Objective
ANAESTHESIA
Review

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Review
A Comprehensive Textbook for the Examinees

Third Edition

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Foreword

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Foreword

The Tata Memorial Center has been in the forefront of educational activities and boasts of good record in holding continuing medical education programs in many branches of medicine. The Department of Anaesthesiology, Critical Care and Pain has been conducting the Anaesthesia Review Course, a popular widely appreciated, continuing medical education (CME) for anaesthesia postgraduate students about to undertake their final examination.

Objective Anaesthesia Review: A Comprehensive Textbook for the Examinees was first published in 2009 with financial support from the Tata Memorial Center. The book became instantly popular and has been appreciated for the help it gives the examinees at a critical juncture in their career. The question-answer format of the book is easy-to-follow. The equipment and table viva topics are thoroughly discussed as well.

I am sure that by partnering with M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, the department has made sure of wider availability of the book across the country.

I wish both the book and the exam-going students a great success.

KS Sharma
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Preface

Objective Anaesthesia Review: A Comprehensive Textbook for the Examinees is a culmination of an idea born 9 years ago. We felt that there was no systematic, condensed yet comprehensive teaching program for the anaesthesia postgraduate about to face the examination. Individual departments conduct their own teaching activity, but these are of varying standards, often sporadic, and spaced out through the three years of curriculum. The art and science of anaesthesia is so dependent on technology that a large part of the practical examination focuses on evaluating the examinee's knowledge of equipment. We anaesthesiologists heed the past, as many of today's standards in safety are achieved slowly over ages from equipment of earlier generations. Again, this plethora of equipment, particularly the old ones, may not be available in all institutes. The examinee, therefore, needed a thorough brushing up; in a short span of time; of the clinical cases and equipment and technology.

To fill this lacuna, the first Anaesthesia Review Course was held in 2004 by Department of Anaesthesiology, Critical Care and Pain, Tata Memorial Hospital, Mumbai, Maharashtra, India. This course consisted of interactive case discussions with experienced teachers and hands-on workshops. The course was appreciated for pattern and content by the postgraduates and the faculty alike and it was deemed to be a resounding success. With the first course came the first workbook, this contained the cases written up by the faculty. The Anaesthesia Review Course has been held annually since then and every year there has been a workbook. However, there were several problems. Since we could only discuss only a few cases every year, each year's workbook contained only some cases. So the students had to obtain and go through multiple workbooks. The previous books were scarce, being printed in limited numbers. The equipment and other photographs were not great, being black and white and reproduced using offset print and photocopying. Many such and more complaints came from the anaesthesia postgraduates attending the course.

The idea thus germinated for this book. Why not write a book which shall contain all the cases and the entire range of anaesthesia equipment, and table viva topics such as ABGs, ECGs, PFTs, etc. normally encountered in the examination? This book, therefore, contains all these and more. There are color plates for the old as well as new equipment, which may not be available to all postgraduates.

All our colleagues worked hard for the first edition of the textbook, everybody contributing. Publishing books can be expensive however and we could not have published the first edition without the encouragement and sanction of funds by Dr RA Badwe, Director, Tata Memorial Center. Dr Raman Sareen, the head of our department then and Dr Sharma also encouraged us. Since then, we have published one reprint and second edition of this book. The problem with privately publishing books is the lack of wide availability. Last year M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, approached us for publication of the next edition of the book. You will find many of the chapters completely revamped. We have new chapters on Management of Acute Postoperative Pain and Videolaryngoscopes, something we will increasingly see being used in our operating rooms.

All our colleagues in the department worked hard and they have also often spared us from our clinical duties so we could work on the book and our sincere thanks to all of them. Mr Ramesh from M/s Jaypee Brothers Medical Publishers (P) Ltd, Mumbai Branch, has been remarkably patient with us, and we also thank him for a job well done. Our families have been particularly supportive while we labored for the sake of the book.

It is our heartfelt hope that the examinee really benefits from this book, and we wait eagerly for the feedback from those true and sincere critics, and the exam-going students. We welcome any suggestions and constructive criticism. It will only make the next edition better!

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Section One

Case Discussion

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1 Mitral Stenosis with Pulmonary Hypertension

M Shetmahajan, V Patil

42-year-old lady, with a history of rheumatic mitral stenosis presents with large pelvic mass suspected to be ovarian cyst. She is scheduled for excision of mass. She gives history of balloon mitral valvotomy 13-year ago. She also gives a history of normal pregnancy 15-year ago. She can climb 2 flights of stairs without difficulty but feels breathless beyond this.

What are the causes of mitral stenosis (MS)?

Mitral stenosis refers to decrease in mitral valve area. Most common cause of MS is rheumatic heart disease, although more than 50% of these patients do not give a history suggestive of rheumatic fever in past. Rheumatic fever leads to cardiac inflammation causing pancarditis which heals with scarring, affecting valves. In the acute phase, rheumatic fever may cause mitral regurgitation. Mitral stenosis develops few years later and symptoms develop after many more years when mitral valve area reduces significantly. The stenosis is characterized by fusion, fibrosis, thickening, and calcification of the leaflets, and thickening, fusion and shortening of the chordae tendineae.

Other causes of MS include congenital mitral stenosis, systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), malignant carcinoid, mucopolysaccharidoses (of the Hunter-Hurler phenotype), Fabry disease, Whipple disease, and methysergide therapy.

Describe the pathophysiology of mitral stenosis

The normal mitral valve area is 4 to 6 cm^2. In very early diastole, there is a small pressure gradient between left atrium (LA) and left ventricle (LV), which rapidly equilibrates in the phase of diastasis followed by phase of atrial contraction.

As the mitral orifice narrows in MS, it obstructs free flow of blood from left atrium to left ventricle during diastole, leading to the development of a pressure gradient between two chambers and an increase in LA and pulmonary venous pressure. This gradient is increased by increases in the heart rate (reducing diastolic time) or cardiac output (increasing flow of blood across mitral valve). As obstruction becomes more and more severe, time required for left ventricular filling increases and left atrial contraction (kick) becomes necessary to fill ventricle. As mitral valve area goes below 1 cm^2, left atrial pressure increases to about 25 mmHg (normal LA pressure is about 5 mmHg). Since there are no valves between pulmonary circulation and left atrium, increased left atrial pressure is transmitted to pulmonary circulation causing pulmonary hypertension. Also this constant pressure overload of the left atrium leads to the increase in left atrial size, which then becomes more prone to develop atrial fibrillation and atrial thrombus formation.

As stenosis worsens, flow restriction also limits left ventricular filling and thus LV preload. In addition, there is increased afterload due to reflex vasoconstriction in response to low cardiac output. Both these factors result in decreased LV function, leading to further reduction in cardiac output and thus may mimic left ventricular failure.

In its early stage, the pulmonary hypertension is often described as "passive" and reversible, as elevation of Pulmonary Arterial Pressure is caused solely by passive backward transmission of the elevated left atrial pressure. Over period of time, this leads to pulmonary artery and arteriole vasoconstriction called as reactive pulmonary hypertension. Over longer period, the muscular layer of pulmonary arterioles hypertrophies in response to elevated

pulmonary vascular pressures, resulting in irreversible pulmonary hypertension. Increased pulmonary artery pressure also leads to pressure overload for right ventricle which hypertrophies and ultimately dilates and fails. Thus, in mitral stenosis, two "stenoses" develop; early at the mitral valve, and later in the arterioles of the lung. Workup of the patient with mitral stenosis should include an assessment of both of these obstructions.

Describe Natural History of MS

- Continuous progressive, life-long disease
- Generally slow but often variable clinical course, with some patients showing little or no clinical progression over prolonged periods and others manifesting a more rapid course.
- Onset of symptoms to disability: 10 years
- Incidence of atrial fibrillation 30-40%
- Causes of death
  - CHF
  - Systemic embolism
  - Pulmonary embolism
  - Infection.

What are the signs and symptoms of MS?

History and physical examination should focus on the patient's effort tolerance, current or past evidence of heart failure, and arrhythmias. In mild disease, the patient may be entirely asymptomatic. As the disease progresses patient will start exhibiting signs and symptoms according to severity of stenosis and underlying cardiac changes. Symptoms can be classified according to the causation.

- Due to decreased CO
  - Low effort tolerance
  - Typical "mitral" facies—mild cyanosis of lips and cheeks and malar prominence
  - Easy fatigability
  - Syncope
- Due to increased LAP
- Pulmonary congestion giving rise to dyspnea, orthopnea
- Hemoptysis as pulmonary venous hypertension results in rupture of anastomoses between bronchial veins
- Pulmonary edema
- Due to LA enlargement
  - Ortner's syndrome (hoarseness due compression of left recurrent laryngeal nerve by left atrium)
  - Atrial fibrillation and thromboembolism
- Due to pulmonary hypertension, RV hypertrophy, and RV failure
  - Chest pain due to RV ischemia in severe pulmonary hypertension
  - Raised JVP
  - Right parasternal heave
  - Tricuspid murmur
  - Hepatomegaly
  - Ascites and edema.

What are the causes of hemoptysis?

- Airway diseases
  - Airway trauma
  - Acute or chronic bronchitis
  - Bronchiectasis
  - Foreign body in airway
  - Lung cancer
  - Tracheal or bronchial tumors
- Parenchymal disease
  - Tuberculosis
  - Pneumonia
  - Lung abscess
  - Mycetoma
  - Goodpasture's syndrome
  - Idiopathic pulmonary hemosiderosis
  - Pulmonary contusion
- Vascular causes
  - Pulmonary embolism
  - Pulmonary hypertension
    - Left ventricle failure
    - Mitral stenosis
  - Pulmonary vascular malformation.

How will you grade the severity of mitral stenosis?

Table 1.1 Grades of MS

Severity	MVA, cm^2	End diastolic pressure Gradient, mm Hg (in sinus rhythm)	PAP (mm Hg)	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	>5	NYHA Class III–IV

What are the cardiac findings on examination in MS?

- Loud S1—The transmitral gradient holds the mitral valve open almost to the end of diastole, so that at beginning of ventricular systole, the mitral valve leaflets move along a long distance for closure. In badly diseased valve, S1 may become soft because it neither opens nor closes well. This manifests as tapping apex beat on palpation.
- The pulmonic component of the second sound (A_2P_2) will be loud in presence of pulmonary hypertension.

- Opening snap: The distance from S2 to the opening snap is a good indicator of MS severity. The more severe the stenosis, higher will be left atrial pressure and sooner will the mitral valve open. An S2-opening snap interval < 0.08 seconds usually indicates severe disease.
- Diastolic murmur (low-pitched) at the apex with a presystolic accentuation in patient with sinus rhythm. A diastolic thrill may be palpated in the left lateral position.
- Diastolic murmur (high-pitched) also called as Graham Steel murmur, is due to the pulmonary regurgitation secondary to pulmonary hypertension. It is heard at the left sternal border in the second intercostal space with inspiration.
- Right parasternal heave due to enlarged right ventricle and tricuspid murmur in patients with pulmonary hypertension and right ventricular dilatation.

What is differential diagnosis for a mid-diastolic murmur?

- Mitral stenosis
- Tricuspid stenosis
- Atrial myxoma
- Ball-valve thrombus in left atrium
- Increased flow across the tricuspid valve in ASD
- MR with increased flow through the mitral valve during diastole
- Austin Flint murmur (in severe AR).

Enumerate ECG findings in a patient with MS

- Left atrial hypertrophy:
  - Causes P mitrale: Bifid appearance of P wave in leads II, III and aVF
  - The second half of the P wave is negative in V1
- Atrial fibrillation
  - Right ventricular hypertrophy
  - Dominant R wave in V1 and V2
- Right axis deviation
- For patients on digoxin digitalis effects may be seen (ST depression with a characteristic sagging appearance, decreased T wave amplitude, shortened QT interval, prominent U waves)

Enumerate X-ray findings in a patient with MS

- Usually normal or slightly enlarged cardiothoracic ratio (normal 0.5)
- Straightening of left heart border due to enlarged LA appendage
- Small aortic knuckle due to decreased cardiac output
- Enlarged pulmonary conus in pulmonary hypertension
- Moustache or antler sign—prominence of upper lobe veins
- Double density in mid portion of cardiac silhouette due to LA enlargement
- Elevation of left main stem bronchus due to enlarged LA
- Left lower lobe collapse due to compression of left lower lobe bronchus
- Miliary shadows of pulmonary hemosiderosis due to multiple pulmonary hemorrhages
- Calcification of mitral valve
- Indentation of esophagus on lateral x-ray with barium swallow, due to enlarged LA.

Describe anatomy of mitral valve

Leaflet

- Small anterior leaflet inserts on about 1/3 of the annulus
- Large posterior leaflet inserts into about 2/3 of the annulus.

Chordae

- Primary attachment to free margin
- Secondary and tertiary attachment away from free margin.

Papillary Muscles

Anterolateral and posteromedial supplying both leaflets.

What causes presystolic accentuation of the murmur?

This would be heard only if patient is in sinus rhythm. As mitral valve area goes on decreasing pressure gradient between LA and LV goes on increasing. In severe MS, LV preload becomes more and more dependent on atrial contraction. During atrial contraction there is increased flow from the LA to the LV through the stenotic valve which gives rise to presystolic accentuation.

How do you diagnose rheumatic fever? What are Jones criteria?

Duckett-Jones criteria

For diagnosis of rheumatic fever there should be either 2 major or 1 major + 2 minor criteria present.

- Major
  - Carditis, Sydenham's chorea, subcutaneous nodules, erythema marginatum, arthritis
- Minor
  - Past history of RHD, fever, arthralgia, prolonged PR on ECG, increased ESR or CRP, leukocytosis.

What is Ortner's syndrome?

When patient develops hoarseness of voice from compression of the recurrent laryngeal nerve from an enlarged left atrium, it is called Ortner's syndrome. It was first described in 1897, by N Ortner an Austrian physician.

What is Lutembacher's syndrome?

It is combination of presence of MS with atrial septal defect (of the ostium secundum type) with a left to right shunt.

Explain basic principles of echocardiography. What do you understand by M mode/B mode and Doppler?

Echocardiographic systems emit high frequency sound (> 20,000 Hz) into the tissues. These waves enter the tissue and are then reflected back from the various tissues based on acoustic impedance of the tissues. Since the speed of sound in tissues is known (approximately 1540 m/sec); a standard ultrasound imaging system can wait for a given time for the transmitted pulse to travel to a target (time x) and then back (time 2x); the given target will be received and recorded. These reflected ultrasound waves received by the transducer are then processed by the ultrasound machine to create an image. The best ultrasound images are made when the target is perpendicular to the sound waves.

In the **M-mode technique**, all the ultrasonic pulses are transmitted along the same axis and different parts of the heart are studied by changing the direction of the beam manually. An M- mode echocardiogram is not a "picture" of the heart, but rather a diagram that shows how the positions of its structures change during the course of the cardiac cycle. It is a method for studying a structure like a heart valve.

Doppler echocardiography is a method for detecting the direction and velocity of moving blood within the heart or vessel. It measures the relative change in the returned ultrasound frequency when compared to the transmitted frequency. Depending on this, the Doppler echocardiographic systems measure flow direction, velocity and turbulence of the flow. This helps clinicians to differentiate between normal and abnormal flow patterns.

What are echocardiographic findings in a patient with MS?

M-Mode

- Thickened mitral valve leaflets
- Decreased E-F slope of the anterior mitral valve leaflet to usually less than 30 mm/sec
- Reduced anterior motion of the posterior mitral valve leaflet
- Decreased A wave of the mitral valve leaflet
- Reduced mitral valve leaflet excursion (D-E excursion)
- Abnormal septal wall motion (paradoxical or flat) due to right ventricular volume and/or pressure overload
- Left atrial enlargement
- Pulmonary hypertension.

2-D Echocardiography

- Thickened mitral valve leaflets, especially at the leaflets and chordae
- Left atrial enlargement
- Left atrial thrombus (if present)
- Normal left ventricle
- Right ventricular and right atrial enlargement in pulmonary hypertension.

What is the role of infective endocarditis prophylaxis in MS?

The current American Heart Association (AHA) recommendations no longer suggest infective endocarditis prophylaxis for patients with rheumatic heart disease. Only high-risk group should receive prophylaxis against infective endocarditis. The high-risk groups are:

- Patients with a prosthetic heart valve or prosthetic material used for valve repair
- Patients with a past history of infective endocarditis
- Patients with cardiac valvulopathy after cardiac transplantation
- Specific patients with congenital heart disease.

What are the treatment strategies available for patient with MS?

- Medical management: Mainly consists of
  - Diuretics if congestive symptoms present
  - In presence of AF: Anticoagulants like warfarin and drugs for ventricular rate control such as mainly calcium channel blockers/β-blockers
- Surgical management: Surgical options mainly consist of balloon mitral valvuloplasty, surgical valvulotomy or repair or mitral valve replacement. Patients with moderate or severe mitral stenosis (mitral valve area < 1.5 cm^2) and suitable valve are good candidates for percutaneous balloon mitral valvuloplasty. The major contraindications to balloon mitral valvuloplasty are the presence of thrombus in the left atrium or its appendage, moderate-to-severe mitral regurgitation, and an unfavorable valve morphology. In this group; options are either open mitral valvulotomy or valve replacement where valve cannot be repaired.

On physical examination, she is comfortable and in good general condition. Her blood pressure is 130/70 mmHg, and pulse rate is 80 beats per minute, regular. There is no jugular venous distention, the lungs are normal, and the point of maximal impulse is in the fifth intercostal space in the midclavicular line. The first heart sound is accentuated, an opening snap and a grade 2 diastolic rumble with presystolic accentuation can be heard at apex. There is no hepatomegaly or peripheral edema.

What investigations would you ask for this patient?

- Complete blood count
- Blood grouping and cross matching
- Liver function test
- Renal function test and electrolytes—effect of diuretics and digoxin if patient is receiving any
- ECG
- Chest X-ray
- Echocardiogram—mitral valve area, presence of mitral regurgitation, LA clot, pulmonary pressures

The ECG is normal. The X-ray chest shows a normal-sized heart, an enlarged left atrium, no calcification in the region of the mitral valve, and normal lung fields. Echocardiogram shows moderate mitral stenosis (MVA 2 cm^2) with evidence of mild pulmonary artery hypertension (PAP 38 mmHg) How would you anesthetize this patient? What premedication would you prescribe?

- *Sedatives and anxiolytics:* Prevention of tachycardia is very important as it worsens the left ventricular filling by decreasing diastolic time. Elimination of anxiety avoids tachycardia, but excessive sedation may lead to respiratory depression and hypercarbia and acidosis, resulting in elevated pulmonary vascular resistance. So, sedative premedication like benzodiazepines must be used judiciously in titrated doses.
- *Antibiotic prophylaxis:* This patient would need antibiotic prophylaxis for her surgery which would include good gram-positive and gram-negative cover. Drug can be given as per institutional protocol.
- *DVT prophylaxis:* Since this patient is active with normal lifestyle, chances of preoperative DVT are less. Patients with severe MS with restricted lifestyle should be screened for DVT preoperatively and put on prophylaxis in perioperative period. If epidural catheter insertion is planned confirm that there is a gap of minimum 12 hours between LMWH and epidural catheter placement as per ASRA guidelines.

What medications these patients are likely to be taking and would you continue these drugs?

As we have seen before, these patients are mostly treated with diuretics to relieve congestive symptoms. Digoxin in case of AF and or failure, β-blockers or calcium channel blockers for rate control and anticoagulants in case of AF.

If patient is on diuretics, care should be taken to evaluate fluid status carefully, as hypovolemia in patient with MS may give rise to precipitous fall in BP during induction of anesthesia. Check electrolytes on day of surgery and correct any existing dyselectrolytemia. It is preferable to withhold diuretics on the night before surgery; especially if massive fluid shifts or blood loss is expected during surgery. Drugs to control AF and rate should be continued in perioperative period, monitor serum potassium in patients receiving digoxin and diuretics. Patients with atrial fibrillation on warfarin should be converted to heparin (infusion) in preoperatively. Heparin infusion should be titrated to maintain APTT 1.5–2 times normal and continued in postoperative period.

Describe principles of anesthetic management

1. Establish intra-arterial BP and CVP monitoring. CVP monitoring is of much less value in patients with right ventricular dysfunction
2. Avoid tachycardia
3. Judicious fluid therapy to maintain preload—hypovolemia may lead to a marked drop in cardiac output and hypervolemia can lead to pulmonary edema
4. Avoid fall in SVR or vasodialators; especially in tight MS
5. Avoid increases in PVR: Avoid hypoxia, hypercarbia, nitrous oxide
6. Maintain sinus rhythm; be prepared to cardiovert if patient gets AF. Volatile anesthetics may lead to junctional rhythm with loss of atrial kick.

How would you anesthetize this patient?

There is no ideal technique or drug. Any technique can be used as long as the hemodynamic goals are met.

- Optimize fluid status before induction and establish invasive pressure monitoring
- Use generous amount of opioids to abolish hemodynamic response to intubation. It will also decrease requirement of induction agents. Remifentanil, alfentanil or fentanyl can be used. Patients with moderate to severe MS generally have slow circulation that prolongs arm-brain circulation time. Induction agents should be double diluted and given slowly in titrated doses (sleep dose) till patient goes under. Etomidate is the best agent for hemodynamic stability but midazolam or thiopentone can also be used. Propofol should be avoided as it can lead to precipitous hypotension. Vecuronium, atracurium or rocuronium can be used as they have least effect on heart rate. Pancuronium and D- tubocurarine should be avoided due to their tachycardic response.
- Use titrated doses of vasopressors like phenylephrine to maintain BP and short acting β-blockers like esmolol or metaprolol or calcium channel blockers like diltiazem to control heart rate.

How will you maintain anesthesia?

Volatile anesthetics such as isoflurane, desflurane, or sevoflurane can be administered. Halothane is best avoided;

due to its arryhthmogenic potential and high incidence of junctional rhythm, which can be disastrous in a patient with mitral stenosis. In patients with pulmonary artery hypertension nitrous oxide is best avoided due to its effects on pulmonary resistance. Monitoring of central venous pressure would help in fluid therapy in absence of right ventricular dysfunction.

What is the role of regional anesthesia?

In valvular heart disease, concern is with moderate to severe grades of aortic or mitral stenosis and neuraxial blockade. Epidural anesthesia can be used as a sole anesthesia technique in patients with mild to moderate MS. A number of case reports have been published and reviewed, supporting this. Care should be taken to optimize fluid status and achieve sensory level with titrated doses of local anesthetic agents until it is adequate for surgery. The basic principles of anesthesia remain same i.e. afterload support, maintenance of sinus rhythm, careful volume management and avoidance of tachycardia. Epidural analgesia with opioids can be safely considered as a supplement to GA for other patients. While inserting the epidural catheter, use of adrenaline in the epidural test dose should be avoided.

What precautions would you take to prevent increase in pulmonary vascular resistance intraoperatively?

Avoid

- Hypoxia
- Hypercarbia
- Acidosis
- Lighter planes of anesthesia
- Hypervolemia.

Describe management of perioperative atrial fibrillation

In mitral stenosis, due to the pressure gradient between the left atrium and left ventricle, ventricular filling is dependent on atrial systole and is deleteriously affected in atrial fibrillation (AF). In addition, AF causes stasis of blood in the left atrium, increasing the risk of thrombosis and systemic thromboembolism.

Control of atrial fibrillation with rapid ventricular response should be considered with any of the following agents:

Metoprolol: 5 mg bolus and repeat if needed.

Esmolol: Bolus of 500 mcg per kg IV over 1 minute; may repeat in 5 minutes.

Diltiazem: 10–15 mg IV over 2 minutes; may repeat in 15 minutes.

Digoxin: 0.25–0.5 mg IV; then 0.25 mg IV every 4–6 hours to maximum of 1 mg.

Amiodarone: 150 mg IV bolus over 15 min, followed by 60 mg/h for 6 hours, then 30 mg/h with repeat boluses of 150 mg IV as needed up to a maximum of 2.0 g/d.

All anti-arrhythmic agents have negative inotropic action and hence if the patient is hemodynamically unstable, immediate cardioversion is indicated (synchronized 100 J, 200 J, 300 J, then 360 J monophasic, or biphasic equivalent, with prior sedation).

In case of acute onset AF there is no need to anticoagulate patient but if AF is present for more than 48 hours, patient should be anticoagulated before trying any form of cardioversion.

Anticoagulation is necessary in patients who are unable to maintain normal sinus rhythm.

Anticoagulation may also be beneficial for patients with normal sinus rhythm with a prior embolic event or a left atrial dimension greater than 55 mm Hg noted by echocardiography.

What are the anatomical considerations for percutaneous mitral commissurotomy (PMC) or balloon mitral valvotomy (BMV)?

Wilkins mitral valve score predicts outcome after percutaneous mitral commissurotomy.

Wilkins mitral valve score (On transthoracic 2-D echocardiography)

Leaflet Mobility

1. Highly mobile valve with only leaflet tips restricted.
2. Leaflet mid and base portions have normal mobility.
3. Valve continues to move forward in diastole, mainly from the base.
4. No or minimal forward movement of the leaflets in diastole.

Leaflet Thickness

1. Leaflets near normal in thickness (4–5 mm).
2. Mid-leaflets normal, considerable thickening of margins (5–8 mm).
3. Thickening extending through the entire leaflet (5–8 mm).
4. Considerable thickening of all leaflet tissue (> 8–10 mm).

Leaflet Calcification

1. A single area of increased echo brightness.
2. Scattered areas of brightness confined to leaflet margins.
3. Brightness extending into the mid-portion of the leaflets.
4. Extensive brightness throughout much of the leaflet tissue.

Subvalvular Thickening

1. Minimal thickening just below the mitral leaflet.
2. Thickening of chordal structures extending to one third of the chordal length.

3. Thickening extending to the distal third of the chords.
4. Extensive thickening and shortening of all chordal structures extending down to the papillary muscles.

Each category is scored 0–4, for a total score ranging from 0 to 16. Generally a score < 9 predicts optimal result while score of > 11 signifies severe disease and a suboptimal result after percutaneous mitral valvulotomy. The score does not perform well when the scores are between 9–11.

What types of valves are available? Discuss briefly about each valve.

Bioprosthetic Valves

1. Low incidence of thromboembolism.
2. Average durability is 10–15 years.
3. Chosen in females desiring to become pregnant.
4. Glutaraldehyde-preserved stented porcine tissue valves are the most common.
5. When bioprosthetic valve degeneration is detected, repair should be completed before the onset of symptoms due to the possibility of rupture and embolism.

Mechanical Valves

1. Durable with valve life up to 20 years
2. Requires anticoagulation (INR 2.5–3.0)
3. Usually indicated in young patients or patients with chronic atrial fibrillation
4. Three types of mechanical valves
   a. Caged- ball (Starr-Edwards)
   b. Tilting disk (Medtronic, omniscience)
   c. Bileaflet (St. Jude, carbomedics)

What are the indications for percutaneous balloon mitral valvotomy?

Class I- symptomatic patients (NYHA II, III, IV), moderate or severe MS (valve area < 1.5) and valve morphology favorable to balloon valvotomy in absence of LA thrombus or moderate to severe MR.

Class IIb—asymptomatic patients with moderate or severe MS (valve area < 1.5) and valve morphology favorable to balloon valvotomy in absence of LA thrombus or moderate to severe MR.

What are the indications for mitral valve repair?

- Symptomatic patients (NYHA III,IV), moderate or severe MS (valve area < 1.5) and valve morphology favorable for repair if balloon valvotomy is not available
- Symptomatic patients (NYHAIII,IV), moderate or severe MS (valve area < 1.5) and valve morphology favorable to repair if LA thrombus is present despite anticoagulation
- Presence of non pliable or calcified valve
- Recurrent episodes of embolic events in spite of adequate anticoagulation.

