rhythm, atrioventricular blockade, ventricular bigeminy, multifocal premature ventricular contractions, wandering pacemaker, idioventricular rhythm, asystole, and ventricular tachycardia. Glycopyrrolate Or atropine via intravenous route can be used to counteract the vagal response and raise heart rate.

SURGICAL TRIGGERS

Trigger	Risk	Clinical Setting
Traction on extraocular muscles	HIGHEST	Medial rectus is the biggest culprit. Strabismus surgery is the prototypical case
Direct pressure on the globe	HIGH	Eye examination, intraocular surgery, ROP screening, blunt trauma, orbital haematoma
Retrobulbar / peribulbar block injection	MODERATE	The injection itself transiently triggers OCR — onset within seconds. Block does attenuate subsequent surgical OCR
Orbital surgery / decompression	MODERATE	Maxillofacial trauma reconstruction, orbital apex surgery, dacryocystorhinostomy
Enucleation / evisceration	MODERATE	Direct manipulation of optic nerve and orbital tissues
Retrobulbar haemorrhage	DELAYED	May trigger OCR hours later as periorbital pressure builds. Continue monitoring post-operatively

NON-SURGICAL TRIGGERS

Trigger	Notes
ROP examination	Retinopathy of prematurity screening — speculum insertion, scleral indentation. Profound bradycardia documented
Eyelid retraction / speculum	Particularly in neonates and infants under examination
Facial trauma (V2, V3)	Le Fort fractures, mandibular fractures, zygomatic injuries — TCR equivalent
Sinus & skull base surgery	Trigeminal nerve manipulation during endoscopic sinus or transsphenoidal surgery
Cold irrigation of the orbit	Rare iatrogenic trigger — temperature mechanoreceptor activation



CHOICE OF ANAESTHESIA TECHNIQUE: Depends on various factors like patient condition, injury type, available facilities and surgeon preferences

REGIONAL ANAESTHESIA: Historically it has been avoided due to its potential risk of extrusion of ocular contents. Additional concern of altered eye anatomy and tissue distortion and tissue acidosis from trauma, bleeding, and oedema making blocks more technically difficult.

In patients with compromised cardiopulmonary functions and potentially difficult airway where vision salvage is unlikely.

RA can also be considered as alternative to GA in specialised centers provided visual acuity is good at presentation and recent trials shows comparable outcome postoperatively as patient's who underwent surgery under GA.

INDICATIONS FOR GENERAL ANAESTHESIA: General anaesthesia is often the first choice of management.

- Corneal laceration with uveal prolapse or significant scleral extension
- Late presentation (more than 2 hours)
- Contaminated wound
- Significant corneal edema
- Extensive corneal rupture with auto-expulsion of intraocular contents and collapsed globe
- Patient with vision in only one eye
- Severe pain or discomfort that cannot be managed with local anaesthesia or sedation
- Uncooperative patient such as young children or those with cognitive impairment
- Hemodynamic instability or other medical conditions that require close monitoring and control, achievable more reliably under general anaesthesia.

SEDATION

Sedation should be used cautiously as it can cause hypoxia and hypercarbia which increases IOP. It should not be used as alternative to general anaesthesia in patients with full stomach.

There is no role of Nasogastric tube as insertion of NG tube can increase IOP.

INTRAOPERATIVE ANAESTHETIC CONSIDERATIONS :

MONITORING : standard ASA monitoring includes-

- ECG
- Pulse oximetry
- Non-invasive blood pressure
- End-tidal CO₂
- Temperature
- Neuromuscular monitoring
- Other monitoring depends on patient's co-existing diseases and age of the patient

AIRWAY MANAGEMENT :

The key to inducing anaesthesia in a patient with an open globe is controlling intraocular pressure with a smooth induction with usual preference of intravenous induction.

Awake tracheal intubation is deferred as it is accompanied by coughing and bucking during intubation and is risky in an open-globe eye injury.

However, when dealing with a possible compromised airway or difficult intubation, awake flexible fiberoptic intubation expertly done under careful sedation and airway blocks might be indicated to safely secure the airway.

Use of supraglottic device in Open Globe Injury : In managing airways during open globe injuries, SGA play a crucial role. They provide effective ventilation and can serve as a definitive airway option when there's no risk of aspiration or difficulty in airway management. Even in cases of difficult airways, they can function as rescue devices. For consideration of IOP, particularly relevant in open eye injuries, second-generation supraglottic devices are preferred

RAPID SEQUENCE INDUCTION :

In situations where surgery is considered necessary for a patient with full stomach, rapid sequence induction technique should be considered. This technique aims to mitigate the risk of aspiration while minimizing the potential for ocular damage.

High dose antiemetics administered to prevent intraocular expulsion due to vomiting. Sedatives used to achieve adequate analgesia and sedation during procedure. To reduce chance of aspiration, induction is



done with patients head up position and preoxygenation with 100% oxygen is performed to improve oxygen reserve. To reduce IOP, administer 1-1.5 mg/kg of lignocaine intravenously.

INDUCTION AGENT : The ideal induction agent for patients with full stomach would provide a rapid onset of action to minimise the risk of regurgitation would be Propofol. Etomidate is useful in cardiac disease patients, but associated with incidence of myoclonus ranging from 10%-60%. Severe myoclonus may lead to complete retinal detachment and vitreous prolapse. Ketamine has conflicting results, so not recommended in penetrating eye injuries because of increased risk of blepharospasm and nystagmus. Thiopentone used as alternative in RSI induction agent as it decreases IOP.

NEUROMUSCULAR AGENT :

Rocuronium produces excellent intubating conditions at RSI doses. Does not cause fasciculation's, unlike succinylcholine. Causes minimal or no clinically significant increase in IOP. Avoids the theoretical risk of ocular content extrusion associated with succinylcholine-induced IOP elevation. Can be rapidly reversed with Sugammadex if needed, at a dose 1.0–1.2 mg/kg IV. Provides intubating conditions comparable to succinylcholine within approximately 60 seconds, currently the choice of muscle relaxant.

Even though Succinylcholine is preferred for its has rapid onset of action (30–60 seconds), making it ideal for rapid sequence induction (RSI). Short duration of action (5–10 minutes), allowing early return of spontaneous ventilation if airway management becomes difficult. Particularly useful in unfasted patients where aspiration risk is high and rapid airway control is required. But Succinylcholine is traditionally avoided as it causes a transient increase in intraocular pressure (IOP) of approximately 8–10 mmHg, elevated IOP may theoretically promote extrusion of intraocular contents in patients with open globe injuries. Consequently, many ophthalmic surgeons prefer to avoid its use whenever feasible.

Current evidence suggests that the increase in IOP is multifactorial and results from: Reduced aqueous humour outflow, increased choroidal blood volume, Elevated central venous pressure.

Although succinylcholine consistently produces a transient rise in IOP, documented cases of actual extrusion of ocular contents attributable solely to succinylcholine are exceedingly rare. Therefore, the anaesthesiologist must balance the theoretical risk of ocular damage against the very real risk of pulmonary aspiration in an inadequately fasted patient.

Most significant increase in IOP is seen during laryngoscopy. Intubation is done carefully and gently to minimise increase in IOP. Repeated attempts of laryngoscopy should be avoided. Premedication can be done to prevent such IOP responses. IV dexmedetomidine 0.2-0.8mcg/kg administered over 10 mins prevents the response.

MAINTENANCE OF ANAESTHESIA AND VENTILATION

Controlled ventilation with mild hypocarbia causes cerebral vasoconstriction and decrease in choroidal blood volume which causes decrease in IOP.

Volatile agents, oxygen-air mixture, opioid supplementation, muscle relaxation. Nitrous oxide should be avoided if intraocular gas bubbles are present or anticipated.

Avoid hypercapnia as it increases choroidal blood volume

EXTUBATION :

Extubation is another critical phase in patients with open globe injury. Increased caution must be taken during awakening time and measures should be taken to prevent coughing, retching and vomiting as these may increase the IOP. In patients having surgery with general anaesthesia, it is a good idea to ask the surgeon to perform a local anaesthetic block before waking up the patient. If stronger analgesia is required this is best given as small intravenous doses of morphine or pethidine. Nausea and vomiting after emergency eye anaesthesia can be a major problem in some patients. Prophylactic antiemetics can be used such as ondansetron 4mg and dexamethasone 4mg. prior to extubation, IV lignocaine can be used to minimize these adverse outcomes and decrease coughing. An infusion of remifentanyl >0.1mcg/kg/min can be used with extubation to avoid sudden arousal of. Deep extubation i.e., endotracheal tube removed while patient is still under GA but maintaining spontaneous breathing is preferred in cases of OGI



Postoperative Care

The postoperative period carries ongoing risks of IOP elevation from pain, nausea, vomiting, emergence agitation and eye rubbing or squeezing. PONV is one of the most significant threats to a repaired open globe; prophylaxis must be multimodal.

Analgesia

Oral analgesia: Paracetamol 1g 6-hourly + NSAID (ibuprofen 400 mg or diclofenac 50 mg 8-hourly) forms the analgesic backbone. NSAIDs also reduce ocular inflammation. Codeine phosphate 0.5 mg/kg 6-hourly may be added in adults. Remifentanyl can be considered along with adequate treatment of PONV. Reserve IV morphine and fentanyl for breakthrough pain: titrated IV morphine 2–4 mg; avoid large boluses.

PONV Management

Ondansetron (5-HT₃ antagonist), dexamethasone target different receptors and work synergistically. Metoclopramide 10 mg IV provides prokinetic benefit. A combination of two agents from different classes is standard; three may be needed in high-risk patients.

What are your concerns in paediatric patients?

Children are the most vulnerable for OGI. They are almost always managed under GA. The OCR is more active and severe in children. Crying children is one of the most important concern because crying increases IOP. Separation anxiety is prevented by premedicating the child in preop area. Inhalational induction with sevoflurane is preferred. Dosing of all agents is weight-based. Preoperative anxiety is managed with parental presence during induction and oral midazolam and atomized intranasal dexmedetomidine. PONV is highly prevalent in children; multimodal prophylaxis is mandatory.

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2. Anaesthesia for Open Globe Injury: Indian J Ophthal Anaesth | Volume 04 | Issue 2 | Jun 2024 Revisited Bhavna Gupta¹, Kunal Bishwakarma¹, Anish

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14th JUNE 2026
10:40AM - 11:20AM

POSTERIOR FOSSA TUMOR / ACOUSTIC NEUROMA

Case summary:

A 42-year-old ASA III patient with complaints of progressive unilateral sensorineural hearing loss, tinnitus and vestibular dysfunction causing imbalance. Examination revealed isolated CN VIII involvement with positive cerebellar signs (ataxia, dysmetria, dysdiadochokinesia, positive Romberg), while higher mental functions, motor system, sensory system, other cranial nerves, and systemic examinations were normal. MRI confirmed a left CPA vestibular schwannoma (20 × 9 mm) extending into the internal auditory canal without clinical evidence of raised ICP or brainstem compression, and the patient was planned for definitive neurosurgical management.

Q1. How do you localize the pathology to posterior cranial fossa and mention the differential diagnosis based on key points in history?

Clinical presentation depends on: Site of the tumor, Rate of tumor growth

Features due to raised intracranial pressure / hydrocephalus - Severe headache, Frequent vomiting.

Symptoms due to brainstem compression

- Cranial nerve dysfunction: Involvement of 3rd, 4th, 6th cranial nerve
- Ocular palsies, Diplopia
- Long tract signs: Hemiparesis

Symptoms due to cerebellar compression

A. Vermian / midline cerebellar syndrome (Medulloblastoma, Ependymoma, Vermian astrocytoma)

- Truncal ataxia (swaying)
- Frequent falls
- Wide-based gait
- Characteristic eye findings

B. Hemi-cerebellar syndrome (Metastasis, cerebellar astrocytoma, cystic hemangioblastoma)

- Limb ataxia

- Nystagmus

- Dysmetria

Differential diagnosis:

1. Vestibular schwannoma
2. Meningioma
3. Epidermoid cyst
4. Brainstem glioma
5. Medulloblastoma
6. Ependymoma
7. Cerebellar astrocytoma
8. Metastasis
9. Hemangioblastoma

Q2. What signs indicate brainstem compression preoperatively and what is the significance of absence of papilledema in this case?

Preoperative signs of brainstem compression:

- Altered consciousness
- Respiratory irregularities
- Cranial nerve palsies (IX, X, XI, XII)

9-Loss of gag reflex, dysphagia

10- hoarseness

11- shoulder droop

12-Tongue deviation towards side of lesion, dysarthria

- Long tract signs

Motor- Spasticity, hyperreflexia

sensory-loss of pain, proprioception

coordination-sensory ataxia

Spastic gait

- Abnormal pupillary responses to light
- Bradycardia and hypertension

Significance of absence of papilledema:

- Does not exclude raised ICP. It may be absent in acute deterioration.

• Chronic slowly growing tumors may accommodate intracranially.

• Posterior fossa lesions can obstruct CSF flow before papilledema develops.



Q3. What are the determinants of intracranial compliance and how do tumors alter it?

Monro–Kellie doctrine:

Intracranial contents include brain tissue (80%), cerebral blood flow (10%), CSF (10%). Any increase in volume of one component must be compensated by a decrease in another component to maintain normal ICP.

Determinants of intracranial compliance:

1. Brain tissue elasticity - Cerebral atrophy with advanced age
2. CSF displacement: Presence of hydrocephalus
3. Venous blood displacement
4. Rate of lesion growth: Smaller volume of posterior fossa means that less mass effect of tumor is enough to produce symptoms from compression

Effect of tumors on intracranial compliance:

- Direct mass effect
- Cause vasogenic edema due to disruption of blood brain barrier
- Obstructive hydrocephalus
- Reduce compensatory reserve
- Midline shift and herniation as compliance worsens

Q4. How do you assess raised ICP clinically? Does your management alter if patient has signs of raised ICP?

Symptoms:

Headache
projectile vomiting
Visual disturbances
Altered sensorium
Seizures

Signs:

might be Papilledema
Cushing's reflex: Hypertension, bradycardia, Cheyne-stokes respiration
Pupillary changes
Sixth nerve palsy
Declining GCS

Management

If raised ICP is present: if it is due to tumors – perioperative steroids are beneficial

If raised ICP features present, a preoperative VP shunt may be done if prolonged waiting time for definitive

posterior fossa surgery

- Mannitol or hypertonic saline
- Avoid hypoxia, hypotension and hypercapnia
- Neck neutral as far as possible
- Smooth induction, adequate depth, avoid coughing/straining (Avoid drugs which will increase ICP like ketamine, succinyl choline)
- Controlled ventilation- Mild to moderate hyperventilation

Q5. How does chronic hypertension affect cerebral autoregulation?

Normal autoregulation:

CBF remains constant between MAP 50-150mmhg normotensive individuals, within this range:

- Cerebral vessels constrict during hypertension
- Cerebral vessels dilate during hypotension

This maintains stable CBF.

In chronic hypertension, there is upregulation. i.e, autoregulation curve shifts to the RIGHT (Instead of 50–150 mmHg, autoregulation may occur at 80–180 mmHg)

- Lower limit increases
- Higher pressures are needed to maintain cerebral perfusion
- Sudden BP reduction can cause cerebral ischemia

Anaesthetic implications:

- Avoid aggressive hypotension
- Maintain CPP near baseline
- Tight hemodynamic monitoring – invasive arterial beat to beat BP
- Vasopressors may be needed earlier

Q6. What is the importance of medication history in posterior fossa tumor surgery/resection?

Medication history is critical because many drugs influence intracranial dynamics, seizures, coagulation, and autonomic responses.

1. Steroids (Dexamethasone)
Reduce edema
Cause hyperglycemia and immunosuppression
2. Antiepileptics (Levetiracetam, Phenytoin, Valproate)



- Coagulation abnormalities (valproate-thrombocytopenia)
- Hepatic enzyme induction- alter anesthetic requirements Phenobarbitone, Phenytoin- resistance to NDMR with chronic use, increased dose requirements of opioids

3. Diuretics: Electrolyte imbalance Furosemide- hypokalemia, hyponatremia, hypomagnesemia

Spironolactone- hyperkalemia

Mannitol- hyponatremia

4. Antihypertensives: Influence cerebral perfusion

5. Anticoagulants/antiplatelets: Increase bleeding risk

6. Chemotherapy/immunotherapy: Organ dysfunction

Q7. How do you assess perioperative anaesthesia risk for this patient?

Perioperative anesthesia risk assessment includes:

1. Neurological assessment

- GCS
- Cranial nerve deficits
- Raised ICP
- Brainstem signs

2. Systemic evaluation

- Cardiovascular status
- Pulmonary reserve
- Renal and hepatic function

3. Airway assessment

- Aspiration risk
- Lower cranial nerve dysfunction

4. Functional capacity

- METS
- ASA grading

5. Imaging review

- Tumor size
- Hydrocephalus
- Midline shift
- Venous sinus involvement

6. Position-related risk

- Venous air embolism- An ECHO for PFO will assess risk of VAE especially in posterior fossa surgery done

in sitting position. Transcranial Doppler with bubble test is also used for screening.

- Pressure injuries: Sitting position- ulnar nerve injury, Prone position- orbital complications, lateral position- brachial plexus injury

7. Blood loss prediction

Risk stratification:

Posterior fossa surgery is considered high-risk neurosurgery requiring ICU preparedness

ASA grade 3 with mortality of 4.5%

Q8. What are the anaesthetic challenges in posterior fossa tumor surgery?

- Raised ICP
- Brainstem compression
- Venous air embolism
- Hemodynamic instability
- Massive blood loss
- Cranial nerve monitoring interference
- Need for ABSOLUTE immobility
- Positioning complications
- Delayed emergence

Specific posterior fossa concern

Proximity to respiratory and cardiovascular centers

- Risk of postoperative apnea
- Lower cranial nerve dysfunction
- Hydrocephalus

ICU and mechanical ventilation

Goals:

- Avoid ET tube kinking due to acute neck flexion
- Adequate depth of anesthesia especially in patients with poor intracranial compliance to prevent severe ICP rise and facilitate intra operative neurophysiological monitoring
- Maintain haemodynamic instability, adequate cerebral perfusion pressure and oxygenation
- Optimize conditions for neurophysiologic monitoring
- Monitor for air embolism
- Facilitate minimization of bleeding
- Smooth emergence and early awakening
- Prevent postoperative pain



Q9. How does anaesthesia affect ICP, CPP, CBF and CMRO2?

Volatile agents increase CBF and ICP dose-dependently

1. ICP

- Propofol reduces ICP. This is neuroprotective
- Hyperventilation lowers ICP via vasoconstriction.

2. CPP

CPP = MAP – ICP

- Hypotension reduces CPP.
- Raised ICP reduces CPP.

Opioids, benzodiazepines

3. CBF

- IV agents reduce CMRO2 and CBF.

Up to 1MAC in the presence of mild hypocarbia, increase in CBF due to inhalational agents can be blunted. Beyond that, ICP and cerebral blood flow increases

4. CMRO2

- Most anesthetics reduce CMRO2.
- Ketamine increases CMRO2.

An ideal neuroanesthetic:

- Reduces CMRO2
- Maintains CPP
- Avoids ICP rise
- Permits rapid recovery

Q10. What is your plan of anaesthesia and how will you prepare the OT?

Anaesthetic Plan

- General anaesthesia with endotracheal intubation
- Preferred: Propofol-based TIVA + short-acting opioid (remifentanyl/fentanyl)

Premedication: avoid sedatives and opioids, risk of hypercarbia

Induction: intravenous induction

Maintenance: IPPV with muscle relaxant avoid muscle relaxant if CN or MEP monitoring is needed), maintenance with O2 (preferably without N2O)

- Maintain normocapnia/mild hypocapnia
- Avoid hypoxia, hypercapnia, hypertension
- Mannitol/steroids if raised ICP
- Smooth emergence for early neurological assessment

OT Preparation

- Difficult airway cart ready
- Infusion pumps for TIVA
- Blood and blood products arranged
- Mannitol, hypertonic saline, vasopressors available
- Air warmer and fluid warmer
- Defibrillator and emergency drugs ready
- VAE management setup: saline irrigation, aspiration syringes

Monitoring

- Standard ASA monitors such as NIBP, HR, SpO2, temperature, ETCO2
- Invasive arterial BP monitoring
- Foley catheter
- Multi-orifice central venous catheter for VAE aspiration
- Precordial Doppler/TEE if sitting position
- Neuromonitoring: BAER, SSEP, facial nerve EMG

Positioning Precautions

- Neutral neck alignment
- Adequate padding of pressure points
- Avoid excessive neck flexion
- Protect eyes, ears, and peripheral nerves

Postoperative Plan

- Awake the patient assess neurological status
- Extubate only if neurologically stable

If not, re-sedate and transport to ICU with Remifentanyl infusion of 0.1 mcg/kg/min

- ICU monitoring
- Watch for airway edema, lower cranial nerve palsy, VAE complications



Q11. TIVA vs inhalational anaesthesia? Advantages of propofol-based TIVA/dexmedetomidine.

Feature	TIVA (Propofol-based)	Inhalational
CBF/ICP	↓ CBF & ICP	↑ CBF, may ↑ ICP
Brain relaxation	Excellent	Less optimal
Neuromonitoring	Better BAEP/SSEP/MEP	Suppresses signals
Emergence	Rapid & clear	Delayed possible
PONV	Less	More
VAE risk	Avoids N ₂ O expansion	N ₂ O worsens VAE
Autoregulation	Better preserved	Impaired at high MAC

Advantages of Dexmedetomidine

- Hemodynamic stability – blunts sympathetic response
- Propofol/opioid sparing effect
- Smooth emergence with less coughing/bucking
- Better postoperative analgesia
- Minimal respiratory depression

Advantages of Propofol-based TIVA

- ↓ CMRO₂, CBF, ICP → excellent brain relaxation
- Better preservation of BAEP/SSEP/MEP & facial nerve monitoring
- Rapid smooth emergence for early neurological assessment
- Lower postoperative nausea and vomiting
- Avoids nitrous oxide-related VAE expansion and tension pneumocephalus

Q12. What monitoring parameters do you use? Comment on significance of each.

Routine Monitoring

NIBP

- ECG
- Pulse oximetry
- Capnography
- Temperature: Tympanic membrane

- Urine output
- Neuromuscular monitoring

Invasive Monitoring

Arterial Line: Beat-to-beat BP monitoring, serial ABG (Transducer will be fixed at the level of EAC- corresponding to level of circle of Willis)
Indications: sitting position, major blood loss, haemodynamic instability

Right subclavian Central Venous Catheter: CVP monitoring, Aspiration of air during VAE

Special Monitoring for detection of VAE

Precordial Doppler, TEE

Transcranial Doppler:

- Detects paradoxical air embolism

Neurophysiological Monitoring

Facial Nerve Monitoring:

- Used in CPA tumors/acoustic neuroma
- EMG + direct stimulation
- Helps preserve facial nerve function

BAER:

- Hearing preservation monitoring
- Loss of wave V → hearing loss

SSEP:

- Monitors sensory pathways/ischemia

MEP:

- Monitors motor pathways
- Muscle relaxants interfere



Q13. Discuss the intraoperative complications and their management.

A. Venous Air Embolism – most lethal complication

Cause: Sitting/semi sitting position, open venous sinuses.

Recognition:

Clinical-Bradycardia, hypotension, arrhythmias, cardiovascular collapse

TEE- most sensitive, detect volumes of 0.02ml/kg

Precordial Doppler- detect volumes of 0.05ml/kg

ETCO₂- decreases

Pulmonary artery catheters- less sensitive, 0.25ml/kg, sudden increase in pulmonary artery pressures

End tidal nitrogen (N₂) – increases

Esophageal stethoscope: low sensitivity -1.7ml/kg, presence of mill wheel murmur

Management: Inform surgeon, 100% O₂, Durant position, flood field with saline, aspirate air via CVC, b/l jugular vein compression, vasopressors/CPR if needed.

Prevention: Avoid excess head elevation, ensure euvolemia, maintain adequate venous pressure, irrigation of operative field, immediate occlusion of open veins

B. Brainstem Manipulation → Hemodynamic Instability

Recognition: Bradycardia, hypotension, hypertension, arrhythmias, asystole.

Management: Stop stimulation, atropine, epinephrine, antiarrhythmics, ACLS if arrest.

C. Massive Blood Loss

Recognition: tachycardia, hypotension, weak peripheral pulses, arrhythmia, cardiovascular collapse, dampened arterial waveform, decrease in ETCO₂

Management: Rapid transfusion with blood and blood products, correct coagulopathy, calcium replacement if indicated.

D. Cranial Nerve Injury

Commonly affected: CN VII, IX, X.

Recognition: Tense dura, pupillary changes, EEG

slowing, Loss of SSEPs

Clinical: Bradycardia, hypertension, arrhythmias

Prevention: Facial nerve & BAEP monitoring; avoid long-acting muscle relaxants.

Management: Airway assessment, delayed extubation if lower CN palsy.

E. Raised ICP / Brain Swelling

Recognition: Bradycardia, hypertension, increased airway pressure

Prevention: Mannitol, steroids, mild hyperventilation, head elevation.

Management: Mannitol/hypertonic saline, controlled ventilation, decompression if required.

F. Tension Pneumocephalus

Risk factors: Sitting position, nitrous oxide.

Recognition: Bradycardia, Hypertension, Seizures, Delayed emergence

Prevention: Avoid N₂O during closure.

Management: 100% O₂, decompression if severe.

G. Position-Related Complications

Includes: Macroglossia more common in prone position, neuropathy, pressure sores

Recognition: At end of surgery on examination

Prevention: Neutral neck position, adequate padding.

Management: Repositioning, airway support.

H. Electrolyte / Fluid Disturbances

Causes: Mannitol, long surgery.

Recognition: Tachycardia, ventricular arrhythmias, bradycardia, tall peaked T waves, wide QRS, seizures, delayed awakening

Prevention: serial ABG/electrolytes.

Management: Correct Na⁺/K⁺ abnormalities, monitor urine output.

Q14. How will cranial nerve/neurophysiological monitoring impact anesthesia management?

Anaesthetic technique directly affects the quality of evoked potentials and EMG signals.

Preferred Anaesthetic Technique

- TIVA (Propofol + opioid infusion)
- Provides stable and reliable monitoring signals



Effect of Volatile Agents

- Volatile agents suppress SSEPs and MEPs dose-dependently
- Decrease amplitude and increase latency
- MEPs highly sensitive to inhalational agents
- Use low MAC (<0.5 MAC) if volatile agents required

Nitrous Oxide

- Suppresses evoked potentials

Neuromuscular Blockers

- Major implication in MEP and EMG monitoring
- Use short-acting relaxant only for intubation, Avoid repeat doses during maintenance

- Facial nerve EMG requires intact neuromuscular transmission

Metabolic Factors Affecting Signals

- Correct hypoxia, anemia, electrolyte imbalance, and hypoglycemia
- Physiological stability improves signal reliability

Response to Sudden Signal Loss

- Inform surgeon immediately
- Check BP, oxygenation, anesthetic depth
- Assess positioning and surgical traction
- Correct physiological disturbances promptly

Position	Uses	Advantages	Disadvantages	Key points
Sitting	Midline posterior fossa lesions	Excellent exposure, ? bleeding, ? ICP, gravity drainage of blood/CSF, good facial nerve monitoring	VAE, paradoxical air embolism, hypotension, pneumocephalus, quadriplegia, nerve injury, macroglossia	Contraindicated in R? L shunt, VA shunt
Prone / Concorde	Midline & medial cerebellar lesions	Good access, ? VAE vs sitting	Facial/tongue edema, ETT kinking, pressure injuries, blindness, ? ICP if abdomen compressed	Avoid excessive neck flexion
Lateral	CPA & lateral cerebellar lesions	Hemodynamically safest, ? aspiration, ? VAE	V/Q mismatch, brachial plexus & peroneal nerve injury	Use axillary roll
Park-bench	CPA surgery	Better lateral exposure, good venous drainage	Similar to sitting position,	Head rotated ~30°
Semi-sitting	Alternative to sitting	Better exposure with less physiologic disturbance	VAE, hypotension	Less severe than full sitting
Supine with neck tilt	Rarely used; MVD/translabrynthine approach	Easy airway access	Limited exposure, brachial plexus stretch injury	Limited role

Q16. What are the postoperative complications? Role of ICU monitoring postoperatively?

Neurological complications: Altered intracranial pressure dynamics, Acute Hydrocephalus, Aseptic meningitis, Residual neuromuscular blockade, Cranial nerve dysfunction

Airway: Macroglossia, airway edema, Respiratory failure

Cerebral Edema, hematoma

Cardiovascular complications: Hypotension, Arrhythmias

Paradoxical cerebral air embolism

ICU role:

- Ventilatory support
- Neurologic monitoring
- Hemodynamic stabilization
- Electrolyte management
- Early complication detection



Q17. How will you manage postoperative analgesia in this patient?

Goals of postop analgesia:

- Adequate pain relief
- Avoid oversedation
- Permit neurologic assessment

Multimodal analgesia:

- Paracetamol and NSAIDS
- Opioid PCA
- Scalp block at the beginning of surgery
- Dexmedetomidine infusion: Intraoperative use can reduce post op analgesia requirement. Postoperative alone has more sedative and less analgesic benefit.

Q18. What are the anaesthetic implications if the same patient is posted for non-intracranial surgeries?

Peripheral nerve block:

Residual neurologic deficit

Neuraxial blockade:

Imaging to be done to rule out tumor recurrence/residual mass

Raised ICP

VP shunt presence – risk of pneumocephalus

General anesthesia: Maintain CPP

H/o Seizure disorder – anaesthetic drug metabolism altered by antiepileptic's

Cranial nerve dysfunction: Aspiration risk if lower cranial nerves affected



14th JUNE 2026
11:20AM - 12:00Noon

MORBID OBESITY WITH OBSTRUCTIVE SLEEP APNOEA – CASE DISCUSSION.

Dr C L Gurudatt, Mysore

Dr Prabhu B G, Davanagere

A 48 years old male patient has a body mass index of 38 kg/m² with class 2 obesity also gives history of obstructive sleep apnoea. He is on CPAP of 10 cm H₂O at night. He is a known hypertensive and on tablet losartan 40 mg once a day and subcutaneous low molecular weight heparin. He does not give any h/o angina or coronary revascularization procedures. He tried medical management for reduction of body weight but failed. His airway assessment has not found any expected difficult airway. He is posted for laparoscopic bariatric surgery.

1) Define obesity?

The World Health Organisation (WHO) has defined obesity as a condition with excess body fat to the extent that health and wellbeing is adversely affected.

2) What is Ideal body weight (IBW)?

Ideal body weight is calculated using Broca's index. It is the weight associated with the lower mortality rate for a given height and gender.

IBW = Height in CM – 100 kg for males

Height in CM – 105 kg for females

3) What is Predicted Body Weight (PBW)?

For Males, PBW (kg) = 50 + 2.3 (height in inches – 60)

For Females, PBW (kg) = 45.5 + 2.3 (height in females – 60)

Or

For Males, PBW (kg) = 50 + 0.91 x (height in cm – 152.4)

For Females, PBW (kg) = 45.5 + 0.91 x (height in cm – 152.4)

PBW is usually used to calculate the tidal volume in patients on mechanical ventilation.

4) What is Adjusted Body Weight (ABW)?

ABW = Ideal body weight + 0.4 (Actual body weight – Ideal body weight)

For e.g. A male patient who is 165 cms and 90 kgs, then

ABW = 65 + 0.4 (90-65)

= 65 + 10 = 75 kgs

ABW can be used to calculate the dose of a majority of

the drugs used during anaesthesia except a few like Succinyl choline, propofol infusion, neostigmine, sugammadex, dexmedetomidine the dosage of which is calculated based on the actual body weight.

5) What is Lean Body Weight (LBW)?

It is the total body weight (TBW) – adipose tissue

LBW in Males = 1.10 x TBW – 0.0128 x BMI x TBW

LBW in Females = 1.07 x TBW – 0.0148 x BMI x TBW

In, morbidly obese patients, increasing the LBW by 20%-30% gives an estimate of LBW, in non-obese and non-muscular patients, TBW approximate LBW.

6) What is Body mass Index?

It is also called as Quetlet index. It is used to estimate the degree of obesity.

BMI = Weight (kg) / Height (m²)

7) How obesity is graded based on BMI?

BMI	GRADE
< 18.5	Underweight
18.5 - 24.9	Normal
25 - 29.9	Over weight
30 - 34.9	Obesity class 1
35 - 39.9	Obesity class 2
40 - 50	Morbid Obesity class 3
50 - 60	Super obesity
>60	Super Super obesity

8) What are android and gynaecoid obesity?

In android (central) obesity, adipose tissue is located predominantly in the upper body truncal distribution and is associated with increased Oxygen (O₂) consumption and increased incidence of cardiovascular system (CVS) disease. In gynaecoid obesity (peripheral), adipose tissue is located predominantly in the hips, buttocks and thighs. This fat is less metabolically active, so it is less likely associated with CVS disease.

9) What are the different anthropometric measurements used to assess increased cardiovascular system (CVS) risks?



Waist circumference > 88cms / 35 inches (women) & > 102 cms / 40 inches (men)

Waist – Hip ratio: > 0.85 (women) & > 0.9 (men)

Waist to height ratio > 0.5

These measurements are more reliable than BMI for identifying CVS risks.

A waist circumference of > 80 cms (30 inches) in Asian females and > 90 cms (35 inches) in Asian males is considered abnormal and carry a higher CVS risk.

10) What are the pathophysiological changes that occur in respiratory system in obese patients?

- Obese patients will have a restrictive type of lung disease with increased fat accumulation on the thorax and abdomen with decreased chest wall and lung compliance.
- Decreased lung compliance is also due to increased pulmonary blood volume. Lung compliance further reduces in supine position leading to shallow and rapid breathing, increased work of breathing and limited maximum ventilatory capacity.
- Decreased lung compliance leads to decreased FRC, vital capacity and total lung capacity. Decrease in Functional Residual Capacity (FRC) is due to decrease in Expiratory Reserve Volume (ERV) (Every unit increase in BMI above 30 kg/m², decreases FRC by 1%). Residual Volume (RV) and Closing Capacity (CC) are unchanged.
- Decreased FRC results in lung volume below CC during normal tidal ventilation leading to airway closure, Ventilation Perfusion (V/Q) mismatch, right to left shunting and arterial hypoxemia.
- Anaesthesia and supine positioning worsen this situation such that up to 50% reduction in FRC occurs in obese anaesthetised patients compared with 20% in the non-obese patients. (Figure 1)
- Ratio of Forced Vital Capacity (FVC) & Forced Expiratory Volume in first second (FEV₁), are usually within normal limits
- Obese patients have increased O₂ consumption and increased CO₂ production which leads to increased cardiac output and increase in alveolar ventilation.
- Chronic hypoxemia leads to – Polycythemia, pulmonary hypertension and cor pulmonale.

11) What are the anesthetic implications of changes in

respiratory system?

- Shortened time to desaturation during periods of apnoea due to decreased FRC and increased oxygen consumption and relative increase in CC.
- Increased oxygen requirement – need of supplemental oxygen
- Hypoventilation with supine spontaneous ventilation
- Extreme caution while administering sedation
- Non supine positioning if possible
- CPAP when indicated

12) What is obstructive sleep apnoea and hypopnea?

OSA is defined as complete cessation of breathing for more than 10 seconds in spite of vigorous respiratory efforts with more than 5 times per hour during sleeping, with oxygen saturation decreasing by > 4%.

Hypopnea is defined as partial obstruction of breathing i.e. >30% for more than 10 seconds, 15 or more times per hour associated with a decrease of saturation greater than 4%

13. What is apnoea hypopnoea index (AHI)?

Results of polysomnography are reported as the apnea–hypopnea index (AHI), which is derived from the total number of apneas and hypopneas divided by the total sleep time.

14. Based on AHI how severity of OSA is classified?

- Mild Disease: AHI of 5 to 15 events per hour
- Moderate Disease: AHI of 15 to 30 events per hour
- Severe Disease: AHI of greater than 30 events per hour

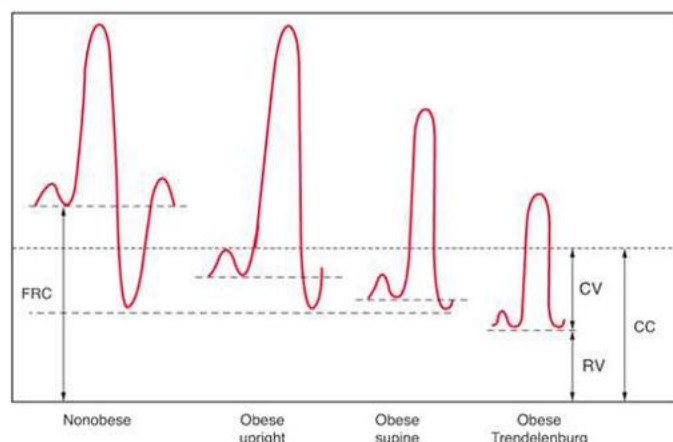


Figure 1 - Effects of obesity , positioning, and anaesthesia on lung volumes. FRC, functional residual capacity; CC, closing capacity; CV, closing volume; RV, residual volume



15) What are the consequences of OSA?

Physiological abnormalities resulting from OSA include - hypoxemia, hypercapnia, pulmonary hypertension, systemic vasoconstriction, hypertension and secondary polycythemia from recurrent hypoxia. There is an increased risk of IHD & cerebrovascular disease. Right ventricular failure can occur from hypoxic pulmonary vasoconstriction. Respiratory acidosis can occur during sleep. increased risk of sudden cardiac deaths due to fatal arrhythmias.

16) What is STOP BANG questionnaire to diagnose OSA?

S – significant snoring

T – Tiredness during day time.

O – Observed apneic spells

P – Pressure – high BP

B – BMI > 35 kg/m²

A – Age older than 50 years

N – Neck circumference >40cm (F), >43cm (M)

G – Gender – male

Using four equally weighted historical questions (STOP) and four equally weighted objective findings (BANG), points are assigned to create an overall score. A total score of 0–2 is low risk, 3–4 is intermediate risk, and 5–8 is high risk of OSA.

How ever the gold standard diagnostic technique is overnight polysomnography (OPS) or sleep study is required.

17) How preoperative CPAP help OSA patients?

Preoperative initiation of CPAP has been found to improve hypertension, glucose and lipid metabolism when added to a weight loss intervention.

18) What is the anesthetic implication of OSA?

Patients with OSA are considered to have difficult airway with difficulty for mask ventilation, laryngoscopy, intubation, supraglottic airway insertion and front of neck access. They are extremely sensitive to sedatives and hypnotics. They are highly prone for postoperative pulmonary complications. For preventing post-op pulmonary complications, these patients should be maintained in semi upright posture with continuous pulse oximetry monitoring, with a focus to avoid opioid based analgesia and therapy with titrated oxygen &/or CPAP as needed to avoid hypoxemia and hypoventilation.

19) What is obesity hypoventilation or Pickwickian syndrome?

It is defined as the presence of alveolar hypoventilation during wakefulness, resulting in pulmonary hypertension and cor pulmonale, increased risk of post-op complications and death. The presence of both obesity (BMI > 30 kg/m²) and awake arterial hypercapnia (PaCO₂ > 45mm Hg) in the absence of known causes of hypoventilation support the diagnosis.

20) What are the changes that take place in cardiovascular and haematologic systems in obesity?

Total blood volume is increased. Most of the extra volume is distributed to adipose tissue. On a volume to weight basis the blood volume is less than in non-obese individuals i.e. 50ml/kg in obese and 70 ml/kg in non-obese. Blood flow to fat is 2-3 ml/ 100 gm of tissue. Adipose tissue requires less blood supply.

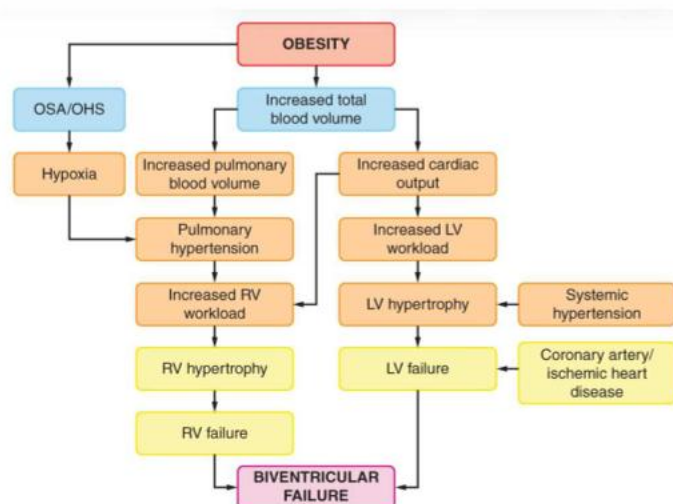


Figure 2 - Interrelationship of cardiovascular and pulmonary sequelae of obesity . OSA, obstructive sleep apnoea ; OHS, obesity hypoventilation syndrome; LV, left ventricular; RV, right ventricular.

Cardiac output increased to 20-30 ml/kg for extra fat. This leads to left ventricular hypertrophy (Eccentric), decreased compliance, diastolic dysfunction with elevated left ventricular diastolic pressure and pulmonary edema. Chronic hypoxia produces increase in pulmonary vascular resistance leading to increased afterload for the right ventricle resulting in right ventricular hypertrophy and dilatation (Figure 2).

Systemic hypertension produces concentric left



ventricular hypertrophy (LVH), systolic dysfunction leading to obesity cardiomyopathy. There can be biventricular failure (Figure 2). Cardiac dysrhythmias may be precipitated by fatty infiltration of the conduction system, hypoxia, hypercarbia, electrolyte imbalance, coronary artery disease (CAD), increased circulating catecholamines, OSA and myocardial hypertrophy.

21) What are the expected ECG changes in obese patients?

Low QRS voltage, LVH strain pattern, left atrial enlargement, T wave flattening in the inferior and lateral leads, Prolonged Q-T interval.

22) What is the cause of hypercoagulability in obese patients?

Adipose tissue releases many bioactive mediators. These can result in abnormal lipids, insulin resistance, inflammation and coagulopathies. Obese patients have higher levels of fibrinogen, factor 8, factor 7, von Willebrand factor and plasminogen activator inhibitor. All these are associated with hypercoagulability.

23) What are the changes in gastro-intestinal system in obese patients?

Gastric volume and acidity are increased, hepatic function is increased, hepatic function is altered, drug metabolism is adversely affected. Delayed gastric emptying occurs because of increased abdominal mass, that causes antral distention, gastrin release and decrease in pH with parietal cell secretion. These patients also will have increased incidence of hiatal hernia and gastro-esophageal reflux which further increases aspiration risk. They should be considered to have full stomach if they have history suggestive of gastro esophageal reflux disease (GERD) and precautions to prevent acid aspiration during induction.

24) What are the changes in hepatic system in obese patients?

Fatty infiltration of the liver, non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH) are common, fatty infiltration reflects the duration rather than the degree of obesity. Features suggestive of NASH include hepatomegaly, elevated liver enzymes, abnormal liver histology, steatohepatitis, fibrosis and cirrhosis

25) What are the endocrinal and renal system changes in obesity?

These patients will have increased incidence of type 2 diabetes mellitus (DM) due to insulin resistance. There is an increase in the risk of developing DM by 2.5% for every 1 kg/m² increase in BMI above 22 kg/m². There is an increase in the risk of myocardial infarction and wound infection. They may require exogenous insulin in the intraoperative period. Subclinical hypothyroidism is also more common. Obesity is associated with increased GFR and glomerular hyperfiltration. There can also be impaired natriuresis and activation of Renin Angiotensin Aldosterone System (RAAS).

26) What is metabolic syndrome?

Metabolic syndrome also called as syndrome X, is diagnosed when 3 out of 5 of these conditions exist.

- a) Central obesity – Waist circumference > 102cm in males and > 88cm in females
- b) Dyslipidemia – Triglycerides > 150mg/dl
- c) HDL – 40mg/dl & 50mg/dl in females or less
- d) Hypertension
- e) Elevated fasting glucose > 100mg/dl

These patients are prone for increased perioperative adverse events including CVS, Pulmonary, renal complications and wound infections.

27) What is inflammatory syndrome of obesity?

These patients are at risk of higher rate of perioperative infection. This phenomenon may be due to inability of neutrophils to activate, mitigate and adhere to sites of inflammation as a result of adipose tissue secretion of various proinflammatory cytokines or adipokines

28) Why obese patients are prone for thromboembolic disorders?

This is mainly due to polycythemia, increased intra-abdominal pressure, increased fibrinogen levels and immobilisation leading to venostasis. There is decrease in fibrinolysis which renders these patients to develop deep vein thrombosis. Perioperative use of low molecular weight heparin and sequential compression stockings is advocated.

29) What degenerative joint diseases are more common in obese patients?

Osteoarthritis and degenerative joint diseases are common. Hyperuricemia and gout and vitamin D deficiency may also contribute. Because of



degenerative joint diseases positioning for central neuraxial blockade may be difficult and also early post operative mobilization.

30) When medical therapy is advocated to treat obesity?

Patients with a BMI of 27 kg/m² with persistent co-morbid conditions like hypertension or diabetes mellitus and in patients with BMI > 30kg/m² with no comorbidities, medical therapy is advocated.

Drugs that suppress appetite – Phentermine, Drugs that prevent the hydrolysis of dietary fat into absorbable fatty acids – Orlistat, Drugs that increase satiety – GLP 1 receptor agonists are commonly used.

Phentermine can induce tachycardia, palpitation and hypertension.

Orlistat may result in fat soluble vitamin deficiency. Vit K deficiency may induce coagulopathy and require correction 24 hours before surgery.

GLP 1 receptor agonists may produce delayed gastric emptying and may have to be stopped 5 -7 days pre operatively.

31) When bariatric surgery is indicated?

Bariatric surgery is indicated with BMI of > 40 kg/m² or BMI of > 35 kg/m² with obesity related co-morbidities not controlled with medical therapy.

32) What are the different types of bariatric surgeries advocated?

Malabsorptive surgeries – jejuno-ileal bypass, biliopancreatic diversion.

Restrictive surgeries – sleeve gastrectomy, vertical banded gastroplasty, adjustable gastric banding.

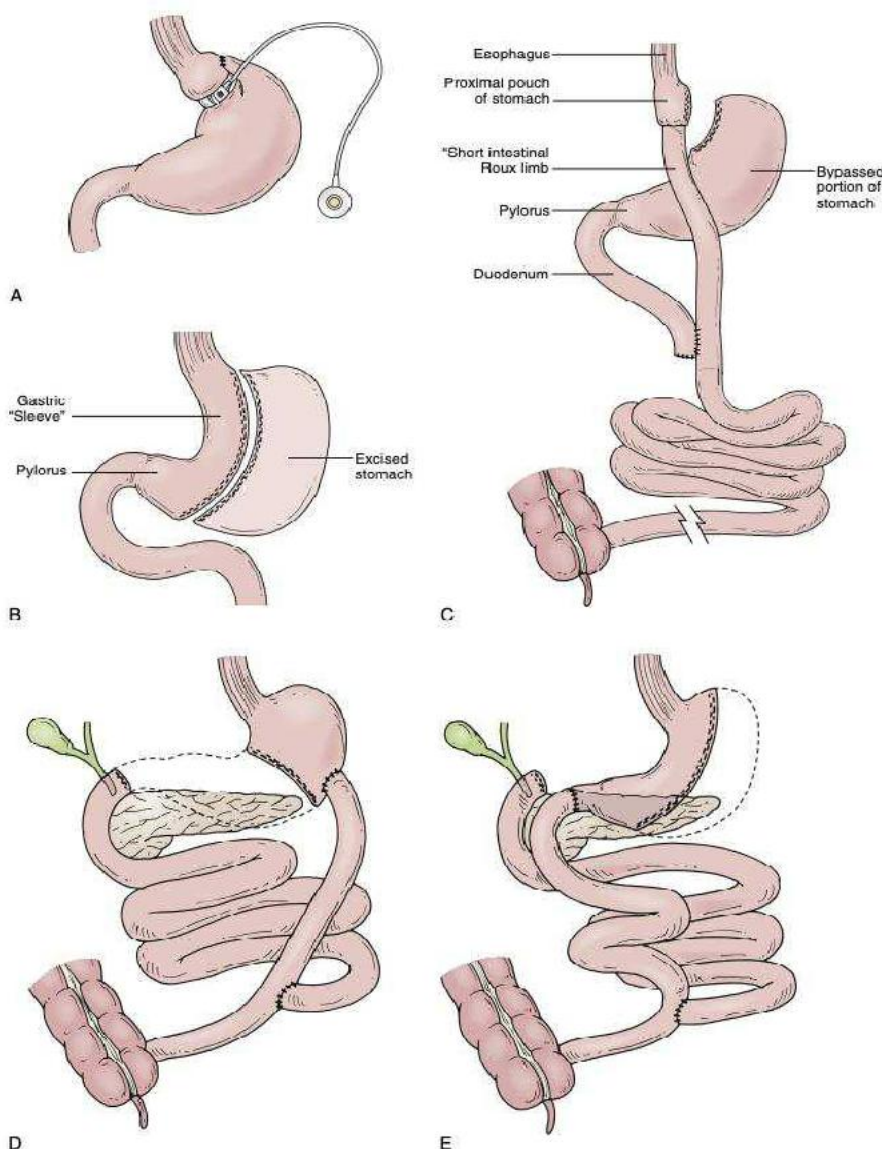


Figure - 3, Adjustable gastric band (AGB). A silicone band is looped around the proximal stomach to create a 15- to 20-mL pouch with an adjustable outlet. The stomach is wrapped around the band anteriorly to prevent the band from slipping out of position. The band consists of a rigid outer ring and an inner inflatable balloon reservoir connected by tubing to a subcutaneous port that can be accessed through the skin to adjust the tightness. B, Sleeve gastrectomy. A narrow gastric sleeve is created by stapling the stomach vertically. The fundus and greater curve of the stomach are removed from the abdomen. C, Roux-en-Y gastric bypass (RYGB). A small gastric pouch (15–30 mL) is created by division of the upper stomach, connected to a 100- to 150-cm limb of jejunum called the Roux limb. The small gastric pouch results in restriction of food intake. D, Biliopancreatic diversion (BPD). Most of the small bowel is bypassed, and only 50–100 cm of a common channel remains for absorption of calories and nutrients. The upper pouch is larger than that of the RYGB to allow for ingestion of larger amounts of protein to prevent malnutrition. E, Bilio-pancreatic diversion with duodenal switch (BPD-DS). To avoid dumping syndrome and maintain the pylorus, the procedure was modified with the pouch based on the lesser curve of the stomach and an anastomosis at the first portion of the duodenum.

33) How drug dosing is calculated in obese patients?

Volume of distribution is affected in obese patients due to:

- Decreased fraction of total body water
- Increased adipose tissue
- Increased lean body mass
- Altered tissue protein binding
- Increased blood volume and cardiac output
- Increased concentration of free fatty acids, cholesterol
- Organomegaly.

Plasma protein binding is changed leading to the following:

- Adsorption of lipophilic drugs to lipoproteins so increased free drug available
- Plasma albumin unchanged
- Increased α_1 -acid glycoprotein.

Drug clearance is affected by:

- Increased renal blood flow
- Increased GFR

- Increased tubular secretion
- Decreased hepatic blood flow in congestive cardiac failure.

Usually, the loading dose depends on the distribution volume and maintenance dose depends on the clearance. A drug that is distributed mainly to lean tissues the loading dose is calculated based on lean body weight. A drug which has similar clearance values in obese and non-obese the maintenance dose is based on Lean body weight. A drug that is equally distributed among lean and adipose tissues, loading dose is calculated based on total body weight and whole clearance increased with obesity is also calculated based on TBW.

Except drugs like succinylcholine, atracurium, dexmedetomidine, midazolam, neostigmine and sugammadex loading doses and propofol infusion which are calculated based on total body weight, all other drugs are calculated based on lean body weight. Lean body weight is roughly calculated as 80% in males and 75% in females of the total body weight. It is approximately same as adjustable body weight = ideal body weight + 0.4 (TBW-IBW).

34) What important considerations to be kept in mind while doing pre-anaesthetic evaluation in obese patients posted for bariatric surgery?

The focus of the history and physical examination should be on the cardiovascular (CVS) and respiratory systems (RS) and on airway examination. Many of these morbidly obese patients lead a very sedentary lives and so eliciting symptoms of CVS & RS diseases may be difficult.

It is very important to enquire about chest pain, shortness of breath at rest or with minimal exertion, palpitations and position with which the patient sleeps. Symptoms of pulmonary hypertension like exertional dyspnoea, fatigue and syncope which reflect an inability to increase cardiac output during activity should be asked. Symptoms of OSA and STOP-BANG questionnaire should be taken. Symptoms of acid reflux, inability to lie flat without coughing or heart burn may indicate GERD or delayed gastric emptying. In diabetic patients, symptoms of immediate complications like hypoglycemic episodes and hyperglycemic non ketotic state and delayed complications like micro and macro angiopathy -



Neuropathy - Peripheral and Autonomic, Nephropathy, Retinopathy and Ischaemic heart disease, Cerebrovascular disease and Peripheral vascular disease respectively, should be noted. Physical capacity in terms of metabolic equivalents (METs) to be assessed.