What are the indications for mitral valve replacement?

- Symptomatic patients (NYHA II,III,IV), moderate or severe MS (valve area < 1.5) who are not candidates for percutaneous balloon valvotomy or valve repair.
- Patients with severe MS (valve area < 1) and severe pulmonary hypertension (pulmonary systolic pressures > 60–80 mm Hg) who are not candidates for percutaneous balloon valvotomy or valve repair.

What are indications for anticoagulation?

- Patients with AF (class Ib)
- Prior embolic event even in sinus rhythm (class Ib)
- Thrombus in left atrium (class Ib)
- Severe MS and left atrial dimensions 55 mm on ECHO (class II b).

Anesthetic Considerations for a Pregnant Patient with Mitral Stenosis and Pulmonary Hypertension

Why does pregnancy aggravate the symptoms of mitral stenosis?

The main cardiovascular changes in pregnancy which worsen features of mitral stenosis are:

1. Increase in blood volume by 30–50% starting at end of 1st trimester to peak at 20–24 weeks. This increases pulmonary capillary hydrostatic pressure thereby increasing risk of pulmonary edema.
2. Decrease in systemic vascular resistance.
3. Increase in heart rate by 10–20 beats/min—reduces diastolic filling time of LV.
4. Cardiac output increases by 30–50% after the fifth month. CO returns to normal within 3 days of delivery. Because transvalvular gradient increases by the square of cardiac output, the transvalvular gradient increases significantly. This also raises LA pressure substantially to give rise to symptoms. During pregnancy, the patient's symptomatic status will generally increase by 1 New York Heart Association class.
5. During labor and delivery, there is sympathetic stimulation causing tachycardia and further increase in cardiac output. Also there is sudden rise in venous return to the heart due to auto-transfusion and IVC decompression. This may lead to decompensation.
6. Enlarged atrial dimension predispose to atrial arrythmias including atrial fibrillation.

7. Pregnancy also induces changes in haemostasis which contribute to increased coagulability and thromboembolic risk.

How is pregnancy managed in a patient with MS?

Pregnancy in a patient with valvular heart disease needs a multidisciplinary approach with involvement of the obstetrician, cardiologist and anesthetist with detailed counseling of the patient and her family.

Pregnancy should be deferred till a symptomatic MS is treated as there is worsening of symptoms and high risk of maternal and fetal mortality and morbidity. Patients with MVA < 1.0 cm^2 fare better with percutaneous mitral commisurotomy (PMC) than medical management if pregnancy is planned. The decision to perform PMC before conception in patients with moderate MS should be on the basis of their valve area, symptoms, and exercise tolerance. Patients with MVA > 1.5 cm^2 usually have a favorable pregnancy outcome. Unfortunately, in many patients in the developing countries, MS is first diagnosed during pregnancy as the physiological stress of pregnancy worsens symptoms.

Dyspnea in pregnancy could be due to various reasons but onset in the 2nd trimester (especially between 3rd and 5th month) corresponding to rise in cardiac output should be evaluated further to detect valvular disease.

Mortality in pregnant women with minimal symptoms is less than 1%. The risk of maternal cardiac complications is high in cases of severe stenotic valvular disease i.e. MVA < 1.5 cm^2 and severe pulmonary hypertension (systolic PAP > 75% of systemic pressure). The risk is increased in women with a history of cardiac events, arrhythmias, LV dysfunction (EF < 40%) or who are in NYHA class II, III, IV.

Fetal risk is also high in stenotic heart valve disease due to growth retardation, preterm delivery, and low birth weight.

Echocardiographic examination should be performed in all pregnant patients presenting with dyspnea or heart murmur (except the soft systolic murmurs) or those who have a prosthetic valve. Valve stenosis should be quantified using valve area measurement. Though transvalvular gradients are not reliable markers of severity in pregnancy (because of increased cardiac output), they have a prognostic value. The patient should also be evaluated for pulmonary hypertension and LV function. Chest X-rays should be limited to when absolutely required with appropriate shielding of the abdomen. CT is contraindicated because of the radiation risk but MRI can be performed during pregnancy. Cardiac catheterization is restricted to the interventional procedures with abdominal shielding

Medical therapy: Fetal side effects should be considered when prescribing drugs. It is advocated in patients with mild to moderate symptoms before pregnancy with the aim is to reduce overload and avoid tachycardia. It is advocated in patients with mild to moderate symptoms before pregnancy with the aim to reduce overload and to avoid tachycardia. Fetal side effects should be considered when prescribing drugs (Table 1.2).

Treatment includes bed rest, salt restriction, β-blockers with β1 selectivity (interfere less with β2 mediated uterine relaxation) with diuretics if appropriate. β-blockers are safe but may induce neonatal bradycardia and possible

Table 1.2 Drug therapy and their implications

Drug	Indication	Fetal side effects
Furosemide	To decrease congestion associated with valvular heart disease levels	Increased urinary sodium and potassium levels
β-blockers	Hypertension, supraventricular arrhythmias, to control heart rate in women with clinically significant mitral stenosis	Possible decreased heart rate, possible lower birth weight
Warfarin	For anticoagulation of mechanical heart valves, valvular heart disease with associated atrial fibrillation during 12–36 weeks of pregnancy	Hemorrhage, developmental abnormalities when used between 6 and 12 weeks of gestation
Unfractionated heparin	For anticoagulation of mechanical heart valves, valvular heart disease with associated atrial fibrillation during 6–12 week and after 36 weeks of pregnancy	Hemorrhage, no congenital defects
Low-molecular-weight hepari	Use during pregnancy is controversial	Hemorrhage
Aspirin	Low-dose aspirin occasionally used as an adjunct in patients with previous embolic events or prosthetic-valve thrombosis	Hemorrhage, prolongation of labor, low birth weight (when taken in high doses)
Digoxin	For suppression of supraventricular arrhythmias	No major adverse effects
Adenosine	For immediate conversion of supraventricular arrhythmias	No major adverse effects
Amiodarone	May be used to suppress atrial or ventricular arrhythmias in high-risk patients	Hypothyroidism, intrauterine growth retardation, premature birth. Rarely used during pregnancy because of side effects

growth retardation. Higher doses of β-blockers are required in pregnancy due to increased heart rate in pregnancy. Metoprolol is better than atenolol due to reports of fetal growth retardation with the latter (Beta-agonist agents are contraindicated).

Atrial fibrillation should be treated with cardioversion only when it is acute in onset or after intervention. (Cause of atrial fibrillation in MS is an enlarged LA. So high chance of reversion to fibrillation prior to definitive treatment) Digoxin, Beta-blockers and calcium channel blockers are used for rate control. Verapamil is preferred over diltiazem due to report of fetal side effects. Anticoagulation therapy is needed to avoid thromboembolism in case of chronic atrial fibrillation. Diuretics can impair fetal perfusion and should be used at the lowest dose possible. Quinidine and procainamide can be given for atrial and ventricular arrhythmias as they have good safety profile in pregnancy.

During pregnancy, clinical and echocardiographic follow-up should be performed at 3 and 5 months and every month thereafter in pregnant patients with severe valve stenosis and whenever there is worsening of symptoms.

In the case of dyspnea or pulmonary artery hypertension not responsive to medical management, percutaneous mitral commisurotomy (PMC) should be considered particularly when anatomical conditions are favorable or in case of uncertainty regarding follow-up.

Termination of pregnancy should be considered in severe symptomatic stenotic lesions where PMC is not an option.

Percutaneous mitral commisurotomy is referred over closed (transatrial) mitral commisurotomy (CMC) or open procedure with CP bypass. PMC is better than CMC in terms of valve area and long-term durability.

It is contraindicated in the presence of a clot in LA.

It is best performed after the 20th week of gestation to reduce fetal risk of radiation. Procedural time should be kept as short as possible and abdominal shield used to reduce radiation risk. Patient should be warned of radiation risk to the fetus. Transesophageal echocardiographic (TOE or TEE) guidance may be useful to reduce radiation.

Closed mitral commisurotomy (CMC): It is still practiced in the developing world as it is less expensive and sophisticated cardiac catheterization facilities are not widely available.

Disadvantages

1. Uncontrolled procedure with risk of mitral regurgitation and even mitral valve rupture needing emergency CP bypass.
2. Cannot correct subvalvular deformity.
3. Risk of general anesthesia.

CPB bypass during pregnancy does affect mortality significantly when compared to in non-pregnant state. However, it is associated with a fetal mortality between 20 and 30%. Meticulous maintenance of blood pressure during bypass is important. Fetal well-being should be monitored continuously with a cardiotocograph.

When valvular surgery is required during pregnancy, cesarean section should be performed first if the fetus is viable.

Open commisurotomy: When the valve can be conserved, it avoids the risks of prosthetic valves and also avoids the need for anticoagulation in patients in sinus rhythm. Choice of mechanical valve and a bioprosthesis is determined primarily by assessing the risk of anticoagulant-related bleeding with a mechanical valve vs. the risk of structural valvular degeneration with a bioprosthesis. Bioprosthetic valves are less durable than mechanical valves but avoid the need for anticoagulation.

Antithrombotic management: Oral anticoagulation is recommended for the following situations

- Lifelong for all patients with mechanical valves
- Lifelong for patients with bioprostheses who have other indications for anticoagulation, e.g. atrial fibrillation
- For the first 3 months after insertion in all patients with bioprosthesis with a target INR of 2.5. Aspirin (low dose: 75–100 mg) is also used as an alternative to anticoagulation for the first 3 months, but there are no randomized studies to support the safety of this strategy.

Delivery

Vaginal delivery is recommended whenever possible if the hemodynamic condition is stable at the end of pregnancy. Epidural analgesia helps in reducing sympathetic stimulation. However, titrated epidural boluses with close hemodynamic monitoring is needed to prevent precipitous drop in blood pressure. Hypotension is treated with cautious boluses of crystalloid solution and pure vasoconstrictors like phenylephrine.

The use of obstetric procedures (vacuum or outlet forceps) to shorten extraction time is recommended to reduce the total duration of labor, diminishing hemodynamic consequences. Hemodynamic monitoring is recommended in women with severe MS or LV dysfunction which is continued for 24 hours after delivery.

Cesarean Section

Advantage

- Avoids the hemodynamic consequences of labor.

Disadvantages

- Hemodynamic changes at induction of anesthesia due to vasodilatation and sympathetic stimulation
- Positive pressure ventilation may worsen pulmonary hypertension and affects venous return
- Blood volume shift.

Close hemodynamic monitoring is needed particularly in severe stenosis as these patients are very sensitive to changes in preload and afterload and fluid and vasopressor therapy must be very carefully titrated against blood pressure. Advanced invasive monitoring including continuous arterial BP monitoring and pulmonary artery catheterization may be needed and should be ideally performed in specialized centers.

Spinal anesthesia is contraindicated in severe stenosis because of the uncontrolled hypotension. In less severe cases, spinal anesthesia with a single injection of 1 mL 0.25% bupivacaine and 10–20 mcg of fentanyl or use of spinal catheter has been described. Routine antibiotic prophylaxis in patients with valvular heart disease undergoing uncomplicated vaginal delivery or cesarean section is not indicated. Prophylactic antibiotic therapy can be given at the beginning of labor and during delivery in patients at high-risk, i.e. with previous endocarditis or when overt infection is present. Prophylactic antimicrobial therapy is indicated for invasive urinary tract or gastrointestinal procedures in pregnant women. Intravenous ampicillin and gentamicin, or oral amoxycillin should be used in the non- penicillin allergic patient. Vancomycin and gentamicin are used in the patient with penicillin allergy.

How will you maintain anticoagulation in a patient with a prosthetic valve?

The risk of embolization is greater with the valve in the mitral position than in the aortic position. Other risk factors for increased risk of embolization include atrial fibrillation, LV dysfunction, clotting disorder, and prior embolic events. With either type of prosthesis (Bioprosthetic or mechanical) or valve location, the risk of emboli is higher in the first few months after valve insertion as the valve is not fully endothelialized. In most patients with a mechanical prosthesis, the target international normalized ratio (INR) is 2.5–3.5. The target INR can be reduced to 2.0–3.0 in those patients with a new-generation AVR and no other risk factors for thromboembolic events.

Aspirin is recommended for all patients with prosthetic heart valves: aspirin alone (75–100 mg per day) in patients with bioprostheses and no risk factors or aspirin (75–100 mg per day) combined with warfarin in patients with mechanical heart valves and high-risk patients with bioprostheses.

For minor surgeries, where bleeding is easily controlled, anticoagulation should be with a target of 2.0.

For major surgical procedures, in which anticoagulant interruption is considered essential (INR < 1.5), patients should be admitted to hospital in advance and transferred to intravenous unfractionated heparin. Heparin is stopped 6 hours before surgery and resumed 6–12 hours after. Low molecular weight heparin (LMWH) can be given subcutaneously as an alternative preoperative preparation for surgery. Despite wide use and the positive results of observational studies, the safety of LMWHs has not been widely established and their efficacy has not been proved by controlled studies, particularly in patients at high-risk of valve thrombosis. When LMWHs are used, they should be administered twice a day, using therapeutic rather than prophylactic doses, adapted to body weight and if possible according to monitoring of anti-Xa activity. LMWHs are contraindicated in case of renal failure.

Despite the low level of evidence for both strategies, the European Society of Cardiology (2007) favors the use of unfractionated intravenous heparin. Effective anticoagulation should be resumed as soon as possible.

In valve disease patients, all surgical procedures, even minor, require scrupulous asepsis and avoidance of wound hematoma formation. Antibiotic prophylaxis should be prescribed for those patients undergoing noncardiac procedures which put patients at high-risk of bacteremia.

How will you maintain anticoagulation during pregnancy?

Patients with MS with AF are high-risk for thromboembolism and Vitamin K antagonists are the treatment of choice in non pregnant patients.

Traditional teaching is that patients should be anti-coagulated with heparin in the first trimester of pregnancy and then converted to warfarin for the remainder of the pregnancy. Warfarin should then be stopped prior to delivery. However, there is some literature which supports use of warfarin < 5 mg in the first trimester. Because of different recommendations regarding anticoagulation during pregnancy, the decision is best taken after detailed discussion with the obstetrician, cardiologist and the patient. Vitamin K antagonists are titrated to an INR of 2–3 for best maternal protection. However, Vitamin K antagonists increase the risk of miscarriage, prematurity, and embryopathy when used between the sixth and 12th weeks. (The risk is lower when warfarin dose is < 5 mg/ day). Vitamin K antagonists are contraindicated during labor and delivery because of the risk of cerebral bleeding in the fetus.

Unfractionated heparin therapy is safe for the fetus, but it is associated with a considerable increase in the thrombo-

embolic risk for the mother, including occlusive prosthetic thrombosis. In the first trimester, the use of warfarin offers better protection to mother against thromboembolism but has twice the number of adverse fetal effects compared to unfractionated heparin.

In patients with prosthetic valves, maternal mortality is estimated between 1 and 4% and is mostly related to thromboembolism. The risks are particularly high in patients with mitral valve prostheses. Therefore, these patients should be informed of the risks and they require careful risk assessment and very close monitoring of anticoagulant therapy if pregnancy occurs. Experience with LMWHs remains limited and controversial.

What are the implications of a prosthetic valve in pregnancy?

Pregnancy in a woman with a mechanical valve is associated with an estimated maternal mortality of 1 to 4 percent, with death usually resulting from complications of prosthetic-valve thrombosis. A cardiology opinion is sought for 2 D echocardiographic examination to assess valve function, clots, LV function and to decide anticoagulation plan during pregnancy.

Vaginal delivery can be recommended if:

1. If the patient is not on oral anticoagulation at the onset of labor.
2. If there is no significant prosthetic dysfunction.
3. If there is no other significant cardiovascular disease, e.g. disease at another valve site, impairment of LV function, aortic dilatation, etc.
4. If a specialist obstetric anesthetist is available to provide epidural anesthesia

Recommendations for the evaluation and care of women of childbearing age with mechanical valve prostheses who are taking anticoagulants.

Before Conception

- Clinical evaluation of cardiac functional status and previous cardiac events
- Echocardiographic assessment of ventricular and valvular function and pulmonary pressure
- Discussion of risks associated with pregnancy
- Discussion of risks and benefits associated with anti-coagulant therapy
- Family or pregnancy planning.

Conception

Change to unfractionated heparin (aPTT ratio should be maintained above 2.0) from time of confirmed pregnancy through week 12.(Use of warfarin < 5 mg/day or low molecular heparin has been recommended by some in the first trimester)

Completion of First Trimester

- Warfarin therapy, 12–36 week.
- There is currently no ideal regimen of anticoagulation in women with mechanical heart valves in pregnancy. Women should be offered a choice between the higher rates of fetal loss associated with the use of warfarin and the higher risk of maternal valve thrombosis with subcutaneous heparin (RCOG study group statement September 2011).

Week 36

- Discontinue warfarin
- Change to unfractionated heparin titrated to a therapeutic activated partial-thromboplastin time or anti-factor Xa level.

Delivery

- Heparin should be discontinued at the start of labor
- Oral anticoagulation should be resumed after 24 hours if no concerns regarding bleeding
- Restart heparin therapy 4 to 6 hours after delivery if no contraindications
- If labor occurs preterm while the patient is still on oral anticoagulants, a cesarean section should be performed after reducing the INR to 2.0. A vaginal delivery should be avoided under oral anticoagulation because of the danger of fetal intracranial bleeding.

Prosthetic Thrombosis

It is a dreaded complication with high mortality, should be suspected in any patient who has a prosthetic valve developing worsening of cardiac condition.

Diagnosis: Transthoracic echocardiography and/or TOE.

Management

Options are surgery or fibrinolysis. Surgery is high-risk because it is most often performed in emergency conditions and is reintervention. Fibrinolysis carries risks of bleeding, systemic embolism, and recurrent thrombosis.

What are the implications of cardiopulmonary bypass in pregnancy?

The risk to mother is same as in non-pregnant state. However, it confers high-risk to the fetus

Fetal risk due to

1. Increased uterine contraction
2. Placental hypoperfusion.

Risk of teratogenesis is highest in first trimester due to drugs and possibly cardiopulmonary bypass and surgery during this period is avoided. High flow, high pressure normothermic bypass, tocolytics and fetal monitoring are advocated. Fetus has bradycardic response to CP bypass and is thought to be due to hypoperfusion. Tocolytics with β agonist action are avoided. Magnesium is preferred.

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2 Ischemic Heart Disease

J Divatia, J Doctor, A Chatterjee

A 70-year old hypertensive male patient with history of chest pain is posted for a total gastrectomy.

Name the known risk factors for the development of ischemic heart disease (IHD)

Age, male gender, and positive family history are risk factors that cannot be modified. Hypercholesterolemia, hypertension, and cigarette smoking are the risk factors for which interventions have been proven to lower IHD risk. Sedentary lifestyle, diabetes mellitus, and obesity are the risk factors for which interventions are likely to lower IHD risk.

Describe the normal coronary blood flow (CBF)

The resting CBF averages about 225 mL/min, which is 4–5% of the total cardiac output in normal adults. The CBF increases three to fourfold to supply the extra nutrients needed by the heart at maximum exercise level. The CBF is determined by the pressure gradient between the aorta and the ventricles. There are phasic changes in CBF during systole and diastole in the left ventricle. The blood flow falls to a low value during systole, especially in the subendocardial area, because of compression of left ventricular muscle around the intramuscular vessels and the small pressure gradient between the aorta and the left ventricle (aortic systolic pressure minus the left ventricular systolic pressure). During diastole, the cardiac muscle relaxes and no longer obstructs the blood flow through the left ventricular capillaries. The pressure gradient (coronary perfusion pressure) is high during diastole (aortic diastolic pressure minus the left ventricular end-diastolic pressure). Thus coronary blood flow occurs maximally in diastole. Conditions and interventions that prolong the diastolic time are beneficial, while those that reduce the diastolic filling time are detrimental. The phasic changes of the CBF are much less in the right ventricle because the force of contraction of the right ventricle is much weaker than that of the left ventricle and the pressure gradient is high between the aorta and the right ventricle during the entire cardiac cycle.

Describe the coronary anatomy?

The right coronary artery system is dominant in 80–90% of people and supplies the sinoatrial node, atrioventricular node, and right ventricle. Right-sided coronary artery disease often manifests as heart block and dysrhythmias.

The left main coronary artery gives rise to the circumflex artery and left anterior descending artery, which supply the majority of the interventricular septum and left ventricular wall. Significant stenosis of the left main coronary artery (left main disease) or the proximal circumflex and left anterior descending arteries (left main equivalent) may cause severely depressed myocardial function.

Explain the determinants of myocardial oxygen demand and delivery?

Myocardial oxygen demand is determined by wall tension and contractility. Wall tension (T) is the product of intraventricular pressure (P), radius (r), and the wall thickness (h): ($T = P \times r/2h$) (Laplace's law). Increased ventricular volume (preload) and increased blood pressure (afterload) increases the wall tension and O_2 demand. Increase in contractility in response to sympathetic stimulation or inotropic medications increases O_2 demand also.

Increase in heart rate increases myocardial contractility but decreases the ventricular diameter and the wall tension. Thus, the increase in oxygen demand caused by enhanced contractility is in large part offset by the reduction in oxygen demand that accompanies the reduced wall tension. However, the oxygen demand increases because of more contractions performed per minute.

Myocardial oxygen delivery is determined by oxygen content and coronary blood flow. Oxygen content can be calculated by the following equation:

O_2 content = [1.39 mL O_2/gm of hemoglobin × hemoglobin (gm/dL) × saturation] + [0.003 × PaO_2]

The oxygen content is decreased in anemia and hypoxemia.

Coronary blood flow is determined by coronary perfusion pressure, the time available for perfusion, and the patency of coronary arteries. Coronary perfusion pressure is altered by diastolic hypotension, left ventricular hypertrophy, and increased left ventricular end-diastolic pressure. The diastolic time for perfusion is decreased with tachycardia. The patency of the coronary arteries can be influenced by vasospasm or coronary occlusion caused by atherosclerosis.

What is the pathophysiology of myocardial ischemia?

Myocardial ischemia occurs when coronary blood flow is inadequate to meet the needs of the myocardium. 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) and 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). Most of the sclerotic lesions are eccentrically located so the remainder of the arterial wall is responsive to vasoactive stimuli and is capable of contraction. Therefore, the severity of the stenosis is dynamic and influenced by the vasomotor activity of the free arterial wall.

Nonstenotic causes of myocardial ischemia include aortic valve disease, left ventricular hypertrophy, ostial occlusion, coronary embolism, coronary arteritis, and vasospasm.

What are the clinical manifestations of myocardial ischemia?

The clinical manifestations of myocardial ischemia are varied. Angina pectoris with or without signs of arrhythmias or heart failure is assumed to be the classic manifestation of myocardial ischemia. However, myocardial ischemia may present as ventricular failure or arrhythmias without angina or may remain clinically silent. The dynamic nature of coronary stenosis accounts for the changes in the caliber of a stenosis that may produce rest pain at one time and angina with varying degrees of exercise at other times.

How can the angina be graded?

The Canadian Cardiovascular Society introduced a grading system for angina:

1. **Class I**: Angina with strenuous or rapid prolonged exertion at work or recreation.
2. **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.
3. **Class III:** Angina with walking one to two blocks on the level and climbing one flight of stairs at a normal pace.
4. **Class IV**: Angina may be present at a very low level of physical activity or at rest.

What is unstable angina? Describe the pathogenesis of a perioperative myocardial infarction?

Unstable angina includes a spectrum of syndromes between stable angina and myocardial infarction. The angina attacks may increase in frequency or severity, persist longer, respond less to nitrates and may occur at rest or on minimal exertion. A myocardial infarction (MI) is usually caused by platelet aggregation, vasoconstriction, and thrombus formation at the site of an atheromatous plaque in a coronary artery. Theoretically, sudden increases in myocardial O_2 demand (e.g. tachycardia, hypertension) or decreases in O_2 supply (e.g. hypotension, hypoxemia, anemia, tachycardia, coronary occlusion) can precipitate MI in patients with IHD. Most perioperative ischemic events are unrelated to hemodynamic perturbations, suggesting that intracoronary events may be important in the genesis of perioperative ischemia. Complications of MI include dysrhythmias, hypotension, congestive heart failure, acute mitral regurgitation, pericarditis, ventricular thrombus formation, ventricular rupture, and death.

What clinical factors increase the risk of a perioperative MI following noncardiac surgery?

There are major, intermediate, and minor clinical predictors based on the algorithm for risk stratification and appropriate use of diagnostic testing of the American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Perioperative Evaluation of Cardiac Patients Undergoing Noncardiac Surgery[1]. The guidelines integrate clinical risk factors, exercise capacity, and the surgical procedure in the decision process.

Major factors (markers of unstable coronary artery disease)

- Acute myocardial infarction (< 7 days) or recent MI (7–30 days)
- Unstable or severe angina class III and IV
- Decompensated heart failure (NYHA functional class IV worsening or new onset heart failure)
- Significant arrhythmias
  - High grade atrioventricular block (AV block)
  - Mobitz type II AV block
  - Third degree AV block
  - Symptomatic ventricular arrhythmias
  - Supraventricular arrhythmias (including atrial fibrillation) with uncontrolled ventricular rate (heart rate > 100 beats per minute at rest)
  - Symptomatic bradycardia
  - Newly recognized ventricular tachycardia.

Intermediate factors (markers of stable coronary disease)

- History of IHD (excluding revascularization)
- History of congestive cardiac failure (CCF)
- History of a stroke or transient ischemic attack (TIA)
- Preoperative insulin therapy (Diabetes)
- Serum creatinine > 2 mg% (renal failure).

Minor factors (increased probability of coronary artery disease)

- Familial history of coronary artery disease
- Polyvascular status
- Uncontrolled systemic hypertension
- Hypercholesterolemia
- Smoking
- ECG abnormalities (arrhythmia, LVH, bundle branch block)
- Postinfarction (> 3 months), asymptomatic without treatment
- Post CABG or PTCA > 3 months and < 6 years, and no symptoms of angina and not on antianginal therapy.

How does the type of surgery influence the risk stratification for perioperative ischemia?

- High-risk procedures (risk of perioperative adverse cardiac events > 5%) includes emergent major operations, aortic and major vascular procedures, peripheral vascular surgeries, and anticipated prolonged procedures associated with large fluid shifts and/or blood loss.
- Intermediate-risk procedures (risk of perioperative adverse cardiac events 1–5%) include carotid endarterectomy, head and neck surgery, intraperitoneal and intrathoracic surgery, orthopedic surgery, and prostate surgery.

Low-risk procedures (risk of perioperative adverse cardiac events < 1%) include endoscopic procedures, superficial procedures, cataract surgery, and breast surgery and ambulatory surgery.

How can cardiac function be evaluated on history and physical examination?

If a patient's exercise capacity is excellent, even in the presence of IHD, the chances are good that the patient will be able to tolerate the stresses of surgery. Poor exercise tolerance in the absence of pulmonary or other systemic disease indicates an inadequate cardiac reserve. All patients should be questioned about their ability to perform daily activities, such as is described in the Canadian classification of angina pectoris or the New York Heart Association classification of exercise tolerance.

Assessment of functional capacity: The Duke Activity Status Index (DASI) and approximate metabolic equivalents (METs – 1 MET represents an oxygen consumption of 3.5 mL/kg/min)

1–4 METs

- Standard light home activities
- Walk around the house
- Take care of yourself—eating, dressing, bathing and using the toilet.

5–9 METs

- Climb a flight of stairs, walk up a hill
- Walk one or two blocks on level ground
- Run a short distance
- Moderate activities (golf, dancing, mountain walk)
- Have sexual relations.

>10 MET

- Strenuous sports (swimming, tennis, and bicycle)
- Heavy professional/domestic work like scrubbing floors, lifting or moving heavy furniture.