During examination, all anthropometric measurements should be taken. Height and weight should be measured and grade of obesity the patient belongs to based on BMI should be done. Examination of the spine & other anatomical landmarks for regional anaesthesia should be undertaken. Patients with severe obesity and at least one risk factor for CHD (diabetes, smoking,

hypertension, or hyperlipidaemia) or poor exercise tolerance should have a 12-lead electrocardiogram (ECG) and chest radiograph prior to surgery. Further cardiac work-up may be indicated based on the results of these tests. Other elements of preoperative cardiac evaluation and management are similar to those for patients with normal BMI.

35) What pre-operative diagnostic tests are ordered?

Investigations like complete blood count, urine analysis, ECG to look for right ventricular hypertrophy (RVH), left ventricular hypertrophy (LVH), dysrhythmias, Ischaemic heart disease (IHD), chest-x-ray for cardiomegaly and signs of heart failure should be done. Transthoracic ECHO to evaluate right and left ventricular systolic and diastolic functioning and pulmonary hypertension, an arterial blood gas analysis (ABG) to know the CO₂ and O₂ status which also helps to guide the ventilatory settings to be done. ABG should be taken in supine and upright positions if underlying respiratory comorbidities are suspected (OSA, OHA) and to evaluate the need for perioperative oxygen administration and postoperative ventilation. A sleep study to find out the severity of OSA and also to find out the requirement of CPAP or BIPAP may be undertaken. If the patient is already using these devices, he should be asked to bring them for use during post-op period.

36) Any specially designed operation table is required in morbidly obese patients?

Specially designed tables or two regularly sized operating room tables may be required. Regular operation theatre tables have a maximum weight limit of approximately 200 kg. In contrast, tables capable of

holding up to 455 kg, with a wider or side attachment to accommodate the extra girth, may be required for safe anaesthesia & surgery in obese patients.

Patients are frequently wider than table and positioning an arm by the side may be difficult. Due to the layer of fat around arm and back, patient's shoulders are frequently far above the table and arm boards, making positioning of arms on arm boards uncomfortable for patient and fastening the arms under tension under anaesthesia will put brachial plexus at risk of hyperabduction injury. To prevent this extra padding may be required on arm board.

Operation table should be powered as manual operating tables become difficult to manage. Standard straps may not reach around patients and may make patient prone to fall.

Postoperative bed should have higher weight limit than that used for normal patients.

37) What are the problems of different positions adopted during surgery in obese patients?

Supine positioning causes ventilatory impairment & inferior vena caval and aortic compression in obese patients. FRC & oxygenation are decreased further with supine positioning. Simply changing the obese patient from a sitting to a supine position can cause a significant increase in O₂ consumption and cardiac output.

Head down positioning often required for bariatric surgeries further worsens FRC & should be avoided if possible.

Head up position provides the longest safe apnoea period during induction of anaesthesia as many times longer time will be required for intubation. Head up position with CPAP or PEEP will both increase PaO₂ and respiratory compliance and also decrease airway pressures. Both head up position and PEEP can decrease cardiac output and partially counteract the benefits of oxygenation.

Lateral decubitus position allows for better diaphragmatic excursion and should be preferred over prone positioning whenever surgical procedure permits.



Whatever may be the position adopted particular care should be given to protect pressure areas to prevent pressure sores, neural injuries and rhabdomyolysis.

38) What difficulties are encountered in monitoring of obese patients?

Careful selection of proper sphygmomanometer cuff size is important for accurate non-invasive monitoring of B.P. If the cuff is small, it can give a falsely elevated B.P measurement. A cuff with bladder that encircles 75% of the upper arm circumference or preferably the entire arm size should be preferred.

If larger cuffs are not available, forearm measurement with standard cuff maybe used. Sometimes invasive BP monitoring may be required for accurate monitoring. Volume clamp system is a new technology that uses finger cuff for non-invasive continuous beat to beat B.P measurement as well as measurement of additional haemodynamic parameters. It can be used for B.P measurements intraoperatively in obese patients.

Central venous catheterisation may be required for IV access as the peripheral venous access may be inadequate for fluid management.

Needle electrodes may be required for ECG monitoring and neuromuscular junction monitoring to decrease the electrical impedance.

39) What are your anaesthetic goals?

- Avoid hypoxia, hypercarbia, barotrauma, and volutrauma. Minimise atelectasis and minimise the use of respiratory depressant drugs
- Maintain stable haemodynamics. Avoid an increase in pulmonary artery pressures. Ensure adequate tissue perfusion. Avoid supply-demand mismatch.
- Prevention of deep vein thrombosis, aspiration
- Adequate analgesia
- Maintain euglycemia
- Preparedness to handle difficult airway
- To ensure adequate intravenous access
- To prevent pressure sores and nerve injuries.

40) What is the anaesthetic technique of choice for laparoscopic bariatric surgeries?

General endotracheal anaesthesia is the technique of choice for bariatric surgeries. Controlled ventilation is preferred over spontaneous ventilation to prevent hypoventilation.

Endotracheal tube is preferred as supraglottic airway may not maintain a good seal as they require higher airway pressures due to the decreased lung compliance. These patients especially having symptoms of GERD or hiatus hernia are more prone for aspiration and SGA do not protect from aspiration of stomach contents.

41) How will you optimise the condition of this patient before surgery?

- A multidisciplinary approach should be taken including a physician, dietician or nutrition specialist, physiotherapist, surgeon, anaesthesiologist, and allied personnel
- Preoperative weight loss should be considered.
- Physiotherapy and exercise Programmes to improve glucose tolerance and respiratory capacity
- Deep breathing exercises and incentive spirometry to improve perioperative oxygenation and prevent atelectasis
- NIPPV- CPAP with 10cm H₂O and BiPAP may be required
- Euglycemia and euthyroid state should be achieved
- Habits such as smoking, and alcohol should be stopped
- Antacid prophylaxis - as these patients are prone to aspiration- PPIs, H₂ receptor antagonists, and prokinetics should be given in the pre-operative period
- Obesity is a risk factor for DVT. The adjusted risk ratio of PE according to weight increases from 1.0 in patients with BMI less than 25 kg/m² to 2.7 in patients with BMI more than 40 kg/m². The reported incidence of PE in patients undergoing bariatric surgery is between 1.4–2.6%. DVT prophylaxis should be initiated before the induction of anaesthesia. Unfractionated heparin; 5000 units subcutaneously, administered before surgery and then twice a day until the patient is fully mobile; has been shown to reduce the risk of DVT. Also, low molecular weight heparins, such as enoxaparin, have been used for thromboembolism prophylaxis. Additionally, appropriately sized compression stockings and use of pneumatic sequential compression devices intraoperatively and postoperatively helps in reducing risk of DVT.
- Drugs for medical management of obesity and those for various comorbidities must be optimised.

42) How will you prepare the operation theatre for this case?

- Equipment for patient positioning and transport



- Vein finder
- Standard monitoring equipment including appropriate size NIBP cuffs or arterial line and IBP and etco2 and temperature monitoring
- Difficult airway cart including an Airway Rescue technique kit
- HFNO or THRIVE for Para oxygenation
- BiPAP or CPAP devices
- Quantitative neuro muscular monitor
- Ultra sound machine with both linear and curvilinear probes
- DVT prophylaxis equipment - intermittent pneumatic compression stockings
- Warming devices- fluid warmer, Bair huggers.
- Emergency drugs including inotropes and vasopressors

43) What are the applications of POCUS in the perioperative management of this patient?

- Airway assessment and marking
- Airway blocks
- Nerve blocks for analgesia
- Gastric ultrasound to assess fasting status
- Intravenous access - peripheral or central
- Arterial line insertion
- Cardiac function assessment in the perioperative period
- Fluid status assessment
- Lung ultrasound intraoperatively and postoperatively for atelectasis
- Spine assessment and marking.

44) What are the problems expected in managing the airway in obese patients?

Many of these obese patients may have difficult airways especially difficulty in mask ventilation, difficulty in introducing & selecting a supraglottic airway, difficulty for laryngoscopy, difficulty for intubation & difficulty in front of neck access. These problems are exaggerated in patients with OSA.

45) What are your plans for the management of the airway?

All these patients are considered an anticipated difficult airway hence all the available modalities along with their risks and benefits should be discussed with both the patient and the attenders and informed consent should be taken.

Thorough preoxygenation with BiPAP or CPAP WITH 10 CM of H₂O for 3-5 minutes.

Apnoeic oxygenation with High Flow Nasal Cannula (HFNC) or at least 15L/min of low flow O₂ for all methods

Ramped up position to bring the sternal notch in line with the tragus.

Consider modified RSI if there is a risk of aspiration
Additional personnel, expert help, and equipment should be available on standby.

Plan A: Ramped up position with VDL / direct laryngoscopy with Macintosh blade with a stubby handle or McCoy blade

Plan B: Fibre optic bronchoscopy with USG-guided airway blocks

Plan C: SGA device insertion - intubating LMA

Plan D: Airway Rescue Technique
Topicalization of the mucosal surfaces prior to intubation with 10% lignocaine sprays can decrease pressor response.

Each of the above-mentioned plans can be used as the sole method or switched between in no particular order as the situation evolves.

46) What is HELP or Ramped position for laryngoscopy in obese patients?

Head elevated laryngoscopic position or ramped position is used with goal of aligning the external auditory meatus & the sternal notch in a horizontal plane. The head can be placed in semi-Fowler's position or a stack of blankets or a preformed or inflatable ramp can be used to achieve ramped position (Figures 4 and 5).

This position will lead to better oxygenation and better comfort to mask ventilate and there is a better view of the airway during direct laryngoscopy.

47) How preoxygenation & apnoeic oxygenation are done in obese patients?

Preoxygenation is needed to increase oxygen reserve to prevent hypoxemia during apnoea as there is increase in oxygen consumption in obese patients. Again, in these patients O₂ reserve is low due to decreased FRC leading them to rapidly desaturate &



thus may not be able to tolerate apnoea during laryngoscopy & intubation. They may also have an extended apnoea during laryngoscopy because of the difficult airway. There is always a possibility of landing into “cannot intubate, cannot oxygenate” situation because of the extreme difficult airway in some of these patients especially OSA patients.

Preoxygenation can be performed via a tight - fitting facemask at a flow rate of 10–12 L/min to prevent rebreathing aiming for an end tidal concentration of O₂ greater than 90% to maximise safe apnoea period.

Preoxygenation with manually applied CPAP or the use of non-invasive ventilation will improve oxygenation. The use of nasal cannula for passive apnoeic oxygenation during laryngoscopy can prolong the time to desaturation.

Heated humidified high flow nasal oxygenation like THRIVE can significantly delay apnoeic desaturation.



Figure 4 - Head elevated laryngoscopic position (HELP) or Ramped position

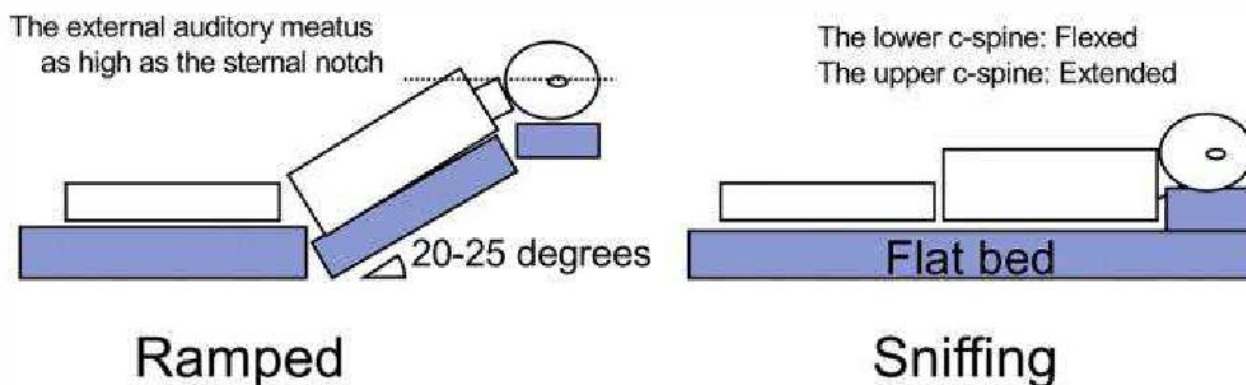


Figure 5 - Comparison between Ramped and Sniffing position

48) What technique of mask ventilation is performed in obese patients?

These patients have a difficulty to mask ventilate. Holding the mask with two hands in the V-E position with the operator's thumbs along the side of the mask and the first & second fingers behind the angle of mandible to perform jaw thrust may be more effective than the C-E position with the thumb & index finger on the mask & the fourth & fifth finger on the patient's mandible (Figure 6).

49) Should the video laryngoscope be the first choice for laryngoscopy & intubation in obese patients?

Since obese patients are considered to have difficult airway, a detailed airway assessment should be done. Compared to direct laryngoscopy the use of video laryngoscopy in patients undergoing bariatric surgery has shown to provide a better glottic view & lesser time required to successfully intubate the trachea.

50) What is check laryngoscopy?

Performance of an awake topicalised direct laryngoscopy with modest sedation can also be utilised to assist laryngoscopic view in deciding whether to proceed with induction of GA or awake sedated fibreoptic intubation.

If any part of larynx is visible with direct awake laryngoscopy, one can induce & get the airway.

An emergency airway cart that provides access to rescue intubating devices such as supraglottic devices, a flexible bronchoscope, a light wand & nebulisation drugs should be immediately available.

For very high-risk patients with extremely limited pulmonary reserve or abnormal airway anatomy, a surgeon with considerable experience in performing tracheostomy should be immediately available to perform emergency tracheostomy if needed.

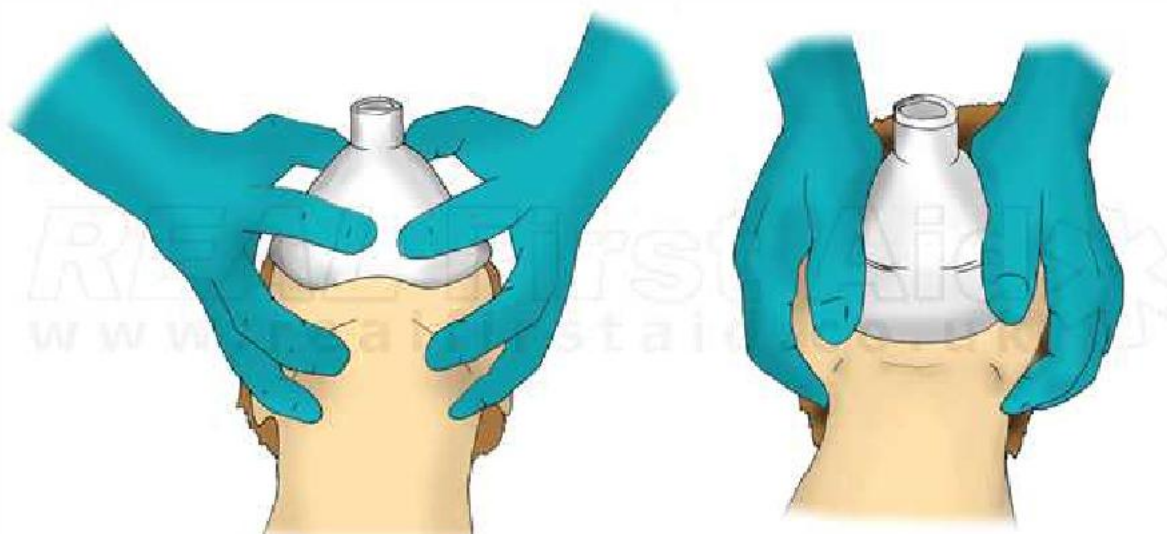


Figure 6– C E and VE technique of holding the mask with 2 hands

51) Should all obese patients be undergoing rapid sequence induction?

Only those obese patients with history suggestive of GERD, hiatal hernia, chronic diabetic mellitus or patients on GLP-1 agonists drugs require RSI.

In all other obese patients preoperative fasting guidelines should be followed- 6hrs for solids & 2 hours for clear fluids may be safe.

Acid aspiration prophylaxis should be used for all obese patients.

52) What premedication drugs would you like to administer?

- Avoid the use of respiratory depressant drugs before shifting the patient to the operation theatre.
- Judicious use of benzodiazepines like midazolam after starting supplemental oxygen

- Aspiration prophylaxis should be administered as these patients are considered full stomach irrespective of fasting durations.

- Antiemetic - Ondansetron 4mg
- Anti-sialagogue- Glycopyrrolate 0.2mg
- Prokinetic agent – Metoclopramide
- H2 receptor blocker / PPI

- Dexmedetomidine in titrated doses of 0.5-1 mcg/kg can be used as it provides anxiolytic, mild sedative, and analgesic effects without respiratory depression while preserving airway reflexes with hemodynamic stability hence reducing the requirements of opioids and benzodiazepines.

- Short-acting opioids such as Fentanyl 1-2mcg/kg LBW can be administered without significant risk of postoperative respiratory depression.

53) What drugs are used for induction & maintenance of these patients?

Anaesthetic induction agents are chosen based on patient factors rather than obesity - Propofol for patient with good left ventricular systolic function & etomidate for patient with poor LV function.

Dose of propofol & etomidate for induction is based on IBW. Succinylcholine is highly recommended in obese patients in which airway management is challenging or with high risk of peri-induction hypoxemia or aspiration due to its rapid onset & limited duration of action.

Dose of succinylcholine is based on actual body weight because of increase in activity of pseudocholinesterase.

Continuous infusion of propofol or any of the inhalational agents or a combination may be used to maintain anaesthesia.

Desflurane, sevoflurane & isoflurane are minimally metabolised & are therefore ideal agents, with Desflurane possibly providing faster washout. The use of Nitrous oxide is limited by high oxygen demand in obese patients.

54) What is your choice of muscle relaxant for intubation?

- Succinylcholine at a dose of 1-1.5mg/kg TBW is to be considered as the relaxant of choice since this subset is generally considered to be full stomach despite preoperative fasting due to delayed gastric emptying time and higher residual gastric volumes with lower pH.
- Succinylcholine has a short onset time of 60 seconds and hence can be used for RSI and is a depolarising agent, so it initially causes contraction followed by relaxation of skeletal muscles. Therefore, during the initial contraction period, it causes the cords to move apart facilitating intubation.
- Succinylcholine is metabolised by pseudocholinesterases in the plasma. The circulating concentration of these enzymes are increased in the morbidly obese relative to BMI hence a higher dose is required.
- Rocuronium is a viable alternative to succinylcholine as its onset time is around 90 seconds however it has a longer duration of action when administered at the intubating dose of 1.2mg/kg TBW.
- Hence in a difficult airway situation when the decision

to abort the procedure has been taken a rapid reversal dose of Sugammadex (16mg/kg TBW) needs to be available on hand if rocuronium is used.

- Other NDMRs have a longer onset period and are not advocated for use in this subset of patients unless it is confirmed by USG that there are no gastric contents.

55) Which muscle relaxants are used for maintenance?

Profound muscle relaxation is important during laparoscopic bariatric surgeries to facilitate ventilation & maintain adequate working space for visualisation and safe manipulation of laparoscopic instruments.

Vecuronium, Rocuronium can be used so that they can be reversed using sugammadex.

Doses of muscle relaxants can be administered based on neuromuscular monitoring. Obese patients are prone for residual neuromuscular palsy. By continuous NM monitoring and using sugammadex for reversal will prevent residual NM palsy and thus preventing many of the postoperative complications like hypoxia, hypercarbia and postoperative pulmonary complications.

56) What is the maximum intra-abdominal pressure for pneumoperitoneum during laparoscopic bariatric surgeries to be used?

Pneumoperitoneum should not be increased above 15 mmHg since intra-abdominal pressures of 20 mmHg or greater can cause caval compression and decrease cardiac output. Cephalad displacement of the diaphragm & carina by the pneumoperitoneum during laparoscopy can cause firmly secured endotracheal tube to displace into a main-stem bronchus & can be a cause for intraoperative hypoxia.

57) What are the effects of pneumoperitoneum creation and how will you counter them?

CVS:

- Sudden insufflation causes parasympathetic stimulation and the decreased venous return due to compression on the IVC and head-up position can cause an initial drop in cardiac output and blood pressure.
- This can be prevented by slow insufflation, fluid supplementation, avoidance of rapid changes of position, and bolus doses of vasoactive agents.
- Increased IAP causes increased sympathetic



stimulation leading to increased SVR and increased afterload and blood pressure.

- Hence beta-blockers such as labetalol or vasodilator agents such as NTG can be used
- CO₂ causes myocardial depression and sensitises the myocardium to the arrhythmogenic potential of catecholamines

RS:

- Cephalad displacement of the diaphragm, decreased FRC, decreased compliance, increased airway pressures, increased physiological dead space, and V/Q mismatch.
- These changes cause an increase in EtCO₂ that can be countered by employing ventilatory strategies.

Renal:

- Increased IAP can cause renal hypoperfusion. This in turn causes increased release of ADH, Renin, and Aldosterone, the result of which could be ATN

Hepatic:

- Increased IAP causes decreased portal venous flow leading to hepatic hypoperfusion which can result in acute hepatocellular dysfunction evidenced by increased transaminase levels.

CNS:

- Hypercarbia causes cerebral vasodilation. Raised intrathoracic pressures translate to elevated CSF pressures and consequently raised ICP.
- Hypoxia and decreased cerebral oxygen tension can trigger catecholamine and vasopressin release.

Coagulation:

- Venous stasis due to venous pooling caused by compression over the IVC by the pneumoperitoneum.
- Hypercoagulable state due to the chronic Pro-inflammatory state in the morbidly obese.
- The venous stasis can be countered by using intermittent pneumatic compression stockings.

Most of the complications can be avoided by limiting insufflation pressures to less than 15mmHg and judicious fluid therapy.

58) What intraoperative ventilatory strategies adopted to prevent atelectasis & hypoxia?

- Set tidal volume of 6–8 ml/kg of predicted body weight. Limit FiO₂ to the level required to maintain

peripheral O₂ saturation (SpO₂) > 92% (ideally FiO₂ below 0.5-0.8) to prevent resorption atelectasis and oxygen toxicity.

- Employ individualized PEEP to optimize plateau and driving pressure as well as lung compliance so as to follow open lung concept.

- Use lung recruitment maneuvers judiciously during anaesthesia (ideally delivered by stepwise increase/decrease of PEEP & VT respectively to a plateau pressure > 40 to < 55 cm H₂O) to improve oxygenation & plateau pressure as needed.

- Adjust respiratory rate to maintain normocarbia (permissive hypercapnoea is acceptable in patients without pulmonary hypertension).

- Maintain head up position whenever possible.

- Recruitment maneuver should not be performed unless patients are haemodynamically stable and euvolemic, as they may lead to transient decrease in preload and hypotension.

- PEEP up to 15cmH₂O is effective in maintaining FRC and improving oxygenation during laparoscopic surgeries in obese patients.

59) How intraoperative fluid management is done?

Fluid requirements for the obese patient are larger than predicted for those of normal BMI and even during relatively short 2-3 hours case. These patients may need 3–4 L of crystalloid fluid to prevent acute tubular necrosis in the kidneys. Normovolemia should be the rule in order to avoid increased haemodynamic instability, postoperative nausea & vomiting & HTN.

Hypervolemia to be avoided as many of these patients have diastolic dysfunction. If possible, fluid therapy can be guided by dynamic parameters which will help in goal directed fluid therapy (GDFT).

60) How intra operative analgesia is maintained?

Remifentanyl and Fentanyl carefully titrated to clinical effect are the most common choices. Dexmedetomidine an alpha 2 agonist with sedative and analgesic properties is an attractive adjunct in obese patients as it doesn't have any respiratory depressant effect, but decreased post op opioid



requirements. For open surgery, epidural analgesia and other regional or fascial blocks can also be used for both intra and post op analgesia.

61) How these patients are extubated after surgery?

Bariatric patients are generally extubated in the operating room. The extubation criteria for patients with obesity are:

- ♦ Intact neurologic status, fully awake & alert with head lift more than 5 sec
- ♦ Haemodynamic stability
- ♦ Normothermia greater than 36°C
- ♦ Full reversal of NM blockers confirmed with TOF ratio > 0.9
- ♦ Respiratory rate > 10 and < 30 breaths per minute
- ♦ SpO₂ > 95% on FiO₂ of 0.4
- ♦ Acceptable blood gas values
- ♦ Acceptable pain control
- ♦ Acceptable respiratory mechanics with maximum inspiratory pressure > 30cmH₂O, vital capacity > 10ml/kg of IBW
- ♦ No surgical complications noted

Patients with preop CPAP/BiPAP should return to their therapy in PACU.

62) How postoperative analgesia is managed?

Because opioid-induced respiratory depression is a concern, a multimodal approach to postoperative pain control is employed. Local anaesthetic wound infiltration or ultrasonographic (USG) guided transverse abdominal plane (TAP) blocks along with supplementation with IV paracetamol, nonsteroidal anti-inflammatory drugs (NSAIDs) & NMDA receptor antagonists like ketamine can be employed as a part of multimodal analgesia.

63) What are your post-operative concerns in a morbidly obese patient?

- Postoperative pulmonary complications - hypoventilation and hypoxemia secondary to atelectasis and diaphragmatic splinting, hypopnoea - apnoea spells
- Need for postoperative ventilatory support in the form of CPAP and BiPAP
- Post Operative Pain - can be reduced by lower IAP, shorter duration of surgery, and evacuation of subdiaphragmatic CO₂ prior to wound closure.
- Patients with active reflux may be at a higher risk of postoperative aspiration and exacerbation of reactive airway diseases

- Venous Thromboembolism may occur due to activation of coagulation cascade and obstruction to venous outflow during pneumoperitoneum. Obesity itself is a hypercoagulable state
- Postoperative nausea and vomiting- laparoscopic surgery is associated with a higher risk of PONV.

Surgical site infection rates and incidence of pneumonia are higher due to their immunocompromised condition

64) What measures to be taken during the postoperative care of these patients?

Issues specific to the patient with obesity in the post anaesthesia care unit (PACU) are largely respiratory & ventilatory.

Respiratory monitoring – patients should have continuous pulse oximetry in PACU until they have demonstrated that they can maintain adequate oxygenation when left unstimulated.

Arterial blood gases is the best assessment for suspected hypoventilation. High level of suspicion for hypoventilation should be maintained in patients who remain sedated or become hypoxic despite administration of O₂. Hypoventilation due to sedative medication should be ruled out: pharmacological reversal of benzodiazepines or opioids may be used as clinically indicated. When upper airway obstruction occurs, oropharyngeal or nasopharyngeal airway maybe used. When these maneuvers are insufficient, it is reasonable to assist with noninvasive ventilation, which may keep them from requiring re intubation.

- ♦ Supplemental oxygen by face mask or nasal cannula titrated to keep SpO₂ > 90%.
- ♦ Positioning of patient in head up or lateral position
- ♦ Use incentive spirometry or chest physiotherapy
- ♦ These patients are kept on their CPAP or BiPAP machines as much as possible with monitoring of end tidal CO₂ and SpO₂ by pulse oximetry. Monitoring capnometry in the PACU is critical to diagnose hypoventilation, since the administration of supplemental O₂ can delay the diagnosis of hypoventilation.

65) What is obesity paradox?

The phenomenon where patients who are overweight or have type I obesity (BMI 30–34.9 kg/m²) may



experience lower mortality or fewer complications during surgery compared to normal weight or underweight counterparts is known as obesity paradox.

Factors contributing to obesity paradox:

- a. Nutritional reserve - overweight or mildly obese patients may have greater metabolic reserves to handle stress of major surgeries
- b. Inflammatory pre-conditioning - chronic low-grade inflammation associated with obesity might prime the immune system, leading to a more controlled response to surgical trauma
- c. Early intervention - Obese patients may be screened more rigorously for CVS issues prior to surgery as they are perceived as higher risk, leading to a better

management of comorbidities before they reach the OT.

Additional Reading –

1. Malhotra G, Eckman DM; Anesthesia for bariatric surgeries, Chapter 54, Miller's anesthesia, 10th edition, editor- Gropper M, Elsevier 2025
2. Bustamante AF, Bucklin BA; Anesthesia and Obesity, Chapter 45, Barash, Cullen and Stoelting's Clinical Anesthesia, 9th edition, 2024, Wolters Kluwer
3. Eipe N, Anesthesia for the patient with obesity, UpToDate 2025
4. Sun Moon T, Jones SB, Nutritional Diseases: Obesity and Malnutrition, Chapter 19, Stoelting's Anesthesia and Coexisting Diseases, 2022, Elsevi



14th JUNE 2026
12:00Noon - 12:30PM

ANESTHETIC CONSIDERATIONS FOR A 3 MONTH OLD INFANT POSTED FOR CLEFT LIP/PALATE REPAIR

BRIEF SUMMARY

1. Embryology, Anatomy & Types

Cleft lip results from failure of fusion of the maxillary and medial nasal processes during the 4th–7th week of gestation, while cleft palate occurs due to failure of palatal shelf elevation and fusion between the 7th–12th weeks. Anatomically, clefts are divided by the incisive foramen into prepalatal defects involving the lip, alveolus, and anterior palate, and postpalatal defects involving the hard and soft palate. They may be unilateral or bilateral, complete or incomplete, with left-sided clefts being the most common. Submucosal clefts represent bony defects with intact mucosa.

2. Incidence & Associations

The incidence is approximately 1 in 1000 live births, with cleft lip more common in males and isolated cleft palate more common in females. There is a strong association with congenital anomalies, especially congenital heart disease, making preoperative cardiac evaluation important. Syndromes such as Pierre Robin sequence and Treacher Collins syndrome are particularly important due to their airway implications.

3. Pathophysiology

In infancy, the inability to generate negative pressure results in feeding difficulty, regurgitation, and aspiration risk. In older children, velopharyngeal dysfunction leads to hypernasal speech, while Eustachian tube dysfunction results in recurrent otitis media and conductive hearing loss. Dental abnormalities and facial growth disturbances are common, along with psychosocial impact.

4. Key Syndromes Affecting Anesthesia

Pierre Robin sequence presents with micrognathia, glossoptosis, and airway obstruction, often causing dynamic airway collapse, particularly during sleep or sedation. Treacher Collins syndrome involves mandibular hypoplasia, facial bone abnormalities, and a significantly difficult airway. These conditions are critical as they influence airway planning and perioperative risk.

5. Timing & Goals of Surgery

Cleft lip repair is usually performed at 3–6 months of age (guided loosely by the “rule of 10”), while cleft palate repair is performed between 6–18 months. The goals include restoration of feeding, speech development, hearing, facial growth, and cosmesis. Early repair improves speech outcomes, while delayed repair may preserve facial growth.

6. Preoperative Evaluation

Preoperative assessment focuses on feeding ability, nutritional status, hydration, and history of regurgitation or aspiration. Symptoms of airway obstruction such as snoring or apnea must be elicited. Upper respiratory tract infections are common and significantly increase perioperative airway complications; elective surgery should be postponed if active infection is present.

Examination must include assessment of anemia, hydration, and syndromic features. Cardiovascular examination is essential to rule out congenital heart disease. Respiratory examination looks for wheeze or infection. Airway assessment is crucial, identifying micrognathia, glossoptosis, and the type of cleft, all of which predict difficulty in ventilation and intubation.

7. Investigations

Hemoglobin estimation is essential as anemia is common in infants. Echocardiography is indicated if a murmur or cardiac anomaly is suspected. Additional tests such as chest X-ray or electrolytes are guided by clinical findings.

8. Fasting & Premedication

Fasting guidelines recommend 2 hours for clear fluids, 4 hours for breast milk, and 6 hours for formula feeds. Premedication must be used cautiously due to airway concerns. Anticholinergics reduce secretions, paracetamol provides baseline analgesia, and dexamethasone reduces airway edema and postoperative nausea. Sedatives like midazolam are often avoided in infants at risk of airway obstruction.

9. Operating Room Preparation

Cleft surgery involves a shared airway, so the operating table is rotated 90–180 degrees away from the



anesthesia machine. A preformed oral RAE tube is preferred as it allows midline fixation and better surgical access. Careful positioning is essential to avoid endobronchial intubation. A difficult airway cart must be ready, including video laryngoscopy, bougie, fiberoptic scope, and supraglottic airway devices. Warming measures are essential to prevent hypothermia.

10. Airway & Intraoperative Challenges

Airway management is the most critical issue. Mask ventilation may be difficult due to poor seal from the cleft. Laryngoscopy can be challenging, particularly in wide clefts where the blade may slip into the defect; packing the cleft or using alternative blade techniques may help. Maintaining spontaneous ventilation during induction is often preferred in anticipated difficult airway.

The airway is shared with the surgeon, summarized by the “BADD” concept:

- Blood
- Access limitation
- Debris
- Dislodgement of tube

The Dingman mouth gag and surgical manipulation can cause tube compression, kinking, or displacement. Continuous monitoring of ventilation and capnography is essential.

11. Induction & Maintenance

Induction is commonly performed using sevoflurane in oxygen, maintaining spontaneous ventilation until the airway is secured. Intravenous access is obtained after adequate depth. Laryngoscopy should only be attempted when sufficiently deep to avoid laryngospasm.

Maintenance may be with inhalational agents or total intravenous anesthesia. Controlled ventilation is often used, aided by non-depolarizing muscle relaxants such as atracurium, which improve surgical conditions and reduce bleeding. Opioids like fentanyl or remifentanyl may be used judiciously to provide analgesia and facilitate smooth emergence.

12. Intraoperative Hazards

Major hazards include airway obstruction, accidental extubation, endobronchial intubation, hypoxia,

hypercarbia, and laryngospasm. Blood and secretions may enter the airway, increasing the risk of aspiration. Hypothermia is common due to large surface area and must be actively prevented. Although blood loss is usually minimal, even small losses are significant in infants.

13. Fluid Therapy

Fluid management is guided by the Holliday–Segar formula (4 mL/kg/hr for first 10 kg), along with replacement of fasting deficits and intraoperative losses. Glucose-containing fluids may be required due to limited glycogen stores in infants.

14. Analgesia

A multimodal approach is preferred. Regional techniques are highly effective, including infraorbital nerve blocks for cleft lip and palatine nerve blocks for cleft palate. Systemic analgesics include paracetamol, NSAIDs, and cautious opioid use. Adjuncts such as dexmedetomidine may reduce opioid requirement and emergence agitation.

15. Emergence & Extubation

Extubation is a critical phase due to risk of airway obstruction. The airway must be carefully suctioned under direct vision to remove blood and debris without disrupting surgical repair. Extubation may be performed awake or deep depending on risk assessment, but full reversal of neuromuscular blockade is essential. Oxygen supplementation and close observation are mandatory.

16. Postoperative Management

The primary concern is airway obstruction, which may occur immediately or within 48 hours postoperatively. Infants are positioned laterally or prone to allow drainage of secretions. Continuous monitoring in PACU is required. Arm restraints may be used to prevent disruption of surgical repair.

17. Postoperative Complications

The most important complication is airway obstruction due to edema, tongue fall, residual anesthetics, or blood clots. Bleeding can compromise the airway and lead to aspiration. Laryngospasm, hypoxia, and respiratory infections are common. Hypothermia delays recovery. Long-term complications include velopharyngeal insufficiency and speech defects.



COMMON VIVA QUESTIONS

A. Anatomy and physiology

1. Describe the anatomic concept of a cleft lip and cleft palate.

A cleft lip results from the failure of fusion between the maxillary process and the medial nasal process during early embryological development. This defect can vary from a small notch in the upper lip to a complete cleft extending through the lip, alveolus, and floor of the nose. In more severe cases, it may be associated with a cleft palate.

A cleft palate occurs due to failure of fusion of the palatal shelves. Anatomically, clefts are divided based on the incisive foramen into prepalatal and postpalatal defects. Prepalatal clefts involve the lip, alveolus, and anterior palate, whereas postpalatal clefts involve the hard and soft palate. A submucosal cleft represents a defect in the underlying bone with intact mucosa. The most common presentation is a unilateral complete cleft, often on the left side.

2. What is the cause of a cleft lip or cleft palate?

The development of the face occurs between the fourth and seventh weeks of gestation. A cleft lip arises when there is failure of fusion of the mesodermal elements forming the upper lip. A cleft palate develops when the palatal shelves fail to elevate, migrate medially, and fuse. The etiology is multifactorial, involving genetic predisposition as well as environmental influences such as maternal illness, nutritional deficiencies, or teratogenic exposure.

3. What is the incidence of cleft lip and cleft palate?

Cleft lip with or without cleft palate occurs in approximately 1 in 1,000 live births, with higher incidence observed in Asian populations and lower incidence in African populations. Cleft lip is more common in males, whereas isolated cleft palate is more frequently seen in females. A significant proportion of cases are isolated, but a notable percentage is associated with other congenital anomalies or syndromes.

4. Discuss the pathophysiology in neonates and older children.

In the neonate, the presence of a cleft palate interferes with the ability to generate negative pressure required

for effective sucking, leading to feeding difficulties and risk of aspiration. The abnormal communication between the oral and nasal cavities results in regurgitation of feeds and predisposes to recurrent infections.

In older children, the consequences extend to impaired speech due to velopharyngeal dysfunction, leading to hypernasal speech and inability to produce certain consonants. Dysfunction of the Eustachian tube results in recurrent middle ear infections and conductive hearing loss. Additionally, abnormalities in dental development and facial growth may occur. Psychosocial issues also become significant as the child grows.

5. What other conditions are associated with cleft lip and palate?

Cleft palate is frequently associated with other congenital anomalies and syndromes. Common syndromic associations include Pierre Robin sequence, Treacher Collins syndrome, Stickler syndrome, and velocardiofacial syndrome. Non-syndromic anomalies may include limb deformities, ear abnormalities, and umbilical hernias. Congenital heart disease is also commonly associated and has important anesthetic implications.

6. What is Pierre Robin sequence?

Pierre Robin sequence is characterized by a triad of micrognathia, glossoptosis, and airway obstruction. The small mandible causes posterior displacement of the tongue, leading to obstruction of the airway, particularly during sleep or relaxation. This condition is often associated with cleft palate and results in significant feeding and respiratory difficulties in infancy.

7. What is Treacher Collins syndrome?

Treacher Collins syndrome is a craniofacial disorder characterized by hypoplasia of the zygomatic bones and mandible, microtia with associated hearing loss, a high-arched or cleft palate, and characteristic facial features such as downward slanting eyes. From an anesthetic perspective, it is particularly important because it is associated with a difficult airway due to mandibular hypoplasia and restricted oral access.

8. What kind of specialized team care is required?

Management of patients with cleft lip and palate



requires a multidisciplinary approach. This includes a pediatrician for overall health, a plastic surgeon for corrective procedures, and an anesthesiologist for perioperative care. In addition, speech therapists, orthodontists, geneticists, psychologists, and social workers play important roles in addressing speech development, dental alignment, genetic counseling, and psychosocial support.

9. Discuss the indications and timing of surgery.

The timing of surgical repair is planned to balance anesthetic safety with optimal functional outcomes. Cleft lip repair is typically performed between 3 and 6 months of age, often guided by the “rule of 10” (10 weeks of age, 10 pounds weight, and hemoglobin of 10 g/dL). Cleft palate repair is usually performed between 9 and 12 months of age to facilitate normal speech development while minimizing interference with facial growth. Early repair improves feeding, speech, hearing, and psychosocial development.

10. What is velopharyngeal incompetence and how is it diagnosed?

Velopharyngeal incompetence refers to the inability of the soft palate to adequately close against the posterior pharyngeal wall during speech. This results in hypernasal speech and air escape through the nose. Diagnosis is primarily clinical, based on speech assessment, but may be confirmed using nasopharyngoscopy or videofluoroscopy to visualize palatal movement.

11. What is the relation of tonsillectomy and adenoidectomy to velopharyngeal incompetence?

Tonsillectomy and adenoidectomy can influence velopharyngeal function. In patients with borderline palatal competence, removal of adenoids may worsen velopharyngeal incompetence. However, in patients requiring pharyngoplasty, removal of tonsillar or adenoidal tissue beforehand may be necessary to optimize surgical outcomes and prevent airway obstruction.

B. Preoperative Evaluation and Preparation

1. What are the preanesthetic considerations in a 3 month old infant posted for cleft lip and palate surgery?

A thorough preoperative evaluation includes a detailed history focusing on feeding difficulties, respiratory

symptoms such as snoring or apnea, and recent upper respiratory tract infections. Physical examination should assess vital signs, nutritional status, hydration, and the presence of anemia or cyanosis. Particular attention should be given to airway assessment, including mouth opening, mandibular size, and tongue position, as these patients often present with difficult airway anatomy. The possibility of associated congenital anomalies, especially cardiac defects, must also be evaluated.

4. What are the important findings in respiratory, cardiovascular, and CNS examination?

Respiratory examination should look for signs of infection such as wheeze or crepitations, as active upper respiratory infection increases the risk of perioperative complications like laryngospasm and bronchospasm. Cardiovascular examination is essential to detect murmurs, which may indicate underlying congenital heart disease requiring further evaluation. Central nervous system examination helps identify developmental delay or syndromic features that may influence anesthetic management and airway planning.

3. What are the preoperative investigations are required?

Baseline investigations include a complete blood count to assess hemoglobin levels and detect infection or thrombocytopenia. Echocardiography is indicated if a cardiac murmur or cyanosis and/or syndromic features are present. Chest radiography may be required in the presence of respiratory symptoms. Additional investigations such as renal function tests and electrolytes may be necessary in cases of dehydration or associated comorbidities.

4. What are the other preoperative considerations?

Other important considerations include optimization of nutritional status, as feeding difficulties are common. The risk of aspiration is increased due to regurgitation of feeds, necessitating strict adherence to fasting guidelines. Hypothermia is a concern in infants due to their large surface area and poor thermoregulation. A difficult airway should always be anticipated, and appropriate equipment must be prepared.

5. How will you perform and interpret airway assessment in this infant?



Airway assessment in a 3-month-old infant with cleft lip and palate begins with careful inspection of anatomical features that may predict difficulty in mask ventilation and intubation. Mouth opening is assessed, although cooperation is limited in infants, and the type and extent of the cleft are noted, as they can interfere with mask seal and laryngoscope blade placement. Particular attention is given to the presence of micrognathia or retrognathia, which displaces the airway anteriorly and makes laryngoscopy difficult. Glossoptosis, commonly seen in conditions such as Pierre Robin sequence, indicates a tendency for the tongue to fall backward and obstruct the airway, especially during sedation or after extubation. The overall assessment should lead to anticipation of a difficult airway, prompting preparation of adjuncts such as an oropharyngeal airway, video laryngoscope, and fiberoptic equipment, along with a well-defined backup plan.

6. What are the fasting guidelines?

Current fasting guidelines recommend a minimum fasting period of 2 hours for clear fluids, 4 hours for breast milk, and 6 hours for formula or solids. These guidelines aim to reduce the risk of aspiration while minimizing dehydration and hypoglycemia.

7. What premedications are given?

Premedication in this infant is selected carefully, balancing anxiolysis with the risk of airway obstruction. Midazolam may be used in a dose of approximately 0.3–0.5 mg/kg orally or 0.05–0.1 mg/kg intravenously for anxiolysis, although it is often avoided in very young infants or those with airway compromise. Anticholinergic agents such as glycopyrrolate (0.004 mg/kg) or atropine (0.01–0.02 mg/kg) are administered to reduce secretions and prevent vagal bradycardia. Paracetamol at 10–15 mg/kg is given for baseline analgesia. Dexamethasone, in a dose of 0.5–1 mg/kg, is administered to reduce airway edema and postoperative nausea. Aspiration prophylaxis may include ranitidine and metoclopramide, especially in patients with significant regurgitation risk.

C. Intraoperative Management

1. What monitors are required for cleft lip and palate surgery?

Standard monitoring is essential and includes electrocardiography, noninvasive blood pressure,

pulse oximetry, end-tidal carbon dioxide monitoring, and temperature monitoring. Among these, continuous capnography is particularly important because the anesthesiologist has limited access to the airway once surgery begins. A precordial stethoscope is extremely useful in infants as it provides continuous auditory monitoring of both ventilation and heart sounds. Temperature monitoring is critical due to the high risk of hypothermia in infants, and active warming measures must be employed throughout the procedure.

2. How will you prepare the operating room for cleft lip/palate surgery in this infant?

Operating room preparation is crucial due to the shared airway and limited access during surgery. The operating table is arranged such that the patient's head is turned 90 to 180 degrees away from the anesthesia workstation to allow the surgeon unobstructed access. A preformed oral RAE endotracheal tube is preferred, as it can be secured in the midline over the chin and does not interfere with the surgical field. Careful selection of tube length is important to avoid endobronchial intubation or accidental extubation. A difficult airway cart, including video laryngoscope, bougie, and fiberoptic bronchoscope, must be readily available. Equipment for managing complications such as tube kinking or dislodgement should be prepared. Special attention is given to warming devices to prevent hypothermia and suction systems to clear blood and secretions effectively.

3. Discuss the induction and anesthetic management.

Anesthesia is usually induced using an inhalational technique with sevoflurane in oxygen, as this allows a smooth and controlled induction while maintaining spontaneous ventilation until the airway is secured. Intravenous access is obtained once an adequate depth of anesthesia is achieved. Laryngoscopy should not be attempted until a deep plane of anesthesia is reached to avoid laryngospasm.

Endotracheal intubation is performed orally, typically using a preformed oral RAE tube, which allows the tube to be secured in the midline and provides optimal surgical access. In patients with anticipated difficult airway, muscle relaxants should be withheld until the airway is secured. Anesthesia is induced using an inhalational technique with sevoflurane in oxygen, which allows a smooth induction while maintaining



spontaneous ventilation until the airway is secured. Intravenous access is established after achieving an adequate depth of anesthesia. Laryngoscopy is performed only when the patient is sufficiently deep to minimize the risk of laryngospasm. The trachea is intubated using an oral RAE tube, secured in the midline.

Maintenance of anesthesia is achieved using inhalational agents with controlled ventilation. Muscle relaxation is provided to facilitate intubation and improve surgical conditions, typically using a non-depolarizing agent such as atracurium in a dose of approximately 0.5 mg/kg. Controlled ventilation helps maintain normocapnia, reduces bleeding, and allows a lighter plane of anesthesia with faster recovery. Careful monitoring and coordination with the surgical team are essential throughout the procedure due to the shared airway.

4. What airway related intraoperative complications can occur?

Intraoperatively, several airway challenges are anticipated due to anatomical abnormalities and surgical factors. Mask ventilation may be difficult because of poor seal caused by the cleft defect. Intubation can be challenging in the presence of micrognathia or glossoptosis, which alter airway alignment. Once intubated, the airway remains at risk due to its shared nature with the surgeon. The use of mouth gags such as the Dingman retractor can lead to kinking, compression, or displacement of the endotracheal tube. There is also a risk of accidental extubation during surgical manipulation. Additionally, blood and secretions in the oropharynx can provoke laryngospasm and airway obstruction. Continuous vigilance and repeated reassessment of tube position are therefore essential.

5. What intraoperative hazards can occur during cleft lip and palate surgery, and how will you manage them?

Intraoperative hazards include airway obstruction due to tube kinking, compression, or displacement, as well as accidental extubation, which may first be noticed by the surgeon or by the disappearance of the end-tidal CO₂ trace. Immediate management involves informing the surgical team, administering 100% oxygen, suctioning the airway, and preparing for reintubation, which may be difficult and require a smaller tube.

Hypoxia and hypercarbia may result from inadequate ventilation and must be promptly corrected. Blood loss, although usually small, can be significant in infants and requires close monitoring and replacement. Hypothermia is another major concern due to the infant's large surface area and must be prevented using warming devices. Laryngospasm may occur due to airway irritation and is managed with oxygen, positive pressure ventilation, deepening anesthesia, or administration of muscle relaxants if severe.

6. What is laryngospasm and how is it diagnosed?

Laryngospasm is a reflex closure of the vocal cords due to stimulation of the larynx. It is more common in pediatric patients, especially in the presence of airway irritation such as blood or secretions. Partial laryngospasm presents with stridor and increased respiratory effort, while complete laryngospasm presents as silent chest with absence of air movement despite respiratory effort.

7. How is laryngospasm managed?

Management begins with immediate administration of 100% oxygen and removal of the triggering stimulus. Gentle continuous positive airway pressure may help relieve partial laryngospasm. Deepening the level of anesthesia with agents such as propofol can be effective. In cases of complete laryngospasm or failure of initial measures, a rapid-acting muscle relaxant such as succinylcholine should be administered to relieve the spasm and allow ventilation.

8. How will you manage intraoperative fluid therapy? Explain using the Holliday–Segar (4:2:1) formula.

Fluid therapy in this infant is guided by the Holliday–Segar formula for maintenance requirements. For a child weighing up to 10 kg, fluids are administered at 4 mL/kg/hour. In addition to maintenance, preoperative fasting deficits are calculated and replaced, typically over the first few hours of surgery. Ongoing losses, including blood loss and third-space losses, are also accounted for. Careful monitoring is essential, as infants have limited physiological reserves and are prone to both dehydration and fluid overload. Balanced isotonic fluids are generally used, and blood transfusion is considered if blood loss becomes significant.

D. Extubation Strategy

1. What is your extubation plan in this patient?



Extubation should be performed only when the child is fully awake with intact airway reflexes and adequate spontaneous ventilation. Before extubation, the oropharynx should be carefully suctioned under direct vision to remove blood and secretions, taking care not to disrupt surgical sutures. The presence of airway edema, particularly involving the tongue or soft tissues, should be assessed, and extubation should be delayed if significant swelling is present.

The child should be positioned in a lateral or semi-prone position to facilitate drainage of secretions and reduce the risk of aspiration. Awake extubation is preferred as it reduces the risk of airway obstruction and laryngospasm.

E. Postoperative Management

1. What are the important postoperative considerations?

Postoperative care focuses primarily on maintaining airway patency and monitoring for complications. Continuous monitoring of oxygen saturation is essential. The child should be positioned laterally or prone to allow drainage of secretions. Supplemental oxygen is often required. Adequate hydration and early feeding, as permitted, are important for recovery.

2. What postoperative complications may occur?

The most significant postoperative complication is airway obstruction, which may result from edema, posterior displacement of the tongue, or residual effects of anesthesia. Bleeding from the surgical site can also compromise the airway. Respiratory

complications such as laryngospasm and bronchospasm may occur, particularly in children with recent respiratory infections.

Other complications include hypothermia, infection, and, in the long term, velopharyngeal insufficiency, which may require further surgical correction.

3. How is postoperative pain managed?

Pain management should be multimodal. Regional techniques such as infraorbital nerve block for cleft lip repair and palatine nerve blocks for cleft palate repair provide effective analgesia and reduce the need for systemic opioids. Non-opioid analgesics such as paracetamol and ibuprofen are commonly used. Opioids may be administered in small, titrated doses, but caution is required due to the risk of respiratory depression and airway obstruction.

RECENT UPDATES

Recent advances in cleft lip and palate anesthesia care include a shift toward opioid-sparing techniques using multimodal analgesia and dexmedetomidine, increased use of regional nerve blocks such as suprazygomatic maxillary block, adoption of video laryngoscopy for difficult airway management, emphasis on maintaining spontaneous ventilation during induction, and improved extubation strategies. Postoperative care now focuses on prolonged airway monitoring due to risk of obstruction. Additionally, total intravenous anesthesia and enhanced recovery protocols are increasingly being utilized.



14th JUNE 2026
07:30AM - 08:30AM

“THE PREGNANT HEART IN PERIL: ANAESTHETIC MASTERY OF PERIPARTUM CARDIOMYOPATHY COMPLICATED BY ACUTE APPENDICITIS”

Dr Sumesh T Rao

Professor & Head, Department of Anesthesiology,
Kasturba Medical College – Mangalore (MAHE)

Key Themes:

- Cardio-obstetric emergencies – bridging obstetric, cardiac and general anaesthesia
- Molecular pathophysiology of PPCM (the “Two-Hit Hypothesis”) as a springboard for rational therapy
- High-stakes intraoperative haemodynamic management in a patient with reduced left ventricular reserve
- Contraindicated drugs in pregnancy (ACEi, ARB, ARNI, spironolactone) – a board favourite and a life-saving distinction
- Latest 2025 updates: ESC mWHO 2.0, bromocriptine evidence, MCS outcomes and the “TUBE” approach
- Socratic Q&A that replicates the pressure and reasoning of postgraduate case discussions

WHY YOU CANNOT MISS THIS SESSION

“Twenty weeks pregnant. History of peripartum cardiomyopathy. Now an emergency appendicectomy. Three high-risk conditions colliding in one operating theatre.”

This half-hour session is not a theoretical review. It is a high-fidelity, case-based masterclass designed for the anaesthesiologist who may be called to the emergency room or obstetrics ward at 3 am. You will leave with a clear, actionable mental framework – one that will serve you in the board examination room and, more importantly, at the patient's bedside.

WHAT YOU WILL TAKE HOME

By the end of this presentation, you will be able to:

- **Confidently stratify** a pregnant patient with prior PPCM using the modified WHO 2.0 classification,

distinguishing between “full recovery” (LVEF > 50%) and “residual impairment” (LVEF < 50%) – a distinction that changes management from “high-risk but possible” to “pregnancy absolutely contraindicated.”

- **Explain the Two-Hit Hypothesis** to a trainee or a colleague, and link it directly to the rationale for bromocriptine therapy – including the mandatory pairing with therapeutic anticoagulation.

- **Recognise the three families of heart-failure drugs that are strictly contraindicated in pregnancy** and articulate the specific foetal risks for each, a topic that recurs in every major anaesthesia and cardiology board examination.

- **Design a tailored anaesthetic plan** that weighs laparoscopic versus open surgery, chooses the safest induction agent (etomidate, not ketamine), and selects a neuraxial technique that avoids catastrophic sympathectomy.

- **Apply recent 2024-2025 evidence** - from the EORP PPCM Registry to the PPCM-Cardiogenic Shock multicentre cohort – to guide bromocriptine use, mechanical circulatory support (IABP, VA-ECMO), and perioperative monitoring.

- **Handle a Socratic Q&A session** that mimics the intense, interactive case discussions we run with our own postgraduate residents. You will be challenged, but you will also learn how to think under pressure.

PART I – LITERATURE COMPILATION: WHAT I READ BEFORE DESIGNING THIS TALK

“A conference talk is only as strong as the evidence behind it.”

Below is a curated list of the textbooks, landmark papers, and recent guidelines that shaped every slide. I encourage you to read or skim them before the session - the Q&A will directly reference key findings.



Core Textbooks & Permanent References

- **Rodriguez Ziccardi, M., & Siddique, M. S. (2022).** Peripartum cardiomyopathy. In StatPearls [Internet]. – The most accessible, evidence-based entry point.
- **Chapman, K., Njue, F., & Rucklidge, M. (2023).** Anaesthesia and peripartum cardiomyopathy. BJA Education, 23(12), 464–472. – A concise, clinically oriented review written by anaesthesiologists for anaesthesiologists.
- **Meng, M.-L., et al. (2023).** Anesthetic care of the pregnant patient with cardiovascular disease: A scientific statement from the American Heart Association. Circulation, 147, e657–e673. – The definitive AHA guidance, updated for 2023.
- **ESC Guidelines on Cardiovascular Disease in Pregnancy (2018 with 2025 updates).** – The European Society of Cardiology framework that introduced the mWHO classification and bromocriptine recommendations.

Landmark Clinical Studies (2019–2025)

- **Jackson, A. M., et al. (2023).** A 20-year population study of peripartum cardiomyopathy. European Heart Journal, 44(48), 5128–5141. – Large-scale epidemiology and long-term outcomes.
- **EORP PPCM Registry (2025).** Bromocriptine use in 552 patients – the first large, real-world evidence showing reduced adverse maternal outcomes (OR 0.47) with no excess thromboembolism when anticoagulation is used.
- **Florea et al. (2024).** Mechanical circulatory support in PPCM. Annals of Thoracic Surgery Short Reports. – 28 patients, 89% survival to discharge.
- **PPCM-Cardiogenic Shock multicentre cohort (856 patients, 2025).** – 180-day mortality 17.9%; ECMO in 11.3%; transplantation in 8.1%.
- **Choudhary, J., et al. (2025).** Approach to perioperative management of peripartum cardiomyopathy with acute heart failure: A case report. JCA Advances, 2, 100121. – Introduces the “TUBE” approach (TEE, Ultrasound TAP block, Bolus

phenylephrine, Early MDT).

Pregnancy & Appendicectomy Specifics

- **Tsang, Y.-P., Tsui, D. K. K., & Yau, K. K. K. (2012).** Laparoscopic appendicectomy in a postpartum woman. Surgical Practice, 16(4), 170–171. – Still cited for the haemodynamic effects of pneumoperitoneum in pregnancy.
- **Raj, R., Kumar, M., & Batra, M. (2014).** Anaesthetic management of a case of dilated cardiomyopathy for emergency appendectomy. Anesthesia: Essays and Researches, 8(1), 105–107. – A classic case report that illustrates the principles we will discuss.

Why this reading list? I have deliberately selected a mix of textbook chapters (for foundational definitions), recent society statements (for best practice), and 2024-2025 primary literature (for cutting-edge updates). You will updated on evidence that changes how we manage patients today.

PART II – RECENT UPDATES & ADVANCEMENTS (2024–2025) THAT CHANGE PRACTICE

This section is the beating heart of the presentation. I will present five practice-changing updates, each backed by the literature you just reviewed.

Update 1 – ESC mWHO 2.0 classification (2025)

- **What's new:** The old mWHO classes have been refined with CARPREG II risk modifiers. For PPCM, a “fully recovered” LVEF is now strictly defined as > 50% – not 45%, not 49%.
- **Clinical impact:** Many women assume they are “low risk” after LVEF improves from 20% to 48%. Under mWHO 2.0, that residual impairment still places them in Class IV – pregnancy absolutely contraindicated.

• **Board relevance:** Expect an SAQ asking you to classify a patient with prior PPCM and current LVEF 52% vs 48%.

Update 2 – Bromocriptine effectiveness confirmed in large registry (EORP 2025)

- **Key numbers:** 552 PPCM patients, 85 treated with bromocriptine. Primary composite outcome (death, readmission, or persistent severe dysfunction)



occurred in 22% of bromocriptine group vs 33% of standard care (adjusted OR 0.47, $P < 0.001$).

- **Safety signal:** Thromboembolism 6.0% (bromocriptine) vs 5.6% (control) – not significant, provided therapeutic anticoagulation (LMWH) is used.

- **Take-home:** Bromocriptine is no longer experimental. It is an evidence-based, pathophysiology-targeted therapy that should be considered postpartum in all patients with severe PPCM.

Update 3 – Mechanical circulatory support (MCS) outcomes (Florea 2024; multicentre 2025)

- **Temporary MCS is now standard of care** for PPCM with cardiogenic shock. IABP first-line; Impella for higher flow; VA-ECMO for refractory cases or cardiac arrest.

- **Key statistic:** Survival to discharge with temporary MCS – 89% (Florea).

- **Practical implication:** Every tertiary obstetric unit should have an MCS escalation protocol. The anaesthesiologist must know who to call and when – “early involvement of cardiac surgery” is not a suggestion; it is a performance measure.

Update 4 – Anaesthetic technique innovations (2025 case series)

- **The “TUBE” approach** (AIIMS, New Delhi) for LVEF 20% undergoing Caesarean section: Transoesophageal echocardiography for real-time guidance, Ultrasound TAP block for opioid-sparing analgesia, Bolus phenylephrine (not infusion) for afterload control, Early multidisciplinary team huddle.

- **CSE with low-dose intrathecal component** is gaining favour over pure epidural for controlled sympathectomy with faster onset and lower failure rate.

- **Remifentanyl infusion** during general anaesthesia: profound suppression of laryngoscopy response without long-acting myocardial depression.

Update 5 – NEJM 2024 PPCM review highlights

- PPCM is now a leading cause of maternal death in the USA and globally – overtaking haemorrhage and pre-eclampsia in some registries.

- Cardiogenic shock in the peripartum period is caused by PPCM in approximately 60% of cases – meaning **every anaesthesiologist must be able to recognise and stabilise PPCM before escalation.**

How this section will be delivered: Slides will show the key graphs (EORP survival curves, MCS survival bar charts). I will walk you through the numbers, but more importantly, I will tell you how to use them in your next clinical shift – e.g., “If you see a postpartum patient with new dyspnoea and LVEF 30%, bromocriptine + LMWH should be on your order set within 24 hours.”

PART III – INTERACTIVE Q&A: WHAT WE ASK OUR POSTGRADUATE RESIDENTS

“The best way to learn is to be asked a difficult question when you are not expecting it.”

In this segment, I will act as a mock examiner. The audience will be the resident on call. I will pose **six Socratic questions** – the same ones we use during weekly case discussions in our department. You are encouraged to whisper your answers to your neighbour, write them down, or (if brave) speak aloud.

Question 1 (Opening – risk stratification)

“This patient has an LVEF of 43% – recovered from a nadir of 28% three years ago. According to the ESC mWHO 2.0 classification, what is her risk category for this pregnancy? What would be different if she had a persistent LVEF of 48%?”

Why we ask this: Many trainees mistakenly call any improvement “recovery.” The exam and real life require precision – $< 50\%$ is still Class IV, pregnancy contraindicated.

Question 2 (Pathophysiology – the “board favourite”)

“She is 20 weeks pregnant. What specific pregnancy-related hormonal pathway is most relevant to her PPCM pathology? And how does this influence your postpartum management plan?”

Why we ask this: Understanding the 16-kDa prolactin fragment leads directly to bromocriptine and explains why we do not use it antenatally (prolactin is needed



for lactation, but the damage occurs postpartum).

Question 3 (Inotropes – advanced pharmacology)

“If the patient deteriorates intraoperatively and requires inotropic support, which agent is most appealing from a myocardial oxygen consumption perspective – and why?”

Why we ask this: The classic trap is to reach for dobutamine. The superior answer is levosimendan (calcium sensitiser, increases contractility without increasing MVO₂).

Question 4 (Contraindicated drugs – high yield)

“List three HF medication classes strictly contraindicated in pregnancy, and for each name the specific foetal risk. Bonus: Which of these could be particularly problematic in a patient presenting with acute pulmonary oedema?”

Why we ask this: ACEi/ARB/ARNI are still prescribed by well-meaning cardiologists who are not obstetric specialists. Recognising them and stopping them is a patient safety issue.

Question 5 (Neuraxial technique – clinical reasoning)

“You have decided on an open appendicectomy. Why is single-shot spinal relatively contraindicated, and how would you perform a safe neuraxial technique instead?”

Why we ask this: The failing LV cannot handle a rapid sympathectomy. The answer must include continuous

epidural or low-dose CSE – not single-shot spinal.

Question 6 (Closing – synthesis)

“One sentence each – name the ONE most important: pathophysiology insight, drug contraindication, monitoring modality, and MCS device you would want available.”

Why we ask this: Forces the resident to prioritize. The correct list: 16-kDa fragment, ACEi/ARB/ARNI, invasive arterial line (plus TEE if available), and IABP as first-line MCS.

What you will gain from this Q&A: Not just answers, but a framework for answering under pressure – the ability to pivot from “I don't know” to “I know how to think about this.”

CLOSING REMARKS – THE “EXAM CHEAT SHEET” AND FINAL PEARL

I will end with a single slide summarising **the seven non-negotiable principles** (printed in your delegate handout). But the final verbal pearl is this:

“The patient with PPCM presenting for emergency non-cardiac surgery has one fundamental vulnerability: the failing left ventricle is exquisitely afterload-sensitive. Protect the afterload, maintain normovolaemia, avoid tachycardia, and have your MCS escalation plan ready. Master these principles, and you will be prepared not just for the board examination, but for the real clinical scenario where a life depends on your decisions.”



14TH JUNE 2026
02:40PM - 03:10PM

MANAGEMENT OF TURP

A 70 YEAR OLD GERIATRIC PATIENT DIAGNOSED WITH BPH POSTED FOR TURP.

PRESENTED BY: SAPTHAGIRI INSTITUTE OF MEDICAL SCIENCES AND
RESEARCH CENTRE

Presenters: Dr Sneha B, Dr Anvitha Anand

Moderator: Dr Narendra Babu

READING MATERIAL AVAILABLE QUESTIONS And ANSWERS

Preoperative Evaluation and Preparation

1. What is TURP? What is the incidence of BPH? How is TURP performed?

TURP is a procedure where prostate tissue is resected by an electrically powered coagulating metal loop passed through a urethroscope. It is used to alleviate the symptoms of bladder outflow obstruction, usually caused by benign prostatic hypertrophy (BPH). This enables the hypertrophied prostate to be resected in pieces and washed out using an irrigation solution.

BPH affects 50% of males at 60 years and 90% of 85-year-olds and so TURP is most commonly performed on elderly patients, a population group with a high incidence of cardiac, respiratory and renal disease.

2. How to preoperatively assess a patient posted for TURP?

Reduced functional reserve should be quantified, and the presence of any organ failure noted. If the patient has ongoing medical problems that can be improved before surgery, then TURP may need to be delayed. A decision can then be made with the patient between regional and general anaesthesia, based on consideration of the advantages and disadvantages of each technique in their particular case. For some patients the risks of anaesthesia and surgery may outweigh the potential benefits of an elective procedure such as TURP.

3. What history and examination is important for a geriatric patient?

- **Cardiovascular** - Hypertension, ischaemic heart disease (IHD) and arrhythmias (particularly atrial fibrillation) are common. Patients with recent onset or poorly controlled heart failure have the highest perioperative mortality. Major risk factors for IHD

(hypertension, diabetes, smoking, hypercholesterolaemia and family history) will raise the likelihood of silent perioperative myocardial ischaemia.

- **Respiratory** - Marked decrease in functional ability (e.g. inability to climb one flight of stairs) suggests severe disease. Inability to lie flat because of dyspnoea from cardiac or respiratory causes will make awake spinal anaesthesia poorly tolerated.

- **Neurological** - Confused patients may be not lie still during spinal anaesthesia.

- **Musculo-skeletal** - Degenerative changes in the vertebral column may make subarachnoid block (SAB) technically difficult. Arthritic joints or joint replacements are susceptible to damage or dislocation when the patient's legs are placed in the lithotomy position for the procedure.

- **Renal** impairment may occur due to obstructive uropathy

- **Airway** - Even if SAB is planned perform a full anaesthetic assessment (e.g. anticipated airway difficulties) in case the regional technique fails or is inadequate.

- **Drug history** - A high proportion of elderly patients take cardiovascular medications. Beta-blockers suppress the compensatory tachycardic response to hypotension associated with SAB or haemorrhage, but should generally be continued for prevention of perioperative myocardial ischaemia. ACE inhibitors limit the renin-angiotensin mediated response to hypovolaemia that may be further impaired by SAB, and most anaesthesiologists omit them for 24 hours preoperatively. Alpha blockers are commonly encountered as first-line medical treatment for BPH. The combined hypotensive effects of these drugs may precipitate severe hypotension after SAB. Warfarin has implications for both the anaesthesiologist (regarding SAB) and the surgeon (intra- and postoperative



haemorrhage). If the INR is greater than 1.4 the procedure should be postponed until the INR is acceptable.

4. What investigations are to be sent before TURP?

- Full blood count or haemoglobin level
- Creatinine and electrolytes - this will detect renal impairment or overt renal failure, commonly secondary to obstructive uropathy.
- ECG for symptomatic patients, and routinely over 60 years
- Group and save - consider cross-matching blood for anaemic patients and those suspected of having large prostates on examination or ultrasound scan.

Other tests may be indicated in particular circumstances:

- Clotting studies (prothrombin time if on warfarin)
- Blood gas and pulmonary function tests (severe respiratory disease)
- Chest radiograph (worsening cardiac or chest disease / suspicion of metastases)

Urinalysis (for glucose, protein, blood, white blood cells)

Blood glucose

- Test for sickle cell disease or haemoglobinopathies in patients of African or Mediterranean extraction respectively.

5. How would you evaluate the patient's cardiac condition? Does he need additional testing?

- The American College of Cardiology/American Heart Association guidelines suggest a stepwise approach that takes into account the clinical markers of cardiac risk, surgery risk, and functional capacity.
- The patient's medical **history** should focus on **information about previous cardiac evaluations and interventions** (coronary stent and automatic ICD [AICD] placement). **Active cardiac conditions** such as **unstable coronary syndromes** (recent MI or unstable angina), **decompensated congestive heart failure (CHF)**, **significant arrhythmias**, or **severe valvular disease** warrant additional **work up prior to surgery**.
- If the patient has stable coronary artery disease (CAD), then risk for **major adverse cardiac event (MACE)** can be determined using combined clinical/surgical risk score.
- If **risk for MACE is low (<1%)**, **no additional information** is required.
- If **MACE is elevated**, the **functional capacity** of the

patient is best evaluated by the **activity history** and assessment of metabolic equivalents (METs), with >4 to **10 METs** being suggestive of sufficient cardiac reserve.

- **Stress testing** may be indicated for patients with **poor exercise tolerance** or **unable to assess METs**, if this would affect the choice to proceed with surgery. Electrocardiogram (ECG) is reasonable in a patient with cardiac history undergoing greater than low-risk surgery but is not required.

6. Would you discontinue any antihypertensives or any medications for angina?

Antianginal agents and β -blockers should be continued perioperatively including the day of surgery to **prevent rebound hypertension and tachycardia** that may result from sudden withdrawal of these drugs. Patients taking **ACE inhibitors or angiotensin II receptor blockers (ARBs)** can be predisposed to refractory intraoperative hypotension due to blunting of the sympathetic effect on vascular tone by anaesthesia, increasing reliance on the renin-angiotensin-vasopressin system to maintain blood pressure and renal perfusion. It is recommended that these medications be **held 24 hours before surgery**.

Intraoperative Management

1. What monitors would you use for this patient?

The following monitors would be used for this patient:

- American Society of Anesthesiologists standard monitors: pulse oximeter, NiBP, ECG including multiple-lead ST-segment analysis.
- Temperature
- Consider invasive arterial blood pressure.

2. Is there a preferred anaesthetic technique for patients undergoing TURP, and why?

The choice of anaesthetic technique should be **guided by the requirements of the surgical procedure, coexisting diseases, need to prevent postoperative complications, and patient preferences**.

Regional anaesthesia has advantages for patients undergoing M-TURP for the following reasons:

- It allows monitoring of the patient's mentation, thereby facilitating early detection of signs of TURP syndrome.
- It can allow for earlier detection of prostate capsular tears or bladder perforation via patient reports of peritoneal or diaphragmatic (referred shoulder) pain.
- It promotes vasodilation and peripheral pooling of



blood, thereby reducing the severity of circulatory overloading.

- It reduces blood loss by lowering arterial and venous blood pressure during surgery.
- However, less emphasis is placed on regional anaesthesia for TURP now because of the increased use of bipolar electrode and laser resection surgical techniques.
- Post operative analgesia.
- Regional anaesthesia may not be a safe option in an anticoagulated patient.

DISADVANTAGES:

- Hypotension
- Obturator reflex cannot be obtunded.

General Anaesthesia:

ADVANTAGES:

- Patient preference.
- Useful for patients who are unable to lie supine.
- Useful for patients with neuromuscular disease and pulmonary compromise.
- No time constraints. Although the procedure should be kept as short as possible.
- May be less haemodynamically challenging than SAB in patients with cardiac problems such as aortic stenosis (and other fixed output states) and IHD.
- Allows better control of CO₂, which may reduce bleeding from the prostatic bed.

DISADVANTAGES:

- Unable to monitor mental status.
- Possibility of difficult airway and risk of aspiration.
- Reduced FRC due to lithotomy position.
- Post operative nausea and vomiting.
- Post operative analgesia needed.

3. Does regional anaesthesia result in a lower incidence of perioperative mortality than general anaesthesia?

Although spinal anaesthesia offers distinct advantages over general anaesthesia for TURP surgery, mortality and many markers of patient outcome are **similar for both groups**, in large part because the safety of the procedure itself has advanced. The 30-day mortality rate associated with TURP has steadily declined from 2.5% in 1962 to current estimates of 0.2% to 0.8%.

Mortality rates are similar in patients receiving regional anaesthesia or general anaesthesia. **Increased morbidity** is more associated with surgical and patient factors than anaesthesia type and has been found in patients with **resections exceeding 90 minutes, gland size greater than 45 g, acute urinary retention, and age older than 80 years.**

The **incidence of postoperative complications**, specifically MI, pulmonary embolism, cerebrovascular accidents, transient ischemic attacks, renal failure, hepatic insufficiency, and the need for prolonged ventilation is **similar in patients receiving regional anaesthesia and those receiving general anaesthesia.**

With the increased use of L-TURP resection surgical techniques, even less emphasis has been placed on regional anaesthesia as the preferred anaesthetic technique given minimal absorption of irrigating fluid.

4. What are the common intravenous fluids used?

Normal saline (0.9% NS), Ringer Lactate, DNS: Dextrose Normal Saline.

5. What intravenous fluid would you use during TURP?

Because dilutional hyponatremia commonly occurs in patients undergoing M-TURP, the intravenous fluid of choice perioperatively is normal saline that contains sodium, 154 mEq per L. Circulatory overloading from absorbed irrigant is common in patients undergoing M-TURP or B-TURP (but not L-TURP); therefore, the amount of intravenous fluid administered during surgery should be carefully monitored.

6. What are the important characteristics of an ideal irrigation solutions used during TURP?

Ideal TURP irrigation solutions

- Isotonic / Iso-osmolar prevents fluid shifts
- Non-toxic → safe on systemic absorption
- Non-hemolytic → no RBC destruction
- Non-electrolyte → compatible with monopolar cautery
- Non-metabolized / minimally metabolized → avoids toxic byproducts
- Rapidly excreted → prevents fluid overload
- Optically clear → good surgical visibility
- Non-conductive → allows effective cautery
- Inexpensive & easily available

Sterile



Electrolyte-containing solutions such as normal saline are avoided in M-TURP because they can conduct electrical current to surrounding tissues and cause burns. Instead, solutions with nontoxic solutes are used for M-TURP; however, the need for transparency necessitates a hypotonic solution. If intravascular hypotonic bladder irrigating solution is absorbed, absorption can lead to hyponatremia, hypoosmolality, and, in severe cases, haemolysis. These complications are avoided when normal saline is used with B-TURP and L-TURP resection.

7. What are the commonly used irrigating fluids?

The most commonly used solution for **M-TURP** is **glycine** (1.2% and 1.5%). Mannitol (3%), glucose (2.5% to 4%), Cytal (a mixture of sorbitol 2.7% and mannitol 0.54%), and urea (1%) solutions are also occasionally used.

8. What are the intraoperative complications associated with TURP?

- TURP Syndrome
- Irrigating fluid related complications
- Bleeding and coagulopathy
- Bladder perforation
- Hypothermia
- Associated with lithotomy position

9. Forty minutes after a sensory level of T10 was established with intrathecal anaesthesia, and monopolar TURP (M-TURP) resection was initiated with the use of glycine as the bladder irrigating solution, the patient becomes agitated and complains of nausea. Further sedation along with an antiemetic is administered. However, shortly thereafter, the patient becomes very restless, blood pressure rises, and heart rate decreases. The patient becomes cyanotic and obtunded, blood pressure precipitously falls, and pupils are dilated and unresponsive to light. What is the most likely cause of these signs and symptoms, and would these occur during bipolar (B-TURP) or laser TURP (L-TURP) resection?

Restlessness and incoherence during TURP are particularly **ominous signs of TURP syndrome**. These are caused by **pulmonary edema, hypoxemia, and cerebral edema**. These signs must not be misinterpreted as signs of inadequate anaesthesia. The administration of sedatives or general anaesthesia in the presence of TURP syndrome has been associated with severe complications including death.

In B-TURP and L-TURP, normal saline can be used as the bladder irrigating solution and hyponatremia is not expected.

In B-TURP, absorption of the intravascular bladder irrigating solution can still occur via the prostatic venous sinuses, resulting in circulatory overload. This is not expected in L-TURP because the treatment zone is rapidly coagulated.

10. What is the definition of TURP syndrome and what is its classical triad?

Rapid absorption of a large-volume, hypotonic bladder irrigation solution during TURP can lead to TURP syndrome. TURP syndrome is characterized by intravascular volume shifts and plasma-solute (osmolality) effects.

An average of 10 to 30mL of fluid is absorbed per minute of resection time, amounting to up to 1800mL per hour. The glycine containing irrigation solution is slightly hypo-osmotic (200mosm/L) and therefore the classical triad of features that make up TUR syndrome are:

- i. **Dilutional hyponatraemia.** Encephalopathy and seizures may develop when the sodium concentration falls below 120mmol/L. Cerebral oedema may occur.
- ii. **Fluid overload.** This causes pulmonary oedema and cardiac failure.
- iii. **Glycine toxicity.** This inhibitory neurotransmitter causes depression of the level of consciousness and visual impairment at toxic levels.

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

The signs and symptoms of TURP syndrome are as follows:

Cardiopulmonary

- Hypertension (fluid overload), then hypotension (cardiac insufficiency)
- Bradycardia
- Angina
- Dysrhythmia (VT/VF)
- Respiratory distress
- Cyanosis
- Hypotension
- Shock
- Death



Hematologic and Renal

- Hyperglycinemia
- Hyperammonemia
- Hyponatremia
- Hypoosmolality
- Hemolysis/anemia
- Acute renal failure
- Death

Central Nervous System

- Nausea/vomiting
- Confusion/restlessness
- Transient blindness - glycine toxicity
- Twitches/seizures
- Lethargy/paralysis
- Dilated/nonreactive pupils
- Coma ($\text{Na} < 100\text{mmol/L}$)
- Death

TURP syndrome can occur as **early as 15 minutes after surgery has started and as late as 24 hours after surgery.**

Patient complaints of dizziness, headache, nausea, chest or throat tightness, and shortness of breath are often **early clinical signs**. Then the patient becomes restless, confused, and nauseated. Some patients complain of abdominal pain. The blood pressure often rises (both systolic and diastolic), and the heart rate decreases. If not treated promptly, the patient becomes cyanotic and hypotensive and ultimately sustains cardiac arrest.

Occasionally, TURP syndrome manifests as neurologic signs. The patient becomes lethargic and then unconscious, and pupils dilate, reacting sluggishly to light. These signs may be followed by short episodes of tonic-clonic seizures and then coma lasting from minutes to hours.

12. How is TURP recognised under general anaesthesia?

If the patient is under general anaesthesia, the presenting signs of TURP syndrome are typically a rise and then a fall in blood pressure, decreasing oxygen saturation, and severe refractory bradycardia. The ECG may show nodal rhythm, ST-segment changes, U waves, and widening of the QRS complex. Recovery from general anaesthesia is delayed.

13. How does the patient absorb irrigation solution during TURP?

The bladder irrigation solution enters the bloodstream directly through **open prostatic venous sinuses** created during monopolar and bipolar resection techniques, particularly if the prostatic capsule is violated during surgery.

If the complication of bladder perforation occurs, significant accumulation of irrigating fluid in the intraperitoneal or retroperitoneal space can occur regardless of technique, resulting in significant intravascular absorption.

14. How does one calculate how much irrigating fluid is absorbed?

The amount of fluid absorbed normally during TURP procedure is about 20mL/min with an average of 1-1.5L by the end of surgery.

The amount of irrigating solution that is absorbed during the procedure can be determined by comparing serum sodium level during the procedure with serum levels at the beginning of the procedure.

Volume absorbed

(Pre operative serum sodium x ECF volume) - ECF volume Measured intra/postop sodium

ECF volume is usually approximated 20% of body weight ($0.2 \times 70 = 14\text{L}$)

15. What factors increase the incidence of TURP syndrome?

TURP syndrome is more likely to occur if the **prostatic gland is atypically large**, the prostatic capsule is violated during surgery, or the **hydrostatic pressure of the irrigation solution is excessively high**.

Large prostatic glands have **rich venous networks** that promote intravascular absorption of irrigation solution. The violation of the prostatic capsule during surgery promotes entry of irrigation solution into the periprostatic and retroperitoneal spaces.

The **hydrostatic pressure of the irrigation solution** is an important determinant of the rate at which the patient will absorb the solution. This pressure **depends** primarily on the **height of the irrigation solution pole**. When the height of the pole **exceeds 60 cm** (approximately 2 ft), the **absorption** of irrigation solution is **greatly enhanced**. Also, an excessively distended bladder during surgery facilitates absorption.



16. What is the effect on cardiopulmonary, renal, and central nervous system (CNS) functions of excessive absorption of irrigation solution during TURP?

Excessive absorption of irrigation solution during TURP causes **hypervolemia and hypertension with bradycardia and may induce angina and pulmonary edema**. It may also have an adverse effect on renal function. *An inverse relation has been observed between the amount of irrigation solution absorbed during surgery and postoperative urinary output.*

Some patients with TURP syndrome **exhibit neurologic signs** resulting from water intoxication if a **hypotonic irrigating solution** is used. They assume a **decerebrate posture, exhibit clonus and positive Babinski reflex, and eventually convulse and lapse into coma**. Examination of the **eyes reveals papilledema and dilated and sluggishly reacting pupils**. The electroencephalogram shows low voltage bilaterally. The patients may remain in a coma from a few hours to several days.

17. What causes CNS dysfunction in patients who have undergone M-TURP?

The physiologic derangement of CNS function in TURP syndrome is not hyponatremia but **acute hypoosmolality**, which is predictable because the blood-brain barrier is **essentially impermeable to sodium but freely permeable to water, leading to cerebral edema**.

The primary factor governing the manifestation of symptoms is the rate at which the serum sodium level falls rather than the total fall itself; the faster the fall in serum sodium level, the greater the incidence of neurologic symptoms. However, under 120 mEq per L, all patients are symptomatic.

Cerebral edema caused by acute hypoosmolality can increase intracranial pressure, which results in **bradycardia and hypertension by the Cushing reflex**. The rise in intracranial pressure is directly correlated to the gain in body weight during TURP.

18. What effect do different serum sodium levels have on the CVS, CNS, and ECG ?

[S. Na]	CVS effects	CNS effects	ECG changes
120 mEq/L	Hypotension, bradycardia	Confusion, restlessness	Wide QRS
115 mEq/L	Cardiac depression	Somnolence, nausea	Wide QRS, ST elevation
110 mEq/L	Congestive heart failure	Seizures, coma	Ventricular tachycardia, fibrillation

19. Suppose a patient weighing 50kgs has a measured serum sodium concentration -125mEq/L. If correction with Inj normal saline (NS) for hyponatremia correction, how much would be required and at what rate?

Step 1: calculate sodium deficit

assuming normal serum sodium concentration to be 135mEq/L, sodium deficit that needs correction (135-125mEq/L).

Step 2: calculate the total sodium correction needed

Correction required = $0.6 \times \text{weight of the patient} \times \text{sodium deficit}$
 Correction required = $0.6 \times 50 \times 10 = 300\text{mEq}$

Step 3: calculate the total volume of normal saline required for correction. Since Inj NS contains 154 mEq/L of Na⁺, total volume of NS required = $300/154=1.94\text{ L}$ (approx 2L)

Step 4: calculate the rate of correction

Maximum rate of correction of Na⁺ allowed = 0.5 mEq/L/hr A deficit of 10mEq/L requires 20hrs for total correction of the deficit ($10\text{mEq/L}/0.5\text{ mEq/L/h} \times r = 20\text{ hrs}$)

Final volume and rate of correction required is 2L of Inj NS at the rate of 100mL/hr for 20hours.

Too rapid correction with 3% hypertonic saline may cause Central pontine myelinolysis due to rapid increase in plasma osmolality.

20. What prophylactic measures may reduce the incidence of TURP syndrome?

The most important preventive measure during surgery is **preservation of the prostatic capsule**.

RULE OF 60s

- Duration of Resection < 60 minutes
- Amount of prostate resected < 60gms
- Height of irrigating fluid < 60cm above the patient (above pubic symphysis or OT table)
- Venous sinus pressure should remain <60mm Hg

Serial serum sodium and osmolality are advisable in order to detect a decreasing trend in M-TURP. If clinical signs of TURP syndrome present, the surgeon should be informed and therapeutic measures immediately instituted. If serum sodium level approaches 120 mEq per L, surgery should be terminated as soon as



possible.

Intravenous fluids should be cautiously administered during M-TURP and B-TURP. A microdrip is recommended particularly for patients with cardiac or renal disease. If regional anesthesia causes hypotension, a small dose of a vasoconstrictor is recommended to raise the blood pressure rather than rapid infusion of intravenous fluids.

21. What therapeutic measures are recommended for patients with TURP syndrome?

When symptoms of TURP syndrome appear, the following therapeutic measures are recommended:

- Terminate surgery as soon as possible.
- Administer **furosemide**, 20 to 40 mg intravenously. Although furosemide results in natriuresis, this effect is negligible relative to the desired acute diuresis and volume offloading of free water.
- Administer **oxygen** by nasal cannula or face mask to maintain oxygen saturation and consider intubation.
- Obtain arterial blood gas, serum osmolality, and sodium analysis. If the serum sodium level is abnormally low and clinical signs of hyponatremia are evident, intravenous administration of hypertonic saline (3% to 5%) may be recommended. The hypertonic solution should be given at a rate no faster than 100 mL per hour. In most cases, no more than 300 mL of hypertonic saline is needed to correct the hyponatremia.
- If the patient develops seizures, a short-acting anticonvulsant such as diazepam (5 to 20 mg) or midazolam (2 to 10 mg) can be administered intravenously. If these drugs do not arrest the seizures, a barbiturate or phenytoin can be added.
- If **pulmonary edema or hypotension** develops, invasive hemodynamic monitoring is recommended. This will serve as a guide for pharmacologic support and fluid administration.
- If **significant blood loss** is suspected, the administration of packed red blood cells should be considered. *In general, intravenous fluids should be administered cautiously because of the propensity of these patients to develop pulmonary edema.*

22. Is hypertonic saline administration necessary to correct hyponatremia? Explain. What are the risks of rapidly correcting hyponatremia?

The administration of hypertonic saline to correct hyponatremia is **not always necessary** and sometimes

can be detrimental. **Unless the patient develops severe clinical signs of hyponatremia**, hypertonic saline administration is not recommended. Spontaneous or induced diuresis usually corrects the hyponatremia within a few hours.

Patients who have undergone TURP often sustain circulatory overloading during surgery; consequently, hypertonic saline administration in these patients can induce pulmonary edema. In addition, **rapid administration** of hypertonic saline has been associated with **central pontine myelinolysis (osmotic demyelination syndrome)**-a poorly understood and fatal neurologic complication.

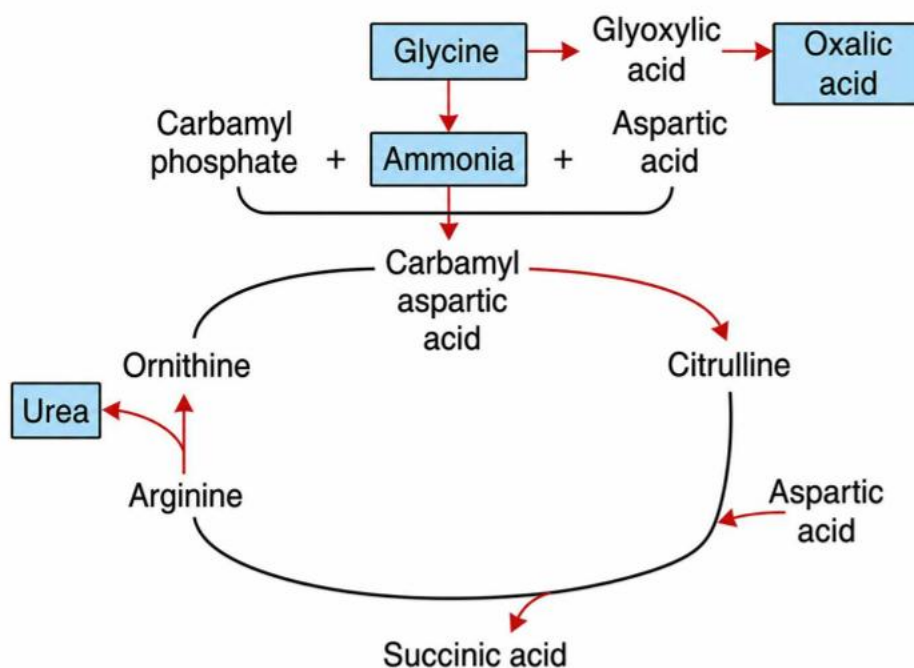
To reduce the hazards of hypertonic saline administration, serum osmolality should be monitored and corrected aggressively only until symptoms substantially subside and then **hyponatremia** should be corrected at a **rate no faster than 1.5mEq/L/hr**.

23. What are the toxic effects of glycine? Is there an antidote to glycine toxicity? What are the metabolic by-products of glycine?

Glycine is an inhibitory neurotransmitter similar to γ aminobutyric acid in the spinal cord and brain. When absorbed by the patient in large amounts, glycine has direct **toxic effects on the heart and retina**. In patients who have undergone M TURP with glycine absorption, it has been shown to cause an average decrease of 17.5% in cardiac output. In animal studies, the administration of the amino acid arginine reversed the myocardial depressing effect of glycine. The mechanism by which glycine depresses cardiac function and the mechanism by which arginine protects the heart is unknown.

Hyperglycinemia may cause **transient visual disturbance** (blindness) during TURP. However, glycine toxicity in patients who have undergone TURP is uncommon, probably because most of the **absorbed glycine is retained** in the **periprosthetic and retroperitoneal spaces**, where access to the circulation is limited. The most common metabolites of glycine are ammonia and glyoxylic and oxalic acids (Fig. 24.4). In some patients, **excessive absorption of glycine** during TURP leads to **hyperammonemia**. Hyperoxaluria could compromise renal function in patients with coexisting renal disease, which is often present in elderly patients undergoing TURP.





24. What are the symptoms and clinical course of TURP-induced hyperammonemia?

The signs and symptoms of hyperammonaemia usually **appear within 1 hour of surgery**. Typically, the patient becomes **nauseated, vomits, and then becomes comatose**. Blood ammonia level can rise to more than 500 mmol per L. The patient **remains comatose for 10 to 12 hours** and eventually *awakens when ammonia blood level decreases to less than 150 mmol per L*. The hyperammonaemia tends to **linger postoperatively**, probably because *glycine absorption from the periprostatic space continues after surgery*.

25. What are the clinical characteristics, causes, and prognosis of TURP-related blindness?

Transient blindness is one of the more alarming complications of TURP. The patient complains of **blurred vision and of seeing halos around objects**. This can occur either **during surgery or later in the recovery room**. Although it is sometimes accompanied by other TURP-related complications, blindness **can occur as an isolated symptom**. Examination of the eyes reveals **dilated and unresponsive pupils**. Postoperatively, TURP related blindness gradually recedes and eyesight returns to **normal within 8 to 48 hours** of surgery.

The cause of TURP-related blindness is unclear. The intraocular pressure and optic discs remain normal. In the past, edema of the cerebral optical cortex was suspected; however, the clinical signs of TURP-related blindness are consistent with retinal rather than cortical dysfunction. **Unlike cortical blindness, TURP-**

related blindness allows perception of light and the blink reflex is preserved. TURP-related blindness is most likely caused by a **toxic effect of glycine on the retina**. This theory is supported by the fact that glycine is a known inhibitory neurotransmitter. In animals, glycine has been shown to inhibit neuronal visual pathways. Also, the **severity of TURP-related blindness is directly related to glycine blood level**. Postoperatively, vision gradually improves as the glycine blood level declines.

26. What are the causes of excessive bleeding during TURP?

Patients undergoing TURP commonly bleed perioperatively, with 2.5% of patients undergoing M-TURP requiring transfusion. One possible cause is **dilutional thrombocytopenia** resulting from excessive absorption of irrigation solution. Another possible cause is **local release of fibrinolytic agents** (plasminogen and urokinase) from the **mucosa of the lower urinary tract**. *During and immediately after TURP, these agents cause local fibrinolysis and promote bleeding from the raw surfaces of the prostatic gland. The existence of a local fibrinolytic mechanism is supported by the observation that prophylactic administration of e-aminocaproic acid (an antifibrinolytic agent) reduces bleeding from the prostatic bed.*

Systemic coagulopathy can also occur during TURP. The coagulopathy is caused by **disseminated intravascular coagulation (DIC)** rather than by primary



fibrinolysis. DIC is triggered during TURP by prostatic particles rich in thromboplastin that enter **the bloodstream during surgery. Characteristically, the platelet count and fibrinogen blood level are abnormally low.** At autopsy, multiple microthrombi are observed in various parts of the body. The high level of fibrin degradation products found in the blood of these patients is caused by **secondary fibrinolysis, which commonly accompanies DIC.**

27. How is blood loss calculated?

A rough estimate of blood loss has been calculated as 15ml blood loss per gram of resected tissue or 2.6-4.6mL/min of resection time.

Blood loss can be estimated by measuring the amount of irrigant blood mixture and its mean haematocrit(Hct)

Hb of irrigating fluid (g/L)

_Patient's Hb (g/L) x Amount of irrigating fluid(L)

28. What are the causes, signs, symptoms, and treatment of bladder perforation during TURP?

The incidence of bladder perforation during TURP is approximately 1% and stems from **trauma by surgical instrumentation, overdistention of the bladder, and, rarely, explosion of traces of hydrogen gas inside the bladder.** An **early sign** of bladder perforation is a sudden decrease in the return of irrigation solution from the bladder. This sign often goes unnoticed until a significant amount of irrigation solution accumulates in the abdomen, causing abdominal distress. Other clinical signs are **hypotension**, followed by **hypertension** and **nausea**. Reflex-type movements of the limbs have been observed under both general and regional anaesthesia. When the perforation is **intrapertitoneal, symptoms develop faster and include severe shoulder pain caused by diaphragmatic irritation.**

Diagnosis of bladder perforation is confirmed by cystourethrography. Treatment is immediate suprapubic cystostomy.

29. What is the effect on body temperature of continuous bladder irrigation during TURP?

Several litres of irrigation solution pass through the bladder **during TURP. This can reduce body temperature at the rate of 1°C per hour.** Approximately half of patients undergoing TURP become hypothermic and shiver at the conclusion of surgery.

30. What are the causes of hypotension during TURP?

Blood loss, severe hyponatremia, and bacteraemia.



CHF, MI or myocardial ischemia, severe anaemia



Circulatory overloading



Hypotension, bradycardia

31. What are the physiological changes and problems associated with lithotomy positioning during TURP?

Physiological changes with lithotomy

- ↓ FRC
- ↑ Venous return on elevation of legs
- Venous return after lowering of legs
- Exaggeration of hypotension with SAB

Problems with lithotomy position

- Nerve injuries: Common peroneal nerve, saphenous nerve, obturator / femoral nerve, sciatic nerve.
- Compression of major vessels at joints
- Lower extremity compartment syndrome
- Aggravation of pre-existing lower back pain.

Postoperative Management

1. What is the source of postoperative bacteraemia in patients who have undergone TURP?

What factors increase the incidence of bacteraemia?

The **prostatic gland** often harbours **large amounts of bacteria.** TURP facilitates the entry of these bacteria into the **bloodstream by opening of venous sinuses,** leading to postoperative bacteraemia. Approximately 30% of patients undergoing TURP have infected urine preoperatively, and half of them have transient bacteraemia postoperatively, although most are asymptomatic; 6% to 7%, however, will develop signs of sepsis. Surgical disruption of the prostatic venous sinuses and excessive hydrostatic pressure of the irrigation solution facilitate the entry of bacteria into the bloodstream. Also, a preoperative indwelling catheter promotes bacterial growth in the urethra.

2. What are the signs of post-TURP septicaemia? What preventive measures are generally recommended?

The signs of septicaemia usually appear postoperatively in the postanesthesia care unit (PACU). They include fever, chills, low blood pressure, and **tachycardia.** Symptoms may be related to **bacterial endotoxins,** in which case they are self-limited, or



direct bloodstream seeding with bacteria. Antibiotics administered prophylactically before TURP do not sterilize the urine because they do not easily penetrate the prostatic gland. Nevertheless, the **preoperative administration of antibiotics is strongly recommended**. If sepsis is suspected postoperatively in a patient who has undergone TURP, broad-spectrum antibiotics should be started immediately without waiting for blood culture results.

3. Is postoperative hypothermia a risk factor for myocardial ischemia?

Yes. Unexpected hypothermia and shivering is associated with a significantly higher incidence of postoperative demand myocardial ischemia. Therefore, it is important to maintain normothermia during the perioperative period, particularly in an elderly population at higher risk for hypothermia due to decreased subcutaneous fat.

4. What is postoperative cognitive dysfunction (POCD)?

POCD is a syndrome defined by worsening cognitive performance postoperatively compared to perioperative baseline. The incidence ranges from **2.2% to 31.5%**. The deficit is present in multiple domains and presents days to weeks after surgery, highly correlated with age. POCD usually resolves within months; however, time course varies and sustained deficits have been reported.

Comparing M-TURP, B-TURP, and L-TURP

1. What differentiates M-TURP from B-TURP?

M-TURP uses high-energy cautery, which allows a fast cutting technique of prostatic tissue with minimal coagulation in the process. The body is the conduit of the energy dispersed from the monopolar electrode to complete the electrical circuit to a large-surface skin grounding pad placed on the patient. It also requires the use of **an electrolyte-free bladder irrigating fluid**.

In B-TURP, the electrical circuit is completely contained within the resectoscope. This prevents energy from traveling through the body. Instead, the energy remains confined to the site of prostatic resection and is returned via the second limb of the bipolar resectoscope. **With the bipolar electrode, less bleeding occurs at the prostatic tissue site and normal saline can be used as the bladder irrigating fluid.**

2. Are decreased morbidity and mortality associated more closely with M-TURP or with B-TURP?

In a meta-analysis that reviewed the safety of M-TURP versus B-TURP in a small number of TURP patients (1,802), a significant benefit was reported in the bipolar group in overall complication rate, need for transfusion, and the absence of TURP syndrome. Subsequent randomized controlled trials have confirmed this morbidity benefit.

3. What differentiates the technologies for L-TURP?

L-TURP delivers light energy to coagulate or vaporize prostatic tissue. **Holmium laser enucleation of the prostate (HoLEP)**, in particular, has been suggested as **the new gold standard for surgical treatment** of benign prostate hypertrophy (BPH). Prostatic tissue is vaporized, and the resulting heat dissipation coagulates small to medium blood vessels. HOLEP can be used safely on patients with larger prostatic glands with similar outcomes as patients undergoing open prostatectomy, and compared to traditional M-TURP, HOLEP demonstrates lower transfusion rates, catheterization time, and shorter hospital stays. Photoselective vaporization and bipolar plasma vaporization have shown similar benefits.

There are five advantages of L-TURP:

- (1) It uses normal saline as the irrigating bladder solution;
- (2) it minimizes the absorption of irrigating fluid;
- (3) it minimizes or altogether eliminates TURP syndrome;
- (4) it produces significantly less bleeding, which allows the procedure to be performed on an anticoagulated patient if needed; and
- (5) it de-emphasizes regional anesthesia as the preferred anesthetic technique.

RECENT UPDATES

Bipolar TURP with saline irrigation has replaced much of the older monopolar "classic TURP syndrome" framework, because it lowers the risk of dilutional hyponatremia and fluid-absorption complications.

Modern management focuses less on rigid "spinal only" teaching and more on matching anesthesia, monitoring, and postoperative vigilance to patient risk and the type of resection system used.

Regional anesthesia remains valuable because it helps detect early neurologic symptoms and may improve



perioperative visibility of complications.

TURP syndrome is still important, but it is less common with bipolar systems; when it occurs, immediate cessation of surgery and correction of hyponatremia/volume issues remain central.

Older articles emphasize avoiding hypotonic fluids, keeping surgery short, and treating TURP syndrome with supportive care and sodium correction. Newer advances add that bipolar systems and saline irrigation have changed the baseline risk, so routine fluid shifts are usually less dramatic than in the older monopolar era. In practical terms, this means anesthetic vigilance is still needed, but the preventive focus has shifted toward procedure type, irrigation strategy, and selective electrolyte monitoring.

TURP syndrome management

The consistent modern message is: stop the procedure, support oxygenation and circulation, check sodium and acid-base status, and treat symptomatic hyponatremia promptly. Furosemide is part of management, but current practice gives priority to correcting symptomatic hyponatremia and treating pulmonary edema or neurologic compromise according to severity. Hypertonic saline is reserved for clinically significant symptomatic cases rather than mild lab changes.

TURP anaesthesia has evolved from a monopolar, hyponatremia-prone procedure toward bipolar saline TURP with lower fluid-absorption risk, while spinal

anaesthesia and early recognition of TURP syndrome still remain important. The older articles are still excellent for the core physiology and emergency response, but the modern update is the reduced incidence and different risk profile created by bipolar technology.

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14th JUNE 2026
02:40PM - 03:10PM

A geriatric patient with BPH posted for TURP

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Case Summary:

A 74year old male with history of fever, increased frequency of micturition, poor urinary stream, easy fatiguability with stable vitals and clinical examination suggestive of urinary tract infection with benign prostatic hyperplasia posted for transurethral resection of the prostate.

1. What is geriatric population?

Geriatric population is defined as patients aged more than 65 years and older. Geriatric Medicine deals with patients in this age group with emphasis on the physiological changes of aging and their impact on anesthesia and perioperative care. It focuses not just on age, but also on decline in organ function, altered pharmacokinetics and pharmacodynamics and increased comorbidities and frailty.

2. Why past history is important in geriatrics?

In geriatric patients, past history is extremely important because ageing is associated with multiple comorbidities, reduced physiological reserve, and altered drug handling.

Past history directly influences risk assessment, drug choice, and perioperative management.

1) Presence of Multiple Comorbidities

Elderly patients commonly have:

- Hypertension
- Diabetes
- Ischemic heart disease
- COPD
- Renal disease

2) Polypharmacy

Elderly patients often take multiple drugs such as antihypertensives, anticoagulants and antidiabetic drugs. The importance of this is the risk of **drug interactions**, some drugs must be continued (e.g., beta-blockers) and some drugs must be stopped (e.g., anticoagulants) before being considered for

anaesthesia and surgery.

3) Functional Status & Exercise Tolerance

It is the ability to perform daily activities and any history of breathlessness. Its importance reflects on cardiopulmonary reserve and predicts perioperative complications.

4) Organ Function of Liver, Kidney and Heart:

Past history gives an idea regarding Coronary heart diseases, disorders of kidney and liver that reveals chronic dysfunction. Its importance guides dose **modification** and hence prevents drug complication.

5) Frailty & Nutritional Status that can be assessed by the history of weight loss and weakness. It is important as it predicts,

- Poor healing
- Increased mortality

Thus, thorough past history is important as it significantly influences **perioperative care**.

3. Why assessment of consciousness and orientation is important in geriatrics age group in preoperative evaluation of patient undergoing TURP surgery?

Assessment of consciousness and orientation is emphasized as a key part of the Geriatric Preoperative Evaluation (GPE) because it directly reflects baseline brain function and helps predict perioperative risk.

Elderly patients often have subclinical cognitive impairment or early dementia. Knowing baseline consciousness and orientation helps in differentiating pre-existing cognitive dysfunction from new postoperative changes especially in cases like TURP surgery where patients are at risk of occurrence of TURP syndrome which may present as confusion, convulsions, impaired consciousness and coma.

4. What are the age-related effects on various body systems?

Aging is associated with anatomical and physiological



changes affecting all body systems.

1. Central Nervous System

- 1) Decreased brain mass and neuronal density
- 2) Decreased number of receptors (acetylcholine, Dopamine, Noradrenaline, 5- Hydroxytryptamine)
- 3) Increased cerebral blood flow
- 4) Increased sensitivity to anaesthetic agents (MAC ↓ ~6% per decade after 40 yrs)
- 5) Increased risk of postoperative delirium & cognitive dysfunction
- 6) Impaired thermoregulation

2. Cardiovascular System:

- 1) Decreased arterial compliance leading to increased systolic BP
- 2) Decreased β -adrenergic responsiveness
- 3) Decreased maximum heart rate & cardiac reserve
- 4) LV hypertrophy and diastolic dysfunction is common
- 5) Cardiac output is maintained at rest but decreases with stress
- 6) Increased risk of hypotension during anaesthesia
- 7) Conduction abnormalities (increased risk of arrhythmias)

3. Respiratory System

- 1) Decreased chest wall compliance, increased chest rigidity
- 2) Decreased lung elasticity leading to decreased residual volume (RV), decreased vital capacity (VC)
- 3) Closing capacity is more than FRC leading to airway closure
- 4) Decreased PaO_2 (age-related hypoxemia)
- 5) Blunted response to hypoxia & hypercapnia
- 6) Increased risk of atelectasis and postoperative pulmonary complications.

4. Renal System

- 1) Decreased renal blood flow (~1% per year after 40)
- 2) Decreased GFR and creatinine clearance
- 3) Serum creatinine may appear normal due to decreased muscle mass
- 4) Impaired fluid & electrolyte regulation
- 5) Increased sensitivity to nephrotoxic drugs.

5. Hepatic System

- 1) Decreased hepatic blood flow
- 2) Mild decreased in liver mass
- 3) Decreased Phase I metabolism

(oxidation/reduction)

- 4) Phase II (conjugation) relatively preserved
- Decreased First-pass metabolism → Increased bioavailability of some drugs.

6. Musculoskeletal System

- 1) Decreased muscle mass (sarcopenia)
- 2) Decreased bone density leading to osteoporosis
- 3) Joint stiffness
- 4) Increased risk of fractures and positioning injuries

7. Endocrine System

- 1) Decreased basal metabolic rate
- 2) Impaired glucose tolerance
- 3) Decreased renin & aldosterone causing impaired fluid balance

8. Thermoregulation

Impaired temperature regulation in turn causing increased risk of hypothermia during anaesthesia.