Signs and symptoms of congestive heart failure, including dyspnea, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, jugular venous distention, a third heart sound, rales, and hepatomegaly must be recognized preoperatively.

What is the definition of recent and prior MI?

Recent monitoring and therapeutic strategies including aggressive recanalization as well as preoperative optimization of patients for surgery significantly decrease the risk for reinfarction even in patients with recent MI. Based on the

decreased risk for perioperative reinfarction; the American College of Cardiology National Database defines an acute MI as one that is ≤ 7 days old. Recent MI is defined as MI occurring within more than 7 days but less than 30 days. Prior MI is defined as an MI that occurred more than 30 days before the surgery.

What ECG findings support the diagnosis of IHD?

The resting 12-lead ECG remains a low cost effective screening tool in the detection of IHD. It should be evaluated for the presence of ST segment depression or elevation, T wave inversion, old MI as demonstrated by Q waves, disturbances in conduction and rhythm, and left ventricular hypertrophy. Ischemic changes in leads II, III and avF suggest right coronary artery disease, leads I and avL monitor the circumflex artery distribution and leads V3–V5 look at the distribution of the left anterior descending artery.

What is the significance of the presence of IHD?

Patients with unstable or severe stable angina (Class III–IV) or recent MI without adequate revascularization are at high risk for developing perioperative MI and prior to undergoing elective noncardiac surgery should be referred for medical evaluation, coronary angiography and revascularization.
Patients with stable angina (Class I–II) or prior MI need further risk stratification and intervention based on their exercise capacity and the severity of the surgery. Patients with poor exercise capacity (< 4 METs) or patients with good exercise capacity (> 4 METs), but who are to undergo a high-risk surgery, need noninvasive testing to evaluate reversible ischemic myocardial areas during exercise. Coronary angiography and appropriate revascularization should be done before noncardiac surgery in patients with exercise-induced myocardial ischemia.

What tests can help further evaluate patients with known or suspected IHD?

Noninvasive cardiac testing of the wide variety of tests advocated, the exercise ECG or stress test is the most cost effective means of detecting coronary artery disease (sensitivity ≈ 70%, specificity ≈ 70%). The exercise ECG simulates sympathetic nervous system stimulation that may accompany perioperative events such as laryngoscopy and surgical stimulation. Interpretation of the exercise ECG is based on: (1) the duration of exercise that the patient is able to perform, (2) the maximum heart rate that is achieved, (3) the time of onset of ST segment depression on the ECG, (4) the degree of ST segment depression, and (5) the time until resolution of the ST segment depression during the recovery period. Certainly, a normal exercise ECG indicates that the coronary circulation is reasonably adequate. The usefulness of this technique is, however, limited by the fact that high-risk patients may be unable to exercise to a level required to achieve target rates, and exercise may be contraindicated in others. Also, approximately 10% of the adult population with normal coronary arteries may develop ST segment changes on the exercise ECG that resemble changes observed in the patient with IHD. For this reason, use of the exercise ECG in an asymptomatic patient is of doubtful value.

Pharmacological stress testing reproduces the cardiovascular effects of exercise and is particularly useful in patients who are unable to exercise. In dipyridamole thallium myocardial imaging, dypyridamole (an adenosine agonist that increases blood flow in normal coronary arteries) is used to simulate the effects of exercise. Images are acquired by the coadministration of a radioisotope, such as thallium, which distributes to myocardium in direct proportion to coronary blood flow. A reversible defect (i.e. a defect seen on the initial stress image that disappears with rest) indicates the presence of coronary artery disease (sensitivity 85–90%, specificity 85–90%).

Dobutamine stress echocardiography: Stress echocardiography has the advantage of being able to assess both regional wall motion abnormalities resulting from induced myocardial ischemia and left ventricular function (in situations of increased myocardial oxygen demand). It offers the best prediction for perioperative events, with a negative predictive value close to 100% (negative or normal stress ECHO rules out coronary artery disease) and a positive predictive value up to 38% among intermediate or high-risk patients.

Coronary angiography: Coronary angiography is an invasive procedure, and is indicated only in cases of unstable coronary syndromes, or uncertain stress tests in high-risk patients undergoing major surgery, or when there is a possible indication for coronary revascularization. Cardiac catheterization has been reported to carry a mortality rate of 0.01% to 0.5% and a rate of serious morbidity ranging from 0.03% to 0.25%[6]

Recommendations for preoperative coronary angiography: The Class I recommendations for preoperative coronary angiography apply only to patients with (i) evidence for high risk of adverse outcome based on noninvasive test results, (ii) angina pectoris unresponsive to adequate medical therapy, (iii) unstable angina, particularly when facing intermediate or high risk non-cardiac surgery, and (iv) equivocal noninvasive test results in patients at high clinical risk undergoing high risk surgery.

Echocardiography can be used to evaluate left ventricular and valvular function and to measure ejection fraction. Stress echocardiography (dobutamine ECHO) can be used to evaluate new or worsened regional wall motion abnormalities in the pharmacologically stressed heart. Areas of wall motion abnormality are considered at risk for ischemia.

How would you stratify risk of anesthesia and surgery for this patient? What are the risk factors in the Lee Revised Cardiac Risk Index?

For quantification of risk, three key questions need to be considered:

1. Are there modifiable operative risk factors?
2. If the patient is at high-risk, should the elective operation be modified, delayed or cancelled?
3. Is coronary revascularization and/or valve surgery indicated?

An overall assessment of perioperative cardiac risk requires consideration of the type of surgery planned, the presence and type of specific clinical indicators of coronary artery disease and the patient's functional status.

Historically, the Goldman Multifactorial Cardiac Risk Index was used to stratify patients according to cardiac risk. The modification of the Goldman Index, proposed by Detsky and colleagues, provides improved risk assessment in patients undergoing vascular surgery. In 1999, Lee and colleagues described revised Cardiac Risk Index (CRI) and is reported to be superior to both previous indices (Table 2.1).

Six risk factors were identified in Lee's revised cardiac risk index (Circulation 1999; 100: 1043-9).This index is now incorporated into the AHA/ACC guidelines for preoperative assessment.

1. High-risk surgical procedure, such as intrathoracic, intraperitoneal or suprainguinal vascular reconstruction.
2. History of IHD (excluding revascularization).
3. History of congestive cardiac failure.
4. History of a stroke or transient ischemic attack (TIA).
5. Preoperative insulin therapy (Diabetes).
6. Serum creatinine > 2 mg% (renal failure).

The presence of each risk factor contributed one point to the index. The risk of cardiac complications depended on the number of risk factors (Table 2.2).

Table 2.1 Risk factors and mortality

No. of risk factors	Event rate
0	0.4%
1	0.9%
2	6.6%
> 3	11%

Other risk indices are not commonly used, but are enumerated below.

Table 2.2 Various cardiac risk indices

	Goldman 1977	Detsky 1986	Eagle 1989	Lee 1999
Age > 70 years	5	5	1	
Emergency surgery	4	10	–	
Intraperitoneal, intrathoracic or aortic surgery	3	–	–	
Canadian Cardiovascular Society Class III angina	–	10	–	
Canadian Cardiovascular Society Class IV angina	–	20	–	
Unstable angina in previous 6 months	–	10	–	
Any angina	–	–	1	
Myocardial infarction in previous 6 months	10	10	–	
Myocardial infarction more than 6 months ago	–	5	–	
Q-wave on ECG	–	–	1	
Third heart sound or jugular vein distension	11	–	–	
Pulmonary edema in previous 7 days	–	10	–	
Any history of pulmonary edema	–	5	–	
Significant aortic stenosis (i.e. gradient > 50 mmHg	3	20	–	
ECG rhythm other than sinus with or without APBs	7	5	–	
> 5 PVBs/min documented on ECG before surgery	7	5	–	

Contd...

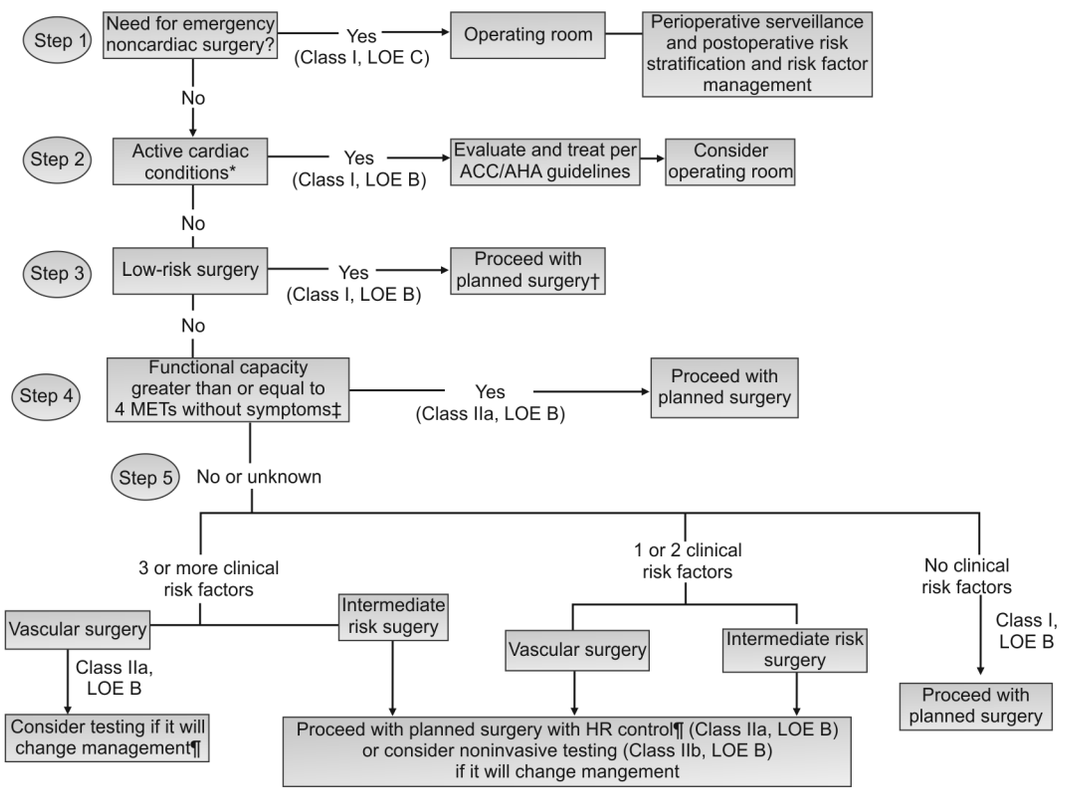
Contd...

	Goldman 1977	Detsky 1986	Eagle 1989	Lee 1999
History of ventricular ectopy	–	–	1	
Diabetes mellitus	–	–	1	
Poor general medical status	3	5	–	
High-risk surgery				1
History of ischemic heart disease				1
History of congestive cardiac failure				1
History of cerebrovascular disease				1
Preoperative treatment with insulin				1
Preoperative serum creatinine > 2 mg%				1
Total possible points	53	120	5	6
Risk Class I	0–5 (1%)	0–15 (0.43)	0 (0–3%)	0 (0.4–0.5%)
Risk Class II	6–12 (6.6%)	20–30 (3.38)	1–2 (6–16%)	1 (0.9–1.3%)
Risk Class III	13–25 (13.8%)	> 30 (10.60)	> 3 (25–50%)	2 (4–7%)
Risk Class IV	> 26 (78%)	–	–	> 2 (9–11%)

Summarize the plan for evaluation of a patient with IHD undergoing noncardiac surgery.

A stepwise evidence-based approach suggested by the AHA/ACC task force[1] is reproduced below:

Flow chart 2.1 Evaluation of IHD patient (AHA)



Should all patients with known or suspected IHD have a preoperative ECG?

According to the above guidelines a resting 12-lead ECG;

- Must be done for patients with;
  - One or more clinical risk factors and vascular surgery
  - Known IHD, Cerebrovascular disease, peripheral vascular disease and intermediate surgery
- Reasonable to do in patients with
  - No clinical risk and vascular surgery
  - One or more clinical risk and intermediate surgery
  - A routine 12-lead ECG is probably not indicated in asymptomatic patients undergoing low-risk procedures.

Which patient's should be subjected to further non-invasive testing?

Patients in whom any of the following two are present should undergo further noninvasive testing:

- Intermediate clinical predictors
- Poor functional capacity (< 4 METs)
- High risk procedure.

This could include:

- Tests for resting LV function
  - Current or poorly controlled CCF
  - Prior LVF and dyspnea of unknown origin
- Exercise or pharmacologic stress test
- Active cardiac conditions
  - Three risk factors and poor functional capacity or 1–2 risk factors and good functional capacity for vascular surgery
  - 1–2 risk factors and poor functional capacity for intermediate surgery
- Noninvasive testing is not indicated for:
  - Low-risk procedures and
  - No risk factors and undergoing intermediate surgery.

Why should stress testing be performed? Which is the method of initial choice?

Resting LV function is not predictive of perioperative ischemic events. Hence, in patients in whom further testing is indicated (as described above) a stress test should be performed. This may include:

- Exercise stress testing
- Non-exercise stress testing
- Dobutamine stress ECHO
- Dipyridamole/ Adenosine thallium testing
- Ambulatory ECG.

The test of choice is an exercise ECG test:

- It provides an objective estimate of functional capacity
- It can detect myocardial ischemia or arrhythmia
- It can be used to estimate perioperative cardiac risk and long-term prognosis.

Regarding strategies to reduce the risk of peri-operative cardiac complications, is it necessary to subject patients with known IHD to coronary revascularization to reduce the risk of major surgery?

CABG: Older studies had shown a significant improvement in survival of revascularized patients, when they undergo subsequent noncardiac vascular surgery. However, when the complication and mortality rates of CABG are taken into account, the combined mortality of cardiac and noncardiac procedures is not different from the mortality of noncardiac surgery when IHD has been managed by medical treatment only. Recently CARP trial by Mc Falls and colleagues has failed to show any benefit of coronary artery revascularization before elective vascular surgery among patients with stable cardiac symptoms. Hence, preoperative CABG should be reserved for patients with:

- Acceptable coronary revascularization risk and suitable viable myocardium with left main stenosis
- Three vessel CAD in conjunction with LV dysfunction
- Two vessel disease involving severe proximal LAD obstruction
- Intractable coronary ischemia despite maximal medical therapy.

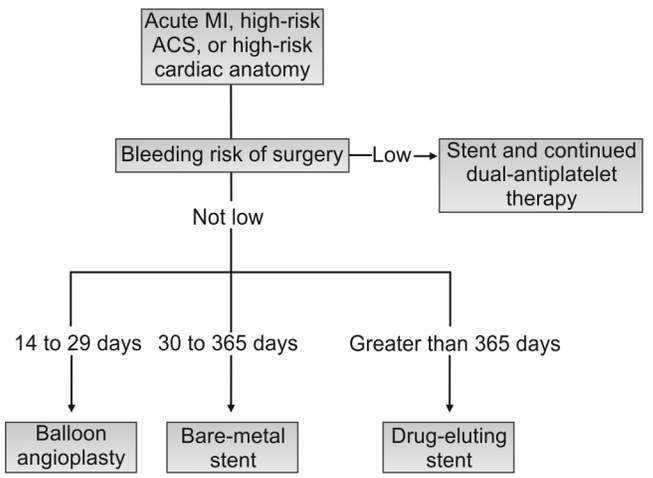
Thus, CABG is indicated only if anatomy or symptoms mandate CABG independent of the planned noncardiac surgery. For major noncardiac surgery following recent CABG, one should keep a gap of at least 4–6 weeks possibly for even up to 6 months.

Percutaneous coronary interventions (PCI): PCI before noncardiac surgery has not been shown to be of value. The indication for PCI depends on the severity of the IHD, rather than impending surgery. In the preoperative period, it may be urgently indicated if there is an acute coronary syndrome (ACS). Unscheduled noncardiac surgery in a patient who has undergone a prior PCI presents special challenges. All patients with stents require dual antiplatelet therapy with aspirin and clopidogrel for a variable period of time. Any surgery performed within 4–6 weeks of a PTCA presents an excessive risk of stent thrombosis and infarction if the antiplatelet medication is stopped, or of major bleeding if the antiplatelet treatment is maintained throughout the perioperative period. The ACC/AHA guidelines now recommend a delay of at least two weeks and ideally 4–6 weeks between implantation of a bare metal stent and noncardiac surgery. This allows 4 weeks of antiplatelet therapy during stent reendothelialization and also a safe option of withholding antiplatelet drugs for 2 weeks prior to the surgery without the risk of stent blockage.

In circumstances where surgery in immediate future is anticipated, it may be preferable to place a bare metal stent or perform a balloon angioplasty without a stent placement. The following algorithm is suggested by the AHA/ACC Task Force:

Management of patients requiring a PCI who need subsequent surgery:

Flow chart 2.2 Patient with PCI needing surgery



Are pharmacological strategies (beta-blockade) useful to reduce perioperative cardiovascular complications?

Rationale for the use of perioperative β-blockade therapy: Perioperative period is a period of physiological stress associated with high levels of catecholamine's in blood. Catecholamines increase each of the four determinants of myocardial oxygen consumption (i.e. heart rate, preload, afterload and contractility). β-blockers have the potential of reducing myocardial O_2 consumption (thus improving the myocardial O_2 supply/demand balance) by decreasing sympathetic tone and myocardial contractility, which in turn decreases the heart rate and arterial pressure. Two randomized, controlled trials have shown that β-blocker therapy reduces perioperative cardiac complications. Mangano and colleagues performed a randomized, double-blind, placebo-controlled trial with atenolol in 200 patients with known coronary artery disease or risk factors for atherosclerosis who underwent noncardiac surgery. No perioperative deaths occurred in patients given atenolol, and only one death was reported in patients receiving the placebo. However by 6 months, there were 8 deaths in the group that received the placebo and none in the group given atenolol ($p < 0.001$). This difference was sustained for the 2-year follow-up period.

In Poldermans and colleagues' study, 112 patients with one or more clinical risk factors and ischemia as identified by a dobutamine stress echocardiography, scheduled to undergo abdominal aortic aneurysm repair or infrainguinal arterial reconstruction were randomly assigned to placebo or bisoprolol. The study was terminated early when investigators noted that bisoprolol markedly reduced perioperative mortality (17% versus 3.4%, $p = 0.02$) and myocardial infarction (17% versus 0%, $p < 0.001$).

However, several other recent trials have not shown any benefit using perioperative beta blockade. The recently published POISE (Perioperative Ischemia Study Evaluation) trial found that while the incidence of cardiac complications was reduced by metoprolol, the overall mortality and the incidence of stroke was significantly higher in patients receiving metoprolol. This may have been due to a higher incidence of hypotension in these patients.

The 2009 ACCF/AHA focused update on perioperative beta blockade[2] summarizes the recommendations (Level of evidence) as follows:

- Class I: β-blockers are recommended in patients who are receiving β-blockers for treatment of conditions with ACC/AHA Class I indication for the drug (I C)
- Class II :
  - β- blockers are probably recommended in patients
    - Undergoing vascular surgery who suffer from coronary artery disease (CAD) or show ischemia on preoperative testing (IIa B)
    - In the presence of CAD or high cardiac risk (i.e. more than one risk factor) undergoing intermediate-risk surgery (IIa B)
    - When preoperative assessment for vascular surgery identifies high cardiac risk (i.e. more than one risk factor) (IIa C)
  - The usefulness of β-blockers is uncertain in patients
    - In patients undergoing vascular surgery with no risk factors who are not currently taking β-blockers. (IIb B)
    - Undergoing either intermediate-risk procedures or vascular surgery with a single clinical risk factor in the absence of CAD. (IIb C)
- Class III:
  - β- blockers are not to be given
    - High dose β- blockers without titration are not useful and may be harmful to patients not currently taking β- blockers who are undergoing surgery (III B)
    - Patients undergoing surgery who have an absolute contraindication to β- blockade (III C).

The guideline recommends that β-blockers be started days to weeks before surgery to titrate the heart rate to 60–80 beats/minute. β-blockers should be omitted if there is hypotension.

Which β-blockers should be used and in what doses? What should be the targeted effect?

Drug of choice: Atenolol or metoprolol can be used. If the patient is on another β-blocker, there is no need to change to a cardioselective β-blocker. However the dose should be adjusted to maintain a heart rate (HR) < 70 beats/minute. Avoid bradycardia and hypotension.

- β-blocker initiation: Target HR between 50–70 beats/ minute
- Preoperatively if HR > 60 beats/ minute and blood pressure (BP) > 100 mmHg : Start Metoprolol 25–50 mg twice daily (BID) dose or atenolol 50–100 mg daily for at least a week prior to surgery
- Immediately preoperative if the HR > 70 beats/ minute and the BP > 100 mmHg: Give metoprolol 2.5–5 mg IV every 10 minutes to get the HR < 70 beats/ minute and BP > 100 mmHg
- Intraoperatively if the HR > 70 beats/ minute and the BP > 100 mmHg: Give metoprolol 2.5–5 mg IV every 10 minutes to get the HR < 70 beats/minute or an esmolol infusion can be started to maintain HR < 70 beats/minute.

What is the role of α_2- adrenergic agonists?

Not everyone with increased perioperative cardiac risk can tolerate a β-blocker. Clonidine, a centrally acting α_2-adrenergic agonist may prove to be an effective alternative. Perioperatively, it is used as a sedative, anxiolytic and an analgesic. It reduces hypertension, tachycardia and norepinephrine release associated with surgical stress.

The effect of α_2- adrenergic agonists has also been studied in the perioperative period. Several small randomized studies comparing clonidine with a placebo failed to demonstrate that clonidine reduced the rate of myocardial infarction and death from cardiac causes. However, in a meta-analysis conducted by Mikawa et al, the trend favored clonidine. The rates for clonidine versus the control groups were 4.4% versus 7.5% for myocardial infarction and 0.7% versus 1.7% for death. However in the largest study involving CABG patients, clonidine was associated with significantly higher odds of bradycardia and hypotension.

What about the use of statins in the perioperative period?

There are no definitive randomized controlled trials regarding the perioperative use of statins. However, AHA makes the following recommendations:

- Recommended: For patients currently taking statins and scheduled for noncardiac surgery, statins should be continued
- Reasonable to use: For patients undergoing vascular surgery with or without clinical risk factors
- May be considered: For patients with at least 1 clinical risk factor and undergoing intermediate-risk procedures.

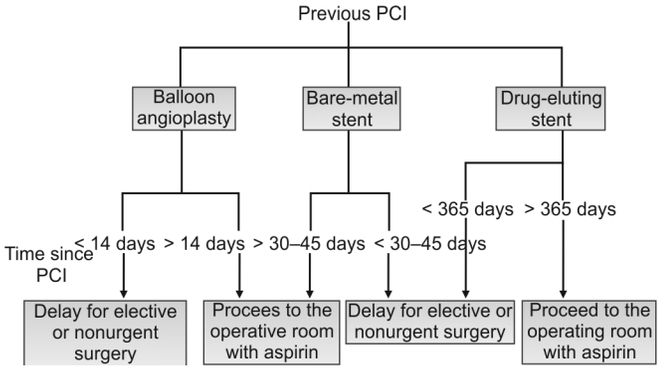
Should all cardiac medications be continued throughout the perioperative period?

Patients with a history of IHD are usually taking medications intended to decrease myocardial oxygen demand by decreasing the heart rate, preload, afterload, or contractile state (e.g., β-blockers, calcium channel antagonists, nitrates) and to increase the oxygen supply by causing coronary vasodilatation (nitrates). These drugs are generally continued throughout the perioperative period. Abrupt withdrawal of β-blockers can cause rebound increases in heart rate and blood pressure. Antagonists of the rennin angiotensin aldosterone system may occasionally cause refractory hypotension that responds only to vasopressin or terlipressin.[3,4] Based on cumulative evidence from randomized controlled trials, it is recommended that angiotensin receptor blockers or ACE inhibitors be discontinued on the morning of surgery.

Aspirin may be continued throughout the perioperative period. Clopidogrel should however be discontinued for a week prior to surgery.

Proposed approach to the management of patients with previous PCI who require noncardiac surgery.

Flow chart 2.3 Plan for patient with previous PCI



How long should a patient with a recent MI wait before undergoing elective noncardiac surgery?

The risk of reinfarction during surgery after a prior MI has traditionally depended on the time interval between the MI and the procedure. The ACC/AHA task force eliminated the arbitrary 6-month time interval and suggested that elective surgery is associated with prohibitive risk only during the

initial 4–6 weeks. The patient's functional status following rehabilitation from an MI is probably more important than the absolute time interval. Patients with ongoing symptoms may be candidates for coronary revascularization prior to their noncardiac procedure.

How is premedication useful in the setting of IHD and surgery?

Patient anxiety can lead to catecholamine secretion and increased oxygen demand. In this regard, the goal of premedication is to produce sedation and amnesia without causing deleterious myocardial depression, hypotension, or hypoxemia. Morphine, scopolamine, and benzodiazepines, alone or in combination, are popular choices to achieve these goals. All premedicated patients should receive supplemental oxygen.

Outline the hemodynamic goals of induction and maintenance of general anesthesia in patients with IHD

The anesthesiologist's goal must be to maintain the balance between myocardial O_2 demand and supply throughout the perioperative period. During induction, wide swings in heart rate and blood pressure should be avoided. Ketamine should be avoided because of the resultant tachycardia and hypertension. Prolonged laryngoscopy should be avoided, and the anesthesiologist may wish to blunt the stimulation of laryngoscopy and intubation by the addition of opiates, β-blockers, or laryngotracheal or intravenous lignocaine.

Maintenance drugs are chosen with knowledge of the patient's ventricular function. In patients with good left ventricular function, the cardiac depressant and vasodilatory effects of inhaled anesthetics may reduce myocardial O_2 demand. An opioid-based technique may be chosen to avoid undue myocardial depression in patients with poor left ventricular function. Muscle relaxants with minimal cardiovascular effects are usually preferred.

Blood pressure and heart rate should be maintained near baseline values. This can be accomplished by blunting sympathetic stimulation with adequate analgesia and aggressively treating hypertension (e.g. anesthetics, nitroglycerin, nitroprusside, β-blockers), hypotension (e.g., fluids, sympathomimetics, inotropic drugs), and tachycardia (e.g., fluids, anesthetics, β-blockers).

What monitors are useful for detecting ischemia intraoperatively?

The V5 precordial lead is the most sensitive single ECG lead for detecting ischemia and should be monitored routinely in patients at risk for IHD. Lead II can detect ischemia of the right coronary artery distribution and is the most useful lead for monitoring P waves and cardiac rhythm.

Transesophageal echocardiography can provide continuous intraoperative monitoring of left ventricular function. Detection of regional wall motion abnormalities with this technique is the most sensitive monitor for myocardial ischemia.

The pulmonary artery occlusion (wedge) pressure gives an indirect measurement of left ventricular volume and is a useful guide to optimizing intravascular fluid therapy. Sudden increases in the wedge pressure may indicate acute left ventricular dysfunction due to ischemia. The routine use of pulmonary artery catheters in patients with IHD has not been shown to improve outcome. However, close hemodynamic monitoring (including pulmonary artery catheter data) may be beneficial depending on the patient's condition and the nature of the surgical procedure.

What are the goals of anesthetic management in this patient?

The primary goal of the anesthetic management of a patient with IHD for noncardiac surgery is the avoidance of myocardial ischemia and myocardial infarction (MI). This is accomplished by preventing ischemia through measures that improve the myocardial oxygen supply-demand balance, primarily by controlling the patient's hemodynamics, and by detecting and treating myocardial ischemia when it does occur. The pathophysiology of perioperative myocardial infarction differs somewhat from that of myocardial infarction occurring in the nonoperated patient, where rupture of a coronary arterial atherosclerotic plaque leads to platelet aggregation and thrombus formation. In contrast, plaque rupture is not always the cause of perioperative myocardial infarctions; most MIs are due to a prolonged imbalance between myocardial oxygen supply and demand in the setting of coronary artery disease.

Myocardial oxygen supply-demand balance

The myocardium extracts most of the oxygen from the blood flowing through the coronary arteries in the normal individual at rest. Increased cardiac work for any reason (e.g. physical exertion, emotional stress, and hypertension) increases myocardial oxygen demand, which is compensated in the healthy person by increased coronary blood flow. With IHD, the arteries are narrowed and the coronary blood flow is already maximal. When oxygen demand by the myocardium exceeds its oxygen supply, myocardial ischemia develops, which, if prolonged, results in myocardial infarction.

Hence, the cornerstone of prevention and treatment of myocardial ischemia is to improve oxygen supply and reduce

Table 2.3 Causes of imbalance in myocardial oxygen supply and demand

Decreased oxygen supply	Increased oxygen demand
Decreased coronary blood flow	Tachycardia Increased wall tension Increased preload Increased afterload (hypertension) Increased myocardial contractility
Tachycardia (diastolic perfusion time) Hypotension (especially diastolic) Increased preload (perfusion pressure) Hypocapnia (coronary vasoconstriction) Coronary artery spasm	
Decreased oxygen content and availability	
Anemia Hypoxemia Reduced oxygen release from hemoglobin (e.g. pH, 2,3-DPG, temperature)	

oxygen demand. It is important to know the conditions detrimental to myocardial oxygen balance because they are encountered frequently in the perioperative period, and can be caused and/or manipulated to some degree by the anesthesiologist (Table 2.3).

Describe perioperative anesthetic management for the above patient?

All anesthetic techniques must aim to keep myocardial oxygen supply greater than demand, and therefore avoid ischemia. The essential requirements of general anesthesia for IHD are avoiding tachycardia and extremes of blood pressure, both of which adversely affect the balance between oxygen supply and demand.

Premedication. A nervous patient may be tachycardic and require an anxiolytic premedication. The patient may already be on medication for angina or hypertension. These drugs include β-blockers, nitrates, and calcium antagonists. These protect against the hemodynamic stresses of surgery and should be continued through the perioperative period. However, general anesthesia may exaggerate the hypotensive actions of such drugs.

Induction. All intravenous anesthetic agents have a direct depressant action on the myocardium, and may also reduce vascular tone. This causes hypotension (especially in the hypovolemic patient), often with a compensatory tachycardia, which may cause myocardial ischemia. To avoid hypotension at the time of induction all agents should be given slowly and in small increments. However, since ketamine causes indirect stimulation of the sympathetic nervous system, leading to both hypertension (increased afterload) and tachycardia, it should be avoided.

Intubation. Laryngoscopy is a powerful stressor, causing hypertension and tachycardia. This can be avoided with a supplemental dose of intravenous induction agent or liberal dose of an opioid like fentanyl prior to laryngoscopy. Also minimize duration of laryngoscopy (15 sec or less) which may be useful in reducing the magnitude of sympathetic stimulation.

Maintenance. Volatile agents have minimal effects on cardiac output, although they do reduce myocardial contractility, especially halothane. They cause vasodilatation, and isoflurane has been implicated in the 'coronary steal' syndrome. The theory is that prestenotic vasodilatation diverts blood away from already ischemic 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, provided blood pressure is maintained. Iatrogenic hyperventilation, which greatly decreases $PaCO_2$, should be avoided as hypocapnia may evoke coronary artery vasoconstriction. It is important to avoid persistent and excessive changes in heart rate and systemic BP. A common recommendation is to strive to maintain patient's heart rate and BP within 20% of normal awake value. Maintain good pain relief and adequate depth of anesthesia to minimize sympathoadrenal response. Pain relief should be provided with liberal use of opioids. Theoretically, nonsteroidal anti-inflammatory drugs (NSAIDs) may have both a useful postoperative analgesic action and an antiplatelet effect which may reduce coronary thrombosis.

Maintain normothermia during intraoperative period as hypothermia will lead to peripheral vasoconstriction thus leading to increased afterload. Also these patients will develop shivering in postoperative period thus increasing myocardial work and oxygen demand.

Anemia is well-tolerated in the general population, but can cause a critical reduction in myocardial oxygen supply in those with IHD—a hematocrit of 30% or more is recommended.

Reversal and recovery. Reversal of muscle relaxation with a combined anticholinesterase/antimuscarinic causes tachycardia, and extubation in itself is a stressor. Problems in the recovery phase which can cause ischemia 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.

The use of regional anesthetic techniques has theoretical advantages: epidural anesthesia reduces preload and afterload, coagulation responses, and in the case of thoracic epidurals, causes coronary vasodilatation. These effects should reduce perioperative myocardial ischemia, but this is not supported by any evidence. However, good epidural analgesia may reduce the incidence of tachycardia arising due to postoperative pain. However, care should be taken to optimally fill these patients before giving a central neuraxial blockade. Despite decrease in myocardial O_2 requirements produced by peripheral sympathetic nervous system blockade it is important to realize that flow in the coronaries narrowed by atherosclerosis is pressure dependent. Therefore, decreases in systemic BP with epidural or spinal anesthesia should not be permitted to persist. In a patient with IHD, local anesthetic techniques whenever feasible such as brachial plexus block should be encouraged in order that the hemodynamic responses to general anesthesia are avoided. However, even under local anesthesia, the patient will be subject to the stresses of the surgical procedure itself as well as anxiety related to it, which can have marked hemodynamic effects.

Describe your strategies to prevent perioperative MI (PMI) in this patient?

The risk of perioperative MI peaks within first 72 hours after surgery with most events occurring on day of surgery or next 24 hours. PMI is due to either acute plaque disruption or due to a myocardial oxygen supply/demand mismatch. Increases in sympathetic discharge with accompanying elevation of heart rate and BP, procoagulant postoperative environment and mobilization of interstitial fluid administered during perioperative period may promote plaque rupture and subsequent cardiac event.

Two principal strategies have been used in an attempt to reduce the incidence of PMI's and other cardiac events and complications: preoperative coronary revascularization, and pharmacological treatment.

What postoperative care would you advice for this patient?

- Continuous ECG monitoring and surveillance for post-operative myocardial ischemia and infarction
- Continuation or institution of beta-blockade
- Temperature control: Decreases in body temperature that occur intraoperatively may predispose to shivering on awakening leading to abrupt excessive increases in myocardial O_2 requirement
- Provision of supplemental O_2
- Adequate postoperative pain relief
- Maintenance of hemodynamic parameters with IV fluids
- Deep venous thrombosis prophylaxis.

How would you diagnose and manage perioperative MI in this patient?

Mechanisms of Perioperative Myocardial Infarction (PMI)

The risk of PMI peaks within first 72 hours after surgery with most events occurring on day of surgery or next 24 hours. Most of these events are silent and of non-ST-elevation myocardial infarction (NSTEMI) variety, as shown by various studies. In PMI, acute plaque disruption and hemorrhage in the infarct-related coronary artery seems to be common. On the other hand, a myocardial oxygen supply/demand mismatch is often believed to be the main trigger of myocardial injury. However, myocardial oxygen supply/demand mismatch and plaque rupture are not mutually exclusive mechanisms, and MI's may develop by different mechanisms at different locations in the same patient. In cases of fatal PMI's autopsy findings have shown plaque rupture as cause of PMI in 45–55% cases whereas half of the patients had no evidence of plaque rupture in their coronaries in spite of having extensive CAD. Increases in sympathetic discharge with accompanying elevation of heart rate and BP, procoagulant postoperative environment and mobilization of interstitial fluid administered during perioperative period may promote plaque rupture and subsequent cardiac event. Most PMI's (> 80%) which occur early after surgery, are asymptomatic, are of the non-Q-wave type and are most commonly preceded by ST-segment depression rather than ST-segment elevation. The majority of PMI's are silent showing no signs or symptoms and may be completely overlooked if continuous ECG monitoring is not performed. Long duration (single duration > 20–30 min or cumulative duration >1–2 h) rather than merely the presence of postoperative ST-segment depression, seems to be the important factor associated with adverse cardiac outcome. The frequent combination of increases in heart rate preceding the ischemic episodes,

ST-segment depression rather than elevation during all ischemic episodes; non-Q-wave rather than Q-wave MIs in almost all cases; the lack of angiographically visible thrombus or ruptured plaques in some patients who underwent coronary angiography following PMI; and complete reversal of ECG changes to baseline are highly suggestive that prolonged stress-induced myocardial ischemia is the likely primary cause of PMI. Repeated brief ischemic episodes may well have a cumulative effect and ultimately cause myocardial necrosis.

In summary, it can be said that both plaque rupture and prolonged perioperative ischemia in presence of stable CAD contribute to PMI. Probably, this could explain the benefit of beta blockade therapy.

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) increased serum concentration of creatine kinase (CK)-MB isoenzyme; and (iii) typical electrocardiographic findings, including development of pathological Q-waves. Perioperative MI is mostly silent, and the ECG is often difficult to interpret as it frequently does not exhibit characteristic ST-segment elevation or Q-waves. Even in nonsurgical patients ECG is diagnostic only in 50% of cases, some abnormality but not classic MI changes in 40% and normal in 10% patients. Also pain if present is often masked by analgesia and residual anesthetics in the immediate perioperative period. Therefore, if the diagnosis of MI is based solely on the classical triad considerable under-reporting could happen.

Routine ICU monitoring with two lead ECG and ST segment trending detects ischemia only in 3% of high-risk postoperative patients when compared to 12 lead ECG[5]. The majority of ischemic events occurred in leads V1, V2, and V4 and not in commonly monitored leads II and V5. Also preexisting bundle branch blocks, digitalis effects or paced beats make interpretation of ECG difficult. All these limitations put heavy emphasis on biochemical markers as diagnostic test in perioperative period. CK-MB is the most routinely available and done marker and has sensitivity of 60–75% and specificity of 80–95% in perioperative period. The cardiac troponins (troponin T and I) are relatively newer cardiac injury markers that are rapidly released into the circulation after myocyte injury. According to some studies cardiac troponins appear to be better suited to identify PMI than the CK-MB isoenzyme. Troponins have nearly absolute myocardial tissue specificity and a high sensitivity. Additional testing with ECHO to assess LV function may be required to establish definite diagnosis. In patients with known CAD undergoing high-risk surgery, the ACC/European Society of Cardiology joint guidelines recommend obtaining ECGs at baseline, immediately after surgery and on the first 2 days following surgery; biomarkers should be obtained for these high-risk patients and those with clinical or ECG evidence of cardiovascular dysfunction.

Management of PMI

The major difference between perioperative patients and nonsurgical patients is the risk of life-threatening bleeding with thrombolytic therapy in the former. Thus thrombolysis is almost always contraindicated. The bleeding risk is high even with aggressive use of antiplatelet agents and anticoagulants. Hence, in the perioperative setting, a more conservative approach is recommended. 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.

a. *Pain relief:* Pain if present should be treated with opioids, most commonly used being morphine, as it decreases preload, thus decreasing myocardial oxygen consumption.
b. *Nitrates:* As certain studies did not show survival benefit for nitrates, use of nitrates is optional for patients without evidence of ongoing ischemia, but for patients with symptomatic ischemia IV nitroglycerine is effective.
c. *Antiplatelet agents:* Aspirin should be administered as soon as MI is suspected in the dose of 375 mg orally/through NGT. Other antiplatelet agents are available, clopidogrel being the widely used one. Patients with allergy to aspirin, clopidogrel can be used. Aspirin being a weak antiplatelet agent it is advisable to combine both however, studies have shown that combination increases the relative risk for major perioperative bleeding by approximately 50%.
d. *Anticoagulants:* Acute MI is often associated with increased thrombin activity. Unfractionated heparin is associated with indirect thrombin inhibition and is frequently used to treat acute MI. Low molecular weight heparins act through their anti-Xa activity and have predictable kinetics but suffer from the longer half life. In the perioperative setting IV UFH is indicated if suspicion of plaque rupture is high and bleeding risk is low.
e. *β-blockers:* β-blockers should be used for all the patients with acute coronary event unless there is significant bradycardia, decompensated CHF, or severe COPD. Calcium channel blockers have no survival benefit in such situations and should not be used.

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3 Hypertension

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49-year-old female with carcinoma esophagus weighing 56 kg is posted for total esophagectomy. She is a known hypertensive and diabetic for the past 10 years, and is on tab losartan 50 mg daily, tab metformin 500 mg daily in two divided doses and mixtard insulin 10 units before breakfast. On examination she has a pulse rate of 84/min, BP 140/90 mm Hg, good effort tolerance (3 flights). She has some missing teeth and also buck teeth.

Define hypertension

The American Society of Hypertension has defined hypertension as a progressive cardiovascular syndrome arising from complex and interrelated etiologies. Early markers of the syndrome are often present before BP elevation is sustained; therefore, hypertension cannot be classified solely by discrete BP thresholds. Progression is strongly associated with functional and structural cardiac and vascular abnormalities that damage the heart, kidneys, brain, vasculature, and other organs and lead to premature morbidity and death.

In other age groups, systemic hypertension is diagnosed when the blood pressure is above the levels mentioned below.

- Adolescent 100/75 mm Hg
- Early childhood 85/55 mm Hg
- Infant 70/45 mm Hg.

Table 3.1 Classification of hypertension

Classification	Normal	Stage 1 hypertension	Stage 2 hypertension	Stage 3 hypertension
BP pattern and cardiovascular risk factors (CVD)	Normal BP or rare BP elevations AND No identifiable CVD	Occasional or intermittent BP elevations OR Risk factors or markers suggesting early CVD	Sustained BP elevations OR Evidence of progressive CVD	Marked and sustained BP Elevations OR Evidence of advanced CVD
Cardiovascular risk factors	None	≥1 risk factor present	Multiple risk factors present	Multiple risk factors present
Early disease markers	None	0–1	≥2	≥2 present with evidence of CVD
Target organ disease	None	None	Early signs present	Overtly present with or without CVD events

What is white coat hypertension?

White coat effect (WCE) or white coat phenomenon, is the transient rise in BP from before to during the visit to the doctors clinic, which settles down after a period of rest. White coat hypertension is due to increased anxiety leading to high blood pressure recorded in a medical setting with normal ambulatory blood pressure response.

What are early markers of hypertensive cardiovascular disease?

Blood Pressure

1. Loss of nocturnal blood pressure dipping
2. Exaggerated blood pressure responses to exercise
3. Salt sensitivity
4. Widened pulse pressure.

Cardiac

1. Left ventricular hypertrophy (mild)
2. Increased atrial filling pressure
3. Decreased diastolic relaxation.

Vascular

1. Increased central arterial stiffness or pulse wave velocity
2. Small artery stiffness
3. Increased systemic vascular resistance
4. Increased wave reflection and systolic pressure augmentation
5. Increased carotid intima—media thickness
6. Coronary calcification
7. Endothelial dysfunction.

Renal

1. Microalbuminuria (urinary albumin excretion of 30–300 mg/d)
2. Elevated serum creatinine
3. Reduced estimated glomerular filtration rate(60–90 mL/min).

Retinal

1. Hypertensive retinal changes.

How would you evaluate hypertensive patient for surgery?

In preoperative evaluation pay special attention to cause and severity of hypertension, current therapy and evidence of end organ damage. When hypertension is detected during preoperative evaluation, by measurement or history, two other issues other than blood pressure control are important—screening for secondary hypertension and extent of target organ dysfunction related to long standing hypertension. Hypertension is also a risk factor for coronary artery disease or cardiomyopathy. When assessing a patient for anesthesia, ask about related illnesses such as ischemic heart disease, renal failure and cerebrovascular disease. Those patients with isolated "white coat" hypertension have not been shown to be at higher risk from anesthesia than controls and therefore surgery should not be delayed unnecessarily. Elderly patients with systolic blood pressures below 180/190 mm Hg should also be considered for surgery, particularly if there is little evidence of end organ damage, as these values are considered in the normal range for elderly patients due to normal physiological change.

Which are the target organs for hypertension and what are the signs of target organ damage?

Cardiac

1. Left ventricular hypertrophy (in moderate to severe hypertension)
2. Systolic or diastolic cardiac dysfunction
3. Symptomatic heart failure
4. Ischemic heart disease—angina pectoris, myocardial infarction.

Vascular

1. Peripheral arterial disease
2. Carotid arterial disease
3. Aortic aneurysm
4. Wide pulse pressure (>65 mm Hg).

Renal

1. Albuminuria (urinary albumin excretion >300 mg/d)
2. Chronic kidney disease (estimated GFR <60 mL/min) or ESRD.

Cerebrovascular

1. Stroke
2. Transient ischemic attack
3. Decreased cognitive function
4. Dementia
5. Loss of vision.

Why is hypertension important?

1. It is common and incidence in our country is increasing with changing lifestyle.
2. It is a risk factor for cardiovascular disease.
3. It causes end organ damage which affects perioperative course—heart, brain, kidneys.
4. It may indicate the presence of serious endocrine related disease—diabetes, thyrotoxicosis, pheochromocytoma, Cushing's, Conn's syndrome, etc. (secondary hypertension).
5. It may indicate the presence of serious renal disease (secondary hypertension).
6. It increases the risk of an adverse anesthetic outcome/prolonged recovery room stay.
7. Perioperatively it may be due to:
   a. Anxiety
   b. Inadequate anesthesia (awareness)
   c. Inadequate analgesia (pain).

What are the causes for secondary hypertension?

1. Chronic kidney disease
2. Coarctation of the aorta
3. Cushing's syndrome and other glucocorticoid excess states including chronic steroid therapy
4. Drug induced or drug related
5. Pheochromocytoma
6. Primary aldosteronism and other mineralocorticoid excess states
7. Renovascular hypertension
8. Sleep apnea
9. Thyroid or parathyroid disease.

Which drugs can cause hypertension?

1. Sympathomimetic drugs like amphetamine, cocaine, migraine medications
2. Corticosteroids
3. Cyclosporine
4. Estrogen and oral contraceptive pills.

Explain the pathophysiology of essential hypertension.

Blood pressure is typically defined as the lateral pressure exerted by blood on the vessel wall, mainly in the arteries. Since mean arterial pressure (MAP) is product of the cardiac output (CO) and systemic vascular resistance (SVR), hypertension can result from either in increase in CO or increased SVR. Of all hypertensive patients, 90% have essential or primary hypertension where underlying pathophysiology is increased SVR. 70–80% patients with primary hypertension have family history of hypertension suggesting strong genetic influence. Since the autonomic nervous system (ANS), plays a central role in maintenance of cardiovascular homeostasis, disturbance of ANS, like sympathetic nervous system over activity, increases blood pressure and contributes to the development and maintenance of hypertension. The increased sympathetic nervous system activity in hypertension is multifactorial and complex and involves alterations in baroreflex and chemoreflex pathways at both peripheral and central levels. Arterial baroreceptors as well as aortic baroreceptors are reset to a higher pressure in hypertensive patients. Angiotensin II, endothelin and reactive oxygen radicals have been found to play major role in resetting baroreceptors.

Peripheral Vasculature: The arterial and arteriolar walls are thickened with resultant high ratio of wall thickness to internal diameter. There is relative hypovolemia due to which vascular relaxation following induction of anesthesia leads to a greater than expected decrease in blood pressure. Similarly vascular contraction leads to an abnormally large increase in blood pressure.

Heart: In order to cope with the increased afterload, the myocardium hypertrophies. This leads to increased wall tension , increased myocardial O_2 requirement, and endomyocardial fibrosis. Cardiac dilatation eventually follows, and subsequent heart failure. ECG shows left ventricular hypertrophy with strain.

Myocardial compliance is decreased with resultant increase in the atrial contribution to diastolic filling and cardiac output. Patients may develop congestive cardiac failure and pulmonary edema. The risk for perioperative myocardial ischemia is increased in hypertensive patients with left ventricular hypertrophy.

Brain: Chronic hypertension shifts cerebral and renal autoregulation to the right resulting in decrease in cerebral blood flow at higher blood pressures than in normotensive patients. With long-term therapy, the autoregulation curve shifts to normal. In patients with mild and moderate hypertension autoregulation returns to normal within 1–2 weeks of treatment. However, in severely hypertensive patients this period may be longer. Remember that hypertensive patients may also have cerebrovascular and carotid arterial disease and in "Watershed" areas ischemia may occur with even relative hypotension.

Kidneys: End organ damage to the kidneys from hypertension can lead to glomerular sclerosis, abnormal distribution of renal blood flow and decreased GFR. "Prerenal" hypoperfusion due to a sudden and sustained decrease in blood pressure (during anaesthesia for major surgery) can lead to postoperative renal insufficiency.

Discuss the relevance of commonly used antihypertensive drugs to the anesthetist. How will you control hypertension in individual patients?

Table 3.2 Antihypertensive drugs and anesthesia relevance

Mechanism of action	Examples	Relevance to anesthesia
Diuretics	Thiazide, frusemide, spironolactone etc.	Electrolyte disturbance
Calcium channel blockers	Nifedipine, verapamil	May cause precipitous hypotension
ACE inhibitors	Captopril, enalapril, ramipril, etc.	Can lead to severe catecholamine resistant hypotension under anesthesia
Beta blockers	Propranolol, metaprolol, atenolol, labetalol	Avoid in asthmatics and patients with overt CHF; peripheral vascular disease; bradyarrhythmias; failure to mount tachycardia which should occur with major blood/fluid loss; rebound hypertension and myocardial ischemia if withdrawn abruptly
Angiotensin receptor blockers	Losartan, valsartan, telmisartan	Same as ACE inhibitors
Vasodilators	Hydralazine,diazoxide	Tachycardia
Centrally acting	Clonidine, methyldopa, reserpine	Rebound hypertension if withdrawn
Alpha blockers	Prazosin Phenoxybenzamine, Phentolamine (not used recently)	Tachycardia

Therapy should begin with lifestyle modification, and if blood pressure goal is not achieved, thiazide-type diuretics should be used as initial therapy, either alone or in combination with one of the other classes (ACE inhibitors, angiotensin receptor blockers, β-blockers, and calcium channel Blockers) that have also been shown to reduce one or more hypertensive complications in randomized controlled trials. The choice of drugs also depends on presence of comorbidity. For example in a patient with coronary artery disease, a β-blocker would be the drug of choice where as in patients with diabetes or renal dysfunction an ACE inhibitor would be a better choice. If the initial drug selected is not tolerated or is contraindicated, then a drug from one of the other classes proven to reduce cardiovascular events should be substituted. Since most hypertensive patients will require two or more antihypertensive medications to achieve their blood pressure goals, addition of a second drug from a different class should be initiated when use of a single agent in adequate doses fails to achieve the goal. Use of more than one drug increases the likelihood of achieving blood pressure goal. The use of multidrug combinations helps in better BP control at lower doses of the component agents, with fewer side effects.

What is the blood pressure response to perioperative procedure in a hypertensive patient?

In a truly hypertensive patient blood pressure response can be considered in two phases—during anesthesia and in the postoperative period. Generally these patients display fall in BP during induction, followed by pressor response and tachycardia during laryngoscopy and intubation, and stimulation during surgery as well as during extubation. Sympathetic activation during the tracheal intubation also occurs in normotensive individuals. However these responses are more pronounced in patients with untreated and inadequately treated hypertensive individuals. These excessive swings in blood pressure and heart rate can increase the risk of ischemia or cardiac failure for patients with CAD or LV dysfunction. Hypertensive patients are known to have increased incidence of ST segment depression during surgery. In addition chronic uncontrolled or poorly controlled hypertension resets cerebral autoregulation at a higher level as mentioned above, so that in these patients, even a moderate fall in blood pressure in perioperative period may lead to cerebral ischemia.

How would you anesthetise this patient?

Premedication: The patient should receive an anxiolytic for premedication. Premedication with benzodiazepines (e.g. diazepam 5–10 mg, or alprazolam 0.25–0.5 mg) on night before and 2 hours prior to surgery will help in allaying anxiety and the associated rise in BP. If the patient shows significant hypertension in the preinduction area a small dose of intravenous sedation (midazolam 1–3 mg) may help to control it. Premedication with α 2-adrenoceptor agonists may be useful. Clonidine provides hemodynamic stability and reduces the risk of myocardial ischemia by reducing sympathoadrenal activity. In addition, clonidine also causes anxiolysis, sedation and decreases the need for both inhalation and intravenous anesthetics. Atropine should be avoided if possible, because of its tendency to cause tachycardia. Anesthetic management should be directed towards avoidance of both hypotension and hypertension.

Induction: Care should be taken not to cause a precipitous fall in arterial pressure at induction. The use of opioids, such as morphine or fentanyl, in liberal doses will attenuate response to laryngoscopy and intubation and also reduce the amount of induction agent required. Etomidate is most cardiostable but thiopentone may be used provided it is given slowly, and titrated against response. Propofol may cause precipitous hypotension and ketamine, which raises the arterial pressure and heart rate, should be avoided. Perform smooth, gentle and fast (<15 secs) laryngoscopy and intubation to attenuate sympathetic response. Spraying of vocal cords is found to be ineffective in preventing this response and no longer recommended. Use of IV lignocaine in the dose of 1.5 mg/kg has been widely practiced for the same purpose, but various studies have shown it to be ineffective or less effective as compared to drugs like beta blockers, vasodilators, calcium channel blockers and high dose opioids. Atracurium, rocuronium and vecuronium are suitable muscle relaxants. Severe hypotension after induction may be more harmful than hypertension and hence one should keep vasopressors ready and treat hypotension promptly. All patients should have continuous monitoring of the ECG and oxygen saturation. Intraoperative blood pressure monitoring may be non-invasive or invasive depending on type of surgery, expected fluid shifts and presence of comorbidity.

Maintenance: High concentrations of volatile agents can cause hypotension by decreasing the systemic vascular resistance and by depressing the myocardium. Liberal use of opiates, which have minimal cardiovascular effects, will reduce the amount of volatile agents required. Nitrous oxide can be safely used. Local anesthetic nerve blocks or infiltration are useful either on their own or in addition to general anesthesia. Hypertension during anesthesia may reflect inadequate depth of anesthesia, which should be corrected before treating blood pressure with antihypertensive drugs. Avoid perioperative hypothermia. Hypotension should be

vigorously treated by reducing the concentration of volatile anesthetic agent (not too low otherwise patient might develop awareness), and correcting any hypovolemia as hypertensives tolerate hypovolemia poorly. The reduced left ventricular compliance, and more rigid vascular tree found in hypertensive patients make them more vulnerable to small changes in blood volume. Furthermore beta blockers prevent the physiological heart rate changes whereas vasodilators prevent vasoconstrictive response. Thus careful monitoring of blood and fluid losses is important and prompt replacement of fluid deficit is necessary to avoid undue hypotension. Bradycardia should be treated using titrated doses of IV atropine. Patients on beta blockers are particularly liable to develop bradycardia, and may develop marked cardiac depression with anesthetic agents. Occasionally, a small dose of a vasopressor may be required in patients not responding to this approach. Normal intravenous fluid replacement should be given.

Recovery: Coughing on the tracheal tube during emergence will increase arterial pressure. Opioids given during anesthesia reduce this tendency. These patients may be extubated in deeper planes if airway is safe.

Role of regional anesthesia: Spinal and epidural anesthesia can cause unpredictable and profound arterial hypotension in poorly controlled hypertensive patients. Well controlled hypertensives, however, have a more predictable response. Peripheral nerve blocks, e.g. brachial plexus blocks or ankle blocks should always be considered in hypertensive patients, as the potential hazards of general anesthesia are avoided.
During emergency surgery aim for intraoperative blood pressure around 140/90 mm Hg but avoid swings > ± 20% from baseline blood pressure. Urine output should be closely monitored.