5. Why is airway examination important in elderly?

Airway examination becomes especially important in older adults, because aging introduces predictable anatomical and physiological changes that can make airway management more difficult and riskier.

1. Age-related anatomical changes

1) Loss of teeth (edentulous mouth)

Poor mask seal

Difficult bag-mask ventilation.

2) Decreased cervical spine mobility (arthritis, spondylosis)

Leading to difficulty in head extension and suboptimal positioning for laryngoscopy

3) Reduced temporomandibular joint (TMJ) movement

Causing limited mouth opening and difficult laryngoscope insertion.

4) Increased pharyngeal soft tissue redundancy

Leads to airway narrowing and increased risk of airway obstruction.

2. Physiological vulnerability

1) Older patients have reduced airway reflexes and hence higher risk of aspiration.

2) Decreased respiratory reserve (lower lung compliance, weaker muscles) causes faster desaturation during apnea, hence less time for laryngoscopy and intubation.



3) Increased sensitivity to anaesthetic drugs causing airway obstruction or hypoventilation at low doses of anaesthetic agents

3. Higher incidence of difficult airway including mask ventilation, laryngoscopy and intubation

Increased prevalence of **predictors of difficult intubation** like:

- Limited neck extension
- Poor dentition
- Reduced mouth opening

Previously normal airway may become difficult with advancing age.

4. Increased risk of complications like:

- 1) Trauma to fragile tissues (gums, mucosa)
- 2) Dental injury
- 3) Postoperative airway obstruction
- 4) Aspiration pneumonia

5. Need for planning and backup

Thorough airway assessment is essential as it helps anticipate difficult airway

6. What are the challenges in old age in central neuraxial blockade with respect to anesthesia?

Aging produces several anatomic and physiologic changes that make **central neuraxial blockade (spinal/epidural)** more challenging and unpredictable in geriatric patients

1. Positioning issues

Presence of arthritis, pain, limited mobility causes difficulty in achieving optimal flexion for spinal/epidural.

2. Technical difficulty (anatomical changes) may be present due to following reasons:

- a) Degenerative spine changes, Osteophytes, calcified ligaments, narrowed intervertebral spaces may be present.
- b) Kyphosis/scoliosis → distorted landmarks
- c) Ligamentum flavum calcification → difficult needle passage
- d) As tissues are fragile, there is risk of traumatic needle insertion

All the above may lead to difficult positioning and multiple attempts at spinal and epidural anaesthesia.

3. Altered spread of local anesthetic

Decreased CSF volume in elderly causes greater cephalad spread.

Narrow epidural space is present resulting in higher-than-expected block level with standard doses. There is also increased risk of high spinal anesthesia in old age.

4. Increased sensitivity to local anesthetics:

Because of age related neural changes, there is enhanced effect of local anaesthetic agents. Hence profound motor block may occur. Also excessive sympathetic block may result as the sympathetic system is already suppressed due to old age. Poor compensation to vasodilation may result in exaggerated haemodynamic instability.

5. Slower onset but prolonged duration

Reduced nerve conduction and decreased drug clearance leads to longer duration of block and delayed recovery.

6. Higher risk of complications

Frequency of complications like hypotension, urinary retention, postoperative cognitive issues like postoperative delirium is more in old age group.

7. What are the challenges in GA in geriatrics?

1. Difficulty in intubation and mask ventilation:

The technique of mask ventilation and intubation can be challenging in old patients due to loss of buccal pad of fat, loss of teeth, decreased neck movements and restricted mouth opening.

2. Early desaturation during GA:

Due to aging, there is increased closing capacity and decreased FRC. Also, there is blunting of response to hypoxia and hypercapnia. All these results in rapid desaturation during laryngoscopy and intubation.

3. Altered pharmacokinetics & pharmacodynamics

Decreased hepatic metabolism and decreased renal clearance contribute to prolonged action of anaesthesia drugs thereby leading to delayed recovery.

4. Cardiovascular instability

These patients may have decreased beta receptor responsiveness, baroreceptor reflex and decreased blood vessel compliance leading to poor compensation to hypovolemia or vasodilation.



Marked hypotension may occur during general anaesthesia following administration of induction agents. There is also increased risk of myocardial ischemia. (Revised cardiac risk index)

5. CNS vulnerability

There is increased sensitivity to sedatives and decreased MAC requirement in old age. (MAC requirement decreases ~6% per decade). There is higher risk of delayed emergence, postoperative delirium and cognitive dysfunction in these patients.

6. Impaired thermoregulation

Reduced ability to maintain body temperature leads to hypothermia during GA, delayed drug metabolism causing delayed recovery from GA. Hypothermia causes increased risk of coagulopathy which may contribute to increased blood loss during surgery.

7. Renal dysfunction

Decreased GFR and renal blood flow lead to drug accumulation, fluid overload and electrolyte imbalance.

8. Positioning and pressure injuries

Fragile skin, osteoporosis may be present. Nerve injuries and pressure sores may occur due to frailty.

9. Polypharmacy & drug interactions

Multiple medications (antihypertensives, anticoagulants, etc.) leads to increased risk of drug interactions and perioperative complications.

10. Reduced physiological reserve

All organ systems have limited ability to compensate. Even minor stress can cause major hemodynamic or metabolic instability.

1. What are the effects of anesthetic agents on old age?

1. Central Nervous System

- ↑ Sensitivity to anaesthetic agents
- ↓ MAC of inhalational agents (~6% per decade after 40 yrs)
- ↓ Dose requirement for most drugs
- ↑ Risk of postoperative delirium & cognitive dysfunction.

2. Intravenous Anaesthetic Agents

↓ Dose requirement, slower redistribution and

prolonged drug action

• Propofol:

- Dose ↓ (~50% less required)
- ↑ Risk of hypotension & apnea.

• Benzodiazepines (e.g., midazolam):

- ↑ Volume of distribution → prolonged half-life
- Dose ↓ (~50%)
- ↑ Sedation

• Opioids:

- ↑ Sensitivity
- ↓ Dose requirement
- ↑ Risk of respiratory depression.

3. Inhalational Agents

- ↓ MAC → reduced requirement
- Faster induction
- Slower recovery (↑ fat stores)
- ↑ Respiratory depression

4. Neuromuscular Blocking Agents

Nondepolarizing agents:

Onset may be delayed (↓ cardiac output) and duration may be prolonged:

- Renal drugs (pancuronium) → ↓ excretion
- Hepatic drugs (rocuronium, vecuronium) → ↓ metabolism

5. Cardiovascular Effects

- ↑ Sensitivity to myocardial depression
- ↓ Baroreceptor response
- ↑ Risk of hypotension

6. Respiratory Effects

- ↑ Respiratory depression
- ↓ Response to hypoxia & hypercapnia
- ↑ Risk of apnea

7. Pharmacokinetic Changes

- ↑ Body fat → ↑ Vd of lipid-soluble drugs
- ↓ Hepatic blood flow → ↓ metabolism
- ↓ Renal function → ↓ drug excretion
- ↓ Plasma proteins → ↑ free drug

8. Pharmacodynamic Changes

- Enhanced drug effect at lower concentrations
- Altered receptor sensitivity



9. What are the challenges with respect to monitoring in geriatrics?

Monitoring an elderly patient after anesthesia is more challenging than in younger adults because physiological reserves are reduced, responses are blunted, and complications are often subtle.

1. Attenuated Physiologic Responses

- Aging leads to reduced **β -adrenergic responsiveness**
- Tachycardia and BP changes may be **blunted despite hypovolemia or hypoxia**
- Hence, routine monitors may **fail to detect early deterioration**.

2. Increased Variability in Baseline Values

Wide interpatient variability due to:

- Hypertension
- Coronary artery disease
- Chronic lung disease

“Normal” values differ → **difficulty in interpretation of monitored parameters.**

3. Reduced Organ Reserve

Decline in:

- Cardiovascular reserve
- Pulmonary compliance
- Renal function

Small physiologic disturbances can cause rapid instability, requiring vigilant monitoring.

4. Impaired Thermoregulation

- Reduced ability to maintain temperature
- High risk of **perioperative hypothermia**
- Continuous temperature monitoring is essential but often underestimated.

5. Altered Pharmacodynamics and Pharmacokinetics

- Increased sensitivity to anesthetic agents
- Delayed drug clearance
- Monitoring depth of anesthesia and recovery becomes difficult
- Higher risk of drug accumulation and delayed emergence.

6. Limitations of Standard Monitoring Techniques

- **Pulse oximetry:**
- Affected by poor peripheral perfusion and vascular

disease

• **Non-invasive BP:**

- Less reliable due to arterial stiffness
- May require invasive monitoring (arterial line, CVP) more frequently.

7. Increased Risk of Cerebral Dysfunction

Higher incidence of:

- Postoperative delirium
 - Cognitive dysfunction
- Requires frequent neurological monitoring (consciousness, orientation)

8. Positioning and Skin Integrity

- Fragile skin and reduced tissue perfusion
- Increased risk of pressure injuries
- Monitoring must include positioning and padding assessment.

10. What is Post operative delirium?

It is an acute, fluctuating disturbance of consciousness and cognition that occurs after surgery, typically within the first few days characterized by:

1. Reduced awareness of the environment
2. Impaired attention
3. Disorganized thinking
4. Altered level of consciousness

11. Why post-operative delirium is common in old age?

Delirium is common in old age because aging reduces the brain's ability to tolerate stress and increased exposure to multiple triggers.

1. Reduced brain reserve

With increasing age, brain becomes less able to compensate for stress.

- Even minor problems—like a mild infection or dehydration—can disrupt normal thinking and attention.
- This is sometimes described as decreased “cognitive reserve.”

2. Higher prevalence of underlying conditions

Older adults are more likely to have illnesses such as Dementia, Parkinson's disease, or Stroke. These conditions already affect brain function, making it easier for delirium to develop.

3. Increased sensitivity to medications



Many common medications—especially sedatives, painkillers, or anticholinergic drugs—can trigger confusion or delirium in older adults, even at standard doses.

4. More frequent medical stressors

- Infections (like urinary or respiratory infections)
- Surgery or hospitalization
- Electrolyte imbalances
- Poor nutrition or dehydration

5. Sensory impairment

Hearing and vision problems are common with age.

When the brain receives less accurate sensory input, it can misinterpret the environment, increasing confusion and risk of delirium.

6. Sleep disruption

Hospital stays, illness, and aging itself often disturb sleep patterns.

Poor sleep can significantly worsen attention and awareness, contributing to delirium.

7. Frailty and overall vulnerability

Older adults often have reduced physical reserves.

This means even small physiological stresses can tip the balance and lead to acute confusion.

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14th JUNE 2026
03:10PM - 03:40PM

ANAESTHETIC MANAGEMENT OF PATIENT WITH CKD POSTED FOR RENAL TRANSPLANTATION

Moderators:

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A 40 year old male, diagnosed with CKD stage 5 on thrice weekly MHD, known hypertension, now electively planned for allogenic living donor renal transplant surgery

What is the definition of End Stage renal disease (ESRD)?

- ESRD refers to the final progression of CKD, when renal function is irreversibly impaired and the development of uremia is imminent
- Clinical syndrome characterized by renal dysfunction that would prove fatal without Renal Replacement Therapy
- GFR <15 ml/min/1.73m²

How do you Estimate of GFR?

- Endogenous filtration marker-S.creatinine and GFR estimating equation
- Modification of Diet in Renal Disease study equation
- Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine equation
- Cockcroft-Gault Formula: $CrCl = \{((140 - \text{age}) \times \text{weight}) / (72 \times SCr)\} \times 0.85$ (if female)

What are the markers of CKD?

- Albuminuria AER ≥ 30 mg/ 24 hrs or ACR ≥ 30mg /g
- Urine sediment abnormalities- WBC, RBC, Granular cast
- Electrolyte abnormalities due to tubular disorder
- Abnormality detected by HISTOLOGY
- Structural Abnormality detected by IMAGING

What are the classification of CKD?

Kidney Disease: Improving Global Outcomes (KDIGO)

GFR categories(ml/min/1.73 m ²)			Persistent albuminuria categories		
Description		Range	*Albumin to Creatinine Ratio		
G1	Normal or high	>90	A1	A2	A3
G2	Mildly decreased	60-89	Normal to mildly increased	Moderately increased	Severely Increased
G3a	Mildly to moderately decreased	45-59			
G3b	Moderately to severely decreased	30-44	<30 mg/g* <3mg/mm ^{ol}	30-300 mg/g* 3-30 mg/mm ^{ol}	>300 mg/g* >30mg/mm ^{ol}
G4	Severely decreased	15-29			
G5	Kidney failure	<15			



What are the causes of CKD?

Relating to comorbidities:

Vascular disease including renal vascular stenosis Hypertension **Type 2 diabetes mellitus- Commonest cause**

Intrinsic renal disease:

Glomerulonephritis, Acute kidney injury, Interstitial nephritis Nephropathies: Infective (e.g. pyelonephritis) Obstructive (e.g. prostatic hypertrophy, nephrolithiasis) Reflux

Genetic: Polycystic kidney disease, Alport syndrome, Fabry disease, Cystinosis

Metabolic:

Hypercalcaemia
Hyperparathyroidism causing nephrocalcinosis
Oxalosis

Systemic disease:

Amyloidosis

Autoimmune disease:

Goodpasture syndrome, Scleroderma
IgA vasculitis, Systemic lupus erythematosus

Neoplasm: Myeloma, Renal tumour

Toxins: Lead poisoning, Drug-related NSAID use
nephropathy Calcineurin inhibitors,
Chemotherapeutic agents

Other Haemolytic uraemic syndrome: Gout

Obstructive uropathy

What are the Functions of Kidney?

- Acid base balance
- Fluid and electrolyte balance
- Toxin and waste product removal
- Blood pressure control
- Erythropoietin production
- Vitamin D activation

What are the systemic effects of chronic renal failure?

Significant declines in glomerular filtration rate (GFR)

and urine production occur when these critical functions are significantly damaged, resulting in the clinical manifestations of uremia.

The common problems related to ESRD are the following:

- **Electrolytes** - Volume expansion, hyponatremia, hyperkalemia, metabolic acidosis, hyperuricemia, hyperphosphatemia, hypocalcemia, hypermagnesemia.
- **Cardiovascular** - Accelerated atherosclerosis, systemic hypertension, left ventricular hypertrophy (LVH), congestive heart failure, pulmonary edema, cardiomyopathy, pericarditis, hyperdynamic circulation
- **Hematologic** - Anemia, B- and T-cell dysfunction, qualitative platelet dysfunction, bleeding diathesis
- **Musculoskeletal** - Muscle weakness
- **Neurologic** - Encephalopathy, loss of memory, seizures, peripheral neuropathy, myoclonus, asterixis
- **Gastrointestinal** - Gastroparesis, uremic gastroenteritis, nausea, vomiting, peptic ulcer disease, gastrointestinal bleeding, pancreatitis
- **Endocrine**. Vitamin D deficiency, secondary hyperparathyroidism, carbohydrate intolerance, hypertriglyceridemia

What are the electrolyte abnormalities?

Electrolyte Abnormalities:

- Hyponatremia
- Hyperkalemia
- Hypocalcemia
- Hypermagnesemia
- Hyperphosphatemia
- Metabolic acidosis

Sodium: In a normal individual more than 25,000 mmol of sodium ions are filtered daily with less than 1% being excreted. CRF can therefore be associated with **sodium retention, sodium wasting or normal sodium balance**, and is further influenced by factors such as diuretic use and cardiac function. Most patients however demonstrate a mild degree of sodium and water retention whilst the extracellular fluid volume remains



isotonic. Ironically, the patient with CRF also has impaired renal concentrating mechanisms and thus extrarenal fluid losses such as vomiting, diarrhoea or pyrexia may rapidly cause hypovolaemia and hypotension.

Potassium in CKD and hemodialysis?

Potassium is the principal cation of the intracellular fluid (ICF), where its concentration is 150 mEq per L. The concentration of K⁺ in the extracellular fluid (ECF) and plasma is lower, between 3.5 and 5.0 mEq per L. Adults ingest approximately 50 to 100 mEq of potassium daily and excrete an equal quantity in the urine. **Chronic hyperkalemia is usually caused by impaired renal potassium excretion.**

In patients with end-stage renal disease (ESRD) or transplant recipients, hyperkalemia can be exacerbated by **excessive dietary intake**.

Other causes being:

- hemolysis, hemorrhage, massive blood transfusion, metabolic acidosis,
- β -adrenergic blockade, angiotensin-converting enzyme inhibitors,
- angiotensin receptor blockers, insulin deficiency/resistance,
- hyperglycemia, rhabdomyolysis, hyperosmolality,
- succinylcholine, digoxin overdose, potassium sparing diuretics, Cyclosporine.

ECG findings of hyperkalemia?

- Peaked T waves,
- Flattened P waves, lengthened PR interval,
- Disappearance of P waves,
- A widened QRS complex that can progress to a "sine wave", ventricular asystole or ventricular fibrillation.

Treatment of hyperkalemia

The most important organ at risk in hyperkalemia is the heart.

- Antagonising the cardiac toxic effects and stabilising the cardiac membrane with IV calcium – 10% calcium chloride/calcium gluconate.

Shifting the extracellular potassium to the intracellular compartment

- Glucose insulin infusion-50 ml of 50% glucose with

10 units of insulin.

- Sodium bicarbonate 1 meq/kg body weight.
- IV furosemide-20-40mg IV push.
- Potassium binding resins.
- Peritoneal/haemodialysis
- Hyperventilation in mechanically ventilated patients
- Inhaled beta agonists

Problems of hypermagnesemia?

- Hypermagnesemia can accompany chronic renal failure, especially when the GFR decreases to less than 10 mL per minute. High blood magnesium levels cause dose-dependent neuromuscular (NM) toxicity.
- Symptoms include diminished deep tendon reflexes, flaccid paralysis which may progress to respiratory failure.
- An increased sensitivity to depolarizing and nondepolarizing NM agents is noted. Bradycardia, hypotension, and heart block are due to the calcium channel blocking effects of magnesium.
- The acute toxicity can be antagonized by intravenous infusion of calcium. Haemodialysis or peritoneal dialysis can normalize blood magnesium levels.

Calcium, phosphate, parathormone and renal osteodystrophy

- Total Plasma calcium concentration is reduced in CRF. Renal production of calcitriol (1,25-(OH)₂D₃) declines causing decreased intestinal absorption of calcium.
- Phosphate excretion is impaired as GFR falls below 20ml/min and hyperphosphataemia develops. As phosphate levels increase, calcium phosphate is deposited in soft tissues such as skin and blood vessels further lowering plasma calcium concentration.
- Hyperphosphataemia also has a negative effect on 1- α -hydroxylase the enzyme responsible for renal calcitriol production.
- Both hypocalcaemia and hyperphosphataemia are potent stimuli to parathormone secretion, leading to hyperplasia of the parathyroid gland, and secondary hyperparathyroidism.
- This causes increased osteoclast and osteoblast activity causing osteitis fibrosa cystica. Patients



usually tolerate hypocalcaemia remarkably well, as long as oral calcitriol is prescribed and calcium carbonate is used both as an intestinal phosphate binder and a source of calcium.

Acidosis–

- Chronic metabolic acidosis is a common feature of ESRD.
- The inability to secrete protons or buffers such as phosphate, or to regenerate bicarbonate limits the clearance of hydrogen ions. Furthermore reduction in glutamine utilisation reduces ammonia production and secretion into the proximal tubule.
- Retention of organic anions causes a progressive increase in the anion gap and a further fall in plasma bicarbonate concentration.
- Although plasma bicarbonate concentrations rarely fall below 12-15mmol/l there is little reserve to counter acute acidosis caused by ketoacidosis or sepsis

Does pH > 7.2 require correction?

As long as the pH is above 7.2, it does not require correction

What are the adverse effects of overt bicarbonate correction?

- Metabolic acidosis with pH <7.2 is treated with HCO₃ replacement.
- HCO₃ replacement = $0.3 \times \text{weight in kg} \times (24 - \text{serum HCO}_3)$
- Only half of the calculated bicarbonate is given because doses exceeding 1mEq/kg/minute may lead to alkaline overshoot. Serial blood gases should be followed to monitor the response to treatment.
- Sodium bicarbonate is a hyperosmolar solution (8.4% preparation contains 1000 mmols /L). It can draw fluid from ICF and can increase the ECF volume producing pulmonary oedema in susceptible patients.
- As it is a hypertonic solution, can produce intraventricular haemorrhage in neonates. Since it can release CO₂ which easily can enter the cells which can exacerbate the intracellular acidosis and can produce more cellular damage.

• Extra cellular alkalosis can shift potassium in to the intracellular compartment producing sudden decrease in extracellular potassium levels leading to fatal ventricular dysrhythmias.

• Extra cellular alkalosis can also shift the oxyhaemoglobin dissociation curve to the left decreasing the release of O₂ to the tissues thereby exacerbating tissue hypoxia. For each 0.1 increase in pH, oxygen availability may decrease by 10%

What are the causes of anaemia in CKD?

A normochromic normocytic anemia is a common finding in CRF, causes:

- Decreased renal erythropoietin production reduces stem cell transformation into erythrocytes
- Uraemic toxins reduce red cell life
- Chronic upper GI tract losses and those from dialysis
- Dietary deficiency in iron and folate also occurs

How is anemia treated in CKD?

The Hb % should be raised to 11-12gm% to optimise the tissue oxygenation.

Further rise will increase viscosity –

- Worsening hypertension
- Thrombosis of AV fistula
- Increased risk of CVA 5% of patients may develop convulsions

Anemia can be treated with recombinant human erythropoietin or with darbopoietin α . Anemia is responsive to treatment with erythropoietin in all stages of CKD.

Recombinant erythropoietin

- Starting dosage 50-150 U/kg/wk IV or SC (once, twice, or three times per week)
- Target hemoglobin 11-12 g/dL
- Optimal rate of correction : Increase hemoglobin by 1-2 g/dL

What are the compensatory mechanisms due to anemia?

- Increased cardiac output.
- Rightward shift of the ODC due to increase in 2,3 DPG



- Decreased viscosity increasing microcirculation.
- Precautions –
- IPPV and central neuraxial block may decrease cardiac output.
- Hyperventilation may shift the curve to the left.
- **Blood transfusion avoided to prevent alloimmunisation** especially if patient later planning transplant.
- **Washed red blood cells (leukocyte poor) that are irradiated and cytomegalovirus (CMV) negative preferred.** This will help decrease leukocyte antigen exposure that can induce the development of alloantibodies predisposing to rejection of the implanted kidney

Why do CKD patients have a high tendency to bleed during surgery? How do you treat?

- Patients with CRF have a tendency to excess bleeding in the perioperative period. Standard tests of coagulation are usually normal (prothrombin time/INR, activated partial thromboplastin time) and platelet count is within normal limits.
- Platelet activity is however abnormal with decreased adhesiveness and aggregation, probably caused by inadequate vascular endothelial release of a von Willebrand factor/factor VIII complex, which binds to and activates platelets
- Increased platelet release of β -thromboglobulin
- Increased vascular production of PGI₂
- Excessive nitric oxide (NO) production
- Bleeding time may be prolonged beyond normal.

Clinical implication:

- Platelet dysfunction is not corrected by platelet transfusion, but if operative bleeding occurs, it can be improved by dialysis
- Cryoprecipitate may enhance platelet aggregation by increasing factor VIII: von Willebrand multimers and/or fibrinogen level

- DDAVP(which enhances release of von Willebrand factor). DDAVP
- (0.3mcg/kg) is effective within 1-2 hours but has a short duration of only 6-8 hours, and is subject to tachyphylaxis
- Intravenous conjugated oestrogens have a slower onset but a longer duration of action (5-7 days).
- Protamine, if indicated to reverse heparin effect – If heparin was recently used, administer an appropriate dose of protamine
- If available, obtain specific tests of platelet function (eg, platelet aggregometry, PFA-100) and point-of-care tests of coagulation and platelet function (eg, thromboelastography [TEG], rotational thromboelastometry [ROTEM]).

What the gastrointestinal manifestations of CKD?

- Gastrointestinal signs and symptoms including nausea, vomiting, abdominal pain, and abnormal gastric motility occur in ESRD patients.
- Using electrogastronomy, impairments in gastric myoelectrical activity exist in patients with symptomatic ESRD as compared to patients with normal renal function.
- Patients with ESRD have delayed gastric emptying, regardless of the timing of their last oral intake. The presence of diabetes, obesity, or both can further impair gastric emptying.

Clinical implications:

Considered full stomach
Aspiration prophylaxis prior surgery
Rapid sequence intubation

What are the cardiovascular manifestations of CKD?

- **Cardiovascular abnormalities** are very common in CRF, and represent 48% of the causes of death in these patients.
- **Systemic hypertension** is the most common with an incidence approaching 80%, although it is often not a feature of sodium-wasting nephropathies such as polycystic kidney disease or papillary necrosis.



- Plasma volume expansion resulting from sodium and water retention is the most frequent cause of hypertension, and may be significantly improved by dialysis.
- Some patients may require beta-blockers, ACE inhibitors, alpha-blockers and vasodilators to adequately control their blood pressure. Alteration in the control of renin and angiotensin secretion may also contribute to hypertension in 30% of patients.

Ischaemic heart disease (IHD) is a frequent cause of mortality in patients with CRF.

Accelerated atherosclerosis results from decreased plasma triglyceride clearance, hypertension and fluid overload causing left ventricular hypertrophy and failure. The elevation in plasma triglyceride levels is caused by a defect in lipoprotein lipase activity and reduced lipolysis.

Metastatic calcific valvular heart lesions : increased incidence. Aortic valve calcification occurs in up to 55% of patients, with aortic stenosis being present in 13%. Mitral valve calcification occurs in 40% (stenosis 11%).

Elevation in the calcium/phosphate product and parathyroid hormone concentrations are the main cause. As a result of these lesions, bacterial endocarditis is much more common in dialysis patients than the normal population.

Haemorrhagic **uraemic pericarditis** was often seen prior to the advent of effective dialysis, but is now uncommon and occurs in patients on inadequate dialysis regimen. If untreated, it may progress to pericardial tamponade with hypotension, elevated jugular venous pressure and signs of falling cardiac output. Pericardectomy may be required but should be reserved for those who fail to improve with immediate dialysis.

Sudden death from acute cardiac arrhythmias is frequent and related to both IHD and electrolyte abnormalities. As GFR decreases, the risk of cardiac mortality increases.

Following successful kidney transplantation, cardiovascular disease remains the most common cause of death. Kidney transplant recipients are at increased risk for the development of myocardial infarction (MI), congestive heart failure, and atrial

fibrillation

Clinical implications:

- Prone for perioperative major cardiac events.
- According to ACC/AHA PERIOPERATIVE GUIDELINES, creatinine $\geq 2\text{mg/dl}$ has risk for perioperative major cardiac event. (MI, pulmonary edema, ventricular fibrillation, primary cardiac arrest, complete heart block)
- Undergoing suprainguinal vascular, intraperitoneal or intrathoracic surgery another risk factor in these patients
- Preoperative optimisation with cardiology consultation

What is cardio renal syndrome?

The **cardiorenal syndrome** is defined by an interconnection between the renal and cardiac systems, where the decline of one organ influences the decline of the other. There is evidence that correction of renal function by renal transplantation can improve systolic dysfunction and reverse left ventricular dilation and hypertrophy

The different interactions that can occur led to the following classification of

CRS that was proposed by Ronco and colleagues :

- Type 1 (acute) – Acute Heart failure results in acute kidney injury (previously called acute renal failure).
- Type 2 – Chronic cardiac dysfunction (eg: chronic Heart failure) causes progressive chronic kidney disease (CKD, previously called chronic renal failure).
- Type 3 – Abrupt and primary worsening of kidney function for example, due to renal ischemia or glomerulonephritis causes acute cardiac dysfunction, which may be manifested by Heart failure.
- Type 4 – Primary CKD contributes to cardiac dysfunction, which may be manifested by coronary disease, Heart failure, or arrhythmia.
- Type 5 (secondary) – Acute or chronic systemic disorders (eg: sepsis or DM) that cause both cardiac and renal dysfunction.



Preoperative investigations:

- ECG: LVH, MI, Arrhythmia
- ECHO: LVH, RWMA, Diastolic dysfunction, valvular lesions, pericardial fluid
- Dobutamine stress ECHO, Coronary angiography

Intraoperative:

- Morning dose antihypertensive agents taken except ARB blockers, ACE inhibitors
- 5 lead ECG with ST-T analysis
- Invasive arterial monitoring if severe LV dysfunction or major fluid shifts expected

What the neurological manifestations of CKD?

Neurological abnormalities

Many patients with CRF have abnormalities in central (CNS) and peripheral nervous system function due to uncleared nitrogen atoms. CNS changes have a wide spectrum from mild personality alterations to asterixis, myoclonus, encephalopathy and convulsions. Peripheral neuropathy is common in advanced

stages of the disease and is initially a distal “glove and stocking” sensory loss progressing later to motor changes. Both dialysis and renal transplantation may improve the neuropathy.

Clinical implication:

The presence of a peripheral neuropathy should alert the anaesthetist to the presence of an autonomic neuropathy with delayed gastric emptying, postural hypotension and silent myocardial ischaemia

Caution during regional anaesthesia, neuropathy to be documented

Two types of neurological disturbances are unique to patients on dialysis

Dialysis dementia with dyspraxia, myoclonus and dementia occurs in patients on dialysis for many years and may be related to aluminium toxicity.

The dialysis disequilibrium syndrome is associated with rapid initial reduction in plasma urea levels at the commencement of dialysis. Urea transport from brain cells is slowed in CRF, leading to large concentration gradient, leading to reverse osmosis.

What are the pulmonary manifestations of CKD?

Pulmonary complications are common in patients with CRF in the postoperative period. Fluid overload, malnutrition, anaemia, impaired humoral and cellular immune function and decreased surfactant production predispose patients to atelectasis and infection. The pulmonary vasculature is also affected by chronic renal failure.

A unique “*lowpressure*” *pulmonary edema* can result from increased permeability of alveolar capillary membranes. Pulmonary congestion and edema may occur in the absence of volume overload and is associated with normal or mildly elevated intracardiac and pulmonary wedge pressures. The chest radiograph is characterized by a “butterfly wing” pattern. This entity responds promptly to dialysis.

Pulmonary hypertension seen in 40% patients ESRD receiving dialysis. Mechanism involves uraemia induced vasoconstriction and increased cardiac output due to AV fistula. It may be reversible post transplant.

CLINICAL IMPLICATION: decreased survival with PHT. Right sided catheterisation important if significant PHT in preoperative ECHO

Pharmacokinetics & CKD

How are anesthetic drug metabolism affected?

- The volume of distribution is usually decreased, but may be increased if there is fluid retention.
- Hypoalbuminaemia and acidosis increase the free drug availability of highly protein bound drugs.
- Uremia can also disrupt the blood–brain barrier.
- Metabolism and renal excretion impaired, so prolonged duration of Action

Induction drugs:

- The doses of benzodiazepines and thiopentone may need to be reduced by 30% - 50%.
- Although the pharmacodynamics of propofol are unchanged in CRF and the metabolites lack sedative activity, changes in volume of distribution and mental state mean that a



reduction in induction dose may also be appropriate. Regardless of blood volume status, these patients often respond to induction of anesthesia as if they were hypovolemic.

- The sympathetic nervous system response may be attenuated by antihypertensive therapy or diabetic autonomic neuropathy. A blunted sympathetic response impairs compensatory peripheral vasoconstriction.
- Some studies have noted higher propofol dose requirement in patients with end-stage renal disease (ESRD) compared to normal patients. Renal failure patients required a higher dose of propofol to reach the clinical end point of hypnosis and bispectral index (BIS) of 50. The hyperdynamic circulation from uremia and higher plasma volume from anemia can counteract the effect of low serum albumin explaining the higher dose requirement.
- Ketamine pharmacokinetics is not significantly changed by renal disease, but its hypertensive effects make it undesirable in patient with underlying hypertension.
- Etomidate is well tolerated and preserves hemodynamic stability.

Midazolam: the dose should be reduced and titrated according to the effect. The elimination of midazolam and its main metabolite, α 1-hydroxymidazolam, is reduced in patients with ESRD

Opioids:

Most lipid soluble analgesics are metabolised by the liver to water soluble metabolites for renal excretion

Morphine – Morphine is avoided in ESRD patients because excessive and prolonged opioid effects may occur due to accumulation of morphine's active metabolites (eg, morphine-6- glucuronide [M6G], an opioid agonist that is 10 times more potent than morphine; and morphine glucuronide [M3G], a mild opioid antagonist) with a risk of sedation and respiratory depression . Also, severe opioid side effects can occur many hours after morphine administration due to accumulation of M6G and its slow equilibration between plasma and the central nervous system

Fentanyl is predominantly metabolized in the liver to norfentanyl, an inactive metabolite. Fentanyl has a short redistribution phase and unchanged free fraction. It has been used in patients with renal failure with no adverse effects, although there are reports of accumulation of the parent drug. **So time interval for bolus doses in PCA needs to be increased for opioids**

Sufentanil is approximately 5 to 10 times more potent than its parent drug fentanyl. Although no significant difference in half-life and clearance of sufentanil has been found between patients with chronic renal failure and healthy controls, there is more variability in patients with renal failure. **Alfentanil** can be used as normal.

Remifentanyl is rapidly broken down by nonspecific plasma and tissue esterases. Since these esterases are abundant, remifentanyl does not accumulate regardless of the duration of administration. It has a predictable offset of action (ie, it has a constant contextsensitive half-time)

Tramadol – Tramadol is used cautiously in ESRD patients, with adjustments in dose and interval between doses. The dose preferred is 50 percent of the usual adult dose, administered at the usual interval.

Buprenorphine – Transdermal buprenorphine has been used for treatment of chronic pain in patients with ESRD Analgesic therapy is stable in patients on dialysis; plasma levels of buprenorphine and its metabolite norbuprenorphine are not elevated, and hemodialysis does not affect plasma levels

Meperidine – Meperidine is avoided in ESRD patients because its renally excreted active metabolite, normeperidine, will accumulate and may cause respiratory depression as well as adverse neuroexcitatory effects (eg, myoclonic activity and/or seizures)

Codeine – Codeine is avoided in ESRD patients. Codeine is metabolized in the liver to codeine-6-glucuronide, norcodeine, and morphine (which is subsequently metabolized to M3G and M6G).

Inhalational agents:

Fluoride ions:

The elimination of most volatile anaesthetic agents is not dependent on renal function and their activity is



unaffected by CRF. The hepatic metabolism of both enflurane and sevoflurane will theoretically produce nephrotoxic fluoride ions and their use should be discouraged for prolonged durations. Metabolism of halothane produces fluoride ions when the liver is hypoxic but has been used safely in patients with renal disease

Typical peak fluoride concentrations after 2 to 3 MAC-hours -

- Sevoflurane : 20 to 30 μM ,
- Isoflurane and Desflurane: $\leq 5 \mu\text{M}$
- Enflurane $\geq 20 \mu\text{M}$

However, none of these anesthetics is associated with clinically significant renal toxicity, suggesting that methoxyflurane is unique in its ability to harm kidneys.

Compound A:

Patients given sevoflurane anesthesia are routinely exposed to compound A in rebreathing circuits. It causes proximal renal tubular acidosis in rats. The inhaled concentration is dependent on the fresh gas flow rate and the type of CO_2 absorbent present.

Fresh gas flows of 1 L/min result in maximum compound A concentrations of approximately 20 ppm with soda lime and 30 ppm with Baralyme. Higher FGF rates result in a smaller accumulation of compound A in the breathing circuit. However, compound A exposure is not associated with clinically significant nephrotoxicity in humans. There is no threshold exposure level known to cause more than subclinical renal damage.

Compound A exposure can be limited by careful selection of fresh gas flows, vaporizer output, and CO_2 absorbent materials.

The use of 2 L/min fresh gas flows ensures that for the vast majority of patients, exposure to compound A will be below the most conservative threshold for nephrotoxicity.

Although clinical studies indicate that sevoflurane is most likely safe even in patients with preexisting renal dysfunction, the drug should be administered in accordance with the approved package labeling guidelines.

The US Food and Drug Administration (FDA) recommends using fresh gas flows $>2 \text{ L/min}$ to administer sevoflurane, or limiting exposure to two hours when fresh gas flow rates are 1 to 2 L/min. Fresh

gas flows $<1 \text{ L/min}$ are not recommended.

Muscle relaxants

Depolarising agents: Succinylcholine –Patients with ESRD have reduced levels of plasma cholinesterase, the enzyme that metabolizes succinylcholine. Hence, the neuromuscular block caused by SCh may be prolonged.

Nondepolarising agents

The prolonged duration of action to nondepolarizing agents is primarily due to slow renal clearance

Mivacurium metabolism is affected by decreased activity of pseudocholinesterase. Its usual duration of action is 15 to 20 minutes, but recovery may be slower in patients with ESRD

Atracurium (0.5 mg/kg) and cisatracurium (0.15mg/kg) mainly undergo Hoffman elimination that is not altered in patients with chronic kidney disease. Renal failure may delay clearance of **laudanosine**, the principal metabolite of atracurium and cisatracurium. Laudanosine does not have effect at the neuromuscular (NM) junction, but at high plasma levels may act as a central nervous system (CNS) stimulant.

Rocuronium is primarily eliminated by direct liver uptake and excretion in bile; some is excreted renally, with reduction in clearance by 33 to 39 percent in patients with ESRD. Hence, neuromuscular blockade is somewhat prolonged after administration of rocuronium unless sugammadex is available to reverse its effect.

Clinical implications:

Initial induction

In patients requiring rapid sequence induction and intubation (RSII) due to risk of aspiration, succinylcholine (SCh) can be used safely as the neuromuscular blocking agent (NMBA) to facilitate laryngoscopy if the potassium concentration is $<5.5 \text{ mEq/L}$ and there are no electrocardiographic changes. The hyperkalemic response after SCh administration is not exaggerated in ESRD patients. As in healthy patients, a transient potassium increase of approximately 0.5 to 1 mEq/L is observed.

Maintenance:

Atracurium and cisatracurium –Drugs of Choice



atracurium (0.5 mg/kg) or cisatracurium (0.15 mg/kg)

Reversal of nondepolarizing NMBAs

Neostigmine : With typical single-dose administration at the end of surgery, neostigmine pharmacokinetics are not different from patients with normal renal function

Sugammadex – Sugammadex is a chelating agent that encapsulates rocuronium or vecuronium in order to rapidly reverse their neuromuscular blocking effect. ESRD patients, sugammadex-rocuronium or sugammadex-vecuronium complexes are retained in the body longer before eventually being excreted by the kidneys. Also, the sugammadex-rocuronium complex is removed by dialysis

What are treatment options for ESRD?

• Medical Management

1. Restrict protein intake [0.6–0.8g/kg/day]
2. Restrict salt intake [<1.5-2g/day]
3. Antihypertensives
4. Diuretics
5. Vitamin D and calcium supplementation, phosphate binders
6. Oral Bicarbonate
7. Erythropoietin
8. Statins

• Definitive treatment – RRT/ Transplant

• RRT: renal replacement therapy-

Replacement of the function of kidneys: filtration and movement of electrolytes & fluid in and out of the body

• Dialysis: Hemodialysis and Peritoneal Dialysis

• Renal Transplant - Treatment of choice

Indications of definitive treatment

- Severe metabolic acidosis
- Severe electrolyte imbalance refractory to diuretics
- Ingestion of dialyzable toxins
- Overload – Pulmonary edema refractory to diuretics
- Uremia – encephalopathy , neuropathy, bleeding, pericarditis/ pleuritis

Haemodialysis:

Access: Catheter- IVC/Femoral- Temporary/

Permacath

AVF - Brachio-Cephalic - common
Radiocephalic/ Femoral artery & Vein

A fistula will take 4-6 weeks to mature

During treatment, two needles are inserted into the vein, one to draw blood and one to return it. The orientation of the needles takes the normal flow of the blood into account. The "arterial" needle draws blood from the "upstream" location while the "venous" needle returns blood "downstream". This sequence prevents partial recycling of the same blood through the dialysis machine, which would lead to less effective treatment

Advantages of the AV fistula use:

- lower infection rates
- higher blood flow rates (which translates to more effective dialysis)
- lower incidence of thrombosis

Hemodialysis, achieves the extracorporeal removal of waste products such as creatinine and urea and free water from the blood when the kidneys are in renal failure

- The principle of hemodialysis is the same as other methods of dialysis; it involves diffusion of solutes across a semipermeable membrane. Hemodialysis utilizes counter current flow, where the dialysate is flowing in the opposite direction to blood flow in the extracorporeal circuit. Counter-current flow maintains the concentration gradient across the membrane at a maximum and increases the efficiency of the dialysis.
- Fluid removal (ultrafiltration) is achieved by altering the hydrostatic pressure of the dialysate compartment, causing free water and some dissolved solutes to move across the membrane along a created pressure gradient.
- The dialysis solution that is used may be a sterilized solution of mineral ions. The dialysis solution that is used may be a sterilized solution of mineral ions. potassium, and phosphate diffuse into the dialysis solution. However, concentrations of sodium and chloride are



similar to those of normal plasma to prevent loss. Sodium bicarbonate is added in a higher concentration than plasma to correct blood acidity. A small amount of glucose is also commonly used.

Haemofiltration

Hemofiltration, also **haemofiltration**, is a renal replacement therapy which is used in the intensive care setting. It is usually used to treat acute kidney injury (AKI), but may be of benefit in multiple organ dysfunction syndrome or sepsis. During hemofiltration, a patient's blood is passed through a set of tubing (a *filtration circuit*) via a machine to a semipermeable membrane (the *filter*) where waste products and water (collectively called *ultrafiltrate*) are removed by convection. Replacement fluid is added and the blood is returned to the patient

Principle

As in dialysis, in hemofiltration one achieves movement of solutes across a semi-permeable membrane. However, solute movement with hemofiltration is governed by convection rather than by diffusion. With haemofiltrate, dialysate is not used. Instead, a positive hydrostatic pressure drives water and solutes across the filter membrane from the blood compartment to the filtrate compartment, from which it is drained. Solute, both small and large, get dragged through the membrane at a similar rate by the flow of water that has been engendered by the hydrostatic pressure.

Peritoneal Dialysis -

Continuous Ambulatory Peritoneal Dialysis (CAPD)

The abdomen is cleaned in preparation for surgery, and a catheter is surgically inserted with one end in the abdomen and the other protruding from the skin.

The total volume is referred to as a *dwell* while the fluid itself is referred to as dialysate. The dwell can be as much as 3 litres, and medication can also be added to the fluid immediately before infusion. The dwell remains in the abdomen and waste products diffuse across the peritoneum from the underlying blood vessels.

	Advantages	Disadvantages
Hemodialysis	Short time, better solute removal	Needs heparin, vascular access, in hospital, poor BP control
PD	Steady state	Peritonitis, risk of hernia.

What are the contraindications for Renal Transplant?

Absolute	Relative
Uncontrolled malignancy Active HIV infection Life expectancy <2 years due to other illness	Age > 65 years Active infection Chronic liver disease, cirrhosis, active hepatitis Active substance abuse Active TB, ATD therapy COPD Severe diffuse atherosclerotic disease Morbid obesity Psychosocial or behavioural abnormalities



How are donors and recipients matched?

Σ ABO compatibility

Σ HLA matching

Σ CDC

Σ They also screened serologically for infections

The three sources of donor kidneys are living related donor, living unrelated donor, and deceased (brain dead) donor.

ABO blood group

The blood group antigen compatible means the recipient and donor have no mismatched ABO antigens. For example, a person with blood type A may donate to a person with blood type A or AB.

Identical means the recipient and donor have the exact same ABO

phenotype. The cadaver kidney should be ABO identical to the recipient. An A donor should be placed in an A type recipient.

A live donor kidney may be either ABO identical or compatible. For example, a donor kidney of either A, B, or O type can be placed in an AB recipient. It is possible to place an ABO incompatible organ in a recipient using protocol such as plasmapheresis and Immune absorption to overcome rejection.

The Rh system is not expressed on graft tissue.

Tissue typing. The human major histocompatibility complex is a cluster of genes on chromosome 6 that encoded human leukocyte antigens (HLA). Before any transplant, specific HLA antigens are identified in all donors and recipients.

Individuals are typed by DNA-based methods. Outcome is best with a perfect HLA matched donor and recipient. Graft survival is similar with a partial HLA matched or -mismatched kidney. The kidney with the least number of mismatched preferred. Again because of improvement in immunosuppression protocols, recipients can receive an organ from a donor with a less than perfect match.

Cross-matching. A cross-match test determines if the recipient has serum antibodies directed against donor HLA lymphocyte antigens. These may arise as a result of exposure through pregnancies or blood transfusions in the past. A positive cross-match is a relative contraindication to transplant, as it will cause rejection of the graft. Currently, there are methods to reduce

HLA allo-sensitization using intravenous immunoglobulin and plasmapheresis.

What do you mean by full house match?

It is a common practice to express the degree of mismatch in terms of whether there are mismatches at each locus for HLA –A , HLA-B , and HLA- DR .If all three are matching ,then its called a '000 mismatch “ or a full house match.

Classification of renal transplants by donor organ source.

(1) **Related:** the donor is genetically related to the recipient (parent, child, sibling, half-sibling, aunt, uncle, niece, nephew, first cousin).

(2) **Paired:** when a donor agrees to give one of their healthy kidneys to a known recipient, the donor and recipient will be assessed to find out if they are suitable for an exchange. In some cases, the donor and recipients' blood groups or tissue types are mismatched or incompatible. If so, as a pair, they can be put forward

for 'paired donation', where they will be matched up with another donor and recipient in the same situation.

(3) **Pooled:** occasionally, more than two donors and two recipients will be involved in a swap. Each recipient gains from a transplant that they would not otherwise have had. The donors might not have given their kidney to the person they know, but that person will have received a kidney from one of the other pooled donors.

How do we prepare a patient for renal transplantation?

- In general, the preoperative evaluation of patients for kidney transplant should focus on the multi organ manifestations of ESRD, with the goals of risk stratification and optimization of the medical status of the patient before transplant
- Before surgery, patients receiving hemodialysis should be maintained on their regular dialysis schedules, whether hemodialysis or peritoneal dialysis. Ideally, dialysis should be performed before surgery, especially in patients with excessive intravascular volume or with documented hyperkalemia or acidosis
- A comprehensive laboratory panel that includes electrolytes, complete blood count, and platelet count is appropriate before surgery. Arterial blood gas may be done before surgery. A type and screen



or type and cross for packed red blood cells should be obtained.

- Although major blood loss during routine kidney transplantation is not common, the surgery involves major vascular structures with the potential for rapid bleeding. Documentation of an increased potassium level immediately before surgery, especially with electrocardiogram (ECG) changes consistent with hyperkalemia, should prompt consideration of immediate dialysis to correct potassium levels before surgery.
- Preoperative vital signs should be closely assessed, especially heart rate and arterial blood pressure trends in hospitalized patients. Preoperative intravascular volume status should be assessed.
- Comparing the patient's current weight to their known euvolemic weight, or "dry weight," may be helpful.
- Patients undergoing surgery immediately after dialysis may be significantly hypovolemic and prone to intraoperative hypotension.
- Orthostatic assessments and the presence of resting hypotension and increased heart rate may identify significant hypovolemia.
- Preoperative volume resuscitation with non-potassium-containing saline solutions or colloids can prevent hypotension on induction of anesthesia.

Review patients medications:

- Continue antihypertensive B blockers, withhold ACE-inhibitors and ARB drugs
- Lipid lowering drugs like statins to be continued
- **DM:** Withhold oral hypoglycaemic agents. Blood glucose monitoring on morning of surgery. i.v insulin to be started if high. Conventional insulin therapy: target glucose levels < 180mg/dl preferred over "tight glucose control" (81-108mg/dl) since increased 90-day mortality with the later and more incidence of perioperative hypoglycaemia
- Mild anxiolytic such as alprazolam given on

night prior surgery

- **Morning:** ABG with blood sugar done

What will be your anesthetic techniques in renal transplantation?

General anesthesia with endotracheal intubation along with regional nerve block like QL Block is the preferred anesthetic method for kidney transplantation in most kidney transplant centers.

The goals of anesthesia:

- Maintain haemodynamic stability
- Maintain renal perfusion and protect the donated kidney by avoiding hypoxia, hypovolemia, hypotension and nephrotoxic drugs
- Facilitate an adequate depth of anesthesia and appropriate muscle relaxation to facilitate surgical conditions
- Altered drug pharmacokinetics kept in mind

AV fistula: AV fistula limb protected: padded, labelled, no lines on that extremity, check fistula patency intermittently.

Monitoring:

Prior Induction:

- 5 lead ECG with ST-T analysis
- Non-invasive BP
- Pulse oximetry
- Wide bore IV line

Post induction:

- Capnography
- Temperature monitoring
- Neuromuscular monitoring
- Urine output
- **Invasive monitoring:** arterial line and CVP line, prior induction inserted in patients with severe cardiovascular compromise. Pulmonary artery catheter or transesophageal echocardiographic monitoring may be considered in patients with advanced CAD, left or right ventricular dysfunction, and pulmonary hypertension.



Static parameters

CVP

- CVP – If a central venous catheter is present, maintain CVP 8 to 15 mmHg throughout the perioperative period. Adjust the range to 8 to 12 mmHg in patients with a history of heart failure. CVP measurements reflect right ventricular filling pressure.
- Although it has limitations, CVP is the most commonly used "static" parameter to assess intravascular volume status (ie, preload) and to guide fluid therapy although may not be completely reliable
- The insertion of a central line provides reliable venous access for intravascular fluid resuscitation and transfusion, and access for administration of immunosuppression drugs and vasoactive infusions.
- USG guided : preferably & rule out thrombosis

Arterial Pressure

MAP – maintain MAP 70 to 90 mmHg. After reperfusion of the allograft, maintain a higher MAP (eg, >90 mmHg) in patients who had higher preoperative MAP values.

Direct intra arterial measurement of blood pressure is usually employed for early detection of decreased MAP. An intraarterial catheter is also useful for qualitative visual estimation of respiratory variation in the tracing. Furthermore, it facilitates intermittent blood sampling to monitor arterial blood gases, hemoglobin/hematocrit, potassium, and glucose.

SaO₂

SaO₂ is tracked as an indicator of perfusion, although this value does not always reflect in cardiac output or tissue oxygen delivery.

Urine output

Although oliguria (urine <0.5 mL/kg/hour) is commonly used as an indicator of hypovolemia in patients with normal kidney function, most patients undergoing renal transplantation are anuric until after the final vascular and ureteral anastomoses have been completed.

Some may remain oliguric due to technical problems (eg, ureteral obstruction or vascular thrombosis) or decreased perfusion of the allograft.

Dynamic parameters

Respiratory variation in the arterial pressure tracing

Respiratory variation in the intraarterial pressure tracing may be an indicator of hypovolemia. Such pulse pressure variation provides information about the likelihood that cardiac output will increase in response to a fluid challenge. In mechanically ventilated patients, large respirophasic variations in arterial systolic pressure and pulse pressure have been qualitatively linked to hypovolemia and underfilling of the right ventricle. Indices of respirophasic pulse pressure variation can be computed manually or automatically.

Airway pressures

Blood sugar

Aspiration prophylaxis: H₂ receptor blocker: iv ranitidine, or proton pump blocker like iv Pantoprazole given before induction ideally prior shifting patient to OT

Antibiotic prophylaxis: 30min prior incision

Induction:

- IV propofol or thiopentone graded doses (watch for hypotension)
- IV opioid like fentanyl 2mcg/kg given
- A rapid-sequence induction of anesthesia with continuous cricoid pressure is the preferred method of induction for general anesthesia. Succinylcholine can be used safely in standard doses in patients with ESRD when potassium levels are within normal limits (usually <5.5 mEq/L) (K increases by 1mEq/L, levels peak in 15 min)
- Some centres use Atracurium /Cisatracurium

Haemodynamic response to laryngoscopy blunted with either:

- Iv Esmolol (preferable since short acting, metabolism esterase dependent (0.5-1 mcg/kg/min)
- IV Lignocaine (1-2 mg/kg)
- Post intubation : patient may develop hypotension in patient rendered hypovolemic from recent dialysis or receiving ACE-inhibitors



Maintenance of anaesthesia: Air-O₂ Isoflurane controlled ventilation

Analgesia: Fentanyl, Remifentanyl, Sufentanil, Alfentanil

Muscle relaxant: Cisatracurium relaxant of choice. Dose 0.15mg/kg. further doses with NM monitoring

Immuno-suppressive therapy:

(Depending on institutional protocol)

Immunosuppressive therapy is to be initiated in the recipient 5 days prior to surgery.

Induction Protocol regimes include administration of oral Tacrolimus, Mycophenolate Mofetil, steroids (Methylprednisolone) and intravenous Basiliximab (Day 0 and Day 4) for Living Transplant or from a cadaveric donor to recipients with low immunological risk

Intravenous Thymoglobulin is to be given to recipient who are deemed at a higher risk for graft rejection (high immunological risk). These include;

- Cases in which prolonged cold ischaemic time is anticipated defined as more than 12 hours).
- In cadaveric transplant where induction time may be short.
- previous history of blood transfusion, previous pregnancy
- Immuno-suppressive plasma drug levels will be checked prior to surgery (Day 2-3 after induction). High levels may cause intraoperative hypotension and in some cases may be refractory.
- There may be an adverse reaction secondary to drug interactions especially with anti- hypertensive drugs. (ACE inhibitors or ACE Receptor Blockers- ARB). Hypotension may be accentuated by hypovolaemia secondary to post dialysis extraction and also anaesthetic agents
- **Incidence of inflammatory/allergic reactions** will be reduced by administration of anti-histamine and steroids prior to surgery.

We classify the recipient into three categories:

Recipient	High risk	Moderate risk	Low risk
	• Spousal • 6 HLA mismatch • previous transplant • Sensitised individual • Younger recipient • Dialysis vintage >3 y • DSA+	• HLA haplo match • Dialysis vintage 1-3 yrs	• HLA Zero mismatch • Older recipient • Dialysis vintage < 1 yr • DSA - -ve
Induction	ATG	ATG / Basiliximab	No induction



Surgical steps:

- Curvilinear incision 20-25 cm extending pubic symphysis to anterior superior iliac spine. Abdominal muscles retracted, peritoneum resected. Adequate anaesthesia essential since exaggerated haemodynamic response seen.
- Renal allograft placed in extraperitoneal space, right side preferred due to long length of renal vein.
- External iliac artery and vein dissected and mobilised
- Heparin may be administered prior clamping vessels
- External iliac vein is clamped first, renal vein anastomosis performed end-to-side
- External iliac artery clamped next, renal artery anastomosis of grafted kidney performed end-to-side

What is total, warm, cold ischemia time?

Ischaemia time starts with clamping of the renal vessels in the donor and ends with vascular anastomosis in the recipient. shorter the ischaemia time better is the organ preservation.

- Cold ischemia time is time from the immersion of the donor kidney in preservative solution to the rewarming in the recipients body on reanastomosis. It should be limited to 24 hrs.
- Warm ischemia time begins with the clamping of the renal vessels in the donor, it is interrupted by cold preservation solution and resumes again when the kidney is placed in the recipient and ends with the vascular anastomosis. To be maintained < 45 mins

Perioperative Fluid Management:

- During anastomosis renal vessels, expansion of intravascular volume should be initiated
- **In transplantation, denervation adds to deterioration in hemodynamic autoregulation of the kidney graft. Thus, even a mild decrease in blood pressure** can further reduce renal perfusion and thereby result in repeat ischemia to the transplanted kidneys At the time of removal of vascular clamps, additional intravascular volume expansion required-

- Perioperative fluid management must **ensure restoration and maintenance of intravascular volume**, in order to obtain good graft function.

Recommendations

- **Balanced solution** (A mixture of normal saline with either lactated or acetated Ringer's solution) may seem to be the ideal fluid for use in renal transplant surgery. Use with caution for cadaveric transplant recipients due to its potassium content in the face of potential delayed graft function.
- To avoid hyperchloremic acidosis and hyperkalemia, a **mixture** of isotonic saline and Hartmann solution may be a better option compared to normal saline alone
- Colloid if required, albumin given, avoid hetastarch

Recommendations

- The ideal range for the CVP and even the role of CVP has **not been clearly identified**.
- Generally, it is still recommended to have a **more liberal rather than restrictive** fluid infusion therapy to ensure adequate graft function for recipient

Goals of therapy include:

- **CVP > 12-15 mmHg**
- **Systolic BP of > 130mm**
- **Mean Arterial BP of > 80mm**
- **Total IV fluids of at least 15-20 ml/kg/hr**
- **Initial target SVV value of < 15%**
- **Higher infusion rates should be given during ischaemic phase (at the time of donor clamping)**
- **Graft turgidity as assessed by surgeon**

Mannitol in a dose of 0.25–0.5 g/kg:

- osmotic diuresis and intravascular volume expansion
- free radical scavenging properties
- protection against ischaemic injury
- promote renal artery dilatation and thus improve



- blood flow through the transplanted organ

Furosemide in low to high dose (40–160 mg) has also been used

- may result in massive diuresis in well functioning grafts, which may make fluid management difficult.
- The use of furosemide maybe beneficial in promoting urine flow and avoiding oliguria but the benefits in terms of improved graft function remain unclear.
- High dose boluses should be used with caution, as they are associated with ototoxicity

Sometimes removal clamps can cause acute bleeding; may need transfusion

- Adequate volume and reduction depth of anaesthesia may be required
- If hypotension persistent vasopressors : Noradrenaline/Dopamine can be used
- After vascular anastomosis, graft ureter is implanted is implanted to recipient bladder mucosa
- Bladder is filled with antibiotic irrigation solution by three way foley allowing for implantation of donor ureter
- Temporary ureteral stent placed Wound closed in layers
- Deep NM blockade till fascial layers closed to prevent coughing and straining to prevent disruption of graft position or vascular anastomosis.
- Reversal with neostigmine single dose, pharmacokinetics is same as any other normal patient.

Peri extubation issues:

- Inadequate reversal NM blockade
- Residual effects of opioids and sedative
- Risk of aspiration should be extubated with intact reflexes
- Exaggerated haemodynamic response, blunted with short acting agent like esmolol
- Most important: urine output

- Continue immunosuppressants

Post op complications:

- Acute renal failure and delayed graft functioning requiring dialysis.
- Delayed graft function maybe due to ischaemic damage prior renal transplant, hypoperfusion during or after transplant or acute tubular necrosis vascular thrombosis, wound haematomas infection

Post operative pain management:

- Acetaminophin
- Patient controlled analgesia with synthetic opioids without active Metabolites(Fentanyl)

Regional:

- **Epidural** : concern with uraemic coagulopathy
- Transversis abdomen plane block / Quadratus lumborum block

Postoperative management:

- ICU or HIGH DEPENDENCY UNIT
- Monitoring: target MAP
- Haemodynamics CVP: 10-12mm Hg
- Continue immunosuppressant
- Fluid management – Previous hour urine output + 100ml



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MASTERING THE MD/DNB ANAESTHESIOLOGY THEORY EXAMINATION

Strategic Writing Techniques + AI-Powered Preparation

A Practical Guide for MD and DNB Anaesthesiology Postgraduates

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The theory examination for MD and MD/DNB Anaesthesiology is not merely a test of knowledge — it is a **structured exercise in communication under time pressure**. Most postgraduates possess adequate knowledge by the time they sit for their examination; what frequently separates a distinction from a pass - or a pass from a failure - is not the depth of that knowledge but the ability to present it in a manner that examiners can read, follow, and reward.

Artificial intelligence has now transformed what was previously impossible: unlimited personalised practice, instant model answers, and examiner-style feedback available at any hour. This guide integrates both dimensions - **how to write excellent examination answers, and how to use AI to practise and perfect that skill**. The two are inseparable: without knowing what a good answer looks like, AI prompts produce mediocre output; without AI-accelerated practice, even good technique remains untested.

This guide is written from the perspective of an examiner and teacher who has set, evaluated, and discussed hundreds of anaesthesia theory papers over three decades. Use it sequentially: first master the craft of examination writing; then deploy AI to sharpen that craft at scale.

PART ONE: THE CRAFT OF EXAMINATION WRITING

I. Understanding the Examination Architecture

The MD/DNB Anaesthesiology theory examination consists of four papers, each of three hours' duration, carrying 100 marks across 10 questions of 10 marks each. The MD examination follows a similar pattern, typically across papers. Every question has defined subparts with explicit mark allocations — these are your most important guide to how much to write and where to focus.

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mark allocations — these are your most important guide to how much to write and where to focus.

Before sitting down to write your first sentence, internalise three structural facts:

- You have approximately 15–18 minutes per question. Time is your most finite resource.
- The examiner who marks your paper will assess 60–80 scripts. Clarity, legibility, and structure are rewarded; repetitive prose is not.
- The mark distribution within each subpart is your syllabus for that answer. A subpart carrying 2 marks requires a maximum of four to six points; one carrying 6 marks warrants a full structured response with a classification, mechanisms, clinical context, and complications.

II. Before You Write — The First Ten Minutes

A. Reading the Question Paper

The first ten minutes of a theory examination should be spent reading — not writing. Most candidates make their most consequential decisions in this window, and most errors stem from not reading carefully.

Read every question completely before you begin writing. The instruction at the top of your answer booklet says 'attempt all questions in order.' It does not say 'begin immediately.' Read all ten questions, identify your strongest four or five, and plan your sequence accordingly.

Pay particular attention to:

- **The action verb:** 'Enumerate' means list. 'Describe' means explain with mechanism and context. 'Discuss' means enumerate, explain, and critically analyse. 'Define' means provide a precise, acceptable definition. Confusing these loses marks.
- **The mark allocation for each subpart.** A 3+3+4 question tells you exactly how much intellectual real estate to allocate to each part.
- **The presence of clinical scenarios.** Case-based



questions reward clinical reasoning, not just recall. The examiner wants you to think through the scenario, not recite from a textbook.

B. Planning Your Answer

For each question, spend 60–90 seconds creating a mental or margin-written outline before you begin. Identify: the key classification (if applicable), the main headings you will use, any table or diagram that would convey information efficiently, and the clinical implication to mention at the end of each section. This prevents the most common error in theory examination writing: a disorganised, stream-of-consciousness answer that contains good information presented in a way that loses marks.

III. The Structure of a Good Answer

A. The Opening Line

Begin with a crisp definition or contextual statement. Do not begin with 'Introduction,' 'This is a very important topic,' or any equivalent. The examiner skips your introduction and looks for content. A strong opening might be: *'Sugammadex is a modified gamma-cyclodextrin that selectively encapsulates steroidal neuromuscular blocking agents, reversing their effect without the cholinergic side effects of neostigmine.'*

B. Classification - The Highest-Value Opening Move

Where the topic permits, begin with a classification. A well-structured classification demonstrates that you understand the architecture of the topic, not just isolated facts about it. It also provides your answer with an automatic framework: once you have classified, you have your headings.

Example: *Asked about 'opioids in anaesthesia,' begin with: 'Opioids may be classified by: (1) receptor activity — pure agonists (morphine, fentanyl, remifentanyl), partial agonists (buprenorphine), agonist-antagonists (nalbuphine, pentazocine), and pure antagonists (naloxone, naltrexone); (2) chemical structure — phenanthrenes, benzomorphans, phenylpiperidines; (3) duration of action — short, intermediate, long.'*

C. Headings, Subheadings, and Flowcharts

Use bold headings and subheadings liberally. They serve two functions: they make your answer scannable for the examiner, and they impose discipline on your writing, preventing repetition and drift. Underline or use a ruler for major headings if writing by hand.

In addition to headings, consider flowcharts for management algorithms, equipment sequences, and clinical decision pathways. A well-drawn flowchart for 'management of difficult airway' or 'intraoperative bronchospasm' communicates decision logic in a way that

prose cannot match efficiently.

D. Diagrams — Labelled, Titled, and Purposeful

A labelled diagram or well-constructed table can convey in ten seconds what would take two paragraphs of prose to explain — and it does so more accurately and memorably. Diagrams are particularly valuable for:

- The oxygen-haemoglobin dissociation curve
- West's zones of the lung
- Brachial plexus anatomy
- The cardiac action potential
- Anaesthetic breathing circuits
- Coagulation pathways
- Pharmacokinetic compartment models

Rules for examination diagrams:

- Draw cleanly, in pencil if preferred, and label every structure.
- A diagram without labels earns half marks at best.
- Include a title and legend where appropriate (e.g., axes on a graph).
- A rough but accurately labelled diagram is worth more than a beautiful unlabelled one.

E. Tables for Comparisons

When a question asks you to 'compare' two drugs, two techniques, or two conditions, a table is almost always superior to prose. The examiner can verify completeness instantly, and you cover more information in less space. A comparison table for sevoflurane versus desflurane, for example, would include rows for MAC, blood:gas coefficient, cardiovascular effects, respiratory effects, metabolism, environmental impact, and clinical uses - all in a fraction of the space that prose would require.

F. The Clinical Relevance Statement

Every pathophysiological or pharmacological answer should conclude its major sections with a statement of clinical relevance. This demonstrates that you are training to be a clinician, not an academician. *'Because sevoflurane has a lower blood:gas solubility coefficient (0.65) than isoflurane (1.4), induction and recovery are faster — this is particularly advantageous in day-case anaesthesia and paediatric practice where rapid awakening is a priority.'*

G. Sample 10-Mark Structured Answer

The following illustrates how these principles apply to a standard question. Study the architecture — classification first, headings, a comparison table, and explicit clinical anchoring at each stage.



SAMPLE MODEL ANSWER: 'Write a note on opioids in anaesthesia.' (10 marks)

Definition & Classification (2 marks)

Opioids are naturally occurring, semi-synthetic, or synthetic compounds that bind to specific opioid receptors (μ , κ , δ) in the CNS and peripheral tissues, producing analgesia, sedation, and dose-dependent respiratory depression.

Classification:

1. By receptor activity: Pure agonists (morphine, fentanyl, remifentanyl) | Partial agonists (buprenorphine) | Agonist-antagonists (nalbuphine, pentazocine) | Pure antagonists (naloxone, naltrexone)
2. By duration of action: Short-acting (remifentanyl), intermediate (fentanyl, alfentanyl), long-acting (morphine, methadone)

Comparison Table — Key Opioids in Anaesthetic Practice (4 marks)

Property	Morphine	Fentanyl	Remifentanyl
Potency (vs morphine)	1×	100×	250×
Onset	Slow	Rapid	Very rapid
Duration	4–6 hr	30–60 min	3–5 min
Metabolism	Hepatic (active M6G)	Hepatic (CYP3A4)	Plasma esterases
Context-sensitive $t_{1/2}$	Long	Increases	Stable ~3 min
Key clinical use	Post-op pain, PCA	Intraop analgesia	TIVA, day surgery
Notable adverse effect	Histamine release	Chest wall rigidity	Post-op hyperalgesia

Clinical Implications & Adverse Effects (4 marks)

Respiratory depression is the most clinically important adverse effect, mediated via μ_2 receptors in the pre-Bötzing complex. Remifentanyl's ultra-short context-sensitive half-life makes it ideal for TIVA and day-case surgery, but demands transition analgesia before discontinuation to prevent post-operative pain surge. Buprenorphine's ceiling effect on respiratory depression makes it relatively safer in opioid-dependent patients.

Examiner's note: This answer earns full marks by — opening with definition + classification, using a comparison table efficiently, anchoring each drug to clinical context, and mentioning mechanisms of adverse effects. Total writing time: ~15 minutes.



A. The Mark-Per-Minute Rule

With 100 marks in 180 minutes (excluding reading time), you have approximately 1.5 minutes per mark. A 5-mark subpart deserves 7–8 minutes of writing; a 3-mark subpart deserves 4–5 minutes. Many candidates write exhaustively on topics they know well and run out of time for the last two or three questions — this is the single most common cause of preventable marks loss in theory examinations.

B. Never Leave a Question Blank

A blank answer scores zero. An incomplete answer may score 2–4 marks. In a competitive examination, those marks can be the difference between passing and failing. If you are running out of time, switch to point form — a structured list of headings and key words tells the examiner that you understand the topic even if you have not had time to develop each point.

C. Prioritise Questions Strategically

Begin with the questions you are most confident about. Early success builds momentum and ensures maximum marks on questions you know. Leave questions you find difficult for later, by which time you will have settled into the examination rhythm. The answer booklet has allocated pages for each question; your answers need not follow alphabetical order as long as you clearly indicate the question number.

V. Question-Type Specific Strategies

A. Basic Sciences Questions - Definitions, Classifications, Mechanisms

These are the 30% of questions designed to give every prepared candidate full marks. Do not underperform on easy questions by rushing them. Structure: definition → classification → mechanism → clinical implication → complications or adverse effects. Allocate full time and do not sacrifice marks for the sake of speed.

B. Applied Clinical Science Questions

These questions test integration. The answer requires you to connect basic science to clinical practice. Structure: brief pathophysiology → clinical presentation → preoperative assessment → anaesthetic plan (induction, maintenance, monitoring, specific concerns) → postoperative care → complications. Use the SOAP framework

(Subjective–Objective–Assessment–Plan) for clinical scenario subparts where it fits.

C. Complex and Analytical Questions

Difficult questions reward the systematic thinker over the memoriser. If you do not know the specific answer, structure your response around what you do know: general principles, pharmacological class properties, physiological mechanisms, and clinical management principles. A well-structured answer that demonstrates logical thinking on a topic you are partially familiar with will score better than a disorganised answer full of memorised facts you cannot relate to each other.

D. Case-Based Analytical Questions

Case-based questions are the examiner's attempt to assess clinical judgement. They are not asking you to recite a chapter — they are asking what you would do with **this specific patient**. Always anchor your answer to the clinical details given in the stem. A preoperative assessment of a 22-year-old primigravida with severe pre-eclampsia is not the same as a generic answer about pre-eclampsia — it requires specific blood pressure targets, drug dosing in the context of magnesium therapy, rapid-sequence spinal anaesthesia considerations, and a postoperative monitoring plan.

Poor Answer vs Good Answer - At a Glance

? Poor Answer Characteristics	? Good Answer Characteristics
Begins with 'Introduction' / generic background	Begins with crisp definition or classification
Stream-of-consciousness prose, no headings	Bold headings and subheadings throughout
Bullets only — no explanatory prose	Bullets for recall; prose for mechanism and reasoning
Diagram drawn but not labelled	Fully labelled diagram with title and legend
No clinical relevance mentioned	Clinical implication stated after each major section
Generic answer ignoring case-specific details	Answer anchored to the specific patient in the stem
Exhaustive on Q1; Q9 and Q10 left incomplete	Strict 15–18 minutes per question; none left blank
Abbreviations used without definition	Full term written first, abbreviation in parentheses



VI. Handwriting, Presentation, and Examiner Psychology

A. Legibility

Illegible handwriting is not just an inconvenience — it is a marker of disrespect towards the examiner's time. Slow down by 10% and write clearly. The examiner cannot award marks for content they cannot read.

A DNB/MD Anaesthesiology examiner typically marks 60 to 80 answer scripts (papers) in a single examination session or sitting (lasting few days).

Good handwriting addresses examiner fatigue and psychology. When someone is reading through 60–80 handwritten answer papers one after another, they are tired and time-pressured. A well-structured, clearly written answer stands out immediately and earns goodwill. A cluttered, prose-heavy, poorly organised answer frustrates them and loses marks — not necessarily because the content is wrong, but because it demands extra effort to extract marks from it: **“Clarity is not a courtesy — it is a strategy.”** In other words, writing clearly and structurally is not just being polite to the examiner — it is a deliberate marks-scoring tactic in your own interest.

B. Ink, Presentation, and White Space

Use blue or black ink. Do not use pencil for written answers (only for diagrams if your institution permits). Cross out errors neatly with a single line — never use correction fluid. Write the question number clearly in the margin at the beginning of each answer.

Do not cram content onto the page. Leave adequate margins, space between headings and text, and space between sections. A well-spaced answer looks like the work of a confident, organised candidate.

C. The First Impression Effect

Research in educational measurement consistently shows that the first half-page of an answer influences the score assigned to the remainder of that answer. Begin every answer with a strong, well-structured opening. A crisp definition, a logical classification, or a concise clinical

context statement sets the tone for everything that follows.

D. The Examiner's Perspective

As someone who has sat on DNB examination panels and evaluated theory papers, I can tell you what examiners look for — and what they find frustrating. We are looking for evidence that you understand the topic, not just that you have memorised a list of facts. We reward structure because it tells us you can think systematically. We reward clinical relevance because it tells us you will be a safe clinician. We reward diagrams that are accurately labelled because they demonstrate spatial understanding.

We are frustrated by answers that pad length without adding content, by handwriting that requires decipherment, and by answers that begin confidently and then lose structure halfway through. What a well-written paper communicates above all is this: the candidate sitting at this desk is ready to be trusted with the care of patients under anaesthesia.

VII. Common Errors to Avoid

- Writing the question number out in full as part of your answer — this wastes time and space.
- Repeating the same information in different words across multiple paragraphs.
- Using abbreviations without first writing the full term.
- Answering a different question from the one asked — always re-read the question before beginning and again midway through your answer.
- Spending disproportionate time on subpart (a) and leaving subpart (b) incomplete.
- Drawing diagrams without labelling them.
- Writing in bullet points throughout without any explanatory prose — bullets demonstrate recall, not understanding.
- Beginning with 'Introduction' followed by generic background not specific to the question.
- Failing to mention clinical implications — basic science answers without clinical context score lower in clinical examinations.



VIII. The Ten Commandments of Theory Examination Writing

I	Read the entire question paper before writing a single word.
II	Allocate time strictly: 15–18 minutes per question, no exceptions.
III	Begin every answer with a definition or classification.
IV	Use bold headings, subheadings, and flowcharts to create visual structure.
V	Draw labelled diagrams and construct tables wherever they add value.
VI	Anchor every basic science answer to a clinical implication.
VII	In case-based questions, always respond to the specific patient in the stem.
VIII	Never leave a question blank — partial answers score partial marks.
IX	Write legibly, space your content, and respect the examiner's reading experience.
X	Practise writing full answers under timed conditions before the examination — and use AI to evaluate what you write.

VIII. The Ten Commandments of Theory Examination Writing

Category	Action Item	Done?
Preparation	Revise the Ten Commandments of Theory Writing	☐
Preparation	Review your AI-generated weak-topic summaries	☐
Preparation	Re-read 3 –5 of your best practice answers (structure check)	☐
Equipment	Pack two pens (blue/black), one pencil, one ruler	☐
Equipment	Check your hall ticket, institution ID, and admit card	☐
Strategy	Remind yourself: read the full paper before writing	☐
Strategy	Set your mental alarm — 15–18 minutes per question, no overrun	☐
Strategy	Plan to attempt your strongest questions first	☐
Wellbeing	Sleep by 10 PM. A rested brain writes better than a cramming one.	☐
Wellbeing	Eat a proper breakfast, carbohydrate rich. Hydrate well. Cognitive performance drops with hunger.	☐



PART TWO: AI-POWERED EXAMINATION PREPARATION

X. What AI Can and Cannot Do For You

Artificial intelligence has transformed the landscape of medical education. For postgraduates preparing for the MD and DNB Anaesthesiology theory examinations, AI platforms now offer capabilities that no textbook, teacher, or coaching class can replicate: unlimited availability, instant feedback, personalised question generation, and the ability to simulate an examiner's perspective on demand.

Before deploying any AI tool, understand its nature and limitations:

- **AI can occasionally be wrong** — particularly on specific drug doses, recent guideline updates, or highly regional content. Always verify key clinical facts against your standard references: Miller's Anaesthesia, Morgan & Mikhail, Stoelting, University/NBEMS syllabus, and relevant society guidelines. For example, always cross-check sugammadex dosing (2 mg/kg for moderate block, 16 mg/kg for immediate reversal) and current ASA fasting guidelines against primary sources.
- **AI cannot replicate the examiner's specific expectations** — unless you provide that context explicitly in your prompt.
- **AI should help you understand, not just reproduce model answers.** The goal is genuine comprehension that will serve your patients — not surface-level pattern-matching that collapses under viva questioning.
- **AI works best as a thinking partner and practice tool** — not as a replacement for reading, clinical experience, or structured teaching.

With those caveats clearly stated, AI platforms can dramatically accelerate and deepen your examination preparation when used systematically — particularly when combined with the writing principles described in Part One.

XI. Choosing Your AI Platforms

Platform	Best For	Free / Paid	Anaesthesia-Specific Strength	Top Prompting Tip
Claude (Anthropic)	Structured essay answers, examiner-style feedback, long-form explanation	Free + Pro	Excellent at maintaining context; best for model answers and multi-turn Socratic dialogue	Ask it to structure answers using classification → headings → table → clinical implication
ChatGPT (OpenAI)	Quick Q&A, topic summaries, question generation	Free (3.5) + Plus (GPT-4)	Widely used; strong MCQ and short-answer generation	Specify 'MD/DNB-style' and request mark allocations in every prompt
Grok (xAI)	Reasoning-heavy questions, up-to-date clinical content	Free + Premium	Strong analytical reasoning; good for pharmacology and recent guideline integration	Ask it to reason step-by-step through clinical scenarios
Gemini (Google)	Guideline cross-referencing, image analysis	Free + Advanced	Integration with Google Search; useful for checking recent updates	Use for verification after generating content with Claude or ChatGPT
Perplexity AI	Evidence-based answers with citations	Free + Pro	References actual papers — essential for checking guideline accuracy	Use after AI generates an answer to verify with cited sources
NotebookLM (Google)	Query your own notes and PDFs	Free	Creates a personal AI tutor from your own study material	Upload your annotated textbook chapters and past answer scripts



Recommendation: Use Claude or ChatGPT as your primary practice partner, Grok for analytical reasoning and current clinical content, Perplexity or Gemini for guideline verification, and NotebookLM to create a searchable AI-powered version of your own notes.

XII. Prompt Engineering for Anaesthesia Theory

The quality of AI output depends almost entirely on the quality of your input. These four golden rules will consistently produce examination-grade responses:

Rule	What to Do	Example
Be examination specific	Always name the examination and format	'Generate a MD/DNB Anaesthesiology Final theory answer, 10 marks, with subpart mark allocation...'
Request the right structure	Specify the architecture from Part One of this guide	'Structure the answer as: classification → mechanism → table → clinical implication → complications'
Set difficulty and depth	Calibrate to your level	'I am a second-year MD/DNB candidate. This question is moderate difficulty...'
Ask for feedback, not just answers	Use AI as an evaluator, not just a generator	'Evaluate my answer against examiner expectations. Tell me what I missed and approximately how many marks it would score.'

XIII. The Five Core Uses of AI for Theory Preparation

XIII.1 - Generate Model Answers

The most powerful use of AI for theory preparation is generating fully structured model answers to past questions and practising your own answers against them - using the structural principles from Part One as your benchmark.

How to do it:

- Open your chosen AI platform (Claude is recommended for this task).
- Paste the question exactly as it appears in the past paper.
- Prompt: 'Generate a model answer for a MD/DNB Anaesthesiology theory examination for this question. Structure it as described in Dr Murthy's writing guide: classification first, bold headings, a comparison table where applicable, and a clinical implication at the end of each major section. Include mark allocation guidance.'

- Study the model answer carefully. Identify the headings, the depth of each section, and the clinical anchoring.
- Close the AI response and write your own

The active learning cycle — generate, study, write, compare, refine — is far more effective than passive reading. This is precisely the practice that the Ten Commandments endorse.

answer from memory under timed conditions (15–18 minutes).

- Return to AI and ask: 'Compare my answer to the model answer and tell me what I missed, what was irrelevant, and what I should prioritise.'

XIII.2 — Use AI as a Personalised Question Generator

Past question papers are a finite resource. AI can generate unlimited fresh questions in the style of MD/DNB examinations, calibrated to any topic or difficulty level.

Prompts that work well:



- 'Generate 5 MD/DNB-style theory questions on thoracic anaesthesia, with difficulty distribution of 2 easy, 2 moderate, and 1 analytical case-based question. Include mark allocations.'
- 'Generate a 10-question theory paper on critical care and emergency anaesthesia in the style of the University/NBEMS MD/DNB Final Examination, with subpart mark allocations.'
- 'I am weak on pharmacokinetics. Generate 3 questions that specifically test applied pharmacokinetics in the context of anaesthesia drug administration.'
- 'Generate a question requiring a labelled diagram of West's zones of the lung, and provide a text description of the diagram an examiner would expect.'

Once questions are generated, use them for timed practice without referring to the AI's model answer until you have completed your written response.

XIII.3 — Master Topics with Socratic Dialogue

For topics you find difficult to understand (not just memorise), use AI as a Socratic dialogue partner — a virtual teacher who answers follow-up questions and builds your understanding progressively.

Example dialogue:

- You: 'Explain the physiological basis of hypoxic pulmonary vasoconstriction and its clinical relevance during one-lung ventilation.'
- AI: [Explains HPV mechanism, inhibiting agents, clinical significance]
- You: 'Which volatile agents inhibit HPV most, and by what mechanism?'
- AI: [Detailed response with dose-dependence]
- You: 'How does the inhibition of HPV by volatile agents compare to propofol in terms of magnitude and clinical significance during one-lung ventilation?'
- AI: [Comparative analysis with evidence — verify against current literature]

This progressive deepening of a single topic builds the kind of integrated understanding that analytical examination questions reward — and that no amount of passive reading can replicate.

XIII.4 — Get Examiner-Style Feedback on Your Written Answers

One of the most valuable features of modern AI is the ability to receive detailed, specific feedback on your written answers — feedback that most postgraduates have no access to between formal assessments.

How to do it:

- Write a full answer to a theory question by hand within the time limit.
- Type or transcribe your answer, or scan the answer script and paste it into your AI platform. Most AI platforms read and understand handwritten answers.
- Prompt: 'I am a MD/DNB Anaesthesiology postgraduate. This is my examination answer to the following question: [paste question]. Please evaluate my answer using the following criteria: (1) Does it open with a definition or classification? (2) Are headings and subheadings used? (3) Is a table or diagram included where appropriate? (4) Is clinical relevance stated after each major section? (5) What key points did I miss? (6) Approximately how many marks out of 10 would this score, and why?'
- Revise your answer based on the feedback and resubmit for re-evaluation.

This feedback loop integrates directly with the writing principles of Part One — it is the examiner's checklist operationalised as a prompt.

XIII.5 — Build and Maintain a Spaced Repetition System

Spaced repetition — reviewing material at increasing intervals — is the most evidence-based method for long-term retention. AI can help you build and manage this system.

How to do it:

- After studying any topic, ask AI: 'Generate 10 high-yield short-answer questions on [topic] that I should be able to answer from memory as a MD/DNB anaesthesiology candidate.'
- Write your answers, check them against AI-generated model answers, and rate your confidence (1–3).
- Add questions rated 1 or 2 to a review list.



- Return to those questions 3 days later, then 7 days later, then 14 days later — each time writing your answer from memory before checking.
- Apps like Anki (flashcard application- free on android and desktop) can be combined with AI-generated question content for automated spaced repetition scheduling.

XIV. Topic-Specific AI Prompt Library

Copy, adapt, and use these prompts directly. They are designed to produce output structured around the writing principles in Part One.

For Pharmacology Questions

- 'Explain the mechanism of action, pharmacokinetics, and clinical uses of [drug] in a MD/DNB-style structured answer with a classification table. Note any doses or guideline-specific content I should verify against primary references.'
- 'Compare [drug A] and [drug B] in a table covering mechanism, onset, duration, metabolism, elimination, and clinical uses - formatted for examination writing.'
- 'What are the most commonly examined pharmacology topics in MD/DNB Anaesthesiology final theory papers? Rank them by frequency and explain why they recur.'

For Physiology Questions

- 'Describe the physiology of [topic] and explain its clinical implications in anaesthesia practice. Include a text description of the diagram I should draw in the examination, with axes and labels specified.'
- 'Generate a MD/DNB-style question on [physiological topic] and provide a model answer with mark allocation, opening with classification and including a clinical implication statement.'
- 'I scored only 3/5 on the physiology section of this answer: [paste answer]. Using Dr Murthy's writing guide criteria, what key physiological concepts and structural elements did I miss?'

For Case-Based and Analytical Questions

- 'I am a MD/DNB candidate. Here is a case: [paste case]. Walk me through how an examiner would expect me to structure my answer — what clinical reasoning framework should I use, and how should I anchor each section to this specific patient?'
- 'What are the haemodynamic goals for [specific condition, e.g., severe mitral stenosis]? Provide them in a table with the rationale for each, and flag any values I should verify in current guidelines.'
- 'Generate a flowchart (described in text) for the management of [condition, e.g., intraoperative anaphylaxis] that I could draw in an examination answer.'

XV. Three-Month AI-Integrated Study Schedule

This schedule merges writing practice and AI-powered preparation into a single cohesive programme. The progression mirrors the same three-phase structure recommended in both components of this guide.



Month	Phase	Writing Practice	AI Integration	Daily Time
Month 1 Weeks 1–4	Foundation Building	Write one answer per day to a past question. Mark against the writing criteria: definition, classification, headings, table, clinical implication.	Use AI to generate model answers for each topic. Compare your written attempt. Use Socratic dialogue for any section you found difficult.	60–90 minutes
Month 2 Weeks 5–8	Application & Integration	Write one full 10-question paper per week under timed conditions (3 hours, by hand). Evaluate your own time management.	Submit transcribed answers to AI for examiner-style feedback using the prompt in Section XIII.4. Use AI to generate spaced repetition questions for weak topics identified in Month 1.	90 minutes
Month 3 Weeks 9–12	Simulation & Consolidation	Full examination simulations weekly. Write by hand, under time pressure, with no notes. Focus on applying all Ten Commandments simultaneously.	Review all AI feedback received over the past two months. Identify persistent gaps. Use AI for final consolidation of your three weakest topics. Review your AI-generated prompt pack.	2 hours

XVI. Ethical and Practical Boundaries

Use AI as a preparation tool, not a substitute for genuine learning. The examination exists to assess your competence as a future clinician - competence that will determine patient safety. Shortcuts in examination preparation are shortcuts in clinical preparation.

- **Do not use AI during the examination** — this constitutes academic dishonesty and, where discovered, results in disqualification.
- **Do not treat AI-generated content as infallible.** Cross-reference drug doses and guideline-specific content against authoritative references — always.
- **AI should help you understand, not just reproduce model answers.** If you cannot explain a concept in

your own words after reading the AI's model answer, you have not yet learned it.

- **Do maintain a record of what you have practised and what AI feedback you have received** — this metacognitive awareness is itself a learning tool..

XVII. A Final Word

The postgraduates who will benefit most from this guide are not those who use it to avoid hard work — but those who use it to make their hard work more targeted, more efficient, and more effectively evaluated. The writing principles in Part One and the AI tools in Part Two are not separate strategies; they are two halves of a single system. You cannot use AI effectively without knowing what a good answer looks like. And you cannot write good answers consistently without the accelerated practice that AI now makes possible.



The examination is still written by you, marked by human examiners, and reflective of your own understanding. What AI offers is an unprecedented degree of personalised practice and feedback that was unavailable to any previous generation of candidates.

Use it wisely. Write diligently. Understand deeply.

The knowledge you carry out of this examination hall
must serve your patients for a lifetime.

Disclosure: The author has no conflicts of interest to declare. The ideas here are generated by the experience gained by nearly three decades of active teaching. In preparation of this manuscript I have taken ample help from various AI tools/agents/platforms with which I have been working for more than a year now. This guide is written to look like a typical answer to a theory question. This guide was prepared for PG Excel 2026, Navodaya Medical College, Raichur, and is submitted for inclusion in the PG Excel 2026 E-Book.

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14th JUNE 2026
04:50PM - 05:25PM

ANAESTHESIA MACHINE-QUESTION & ANSWER FORMAT

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Anaesthesia machine in a Q&A format:

1. Evolution of Boyle's machine:

1. What are the forerunners of current Boyle's machine?

Teter's apparatus, Cotton and Boothby apparatus and Gwathmy's apparatus

2. On which anaesthetic apparatus, Boyle's has modified and designed his own apparatus?

Gwathmy's apparatus.

3. Can you tell something about Boyle whose name is attached to our anaesthesia machine?

His full name is Henry Edmund Gaskin Boyle who headed the anaesthesia department in St.Bartholemew's Hospital, England. In 1916, he imported Gwathmey's Nitrous Oxide-Oxygen-Ether machine from America and modified it. He is also involved in the invention of Boyle-Davis mouth gag which is still used in ENT surgeries.

4. Who are the pioneers in the designing of anaesthesia apparatus even before Henry Boyle?

James Taylor Gwathmey and Geoffrey Marshall.

5. When and by whom "to and fro" canister was introduced?

Dr.Ralph Waters had introduced "to and fro canister" in the year 1921.

6. When and by whom the circle absorber was introduced in to the anaesthetic practice?

Brian Sword had introduced the circle absorption system in the year 1930.

7. Mention some wired masks which were used for the administration of inhalational anaesthesia during historic times:

1. Skinner mask
2. Schimmelbusch mask
3. Ferguson mask
4. Yankaaauer mask

5. Bellamy-Gardner mask

8. Who introduced the metal gas cylinders in to the anaesthetic field?

S.S.White of Philadelphia and Coxeter of London

9. Who invented the reducing valve to reduce the high cylinder pressure to a working pressure ?

Jay Albion Heidbrink introduced the reducing valve concept. In Germany, Heinrich Draeger and his son Bernhard Draeger invented a reducing valve to be used in the Beer industry. This valve was modified and used in the anaesthesia machine.

10. Who introduced the Rotameter?

Rotameter was introduced by Karl Kuppers in 1908.

11. What do you mean by "Water Sight Flow Meters"?

In the earlier days before the introduction of dry flow meters, oxygen was made to flow through the ether liquid through a tube with multiple holes. The flow of O₂ was measured depending upon the number of holes generating bubbles.

12. Describe the early development of soda lime canisters:

Dennis Jackson demonstrated the usage of sodalime to conserve anaesthetic gases in animals. Ralph Waters after discussing with Jackson developed his "To and Fro" canister. Brian.C.Sword modified the canister and fitted in to the anaesthetic machine.

13. Describe the Boyle's Ether vaporizer:

It is a flow over, variable Bye-pass vaporizer. There is no automatic temperature compensation mechanism. There is a plunger rod in the ether bottle which can be depressed close to the surface of the liquid ether so that the fresh gas flow is made to go near the surface of the liquid ether or bubble through it. This manoeuver will increase the ether output concentration. So whenever the ether output decreases because of heat loss, the plunger can be manually depressed to maintain the output concentration.



14. Why ether has gone out of anaesthetic practice?

Ether is a slow inducing agent with prolonged recovery time. It is an airway irritant predisposing to Bronchospasm and Laryngospasm. It is highly inflammable which precludes the use of diathermy.

15. Describe the "Copper Kettle Vaporizer":

It is a measured flow, bubble through, out of system, temperature compensation by supplied heat and manual flow alteration vaporizer. Multiple anesthetic agents can be used in this vaporizer.

16. Describe the "E.M.O"(Epstein-MacIntosh-Oxford) vaporizer:

It is a variable bye-pass, flow over with wick, temperature compensation by supplied heat by a water bath and flow alteration and an agent specific (ether) vaporizer. It was mainly used for field operations.

17. Describe "Goldman vaporizer":

It is a variable bye-pass, flow over without wick, in or out of system, no temperature compensation and multiple agent vaporizer. Commonly it was used as a halothane vaporizer which when fully charged and the dial is kept maximum, it used to deliver 2 to 2.5% of halothane vapour.

18. When was the first ether anaesthesia conducted in India?

March 22,1847 at Calcutta.

19. Where was the first hospital piping line done in India?

Christian Medical College,Vellore, Tamilnadu.

20. How will you classify the anaesthetic machines?

1. Intermittent flow or demand flow machines
2. Continuous flow machines

21. Give an example of intermittent flow anaesthesia machine:

McKesson's apparatus - mainly used in dental practice.

22. How will you classify the basic components of a Boyle's machine?

High pressure system and a low pressure system.

2. Compressed Gas cylinders:

25. What is the material used for the manufacture of

cylinders?

Molybdenum steel

Aluminium for MRI compatible cylinders

26. What are the sizes of cylinders used in India?

1. A size- used in Boyle's machine- O₂ content in this cylinder-660 lit
2. B-size-used in the wards-O₂ content- 1320 litres
3. D-type used in the manifold room - O₂ content 6900 litres
4. AA size- small size - content -330 litres of O₂

27. What are the parts of a cylinder?

Body, shoulder, neck, base and cylinder valve.

28. What do you mean by "Woods metal" and where it is used?

It is an alloy of bismuth, tin, cadmium and lead. In some cylinders it is used as a safety relief device being situated in the valve body. In some, this alloy is used to seal the valve body into the neck of the cylinder. If the cylinders are exposed to high temperature, this alloy will melt and lets the gas goes out, avoiding an explosion.

29. What is the metal used to construct the valve body?

Bronze or brass

30. What is a Bodok seal?

It is a washer with a metal ring, made up of neoprene. It is used to provide air tight fitting of the cylinders with yoke assembly.

31. What do you mean by "cracking of cylinders"?

Before fitting the cylinders to the yoke assembly, the cylinder valve should be opened a little which clears any dust in the gas outlet port. This procedure is called "cracking of cylinders".

32. What will happen if the dust is carried inside the Boyle's circuit?

The flow meter bobbin can get struck by the presence of dust which will give a false sense of flowing of gases.

33. What do you mean by "Yoke Plug"?

Yoke plug is a metal dummy which can be placed in the cylinder port when it is not fitted with a cylinder. It prevents dust being settle in the gas entry port of the yoke assemble.



34. How many possible combinations are there for pin index system and how many are allotted to gases?
10 possible combinations are there with 6 pins. All the 10 combinations of numbers are allotted to gases.

35. Which pin index number is allotted to the combined N₂O : O₂ (Entonox) cylinder?

Seven. (A single pin)

36. What are the pin index numbers for the various gas cylinders?

Oxygen: 2, 5

Nitrous Oxide: 3, 5

Air: 1, 5 Cyclopropane: 3, 6

Nitrogen: 1, 4

37. What is the pressure inside the cylinders of various gases?

N₂O & CO₂ = 750 psi or 5000 kPa

O₂ = 2000 psi or 150 kgf/cm² (Kilogram-force per square cm)

38. What do you mean by critical temperature of a gas? What is its significance?

The critical temperature of a substance is the temperature at and above which vapour of the substance cannot be liquefied, no matter how much pressure is applied. N₂O cylinder when it is exposed to a temperature above its critical temperature (36.5°C), it can't exist as a liquid and will get converted in to gas. In case if the N₂O cylinder is filled up to the brim, this conversion will lead to enormous pressure rise inside the cylinder, leading to an explosion. In order to avoid this N₂O cylinders are filled up to 67% of the volume of the cylinder.

39. What are the critical temperatures of O₂ and N₂O?

O₂ = -119°C N₂O = 36.5°C

40. What is the storage temperature of liquid oxygen?

It is stored between - 150° to -170° C at a pressure of 5-10 atmospheres.

41. What is the pressure inside the Entonox cylinder?

13700 kPa or 2000 psi.

42. How many types of cylinder valves are there?

i. Flush type

ii. Bull nose type

43. What are the different types of cylinder valve

interior designs?

i. Packed type

ii. Diaphragm type

44. What do you mean by "Filling ratio" of cylinders?

The filling ratio is the ratio between the weight of the gas with which the cylinder is filled divided by the weight of the water the cylinder could hold. The cylinders can't be filled with anaesthetic gases which are in liquid form to its full brim. Otherwise a rise of temperature may cause a rise of pressure leading to bursting of cylinders.

45. What are the anaesthetic gases which are in liquid form?

i. N₂O

ii. CO₂

iii. cyclopropane

46. What are the filling ratios of them?

N₂O and CO₂ -0.66 Cyclopropane – 0.48

47. What is the filling ratio of Oxygen?

Oxygen cannot have filling ratio as it is a gas which will fill the cylinder as a whole.

48. What do you mean by "transfilling of cylinder"? How it is avoided to happen?

If an empty cylinder is left out in the yoke assembly, O₂ from the nearby full cylinder/pipeline can enter in to it. This is called "transfilling of cylinder". This is prevented from happening by incorporating an one-way non-return valve between the cylinder and pressure gauge.

49. When a used cylinder should be condemned?

Any cylinder which fails to pass the periodic test, loses its tare weight by over 5%, has any other defect which makes it unsafe and after expiry of its service life. to be condemned.

50. What is the colour coding of various cylinders?

Oxygen: Black body and white shoulder

Nitrous oxide: French blue body and shoulder

CO₂: Grey body and shoulder.

Entonox: Blue body and blue and white shoulder.

51. When will you submit the cylinders for checking?

All the gas cylinders should be submitted for checking every 5 years. The year of checking must be permanently stamped on the cylinder.

52. What is the pressure to be raised to check the integrity of cylinder?



1.66 times the service pressure should be raised inside the cylinder to check for the leaks. The service pressure is the maximum pressure to which the cylinder may be filled at 700 F.

53. What is a "hydrostatic test" for the cylinders?

Cylinder is filled with water and pressurized to 1.66 times the working pressure. Excessive expansion or presence of leaks precludes the usage of that cylinder.

54. What is the storage instruction for the Entonox cylinders?

They should be stored in a horizontal position in a dry and non-humid area. They should be inverted twice or thrice before it is being used to ensure proper mixing of gases. On storage, N₂O tends to settle in the bottom and O₂ in the top of the cylinder. When this cylinder is used without ensuring proper tilting, there is a possibility to deliver oxygen rich gases first and hypoxic N₂O mixture at the last.

55. What are all the common clinical situations where Entonox inhalation be used for pain relief?

Burns dressing and labour analgesia.

56. How will you know that the given Oxygen cylinder will last for the given surgical duration?

The 'A' type oxygen cylinder which is usually fitted to the anaesthesia machine will hold 660 litres of oxygen, generating a pressure of 1950 psi. (For practical calculation, we can take it as 2000 psi). If the pressure gauge shows 2000 psi, then it is a full cylinder with 660 litres of oxygen. If we draw 2 litres of oxygen per minute, then it should last for 330 min (5.5 hours). But usage of anesthesia ventilator and emergency oxygen flush will empty the cylinder fast.

57. How will you know that the fitted N₂O cylinder will last for the given surgical duration?

The pressure shown in the N₂O pressure gauge cannot be approximated with the quantity of N₂O as we do it for oxygen as part of N₂O will be in the liquid form. Here we have to employ the Avagadro's principle to calculate the amount of N₂O gas inside the cylinder.

58. Explain 'Avagadro's principle':

Avagadro's law states that under the same condition of temperature and pressure, equal volumes of different gases will contain an equal number of molecules. In other words, at STP one mole of any ideal gas will

occupy 22.4 litres and will contain 6.023×10^{23} numbers of molecules.

59. How to apply Avagadro's law in calculating the amount of N₂O gas in the cylinder?

N₂O being an ideal gas, one mole of it will occupy 22.4 litres. The weight of one mole of N₂O is its gram molecular weight. (Gram molecular weight is nothing but molecular weight expressed in grams). The gram molecular weight of N₂O is 44. (Two Nitrogen atoms + One oxygen atom constitute N₂O molecule. The atomic weight of Nitrogen is 14 and for oxygen 16. The molecular weight of N₂O is $14 + 14 + 16 = 44$). So 44 grams of N₂O will occupy 22.4 litres.

To calculate the quantity of N₂O in the cylinder, we have to weigh the cylinder with its contents. If we know the tare weight of the cylinder (It will be inscribed in the shoulder of the cylinder as TW), the contents can be calculated.

Subtracting the tare weight from the weight of the full cylinder will give you the weight of the N₂O. If weight of the N₂O is known (say for example 2500 grams), then Avagadro's law can be applied. If 44 grams occupies 22.4 L, then 2500grams will occupy $22.4/44 \times 2500 = 1272$ L. If 2 L of N₂O is drawn per minute, it should last for 21 hours.

3. Pressure Gauge:

60. What is the proper name of the pressure gauge?
Bourdon Pressure Gauge.

61. What is the principle used in it?

It uses a curved, flattened tube that gets straightened on the entry of a pressurized gas. This tube is connected to rack and pinion mechanism which is activated by the straightening tube. The degree of straightening is converted to a measure on a calibrated scale.

62. What is the safety mechanism employed in the older type pressure gauges?

There is a vent in the back of the gauge which will release the gas in the event of rupture of the curved tube not exposing the Anaesthesiologist who stands in front of the gauge.

63. What is the colour coding of the pressure gauge?



It is white for oxygen and French blue for the Nitrous oxide.

4. Pressure regulators:

64. What is the need for a pressure regulator in an anesthesia machine?

The gas containing cylinders such as Oxygen, carbon dioxide, medical air and Nitrous oxide are supplied in high pressure containers. This high pressure has to be reduced to a working pressure so that barotrauma doesn't occur. The pressure has to be regulated to a constant pressure so that the flow meters work properly.

65. What is the outlet pressure of the pressure regulators in the anaesthetic machine?

50 psi/ 4 bar / 420 kPa

66. If there are 2 dedicated lines for piped O₂ supply and O₂ cylinders in an anaesthesia workstation, how the O₂ from the pipeline is made to be used preferentially?

The outlet pressure of the pressure regulator situated in the cylinder line (50psi) is made little lower than the piped line supply. As the pressure from the regulator on the pipeline (60psi) is higher, it is preferentially used.

67. What do you mean by "pre-set" regulator?

The outlet pressure is pre-set at the factory in this type of regulators. They can't be altered by the Anaesthesiologists. That's why they are called as "pre-set" regulators.

68. What do you mean by "master and slave" regulators?

The N₂O pressure regulator is connected with the O₂ pressure regulator in such a way that the outlet pressure from the O₂ regulator is needed for the functioning of the N₂ regulator. If O₂ pressure drops, N₂O regulator also shuts off preventing the N₂O flow.

69. What is the basic working principle of pressure regulators?

Large force acting on a small surface area can be balanced by small force acting on a large surface area.

70. Which primary factor decides the magnitude of pressure reduction in the pressure regulators?

The ratio of the large diaphragm to the small diaphragm decides the amount of pressure reduction.

If the small diaphragm is one tenth of the diameter of the larger one, the pressure reduction will be one tenth of the cylinder pressure. To explain further, if the large diaphragm measures 100 mm and small one 10 mm, the ratio of their dimension is 10:1. If the cylinder pressure is 2000 psi, then it will be reduced to 200 psi.

71. What do you mean by "adiabatic compression" of gases?

When an O₂ cylinder is opened, the gas expands and gushes into the machine. If the flow meter is not opened, the gas again gets compressed in the pressure gauge within a very short time. As the gas gets compressed in a short time, the temperature rises enormously. This type of gas compression where there is no time for heat dissipation is called "adiabatic compression".

72. Why oil or grease should not be used in the O₂ valve cylinder?

If oil / grease molecules are taken inside the O₂ line and exposed to the high temperature because of adiabatic compression, there is a chance of fire mishap. That's why oil/grease should not be used to lubricate any part of the anaesthetic machine.

73. Which part of the pressure regulator is amenable for wear and tear?

The large diaphragm which is situated inside the regulator is amenable for wear and tear. The leakage if any is present in the regulator is caused by the worn out large diaphragm which has to be changed by authorized personnel.

5. Flow meters:

74. What is the name of the flow meter in use in an anaesthetic machine?

Rotameter

75. What is the name of the flow meter tube?

Thorpe tube

76. What is the speciality in Thorpe tube?

It is a conical tube with the diameter increasing from the bottom to top

77. How the bobbin is made to rotate in the flow meter?

These are slanting cuts in the base of the bobbin called



"flutes". When the gas flows across the flutes, it imparts a spinning action to the bobbin.

78. Why the bobbin has to rotate?

A rotating bobbin indicates gas flow, which differentiates from a struck bobbin.

79. What are the types of bobbins?

Ball type, H type, skirted bobbin, Non-skirted, plumb bob.

80. What are the reasons for the sticky, non-rotating bobbins in spite of gas flow?

1. Slanting flow meter tubes
2. Dusts in the gases
3. Static electricity

81. What is the type of flow in the flow meter when gas flows in low flow range?

It is a tubular flow pattern because the gap between the bobbin and the wall of flow meter tube is tubular in the low flow range. The length of the gap is more than the diameter of the gap.

82. In high flow range, what is the flow type?

It is an orificial flow because the diameter of the gap is more than the length.

83. Which factors decide the amount of flow in the tubular flow (low flow range)?

Apart from the pressure gradient, viscosity of the gas decides the amount of flow.

84. Which factors decide the amount of flow in the high flow range?

Pressure gradient and density.

85. Give an example for variable orifice flow meter?

Rotameter

86. Give an example for fixed orifice flow meter?

Bourdon Pressure gauge.

87. What is the impact of high altitude on the flow meter?

In high altitude, flow meter will deliver more gas than indicated in the scale. As the flow of gas through the flow meter depends upon the physical factors like density and viscosity, these physical factors of the gases can vary in high altitudes. The flow meters are

calibrated for a particular density/viscosity at the factory conditions. If they are taken to high altitudes, the flow will also be affected.

88. What do you mean by static electricity? Why it is important for an Anaesthesiologist?

It is nothing but accumulation of electrons on a material surface because of continuous friction. This leads to a development of negative charge compared to the object nearby. When the potential difference exceeds certain limit, the electrons tend to jump through the air creating a spark. This spark can ignite an inflammable anaesthetic mixture.

Static electricity development near the bobbin also retards the rotation of the bobbin during gas flow.

89. How the accumulation of static electricity can be avoided in the flow meter tube?

By incorporating Tin Oxide during the manufacture of pyrex glass tubes, it can be made conductive. The electrons will pass through this conducting tube to the metal parts of Boyle's machine and will get discharged thus avoiding a fire accident.