What are the causes of intraoperative hypertension? How would you manage it?

Hypertension occurs commonly during anesthesia. Main reasons are—light plane of anesthesia, preexisting hypertension, surgical factors (direct sympathetic stimulation), hypercarbia, airway problems (malfunctioning valves, hypoventilation), endobronchial intubation, infiltration with vasopressors, drug errors (inadvertent injection of vasopressors), fluid overload, awareness or uncommon conditions (pheochromocytoma, hyperthyroidism, malignant hyperthermia) and raised intracranial pressure.

Management: First confirm that blood pressure change is real (check BP cuff/transducer level in case of invasive BP), check for ventilation malfunction, ensure adequate alveolar minute ventilation, give adequate analgesia, deepen plane of anesthesia, inform surgeons and interrupt surgery if necessary, recheck for drug errors. Only after these steps have been taken consider using antihypertensive drugs. One can use nitroglycerine 20 mg diluted in 20–50 mL 5% dextrose or normal saline and at 0.1 ml/kg/h dose through syringe pump and titrate as per response. If tachycardia is troublesome use beta blockers like esmolol or metaprolol. Intravenous infusion of labetalol at rate of 0.5 mg/kg/h and titrated to response is another option. Labetalol being an alpha and beta blocker does not give rise to tachycardia as seen with nitroglycerine or sodium nitroprusside.

What are the perioperative issues in patients on antihypertensive drugs?

Most antihypertensive agents will usually be continued prior to anesthesia, but cessation should be considered if marked blood loss is anticipated or regional techniques such as epidurals are carried out, as the cumulative hypotensive effects may be detrimental. Renal function should be carefully monitored in those patients taking ACE inhibitors, particularly in the perioperative period. Non-steroidal anti-inflammatory drugs should be used with caution in patients taking ACE inhibitors. Some authors recommend stopping ACEI and ARBs and substituting other antihypertensive agent one day prior to surgery to prevent precipitous fall in blood pressure and catecholamine resistant hypotension. Beta blockade has been previously shown to improve perioperative morbidity and mortality in patients with ischemic heart disease; however the POISE study showed increased incidence of stroke and higher overall mortality. Beta blockers are better avoided in patients with reactive airway disease. Also particular caution should be exercised in patients on beta blockers as these drugs mask manifestation of tachycardia which may be due to pain, reduced cardiac output, awareness, anaphylaxis or even malignant hypertension during anesthesia.

Enumerate problems of anesthetizing a patient with uncontrolled hypertension.

- Cardiac ischemic events (especially in presence of LVH)
- Arrhythmias
- Cerebral autoregulation reset (shift to right) making patients prone for CVA
- Exaggerated cardiovascular response to intubation and extubation
- Presence of diastolic dysfunction (poor left ventricular relaxation) make patients prone for pulmonary edema
- Excessive surgical bleeding

A 52-year-old man presents to surgery for inguinal hernia repair. In preoperative check you find his BP 200/120 mm Hg. What would you do?

Recheck the BP after assuring the patient and adequate period of rest to rule out white coat effect. Check that BP cuff size is adequate for the patient. Ask a detailed history whether he has pre-existing hypertension and look for symptoms of any end-organ damage.

On rechecking, his BP is the same. The patient does not give history of hypertension, but complains of headaches, decreased effort tolerance and easy fatigability since 6–8 months. What will you do next?

We should not go ahead with the planned surgery and will have to wait till his blood pressure is under control, and assess the patient further for end-organ damage. Explain to the patient about the need to control blood pressure, implications for anesthesia as well as the long-term risk to life in case of uncontrolled hypertension. The patient should undergo investigations to rule out presence of secondary hypertension.

A recent American College of Cardiology/American Heart Association guideline states that uncontrolled systemic hypertension is a minor clinical predictor of increased perioperative cardiovascular risk. If the initial evaluation establishes hypertension as mild or moderate, and there are no associated metabolic or cardiovascular abnormalities, there is no evidence suggesting beneficial effects of delaying surgery. However for Stage 3 (severe) hypertension (>180/>110 mm Hg) the potential benefits of delaying surgery to optimize the effects of antihypertensive medications should be weighed against the risk of delaying the surgical procedure. Control can be achieved over several days to weeks of preoperative outpatient treatment. Amongst the agents available, beta-blockers appear to be particularly attractive. The guideline emphasizes that continuation of preoperative antihypertensive treatment throughout the perioperative period is critical.

How soon the patient can undergo surgery?

There are no definite recommendations about how long you need to have patients numbers normalized before subjecting them to anesthesia. One has to keep in mind that the numbers may normalize quickly but several weeks of treatment is necessary to reduce abnormal vascular tone and autoregulatory threshold to get resettled.

You are called to recovery to review a 77 years old patient who has undergone laparoscopic cholecystectomy. His blood pressure is 210/110 mm Hg. How will you manage this patient?

The common causes of postoperative hypertension are:

- Pain
- Emergence phenomenon
- Hypercarbia
- Intolerance of endotracheal tube
- Full bladder
- Hypervolemia
- Peripheral vasoconstriction due to hypothermia and hypovolemia
- Withdrawal of chronic therapy

Postoperative hypertension can lead to:

- Damage to vascular anastomoses
- Postoperative bleeding
- Intracranial bleeding
- Myocardial ischemia (particularly when associated with tachycardia)

Treatment of postoperative hypertension depends on the clinical situation, etiology and the degree of hypertension.

Analgesia must be optimized. Hypertension, where worrisome, should be treated with nitroglycerine, labetolol, esmolol, nitroprusside, hydralazine, etc. Sublingual nifedipine should be avoided as it can lead to uncontrolled and precipitous fall in BP.

This patient has severe hypertension. Rule out above-mentioned causes. Repeat blood pressure measurements regularly in addition to other routine monitoring. If hypertension persists, treat with labetolol or nitroglycerine.

What is malignant hypertension or hypertensive crisis? How will you manage it?

Malignant hypertension is a syndrome associated with an abrupt increase of blood pressure in a patient with underlying hypertension or related to the sudden onset of hypertension in a previously normotensive individual. It is accompanied by an encephalopathy or nephropathy. It is best described as a hypertensive emergency. The absolute level of blood pressure is not as important as its rate of rise. Common causes of hypertensive crises are:

- Antihypertensive drug withdrawal (e.g. clonidine)
- Autonomic hyperactivity
- Drugs like cocaine, amphetamines
- Acute glomerulonephritis
- Head trauma
- Pheochromocytoma
- Preeclampsia and eclampsia
- Renovascular hypertension.

Pathophysiology

- Abrupt increases in systemic vascular resistance due to humoral vasoconstrictors
- Endothelial injury
- Fibrinoid necrosis of the arterioles
- Deposition of platelets and fibrin

- Breakdown of the normal autoregulatory function
- Ischemia causing release of vasoactive substances leading to a vicious cycle.

Clinical Manifestations

The manifestations of hypertensive crises are those of end-organ dysfunction:

- Hypertensive encephalopathy
- Acute aortic dissection
- Acute myocardial infarction
- Acute cerebral vascular accident
- Acute hypertensive renal injury
- Acute congestive heart failure.

It is important to recognize that the absolute value of blood pressure may not be as important as the rate of increase in blood pressure. Patients with longstanding hypertension may tolerate systolic blood pressure of 200 mm Hg or diastolic blood pressure of up to 150 mm Hg without developing hypertensive encephalopathy, while children or pregnant women may develop encephalopathy with diastolic blood pressure 100 mm Hg.

Hypertensive Encephalopathy

- Headache
- Altered level of consciousness and confusion
- Acute CVA with intraventricular bleeding or ischemic infarction resulting in focal neurological abnormalities
- Fundoscopy showing advanced retinopathy with arteriolar changes, hemorrhages, exudates, papilledema.

Cardiovascular manifestations:

- Angina
- LVF
- Acute myocardial infarction.
- Aortic dissection: Propagation of the dissection is dependent not only on the elevation of the blood pressure itself, but also on the velocity of left ventricular ejection.

Renal Manifestations: Renal failure with oliguria and/or hematuria.

Treatment: Intravenous sodium nitroprusside, labetolol, esmolol, nitroglycerine can be used for the treatment of malignant hypertension. Their doses, duration of action, adverse effects, etc. are given below.

Drugs for treatment of hypertensive emergencies

In patients with malignant hypertension without encephalopathy or another catastrophic event, it is preferable to reduce

Table 3.3 Drugs for hypertensive emergencies

Drug	Dose	Onset of Action	Duration of Action	Adverse Effects	Special Indications
Vasodilators					
Sodium nitroprusside	0.25–10 µg/kg/min as IV infusion	Immediate	1–2 minutes	Nausea, vomiting, muscle twitching, sweating, thiocynate and cyanide intoxication	Most hypertensive emergencies; caution with high intracranial pressure or azotemia
Nitroglycerin	5–100 µg/min as IV infusion	2–5 minutes	5–10 minutes	Headache, vomiting methemoglobinemia, tolerance with prolonged use	Coronary ischemia
Fenoldopam	0.1–0.3 µg/kg per min IV infusion	<5 minutes	30 minutes	Tachycardia, headache nausea, flushing	Most hypertensive emergencies; caution with glaucoma
Nicardipine hydrochloride	5–15 mg/h IV	5–10 minutes	15–30 min may exceed 4 hours	Tachycardia, headache flushing, local phlebitis	Most hypertensive emergencies except acute heart failure; caution with coronary ischemia
Hydralazine hydrochloride	10–20 mg IV 10–40 mg IM	10–20 minutes IV 20–30 minutes IM	1–4 hours IV 4–6 hours IM	Tachycardia, flushing, headache, vomiting, aggravation of angina	Eclampsia
Adrenergic Inhibitors					
Labetalol hydrochloride	20–80 mg IV bolus every 10 min 0.5–2.0 mg/min IV infusion	5–10 minutes	3–6 hours	Vomiting, scalp tingling, bronchoconstriction, dizziness, nausea, heart block, orthostatic hypotension	Most hypertensive emergencies except acute heart failure
Esmolol hydrochloride	250–500 µg/kg/min IV bolus, then 50–100 µg/kg/min by infusion; may repeat bolus after 5 min or increase infusion to 300 µg/min	1–2 minutes	10–30 minutes	Hypotension, nausea, asthma, first degree heart block, heart failure	Aortic dissection, perioperative
Phentolamine	5–15 mg IV bolus	1–2 minutes	10–30 minutes	Tachycardia, flushing, headache	Catecholamine excess as in pheochromocytoma

blood pressure over hours or longer rather than minutes. This goal may effectively be achieved initially with frequent dosing of short-acting oral agents such as captopril, clonidine, and labetalol.

How would you manage patients with uncontrolled hypertension for surgery?

Uncontrolled hypertension is associated with wider fluctuations of blood pressure during induction of anesthesia and intubation, and may increase the risk for perioperative ischemic events. Blood pressure levels of >180/110 mm Hg should be controlled prior to surgery. In urgent situations, rapidly acting parenteral agents, such as sodium nitroprusside, and labetalol, can be utilized to achieve effective control very rapidly. Care should be taken not to cause precipitous fall. Most of the times; these patients have occult hypovolemia and would need proper fluid management. Continuous beat to beat monitoring of BP is advisable while using these drugs. Candidates with controlled hypertension should maintain their medications until the time of surgery, and therapy should be reinstated as soon as possible postoperatively. Since most hypertensive patients are on diuretic drugs, hypokalemia may be present and should be corrected prior to anesthesia. Older patients may benefit from treatment with selective beta-1 blockers before and during the perioperative period. Sudden intraoperative hypertension is managed by same antihypertensive agents that are utilized in the management of hypertensive emergencies. Nitroglycerin is often an agent of choice in patients with coronary ischemia, while the very short-acting β-blockers like esmolol, may be of benefit in managing intraoperative tachycardia and hypertension. Hypertension is very common in the early postoperative period and is related to increased sympathetic tone and vascular resistance. Contributing factors include pain and increased intravascular volume, which may require parenteral dosing with a loop diuretic such as furosemide. If resumption of oral treatment must be interrupted postoperatively, periodic dosing with intravenous antihypertensive agents should be considered.

Suggested Reading

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4 Tetralogy of Fallot

V Agarwal, R Ambulkar, M Desai

An 8-month-old boy is admitted with history of mild cyanosis since 2 weeks after birth and gradually increasing over the last 3 months and worsening on crying. He was diagnosed to have Tetralogy of Fallot (ToF) on cardiac catheterization.

How is a pediatric cardiac surgery patient different from an adult cardiac surgery patient?

- The cardiovascular system in a neonate and infant has reduced myocardial reserve. This is due to the following reasons:
  - Limited or restricted number of beta receptors
  - High resting levels of circulating catecholamines
  - An immature calcium transport system. They depend on the extracellular calcium levels in contrast to the adult myocardium
  - Reduced ventricular compliance
  - The stroke volume does not increase with preload augmentation. As a result they are more dependent on the heart rate rather than the stroke volume to increase the cardiac output.
- Pediatric patients are physically smaller than adults. To establish invasive monitoring is challenging in these small patients. Sometimes it may not be possible to use conventional monitoring such as pulmonary artery catheter (in patients < 7kg 5.0 Fr pulmonary artery catheter may have to be inserted through the femoral vein rather than internal jugular vein and may require fluoroscopic guidance). One may have to rely on transthoracic catheters inserted during the time of surgery as well as specialized monitoring such as transesophageal or epicardial echocardiography with Doppler color flow imaging.
- During cardiopulmonary bypass (CPB), these patients are cooled to 15° to 18°C; hemodilution occurs to more than 50% of their extracellular volume. They may undergo periods (more than 1 hour) of deep hypothermic cardiac arrest. They have exaggerated inflammatory response to CPB due to the disproportionate exposure to the non-endothelialized surfaces of the CPB circuit per body surface area compared to adults.

What is Tetralogy of Fallot (TOF)?

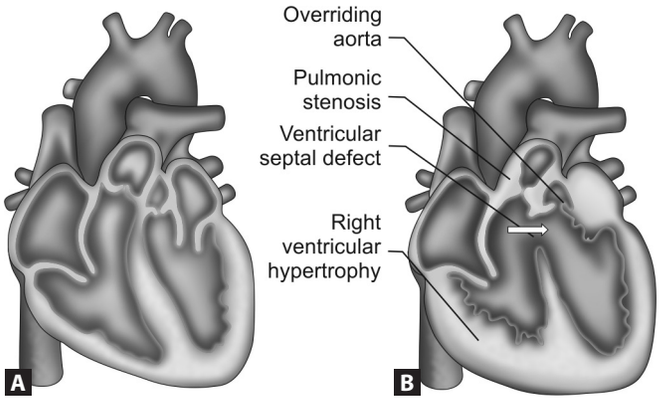
It is a tetrad of anatomical malformations resulting from a single morphologic defect, i.e. mal-alignment of the anterior part of the infundibular septum. The four components are:

- Right ventricular outflow tract obstruction (RVOTO)
- Ventricular septal defect (VSD)
- An overriding aorta
- Right ventricular hypertrophy.

The RVOTO may be infundibular spasm, pulmonary stenosis or pulmonary atresia. ToF with pulmonary atresia is much more severe form of congenital heart disease and cyanosis develops almost immediately after birth and worsens progressively as the ductus arteriosus closes. These patients depend on the patent ductus arteriosus until definitive corrective surgery is done. Prostaglandins are added to maintain the patency of ductus arteriosus. ToF with pulmonary atresia may be associated with other syndromes such as acronyms:

- CATCH22 (cardiac defect, abnormal face, thymic hypoplasia, cleft palate, hypocalcemia, associated with micro deletion of band 22q11).
- VATER syndrome include the vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, and renal and radial anomalies.

- CHARGE syndrome includes the coloboma, heart disease, atresia choanae, retarded growth and retarded development and/or CNS anomalies, genital hypoplasia, and ear anomalies and/or deafness.



Figs 4.1A and B (A) Normal heart; (B) Tetralogy of Fallot

The diagnosis can be done prenatally with fetal ultrasound. In acyanotic patients, a crescendo-decrescendo systolic murmur; best heard at the upper left sternal border; is audible at birth. They may be asymptomatic and later on develop signs and symptoms of congestive heart failure secondary to large left to right shunt. Most patients become cyanotic after birth. Subsequently echocardiography can be done to confirm the diagnosis.

The nature of the RVOT dictates the onset and severity of symptoms. If untreated the infant progressively becomes cyanotic because of worsening infundibular stenosis, develops polycythemia in an attempt to increase oxygen delivery to the tissues. Hypoxic spells may occur in infants with agitation and crying. If cyanosis is severe it can lead to hypotension, hemodynamic instability and finally loss of consciousness. Persistent cyanosis may cause growth retardation. Polycythemia predisposes to brain abscess, stroke.

ToF is the most common cyanotic heart defect seen in children, if treated results in survival to adulthood. Also, it is the most common complex lesion encountered after repair in adult population. If untreated 30% of the infants with ToF and pulmonary stenosis die within the first year, 40% by 4 years, 70% by 10 years, and 95% by 40 years.

What is the pathophysiology of TOF?

There are two pathophysiologic problems:

1. Reduced pulmonary blood flow results in systemic hypoxemia and cyanosis.
2. Increased impedance to RV ejection results in pressure overload, ventricular dysfunction and failure of the RV.

The VSD leads to an increase in blood flow and pressure in the right ventricle causing right ventricular hypertrophy. The severity of the shunt increases with the severity of RVOTO. Chronic hypoxia occurs because of shunting of blood. Exacerbations of hypoxia following infundibular spasm may be life threatening. Chronic hypoxia leads to polycythemia which predisposes to hypercoagulability and thrombosis. Since right-to-left shunting of blood bypasses the filtering effect of the pulmonary capillaries these patients have a higher incidence of systemic infection such as a brain abscess.

What are the clinical features?

- Cyanosis may be present immediately or shortly after birth and increases progressively thereafter as right ventricular hypertrophy develops increasing the shunt fraction and worsening systemic hypoxemia.
- The neonate or infant may present with cyanosis during feeding, fatigue during feeding, irritability and tachypnea. Fatigue during feeding and tachypnea decreases the time for swallowing leading to inadequate feed intake and therefore failure to thrive.
- Dyspnea on exertion such as following crying, feeding in an infant or during playing or running in an older child.
- Hypoxic spells. The parent may mention that the child squats when he/she becomes blue.
- Physical growth retardation.

What are the compensatory mechanisms in patients with TOF?

- RV hypertrophies to work against narrow RVOT.
- Polycythemia in face of chronic hypoxia.
- Squatting may be considered a compensatory mechanism by the child to attenuate cyanotic spells

What is a pink tet?

The term pink tet refers to TOF with minimal valvular or subvalvular stenosis. Blood flow to the pulmonary circulation is sufficient to maintain the deoxygenated hemoglobin concentration less than 5 g%. As a result these patients are acyanotic. This term is also applied to patients with ToF with pulmonary atresia or severe pulmonary stenosis with patent ductus arteriosus and those with major aortopulmonary collateral arteries. An acyanotic patient with TOF (pink tet) has a long, loud, crescendo-decrescendo systolic murmur; best heard at the upper left sternal border. The loud murmur is a reflection of mild RVOT. Infants with acyanotic ToF (pink ToF) may be asymptomatic or may show signs of congestive heart failure (CHF) from a large left to right shunt. In acyanotic patients with CHF, medical management is similar to that of a patient with a ventricular septal defect (VSD) and includes

diuretics (furosemide), digoxin and afterload reduction with angiotensin converting enzyme inhibitors (captopril).

What is a hypoxic/hypercyanotic/tet spell?

"Tet spell" is paroxysmal exacerbation of hypoxemia usually preceded by hyperpnea. The exact etiology is not fully understood; it occurs probably due to infundibular spasm aggravating the RVOTO which leads to worsening of right to left shunt causing acute severe cyanosis and may lead to loss of consciousness. Hypoxemia further increases the right to left shunt by decreasing the SVR.

It is characterized by paroxysms of hyperpnea, prolonged crying, intense cyanosis, and decreased intensity of the murmur of pulmonic stenosis as the blood is flowing past the VSD from right to left ventricle. It is precipitated by activity or distress (crying, defecating, agitation or fright) and squatting relieves the spell. Squatting causes compression of the femoral vessels thus increasing the systemic vascular resistance (SVR) which reduces shunt from right to left across the VSD and therefore reduces cyanosis. Other factors which can worsen cyanosis are anemia, acidosis, infection and stress.

"Tet spells" can also be precipitated by an acute reduction in SVR, such as during induction of anesthesia (especially if the patient is hypovolemic). The anesthetist should be prepared to manage a hypoxic spell.

How do you manage tet spell?

The main aim of management is to increase the SVR and decrease the PVR.

- Knee-chest position. It causes compression of the femoral vessels increasing the SVR and thus decreasing the right to left shunt. Place the baby on the mother's shoulder with the knees tucked up underneath. This also provides a calming effect.
- Oxygen is of limited value since the primary abnormality is reduced pulmonary blood flow and increased right to left shunt. Administer oxygen.
- Morphine sulfate, 0.1–0.2 mg/kg IM/SC, can be given to reduce respiratory drive and hyperpnea.
- Phenylephrine, 5–10 μg/kg IV or as an infusion 2-5 μg/kg/min can be used to increase SVR.
- Dexmedetomidine infusion, 0.2 μg/kg/min, has recently been shown to ameliorate symptoms during the hypoxic spell.
- Treating metabolic acidosis with sodium bicarbonate (1–2 mEq/kg, IV) reduces the respiratory centre stimulating effect of acidosis and also decreases the SVR.
- Volume resuscitation either with crystalloids or colloids (15-30mL/kg) augments the right ventricular preload, which may increase the diameter of the heart and thus physically decrease the RVOTO.
- Beta-blockers can be administered to decrease infundibular spasm. They have a myocardial depressant effect and thus relieve the infundibular spasm. Also decrease in the heart rate improves the diastolic filling of the ventricle and thus decreases RVOTO. Esmolol 0.5 mg/kg bolus IV followed by an infusion of 50–300 μg/kg/min or propranolol 0.1mg/kg can be used.
- Intraoperatively, if a tet spell occurs, manual compression of the aorta can be done to increase the SVR.

What surgical procedures are available to correct this condition?

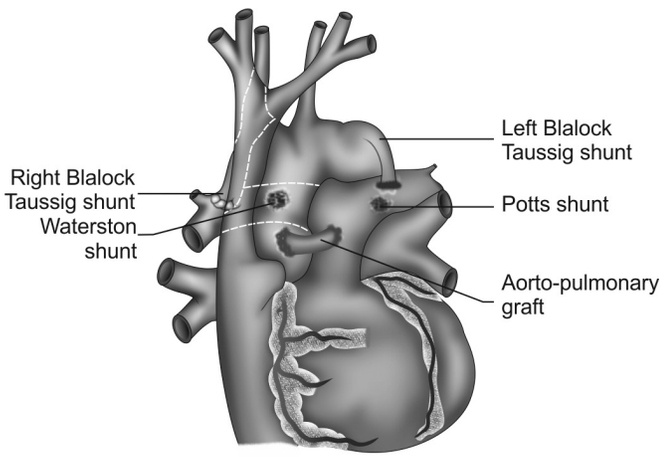


Fig. 4.2 Systemic to pulmonary shunts

Palliative surgery

The principle is to increase the pulmonary blood flow by creating a shunt between systemic and pulmonary circulation. Shunt surgery improves oxygenation and promotes pulmonary vascular growth but predisposes to right ventricular failure secondary to increased pulmonary blood flow. Various procedures have been described such as

- Blalock-Taussig shunt (BT shunt) is a direct end to end anastomosis of subclavian artery to pulmonary artery.
- Pott's procedure is anastomosis of descending aorta to pulmonary artery
- Waterston shunt is anastomosis of ascending aorta to pulmonary artery. Both the Pott's and Waterston shunts resulted in excessive pulmonary blood flow, distortion of the pulmonary artery, and problems during subsequent complete tetralogy of Fallot repair. Therefore they are no longer used.
- Modified BT shunt: Professor Marc deLeval modified the original BT shunt using an interposition conduit between

subclavian artery and pulmonary artery. Also known as the modified BT shunt, the deLeval shunt and the GOS (Great Ormond Street) shunt. Most commonly used systemic to pulmonary artery shunt.
- Balloon dilatation of the RVOT has been attempted.

Definitive Surgery

Earlier only the palliative aorto-pulmonary shunt operations were done because of high mortality in the very young patients. This was later followed by corrective surgery. However, with the development of deep hypothermic cardiac arrest, low flow hypothermic CPB, advances in anesthetic management of infants and children and their postoperative care, the morbidity and mortality has decreased dramatically after early one-stage repair. One stage repair is preferred as it avoids second surgery and eliminates complications of shunt operations. Systemic-to-pulmonary artery shunt is indicated in patients in whom the risk of complete repair is considered greater than the cumulative risk in 2-stage repair.

Timing of Surgery

- Most centres advocate correction surgery in asymptomatic patients between 4–6 months of age whereas some centers prefer to operate in the early neonatal period and others wait until 1 year of age. Symptomatic patients or those refractory to medical management require urgent surgery irrespective of the age.
- Repair at older age is associated with complications of both the palliative and definitive surgery and thus increases long-term morbidity.

Repair

Complete surgical repair involves closure of the VSD and relief of the RVOTO. A median sternotomy approach is used with cardiopulmonary bypass. There are two surgical approaches:
- Transventricular approach which involves right ventriculotomy at the infundibulum which allows exposure of the ventricular septal defect and patch closure.
- Transatrial approach through which the VSD and RVOTO can be approached. Muscle resection is performed to relieve the RVOTO.

Approximately 5% of the patients with ToF have abnormal coronary artery which may modify the surgical approach for repair. The most common is the origin of left anterior descending artery from the right coronary artery.

How will you evaluate these patients preoperatively?

In addition to the usual pediatric anesthetic evaluation, relevant information should be elicited from the parent as mentioned in the clinical features of TOF.

History: It is important to determine the following:
- Whether the child has normal or impaired functional status?
- Are they gaining weight appropriately? Look for signs of cardiac cachexia.
- Does the child have signs and symptoms of right heart failure (sweating, tachypnea, poor feeding and recurrent respiratory infections)?
- How is the cyanosis progressing? What is the frequency of cyanotic spells?
- These patients are particularly prone to recurrent respiratory tract infection after palliative shunt surgery secondary to increased pulmonary blood flow and altered lung compliance. Recent upper respiratory tract infection or pneumonia should be ruled out. If a patient suffers from upper respiratory tract infection, surgery may be deferred for a few days till symptoms improve. Failure to do so may result in increased airway reactivity and increase the likelihood of postoperative respiratory complications.
- Other associated congenital anomalies should be identified and relevant knowledge regarding the same and their anesthetic implications should be understood prior to conduct of anesthesia.
- Previous surgical interventions if any. Look at the anesthesia records for any problems encountered. If patient has undergone a BT shunt, then arterial line should not be placed on the same side as the information may not be accurate.
- What medications the patient is on at present?
- Lastly, family history of anesthetic difficulties.

Examination: cyanosis and clubbing may be present in infants older than 6 months. A loud murmur of pulmonary stenosis is audible over the pulmonary area.

Investigations

Chest x-ray: It may reveal a classic boot-shaped heart.

Echocardiography and Doppler: It is a non-invasive tool to assess intra-cardiac anatomy, blood flow patterns, and estimates of physiologic data.