By spraying antistatic sprays like croxetene or sphirol-H on the glass tubes.

90. What is the difference between flow control knob of O₂ and other gases?

Oxygen flow control knob projects outside more than the other knobs so that it can be operated easily. It is touch coded (has broad serrations), colour coded (White) and level coded.

91. Summarise the safety measures in the flow meter assembly:

1. Flow control knobs are touch and colour coded
2. Oxygen flow control knob projects out from outlet knobs
3. Oxygen in the down stream
4. ORMC
5. Link-25
6. Minimum mandatory flow of O₂ - 200 -300 ml.
7. Luminescent back screen
8. Luminescent dot in the bobbin which helps to identify the rotation of the bobbin
9. Bobbin stop at the top and bottom end of flow meter tube.



92. Why Oxygen is preferred to be placed downstream of all the flow meters?

If there is a leak in the other flow meters, if Oxygen is placed upstream it will also go out through the leak. If placed downstream, oxygen flow will not be affected if there is leak in other flow meters.

93. In some of the new machines, Oxygen flow meter is found to be placed in the upstream? What is the reason?

Baffles are provided after the flow meter for every gas so that one gas cannot enter in to another gas flow meter. It doesn't matter where the oxygen flow meter is kept as it is directed by baffle system to the end of the flow meter sequence.

94. What is the purpose of Link-25?

The flow control valves of oxygen and Nitrous oxide are interlinked mechanically by chained gear system. If oxygen flow control valve is operated separately, the gear will not get engaged with N₂O. So Oxygen flow meter can be operated individually. Operator can rise the O₂ flow meter at his will.

If N₂O flow meter is attempted to give a flow, Oxygen flow meter will also get opened because of the gear system. The operator will not be able to give N₂O alone. If both the gases are flowing, and the operator reduces the O₂ flow automatically N₂O flow will also get reduced. Oxygen is ensured to flow at a minimum percentage of 25% along with N₂O.

95. When the gases exit from the pressure regulators, the pressure will be around 50-60 psi. Then where will this pressure get reduced before it goes to the patient? Pressure drops at the flow control valve of the flow meters drastically.

96. Why the link-25 is named so?

It is a mechanical link between O₂ and N₂O flow meters so that minimum 25% O₂ flow is ensured at all times.

97. What do you mean by 'Minimum mandatory flow of O₂'?

Some anaesthesia machines are fitted with this option which gives an oxygen flow of 200 ml/min even after closing the Oxygen flow control valve so that hypoxic mixtures will not be delivered at any time.

This didn't become popular as the 200 ml/min flow

dries up the soda lime even when the machine is not in use.

98. What do you mean by ORMC-Oxygen Ratio Monitor Controller?

This controller monitors the oxygen and N₂O pressure across a diaphragm and cuts off N₂O if oxygen pressure drops below 10 psi.

6.Vaporizer:

99. What do you mean by saturated vapour pressure?

It is the maximum partial pressure that can be achieved by a vapour at any given temperature. For a given temperature, some of the molecules of a volatile liquid escape in to vapour phase. The pressure created by these vapour is called "Vapour pressure". When the vaporization of that liquid stabilizes (Meaning the number of vapour molecules leaving the fluid and the number of molecules getting dissolved back in to the liquid are same), the vapour pressure measured at that point is called "Saturated Vapour Pressure".

100. What is the SVP of desflurane at 20° C?
664 mm of Hg

101. What is the impact of atmospheric pressure on the SVP of a volatile substance?
Atmospheric pressure doesn't affect the SVP.

102. How will you calculate the amount of liquid anaesthetic used by the vaporiser per hour?
EhranWorth and Eisen Kraft formula = ml of liquid used per hour = 3 x fresh gas flow in L/min x dial setting (V/V%)

103. What are the boiling points of commonly used inhalational anaesthetics?
Halothane: 50° C, Isoflurane: 48.5° C, Sevoflurane: 58.5° C, Desflurane:22.8°C.

104. What are the differences between draw over and plenum vaporisers?
Draw over vaporisers are low resistant, bidirectional flow vaporisers which are mainly used with patients on spontaneous ventilation. Eg: Goldman vaporiser

Plenum vaporisers are high resistant, unidirectional flow vaporisers which needs positive pressure gas flow for its proper functioning.



105. What are the main advantages of flow over vaporizers?

The design is simple, cheap and having least resistance for the gas flow. They are ideal for the patients who are left on spontaneous ventilation as the resistance is very low.

106. What are the disadvantages of flow over vaporizers?

The vapour output decreases after some time of usage, even with a constant dial position.

107. What are the reasons for the decreased vapour output in a flow over vaporizer on usage?

The continuous vaporization which occurs after usage brings out a fall in the temperature of the remaining liquid anaesthetic agent. This fall in the temperature of the liquid decreases the vaporization and reduces the vapour output.

108. How can the reduction in the vapour output in the flow over vaporizer be tackled?

There are many methods to tackle this reduction in the vaporization process.

1. By making the fresh gas flow bubbling through the liquid anaesthetic agent.

2. By incorporating a mechanism which increases the flow of fresh gas in to the vaporization chamber once the temperature of the system falls. This thermo compensation mechanism could be a bellows or a strip of metal. This mechanism acts in a way that when it gets cooled, it tends to shrink allowing more fresh gas to enter.

3. By using a thermo buffer mechanism like water jacket which can supply some heat to the liquid anaesthetic agent.

109. What do you mean by “latent heat of vaporization”?

The latent heat of vaporization of a given liquid is the heat energy required to convert one kilogram of the liquid into its vapour at its boiling point without any change of its temperature. It is measured in Joules per kilogram.

110. What is the significance of latent heat in anaesthesia?

A liquid with less latent heat energy is more volatile. It can get easily vaporized in clinical conditions. Usually the latent heat of vaporization is supplied by the

remaining liquid anaesthetic and the container of the vaporizer. If more latent heat is supplied by the remaining liquid, then it will get cooled fast and the vaporization process will tend to decrease as the vaporization entirely depends upon the temperature.

111. What do you mean by “Specific heat capacity” of a substance? What is the importance of it in anaesthesia?

The quantity of heat required to raise the temperature of one gram of a substance by one celcius degree. The units are Joules per gram per Celcius degree. Any substance will require calories to raise its temperature as per the above definition. At the same time, it will lose energy on cooling. The same amount of energy will be released on cooling. As the container material gets cooled during the evaporation of the liquid anaesthetic, it tends to give some energy to the liquid from its specific heat capacity. So more the specific heat capacity of a material, more the energy it gives. The material, selected to be used for the construction of the vaporizer needs to have a good amount of specific heat capacity.

112. Why thermal conductivity gains importance in selecting a material to fabricate the vaporizer?

On vaporisation of the liquid anaesthetic, the materials around it get cooled. The heat from the environment has to get transmitted to the liquid to sustain the vaporization. So the material of construction should have high thermal conducting property to enable this.

113. On considering all the physics involved in the construction of vaporizer, which metal seems to be ideal for the construction of a vaporizer?

Copper with its moderate specific heat capacity (385 J/Kg/CO) and good thermal conductivity fits into the groove well. Additionally it is malleable and rust proof.

114. What do you mean by “Vaporizer inside the circle” and “Vaporizer outside the circle”?

Positioning the vaporizer in the back bar is called “Vaporizer outside the circle”. That means it is outside the circle absorber circuit. It takes some more time to saturate the circle system with the anaesthetic vapour when the vaporizer is outside the circle. When it is positioned in the expiratory limb of the circle system near the canister, it is called “Vaporizer inside the circle”. The saturation of the circuit takes place faster than the previous one.



115. What is another main difference between vaporizer inside the circle (VIC) and vaporizer outside the circle system (VOC) vaporiser?

Vaporiser output can be more than the dial setting in the VIC vaporiser as the inflowing expiratory gas into the vaporiser in the circle contains anaesthetic vapour.

116. What change will you make in the vaporiser dial setting in high altitudes?

There need not be any change in the dial setting of the vaporiser. If we calculate the volume percentage delivered by vaporiser in the high altitudes, there will be an apparent increase of vaporiser output. But the number of molecules getting vaporised at that temperature doesn't change and the partial pressure remains the same. So we need not change the dial setting.

117. Describe the "Aladin Cassette" vaporiser?

Plenum, variable by-pass, acts as an injector when cassette contains only vapour, flow over with wick, electronically temperature compensated, out of system, agent specific and colour coded vaporizer.

118. What is different with the desflurane vaporiser?

It is a plenum, dual circuit gas vapour blender, with thermo compensation provided by supplied heat. It is an absolute pressure generator.

119. Why does desflurane need a special vaporiser?

The SVP of desflurane is 664 mm of Hg at 20°C. Large volumes of desflurane vapour will be available at room temperature to get mixed with the fresh gas. The potency of desflurane is modest. So to give a clinical useful concentration (4-6%), lot of fresh gas has to be used. If very high fresh gas flow is used, rapid vaporisation of desflurane will lead to loss of latent heat of vaporisation. Thus vaporisation will be unpredictable. So a heated coil converts the liquid agent into vapour and a measured flow is mixed with fresh to make the final desired concentration.

120. What are the safety measures in the vaporisers?

- Colour coding indicator on the vaporiser
- Agent specific filling systems with sealed bottles
- Agent level indicators
- Interlocking mounting system to prevent simultaneous use of two vaporisers.
- Audio-visual alarm system in electronic vaporisers.
- Technologies to overcome the effect of pumping

effect.

121. Why simultaneous use of two vaporisers is to be discouraged?

When two vaporizers are open at the same time, the inhalational agent in the upstream will enter in to the next vaporizer. The inhalational agent in the upstream, if it is allowed to enter the downstream vaporiser, it may condense in it. This leads to contamination of the downstream vaporiser which leads to inadvertent administration of the contaminated agent to the patient.

122. What is a "Trilene lock" in the older type of Boyle's machine?

Trilene bottle which was fitted in the back bar of the Boyles machine was not allowed to get operated when closed circuit with soda lime canister is in use. This lock is linked with the opening lever of the Trilene Vaporiser and the control knob of the back bar which selects the open and closed circuit. This lock was designed to prevent the inadvertent use of Trilene with the soda lime which leads to the formation of toxic gases- Phosgene and carbon monoxide.

123. Name two inflammable inhalational agents?

1. Di-ethyl ether
2. Cyclopropane.

124. What is a "Schimmelbusch" mask?

It is a wire frame mask with a gutter to collect excess liquid agent, over which gauze or lint is applied and the liquid anaesthetic agent like ether or chloroform is dropped by a drop bottle. It was used to administer open drop ether/ chloroform anaesthesia.

125. Name one ether liquid drop bottle?

Bellamy- Gardner bottle.

126. Describe the Goldman vaporiser?

It is a draw over, non-temperature compensated, multiple agents, non-calibrated, VIC or VOC vaporiser.

127. Describe Epstein-MacIntosh Oxford vaporizer:

Draw over, variable by-pass, flow over, temperature compensation provided by water bath and agent specific vaporizer. Designed for field use.

128. What is the thermostat kept in the E.M.O vaporizer which acts as a controller of outflow of ether vapour from the vaporizing chamber?



A small metal bellows containing liquid ether attached to a spindle acts as the controlling mechanism.

129. What is the name of the vaporizer designed for Trilene based on the principle of EMO vaporizer?
EMOTRIL.

130. Who discovered the Oxford Miniature Vaporizer?
Epstein-MacIntosh introduced OMV in the year 1966. Four agents could be administered individually with that vaporizer by selecting a swivel knob.

131. What do you mean by "Hyderabad cone"?
The device in the shape of a cone was used to investigate the effects of chloroform by Hyderabad Chloroform commission, headed by Edward Lawrie.

132. Name one measured flow vaporizer in which liquid anaesthetic agent bubbles through the liquid?

Copper kettle vaporizer meant for ether.

133. What do you mean by "pumping effect" seen in vaporisers?

The back pressure created by ventilators or manual squeezing of reservoir bag forces some of the gas exiting the outlet port of a vaporizer back into the vaporizing chamber, where more vapour is added. Retrograde flow may also contaminate the bypass channel. These effects cause an increase in the inspired concentration of the agent which may be harmful to the patient. This is called "pumping effect"

134. How will you minimise the effects of "pumping effect"?

1. By keeping a long inlet port into the vaporising chambers

2. By keeping a flow restrictor downstream after vaporisers.

3. By avoiding the wicks near inlet port

4. By decreasing or minimising the size of bypass chambers.

5. By keeping a one-way valve after the vaporisers, preventing the back flow.

135. What do you mean by "pressurising effect"?

As described in pumping effect, the gas is pumped back into the vaporising chamber when reservoir bag is squeezed. This returned gas may not pick up additional vapour, as vapourization depends upon temperature of the remaining fluid rather than presence of excess gas. This in turn dilutes the vapour concentration thereby reducing the vapour output. This is in contrast to the pumping effect.

136. In which clinical situations these effects occur?

In high flow of fresh gases with high dial setting, pressurising effect occurs whereas in low flow anaesthesia, pumping effect operates.

137. What is an "Ana Con Da" device?

This system "Anaesthetic Conserving Device" was designed to be used in ICU for sedating the intubated and ventilated patients. It's a modified HME filter where liquid anaesthetic is supplied by a specialised syringe pump.

138. What is the preservative used in halothane and what is its significance?

Thymol is the preservative in halothane which causes stickiness of the vaporiser dial and bimetallic strips in the Tec series vaporisers.

139. What is the preservative used in Isoflurane?

Isoflurane doesn't have a preservative.

140. In some of the vaporizers, we can see an English letter 'T' after the zero mark of the dial. What does it mean?

If the dial is set at this "T" mark, the vaporizer can be removed from the back bar and may be transported to another machine. During this transport, if the dial is set at this "T" mark, the vaporizer becomes tip proof. Even if the vaporizer is inverted, the liquid agent cannot come in to the by-pass chamber.

141. What is the colour coding of vaporizers?

Sevoflurane - Yellow Isoflurane - Purple
Halothane - Red Desflurane - Blue

142. What is Heliox?

Heliox is a mixture of oxygen and helium. A mixture of 21% oxygen and 79% of helium, named Heliox is used to improve gaseous exchange in acute exacerbation of asthma and COPD. As this mixture is less dense than room air, it flows better through narrow orifices. Hence it is used to treat the hypoxia induced by upper airway obstruction as in carcinoma larynx.

7. Anaesthesia ventilators:

143. What are the types of anaesthesia ventilators?

- Ascending bellows type
- Descending bellows type
- Piston type



144. On what basis the anaesthesia ventilators are named as ascending or descending type?

They are named after the status of them during the expiratory cycle. Ascending bellows ventilator descends during inspiration and ascends during expiration. The descending bellows ventilator does it in opposite direction

145. Which type of anaesthetic ventilator is safe?

Ascending bellows type is safe because the bellows fail to rise in an event of circuit disconnection which will be noticed by the attending Anaesthesiologist immediately.

146. What is the advantage of having a descending type of anaesthesia ventilator?

Descending bellows ventilators allow ventilation with air during gas supply failure, as they can suck the atmospheric air.

147. How the ventilator bellows are compressed during inspiration?

The compression of the bellows is done by the compressed oxygen from the cylinder/pipeline which comes in a separate line without getting mixed with the patient's fresh gas. After compressing the bellows, this driving gas is let out through a separate exit valve.

148. Will there be a mixing of fresh gas and driving gas in bellows ventilators?

No. There will be no mixing of these gases. There will be separate circuits for both of them.

149. How much of oxygen is used to drive the ventilator?

It varies with the different kind of workstations. It typically varies between 3 to 6 litres of oxygen per minute.

150. What do you mean by "ventilator-fresh gas flow coupling"?

As the fresh gas flow from the flow meter is continuous, it is getting added to the ventilator set tidal volume. This is called as "fresh gas coupling".

151. What do you mean by "Fresh gas decoupling"?

During the inspiratory phase, the fresh gas from the flow meter will be sent to a collecting reservoir bag by a decoupling valve, so that this gas is not added to the inspiratory gas. This is called "fresh gas decoupling".

During expiratory phase, this valve opens and allows the collected fresh gas to go into the ventilator bellow.

152. What are the advantage and disadvantages of Fresh gas decoupling?

Advantages: Decreased risk of barotrauma and volutrauma

Disadvantages:

a) Generation of negative pressure in the patient's airway, which may entrain the atmospheric air through the relief valve.

b) Reliance on the reservoir bag as gas accumulator.

153. What will happen to the ventilator pressure dynamics in a patient with pulmonary oedema?

In pulmonary oedema, there is decrease in pulmonary compliance. So there will be rise of both peak inspiratory pressure and plateau pressure.

154. What will happen to the ventilator pressure dynamics in a patient with bronchospasm?

There will be steep rise of peak inspiratory pressure without a raise in plateau pressure.

155. What is the advantage & disadvantage of piston type ventilators?

It is precise and less noisy. It doesn't require a driving gas and so economical. But as it is not visible, it may not act as a visible monitor for proper ventilation.

156. In what way, the anaesthesia ventilators are better than hand ventilation?

As they produce constant ventilation with constant tidal volume and respiratory rate, the CO₂ level is uniform without the chances for hypo or hyperventilation. And they are fatigue free. But the bellows type of ventilators consumes lot of oxygen as driving gas.

8. Breathing circuits:

157. Which anaesthetic agent prompted the development of circle system?

Cyclopropane and Acetylene.

158. What is the primary function of reservoir bag?

It acts as a reservoir of gases so that Peak Inspiratory Flow Rate (PIFR) is met with.



159. How will you classify the breathing circuits?

- Breathing system with CO₂ absorber
- Breathing systems without CO₂ absorber

I. Without CO₂ absorber

- uni-directional flow
- Non Re-breathing system
- Circle absorber without absorber
- bi-directional flow
- Afferent reservoir system
- Enclosed afferent reservoir system
- Efferent reservoir
- combined system

II. Breathing system with CO₂ absorption

- unidirectional flow
- bidirectional flow

160. How is CO₂ eliminated in ICU Ventilator? (Which breathing circuit is used in ICU Ventilator?)

ICU Ventilators use non-rebreathing circuits. There is no mixing of inspiratory and expiratory gases. Expiratory gas goes to atmosphere. There is no need for CO₂ absorption as there is no rebreathing in ICU ventilator.

161. What do you mean by Non-rebreathing valves?

It's a one-way valve which ensures unidirectional flow of gases. This valve lets the inspiratory gas to go in to the patient's lungs and expiratory gas to go out to the atmosphere.

162. Can you tell some examples of Non-rebreathing valve?

Ruben valve, Stephen-Slater valve and Leigh Valves.

163. Where are the Non-rebreathing valves commonly used now?

These valves are commonly used in the Manual Resuscitator bags like AMBU bag.

164. What are the different types of mechanism by which the unidirectional flow of gases is ensured in these non-rebreathing valves?

Spring disc, spring ball, spool with a spring, Flap valve, Fish mouth valve, Diaphragm flap or Mushroom flap valve mechanisms. The flap/disc/ball is pushed by the inspiratory gas from the bag against a spring thus allowing the gas go in to the patient. Once the expiration is allowed, the spring pushes the flap/ball/disc back in to the original position thus allowing the expiratory gas to go out to the

atmosphere.

165. What do you mean by "Forward creep"?

In a non-rebreathing valve, when inspiration occurs during controlled ventilation, the spool in the non-rebreathing valve has to completely close the expiratory port. If it doesn't close properly, the part of inspiratory gas escapes into the expiratory port without going to the patient's lung. This is called forward creep.

166. Describe the Heidbrink valve:

These valves are designed for Mapleson type of circuits for the expulsion of the expiratory gases during spontaneous breathing. It has a disc which opens to atmosphere when the circuit pressure exceeds 2 cm of H₂O. This pressure rise occurs during the expiratory phase of the spontaneously breathing patient, thereby allowing the expiratory gas to go out to the atmosphere. During inspiration, it gets closed by the push of a spring which is behind the disc. This valve plays an important role in the CO₂ elimination of spontaneously breathing patients.

167. Up to which point, the apparatus dead space extends in a Magill's circuit?

Up to the APL valve (Heidbrink's valve)

168. Up to which point, the apparatus dead space extends in a closed circuit?

Up to the bifurcating point in the Y piece

169. Through which tube the expiratory gases traverse in a co-axial Lack system?

Through the inner tube

170. Which is the most efficient system among Mapleson circuits for spontaneous ventilation? What is the minimal FGF in that circuit?

Mapleson A or Magill circuit is the most efficient circuit for spontaneous ventilation. The FGF should be at least equivalent to patient's alveolar ventilation.

171. Which circuit can be called as "controlled rebreathing circuit?"

Bains circuit

172. In Ayre's T piece, what should be the length of the expiratory limb?

The length of the expiratory limb should be equal to the patient's tidal volume.



173. What are the factors deciding the CO₂ elimination from the Mapleson breathing circuits?

1. Fresh gas flow rate
2. Minute ventilation
3. Mode of ventilation
spontaneous/controlled
4. Tidal volume
5. Respiratory rate
6. I:E ratio
7. Duration of end-expiratory pause
8. Peak inspiratory flow rate
9. Volume of the breathing tube

174. How do you check the integrity of the inner tube of the Bain's circuit?

By performing Pethick test. An already filled reservoir bag collapses fast, when emergency O₂ flush is activated to produce fast O₂ flow at the patients end. This gas flow will deflate the bag because of venturi effect.

175. Explain the Smith-Foex-Crampton manoeuvre to test for the integrity of the Bain circuit:

Keep a flow of 2 litre oxygen with a Bain circuit. Occlude the inner tube end at the patient's end with your finger or a pen. The oxygen bobbin has to dip a little because of the conducted pressure back through the inner tube of the circuit. If it doesn't go down on occlusion, it means there is a leak in the inner tube of the Bain circuit or in the back bar.

176. Why it is necessary to check for the leak in the inner tube of Bain circuit?

If there is a leak in the inner tube, the fresh gas will enter the circuit in the midway of the breathing hose. This will trap the expired gas between the leak point and the original fresh gas end. This trapped expired gas will get re inhaled to produce hypercapnia. That's why it becomes necessary to check for the leak in the inner tube.

177. What are the primary factors which decide the CO₂ elimination in Bain circuit?

1. Amount of fresh gas flow
2. Duration of the end-expiratory pause.

178. What do you mean by Bain hump in the capnography?

The down slope of the capnographic trace may show a small increase of CO₂ concentration because of the

rebreathing of the gas during the beginning of inspiration in Bain circuit. A small amount of exhaled alveolar gas gets trapped between the fresh gas entry and endotracheal tube. This gas will appear in the capnographic trace as the inspiration begins.

179. What is the suggested fresh gas flow rate in a Bain circuit?

Fresh gas flow: 150 to 200 ml/kg/min during spontaneous ventilation
100 to 150 ml/kg/min during controlled ventilation

180. If you desire to induce hypocapnia with a Bain circuit, what will you do? Will you increase the respiratory rate or fresh gas flow?

Fresh gas flow has to be increased with adequate end-expiratory pause. Increasing the respiratory rate will decrease the duration of end-expiratory pause thereby favouring rebreathing of the expiratory gases.

181. What are the components of circle system?

Soda lime canister forms the back bone of the circle system. Fresh gas entry, reservoir bag, inspiratory and expiratory uni-directional valves, corrugated breathing tubes, Y adapter and expiratory spill valve.

182. Among these components which is the most important one in a closed circuit?

The uni-directional valves are the one which impart the circular path to the anaesthetic gases. Their malfunctioning leads to accumulation of CO₂.

183. What is the ideal position of the APL valve (Expiratory Spill valve)?

It should be placed in the expiratory limb before the canister so that expiratory gas, rich in CO₂ will be lost to the atmosphere before entering into the canister. This extends the life of soda lime.

184. What is the ideal size of soda lime canister?

Various machines have various sizes of canister. Ideally it should accommodate at least one tidal volume of the patient. Larger canister last long without necessitating frequent soda lime change. But desiccation can occur on prolonged usage. Accumulation of toxic materials like Compound-A can occur. So small size canisters are preferred. Small size canisters reduce the total internal volume of the closed circuit. This will help in saturating the circuit fast with the anaesthetic agents.



185. What is the influence of closed circuit volume on the conduct of anaesthesia?

The higher internal volume of the circuit increases the time taken for the induction and recovery from inhalational anaesthesia. If the circuit volume is 10 L and the fresh gas flow is 2L, then it will take 5 minutes to saturate 65% of the circuit (Time taken = Circuit volume/fresh gas flow). It will take another 5 minutes to saturate 85% of the volume of the circuit. And it takes another 5 minutes to saturate 96% of the circuit volume. In total, it will take 3 time constants (15 minutes in that example) to saturate the circuit reasonably.

186. Name any two inhalational agents which are not compatible to soda lime:

Trilene (An inhalational agent of historic interest) reacts with soda lime and forms toxic gases like phosgene and acetylene.

Sevoflurane to certain extent reacts with soda lime and forms Compound-A which is nephrotoxic.

187. What are the favourable conditions for the formation of Compound-A with sevoflurane inhalation through a closed circuit?

1. Prolonged sevoflurane inhalation
2. Low flow anaesthesia
3. Dry, desiccated soda lime
4. High temperature production with soda lime
5. High alkali content. (Sodium and Potassium hydroxide)

188. What is the purpose of corrugation in the breathing hoses?

1. To make it kinking resistant
2. To increase the surface area of contact between the dry and cold fresh gas and atmosphere. The fresh gas attains atmospheric temperature when travelling in the breathing hoses.

189. Why the old time breathing tubes were black in colour?

These rubber hoses were made anti-static by adding carbon in it. That's why they are looking black. A yellow line running in these hoses indicate that they are made antistatic.

190. What is the compliance of the adult breathing circuits?

It is usually 5ml/cm of H₂O - If an airway pressure of 20 cm is reached, the breathing circuit will expand to hold 100ml without delivering it to the patient.

191. Give some examples of breathing systems which can be used for both spontaneous and controlled ventilation.

1. J-R modification of Ayre's T piece
2. Bain circuit
3. Humphrey ADE breathing system
4. Closed circuit with CO₂ absorbent.

192. What is the modification added to Ayre's T piece by Jackson-Rees?

He added an open ended bag to the expiratory limb of Ayre's T piece.

193. What is the fresh gas flow recommended for the Ayre's T piece?

For spontaneous ventilation: 2 to 3 times the minute volume of the baby.

Controlled ventilation : 1.5 to 2 times the minute ventilation.

194. What are the most important factors which decide the CO₂ elimination in Ayre's T piece?

1. Fresh gas flow
2. Duration of end-expiratory pause
3. The length of the expiratory tube.

195. What are the advantages of Ayre's T piece?

It is a light weight circuit which doesn't drag the endotracheal tube much. As there are no valves, the resistance for spontaneous breathing is less.

196. Which face mask is ideal for the paediatric patients?

Rendall-Baker-Sousek masks are ideal as they have lesser dead space volume. Scented, transparent masks are also suitable for paediatric patients.

197. What do you mean by "Maxima" circuit?

It is an enclosed afferent reservoir system, designed by Miller in 1995.



14th JUNE 2026
05:25PM - 05:40PM

CHEST X-RAY -VIVA-VOCE

Introduction

Chest X-ray is one of the most commonly performed and essential bedside investigations in anaesthesia, critical care, emergency medicine, and perioperative practice. A systematic approach to chest radiograph interpretation is extremely important for postgraduate students, especially during viva voce examinations, clinical discussions, and emergency situations.

Many times, important findings are missed because the film is viewed in a random manner. Hence, using a structured flow-chart approach helps in developing a consistent, organized, and reproducible method of interpretation. This flow chart is designed to simplify chest X-ray reading by guiding the examiner through a stepwise assessment - starting from patient identification and film quality, followed by airway, lung fields, cardiac shadow, diaphragm, mediastinum, pleura, bones, and finally tubes and lines. For anaesthesia postgraduates, this approach is particularly useful in identifying perioperative complications such as endobronchial intubation, pneumothorax, pleural effusion, pulmonary edema, aspiration, lung collapse, and malposition of central venous catheters or other devices.

By following this systematic method, we can improve diagnostic accuracy, avoid missing critical findings, and present chest X-ray interpretation confidently during viva voce and clinical practice.

Present a simple algorithm:

Patient Details



Check X-ray Quality (RIPE)



Airway



Breathing (Lungs & Pleura)



Cardiac Silhouette



Diaphragm



Everything Else (bones, tubes, lines)

Use the ABCDEF rule

1. Confirm Patient & Film Details

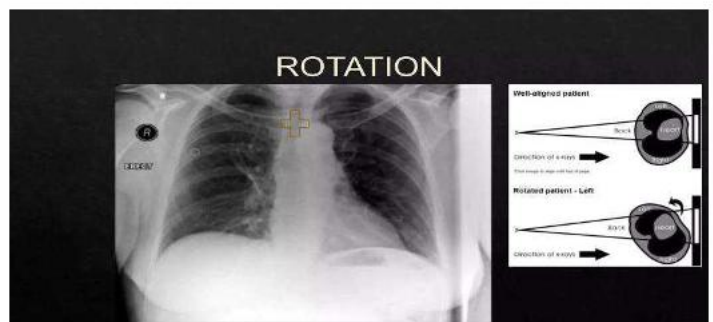
Start systematically—this avoids basic errors:

- Name, age, ID
- Date & time (compare with previous films)
- View: PA / AP / Portable / Lateral
- Indication (trauma, ICU, pre-op, line placement)

2. Check Image Quality (RIPE Approach)

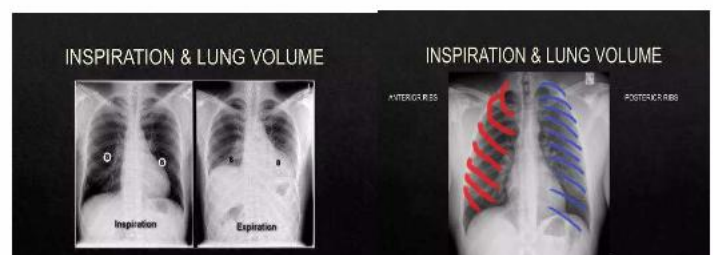
R - Rotation

- Medial ends of clavicles equidistant from spinous processes
- Rotation can mimic mediastinal shift.



I - Inspiration

- Count ribs: ≥ 6 anterior or ≥ 10 posterior ribs visible
- Poor inspiration $\rightarrow$ false cardiomegaly, basal opacities



P - Projection

- PA (preferred) vs AP (ICU films $\rightarrow$ heart appears enlarged)



E – Exposure

- Vertebrae just visible through cardiac shadow
- Overexposed → lung markings lost
- Underexposed → appears falsely opaque

• **Adequate exposure:** Inter-vertebral spaces barely visible through the heart shadow

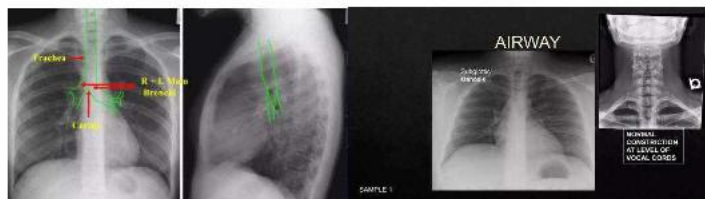


Over-exposed film	Under-exposed film
Inter-vertebral spaces clearly visible through heart shadow	Inter-vertebral spaces clearly visible through heart shadow

3. Systematic Review (ABCDE Approach)

A – Airway

- Trachea: midline or deviated?
- Carina visible?
- Check ETT position:
 - ▶ Ideal: 2–5 cm above carina
- Look for bronchial obstruction



B – Breathing (Lungs & Pleura)

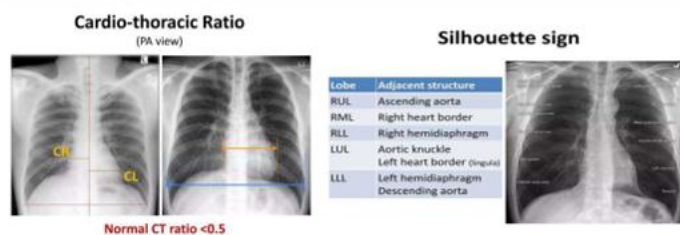
Compare both sides:

- Symmetry of lung fields
- Opacities:
 - ▶ Consolidation (pneumonia)
 - ▶ Collapse (atelectasis)
 - ▶ Interstitial patterns
- Pleura:
 - ▶ Pneumothorax (absent lung markings)
 - ▶ Effusion (meniscus sign, blunting of costophrenic angle)



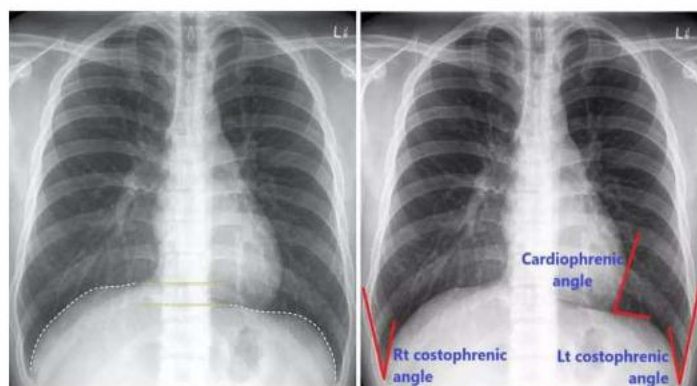
C – Cardiac

- Cardiac size (CTR < 50% in PA film)
- Borders:
 - ▶ Loss of silhouette → localizes pathology
- Shape abnormalities



D – Diaphragm

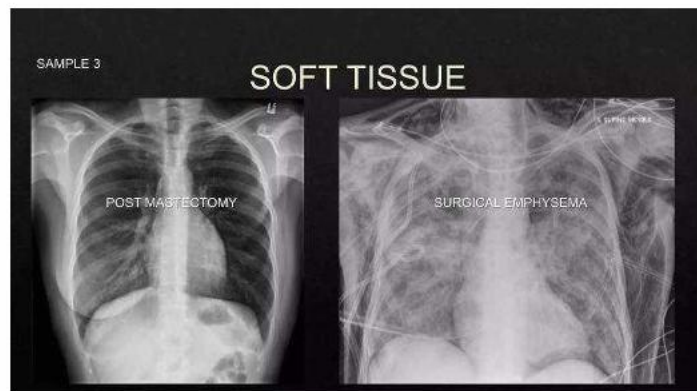
- Right dome higher than left (normal)
- Free air under diaphragm → perforation
- Flattening → COPD
- Elevated diaphragm → phrenic nerve palsy, collapse



E - Everything Else [bones, tubes, lines]

Soft tissues:

- Subcutaneous emphysema

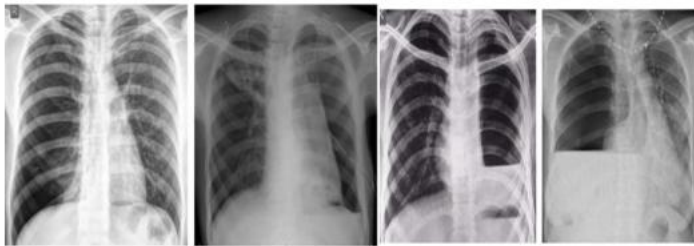


4. Deadly X-ray findings an anaesthesiologist should never miss"

1. Tension pneumothorax
2. Massive pleural effusion
3. Malpositioned ETT and Central line (tip at SVC-RA junction)
4. Mediastinal widening
5. Pulmonary edema

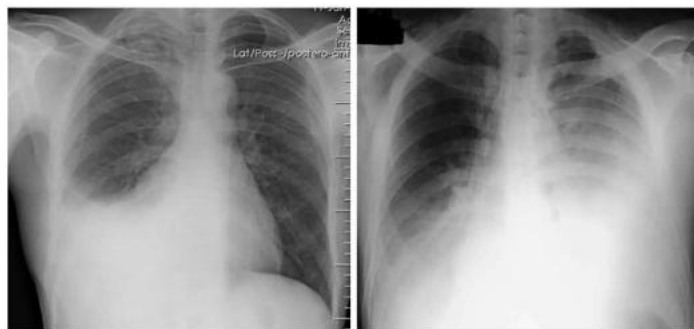
Sample Cases

Pneumothorax



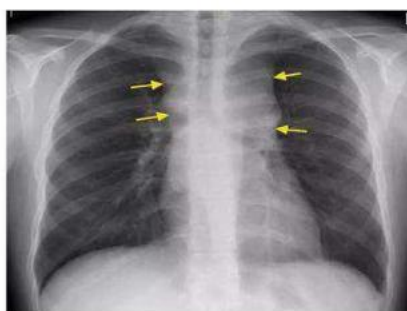
Hydropneumothorax

Pleural Effusion



Mediastinal widening

Definition:
Mediastinum width greater than 6 cm on erect PA view or 8 cm on supine AP view



Pulmonary Edema



OTHER CHEST PATHOLOGY

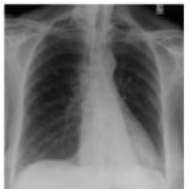
Consolidation



Consolidation

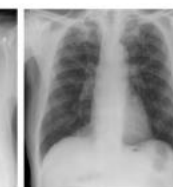
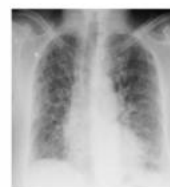


Collapse



Collapse

Fibrosis



Lung Mass

Solitary Pulmonary Nodule (Coin lesion)

- Granulomas: tuberculoma, histoplasmosis, aspergilloma
- Bronchial carcinoma
- Bronchial adenoma
- Lung abscess
- Encysted pleural effusion
- Pseudotumor
- Pulmonary hamartoma
- Hydatid cyst
- Rheumatoid nodule
- Wegner's nodule



40% SPN are malignant

Miliary lesions

- Miliary tuberculosis
- Sarcoidosis
- Pulmonary eosinophilia
- Histoplasmosis
- Pneumoconiosis
- Hemosiderosis
- **Miliary metastasis**
thyroid, renal, breast, prostate, osteosarcoma



Pulmonary Metastasis



Miliary nodules: <2 mm
Pulmonary nodule: 7-30 mm

Pulmonary micronodule: 2-7 mm
Pulmonary mass: >30mm

Hilar Lymphadenopathy

Bilateral hilar lymphadenopathy

- Sarcoidosis
- Lymphoma
- Tuberculosis
- Histoplasmosis
- Pneumoconiosis: silicosis

Unilateral hilar lymphadenopathy

- Lymphoma
- Carcinoma
- Tuberculosis
- Histoplasmosis



Hemithorax

Mediastinum pushed away from the opacified side

- Pleural effusion
- Large lung mass
- Diaphragmatic hernia

Mediastinum pulled toward the opacified side

- Total lung collapse
- Pneumonectomy
- Pulmonary hypoplasia/agenesis

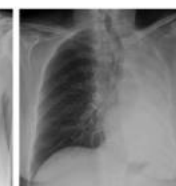
Mediastinum remains central in position

- Consolidation
- Pleural/chest wall mass
- Combination of pathologies

Hemithorax



Hemithorax



Pulmonary Cavity

- **Carcinoma**
- **Autoimmune:** Wegner granulomatosis and rheumatoid nodule
- **Vascular:** emboli (septic/bland)
- **Infection/abscess:** bacterial (Klebsiella, Staphylococcus, anaerobic infections), fungal (histoplasmosis), amebic, hydatid cyst
- **Trauma:** pneumatocele
- **Young:** congenital, bronchogenic cyst



Pulmonary Cavity



Pathognomic sign ???

Emphysema

Pulmonary Tuberculosis

- Apical or posterior segment of upper lobes or superior segments of lower lobes mostly involved
- **Active tuberculosis:** Infiltrates, consolidations, cavities, mediastinal or hilar lymphadenopathy
- **Healed tuberculosis:** Pulmonary nodules, fibrotic scars, bronchiectasis and pleural scarring



Cardiac X-rays

- Right ventricular enlargement
- Left ventricular enlargement
- Mitral stenosis
- Congestive heart failure
- Pericardial effusion
- Pulmonary hypertension



Right Ventricular Enlargement

- CT ratio >0.5
- Cardiac apex is round and elevated above diaphragm
- Cardio-phrenic angle is acute



Congestive Heart Failure

- Increased interstitial markings
- Upper zone vascular redistribution
- Bilateral Pleural effusion
- Cardiomegaly (LV type)



Left Ventricular Enlargement

- CT ratio >0.5
- Cardiac apex displaced downwards & to left
- Cardio-phrenic angle is obtuse & merges with diaphragm



Congestive Heart Failure

- "Bat-wing" appearance
- Kerley B lines
- Cardiomegaly (LV type)
- Min pleural effusions



Mitral Stenosis

- Cardiomegaly (RV type)
- Straightening of left heart border
- Double right heart border
- Splaying of carinal angle



Pericardial Effusion

- CT ratio >0.5
- Globular heart shadow
- "Water bottle" sign



Mitralisation of heart

Mitralisation of heart means straightening of the left border of heart

1. Aortic knuckle: small
2. Pulmonary conus: enlarged
3. Left atrial appendage: prominent
4. Left border of left ventricle: no change

Pulmonary Hypertension

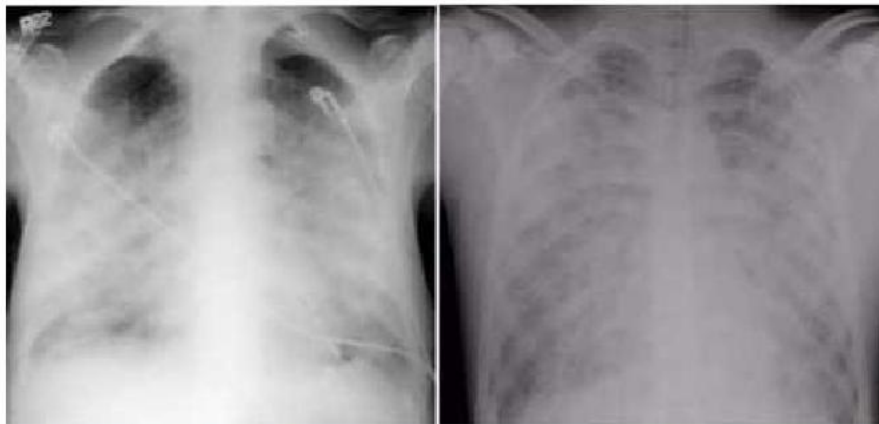
- Enlarged pulmonary arteries
 - ❖ >16 mm right descending pulmonary artery (PA view)
 - ❖ >18 mm left descending pulmonary artery (lateral view)
- Prominent pulmonary outflow tract
- Peripheral pulmonary vessels pruning
- Right ventricular hypertrophy



Guess what ???



TETRALOGY OF FALLOT



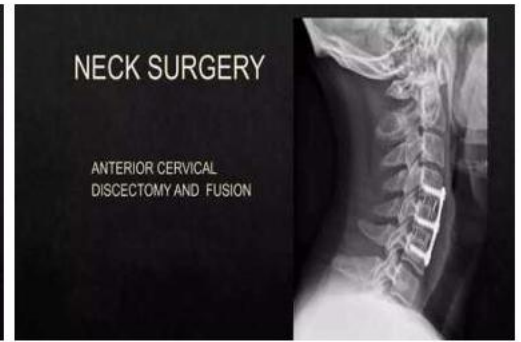
Radiographic feature	Cardiogenic pulmonary edema (LVEF)	Noncardiogenic pulmonary edema (ARDS)
Heart size	Enlarged	Normal
Vascular distribution	Balanced or inverted	Normal or balanced
Distribution of edema	Even or central	Patchy or diffuse
Pleural effusion	Present	Not usually present
Peribronchila cuffing	Present	Not usually present
Septal lines	Present	Not usually present
Air bronchograms	Not usually present	Usually present

X RAY OF NECKS

Certain conditions that affect the Craniocervical junction and cervical spine are clinically relevant to anaesthetists. Cervical spine injuries occur in 2%-5% of blunt trauma patients and of these 7%-14% are unstable.

Indications for Anaesthetist

- Thyroid patients
- Prevertebral mass/Swelling
- TMJ Ankylosis
- Retropharyngeal abscess
- FB neck



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14th JUNE 2026

05:40PM - 05:55PM

PULMONARY FUNCTION TESTS

Compiled by

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Introduction

Pulmonary Function Tests (PFTs) provide objective assessment of respiratory mechanics, airflow, lung volumes, gas exchange, and exercise capacity. They are invaluable in diagnosing respiratory disease, monitoring progression, evaluating treatment response, and assessing perioperative pulmonary risk. In anaesthetic practice, PFTs help identify patients at increased risk of postoperative pulmonary complications and guide perioperative optimization.

Postoperative pulmonary complications remain a major cause of perioperative morbidity and mortality. Accurate assessment of respiratory function is therefore essential, particularly in patients undergoing thoracic, upper abdominal, and major surgical procedures. PFTs quantify respiratory impairment and assist in risk stratification.

Uses of Pulmonary Function Testing

- Diagnose respiratory disorders.
- Differentiate obstructive and restrictive disease.
- Assess disease severity.
- Monitor progression and treatment response.
- Assess disability.
- Evaluate perioperative risk.
- Determine suitability for lung resection surgery.

Classification of PFTs

1. Bedside pulmonary function tests
2. Static lung volumes and capacities
3. Dynamic spirometry tests
4. Flow-volume loops
5. Diffusion studies (DLCO)
6. Gas exchange assessment
7. Cardiopulmonary reserve testing

INDICATIONS AND CONTRAINDICATIONS FOR PULMONARY FUNCTION TESTS

Indications	contraindications
Diagnostic purpose • Dyspnoea • Chronic cough • Wheeze • Asthma, • COPD, • Interstitial lung disease occupational lung disease	• Recent myocardial infarction • Unstable angina • Active pneumothorax • Thoracic aneurysm • Recent thoraco-abdominal surgery, recent ophthalmic surgery • Severe haemoptysis
Prognostic indications • monitoring disease progression and treatment response	
Perioperative indications- • thoracic surgery, upper abdominal surgery, smokers with symptoms, and unexplained breathlessness	

Bedside Pulmonary Function Tests

- **Sabrasez Breath Holding Test:** Patient takes a full breath and holds it. Normal cardiopulmonary reserve (CPR) is >25 secs. Limited is 15-25 secs. <15 secs indicates very poor CPR and is a contraindication for elective surgery.
- **Schneider's Match Blowing Test:** Measures maximum breathing capacity (MBC). Patient blows out a match from 6 inches away with mouth wide open (no pursing). Inability indicates MBC < 60 L/min and FEV1 < 1.6L.
- **Greene and Berowitz Cough Test:** Assesses ability and strength of cough. A wet, productive cough or paroxysms indicate susceptibility to post-op pulmonary complications. Vital capacity must be ~3 times Tidal Volume for an effective cough.
- **Single Breath Count:** Patient takes a deep breath and counts as far as possible before the next breath. Normal is 30-40 counts. It acts as an indicator of vital capacity.
- **De Bono Whistle Blowing Test & Wright Peak Flow Meter:** Used to measure Peak Expiratory Flow Rate (PEFR). Normal PEFR for males is 450-700 L/min, and 350-500 L/min for females.



PULMONARY FUNCTION TESTS

Classified broadly into Static and Dynamic lung function tests.

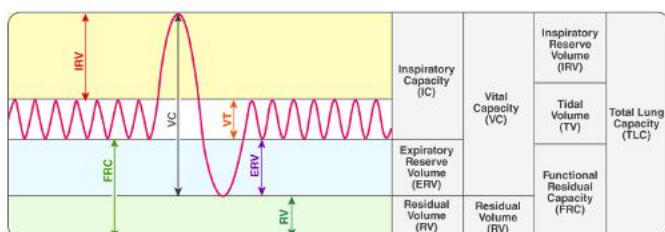
Static tests evaluate the absolute dimensions of the lungs. Because a person cannot voluntarily exhale all the air in their lungs, standard spirometry cannot measure these metrics, requiring specialized testing environments

Dynamic tests are dependent on time and the patient's breathing effort. They are the frontline tools for identifying if there is a blockage or limitation in the airways. Measure the The volume of air exhaled forcefully and the speed at which it is expelled

Feature	Dynamic Lung Function Tests	Static Lung Function Tests
Primary Focus	Airflow rates and exhalation speed.	Absolute lung capacities and volumes.
Key Parameters	FEV ₁ , FVC, FEV ₁ /FVC ratio, VCmax.	TLC (Total Lung Capacity), RV (Residual Volume), FRC (Functional Residual Capacity).
Primary Diagnostic Use	Identifying obstructive patterns and assessing disease severity.	Diagnosing restrictive patterns, lung hyperinflation, and air trapping.
Standard Method	Forced spirometry (flow-volume curve).	Plethysmography (body box), gas dilution, or mini-box.

Static Lung Volumes and Capacities

Static tests measure the total volume of air in the lungs at various phases of the breathing cycle, irrespective of how fast the air is moving. Tidal Volume (TV), Inspiratory Reserve Volume (IRV), Expiratory Reserve Volume (ERV), Residual Volume (RV), Vital Capacity (VC), Functional Residual Capacity (FRC), Inspiratory Capacity (IC) and Total Lung Capacity (TLC) are the derivatives.



Terminology	Explanation	Normal Values (M.) mL	Normal Values (F.) mL
Tidal volume	Volume of air inspired or expired at each breath.	660 (230)	550 (160)
Inspiratory reserve volume	Maximum volume of air that can be inspired after a normal inspiration.	2240	1480
Expiratory reserve volume	Maximum volume of air that can be expired after a normal expiration.	1240 (410)	730 (300)
Residual volume	Volume of air remaining in the lungs after a maximum expiration.	2100 (520)	1570 (380)
Vital capacity	Maximum volume of air that can be expired after a maximum inspiration.	4130 (750)	2760 (540)
Total lung capacity	The total volume of air contained in the lungs at maximum inspiration.	6230 (830)	4330 (620)
Inspiratory capacity	The maximum volume of air that can be inspired after a normal expiration.	2900	2030
Functional residual capacity	The volume of gas remaining in the lungs after a normal expiration.	3330 (680)	2300 (490)

Table 1 Differences between obstructive and Restrictive lung disease in spirometry

	OBSTRUCTIVE	RESTRICTIVE
Vital capacity	N or ↓	↓
Total lung capacity	N or ↓	↓
Residual volume	↑	↓
FEV ₁ /FVC	↓	N or ↑
Maximum midexpiratory flow rate	↓	N
Maximum breathing capacity	↓	N

↓, decreased; ↑, increased; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; N, normal.

The TLC is increased in obstructive lung disease and decreased in restrictive lung disease. However, TLC cannot be obtained by routine screening spirometry.

Clinical importance of FRC

- Oxygen reservoir during apnea
- Prevents alveolar collapse
- Maintains oxygenation
- Decreases work of breathing

Reduced FRC occurs in obesity, pregnancy, supine position and general anaesthesia.



Methods of Measuring RV and FRC

Nitrogen washout technique, helium dilution technique and body plethysmography. Body plethysmography is based on Boyle's law and is the most accurate technique. (Refer Wylie 5th, Pulmonary ventilation)

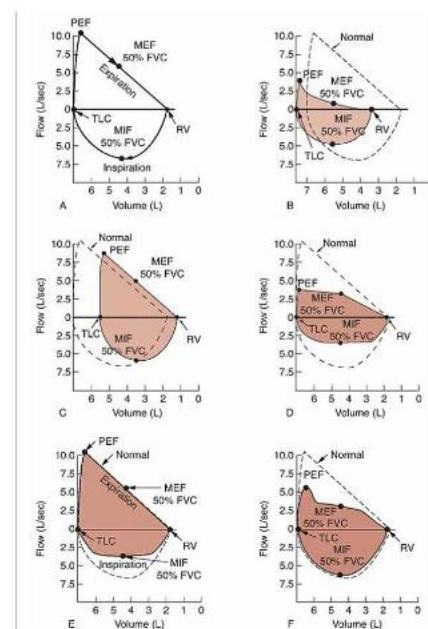


FIGURE 1.3 Flow-volume loops in a healthy person (A), a patient with obstructive lung disease (B), a patient with restrictive lung disease (C), a patient with a fixed obstruction (D), a patient with a variable intrathoracic obstruction (E), and a patient with a variable extra-thoracic obstruction (F). FVC, forced vital capacity; MEF, maximal expiratory flow; MIF, maximal inspiratory flow; PEF, peak expiratory flow; RV, residual volume; TLC, total lung capacity. (From *The Merck Manual: Professional Edition*, with permission. http://www.merckmanuals.com/professional/pulmonary_disorders/tests_of_pulmonary_function_pft/airflow_lung_volumes_and_flow-volume_loop.html?q=flow%20volume%20loops&alt=sh. Accessed November 11, 2014.)

DYNAMIC LUNG FUNCTION TESTS

Flow Volume Loops

Flow-volume loops provide a graphic analysis of flow at various lung volumes. To perform the test, subjects are asked to inhale maximally (to TLC) and then exhale as forcefully and maximally as possible (to RV). This cycle is repeated. The flow (L/sec) is plotted on the y-axis and volume (L) on the x-axis (Fig. 1.3A). The test requires a compliant patient for accurate results—the majority of the test is effort dependent, including the entire inspiratory curve and both ends of the expiratory curve (near TLC and RV).