Cardiac catheterization remains the gold standard for assessing anatomy and physiologic function in congenital heart disease. It is indicated before repair of ToF in patients with previous palliation and when aortopulmonary collaterals, coronary artery and pulmonary artery branching abnormalities are suspected. The anesthetist should have access to the following important catheterization data:
- Location, size, and direction (left to right or right to left) of intra-cardiac shunting

- Pulmonary and systemic arterial pressures
- Ventricular and atrial pressures with specific attention to left and right ventricular end-diastolic pressure
- Oxygen saturation data
- Cardiac chamber size
- Pulmonary vascular resistance
- Valve anatomy and function
- Anatomy, location, and function of previously created shunts
- Anatomic distortion of systemic or pulmonary arterial vessels, especially as it relates to previously placed shunts
- Coronary artery anatomy.

Full blood count: Polycythemia, raised white cell count may be suggestive of infection.

ECG: Right axis deviation secondary to right ventricular hypertrophy.

Arterial blood gas: Metabolic acidosis, hypoxemia and normal CO_2 tension.

What are the anesthetic considerations for this child undergoing modified BT shunt surgery?

Premedication: The goal of premedication is to achieve adequate sedation maintaining respiratory and hemodynamic stability allowing for easy separation from parents in the young and older children to reduce anxiety.

It can be administered orally or parenterally. Oral premedication is preferred and parenteral route is usually reserved for children who refuse or are not willing to take oral premedication. Midazolam 1 mg/kg orally can be given about 20 minutes prior to induction of anesthesia. Ketamine 5–10 mg/kg orally along with midazolam 1mg/kg is sufficient in children who have undergone previous surgical interventions as they get tolerant to midazolam. It can be administered intramuscularly in a dose of 2–3 mg/kg along with glycopyrrolate 10 μg/kg. Midazolam can be administered in a dose of 0.1mg/kg.

Operating Room Preparation: Meticulous attention should be paid to make sure that syringes and intravenous tubing is free from air bubbles to prevent paradoxical air embolism. Resuscitative drugs such as calcium gluconate, sodium bicarbonate, atropine, phenylephrine, lignocaine, and epinephrine should be labelled and ready for administration. An inotropic infusion, usually adrenaline or noradrenaline, should be ready for administration in high-risk cases. For all pediatric patients, certain anesthetic drugs such as thiopental, ketamine, succinylcholine, and atropine should be kept ready for use on an emergency basis because of the potential for airway reactivity, hypotension, and bradycardia during anesthetic induction.

Monitoring: It should be placed prior to induction of anesthesia, however, in a crying child these can be established soon after induction of anesthesia. Monitoring includes seven-lead ECG, non-invasive blood pressure, pulse oximetry, end tidal capnometry, invasive arterial and central venous pressure lines, temperature, an esophageal stethoscope and urine output. Arterial line should not be placed on the side of the shunt surgery. End tidal capnometry is useful in identifying a tet spell intraoperatively. As the pulmonary blood flow decreases so does the end tidal CO_2 and this is followed by arterial desaturation.

Conduct of anesthesia: The anesthetic concerns are to maintain the SVR and reduce PVR and prevent hypercyanotic episodes intraoperatively.

- Choice of induction depends on the availability of vascular access. If intravenous induction is chosen then Ketamine is favored as it increases SVR.
- Hypoxia, hypercapnea, and acidosis (both respiratory and metabolic) increase pulmonary vascular resistance and should be avoided. Patients should be ventilated in such a way that there is minimal increase in PVR. PVR is lowest at or near the functional residual capacity. PEEP may increase the PVR by increasing the alveolar pressure, however in patients with severe pulmonary stenosis, positive pressure ventilation and PEEP does not cause a clinically significant increase in the PVR.
- Nitrous oxide and inhalational agent such as halothane can be used. Halothane is preferred as it relieves infundibular spasm during hypercyanotic spell.
- Opioid based anesthesia ensures stable hemodynamics.
- Infective endocarditis prophylaxis should be given preoperatively.
- Euvolemia should be maintained to prevent dynamic RVOTO.
- As soon as the shunt is open, saturations improve dramatically, but diastolic blood pressure reduces drastically and this can precipitate myocardial infarction in an infant.
- Heparin infusion and aspirin are started to maintain shunt patency.
- The shunt flow may be assessed:
  - Oxygen saturation is around 80% then flow is good.
  - Oxygen saturation greater than 80%, it implies increased pulmonary blood flow and congestion and can lead to unilateral pulmonary edema on the side of the shunt.
  - Oxygen saturation less than 80%, implies poor pulmonary blood flow.

What are the anesthetic considerations for this child undergoing corrective surgery?

The anesthesia considerations are similar to that mentioned above; the only difference is that patients posted for corrective surgery require cardiopulmonary bypass with total body heparinization. Following complete repair with no residual or minimal intrapulmonary shunt the arterial saturation improves to 100%. Protamine should be administered as patient comes off bypass to reverse the effect of heparin. In patients with right ventricular dysfunction due to right ventricular hypertrophy or following ventriculotomy, surgeon may prefer to leave a residual defect at the atrial level to augment the cardiac output at the cost of arterial desaturation until right ventricular function improves. In such case the patient may have arterial saturation of around 80%. Inotropes may be required intraoperatively and in the postoperative period.

What are the anesthetic considerations in a patient with ToF for non-cardiac surgery?

Understanding the pathophysiology of the lesion, its hemodynamic effects and pharmacology of anesthetic drugs will enable the anesthetist to tailor an anesthetic for the child.

- Evaluation of the child's heart and lungs
  - Cardiac function: Child's activity- sweats when nursing, slow to finish bottle
  - Rule out CCF: Puffy eyes, enlarged liver (lungs may sound clear)
  - Effect of cyanotic spells: Passing out indicates very serious R to L shunt
  - Vd/Vt may be very high as the lungs are not well perfused and most of it may be in zone 1. This may be exaggerated by hypovolemia, high pressure during IPPV, PEEP and positioning the child.
- Appropriate tailoring of the anesthetic
  - Oral pre-med sedation; midazolam with ketamine is a good option.
  - Infective endocarditis prophylaxis
  - Promoting L to R shunt or reducing R to L shunt
  - Reducing pulmonary vascular resistance
  - Maintaining SVR and blood pressure
  - Maintaining contractility of the heart
  - Avoid hypoxia, hypercarbia, acidosis, hyperthermia, pain, inadequate depth of anesthesia
  - Maintain hydration especially if they are polycythemic
  - IV induction in the presence of R to L shunt may be faster whereas inhalation may be slower secondary to poor pulmonary flow and dilution from right side
  - Avoid air bubbles in syringes and infusions.

If pallitative shunt is in place

- Consider appropriate site for monitoring NIBP and IBP
- Antibiotic prophylaxis
- Maintain SVR to promote pulmonary blood flow.

Postoperative Pain Management

- For corrective or palliative surgery, the child is usually ventilated and so opioids can be used.
- For non-cardiac procedures, appropriate analgesia reduces stress on the child and hence reduces cyanotic spells.
- Regional blocks should be considered for analgesia.
- Paracetamol and NSAIDs if suitable.

Suggested Reading

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5 Patent Ductus Arteriosus

R Ambulkar, V Agarwal, M Joshi

You are asked to evaluate a neonate 22-day-old weighing 1.2 kg for surgery next week. The baby was born preterm at 32 weeks of gestation. She was diagnosed at birth to have a murmur. She is referred to your cardiothoracic center.

Enumerate the common Left to Right shunts.

The common Left to Right shunts:

- Atrial Septal Defect (ASD)
- Ventricular Septal Defect (VSD)
- Patent ductus arteriosus (PDA)

What are common complications associated with congenital heart disease?

- Infective endocarditis
- Cardiac arrhythmias
- Complete heart block
- Thromboembolism
- Polycythemia.

What is a Patent ductus arteriosus (PDA)?

Patent ductus arteriosus (PDA) is a congenital heart disease where there is a persistence of a vascular structure connecting the proximal descending aorta to the main pulmonary artery.

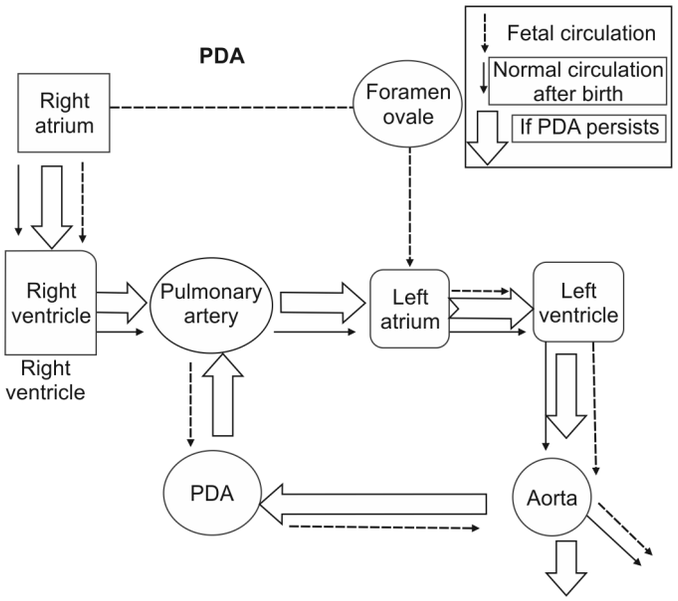

Fig. 5.1 PDA, foetal and normal circulation

Discuss normal fetal circulation.

Umbilical vein carries oxygenated blood and divides into two branches—portal sinus and ductus venosus. Ductus venosus bypasses the liver and enters the IVC; whereas the portal sinus enters the liver. The blood then enters the right atrium, where majority of the blood enters the left atrium through the foramen ovale. From the left atrium blood passes to the left ventricle and subsequently supplies brain and heart. Part of the blood which enters the right atrium mixes with blood from superior vena cava and enters the right ventricle. From the right ventricle blood enters the pulmonary artery, but because of high pulmonary vascular resistance blood is shunted to the aorta via the ductus arteriosus. Mixing of saturated and desaturated blood takes place and this supplies the lower body.

What changes occur in fetal circulation at birth?

When the newborn baby takes the first breath, there is a sharp drop in the intrapleural pressures which may be as low as 25 to 50 mm of Hg. This leads to lungs expansion and a dramatic drop in the pulmonary vascular resistance; with a substantial increase in the pulmonary blood flow. There is a drop of right atrial pressure below the left atrial pressure causing the functional closure of foramen ovale. Also the umbilical cord clamping increases the systemic vascular resistance (SVR) which maintains the left atrial pressure at a higher level (than RA). With the rise in PaO_2 from the low fetal levels; the flow through the ductus arteriosus decreases and eventually leads to closure.

Why is the size of PDA important?

The clinical significance of the PDA and the physiological impact caused by it depends largely on its size; as it decides the magnitude of the left to right shunt. The other factors which determine the flow through the ductus are the pressure gradient from aorta to pulmonary artery, pulmonary vascular resistance and systemic vascular resistance.

Discuss the embryological development of heart and the ductus arteriosus.

Embryology of heart: Cardiac formation is almost complete by 55–60 days post-conception but by age 2–3 years, cardiovascular system essentially is that of a fit adult.

- Cardiovascular system starts to develop at 3 weeks of gestation
- Beating of the heart starts soon after 3 weeks of gestation
- Chamber formation is as early as 5 weeks
- Valve formation is delayed till about 7 weeks.

Embryology of ductus arteriosus: The proximal portion of the sixth pair of embryonic aortic arch forms the proximal branch of pulmonary arteries, and the distal portion of the left sixth arch forms the ductus arteriosus, connecting the left pulmonary artery with the left dorsal aorta.

What are the causes and risk factors associated with PDA?

Prematurity: The increased incidence of PDA is due to developmental immaturity in premature babies.

Genetic Factors: The exact mechanism is unclear. However, it is believed to be multifactorial, with genetic predisposition and timely environmental trigger. In some patients may be autosomal recessive inheritance. If one sibling has PDA there is approximately 3% chance that the subsequent sibling will have PDA.

Rubella infection: There is a good association between rubella infection during the first trimester of pregnancy and increased incidence of PDA.

Teratogens: Drugs like amphetamine and phenytoin when consumed by mother in any trimester are associated with increased incidence of PDA.

Poorly controlled diabetes in mother also predisposes to birth of babies with PDA.

How would you define prematurity and what are the consequences of it?

When the gestational age is less than 37 weeks, the baby is termed as premature.

Moderately premature: 31–36 weeks of gestation.

Severely premature: < 31 weeks of gestation.

Prematurity is common and occurs in 5–10% of live births.
Significance: Due to immature development of organs, there is a higher incidence of mortality and morbidity in these babies and it is associated with the following complications:

- Respiratory failure
- Recurrent pneumonia
- Apnea spells
- Respiratory distress syndrome
- Hyaline membrane disease
- Bronchopulmonary dysplasia
- PDA
- Malnutrition
- Necrotising enterocolitis
- Poor GI motility and function
- Anemia

- Growth failure
- Metabolic disturbances
- More prone to infection
- Kernicterus
- More prone to hypothermia
- Fractures
- Failure to thrive
- Hypoxic ischemic encephalopathy
- Retinopathy.

What causes the closure of DA? When does it occur normally?

The patency of the ductus arteriosus during fetal life is due to low fetal oxygen tension and cyclooxygenase mediated products of arachidonic acid metabolism, primarily prostaglandin [PGE_2] and prostacyclin [PGI_2]. High levels of PGE_2 and PGI_2 are due to production by placenta and decreased metabolism by the fetal lungs. PGE_2 and PGI2 interact with ductal prostanoid receptors resulting in vasodilatation of ductus arteriosus. After birth, the abrupt increase in oxygen tension inhibits ductal smooth muscle potassium channels; resulting in calcium influx and ductal constriction. PGE_2 and PGI_2 levels also fall because of increased metabolism by the lungs and cessation of production by the placenta. The medial smooth muscle fibers in the ductus contract resulting in lumen obliteration of the ductus arteriosus. Functional closure of the ductus occurs within 24 to 48 hours of birth. Within 2 to 3 weeks fibrosis occurs leading to a permanent closure.

Discuss pathophysiologic consequences of left-to-right shunt.

The magnitude of shunt depends on the resistance to flow through the ductus. The elasticity of the ductus as well as the length and diameter would decide the resistance to the flow. The other important factor is the pressure gradient between the two vessels—the aorta and the pulmonary artery. The left-to-right shunting of blood through the ductus results in pulmonary circulatory overload and left heart volume overload. The increased pulmonary circulation results in decreased lung compliance and increased work of breathing. In older patients pulmonary edema may result. The volume overload of the left heart results in an increase in end diastolic pressures of both the atria as well as the ventricle. The left ventricle initially compensates by increase in stroke volume but left ventricle hypertrophies over time. There is also an accompanying increase in heart rate due to increased levels of catecholamines which is a part of neuroendocrine adaptation. All these factors may result in subendocardial ischemia owing to a mismatch in oxygen demand and supply.

What is Eisenmenger's Syndrome?

The pulmonary vasculature undergoes changes with time owing to high volume as well as pressure. There is a progressive increase in pulmonary vascular resistance due to hypertrophy and fibrosis in the vascular system resulting in luminal obliteration in pulmonary arterioles and capillaries. When the pulmonary vascular resistance exceeds systemic vascular resistance, shunting of blood reverses and changes from left-to-right to right-to-left. This is called Eisenmengerization and the syndrome is called Eisenmenger's syndrome or Eisenmenger's reaction.

What is the clinical presentation of a patient with PDA?

The child presents with the following and rarely the child may be asymptomatic:

- Poor growth
- Breathlessness
- Easy fatigability
- Tachycardia
- Frequent lung infections.

The physical examination reveals:

- Prominent or bounding peripheral pulse
- Systolic hypertension and low diastolic pressure due to increased left ventricular stroke volume and flow from aorta to pulmonary circulation.
- The first heart sound (S_1) is normal. The second heart sound (S_2) often is masked by the murmur.
- Continuous machinery murmur is best heard along the upper left sternal border, radiating down the left side of the sternum and into the back. A thrill may be present.
- Apical flow rumble: The pulmonary blood flow exceeds the systemic blood flow causing a left sided volume overload which leads to increased blood flow from the left atrium to left ventricle across the mitral valve causing a flow rumble.
- Cyanosis: Babies with Eisenmenger's syndrome present with differential cyanosis. In differential cyanosis there is cyanosis and clubbing of the toes but not the fingers; because the right-to-left ductal shunting is distal to the subclavian arteries.

How would you investigate such a child?

Chest Radiography

- In significant left-to-right shunt, cardiomegaly with left atrial and ventricular enlargement is present.
- With marked pulmonary over-circulation, signs of pulmonary edema are seen.

- In elderly individuals, calcification of PDA may be seen.
- Rarely may be normal.

Electrocardiogram

- Sinus tachycardia or atrial fibrillation.
- Small PDA—the ECG findings may be normal.
- Larger PDA—Left ventricular hypertrophy.
- In the neonate, especially the premature neonate with a large PDA, T-wave inversion and ST segment depression may be present suggesting ischemia or a supply-demand mismatch.

Two Dimensional and Doppler Echocardiography

- It helps to confirm the diagnosis.
- It is useful in describing the PDA as silent, small, moderate or large.

M-mode Echocardiography

- It is used to measure the cardiac chamber sizes and quantify left ventricular systolic function.
- Small PDA—chamber sizes are usually normal, although mild left atrial and/or left ventricular enlargement may be present.
- Moderate or large PDA, the left atrium and left ventricle are enlarged.

Cardiac Catheterization

- Therapeutic catheterization is the treatment of choice for PDA. It helps in complete diagnostic evaluation of hemodynamics of the patient as it is important to assess the pulmonary vascular resistance and the amount of shunting before trans-catheter closure.
- It also helps in assessing the response of vasodilating agents such as oxygen, nifedipine, prostacyclin, sildenafil, and nitric oxide in patients with raised pulmonary vascular resistance.

Enumerate the complications associated with PDA.

- Congestive heart failure: In moderate to large patent ductus arteriosus, congestive heart failure due to pulmonary over-circulation and left heart volume overload can occur.
- Atrial flutter or fibrillation: Heart failure is frequently associated with atrial flutter or fibrillation in adults.
- Hypertensive Pulmonary Vascular Disease: Secondary to long-standing pressure and volume overload in the pulmonary circulation.
- Infective Endarteritis: Vegetations usually occur in the pulmonary artery end of the ductus and embolic events are usually of the lung rather than the systemic circulation.
- Aneurysm of Ductus Arteriosus: Ductal aneurysm most commonly presents in infancy but has also been reported in adults and may develop after infective endarteritis, surgical closure, or trans-catheter coil occlusion.
- Rare complications: Dissection and/or spontaneous rupture of a dilated pulmonary artery in PDA associated with pulmonary hypertension.

What are the treatment options for PDA?

Medical Management consists of attempts to close the PDA and treatment of complications.

- Indomethacin: Intravenous indomethacin is effective in closing a PDA if it is administered in the first 10–14 days of life. Indomethacin is a potent inhibitor of prostaglandin-forming cyclooxygenase; hence there is a decrease in synthesis of prostaglandins which leads to ductal closure.
- Cautious fluid restriction
- Antiarrhythmia drugs: Treatment of atrial fibrillation or flutter.
- Vasodilators: Patients with PDA and pulmonary vascular disease who cannot undergo definitive closure are managed with pulmonary vasodilating agents such as PGI_2, calcium channel blockers, endothelin antagonists, and phosphodiesterase type V inhibitors (such as Sildenafil).
- Other drugs: Diuretics and digoxin.

Surgical management options are

Transcatheter Closure: Advance a catheter or delivery sheath across the ductus arteriosus from either the pulmonary artery or the aorta and position a closure device in the ductus to occlude it.

Success rate: Complete closure rates of more than 90% to 95% are seen with this method.

Complications of this procedure are:

- Bleeding at the catheterization site
- Rupture of blood vessels
- Tachyarrhythmias
- Bradyarrhythmias
- Vascular occlusion
- Inappropriate deployment of the device
- Migration of the device
- Incomplete closure of the ductus.

Surgical closure: Surgical ligation or division of the PDA remains the treatment of choice for the very large PDA. When the PDA does not permit ligation due to inadequate length, patch closure on cardiopulmonary bypass is necessary. Success rate: Complete closure rates between 94% to 100% are seen.

Complications of this procedure are:
- Bleeding
- Injury to the recurrent laryngeal nerve
- Disruption of the thoracic duct with resultant chylothorax
- Injury to the vagus nerve
- Ligation of the left pulmonary artery
- Ligation of the descending aorta or other arterial structures within the chest
- Pneumothorax
- Infection
- Mortality: 0% to 2%.

Discuss Anesthetic considerations in a patient of PDA.

If the shunt is nonrestrictive and does not impede blood flowing freely in each direction then the main determinant of blood flow is the resistance of the pulmonary and systemic vascular beds. Left-to-right shunts results in increased pulmonary blood flow and hence three pathophysiologic problems:
- Volume overload of the pulmonary circulation
- Increased cardiac work of left ventricle which is required to increase stroke volume and heart rate to ensure adequate systemic perfusion
- Excessive pulmonary blood flow resulting in progressive elevation in PVR.

There are concerns about the increased workload on immature heart of the baby because of the following reasons:
- Relatively noncompliant
- Relatively restricted ability to change stroke volume
- Plateau of pressure-volume curve reached early
- Cardiac output more rate-dependent.

Persistence of large left-to-right shunts may outstrip the capacity of the left heart to maintain adequate systemic perfusion and result in congestive heart failure. The chronic effects of congenital heart disease are a consequence of the imposed hemodynamic stress of the defect and sequelae after interventional cardiac surgery; which alters normal growth and development of the cardiovascular system as well as other organ systems throughout life. Multidisciplinary team-oriented approach involving cardiac surgeons, cardiologists, anesthesiologists, intensivists, and nurses working as a team in preparing the patient and the family for surgery and postoperative recovery is ideal and beneficial. Anesthetic management should be based on sound physiologic principles.

History: Detailed history regarding birth history and maternal drug history
- Cyanosis
- Exercise tolerance
- Feeding history
- Medications
- Previous cardiac/other surgery.

Proper management of the physiologic abnormalities is more important than the choice of specific anaesthetic and pharmacological approaches.

Premedication: The goal of premedication is to achieve adequate sedation, maintain respiratory and hemodynamic stability. In a premature neonate, sedation is not required.

Monitoring
- Pre-induction
  - Precordial stethoscope
  - Pulse oximetry
  - ECG(II +/- V5)
  - Non-invasive blood pressure.
- Pre-incision
  - Invasive blood pressure
  - Hourly urine output measurement
  - Esophageal stethoscope
  - Temperature monitoring
  - Central venous pressure measurement may or may not be used.
- Other monitors which may be used are
  - Doppler transducer
  - $EtCO_2$
  - ABG
  - Inspired gas monitoring.

Infective endocarditis (IE) prophylaxis is not required (AHA 2008 guidelines on IE prophylaxis).

Anesthesia induction: Preoxygenate with 100% O_2. Inhalational induction is done with sevoflurane which decreases systemic vascular resistance (SVR) and improves systemic blood flow by decreasing the magnitude of left to right shunt. Intubate with the appropriate size ETT.

Maintenance of anesthesia: Analgesia with Fentanyl: 1–3 μg/kg. High dose opioids are not well tolerated in neonates. Muscle relaxation can be with either vecuronium or atracurium.

Ventilation: Positive pressure ventilation is well tolerated as a rise in airway pressure increases pulmonary vascular resistance (PVR) decreasing the shunt.

Maintenance of anesthesia with air: O_2 mixture and volatile anaesthetics. Make sure that the anesthesia machine has the capacity to provide air as well as oxygen and nitrous oxide to help balance pulmonary and systemic blood flow.

Reversal: Postoperative ventilation may be needed due to the prematurity of the baby.

Hypoglycemia is a frequent concern in neonates during the perioperative period.

In summary, anesthetic goal in patients with PDA whether for corrective surgery or non-cardiac surgery is to prevent increase in shunt (left-to-right) or reversal of flow (right-to-left)

- Avoid increase in the pulmonary vascular resistance: Avoid hypoxia, hypercarbia, acidosis, nitrous oxide, pain related catecholamine release.
- Prevent decrease in SVR, particularly drug induced cardiac output fall.

In what scenarios would you like to keep DA open and how will you do it?

In transposition of the great vessels (the pulmonary artery and the aorta), a ductus arteriosus may need to remain open as it is the only way that oxygenated blood can mix with deoxygenated blood. Prostaglandins are used to keep the patent ductus arteriosus open.

How do you calculate weight of the paediatric patients?

3-12 months: (age (months) +9)/2)
1-6 years: (Age (yrs) + 4) × 2)

What are the normal cardiovascular parameters in neonates infants and children? How do you estimate MAP and heart rate?

Table 5.1 Cardiovascular parameters in neonates and infants

Age	Blood Pressure	HR
Neonate Wt: 1kg	50/25	100–180
2 kg	55/30	100–180
3 kg	60/35	100–180
1 month	85/65	100–180
3 months	90/65	100–180
6 months	90/65	100–180
9 months	90/65	100–160
12 months	90/65	80–160

In children over 1 year

Mean systolic blood pressure = 90 + [Age (years) × 2]

Lower percentile: 70 + [Age (years) × 2]

Table 5.2 Heart rate calculation in children > 1 year

Age (years)	HR
2	80–130
6	70–120
12	60–100

Suggested Reading

1. Millers Textbook of Anaesthesia: 6th Edition
2. Oxford Handbook of Anaesthesia: 3rd Edition
3. Schneider DJ, Moore JW. Patent Ductus Arteriosus : Circulation 2006;114:1873–82

6 Permanent Pacemaker

S Bakshi, V Patil, S Myatra

65-year-old patient with a pacemaker comes for Total Knee Replacement. The patient gives a history of hospitalization for myocardial infarction 4 years back during which a pacemaker was inserted. The patient is asymptomatic now, however his daily activities are restricted in view of his arthritis. Describe the perioperative management of this patient.

What are the indications for a permanent pacemaker?

Artificial Pacemaker is indicated for treatment of symptomatic bradycardia of any origin. Indications of permanent pacemaker implantation are as follows:

- Acquired Atrioventricular (AV) block:
  - A. Third degree AV block
  - – Bradycardia with symptoms
  - – After drug treatment that cause symptomatic bradycardia
  - – Postoperative AV block not expected to resolve
  - – Neuromuscular disease with AV block
  - – Escape rhythm < 40 bpm or asystole > 3s.
  - B. Second degree AV block
  - – Permanent or intermittent symptomatic bradycardia
- After Myocardial infarction:
  - – Persistent second degree or third degree block
  - – Infranodal AV block with left bundle branch block (LBBB)
  - – Symptomatic second or third degree block.
- Bifascicular or Trifascicular block:
  - – Intermittent complete heart block with symptoms
  - – Type II second degree AV block
  - – Alternating bundle branch block.
- Sinus node dysfunction:
  - – Sinus node dysfunction with symptoms as a result of long-term drug therapy
  - – Symptomatic chronotropic incompetence.
- Hypertensive carotid sinus and neurocardiac syndromes:
  - – Recurrent syncope associated with carotid sinus stimulation
  - – Asystole of > 3s duration in absence of any medication.

A small minority of patients with bifascicular block may develop transient or established complete heart block. The risk of progression from bifascicular block to complete heart block in asymptomatic patients is very small. Insertion of temporary transvenous pacemaker before anesthesia is not necessary; an external pacemaker can be made available.

What are the indications of pacing after myocardial infarction?

Damage to the impulse formation and conduction system of the heart from MI can result in bradyarrhythmias and conduction disturbances. Disturbance of conduction distal to the atrioventricular node and bundle of His are worrisome. Ventricular escape rhythms in the setting of MI are unstable and unreliable. Indications for pacemaker after MI are:

- Class I
  - – Persistent complete heart block
  - – Persistent type II second-degree AV block
- Class II
  - – Newly acquired bundle branch block with transient high grade AV or complete heart block
  - – Newly acquired bundle branch block with first degree AV block
  - – Newly acquired bifascicular bundle branch block

What are the various types of pacemakers?