The forced expiratory flow during 25% to 75% of VC (FEF25%-75%), is reflective of the small to medium sized airways and is considered a relatively effort-independent value.

The derivatives are -Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV1), FEV1/FVC ratio, Peak Expiratory Flow Rate (PEFR), Forced

Expiratory Flow 25–75% and Maximum Voluntary Ventilation (MVV).

(Refer to images on F-V loops, referred from book - Yao & Artusio)

Severity of Airflow Obstruction based on FEV

FEV1 >80% predicted – Mild

50–79% predicted – Moderate

30–49% predicted – Severe

<30% predicted – Very severe

Bronchodilator Testing

An increase in FEV1 by at least 12% and 200 mL following bronchodilator administration indicates significant reversibility and supports the diagnosis of asthma.

Simple analysis

Fixed obstruction – flattening of inspiratory and expiratory limbs.

Variable intrathoracic obstruction – flattening of expiratory limb.

Variable extra-thoracic obstruction – flattening of inspiratory limb.

Restrictive disease – narrow loop with reduced lung volumes

Approach to Interpretation

Step 1: Assess technical quality.

Step 2: Examine FEV1/FVC ratio.

Step 3: Determine severity.

Step 4: Assess lung volumes.

Step 5: Evaluate DLCO.

Step 6: Correlate clinically.

Assessment of Gas Exchange

The alveolar-arterial oxygen gradient assesses ventilation-perfusion mismatch. Increased values occur in diffusion defects, pulmonary embolism, chronic bronchitis and shunt physiology.

1. Diffusing Capacity (DLCO)

DLCO assesses transfer of carbon monoxide across the alveolar-capillary membrane.

Reduced DLCO	Increased DLCO
• Interstitial lung disease • Pulmonary fibrosis • Emphysema • Pulmonary embolism • Anaemia	• Polycythaemia • Exercise • Pulmonary haemorrhage



2. Cardiopulmonary Exercise Testing

CPET is the gold standard assessment of functional capacity.

Important parameters:

- VO₂ max
- Anaerobic threshold
- Oxygen pulse
- Ventilatory equivalents

VO₂ max <15 mL/kg/min predicts increased perioperative risk.

3. Six Minute Walk Test and Shuttle Walk Test

The 6-minute walk test measures functional exercise capacity. Distances below 600 m indicate reduced reserve. Shuttle walk distances below 250 m are associated with increased perioperative risk.

Preoperative Assessment Before Thoracic Surgery for lung resection

Predicted postoperative FEV₁ (ppoFEV₁) and DLCO are calculated to estimate residual pulmonary function after lung resection.

$$\text{ppoFEV}_1 = \text{Preoperative FEV}_1 \times \frac{\text{Remaining Functional Segments}}{19}$$

Risk: >40% predicted – acceptable risk
 30–40% – intermediate risk
 <30% – high risk requiring detailed CPET evaluation.

Important Viva Questions

1. Define spirometry.
2. Why cannot residual volume be measured by spirometry?
3. What is functional residual capacity?
4. What happens to FRC and CC during anaesthesia?
5. Define DLCO.
6. Explain bronchodilator reversibility testing.
7. What is ppoFEV₁?
8. What is the significance of CPET?
9. Differentiate obstructive and restrictive disease.
10. What factors reduce FRC?
11. Explain flow-volume loop abnormalities.
12. What is closing capacity?

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14th JUNE 2026
05:40PM - 05:55PM

CAPNOGRAPHY

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Capnography is the continuous measurement and graphical display of carbon dioxide concentration during the respiratory cycle.

Terminologies

Capnography

The numerical measurement and graphic waveform display of the CO₂ concentration versus time or expired volume.

Capnograph

The machine that generates the waveform.

Capnogram-

continuous graphic waveform over time

Capnometry-

The measurement and numerical display of maximum inspiratory and expiratory CO₂ concentrations during a respiratory cycle.

Capnometer –

The device that performs the measurement and displays the reading

Principles of Capnography

Infrared spectroscopy forms the basis of most capnographs. Carbon dioxide absorbs infrared light at a characteristic wavelength, allowing estimation of CO₂ concentration.

Types of Capnography

Mainstream and sidestream capnography are the two major systems.

Sampling Techniques:

- Sidestream: Samples gas via an aspirator at 50-200 mL/min. It is advantageous for spontaneously breathing patients and positioning flexibility, though prone to slight delays (flight time) and potential leaks.

- Mainstream: Positions the sample chamber directly within the breathing circuit, offering zero delay and minimal leakage, but adds weight to the circuit and requires heating to prevent condensation

Feature	Sidestream Analysers	Mainstream Analysers
Positioning	Aspirates gas from the circuit via a sampling tube to a separate chamber.	Sensor cuvette is placed directly within the patient's breathing circuit.
Flow Dynamics	Optimal sampling rate is 50-200 mL/min.	Does not remove gas from the circuit.
Response Time	Subject to a delay (flight time) dependent on tube length and flow rate.	Extremely fast response time with zero flight-time delay.
Advantages	Easily used in non-intubated patients receiving supplemental SO_2 ; ease of sterilization.	Minimal leakage risk; no vacuum pumps required.
Disadvantages	Vulnerable to water/secretions; high flow rates may entrain fresh gas or trigger mechanical breaths.	Bulky sensors add dead space and weight, increasing the risk of endotracheal tube (ETT) kinking or accidental extubation; requires heating to prevent condensation.
Feature	Sidestream Analysers	Mainstream Analysers
		requires heating to prevent condensation.

The Normal Capnogram Physiology

The timed Capnogram is broken down into distinct physiological phases:

- Phase I: Exhalation of anatomic dead space gas containing no CO₂.
- Phase II: A rapid S-shaped upstroke representing the mixing of dead space and alveolar gas.



The slight positive slope occurs because under-ventilated alveoli (low V/Q ratio) empty slower and contribute to a rising CO₂ concentration. The peak of this phase is the End-Tidal CO₂ (PETCO₂).

- Phase IV: The rapid descent to baseline as inspiration begins.

- Key Angles: The alpha angle (normally 100-110°) indirectly indicates V/Q status, while the beta angle (normally 90°) can indicate rebreathing if increased.

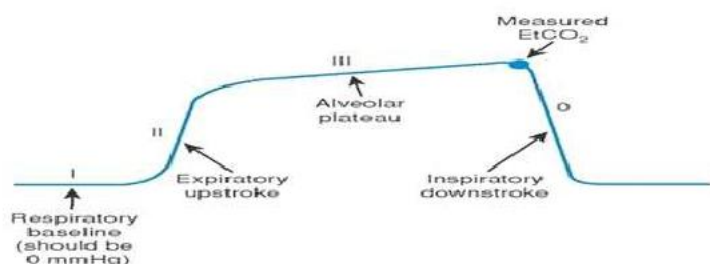
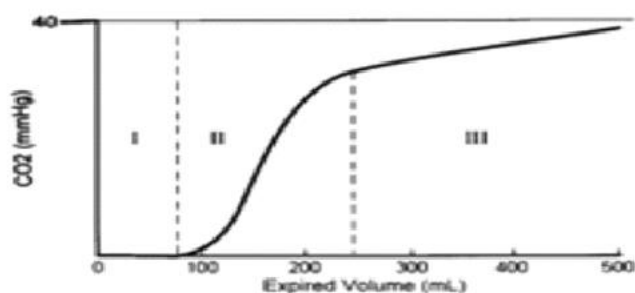


Figure : Normal Capnogram



Volume Capnogram Plots CO₂ against expired volume and helps assess dead space ventilation and V/Q mismatch.

Clinical Applications

1. Endotracheal Intubation: Verifying correct placement of the endotracheal tube. (blind nasal or oral intubation, needle placement during transtracheal circothyrotomy, Placement of Double lumen tube)
2. Procedural Sedation: Ongoing assessment of breathing patterns during sedation.
3. Mechanical Ventilation: Tracking patient response to adjustments in ventilator settings.
4. General Anesthesia: Observing lung function and cardiac output throughout anesthesia.
5. Cardiopulmonary Resuscitation (CPR): Evaluating the quality of chest compressions and helping predict CPR outcomes. Initial PETCO₂ predicts CPR success: below 8 mmHg means <1% survival; above 18 mmHg means 70% higher survival chance.

WAVEFORM INTERPRETATIONS{FIGURE 3}

Clinical Condition	Capnographic Presentation	Pathophysiological Mechanism
Bronchospasm / Asthma	Prolonged Phase II, steeper Phase III slope, increased alpha angle (often "shark-fin" appearance).	Delayed, asynchronous emptying of alveoli due to high expiratory airway resistance.
Rebreathing	Elevated baseline above zero.	Inadequate fresh gas flow, exhausted CO ₂ absorbent, or incompetent expiratory valve. ⁸⁰⁹¹
Curare Clefts	Small downward notches ("clefts") observed in the Phase III alveolar plateau.	Spontaneous diaphragmatic contraction attempting to draw a breath as neuromuscular blockade begins to wear off.
Hypermetabolic States	Abrupt or gradual increase in overall ETCO ₂ HEIGHT.	Increased cellular CO ₂ production (e.g., malignant hyperthermia, shivering, reperfusion after tourniquet release).
Systemic Leakage	Gradual or sudden loss of the waveform; decreasing ETCO ₂	Circuit disconnection, ventilator malfunction, or sampling tube leak.

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Systemic Leakage	Gradual or sudden loss of the waveform; decreasing ETCO ₂	Circuit disconnection, ventilator malfunction, or sampling tube leak.

Advantages	Limitations
Non-invasive, continuous, real-time, sensitive useful for early detection of respiratory events.	Affected by V/Q mismatch sampling errors condensation secretions equipment malfunction.

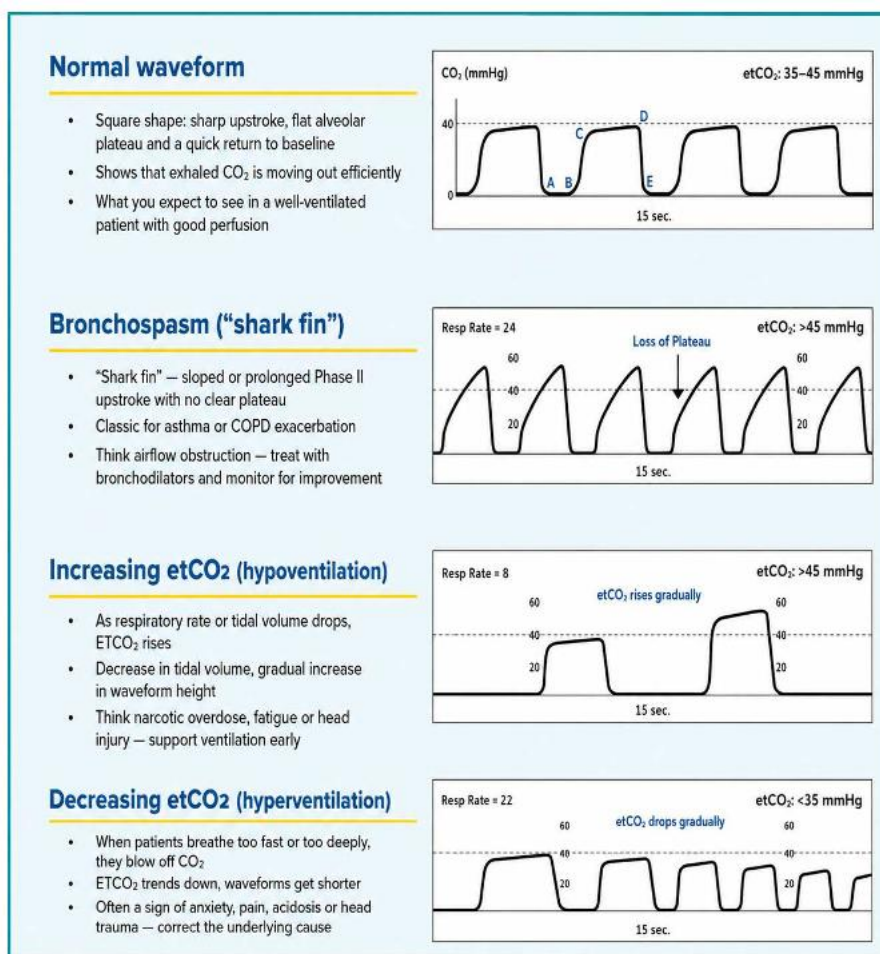
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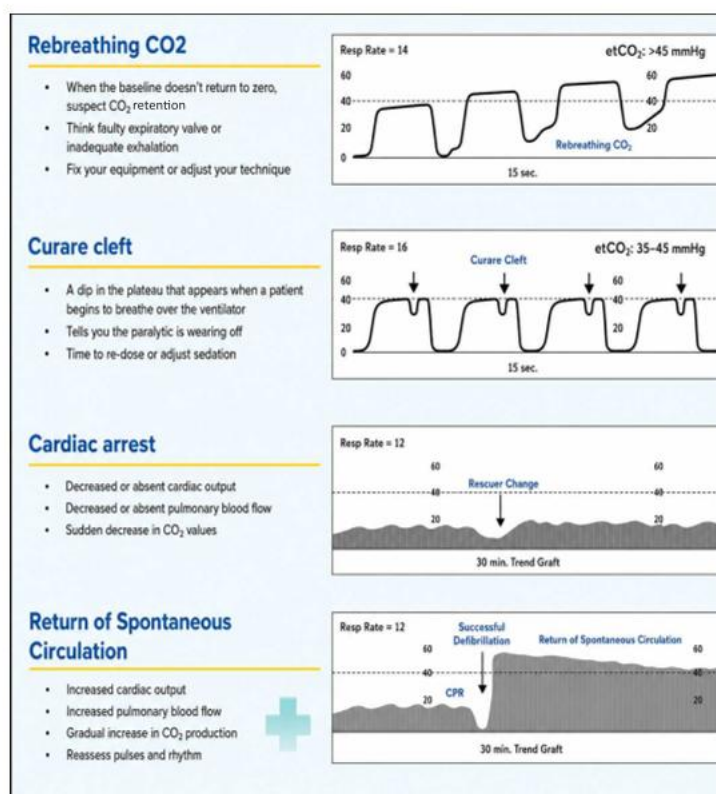


4. Jessica A. Lovich-Sapola, , Capnography, Anesthesia Oral Board review

Figure . WAVEFORMS INTERPRERTATION



[adopted from EMS 1- Infographic: Understanding normal and abnormal capnography waveforms]



[adopted from EMS 1- Infographic: Understanding normal and abnormal capnography waveforms]



14th JUNE 2026
05:55PM - 06:10PM

Arterial blood gas (ABG) analysis

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Arterial blood gas (ABG) analysis is an essential part of diagnosing and managing a patient's oxygenation status and acid-base balance

Indications for obtaining a peripheral arterial blood sample for ABG analysis:

- To evaluate adequacy of a patient's oxygenation, ventilatory, acid-base status, oxygen carrying capacity, and/or intrapulmonary shunt
- To assess the results of a therapeutic intervention or diagnostic evaluation
- To assess adequacy of circulation, as in situations of shock, poor cardiac output, during cardiopulmonary resuscitation, and after cardiopulmonary bypass
- To evaluate the impact of a dyshemoglobinemia (abnormal hemoglobin structure or behavior) on oxygenation

Interpretation:

- Assess internal consistency
- Evaluate each major category of results:
 - ▶ Oxygenation
 - ▶ Ventilation
 - ▶ Acid-base status

Assess Internal Consistency:

- Instrumental and other laboratory errors in managing the blood sample can occur before proceeding to medical analysis of the ABG; a first step is to evaluate the consistency of the reported values
- Using the modified Henderson-Hasselbalch equation, calculate the expected approximate H^+ concentration (nmol/L)
 - ▶ $H^+ = 24(PaCO_2) / HCO_3^-$

- Compare the calculated H^+ with the expected pH (Table 2)

- If the numbers are approximately consistent, proceed with interpretation

- If the numbers are inconsistent, the ABG result is not valid. Alert the laboratory and repeat the sample

- Table 2. **Estimate of arterial pH versus hydrogen ion concentration (nmol/L).**

pH	H^+ (nmol/L)
7	100
7.05	89
7.1	79
7.15	71
7.2	63
7.25	56
7.3	50
7.35	45
7.4	40
7.45	35
7.5	32
7.55	28
7.6	25
7.65	22

Ventilation Assessment:

- Evaluate for hypercarbia or hypocarbia (ie, hypercapnia or hypocapnia)
 - ▶ Hypercarbia
- $PaCO_2$ above reference range (generally more than 45 mm Hg)
- Underlying mechanism for respiratory acidosis include but are not limited to:



- Decreased removal of CO₂ by the lungs (eg, decreased tidal volume of breathing, decreased frequency of breathing, apnea, and increased dead space, as in some chronic pulmonary diseases)

- Increased inspiration of CO₂ (ie, rebreathing on a ventilator or other oxygen delivery device) or nonpulmonary intake of CO₂ (eg, a higher CO₂ mixture is "dialed in" in the sweep gas at the membrane oxygenator on a machine, during either cardiopulmonary bypass or extracorporeal membrane oxygenation)

- Increased entry of CO₂ into the blood from the tissues (eg, after restoration of circulation following cardiopulmonary resuscitation, when CO₂ that has built up in body tissues due to lack of circulation is suddenly released into the bloodstream when circulation is restored)

- Increased production of CO₂ (eg, fever). Note: increased production of CO₂ (eg, during fever, sepsis, malignant hyperthermia, or thyrotoxicosis) seldom raises CO₂ significantly in the presence of normal respiratory status—hyperventilation is usually sufficient to lower CO₂ to within normal limits⁵¹

▶ Hypocarbica

- PaCO₂ below reference range (generally less than 35 mm Hg)

- Almost always due to increased removal of CO₂ by the lungs (eg, increased tidal volume of breathing or increased frequency of breathing), either due to changes in respiratory effort, or to mechanical hyperventilation of a patient receiving respiratory assistance

Acid-Base Approach

Figure 5. The Boston approach to acid-base assessment.

Delta ratio = $\Delta AG / \Delta HCO_3 = (AG - 12) / (24 - HCO_3)$. AG, anion gap. From Neligan P. Perioperative acid-base balance. In: Gropper MA et al, eds. Miller's Anesthesia. 9th ed. Elsevier; 2020:1524-1545, Figure 48.

- Various algorithms for assessing acid-base status

have been published. The suggested approach outlined in this Clinical Overview is based on the Boston approach by Peter Neligan⁵² (Figure 5)

- Four values on the ABG relate to acid-base status:³

- ▶ pH
- ▶ PaCO₂
- ▶ HCO₃⁻
- ▶ Base excess

- Physiologically normal pH is determined by the concentration of hydrogen ions (H⁺) in the blood, via the modified Henderson-Hasselbalch equation:^{38,52}

$$H^+ = 24 \times PaCO_2 / HCO_3^-$$

- For a change in pH value by 0.1, the H⁺ concentration changes approximately 20%

- Normal pH of arterial blood is between 7.35 and 7.45³

- Nomenclature of acid-base disorders³⁸

- ▶ Acidemia and alkalemia are conditions in which the pH value of the blood is less than 7.35 or more than 7.45, respectively

- ▶ Acidosis and alkalosis are the underlying processes that result in acid-base disturbances

Oxygenation Assessment

Detection of Hyperoxemia or Hypoxemia

PaO₂ is expressed in kilopascals. To convert to millimeters of mercury, multiply PaO₂ value by 7.5.

- Determine if hyperoxemia is present

- ▶ Defined as PaO₂ more than 100 mm Hg

- ▶ In some texts, PaO₂ levels more than 300 mm Hg are considered harmful it is debated whether lower levels of hyperoxemia may also be harmful

- ▶ Hyperoxemia can produce hyperoxia, or increased oxygen in the tissues, which may enhance increased production of reactive oxygen species with deleterious results. Hyperoxemia results in pro- and antiinflammatory responses in humans



- Hyperbaric treatment raising the PaO₂ to levels more than 100 mm Hg may be beneficial to wound healing and in treatment of gangrene

- On the other hand, hyperbaric treatment can also cause neurotoxicity

- Pulmonary toxicity has been seen at hyperbaric pressures, and at atmospheric pressure with longer exposure

- Mortality in ICU patients is lower if conservative oxygen therapy is used, for example²²

Determine if hypoxemia is present

- Normal average PaO₂ at sea level in healthy adults breathing room air is between 80 and 100 mm Hg. However, normal PaO₂ can vary with age

- PaO₂ of less than 80 mm Hg is generally defined as hypoxemia, whether the patient is breathing room air or receiving supplemental oxygen

- The SaO₂ threshold that defines hypoxemia varies with different authors from 88% to 95%²⁰

- Hypoxia is inadequate oxygen levels in body tissues, which may or may not be due to hypoxemia (low oxygen levels in the blood). For example, limb tissue hypoxia can occur even in the face of normal blood levels of oxygen if the circulation to the limb is obstructed

- The severity of hypoxia depends on the PaO₂ value, as well as the FIO₂ at the time of ABG sampling (Figure 2)⁷

- Remember: normal PaO₂ at higher elevations can differ significantly from that at sea level, so when analyzing ABG results, altitude must be taken into account

- ▶ Expected PaO₂ levels decrease with increasing altitude; PaO₂ for a healthy adult male breathing room air might be around 97 mm Hg at sea level but only 75 mm Hg at 1400 m altitude²⁴

Pseudohypoxemia

Can occur when the measured PaO₂ in the gas analyzer is lower than in the body, even though the blood sample was handled correctly. This can occur when leukocyte and/or platelet concentrations in the blood sample are extremely elevated, leading to significant oxygen consumption even if the sample is held on ice and analyzed quickly^{25,26,27}

Next Actions If Hypoxemia Is Found

Calculate PAO₂

- PAO₂ (partial pressure of oxygen in alveolar gas [mm Hg]) is estimated from the alveolar gas equation:

$$\text{PAO}_2 = (P_{\text{atm}} - P_{\text{H}_2\text{O}})(\text{FIO}_2) - (\text{PaCO}_2/R)$$

- ▶ PAO₂ = partial pressure of oxygen in the alveoli

- ▶ P_{atm} = atmospheric pressure

- ▶ P_{H₂O} = water vapor pressure (ie, the pressure at which liquid is in equilibrium with gas phase)

- ▶ FIO₂ = inspired oxygen concentration

- ▶ PaCO₂ = arterial partial pressure of carbon dioxide

- ▶ R = the average respiratory exchange quotient (0.8) (ie, the rate of CO₂ excretion to oxygen consumption)²⁹

- Sample PAO₂ calculation for a person breathing room air at sea level:

- Atmospheric pressure: Patm = 760 mm Hg (sea level)

- Partial pressure of water vapor: PH₂O = 47 mm Hg at 37 °C

- FIO₂ = 0.21 (room air)

- Partial pressure of CO₂ in the alveoli is estimated with PaCO₂/R

- Measured PaCO₂ = 40 mm Hg

- ▶ PAO₂ = (Patm - PH₂O)(FIO₂) - (PaCO₂/R)

- ▶ PAO₂ = (760 - 47)(0.21) - (PaCO₂/0.8)

- ▶ PAO₂ = 713(0.21) - (40/0.8)

- ▶ PAO₂ = 149.73 - 50 = 99.73 (or approximately 100)

Calculate alveolar-arterial oxygen difference, or AaDO₂

- Under normal conditions, a small portion of the blood



flow to the lungs does not reach air-containing alveoli (2%-3%),³ and some areas of lung are not fully ventilated, leading to a difference in $PAO_2 - PaO_2$ (ie, oxygen partial pressure in the alveoli versus in the blood) of 5 to 15 mm Hg (at sea level, breathing room air). This is called the alveolar-arterial oxygen difference, or $AaDO_2$

- Also known as the alveolar-arterial oxygen gradient, or A-a gradient; it can be used to assess, for example, the presence of pulmonary shunt and changes in cardiac output⁴²

- The normal A-a O_2 gradient increases with age, and can be estimated from the following equation:⁴¹

- $AaDO_2 = 2.5 + (0.21 \times \text{age})$

- ▶ Thus, a normal A-a gradient for an 80-year-old person would be approximately 19 mm Hg

- Actual $AaDO_2$ can be calculated from the ABG result by subtracting the PaO_2 from the estimated PAO_2 :

- $AaDO_2 = PAO_2 - PaO_2$

- As calculating $AaDO_2$ relies on calculating the PAO_2 , the same caveats regarding supplemental oxygen therapy during ABG sampling apply

- A difference between estimated and calculated $AaDO_2$ can indicate the presence of increased dead space, shunt, and/or $\dot{V}/\dot{Q}$ mismatch

Consider Other ABG-Derived Values Used to Evaluate Oxygenation

- PaO_2/FIO_2 ratio

- An alternative to $AaDO_2$ in defining hypoxemia, and is used to define acute respiratory distress in some clinical criteria⁷⁸

- A normal PaO_2/FIO_2 ratio is 300 to 500 mm Hg⁴³
 - Values less than 300 indicate abnormal gas

exchange

- Values less than 200 indicate moderate to severe hypoxemia⁴³
- Values less than 100 indicate severe hypoxemia⁸⁴³

- PaO_2/PAO_2 ratio

- Also sometimes used to define hypoxia⁴⁴⁵

- A normal PaO_2/PAO_2 ratio is 0.77 or 0.82 for young male adults breathing room air or 100% oxygen, respectively, and is little affected by age⁴⁴

- The ratio can be used to estimate the appropriate amount of supplemental oxygen to deliver and to reduce the number of ABG measurements during changes in the inspired oxygen

- These calculations assume that there is no change in $PaCO_2$, and that the PaO_2/PAO_2 ratio is stable when FIO_2 is altered (a concept that has been validated in clinical studies as being generally true).⁴⁵ Calculations assume sea level

Use AaO_2 Difference to Categorize Mechanism of Hypoxemia²⁰⁴⁷

- Increased AaO_2 difference

- ▶ Increased physiologic dead space

- Examples include:

- Obstruction of pulmonary arteries by embolism
 - Pulmonary vascular changes due to chronic lung disease
 - Completely consolidated alveoli as a result of pneumonia
 - Areas of pulmonary edema

- $\dot{V}/\dot{Q}$ mismatch

- Examples include:

- Pulmonary embolism
 - Intrinsic lung disease
 - Obesity



- Atelectasis
- Patient position
- Decreased tidal breathing
- Occupation of the pleural space with fluid or other substance that presses on the lung parenchyma (eg, effusion, empyema, tumor)
- Occupation of the alveoli by fluid (pulmonary edema)
- Increasing FIO_2 usually results in significant improvement in the PaO_2 , by raising the PAO_2 in perfused alveoli
- Interestingly, even with large pulmonary emboli, the main mechanism of hypoxemia appears to be $\dot{V}/\dot{Q}$ mismatch rather than shunt²⁰⁴⁹

• Shunt

- ▶ Examples include:
 - ▶ Intrapulmonary
 - ▶ Pulmonary edema
 - ▶ Completely consolidated pneumonia
- Extrapulmonary
 - ▶ Congenital intracardiac shunts (eg, tetralogy of Fallot, certain atrial septal defects, patent ductus arteriosus)
 - ▶ Intrapulmonary arteriovenous malformations
 - ▶ Hepatopulmonary shunt (hepatopulmonary syndrome)

• Resistant to increases in FIO_2

▶ Significant unrecognized shunt can be present in the face of normal arterial oxygen saturation, particularly if the patient is breathing supplemental oxygen

• Diffusion limitation

- ▶ Example: interstitial lung disease

• Low mixed venous O_2

- ▶ Example: low cardiac output state

• Normal A-a O_2 difference

- ▶ Hypoventilation
- As PACO_2 rises in hypoventilation, for each increase of

10 mm Hg, PAO_2 falls 12.5 mm Hg²⁰

- Examples: chronic pulmonary diseases, central hypoventilation, and drugs

▶ Decreased PIO_2 (inspired partial pressure of oxygen) or FIO_2

- Examples: high altitude (where PIO_2 is decreased) and iatrogenic cause (ie, the physician has lowered the FIO_2 on ventilatory settings)

Acid-Base Assessment

- Determine if acidemia or alkalemia is present

• Acidemia (pH less than 7.35)

▶ When acidosis occurs, it is accompanied by partially compensatory changes—a rise in HCO_3^- and alterations in respiratory function (accelerated rate and/or increased tidal volume) that lower PCO_2

▶ When the changes in HCO_3^- and PCO_2 follow, or are proportionally smaller than the change in pH, these are both called compensatory changes, and the acidosis is the primary disorder

• Alkalemia (pH more than 7.45)

▶ When alkalosis occurs, it is accompanied by partially compensatory changes—a fall in HCO_3^- and alterations in respiratory function (slower rate and/or decreased tidal volume) that raise PCO_2

▶ When the changes in HCO_3^- and PCO_2 follow, or are proportionally smaller than the change in pH, these are both called compensatory changes, and the alkalosis is the primary disorder

- If significant acidemia or alkalemia is present, this is usually also the primary disorder:18 that is, the patient who has severe acidemia usually has primary respiratory or metabolic acidosis with or without compensatory metabolic or respiratory alkalosis, and the patient who has severe alkalemia usually has primary respiratory or metabolic alkalosis with or without compensatory metabolic or respiratory acidosis



- The presence of a normal pH excludes acidemia and alkalemia, but it does not rule out acidosis or alkalosis. To fully characterize the patient's acid-base status, the clinician must also examine the PCO_2 , HCO_3^- , and anion gap

- A person can have mixed acidosis and alkalosis, but can only have either acidemia or alkalemia at any given time; those terms are mutually exclusive

Next Actions If an Acid-Base Disorder Is

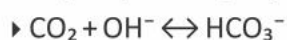
Use PaCO_2 to Determine If the Primary Disorder Is Respiratory or Metabolic

• Buffering systems

- ▶ These systems are means by which the body prevents rapid changes in acid-base status. Examples of buffering systems in the human body include:

• Chemical buffering⁸

- The formation of HCO_3^- occurs in the presence of carbonic anhydrase almost immediately after H^+ ions begin to change in concentration via 2 pathways:



• Respiratory system

- ▶ Responds within minutes to changes in H^+ ion concentration with changes in ventilation (ie, by changing the depth and frequency of respiration to remove CO_2 more quickly or slowly to compensate for upward or downward movements, respectively, in H^+ ion concentration)

• Renal system

- ▶ May not be maximally effective for up to 5 days after onset of acidemia/acidosis begins to alter the clearance of H^+ ions and the retention of HCO_3^- to compensate for changes in blood pH, and NH_3 (ammonia) combines with H^+ to form NH_4^+ (ammonium), which is excreted in urine

• Acute versus chronic disorders

- ▶ Acid-base disorders are also subcategorized as acute or chronic, based primarily on changes in the buffering systems as measured by pH, PaCO_2 , HCO_3^- , and base excess

• Acute disorders

- ▶ Heralded by changes in the acute buffering systems (ie, chemical buffering combined with changes in respiratory removal of CO_2) and are seen on ABG results primarily as changes in pH and PaCO_2

• Chronic disorders

- More longstanding and are seen on the ABG primarily as changes in pH, PaCO_2 , and HCO_3^-

- ▶ Note that respiratory compensation for metabolic acidosis and alkalosis occurs essentially immediately with the onset of the metabolic disorder. In other words, the respiratory response does not ramp up over time at a given pH, and does not depend on whether the metabolic disorder is acute or chronic

- ▶ Metabolic compensation for respiratory acidosis and alkalosis, on the other hand, does not reach full potential immediately, because it engages both chemical buffering systems and renal compensatory mechanisms (which take time to reach full potential). Thus, the alterations in HCO_3^- and PaCO_2 will change over time, and acute compensation values differ from chronic compensation values

- ▶ If the expected amount of compensation for either metabolic or respiratory acid-base disorders has not occurred, then usually 2 disorders are present together (eg, metabolic acidosis and respiratory acidosis)

- ▶ Note: compensation does not usually completely normalize the pH except in the physiologic chronic respiratory alkalosis of pregnancy

- Another simple approach to assessing whether the decrease in PaCO_2 is appropriate for the degree of metabolic acidosis is to look at the last 2 digits of the



pH—the last 2 digits should approximately equal the PaCO₂. For example, if the pH is 7.28, the expected compensatory value for PaCO₂ should be approximately 28 mm Hg³

- Assessing the severity of acid-base disturbances

► While it is tempting to judge the severity of an acid-base disturbance on the value of the pH alone, compensatory mechanisms that alter levels in PaCO₂ and HCO₃⁻ can be deceiving. It is important to evaluate all of these values (pH, PaCO₂, and HCO₃⁻) to determine severity.

Expected compensation for primary acid-base disorders.

Primary disorder	Compensatory response	Expected magnitude of response
Metabolic acidosis	? PCO ₂	PCO ₂ = $1.5 \times (\text{HCO}_3^-) + 8 \pm 2$
Metabolic alkalosis	? PCO ₂	PCO ₂ increases 6 mm Hg for each 10 mEq/L increase in HCO ₃ ⁻
Respiratory acidosis	? HCO ₃ ⁻	Acute: HCO ₃ ⁻ increases 1 mEq/L for each 10 mm Hg increase in PCO ₂ Chronic: HCO ₃ ⁻ increases 3.5 mEq/L for each 10 mm Hg increase in PCO ₂
Respiratory alkalosis	? HCO ₃ ⁻	Acute: HCO ₃ ⁻ falls 2 mEq/L for each 10 mm Hg decrease in PCO ₂ Chronic: HCO ₃ ⁻ falls 5 mEq/L for each 10 mm Hg decrease in PCO ₂

If a Metabolic Acidosis Is Present, Calculate the Anion Gap

- Anion gap calculation will require additional blood sample for electrolyte levels
- Anion gap is calculated by subtracting the total of chloride plus bicarbonate from the total of sodium plus potassium:
 - Anion gap = $(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$
- Note: some authors simplify the equation by eliminating K⁺, which is a relatively small contributor to the anion gap³
- Consider correcting for hypoalbuminemia if appropriate (note: correcting for albumin is controversial, and many authors consider it unnecessary, particularly because albumin values may not be readily available)⁵⁴
 - Anion gap is about 2.5 mEq/L lower for every 1 g/dL decrease in plasma albumin⁸



- Unmeasured anions (eg, albumin, lactate, pyruvate, ketones, phosphates, sulfates) account for a normal anion gap of between 8 and 16 mEq/L³

- ▶ Higher than normal anion gap values can indicate a source of acidosis related to one or more of the unmeasured constituents, or to toxins that are not normally found in blood in significant quantities (eg, ethylene glycol)

- ▶ Lower than normal or even negative anion gap values are unusual, and can indicate, for example, the following:

- ◆ Laboratory error
- ◆ Lower than normal serum anion levels (eg, hypoalbuminemia)
- ◆ Higher than normal serum cation levels (eg, Ca^{2+} , Mg^{2+} , Li^{+})
- ◆ Paraproteinemia (eg, multiple myeloma)

- If anion gap is elevated, consider the relationship between the increase in anion gap and the change in HCO_3^-

- ▶ The ratio of the change in HCO_3^- to the change in anion gap should be between 1 and 2

- ◆ If the ratio is less than 1, then a concurrent non-anion gap metabolic acidosis is likely to be present with a positive anion gap metabolic acidosis

- ◆ If the ratio is more than 2, then a concurrent metabolic alkalosis is likely to be present

Consider Possible Causes of Identified Disorders

- Respiratory acidosis

- ▶ Alveolar minute ventilation is inadequate relative to CO_2 production (resulting in hypercarbia) due to:

- Decrease in the rate of CO_2 removal from the body by the lungs (ie, decreased ventilation or hypoventilation), which can be caused by:

- Decreased the respiratory rate and/or tidal volume of breathing

- Examples: shallow breathing, airway obstruction, iatrogenic (due to mechanical ventilation settings), and central hypopnea

- Increased concentration of CO_2 in inhaled gas

- ▶ Increased production of CO_2 that is not accompanied by an appropriately sized increase in the rate of CO_2 removal from the body by the lungs. This can occur with low, normal, or increased ventilation, if the increase is insufficient to remove the elevated amounts of CO_2

- ▶ Examples: hyperthyroidism, malignant hyperthermia, and sepsis

- ▶ Compensatory changes include increased renal clearance of H^+ and resorption of HCO_3^-

- ▶ Absorption of CO_2 from other sources (eg, during laparoscopic surgery, owing to intraperitoneal insufflation with CO_2)

• Respiratory alkalosis⁸

- Alveolar minute ventilation is increased relative to CO_2 production (resulting in hypocarbia). This can occur through:

- ▶ Increasing the respiratory rate and/or increasing the tidal volume of breathing, (with resulting hypocarbia)

- ▶ Examples: hyperventilation during pain/anxiety or iatrogenic hyperventilation during mechanical ventilation

- ▶ Compensatory metabolic changes include decreased renal resorption of HCO_3^-

- ▶ It is important to remember that *one cause of hyperventilation is hypoxia*³

• Metabolic acidosis³⁸

- Can occur through:



- ▶ Elevation of acidic substance levels in the bloodstream other than CO_2 , due to:
 - ▶ Increased production of acid
 - ▶ Decreased clearance of acid
 - ▶ Ingestion of acidic toxins or drugs (eg, aspirin, paraldehyde, methanol, ethylene glycol)
 - ▶ Loss of HCO_3^- (eg, diarrhea)

• For metabolic acidosis with elevated anion gap and no obvious cause (eg, DKA, lactic acidosis), consider toxic ingestion, such as ethylene glycol, propylene glycol, methanol, aspirin, or oxoproline (associated with acetaminophen poisoning)

• One mnemonic for various causes of an anion gap acidosis is GOLDMARK, as follows:³

- ▶ Glycols: ethylene glycol, propylene glycol
- ▶ Oxoproline (pyroglutamic acid)
- ▶ Lactic acidosis
- ▶ D-lactate/DKA
- ▶ Methanol
- ▶ Aspirin
- ▶ Renal failure
- ▶ Ketoacidosis

• Metabolic alkalosis³⁸

• The primary changes responsible for alkalemia are usually (1) loss of H^+ ions via the gastrointestinal tract or the kidneys or (2) increased resorption of HCO_3^-

• Causes include but are not limited to:

- ▶ Hypovolemia with chloride ion depletion
- ▶ GI causes: vomiting, gastric suction, villous adenoma, diarrhea with loss of chloride-rich fluid
- ▶ Renal causes: loop and thiazide diuretics
- ▶ Lag after correction of hypercapnia (eg, after institution of mechanical ventilation), when compensatory HCO_3^- elevation has not had time to correct
- § ▶ Hypervolemic states that cause bicarbonate resorption (eg, edematous states)
- ▶ Other stimuli for bicarbonate resorption
- ▶ Hyperaldosteronism
- ▶ Hypercortisolism
- ▶ Excess ACTH
- ▶ Exogenous steroid administration

- ▶ Hyperreninemia
- ▶ Severe hypokalemia

• Exogenous HCO_3^- administration

- Mixed acid-base disorders

▶ Because compensatory mechanisms for both metabolic and respiratory acid-base disorders begin rapidly after onset, acid-base disorders are seldom "pure" but are usually "mixed," involving a primary disorder plus a compensatory response (eg, respiratory alkalosis with compensatory metabolic acidosis)

▶ However, in some cases, both a primary metabolic and respiratory disorder may occur at the same time (eg, metabolic acidosis due to DKA combined with a preexisting primary hypoventilatory disorder due to lung disease)

▶ In addition, 2 different primary metabolic disorders can also exist at the same time (eg, an anion gap acidosis with a non-anion gap acidosis)

▶ Mixed primary respiratory acidosis/alkalosis cannot exist, because they are based on mutually exclusive primary changes in PaCO_2 (ie, hypercarbia and hypocarbia)

Possible Sources of Error

• Various aspects of blood sample handling can result in erroneous ABG values, as explained in the following sections¹⁰

Time From Gas Sampling to Gas Analysis

• Once an arterial blood sample is collected, prompt laboratory analysis should occur: the longer the delay, the more measured values can potentially differ from true values

• Errors in handling can lead to both false increases and false decreases in PaO_2

▶ Two mechanisms can lower the PaO_2 value in the arterial sample after collection:

- Diffusion of gas through the plastic syringe²⁵



- However, if the sample is refrigerated, the decrease in oxygen tension in blood samples is not significantly different between glass and plastic syringes at 30 minutes⁵⁷

- ▶ PaO₂ can also be falsely elevated if blood gas analysis is delayed^{59,60}

- In one study, PaO₂ in blood samples rose over 180 minutes, although the magnitude of rise was deemed clinically insignificant⁶⁰

- pH can decrease and PaCO₂ can be increased significantly by delayed analysis

- ▶ The same study cited above found a significant decline in pH at 30 minutes, and rise in PaCO₂ at 60 minutes, with exaggerated changes in pH seen in patients with chronic pulmonary disease versus healthy controls⁶⁰

- ▶ The decrease in pH is delayed if the specimen is placed on ice⁵⁹

Temperature

- Decreases in pH, PaO₂, and PaCO₂ were greater and occurred more quickly in arterial blood samples that were kept at room temperature (24-26 °C) versus kept on ice (0-4 °C)⁵⁹

Air Bubbles in the Syringe

- Such bubbles can falsely increase PaO₂ and decrease PaCO₂ values. The volume of air need only be about 1% of the sample volume.⁶¹ This can occur by accidentally drawing air into the syringe, or agitating the syringe after drawing the sample

- ▶ After collection, the sample should be gently tapped, and visible air expelled

Heparin in the Syringe

- Heparin can increase the acidity of the sample (decrease pH) if the syringe is not completely emptied of heparin before sampling. PaCO₂ may also decrease in the presence of heparin

References: Millers anaesthesia



Dysrhythmia Recognition

Moderator: **Dr Yoganarasimha N**
Professor, Department of Anaesthesiology,
BGS GIMS , Bengaluru

1. What is Dysrhythmia?

It's due to the abnormality in the cardiac conduction system:

because of Impulse generation, Impulse conduction, both which may lead to life threatening complications due to reduction in cardiac output and reduction in myocardial blood flow.

LOOK FOR

- Rate (bradycardia or tachycardia)
- Rhythm (regular or irregular)
- Origin of impulse (supraventricular or ventricular)
- Impulse conduction (atrioventricular, ventriculoarterial or block)
- Ventricular rate
- Special phenomenon (like preexcitation)

2. Tachydysrhythmia and its classification A cardiac rhythm with greater than 100bpm is considered as Tachydysrhythmia

3. CLASSIFICATION:

a) Based on anatomical location of source involved in generation

- Above the level of Bundle of His -- Supraventricular
- Below the level of Bundle of His -- Ventricular

b) Based on QRS complex

- Narrow complex -- Usually occurs when originates at or above AV node
- Broad complex -- Usually occurs when originates below AV node

4. Tachydysrhythmia seen in narrow/broad QRS complex Narrow QRS complex includes:

- Sinus tachycardia
- Atrial flutter
- Atrial fibrillation

- Junctional tachycardia
- Paroxysmal atrial tachycardia
- Accessory pathway mediated re-entrant tachycardia

5. Tachydysrhythmia with Broad QRS complex includes: -

- Ventricular tachycardia
- SVT with Bundle branch block
- SVT with metabolic or electrolyte disorder
- SVT with intraventricular conduction defect
- SVT with preexcitation through accessory pathway

6. Ectopy & types - extra beats or contractions referred as extra systoles which occur when there is electrical discharge from somewhere in the heart other than sinoatrial node. It may occur in atrium or ventricle referred as

- a) Premature atrial contractions (PAC)
- b) Premature ventricular contractions (PVC)

7. Premature atrial contractions (PAC) may occur in all age groups either with or without heart disease:

CHARACTERISTICS IN ECG –

- P wave – vary
 - PR interval – Variable
 - RR interval - irregular
 - QRS complex –
- 1) Narrow if conduction pathways are normal
 - 2) Broad in presence of aberrant pathway



PRECIPITATING FACTORS: -

Sympathetic stimulation (Alcohol / Excessive caffeine/nicotine/recreational drugs)

- Hyperthyroidism
- Chronic lung disease
- Ischemic heart disease
- Digitalis toxicity

COMPLICATIONS: -

Usually hemodynamically insignificant May predispose to tachydysrhythmia

TREATMENT: -

- Avoid excessive sympathetic stimulation
- Treatment of underlying predisposing conditions
- Beta blockers
- Calcium channel blockers
- Treatment of secondary dysrhythmias with drugs/manoeuvres to correct rate/rhythm



8. Premature Ventricular contractions (PVC) Origin – either unifocal (single) or multifocal (multiple) foci located below the AV node

CHARACTERISTICS IN ECG: -

- No preceding P wave
- QRS complex – wide
- ST and T wave – opposite to QRS deflection

Types: -

- 1) Unifocal – constant interval between PVC and sinus rhythm
- 2) Multifocal – Varied interval between PVC and sinus rhythm
- 3) Ventricular bigeminy – Two/Three PVC separated by one regular sinus beat
- 4) Ventricular trigeminy -- Two/Three PVC separated by two regular sinus beats
- 5) Ventricular tachycardia – more than three consecutive PVC

PREDISPOSING FACTORS: -

- Electrolyte imbalances (hypokalaemia, hypomagnesemia)
 - Acidosis
 - Myocardial ischemia/infarction
 - Digitalis toxicity
 - Alcohol/caffeine/cocaine
 - Mechanical irritation (central venous / pulmonary artery catheter/cardiac surgery)
 - Arterial hypoxemia

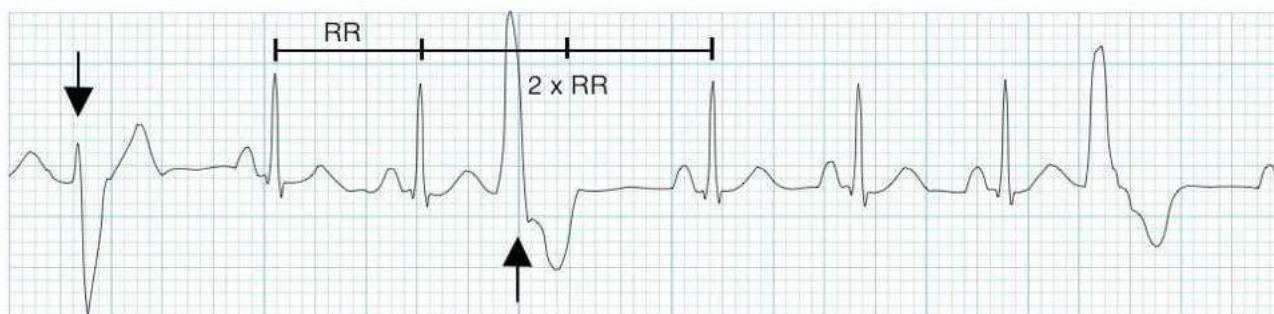
COMPLICATIONS: -

- PVC can be self-limiting/ progressive
- R ON T PHENOMENON – PVC in the middle third of T wave which may predispose to VT/VF
- Presyncope / syncope (lack of atrial contribution to ventricular filling)
- > 6 PVC PER MINUTE / REPETITIVE / MULTIFOCAL PVC may predispose to life threatening dysrhythmias



TREATMENT: -

- Treat the underlying cause Pharmacological treatment – Amiodarone, Lignocaine, Procainamide, Beta blockers only if it progresses to VT or causes hemodynamically instability
- Availability of defibrillator is a priority in presence of PVC



9. Atrial fibrillation – is a type of supraventricular tachycardia associated with dys-coordinated atrial contractions and irregular ventricular rate.

- Incidence increases with age
- Most common tachydysrhythmia post operatively occurring in the early post-operative ward (2-4 days) after cardiothoracic surgery predominantly in elderly patients

CHARACTERISITICS IN ECG:

- absent P waves
- RR interval – irregularly irregular
- QRS complex – Narrow
- Rate – Normal or Rapid depending on conduction system (60 – 170/bpm of ventricular)

Causes –

a) Comorbidities associated with AF

- Systemic hypertension
- Ischemic heart disease / heart failure
- Hyperthyroidism
- Chronic kidney disease
- Chronic obstructive lung disease
- Rheumatic heart disease (common in involving mitral valve)

b) Structural /electrophysiological abnormalities

- associated with AF
- Atrial septal defect
- Pericarditis / pulmonary embolus
- Obesity / sleep apnoea / recreational drug usage

10. Complications of AF

- AF causes loss of atrial kick which reduces ventricular filling
- Loss of coordinated atrial contraction promotes stasis of blood within left atrium leading to formation of atrial thrombi. The major complications of AF are
- Thromboembolism (cerebral vascular accidents - embolic stroke (most common)
- hemodynamically compromise (myocardial infarction, cardiogenic shock)
- Dysrhythmogenesis (predispose to fatal dysrhythmias)
- Acute Pulmonary oedema

11. Management of atrial fibrillation

New onset AF: -

- Usually, spontaneous conversion occurs to sinus rhythm within 24 – 48 hours
- After conversion to sinus rhythm beta blockers can be used to prevent recurrent AF

12. AF in intraoperative period: -

- Hemodynamically stable – Pharmacological therapy for rate control (beta blockers, calcium channel blockers, Amiodarone, Digoxin)
- Hemodynamically unstable – Synchronised cardioversion (100 – 200 J)

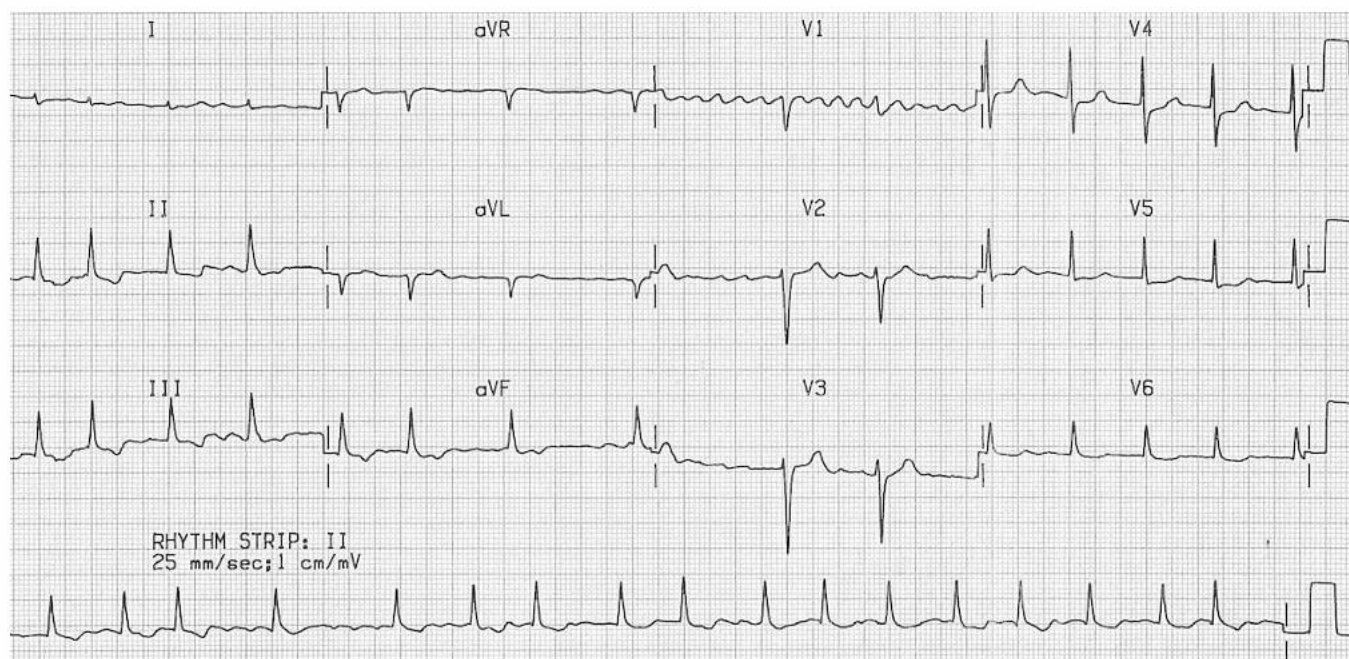


1. Chronic AF: -

- Rate control therapy
- Long term anti-coagulant needed to avoid embolic stroke
- Warfarin or newer anticoagulants (Clopidogrel, Dabigatran, Apixaban, Rivaroxaban) are used.
- IV standard Heparin or LMWH used in perioperative prophylaxis and bridging therapy.

Others: -

- a) Catheter ablation for patient's intolerant to drug therapy
- b) Surgical maze procedure and left atrial appendage exclusion



14. Ventricular tachycardia and its predisposing factors

- Three or more consecutive ventricular premature beats occurring at a heart rate > 120bpm is referred as Ventricular tachycardia
- Presents as monomorphic or polymorphic

- Myocardial ischemia / infarction
- Acute pulmonary oedema
- Sudden cardiac arrest
- Tachycardia induced cardiomyopathy (in persistent VT).