Pacemakers can be classified as per the method of insertion, location of leads, etc.

- Transvenous
- Transcutaneous
- Epicardial
- Transesophageal.

The original nomenclature by ICHD (Intersociety Commission for Heart Disease) Resources involved a three letter code. This code has been extended to five letters. Table 6.1 gives these generic codes for pacemaker.

Table 6.1 Generic codes of pacemaker

I Chamber Paced	II Chamber Sensing	III Response	IV Programmability	V Tachycardia
O- None	O- None	O- None	O- None	O- None
A- Atrium	A- Atrium	I- Inhibited	C- Communicating	P- Pacing
V- Ventricle	V- Ventricle	T- Triggered	P-Simple programmable	S-Shock
D- Dual (A+V)	D- Dual (A+V)	D- Dual (I+T)	M- Multiple programmable	D- Dual(P+S)
			R- Rate modulation	

Modern day pacemakers are programmable into one of three modes

- Asynchronous pacing or fixed rate (AOO, VOO, DOO) - pace at preset rate that is independent of the inherent heart rate. The most dangerous complication is ventricular fibrillation due to R on T phenomenon.
- Single Chamber demand pacing (AAI, VVI) paces at a preset rate when spontaneous heart rate falls below preset rate. The firing of the pacemaker is inhibited by electrical activity in the chamber sensed.
- Dual chamber AV sequential pacing requires two pacemaker leads, one in atrium and one in the ventricle. The atrium is stimulated to contract first, and after an adjustable PR interval the ventricle is stimulated to contract.

Dual chamber pacing is indicated when ventricular pacing alone cannot maintain adequate cardiac output and atrial pacing alone cannot be done as in third degree AV block. Major advantage is ability to increase cardiac output and reduce incidence of atrial fibrillation.

Explain some common terminology used with pacemaker

Pulse Generator: It includes the energy source (battery) and electric circuits for pacing and sensory function. Mercury–Zinc batteries that were used in the early days had a short useful life (2–3 years). Currently Lithium-iodide batteries are being used which have longer life (5–10 years) and high energy density.

Leads: Insulated wires connecting pulse generator and electrodes.

Electrode: An exposed metal end of the lead in contact with the endocardium or epicardium.

Unipolar Pacing: There is one electrode, the cathode (negative pole) or active lead. Current flows from the cathode, stimulates the heart and returns to anode (positive pole) on the casing of pulse generator via the myocardium and adjacent tissue to complete the circuit. A unipolar pacemaker is more likely to pick up extracardiac signals and myopotentials. Pacemaker spikes are large in this pacing (Figure 6.1).

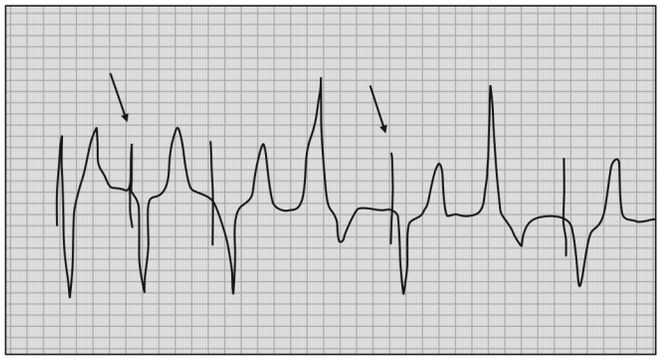

Fig. 6.1 Unipolar pacing

Bipolar Leads: Two separate electrodes, anode (positive pole) and cathode (negative pole), both located close to each other within the chamber that is being paced. As the electrodes are very close, the circuit is small and the possibility of extraneous noise disturbance is less, and the signals are sharp. Pacemaker spikes are very small in this type of pacing (Figure 6.2).

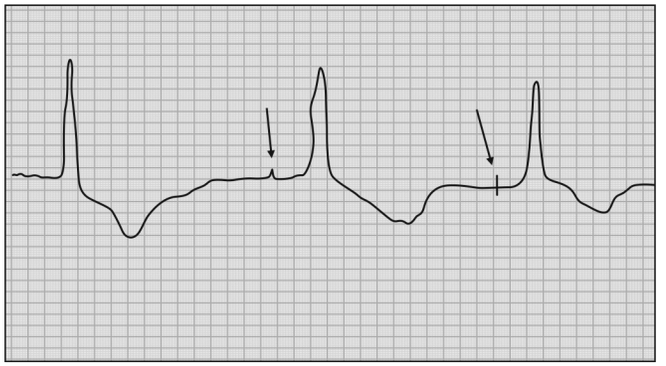

Fig. 6.2 Bipolar pacing

Endocardial Pacing: Also called as transvenous pacing which implies that the leads/electrodes system has been passed through a vein to the right atrium or right ventricle. It can be unipolar or bipolar.

Epicardial Pacing: This type of pacing is accomplished by inserting the electrode through the epicardium into the myocardium and is generally done following cardiac surgery. This can also be unipolar or bipolar.

Pacing Threshold: This is the minimum amount of energy required to consistently cause depolarization and contraction of the heart. Pacing threshold is measured in terms of both amplitude and duration for which it is applied to the myocardium. The amplitude is programmed in volts (V) or in milliamperes in some devices and the duration is measured in milliseconds.

Sensitivity: The minimal voltage level of the patient's intrinsic R wave or P wave that must be exceeded for the pacemaker to sense that R or a P wave and to activate the sensing circuit of the pulse generator, resulting in inhibition or triggering of the pacing circuit.

Resistance: The impedance to the flow of current. In the pacemaker system it amounts to a combination of resistance in lead, resistance through the patient's tissue and polarization that takes place when voltage and current are delivered into the tissues. Abrupt changes in the impedance may indicate problem with the lead system. Very high resistance can indicate a conductor fracture or poor connection to the pacemaker. A very low resistance indicates an insulation failure.

Hysteresis is particularly useful in patients with sick sinus syndrome. This feature allows a longer escape interval after a sensed event, giving the heart a greater opportunity to beat on its own. Here pacemaker is programmed to upper and lower rate and a programmable lower hysteresis rate.

Runaway Pacemaker is pacemaker dysfunction characterized by fast and erratic spikes. This occurs with generator dysfunction due to battery failure or damage produced by leakage of the tissue fluids into the pulse generator. Treatment with antiarrhythmic drugs or cardioversion may be ineffective in such cases. It is necessary to change the pacemaker to an asynchronous mode, or reprogram it to lower outputs. If the patient is hemodynamically unstable temporary pacing should be done followed by changing of pulse generator.

What do we understand by programmable pacemaker?

Our physiologic pacemaker, SA node responds to body's changing demands by increasing or decreasing heart rate. Patients on modes like DDD, VVI, and AAI modes cannot increase or decrease heart rate according to the metabolic demands. To overcome this problem recent generation pacemakers provide flexibility to adapt the device to patient's changing metabolic needs. Sensors capable of detecting body movements (vibrations), changes in ventricular repolarisation, central venous temperature, central venous oxygen saturation, respiratory rate and depth, and right ventricular contractility are commonly used in clinical practice. The various factors, which can be programmed are pacing rate, pulse duration, voltage output, R wave sensitivity, refractory periods, PR interval, mode of pacing, hysteresis (difference between intrinsic heart rate at which pacing begins and pacing rate), and atrial tracking rate.

What is a biventricular pacemaker? What is cardiac resynchronisation therapy (CRT)?

CRT devices have been shown to reduce mortality and improve quality of life in patients with heart failure symptoms. Electrical depolarization is normally initiated throughout both ventricles by the His–Purkinje system. In patients with systolic dysfunction with conduction disturbances as manifested by prolonged QRS complex, conduction of the wave of depolarization in the left ventricle is markedly altered. As a result left ventricular contraction becomes dys-synchronous, with resultant decrease in stroke volume, increased wall stress, and delayed relaxation. A biventricular pacemaker, also known as CRT (cardiac resynchronization therapy) is a type of pacemaker that paces both the septal and lateral walls of the left ventricle. By pacing both sides of the left ventricle, the pacemaker can resynchronize a heart to contract in synchrony. This has been shown to be useful in heart failure. CRT devices have at least two leads, one in the right ventricle to stimulate the septum, and another inserted through the coronary sinus to pace the lateral wall of the left ventricle. For patients in normal sinus rhythm, there is also a lead in the right atrium to facilitate synchrony with the atrial contraction. Thus, timing between the atrial and ventricular contractions, as well as between the septal and lateral walls of the left ventricle can be adjusted to achieve optimal cardiac function. The American Heart Association Science Advisory in 2005, has stated that patients with dilated cardiomyopathy, with LVEF ≤ 0.35, a QRS complex ≥ 120 ms, and sinus rhythm, and are NYHA functional class III or IV despite maximal medical therapy for heart failure are candidates for CRT.

What is Pacemaker syndrome?

Pacemaker syndrome also known as AV dyssynchrony syndrome represents the clinical consequences of AV dyssynchrony or suboptimal AV synchrony, regardless of the pacing mode. This leads to a variety of clinical signs and

symptoms resulting from deleterious hemodynamics. These include hypotension, syncope, vertigo, light-headedness, fatigue, exercise intolerance, malaise, weakness, lethargy, dyspnea, and induction of congestive heart failure. Cough, awareness of beat-to-beat variation of cardiac response from spontaneous to paced beats, neck pulsation or pressure sensation in the chest, neck, or head, headache, and chest pain are the other symptoms. Symptoms may vary from mild to severe, and onset may be acute to chronic. The main mechanisms behind these symptoms are—loss of atrial kick with resultant drop in cardiac output, increased atrial pressure leading to symptomatic pulmonary and hepatic congestion, retrograde ventriculo-atrial conduction leading to delayed, nonphysiologic timing of atrial contraction in relation to ventricular contraction and nonphysiologic ventricular depolarization pattern.

How will you evaluate this patient preoperatively?

Preoperative evaluation includes

- *Detailed evaluation of the underlying cardiovascular disease* responsible for the insertion of pacemaker
- *Other associated medical problems:* Since substantial number of these patients suffer from coronary artery disease (50%), hypertension (20%) and diabetes (10%), one should know:
  - Severity of the cardiac disease,
  - Current functional status,
  - Medications the patient is on.
- The initial indication for the pacemaker:
  - Preimplantation symptoms such as lightheadedness, dizziness or fainting. If these symptoms occur even after the pacemaker insertion, cardiology consultation is needed.
- Bruits, signs of CCF
- Evaluation of Cardiac status:
  - Dobutamine stress test: Despite the presence of chronotropic incompetence among pacemaker-dependent patients, there is preservation of both a positive inotropic response to dobutamine with increased myocardial contractility and changes in left ventricular loading conditions leading to increased myocardial oxygen demand. The demonstration of inducible ischemia during these conditions suggests that HR increase may not be necessary to provoke ischemia. Because dobutamine can provoke ischemia on the basis of factors independent of HR, tests may still be diagnostic despite a blunted HR response and failure to achieve a specific target HR.
- Evaluation of the pacemaker
  - Establish whether a patient has a CRMD (cardiac rhythm management device)
  - Focused history: Details of manufecturer and identification card
  - Physical examination for scars, palpating the device (Generally, generator for the epicardial electrodes is kept in the abdomen and over one of the pectoris muscles for the endocardial electrodes.
- Investigations
  - Routine biochemical and hematological investigations should be performed as indicated on an individual basis
  - Measurement of serum electrolytes (especially K+) should be performed
  - A 12 lead electrocardiogram: In atrial pacing, an electrical spike appears before the P wave and the QRS complex is usually normal. In ventricular pacing, there are two spikes, one before the P wave and another preceding the QRS complex
  - X-ray chest-(for visualization of continuity of leads).

How will you evaluate the function of the pacemaker in the preoperative period?

Assistance from the cardiologist and the manufacturer's representative should be obtained for the purpose.

Most of the information about the pacemaker, such as type of pacemaker (fixed rate or demand rate), time since implanted, pacemaker rate at the time of implantation, and half-life of the pacemaker battery can be taken from the manufacturer's book kept with the patient. Details of pacemaker function can be elicited from the patient regarding last battery and pulse generator check. 10% decrease in the rate from the time of implantation indicates power source depletion. In patients with VVI generator, if intrinsic heart rate is greater than pacemaker set rate, evaluation of pacemaker function can be done by slowing down the heart rate by carotid sinus massage, while the patient's ECG is continuously monitored. Carotid sinus massage should be avoided in patients with a history suggestive of cerebrovascular disease or carotid artery disease, because the atherosclerotic plaque may embolise to the cerebral circulation. Other methods to slow the heart rate are Valsalva maneuver and use of edrophonium. Reprogramming the pacemaker is generally indicated to disable rate responsiveness. Patients with Implanted Cardioverter Defibrillator (ICD) should have the tachycardia therapy (anti-tachycardia pacing and shocks) deactivated before surgery that may require cautery. If the risk of electromagnetic interference (EMI) is high, such as, when

the electricity is in close proximity to the generator, alternative temporary cardiac pacing device should be kept ready.

Describe the effect of magnet application on pacemaker function.

During surgery, electromagnetic interference from electrocautery and similar devices can seriously interfere with the function of pacemaker working in a demand mode. This can be avoided by converting the pacemaker to a fixed rate asynchronous mode by application of a magnet. Magnet application is an extremely important function. Most pacemakers and ICDs have built-in magnetic reed switches that are designed to switch 'ON' or 'OFF' circuitry in response to magnets. The magnet is placed over the pulse generator to trigger the reed switch present in the pulse generator resulting in a non-sensing asynchronous mode with a fixed pacing rate (magnet rate). Magnet operated reed switches were originally incorporated to produce pacemaker behavior that would demonstrate remaining battery life and sometimes pacing thresholds. Activation of the reed switch shuts down the demand function so that the pacemaker stimulates asynchronous pacing. Thus, magnets can be used to protect the pacemaker dependent patient during EMI, such as diathermy or electrocautery. Also if no information is available from the patient about the pacemaker, magnet may identify the particular model with the help of magnet rate, which varies among different manufacturers and thus provide clue for its identification. However, not all pacemaker's switch to asynchronous mode on the application of magnet. The response varies with the model and the manufacturer, and may be in the form of no apparent change in rate or rhythm, brief asynchronous pacing, continuous or transient loss of pacing, or asynchronous pacing without rate response. In an ICD system magnet cannot convert the pacemaker to asynchronous mode, it only disables tachycardia detection and therapy of the ICD. Clinical magnets, made of ferrous alloy, come in various shapes (ring or doughnut, horseshoe, and rectangle or bar).The site of magnet placement is important since a poorly positioned magnet may not produce the desired effect. Magnets are best placed directly on top of the device. It is advisable to consult the manufacturer to know the magnet response before use. The patient must be connected to an electrocardiograph recorder before the magnet is applied and, remain connected, until after the magnet is removed. The first few paced complexes after magnet application provide information regarding the integrity of the pulse generator and its lead system. A 10% decrease in magnet rate from the time of implantation indicates power source depletion and is an indicator of end of life requiring elective replacement of battery. If magnet application on a pacemaker site does not produce any response on the surface ECG pacing rate or mode, the magnet may be repositioned. If no change is still observed, reasons may be: (i) A depleted pacemaker battery; (ii) The pacemaker is programmed to ignore the magnet (St. Jude, Boston Scientific, and Biotronik synchronous mode); (iii) The magnetic field does not reach the device, as in the case of those with deeper (abdominal or submuscular) implants or in very obese patients; (iv) End Of Life or lower battery life. The most current devices should be considered programmable unless known otherwise.

How do you test for the chronotropic response of the paced heart?

In patients with bundle branch block (except for cases with complete heart block) and endogenous activity—a small dose of adrenaline is given intravenously; and the pacemaker demand mode is reduced—the chronotropic response of SA node is noted. If the response is adequate then it can be concluded that patient is not entirely pacemaker dependant.

How would you reprogram the Implantable Cardioverter- Defibrillator (ICD) preoperatively?

ICD can be reprogrammed to disable the anti-tachycardia function before surgery in operating room where a defibrillator is readily available. A magnet can be placed over the ICD to disable the anti-tachycardia function in the operating room. The advantage of placing a magnet on an ICD is that the magnet can be easily removed and anti-tachycardia function is quickly enabled in case of VT/VF during surgery.

How do you preoperatively prepare the patient for surgery and anesthesia?

Preoperative preparation includes the following:

- Determine whether electromagnetic interference (EMI) is likely to occur during the planned procedure.
- Determining whether reprogramming the cardiac rhythm management device (CRMD), i.e. changing to an asynchronous pacing mode or disabling any special algorithms, including rate-adaptive functions is needed.
- Suspending anti-tachyarrhythmia function if present.
- Advising the surgeon performing the procedure to consider use of a bipolar electrocautery system or ultrasonic scalpel to minimize potential adverse effects of EMI on the pulse generator or leads.

- Assuring the availability of temporary pacing and defibrillation equipment and drugs for resuscitation.
- Evaluating the possible effects of anesthetic technique on CRMD function and patient CRMD interactions.

Intraoperatively you can see the pacing spikes but they are not followed by QRS complex or ventricular activity. What has gone wrong? What is pacing threshold and what are the factors affecting it?

Pacing threshold is the minimum amount of energy required to consistently cause depolarization and therefore contraction of the heart. Pacing threshold is measured in terms of both amplitude and duration for which it is applied to the myocardium. The amplitude is programmed in volts (V) or in milliamp in some devices, and the duration is measured in milliseconds. Table 6.2 lists the factors affecting pacing threshold.

Table 6.2 Factors affecting pacing threshold

Factors which increase pacing threshold	Factors which decrease pacing threshold
1–4 weeks after implantation	Increased catecholamines
Myocardial ischemia/infarction	Stress, anxiety
Hypothermia, hypothyroidism	Sympathomimetic drugs
Hyperkalemia, acidosis/alkalosis	Anticholinergics
Antiarrhythmics (class IA,IB,IC)	Glucocorticoids
Severe hypoxia/hyperglycemia	Hyperthyroidism
	Hypermetabolic status

If pacing threshold of myocardium increases more than that of pacemaker due to any of the above mentioned reasons, the pacemaker spike will be seen on the ECG but it will not be followed by a QRS complex or cardiac contraction, i.e. failure to capture (Figure 6.3). This is different from pacemaker failure, where the pacemaker does not fire, and no spike is seen on the ECG (Figure 6.4).

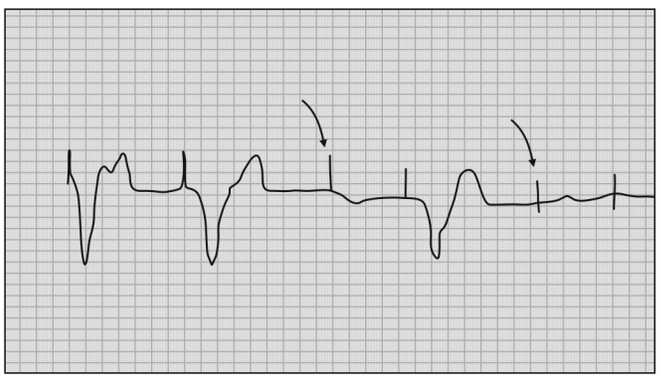

Fig. 6.3 Failure to capture

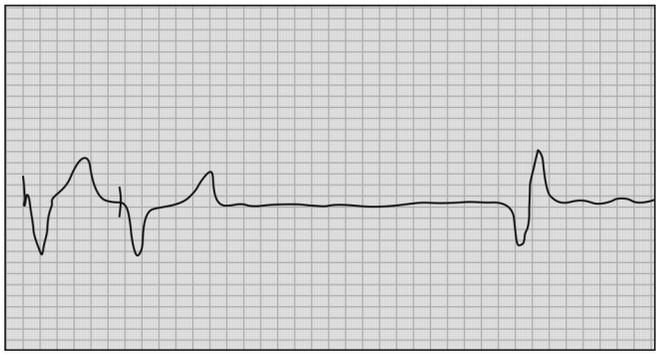

Figure 6.4 Failure to pace (absence of spikes)

Factors Important from Anesthesia Point of View

- *Physiological effects:* During the first 2 weeks, there is an initial sharp increase in the pacing threshold, i.e. up to ten times the acute level because of the tissue reaction around the electrode tip. Then it decreases to two to three times the acute level because of the scar formation. In chronic state, it reaches the initial level in 80% of patients. But this has become far less of a problem with the introduction of steroid-eluting leads and other refinements in the lead technology.
- *Potassium:* This is an important electrolyte that determines the resting membrane potential (RMP). A mild to moderate increase in serum potassium causes an increase in myocardial excitability, but further increase leads to impaired myocardial responsiveness, including that to pacing stimulation.
- *Myocardial Infarction:* Its scar tissue is unresponsive to electrical stimulation and may cause loss of pacemaker capture.
- *Antiarrhythmic Drug Therapy:* Class Ia. (quinidine, procainamide), Ib. (lidocaine,diphenylhydrantoine), and Ic. (flecainide,encainide, propafenone) drugs have been found to increase the pacing threshold.
- *Acid Base Status:* Alkalosis and acidosis both cause increase in pacing threshold.
- *Hypoxia* increases pacing threshold.
- *Anesthetic Drugs* are not likely to change the pacing threshold. It is notable that addition of equipotent halothane, enflurane, or isoflurane to opiate based anesthesia after cardiopulmonary bypass did not increase pacing threshold.

How can you establish temporary pacing in this case?

Transcutaneous: Defibrillating electrodes such as Zoll Pads are preferred. They can be connected to a defibrillator or pacemaker. The electrodes should be placed as far (more than

6 in. or 15 cm) from a CRMD. Three recommended electrode placements are as follows:

Anteroposterior placement: The right arm (RA) electrode placed under the left scapula and the left leg electrode at apex of the heart

Postero-anterior placement: The RA electrode placed under the right clavicle and the LL electrode at the apex of the heart

Apex posterior placement: The RA electrode placed over the right scapula and the LL electrode at the apex of the heart

Transesophageal: Transesophageal atrial pacing is feasible because of the proximity between the esophagus and the posterior aspect of the left atria.

What is Transesophageal Pacing?

Transesophageal atrial pacing is feasible in almost all patients because of the proximity between the esophagus and the posterior aspect of the atria. Transesophageal pacing and recording is done using specialized or non-specialized catheters. There are two different lead types:

1. The pill electrode, connected to a flexible wire that the patient swallows with water
2. A flexible catheter that can even be used in comatose or intubated patients and passed nasally.

It is positioned into the esophagus in order to record the posterior paraseptal atrial electrogram.

There is a relationship between the site of maximal atrial amplitude and the lowest atrial pacing threshold. The optimal atrial pacing site is usually found around 40 cm from the nares. There are a few limitations to the technique:

1. There is only one site of atrial pacing and recording,
2. There is no ventricular pacing,
3. Sometimes atrial capture can be difficult to assess on the surface ECG
4. Leads to patient discomfort most frequently described as mild burning or chest discomfort like indigestion.

Discuss Intraoperative monitoring for patients with pacemakers.

Intraoperative monitoring should be based on the patient's underlying disease and the type of surgery. Continuous ECG monitoring is essential to monitor pacemaker functioning. The artifact filter on the ECG monitor should be disabled in order to detect the pacing spikes. In addition, mechanical evidence of the cardiac contraction should be monitored by palpation of the pulse, pulse oximetry, precordial stethoscope and arterial line, if indicated. Presence of pacemaker is not an indication for insertion of pulmonary artery (PA) or central venous catheter. If these are indicated, care should be taken during insertion of the guide wire or central venous catheter as they are potentially arrhythmogenic and can also dislodge pacemaker leads, especially in recently implanted CRMDs. It is best to avoid the insertion of PA catheter. During use of unipolar electrocautery the ECG is frequently unreliable because of interference. The best monitor to determine if pacemaker is functioning properly is a hand on the pulse. The precordial or esophageal stethoscope or pulse oximeter can also be used. Since these patients frequently have underlying cardiac dysfunction other parameters of perfusion should also be monitored. These include capillary refill, urine output, peripheral temperature, etc.

What would be your choice of anesthesia for this patient? What precautions will you take during anesthesia?

The anaesthetic technique should be used according to the need of the patient. Though there are no guidelines favoring or contradicting use of regional anesthesia in patients with pacemaker, few case reports of use of regional techniques in obstetric are available. One must remember that a paced heart cannot compensate for hypotension by tachycardia and hence spinal anesthesia should be used cautiously and preferably avoided in cases of anticipated blood loss or fluid shift. Since our patient is undergoing TKR surgery, femorosciatic block may be given. Spinal anesthesia aimed for block below T12 level or graded epidural analgesia can also be safely used provided there are no other contraindications for central neuraxial blockade.

Intraoperative management in case of GA: Both narcotics and inhalational techniques can be used safely. These anesthetic agents do not alter current and voltage thresholds of the pacemaker. Skeletal myopotentials commonly encountered with succinylcholine, myoclonic movements, or direct muscle stimulation can inhibit or trigger pacemaker and should be avoided. Avoid perioperative hypothermia as muscle activity caused by shivering may affect pacemaker functioning. Use of nondepolarizing muscle relaxants is safe. In induction agents etomidate and ketamine should be avoided as these can sometimes cause myoclonic movements.

Is there a need to reprogram the pacemaker and convert it to asynchronous mode for every surgery?

Reprogramming the pacemaker to asynchronous fixed mode puts patient at risk of developing R on T phenomenon and ventricular tachycardia especially for patients with good underlying rate. When the risk of electromagnetic interference (EMI) is unlikely to interfere with pacemaker as in surgeries below umbilicus, contralateral arm, eyes or on

face or surgeries not requiring use of cautery, pacemaker need not be reprogrammed. Also for patients who are not dependent on pacemaker as elicited from history and preoperative assessment, it is better to leave pacemaker on demand mode. But in all situations rate responsive function should be suspended.

What are the effects and how will you minimize the effect of electrocautery?

The responses of pacemakers to electrocautery include:

- Inhibition of pacing
- Asynchronous pacing
- Reset to backup mode
- Myocardial burns (rare)
- VF (rare).

Responses of ICD include:

- Inhibition of pacing
- Asynchronous pacing
- Inappropriate tachytherapy
- Inhibition of tachytherapy.

One should apply the following measures to decrease the possibility of adverse effects due to electrocautery.

1. Bipolar cautery should be used as much as possible as it has less EMI.
2. If unipolar cautery is to be used during operation, the grounding plate should be placed close to the operative site (active cautery tip) and as far away as possible from the site of pacemaker, usually on the thigh and should have good skin contact.
3. Electrocautery should not be used within 15 cm of pacemaker.
4. Frequency of electrocautery should be limited to 1-second bursts in every 10 seconds to prevent repeated asystolic periods. Short bursts with long pauses of cautery are preferred.
5. Pacemaker may be programmed to asynchronous mode by a magnet or by a programmer. Before using cautery, the programmer must be available in the operation theatre (OT). During the use of cautery, magnet should not be placed on pulse generator as it may cause pacemaker malfunction.
6. If defibrillation is required in a patient with pacemaker, paddles should be positioned as far away as possible from the pacemaker generator. If possible, anterior to posterior positioning of paddles should be used. Although permanent pacemakers have protective circuits to guard against externally applied high voltage, pulse generator malfunction has been reported.
7. In elective cardioversion, the lowest voltage necessary should be utilized. However, even with these precautions, defibrillation may result in acute increase in the stimulation threshold, with resultant loss of capture. If this occurs, immediate reprogramming or temporary pacing should be done with increased generator output.
8. Careful monitoring of pulse, pulse oximetry and arterial pressure is necessary during electrocautery, as ECG monitoring can also be affected by interference.

The device should always be rechecked after operation.

In the middle of surgery, the patient with an ICD develops ventricular tachycardia. What would you do?