OCCURENCE: - More common in age group > 60 years

PRE-DISPOSING FACTORS: -

- Electrolyte abnormalities (hypokalaemia, hypomagnesemia) Acute myocardial infarction
- Myocarditis
- Cardiomyopathy
- Mechanical stimulation (perioperative-catheter insertion, interventional procedures)
- Digitalis toxicity

15. COMPLICATIONS: -

- Hemodynamically instability (cardiogenic shock)
- Predisposition to ventricular fibrillation

16. Management of ventricular tachycardia: - Timely termination of VT is must even if it is well tolerated to avoid complications

PULSATILE VT: -

- Treatment of underlying cause (correction of electrolyte abnormalities and reversible factors)
- Pharmacological therapy Procainamide, Amiodarone, Lignocaine)

SYMPTOMATIC / UNSTABLE VT: -

- Immediate electrical cardioversion
- Initiated with 100J (monophasic) and increased in increments of 50 – 100 J as necessary

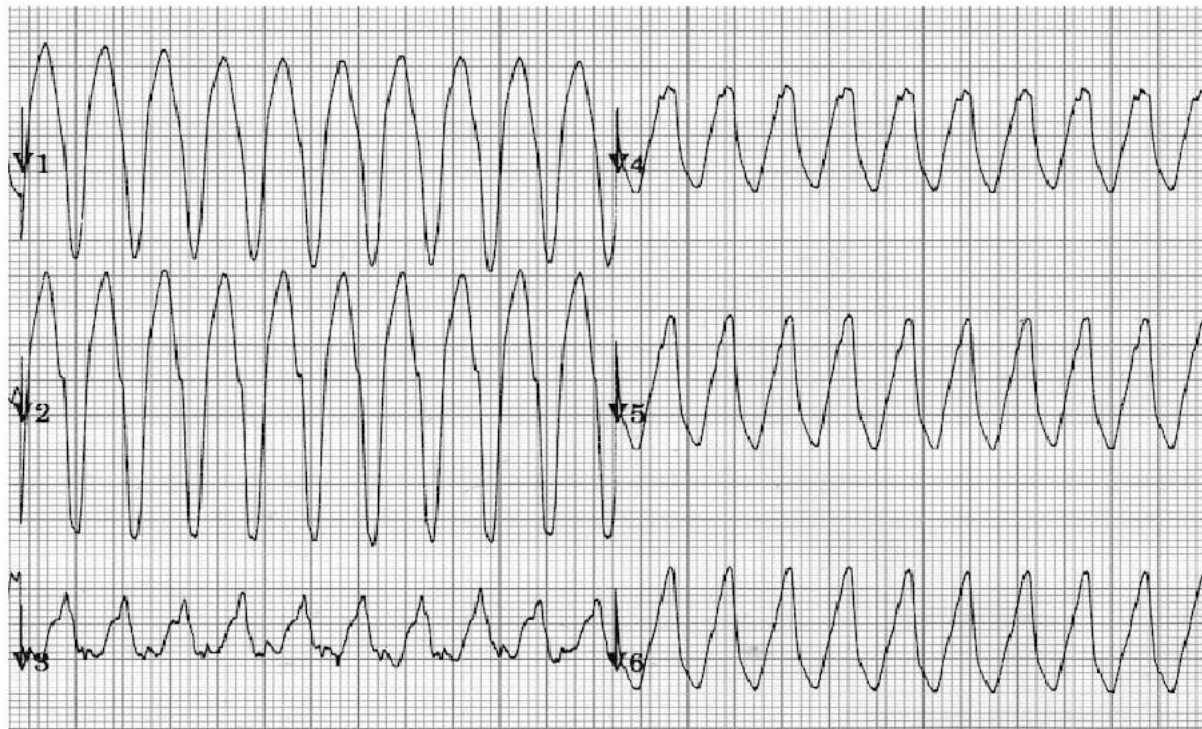


PULSELESS VT: -

- Immediate initiation of cardiopulmonary resuscitation (CPR) and defibrillation with 120 – 200J (Biphasic) or 360 J (Monophasic)

DRUG REFRACTORY VT: -

- Catheter ablation of causative ectopic pathway
- Implantable cardioverter defibrillator therapy



17. Ventricular fibrillation and its predisposing factors

VF is a rapid, grossly irregular ventricular rhythm due to total disorganization of electrical activity in the heart.

CHARACTERISTICS IN ECG: -

- Rapid rate and irregular rhythm
- QRS – marked variability in cycle length morphology and amplitude

18. Predisposing factors of ventricular fibrillation

- Myocardial infarction
- Cardiomyopathy
- Electrolyte abnormalities (hypokalaemia, hypomagnesaemia)
- Electrocutation • Digitalis toxicity
- Mechanical stimulation of the heart.

19. Complications and management of ventricular fibrillation

COMPLICATIONS: -

- VF is associated with a NO STROKE VOLUME STATE
- Most common cause of Sudden Cardiac Death

MANAGEMENT:

- Immediate initiation of cardiopulmonary resuscitation (CPR) and electrical defibrillation according to ACLS guidelines.
- Defibrillation within 3 – 5 minutes of cardiac arrest is associated with better survival
- Contributing factors should be found and treated (Drug or environmental toxins, 5H and 5T).
- VF refractory to electrical treatment – IV administration of INJ. Adrenaline 1mg or Amiodarone 150 – 300 mg may improve the response to electrical defibrillation.



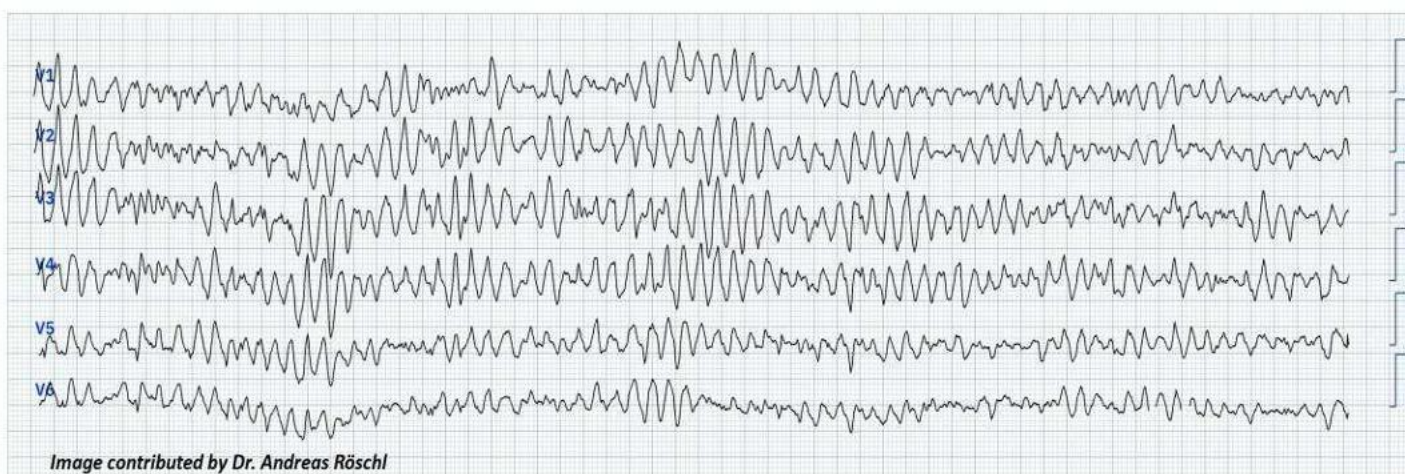
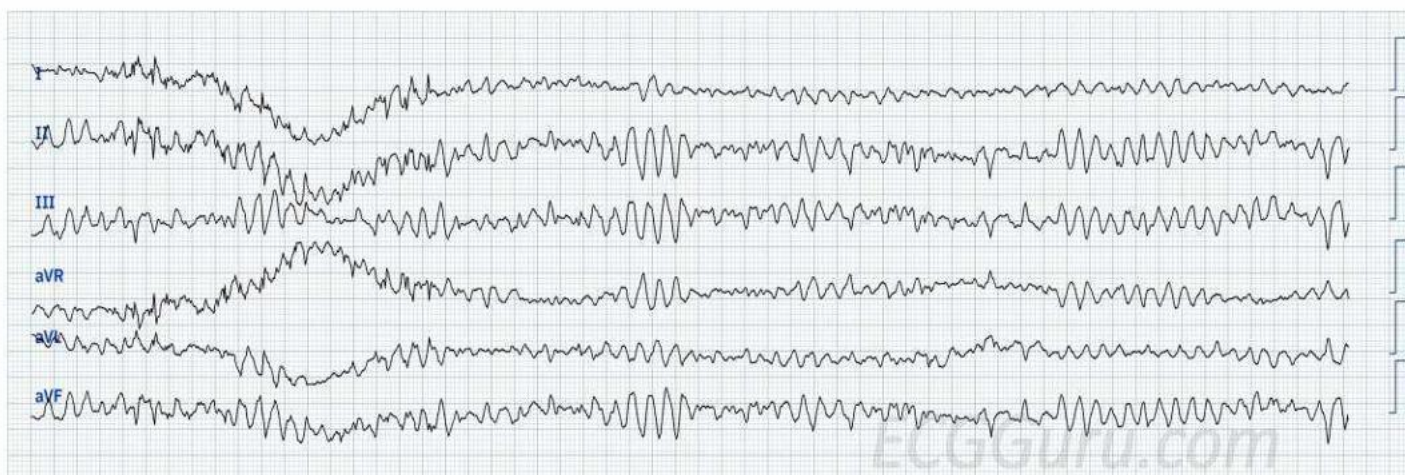
20. Electrical cardioversion and defibrillation

ELECTRICAL OR SYNCHRONIZED CARDIOVERSION

- It is delivery of electrical charge synchronized to the R WAVE of ECG
- It's a single dominant burst of electricity to re coordinate the electrical pathways of the heart
- Stimulus co-ordinated with R wave to avoid delivery on relative refractory period (T wave) which may cause R on T phenomenon
- Used to treat acute unstable dysrhythmias –
 1. Supra ventricular Tachycardia
 2. Atrial flutter
 3. Atrial fibrillation
 4. Symptomatic Pulsatile VT

• CONTRAINDICATIONS: -

1. Digitalis toxicity and associated tachycardia – may predispose to VT / VF
2. Stable AF > 48 HRS without anticoagulation or TOE exclusion of atrial thrombus due to risk of thromboembolism and stroke.



1. ANAESTHESIA FOR ELECTIVE CARDIOVERSION: -

Cardioversion is an intensely painful and stimulating process. Preferably done under deep sedation in conscious patients

- 1) Pre anaesthesia check-up for significant cardiovascular disorders/respiratory disorders/obesity/OSA/other illness
- 2) Informed consent
- 3) Investigations (Complete blood count, Serum electrolytes, ECG, ECHO, Coagulation profile)
- 4) Patient must be anticoagulated for a minimum of 3 weeks prior to cardioversion and at least 4 weeks thereafter
- 5) Alternative strategy to exclude presence of atrial thrombus with TOE.
- 6) All emergency drugs and airway cart to be kept ready before induction
- 7) Patient must be preoxygenated at least 3 minutes prior to cardioversion
- 8) Cardioversion mostly done under deep sedation by administration of slow bolus dose of INJ.Propofol (0.5 – 1mg/kg) with further doses of 0.25 mg /kg
9. Other sedative agents like Etomidate, Benzodiazepines, opioids and inhalational agents can also be used according to hemodynamic stability
10. For emergency cardioversion anaesthesia technique should be tailored individually based on hemodynamically stability and recent ingestion status to avoid complications.

DEFIBRILLATION: -

- Correct dysrhythmias when it is not possible to synchronize to the ECG because of absence of R wave
- Monophasic (360J) or Biphasic (120-200J)
- Used to revert Ventricular fibrillation.
- Patients undergoing defibrillation within initial 3 minutes of VF has a better survival rate



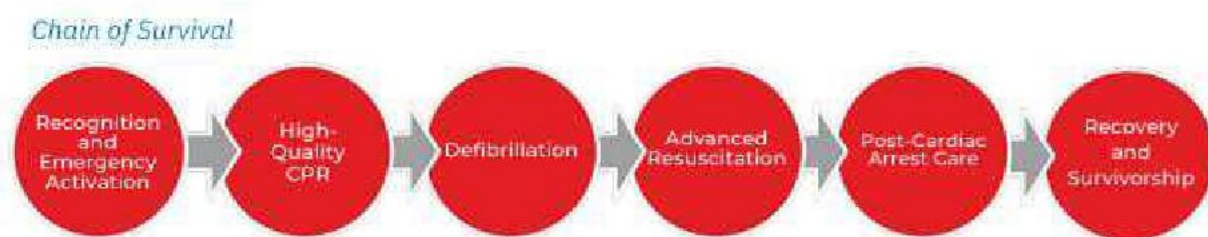
OSCE – Cardio Pulmonary Resuscitation (CPR)

1. Define BLS

Basic life support(BLS) is the component of the immediate care provided for the victims of life-threatening conditions, leading to cardiac arrest, and injuries till the patient can be shifted to a hospital. It can be given by doctors, nurses, paramedics, or even by a trained bystander.

2. What is Chain of Survival?

To increase the odds of surviving a cardiac event, the rescuer should follow the steps in the Chain of Survival . As of **2025 guidelines** , the **Pediatric and Adult Chains of Survival have been unified into a single chain, for a total of 6 steps.**



3. What are the components of High Quality CPR?

- **Compress Hard & Fast:** Push at a rate of 100 to 120 compressions per minute.
- **Adequate Depth:** Compress the chest at least 2 inches (5 cm)
- **Full Chest Recoil:** Allow the chest to return to its normal position between compressions; do not lean on the victim.
- **Minimize Interruptions:** Keep breaks in chest compressions to less than 10 seconds
- **Avoid Excessive Ventilation:** Give breaths that only produce visible chest rise and do not hyperventilate, use pocket mask if available



4. What to do when only one rescuer is available?

Verify scene safety, assess the person, call for help and start CPR.
Use AED when available.

5. BLS when 2 rescuers are available?

1st rescuer – assess the patient and starts CPR

2nd rescuer – calls for EMS , prepares AED for use, applies AED pads , opens airway and gives breaths.

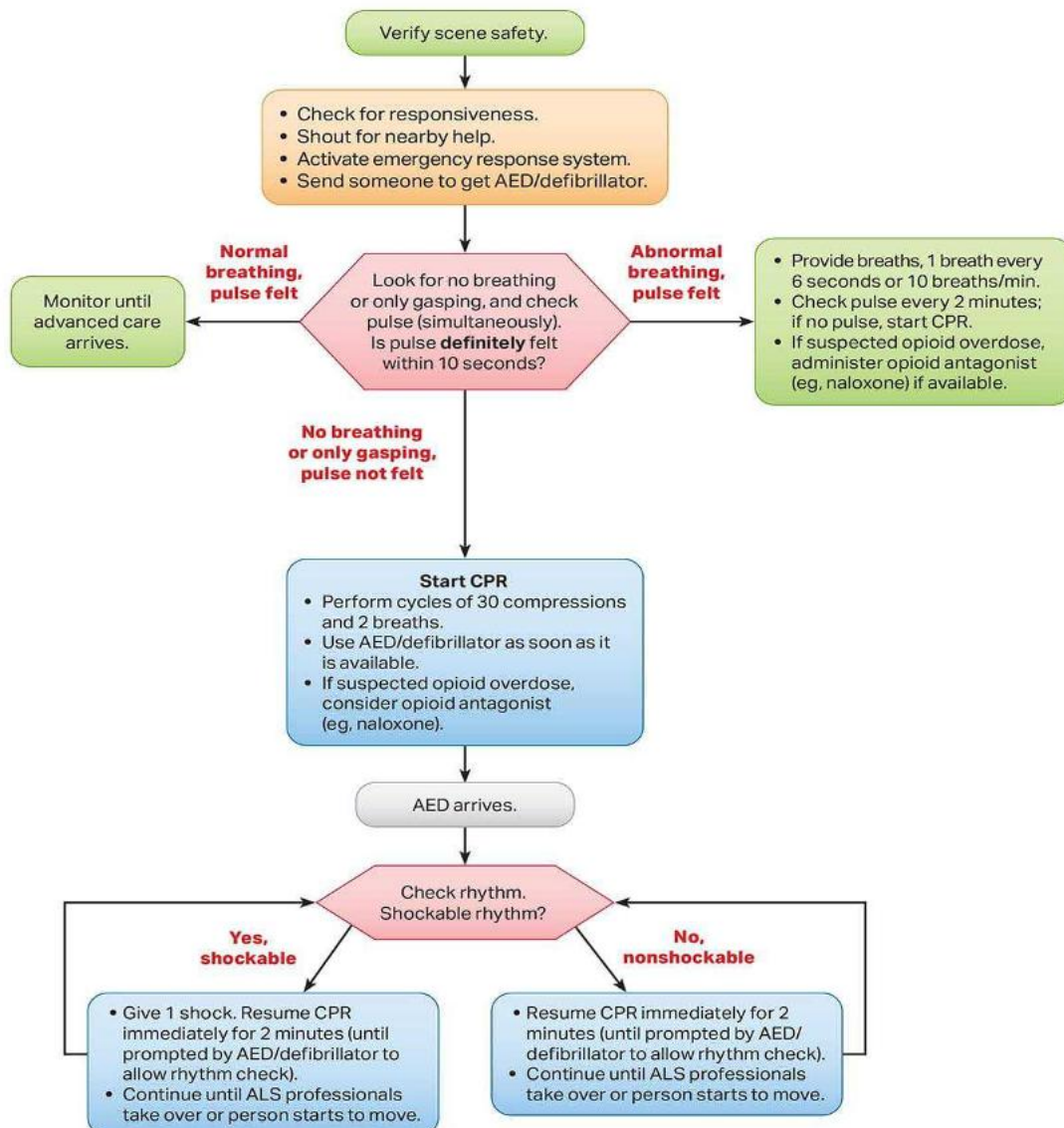
6. What is one cycle of CPR and when to switch roles?

1 cycle of CPR consists of 30 compressions followed by 2 breaths. Switch roles after 5 cycles or early if tired with minimum interruptions <10 secs in chest compressions.



7. BLS Algorithm

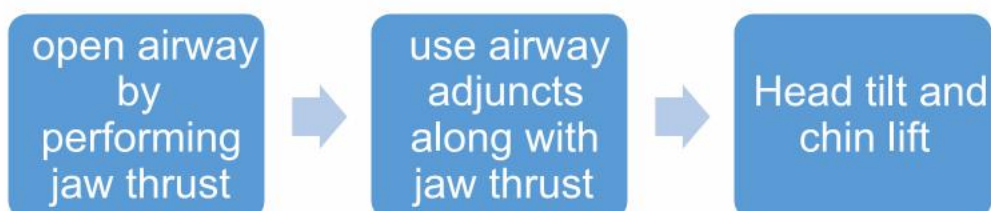
Adult* Basic Life Support Algorithm for Health Care Professionals



*If signs of puberty, treat as adult.

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8. How to open airway if head and neck trauma is suspected?



In trauma patients, establishing and maintaining a patent airway is the highest priority. A jaw-thrust maneuver should be attempted first when head or neck injury is suspected, as it minimizes cervical spine movement. However, **if the airway cannot be adequately opened with a jaw thrust and airway adjuncts, a head-tilt–chin-lift maneuver should be performed, in accordance with 2025 guidelines, to ensure effective oxygenation and ventilation.**

Although jaw thrust is preferred in the setting of suspected cervical spine injury, the primary emphasis in trauma care remains on securing a patent airway, as adequate oxygenation and ventilation takes priority.

9. What is Philadelphia Collar?

A Philadelphia collar is a rigid cervical collar used to immobilize and support the neck (cervical spine) after injury, surgery, or in suspected cervical spine fractures.

Key Features:

- Made of semi-rigid plastic with foam padding
- Has anterior and posterior parts joined by Velcro straps
- Includes a chin support and an opening in the front for:
 - tracheostomy access
 - checking the carotid pulse
 - ventilation if needed

Uses:

- Cervical spine trauma
- Neck fractures or instability
- Postoperative cervical spine support
- Emergency transport of trauma patients

What is log roll?

A log roll is a technique used to turn a patient while keeping the head, neck, and spine aligned to prevent spinal movement in suspected spinal injury.

Uses:

- Shifting trauma patients
- Examination of the back
- Placing/removing a spine board
- Preventing further spinal cord injury

10. What is a spine board?

A spine board (hard board) is a rigid board used to immobilize and transport patients with suspected spinal injuries.

Features:

- Made of rigid plastic or wood
- Used with cervical collar and straps
- Maintains spinal alignment during transport

Uses:

- Trauma and accident victims
- Suspected cervical or spinal fractures
- Emergency patient transport



11. What is COLS?

COLS stands for Compression-Only Life Support in which continuous chest compressions are given without rescue breaths by untrained bystanders. In India, COLS has been promoted by the Indian Resuscitation Council as a standardized, practical technique for laypersons mainly on focus to circulation.

12. Is there any changes in CPR for obese individuals?

No

13. What is AED?

AED stands for Automated External Defibrillator which analyses the heart rhythm and delivers a controlled, precise shock to the heart.

An AED may be located in campus or close by at a community facility, all airports, shopping malls and other public places.



14. How to use an AED in these situations?

- If the person is wet- quickly wipe dry before applying pads
- If the person has pacemaker -Place the defibrillator pads as close to the correct position as possible without being directly over the device
- If the person is in snow- It is safe to use an AED if the person is lying in snow
- If the person has medication patches-remove the patch, wipe the skin dry, and apply the AED pad.

15. When to do pulse check after delivering a shock?

After delivery of a shock, two minutes of high-quality CPR is performed. Do not perform a rhythm or pulse check at this point as even if the shock is successful, heart may not generate effective circulation immediately.

16. What is No-No-Go framework ?

It is to quickly recognise cardiac arrest and guide bystander CPR

No response, No Breathing, Go call Emergency services and start CPR.

17. What are the differences in BLS from 1 yr till puberty?

- The compression to breaths ratio is 30:2 for one rescuer and 15:2 for two rescuers.
- compress the chest at least one-third the depth of the chest. This may be less than two inches for small children (4 -5 cm)
- If only rescuer at the scene and find an unresponsive child, perform CPR for two minutes before calling EMS or before looking for an AED.
- In children, primary cardiac events are not common. Cardiac arrest is most commonly preceded by respiratory problems. Survival rates improve with early intervention for respiratory problems.
- If you witness a cardiac arrest in a child, call EMS and get an AED as you would in the Adult BLS sequence.
- Use pediatric pads if <8yrs or <25kgs. If not available, use adult pads ensuring they donot touch each other.



- Anterior–posterior AED pads one pad is placed on the front of the chest and the other on the back. It is used when:
 - o The patient is a small child/infant and pads may touch each other on the chest
 - o There is limited chest space
- Pad Position:
 - o Anterior pad: left side of chest over the precordium
 - o Posterior pad: on the back between the scapulae

18. How does BLS in infants (0-12 months) differ?

BLS for both children and infants is almost identical. Following are the main differences :

- Check the pulse in the infant using the brachial artery
- During CPR, perform compressions on an infant using the two thumb-encircling hands technique. The 2 finger technique is no longer recommended according to 2025 guidelines.
- Compression depth should be one-third of the chest depth; for most infants, this is about 1.5 inches (4 cm) at the center of chest, not on the end of sternum.
- If only rescuer at the scene and find an unresponsive infant, perform CPR for two minutes before calling EMS or using an AED.
- Begin CPR if no pulse or PR < 60bpm.

19. What is the compression to breath ratio in different age groups?

COMPRESSION TO BREATH RATIO	NO ADVANCED AIRWAY	ADVANCED AIRWAY
Adult	30 compressions followed by two breaths	One breath every 6 seconds without pauses in compressions
Child/Infant	30 compressions followed by two breaths for one rescuer 15 compressions followed by two breaths for two rescuers	One breath every 6 seconds without pauses in compressions One breath every 2-3 seconds (20-30 breaths per minute)

20. When to give rescue breaths?

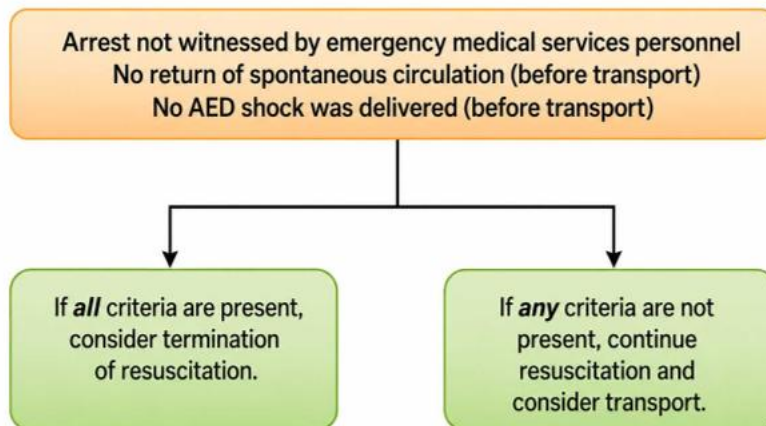
When a person of any age has a pulse but is not breathing (or is not breathing well), immediately open the airway using the head-tilt/chin-lift maneuver or the triple maneuver that includes head tilt, chin lift and jaw thrust, and begin rescue breathing.

AGE GROUP	HOW OFTEN	BREATHS PER MINUTE	DURATION	EVALUATION
Adult	Every 6 seconds	10 to 12 breaths per minute	Each breath should last one second	Check for chest rise and breathing; Check pulse and begin CPR if necessary
Child/Infant	One rescuer: Every 6 seconds Two rescuers: Every 2 to 3 seconds	One rescuer: 10 to 12 breaths per minute Two rescuers: 20 to 30 breaths per minute		



21. When to Terminate Resuscitation?

BLS/Universal Termination of Resuscitation Rules



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22. How to manage a person with choking?

Choking In Adults And Children

DEGREE OF OBSTRUCTION	PERSON'S RESPONSE	RESCUER'S ACTION
<i>Mild Obstruction</i>	- Breathing but may also be wheezing - Coughing and making noise	- Stay with the person and try to keep them calm - Encourage them to cough - Call 911/EMS if the person seems to be getting worse
<i>Severe Obstruction</i>	- Clutching the neck (universal sign of choking; figure 15) - Weak or no cough - Unable to make noise or talk; may make high-pitched noise - Little or no breathing - May be cyanotic (blue around lips and fingertips)	- Deliver 5 back blows between the shoulder blades, then 5 abdominal thrusts. Alternate until obstruction is relieved or person becomes unresponsive. - Call 911/EMS - Begin BLS if the person becomes unresponsive

Heimlich Maneuver in adults:

The Heimlich maneuver is a first-aid technique used to remove a foreign body airway obstruction (choking) by giving abdominal thrusts.

Procedure

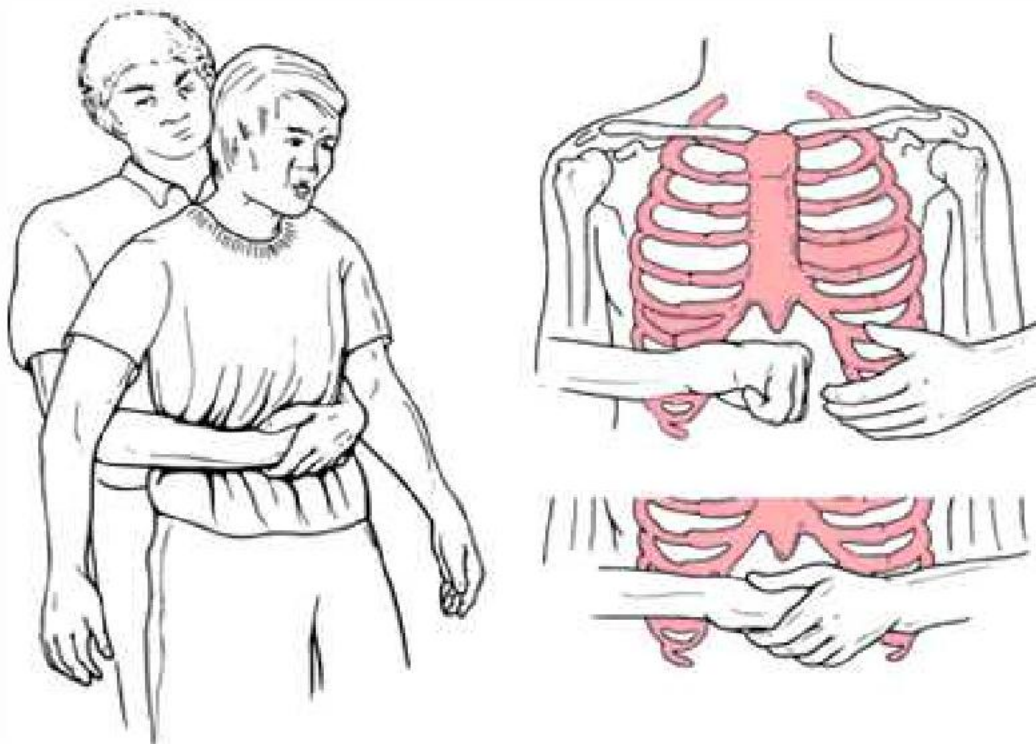
Stand behind the person

Place a fist above the umbilicus and below the sternum

Grasp the fist with the other hand

Give quick upward and inward thrusts until the object is expelled





Choking In Infants

DEGREE OF OBSTRUCTION	INFANT'S RESPONSE	RESCUER'S ACTION
Mild Obstruction	- Breathing but may also be wheezing - May be coughing and making noise	- Stay with the infant and try to keep them calm - Do not do a blind finger sweep - Call 911/EMS if infant does not quickly clear the obstruction
Severe Obstruction	- Weak or no cough - Unable to make noise; may make high-pitched noise - Little or no breathing - May be cyanotic (blue around lips and fingertips)	- Use back blows/chest thrusts to attempt to remove obstruction - Call 911/EMS - Begin BLS if infant becomes unresponsive - Assess if obstruction is visible; if so, remove it



```

graph TD
    1[1 Start CPR  
• Begin bag-mask ventilation and give oxygen  
• Attach monitor/defibrillator] --> 2{Rhythm shockable?}
    2 -- Yes --> 3[2 VF/pVT]
    3 --> 4[3 Shock]
    4 --> 5[4 CPR 2 min  
• IV/IO access]
    5 --> 6{Rhythm shockable?}
    6 -- Yes --> 7[5 Shock]
    7 --> 8[6 CPR 2 min  
• Epinephrine every 3-5 min  
• Consider advanced airway, capnography]
    8 --> 9{Rhythm shockable?}
    9 -- Yes --> 10[7 Shock]
    10 --> 11[8 CPR 2 min  
• Amiodarone or lidocaine  
• Treat reversible causes]
    11 --> 12{Rhythm shockable?}
    12 -- Yes --> 13[Go to 5]
    12 -- No --> 14[12 If no signs of ROSC, go to 10  
• If ROSC, go to post-cardiac arrest care  
• Consider appropriateness of continued resuscitation]
    2 -- No --> 9[9 Asystole/PEA]
    9 --> 10[10 Epinephrine ASAP]
    10 --> 11[10 CPR 2 min  
• IV/IO access  
• Epinephrine every 3-5 min  
• Consider advanced airway, capnography]
    11 --> 12{Rhythm shockable?}
    12 -- Yes --> 13
    12 -- No --> 14
  
```

Flowchart for the management of cardiac arrest:

- Start CPR**
 - Begin bag-mask ventilation and give oxygen
 - Attach monitor/defibrillator
- Rhythm shockable?**
 - Yes:**
 - VF/pVT**
 - Shock**
 - CPR 2 min**
 - IV/IO access
 - Rhythm shockable?**
 - Yes:** Shock → CPR 2 min (Epinephrine every 3-5 min, Consider advanced airway, capnography) → Rhythm shockable?
 - Yes:** Shock → CPR 2 min (Amiodarone or lidocaine, Treat reversible causes) → Rhythm shockable?
 - Yes:** Go to 5
 - No:** 12
 - No:** 12
 - No:** 12
 - No:**
 - Asystole/PEA**
 - Epinephrine ASAP**
 - CPR 2 min**
 - IV/IO access
 - Epinephrine every 3-5 min
 - Consider advanced airway, capnography
 - Rhythm shockable?**
 - Yes:** Go to 5
 - No:** CPR 2 min (Treat reversible causes) → Rhythm shockable?
 - Yes:** Go to 5
 - No:** 12
- 12**
 - If no signs of ROSC, go to 10
 - If ROSC, go to post-cardiac arrest care
 - Consider appropriateness of continued resuscitation

High-Quality CPR

- Push hard (at least 2 inches [5 cm]).
- Push fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, use 30:2 compression-ventilation ratio.
- If advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions.
- Continuous waveform capnography
 - If ETCO_2 is low or decreasing, reassess CPR quality.

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus
Second dose: 150 mg
or
Lidocaine IV/IO dose: First dose: 1-1.5 mg/kg
Second dose: 0.5-0.75 mg/kg

Advanced Airway

- ET intubation or supraglottic advanced airway
- Continuous waveform capnography or capnometry to confirm and monitor ET tube placement

Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

24. Which are the shockable and non shockable rhythms?

Shockable : Ventricular fibrillation, Pulseless ventricular tachycardia

Non Shockable : Pulseless Electrical Activity, Asystole

25. What are the reversible causes of Cardiac Arrest?

REVERSIBLE CAUSES OF CARDIAC ARREST	
THE H'S	THE T'S
<i>Hypovolemia</i>	<i>Tension pneumothorax</i>
<i>Hypoxia</i>	<i>Tamponade</i>
<i>H+ (acidosis)</i>	<i>Toxins</i>
<i>Hypo/Hyperkalemia</i>	<i>Thrombosis (coronary)</i>
<i>Hypoglycemia</i>	<i>Thrombosis (pulmonary)</i>
<i>Hypothermia</i>	<i>Trauma (unrecognized)</i>

26. How does return of circulation differ from return of spontaneous circulation?

Return of circulation is achieved by mechanical means like ECMO whereas ROSC is due to cardiac function.

27. What is the difference between Defibrillation and Cardioversion?

Defibrillation is the nonsynchronized delivery of a shock that depolarizes the entire cardiac tissue into a refractory period, making it unable to sustain or propagate an aberrant circuit. It is performed during a cardiac arrest in a pulseless patient.

Cardioversion is the delivery of energy that is synchronized to the QRS complex, thus only depolarizing the active circuit causing arrhythmia. It is used to revert arrhythmias in awake patients

Cardioversion	Defibrillation
Elective procedure	Emergency procedure
Victim is awake but frequently sedated	Victim is unconscious
Shock is synchronized with QRS	Shock is not synchronized
Indications - Refractory supraventricular tachycardia - Unstable atrial fibrillation/flutter - Unstable ventricular tachycardia with pulse	Indications - Pulseless ventricular tachycardia - Ventricular fibrillation
50–200 J (biphasic)	200 J (biphasic) 360 J (monophasic)



28. What are monophasic and biphasic defibrillators?

In monophasic defibrillators, current flows in one direction between paddles and doesn't change polarity hence require higher energies.

Biphasic defibrillators deliver a charge in one direction for half of shock and electrically opposite direction for second half hence require lower energies.

29. What are the advantages of biphasic over monophasic defibrillators?

- Lower energy requirements- less myocardial damage, less post shock arrhythmias
- Higher efficacy – fewer shocks to achieve ROSC
- Less skin burns
- Lighter and more compatible
- Universal standard

30. Differentiate between single shock strategy and stacked shocks.

In single shock strategy, CPR is immediately resumed after first shock whereas in stacked shocks, series of shocks are delivered without CPR in between.

ACLS guidelines recommend single shock strategy due to following reasons,

- High success rate of first shock with biphasic defibrillators
- Interruption in CPR is required for series of stacked shocks
- Declining success rate of 2nd, 3rd immediate shock when first shock has failed

31. What are the routes to administer drugs?

Vascular – first preferred is intravenous, intraosseous when iv line couldn't be secured. Central venous access if available

Intracardiac injections- discouraged as it requires specialized skills, potential for morbidity and availability of other access options.

Via ET Tube – discouraged as it results in low blood concentrations and unpredictable pharmacological effects, removed from guidelines.

32. Pharmacotherapy in ACLS

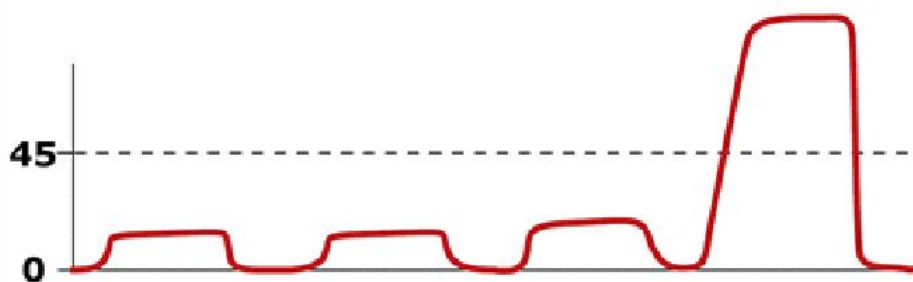
DRUG	MAIN ACLS USE	DOSE/ROUTE	NOTES
Adenosine	• Narrow PSVT/SVT • Wide QRS tachycardia, avoid adenosine in irregular wide QRS	• 6 mg IV bolus, may repeat with 12 mg in 1 to 2 min.	• Rapid IV push close to the hub, followed by a saline bolus • Continuous cardiac monitoring during administration • Causes flushing and chest heaviness
Amiodarone	• VF/pulseless VT • VT with pulse • Tachycardia rate control	• VF/VT: 300 mg dilute in 20 to 30 mL, may repeat 150 mg in 3 to 5 min	• Anticipate hypotension, bradycardia, and gastrointestinal toxicity • Continuous cardiac monitoring • Very long half-life (up to 40 days) • Do not use in 2nd or 3rd-degree heart block • Do not administer via the ET tube route
Atropine	• Symptomatic bradycardia • Specific toxins/overdose (e.g. organophosphates)	• 0.5 mg IV/IO every 3 to 5 minutes • Max dose: 3 mg • 2 to 4 mg IV/IO may be needed	• Cardiac and BP monitoring • Do not use in glaucoma or tachyarrhythmias • Minimum dose 0.5 mg
Dopamine	• Shock/CHF • Symptomatic bradycardia	• 2 to 20 mcg/kg/min • Titrate to desired blood pressure	• Fluid resuscitation first • Cardiac and BP monitoring



Epinephrine	• Cardiac Arrest	- Initial: 1.0 mg (1:10000) IV or 2 to 2.5 mg (1:1000) - Maintain: 0.1 to 0.5 mcg/kg/min Titrate to desire blood pressure	- Continuous cardiac monitoring - Note: Distinguish between 1:1000 and 1:10000 concentrations - Give via central line when possible
	• Anaphylaxis	0.3–0.5 mg IM - Repeat every five minutes as needed	
	• Symptomatic bradycardia/Shock	2 to 10 mcg/min infusion - Titrate to response	
Lidocaine (Lidocaine is recommended when Amiodarone is not available)	• Cardiac Arrest (VF/VT)	- Initial: 1 to 1.5 mg/kg IV loading - Second: Half of first dose in 5 to 10 min - Maintain: 1 to 4 mg/min	- Cardiac and BP monitoring - Rapid bolus can cause hypotension and bradycardia - Use with caution in renal failure
	• Wide complex tachycardia with pulse	- Initial: 0.5 to 1.5 mg/kg IV - Second: Half of first dose in 5 to 10 min - Maintain: 1 to 4 mg/min	
Magnesium Sulfate	• Cardiac arrest/ Pulseless torsades	Cardiac Arrest: 1 to 2 gm diluted in 10 mL D5W IVP	- Cardiac and BP monitoring - Rapid bolus can cause hypotension and bradycardia - Use with caution in renal failure - Calcium chloride can reverse hypermagnesemia
	• Torsades de Pointes with pulse	- If not cardiac arrest: 1 to 2 gm IV over 5 to 60 min - Maintain: 0.5 to 1 gm/hr IV	
Procainamide	- Wide QRS tachycardia - Preferred for VT with pulse (stable)	20 to 50 mg/min IV until rhythm improves, hypotension occurs, QRS widens by 50% or MAX dose is given - MAX dose: 17 mg/kg - Drip: 1 to 2 gm in 250 to 500 mL at 1 to 4 mg/min	- Cardiac and BP monitoring - Caution with acute MI - May reduce dose with renal failure - Do not give with amiodarone - Do not use in prolonged QT or CHF

33. Role of ETCO₂ in Cardiac arrest

- It shows the adequacy of CPR- 10 – 20 mm of Hg shows adequacy i.e, high quality CPR with better chances of ROSC with values more than 20 mm Hg
- Values serve as surrogate to cardiac output
- Sustained value in ETCO₂ >40 mm Hg highly suggests ROSC
- When EtCo₂ is <10mm Hg even after 20 mins of CPR in an intubated individual, to stop resuscitation.



34. Does head up CPR has an advantage?

Head up CPR is assumed to increase cerebral perfusion with goal of improving neurological outcome , but not recommended except for clinical trials.

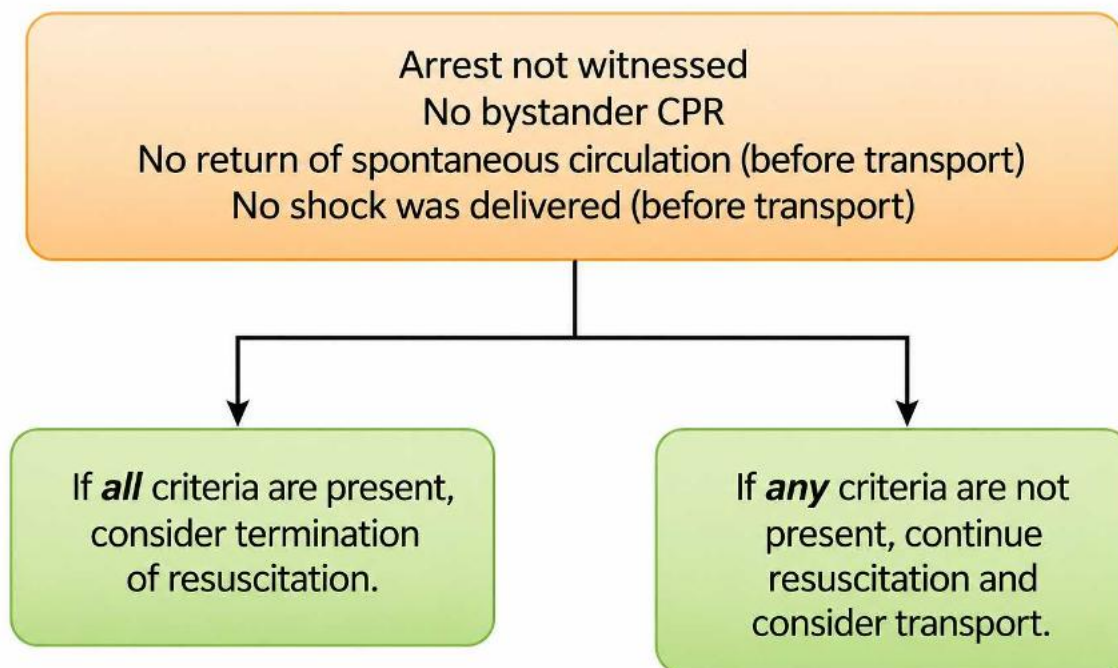
35. Role of POCUS as an adjunct in ACLS

To identify reversible causes like Pulmonary Embolism, Cardiac Tamponade, Effusion, MI, Aortic dissection
Attention to minimize CPR interruptions while performing POCUS

To terminate CPR in absence of cardiac motion



36. Termination of Resuscitative measures in ACLS

ALS Termination of Resuscitation

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37. What are the components of post cardiac arrest care?

THERAPEUTIC HYPOTHERMIA : Recommended for comatose individuals with return of spontaneous circulation after a cardiac arrest event. Individuals should be cooled to 89.6 to 93.2 degrees F (32 to 36 degrees C) for at least 24 hours.

OPTIMIZATION OF HEMODYNAMICS AND VENTILATION

- 100% oxygen is acceptable for early intervention but not for extended periods of time.
- Oxygen should be titrated, so that individual's pulse oximetry is greater than 94% to avoid oxygen toxicity.
- Do not over ventilate to avoid potential adverse hemodynamic effects.
- Ventilation rates of 10 to 12 breaths per minute to achieve ET_{CO2} at 35 to 40 mmHg.
- IV fluids and vasoactive medications should be titrated for hemodynamic stability to maintain a MAP of 65 mm Hg.

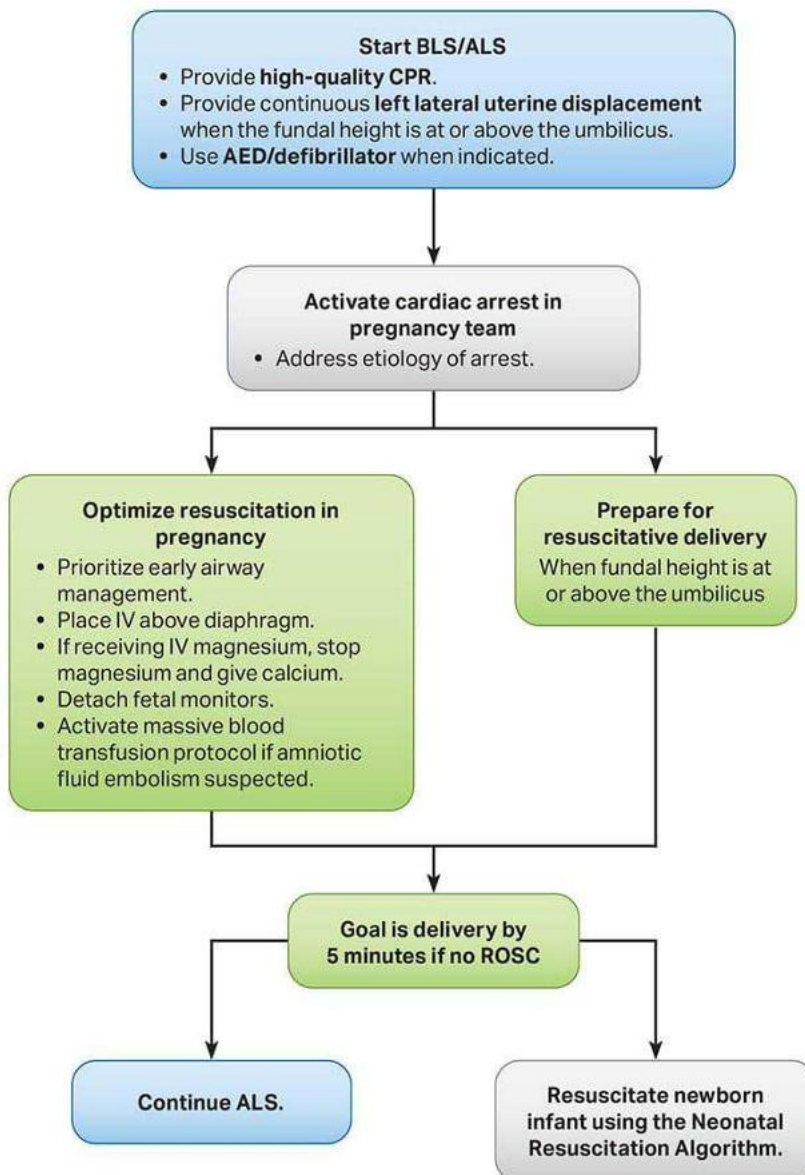
PERCUTANEOUS CORONARY INTERVENTION Percutaneous coronary intervention (PCI) is preferred over thrombolytics.

NEUROLOGICAL CARE Neurologic assessment is key, especially when withdrawing care (i.e., brain death) to decrease false-positive rates. Specialty consultation should be obtained to monitor neurologic signs and symptoms throughout the post-resuscitation period.



38. Explain management of cardiac arrest in pregnancy

Cardiac Arrest in Pregnancy Algorithm



Explanation of Cardiac Arrest Interventions

- Cardiac arrest in pregnancy team will vary according to local resources but may include
 - Team Leader
 - Anesthesiologist
 - Obstetrician
 - Neonatologist
 - Nurses
 - Pharmacists
 - Other professionals
- The goal of left lateral uterine displacement is to relieve aortocaval compression and to facilitate effective chest compressions.
- The goal of resuscitative delivery is to improve the pregnant patient's outcome, and when feasible, the newborn infant's outcome.
- Ideally, perform resuscitative delivery by 5 minutes, depending on local resources.
- In pregnancy, difficult airway is common and is managed (eg, endotracheal intubation or supraglottic airway) by the most experienced professional.

Etiologies of Cardiac Arrest

- A** Anesthetic complications
- B** Bleeding
- C** Cardiovascular
- D** Drugs
- E** Embolic (amniotic fluid or pulmonary embolism)
- F** Fever
- G** General causes (H's and T's)
- H** Hypertension (eg, preeclampsia)

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39. What are the causes of cardiac arrest in pregnancy?

Anesthetic complications (e.g., failed intubation, total spinal, high epidural block, drug toxicity)

Bleeding (e.g., uterine atony, placenta previa, placental abruption, DIC, severe postpartum hemorrhage)

Cardiovascular (e.g., peripartum cardiomyopathy, aortic dissection, myocardial infarction, coronary artery disease)

Drugs (e.g., magnesium toxicity, local anesthetics)

Embolic (e.g., pulmonary embolism, amniotic fluid embolism)

Fever (e.g., sepsis/chorioamnionitis)

General / Non-obstetric / Trauma

Hypertension (e.g., pre-eclampsia, eclampsia)



40. What are the key points in obstetric CPR?

- Start chest compression at the earliest
- Continuous left lateral uterine displacement when fundal height is at or above umbilicus
- Use AED/Defibrillator when indicated
- Secure airway early and an IV access above diaphragm
- Stop magnesium infusion and give calcium
- Activate massive transfusion protocol if suspecting amniotic fluid embolism
- To perform Resuscitative delivery (replacing the term post-mortem caesarean delivery) within 5 mins if no ROSC

41. Key differences between 2020 and 2025 BLS and ACLS guidelines

Topic	2020BLS Guidelines	2025 BLSGuidelines
Chain of Survival	Separate adult and pediatric chains of survival	Unified chain of survival for adults, children, and infants (except neonates)
uCompression-Ventilation Strategy	Compression-focused CPR strongly emphasized	30:2 CPR reinforced as preferred for trained rescuers rather than compression-only CPR
Rescue Breathing Rate	Variable respiratory arrest ventilation recommendations	Standardized to 1 breath every 6 seconds
Infant CPR Technique	Two-finger compression allowed for single rescuer	Two-finger method largely removed; thumb-encircling preferred
Choking Management	Abdominal thrusts prioritized	Alternating 5 back blows + 5 abdominal thrusts
Opioid Overdose Algorithm	Naloxone incorporated but limited emphasis	Expanded opioid-associated emergency algorithms
AED Use	Early AED emphasized	Continued emphasis with improved pad placement guidance
Mechanical CPR Devices	May be considered when manual CPR difficult	Routine use NOT recommended due to insufficient superiority evidence



Topic	2020 BLS Guidelines	2025 BLS Guidelines
Post-Resuscitation Recovery	Recovery mentioned minimally	Formal recovery phase added to chain of survival
Debriefing After Resuscitation	Suggested in some settings	Stronger recommendation for structured debriefing
Pregnancy Cardiac Arrest	New pregnancy algorithm introduced	Greater focus on maternal resuscitation and early perimortem cesarean
Head-Up CPR	Limited evidence	Discouraged outside research settings
Foreign Body Airway Obstruction (FBAO)	Traditional sequence maintained	Updated severe airway obstruction algorithm

Topic	2020 ACLS	2025 ACLS
Epinephrine Timing (Nonshockable Rhythms)	Early epinephrine strongly emphasized	Continued emphasis maintained
Epinephrine Timing (Shockable Rhythms)	Administer after defibrillation attempts	Delayed epinephrine after second shock reaffirmed
Cardioversion Energy	Lower initial energy acceptable	≥200 J biphasic preferred for AF/flutter
Termination of Resuscitation (TOR)	Less standardized	Clear BLS TOR, ALS TOR, and Universal TOR rules added
ETCO ₂ in CPR	ETCO ₂ used to guide quality and prognosis	ETCO ₂ should NOT be sole criterion for stopping CPR
Temperature Management	Targeted Temperature Management (TTM) emphasized	Terminology shifted to “Temperature Control”
Mean Arterial Pressure (MAP)	Avoid hypotension post-ROSC	Specific MAP optimization emphasized
Vascular Access	IV or IO acceptable	IV access preferred over IO when feasible
Advanced Airway	Endotracheal intubation accepted	Greater focus on minimizing interruptions during airway placement
Endotracheal Drug Administration	Rarely used but still discussed	Removed from modern practice recommendations
Mechanical CPR	Acceptable in selected scenarios	Routine use not recommended
Double Sequential Defibrillation	Limited evidence	Still insufficient evidence for routine use
Neuroprognostication	Introduced in post-ROSC care	Expanded multimodal neurological assessment guidance





Thank you