For a patient with an ICD and magnet disabled therapies/ programming disabled therapies:

- Avoid all sources of EMI (electro-magnetic interference)
- Remove the magnet/re-enable therapies through programming if programmer available
- Observe patient for appropriate CRMD therapy
- If above activities does not start the ICD function proceed with external defibrillation/cardioversion. For external cardio version place pads or paddles as far as possible from CRMD. Place defibrillator pads perpendicular to the major axis of CRMD to the extent possible by placing them in an anteroposterior location. If technically impossible to place the pads or paddles in location that help to protect the CRMD, Defibrillate/cardiovert the patient in the quickest possible way and be prepared to provide pacing through other routes.

Are there any special concerns in the postoperative period?

In patients with rate responsive pacemakers, rate responsive mode should be deactivated before surgery. If this is not possible for some reason, the mode of rate response must be known so that conditions causing changes in paced heart rate can be avoided. For example, shivering and fasciculations should be avoided, in the postoperative period. If the pacemaker is 'activity' rate responsive- ventilation should be kept controlled and constant and temperature must be kept constant in 'temperature' rate responsive pacemakers.

In a patient with pacemaker for ECT, what care would you take?

ECT appears safe for patients with pacemakers, since little current flows within the heart because of the high impedance of body tissue, but the seizure may generate myopotentials which may inhibit the pacemaker. Thus ECG monitoring is essential and pacemakers should be changed to non-sensing asynchronous mode (fixed mode).

Any others areas where one needs to excise caution with patient with pacemaker?

Transuretheral Resection of Prostate (TURP) and Uterine Hysteroscopy: Coagulation current used during TURP procedure has no effect, but the cutting current at high frequencies (up to 2500 kc/sec) can suppress the output of a bipolar demand ventricular pacemaker. When electrosurgical unit (ESU) use is anticipated reprogramming of pacemaker preoperatively to the asynchronous (fixed rate) mode should be performed.

Radiation: Cases where radiation therapy is planned for deep seated tumors, therapeutic radiation can damage the complementary metal oxide semiconductors (CMOS) that are the parts of most modern pacemakers. Generally, doses in excess of 5000 rads are required to cause pacemaker malfunction but as little as 1000 rads may induce pacemaker failure or cause runaway pacemaker. If pacemaker cannot be shielded from the field of radiation, consideration should be given to reimplanting the pacemaker generator at distant site. Temporary damage to pacemaker may recover after reprogramming but there may be permanent damage to the pacemaker as well. This effect could be attributed to charge accumulation inside CMOS after radiation therapy leading to failure of various components in the circuitry and therefore, cause pacemaker failure.

Transcutaneous Electronic Nerve Stimulator Unit (TENS): TENS is now a widely used method for pain relief. TENS unit consists of several electrodes placed on the skin and connected to a pulse generator that applies 20 μsec rectangular pulses of 1 to 200 V and 0 to 60 mA at a frequency of 20 to 110 Hz. This repeated frequency is similar to the normal range of heart rates, so it can create a far field potential that may inhibit a cardiac pacemaker. Adverse interaction between these devices has been frequently reported, so these patients should be monitored during initial application of TENS. Pacemaker mediated tachycardia has been induced by intraoperative somatosensory evoked potential stimulation.

Studies and case reports suggest that unipolar electrode seems to be most susceptible to interferences.

Nerve Stimulators used for Regional nerve blocks: One case of pacemaker interference caused by activation of a nerve stimulator has been reported. However, with the advancements of modern pacemaker's technology, a prospective study from Mayo clinic shows that interscalene nerve blocks and other peripheral nerve blocks using the nerve stimulator can be performed in patients with pacemakers without notable interferences with pacemaker functions.

Lithotripsy: Anesthesia may be required in patients undergoing extracorporeal shock wave lithotripsy (ESWL) for immobilization and to avoid pain in flank at entry site of waves. There may be electrical interference from hydraulic shock waves and can cause mechanical damage. High-energy vibrations produced by lithotripsy machine can cause closure of reed switch causing asynchronous pacing. 'Activity' rate responsive pacemaker can be affected due to the damage caused to the piezoelectric crystals by ESWL. The shock waves can produce ventricular extrasystoles, if not synchronized with R wave. Pacemaker malfunction can occur in patients undergoing ESWL, requiring adequate preparation prior to procedure. One should have cardiologist's opinion, perioperative ECG monitoring, device programmer and a standby cardiologist to deal with any device malfunction. Rate responsive pacemaker should have their activity mode deactivated. Focal point of the lithotriptor should be kept at least six inches (15 cm) away from the pacemaker. Patient with abdominally placed pacemaker generators should not be treated with ESWL. Low shock waves (< 16 kilovolts) should be used initially followed by a gradual increase in the level of energy.

Dual chamber demand pacemaker is especially sensitive to shock waves and should be reprogrammed to a simpler mode (VOO, VVI) preoperatively.

MRI: Three types of powerful forces exist in the MRI suite.

Static Magnetic Field: An intense static field is always present even if the scanner is not imaging. Most of the pacemakers contain ferromagnetic material, which gets attracted to the static magnetic field in the MRI and may exert a torque effect leading to discomfort at the pacemaker pocket. The reed switch activation by high static field of 0.5–1.5 T can result in switching of pacemaker to a non-sensing asynchronous pacing.

Radiofrequency Field (RF): This field is switched on and off during magnetic resonance imaging and has a frequency of 21–64 MHz depending on the strength of magnetic field. The radiofrequency signals can cause interference with pacemaker output circuits resulting in rapid pacing at multiple of frequency between 60–300 bpm causing rapid pacing rate. It may cause pacemaker reprogramming and destruction of electronic components. It may also cause heating at the electrode-tissue boundary, which may cause thermal injury to endocardium and myocardium.

Gradient Magnetic Field: used for spatial localization changes its strength along different orientations and operates at frequencies in order of 1 kHz. Gradient magnetic field may also interact with reed - switch in pacemaker. Inappropriate sensing and triggering because of the induced voltages can occur.

Patients with pacemakers should not routinely undergo MRI scanning. Further studies are necessary to refine the appropriate strategies for performing MRI safely in a patient with implanted

pacemaker. The risk benefit ratio must be individually evaluated in every patient with a pacemaker. Patients, who require head MRI scanning without alternative diagnostic possibilities, may be best served in a carefully monitored setting. Appropriate patient selection should be done and equipment for resuscitation and temporary pacing should be available. A cardiologist should be present. Also patients should be closely monitored with ECG and oxygen saturation.

What is an implantable cardioverter-defibrillator (ICD)?

A ICD system consists of a pulse generator and leads for tachyarrhythmia detection and therapy. It provides antitachycardia and antibradycardia pacing, synchronized or nonsynchronized shocks, telemetry and diagnostics including storing even electrograms and history logs. Modern ICD use transvenous lead systems for sensing, pacing and shocks. Epicardial leads are still in use for infants and children. Current ICD have any programmable features, essentially measure each cardiac R-R interval and categorize the rate as normal, too fast or too slow. When the device detects a sufficient number of short R-R intervals within a period of time it will declare a tachycardia episode. The internal computer will decide between antitachycardia pacing and shock based on its programmed algorithm. Typically ICD delivers no more than six shocks per episode. Once a shock is delivered, the ICD will redetect to determine whether the shock successfully terminated the arrhythmia (Table 6.3).

Table 6.3 Types of ICD-NASPE/BPEG generic defibrillator (NBD) code

Letter I shock chamber (s)	Letter II Antitachy-cardia pacing chamber	Letter III tachycardia detection	Letter IV Antibradycardia pacing chamber(s)
O = None	O = None	E = Electrogram	O = None
A = Atrium	A = Atrium	H = Hemodynamic	A = Atrium
V = Ventricle	V = ventricle		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)		D = Dual (A+V)

Indications

- Hemodynamically significant ventricular tachycardia or ventricular fibrillation
- Spontaneous sustained VT with structural heart disease
- Syncope of undermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at EPS.

What are the considerations while giving anesthesia for insertion of pacemaker?

Pacemaker is generally inserted under local anesthesia with monitored anesthesia care. Conventionally, cardioverter/defibrillator systems are implanted under general anesthesia. With the development of single-lead transvenous unipolar cardioverter/defibrillator systems for subpectoral implantation a pacemaker-like approach for device implantation appears applicable. Implantation of a single-lead transvenous unipolar cardioverter/defibrillator under local anesthesia with sedation for defibrillation threshold testing is well tolerated by patients.

In case if general anesthesia is used in an uncooperative patient; nitrous oxide could cause pacemaker malfunction by increasing gas in the pectoral pacemaker pocket. Despite air evacuation with antibiotic solution before closure of the pectoral pocket, a small amount of air remains entrapped in the pocket. In general this amount of air should not have clinical significance. However nitrous oxide is 35 times more soluble in blood than nitrogen. This causes an expansion of the gas in the pocket which leads to loss of a nodal contact and pacing malfunction.

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7 Peripheral Vascular Disease

P Jain, A Kulkarni, S Bhosale

A 60-year-old male patient, smoker with painful claudication having gangrene of left foot is scheduled for amputation.

What is peripheral vascular disease (PVD)?

PVD is segmental inflammatory vasculitis leading to occlusion of small and medium size arteries and veins in extremities. Thromboangiitis obliterans is a common example. Disease can affect vessels in upper or lower extremities and sometimes that of brain, myocardium, lungs, kidney, and intestine. Patient mostly presents with claudication, rest pain, gangrene, and ulcer. Reynaud's phenomenon may accompany the disease. Superficial thrombophlebitis may lead to pain and edema. Ischemic Pain is more during night and is aggravated by elevation of limb. There may be paresthesias due to nerve involvement. The disease progress is slow and symptoms are intermittent. PVD is a powerful predictor of future cerebrovascular and cardiovascular events such as myocardial infarction and stroke, and of increased mortality.

What is the difference between Buerger's disease and Thromboangiitis obliterans?

Buerger's disease	Thromboangiitis obliterans
Age: 20–45	Age 45 and above
Mostly males	Mostly males. Female 10%
Upper limbs	Upper limbs seldom involved
Superficial veins	Deep veins occasionally
Onset ~ foot pain, claudication	Onset ~ calf claudication
Gangrene often early, nearby pulses present	Gangrene 10%, later, loss of pulses
Smoking +	Smoking +, other factors
Sympathectomy/anticoagulants	Sympathectomy and anticoagulation doubtful

Contd...

Contd...

Cholesterol normal	Cholesterol high
No calcification	Calcification present
Normal ECG	Diabetes mellitus
Life prognosis good	Life prognosis reduced

What is Takayasu's arteritis?

Takayasu's arteritis is an idiopathic, chronic, progressive occlusive vasculitis that causes narrowing, thrombosis, or aneurysms of systemic and pulmonary arteries. Inflammatory changes are seen preferentially in large blood vessels such as the aorta and its branches.

What is Reynaud's phenomenon? Why does it occur?

It is episodic vasospastic ischemia of the digits. It is seen more often in women. It is characterized by digital blanching, cyanosis, and rubor after cold exposure and rewarming. Blanching represents the ischemic phase of the phenomenon caused by digital vasospasm.

Define Claudication.

Claudication can be described as functional ischemia characterized by reproducible lower-extremity muscle pain induced by a defined amount of activity. The pain is relieved by rest. The symptoms are due to the inability of the vasculature to provide blood flow according to the metabolic demand of the exercising tissue.

Claudication at rest is described as critical ischemia where arterial perfusion is inadequate to meet the resting demands of tissue and is indication for surgical intervention.

What are pre-anesthetic considerations in PVD?

Respiratory: Patients with PVD frequently have history of smoking and COPD. Preoperative evaluation of pulmonary function helps to determine whether regional v/s GA is appropriate, while providing baseline values for postoperative comparison.

Tests: CXR; consider PFTs and ABG

Cardiovascular: Vascular surgery of the lower extremities is often associated with morbidity and mortality secondary to incidence of CAD and HTN in this patient population.

Tests: ECG; 2D Echo, Dipyridamole Thallium Imaging (DTI), Radionuclide Ventriculography and Dobutamine stress Echocardiography.

Neurological: Patients with PVD have increased incidence of cerebrovascular disease. Careful neurological assessment is necessary to document existing deficits.

Endocrine: Increase incidence of diabetes mellitus may be associated with peripheral and autonomic neuropathies (silent MI, labile BP) and delayed gastric emptying. Frequent blood glucose measurements should be made preoperatively.

Renal: There is a higher incidence of renal artery disease and renal insufficiency in this patient population. Test: BUN and creatinine; consider creatinine clearance; electrolytes and uric acid.

Hematologic: Many patients presenting for this surgery are taking anticoagulant/antiplatelet medication. Inquire as to bleeding or bruising tendency. Tests: Hematocrit, platelet count; PT and PTT.

How do you investigate a case of PVD?

The PVD patients commonly come for emergency surgeries and optimum workup may not be always possible due to the associated comorbidities. Patients with PVD are at high risk for developing postoperative complications and the long-term survival in such patients is less than 65%.
Aim of pre-anesthetic evaluation is:

1. To rule out other causes of vascular obstruction like atherosclerosis, collagen vasculitis and hypercoagulability.
2. To exclude proximal source of emboli and involvement of larger vessels.
3. To find out organ involvement, to find out systemic effects due to smoking.

How will you investigate a patient with PVD?

- *CBC:* Polycythemia, infection, ESR, blood grouping and Cross matching.
- *Urine:* Albumin, sugar, blood sugar: serum creatinine, LFT.
- *Lipid profile:* Triglycerides, serum cholesterol , high and low density lipoproteins.
- ANA, rheumatoid factor, antiendothelial cell antibodies.
- *Prothrombotic states:* Decreased antithrombin protein C, protein-S, prothrombin.
- Anti-phospholipid antibodies, complement, fibrinogen.
- ECG, dobutamine stress echo.
- *X-ray chest:* COPD and calcified aorta.
- *ABG:* PCO_2, PaO_2: Baseline.
- Doppler ultrasound – Blood flow detection (ABP).
- An efficient method of objectively documenting the presence and severity of lower extremity PVD is determination of the Ankle Brachial Pressure Index. This is done by measuring blood pressure at the ankle and in the arm while a person is at rest. Measurements are usually repeated at both sites after 5 minutes of walking on a treadmill. The ratio of ankle systolic pressure to brachial artery systolic pressure is called ankle-brachial pressure index. During exercise, the increased flow through the narrowed vessels leads to decrease in pressure causing fall in ankle pressures. An ABPI between 0.9 and 1.2 is considered normal, 0.3 to 0.9 indicates arterial disease and less than 0.3 indicates impending gangrene. When compared to angiography, the sensitivity of the ABI is about 90%, and the specificity is about 98% for stenosis of 50% or more in leg arteries.
- Duplex imaging can show image of vessel, velocity of blood flow as good as arteriography.
- *Isotope scanning:* Perfusion of limb.
- Digital subtraction angiography.
- *Angiography:* Cork screw or root tree appearance due to collaterals.

What are the factors, which predict perioperative morbidity and mortality?

ACC/AHA clinical predictors of perioperative risks

- Major
  - Unstable angina/MI in 30 days
  - Congestive cardiac failure
  - Arrhythmias
  - Severe valvular disease
- Intermediate
  - Mild angina
  - MI (30 days earlier)
  - Compensated CCF
  - Diabetes mellitus
  - Renal insufficiency
- Minor
  - Age >70 years
  - Abnormal ECG

- Rhythm other than sinus
- H/o stroke
- Uncontrolled hypertension.

What is medical treatment of PVD?

- Control hypertension, coronary artery disease, diabetes mellitus, dyslipidemia, hypothyroidism, atherosclerosis, bronchospasm, respiratory infection.
- Stop smoking, protection from trauma, infection and cold.
- Analgesics: NSAIDs, narcotics.
- Warfarin, Antiplatelet (aspirin, clopidogrel), ACE inhibitors, pentoxiphylline
- Statins decrease the incidence of intermittent claudication.
- *Cilostazol:* 100 mg BD (Phosphodiesterase inhibitor) increase exercise time to development of intermittent claudication.
- Prostaglandin E_1 and E_2 infusions.
- Intra-arterial urokinase.
- Omentopexy, subtotal adrenalectomy.
- Angioplasty, excisional atherectomy, bypass surgery, and even amputation.

What are indications for surgery in PVD? (Lower extremity percutaneous transluminal angioplasty or bypass surgery)

- Incapacitating claudication in persons; interfering with work or lifestyle.
- Limb salvage in persons with limb-threatening ischemia as manifested by rest pain, non-healing ulcers, and/or infection or gangrene.
- Vasculogenic impotence.

What are anesthetic implications of PVD surgery?

Anesthesia is generally required for:

- Distal bypass procedures (infra-inguinal artery bypass, lumbar sympathectomy, venous thrombectomy or vein excision, revascularization (Tibioperoneal/saphenous vein graft or PTFE), amputation.
- Diagnostic and therapeutic sympathetic block.
- Radiofrequency thermo-coagulation of sympathetic chain.
- Spinal cord stimulation/implantation, treatment of post-amputation pain and phantom limb pain.

How do you give anaesthesia in PVD? How do you monitor intraoperatively?

- Anesthesia for the acutely ischemic lower limb is challenging because of inadequate time to evaluate and treat comorbidities and the physiological disturbances from an ischemic limb.
- Local anesthesia with invasive monitoring by an anesthesiologist is the preferred technique for embolectomy
- Revascularization may still require general anesthesia.
- Regional anesthesia can reduce postoperative thrombotic events, minimize cardiac morbidity and maintain effective postoperative analgesia.
- Peripheral nerve blocks are utilized in special situations with minimal interference with cardiovascular function.
- For infra-inguinal arterial bypass, there is evidence that regional anesthesia is superior for promoting graft survival.
- For GA gradual induction (small increments of fentanyl 1–2 mcg/kg and thiopentone 50–75 mg until asleep) is desirable. If LV function is poor, high dose fentanyl induction is preferred.
- Use warm fluids and humidified gases to prevent hypothermia.
- Essential to use perioperative fluid regimen, which reduce blood viscosity and enhance blood flow. Colloid-containing fluids are preferred to crystalloids owing to better intravascular volume supporting capacity and specific colloid-associated pharmacological effects on rheological variables.

Monitoring

- ST-segment analysis
- Arterial line ± CVP/PA catheters.
- Be careful in patient positioning and pad pressure points, eyes. Diabetic patients may be at risk of skin ischemia due to poor positioning or inadequate padding of limbs, etc.

Does regional blockade alter outcome?

Regional anesthesia does have several advantages over general anesthesia that it modifies the cardiovascular and metabolic responses to the stress of surgery and helps to diminish myocardial ischemic episodes. The sympathetic blockade and vasodilatation produced improves the graft perfusion and decreases the chances of graft failure. The circulating levels of catecholamines are also reduced, which decreases the probability of vasoconstriction. There is increased fibrinolysis too due to decrease in procoagulant factors. All these factors put together definitely improve the graft survival.

What is the role of lumbar sympathetic block in PVD? How do you perform the block?

- *Anatomy:* Sympathetic contribution to lower extremity arises from cell bodies originating in lateral horn of spinal cord (T10 – L2). This segment of sympathetic chain runs along L2, 3, 4 vertebral bodies.
- *Contraindications:* Coagulopathy, infection.
- *Position:* Lateral or prone

- *Diagnostic block/therapeutic block:* 10 mL 0.25% bupivacaine is injected at 2 sites, with 22 G needle of 15 cm length.
- *Sympathetic block success:* Temperature rise of > 20°C on ipsilateral leg, improvement in pain, motor function and color usually occurs within minutes establishing diagnosis.
- No reliable evidence to support its use in Buerger's disease, intermittent claudication, diabetic vascular disease or ischemic ulceration or gangrene.

What are the postoperative complications in PVD surgery?

- Hyper-/hypotension, hypothermia, tachycardia, hypoxemic episodes, hypercapnia, myocardial ischemia/infarction and renal functional disturbances. CHF, hypothermia,
- Graft occlusion; hypothermia causes vasoconstriction and also may limit outflow to the graft.
- Avoid overhydration; when epidural sympathectomy fades; increased risk for CHF.
- Maintain normovolemia; so that peripheral vasoconstriction does not occur.

Summary

Patients with PVD are generally associated with CAD and/or carotid artery disease. Though exercise tolerance is a useful indicator of the severity of CAD, it is almost always limited by intermittent claudication or other comorbidities. Vascular reconstructions are of prolonged duration and are associated with hypovolemia and hypothermia. Myocardial ischemia is very common and often associated with infarction.

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8 Pneumonectomy

P Ranganathan, B Trivedi

A 55-year-old male patient presents with the history of cough with mucoid expectoration and progressive weight loss since 2 years. After bronchoscopy, CT scan and biopsy, he is diagnosed to have bronchogenic carcinoma involving the left lower bronchus, extending into the left upper and left main bronchus. The patient has been smoking 15 to 20 cigarettes per day for the last 30 years. He gives a history of breathlessness since 5 to 6 years for which he is taking Salmeterol inhaler twice daily. He has a limited effort tolerance and cannot climb more than 1 flight of stairs without becoming breathless. There is no other medical history. On examination, his height is 158 cm and weight is 48 kg. He has decreased air entry over the entire left hemithorax with bronchial breath sounds and scattered wheeze. Blood investigations are normal. Chest X-ray shows left middle and lower zone opacity with left mediastinal shift. ECG shows left bundle branch block. Echocardiography shows normal biventricular function with no evidence of pulmonary hypertension. The patient is posted for a left pneumonectomy.

Describe the pre-anesthetic evaluation of a patient posted for lung resection surgery.

Pre-anesthetic evaluation should include:

- A detailed medical history for any coexisting disease. Optimal treatment and control of associated medical conditions should be achieved.
- The patient's functional capacity should be assessed.
- Since patients with lung cancer are usually smokers, a history of smoking and of symptoms suggestive of COPD should be elicited.
- The patients should be evaluated for ischemic heart disease since they tend to be chronic smokers, which predispose them to atherosclerosis. Patients with major factors for increased perioperative cardiovascular risk should undergo a preoperative cardiologic evaluation.
- Airway evaluation should be performed keeping in mind that these patients are candidates for lung isolation intra-operatively.
- Patients may receive chemotherapy preoperatively, and should be evaluated for chemotherapy related toxicity.
- Investigations should include complete blood count. This may show polycythemia due to COPD or leukocytosis, which may indicate active pulmonary infection. Sputum cultures and sensitivity studies can be used to guide appropriate antibiotic therapy. Liver and renal function should be assessed in view of age.
- The chest x-ray should be evaluated for tracheal deviation or obstruction, which could predict difficulty with intubation or ventilation, mediastinal mass, which could lead to difficulty with ventilation or superior vena cava syndrome, pleural effusions or areas of consolidation.
- Pulmonary function tests should be carried out to diagnose obstructive or restrictive abnormalities, to assess responsiveness to bronchodilators and to confirm suitability for resection.
- The ECG should be evaluated for signs of left or right heart dysfunction.
- Transthoracic echocardiography to rule out pulmonary hypertension.
- Further cardiopulmonary testing may be indicate if warranted by the history/above investigations.

Why is important to assess suitability for lung resection?

Lung resections are associated with a high risk of morbidity and mortality. Smoking leads to abnormal pulmonary

function and predisposes to both immediate perioperative complications and long-term disability following surgical resection. Loss of lung parenchyma can lead to hypoxemia. In addition, the entire pulmonary blood flow is delivered to the remaining lung, leading to pulmonary hypertension and right heart strain. This can lead to right ventricular failure. Patients need to be evaluated to see if they can withstand the loss of the resected lung without becoming pulmonary cripples.
In addition, the following points need to be kept in mind:

- Resections may be more extensive than planned.
- Handling of the lung/trickling of blood or secretions may cause postoperative dysfunction of the remaining "normal" lung.

What are the 4Ms, which need to be assessed in patients with lung cancer?

The 4 Ms, which need to be assessed in patients with lung cancer are:

- **M**ass effects: Pneumonia, SVC syndrome, nerve palsy
- **M**etabolic effects: Eaton-Lambert syndrome, hypercalcemia
- **M**etastases: to distant organs—bone, adrenal
- **M**edications: Effects of chemotherapy and radiation therapy.

Describe the preoperative respiratory assessment of this patient to assess suitability for pneumonectomy.

Three aspects of respiratory function need to be assessed preoperatively. These include:

- Lung mechanics
- Lung parenchymal function
- Cardiopulmonary interaction.

How will you assess lung function at the bedside?

Refer to Chapter on anesthetic management of a patient with COPD.

How is adequacy of lung mechanical function assessed?

The assessment of lung mechanics includes spirometry and determination of lung volumes. Traditional cut-off values for lung resection are shown in Table 8.1.

Table 8.1 Traditional cut-off values for lung resection

Test	Normal	Cut off Criteria		
		Pneumonectomy	Lobectomy	Segmental resection
MVV (L/min)	> 100	> 70	40 – 70	40
MVV (% pred)	100	> 55	> 40	> 35
FEV_1 (L)	> 2	> 1.7 – 2.1	> 1.0 – 1.2	> 0.6 – 0.9
FEV_1 (%)	> 80% FVC	> 50% FVC	> 40% FVC	> 40% FVC
$FEV_{25–75\%}$ (L)	> 2	>1.6	> 0.6 – 1.6	> 0.6

However, a more valid method of assessing suitability for lung resection is calculation of the predicted postoperative FEV_1.

How is postoperative lung function after lung resection calculated?

The lungs contain 42 sub-segments as shown in Figure 8.1.

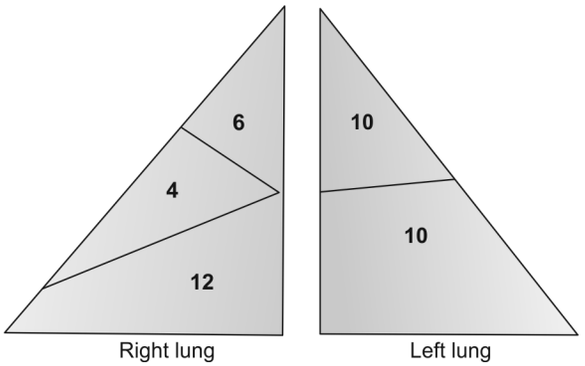


Fig. 8.1 Sub-segments of lungs

Predicted postoperative FEV_1 ($ppoFEV_1\%$) is calculated by counting the number of segments to be removed.

$PpoFEV_1\% = \text{Preoperative } FEV_1\% \times [1 - (\text{no of subsegments removed}/42)]$

For example, in a right upper lobectomy 6 segments are resected

$$\text{Therefore, } ppoFEV_1\% = \text{preoperative } FEV_1\% \times [1 - (6/42)]$$
$$= \text{preop } FEV_1 \times 36/42$$

Patients with a $ppoFEV_1$ less than 40% are at high risk for postoperative pulmonary complications.

How is lung parenchymal function assessed?

Conventionally, preoperative arterial blood gas values were used to assess patients undergoing lung resection, with $PaCO_2$ more than 45 mm Hg, PaO_2 less than 60 mm Hg and SaO2 less than 90% being considered risk factors. However, the diffusing capacity of the lung for carbon monoxide (DLCO) has been shown to be an independent predictor of poor

outcome after lung resection and is the most useful test of gas exchange.

Predicted postoperative DLCO values (ppoDLCO %) can be calculated using the same formula as for ppoFEV$_1$. ppoDLCO less than 40% is a predictor of increased respiratory and cardiac complications.

What are the cut-off values for predicted post-operative lung function?

Conventionally used cut-off values suggest that ppoFEV$_1$ less than 1 liter results in sputum retention and ppoFEV$_1$ less than 800 mL can leave the patient ventilator-dependent. A more accepted estimate of predicted postoperative lung function is that ppoFEV$_1$ less than 40% or ppoDLCO less than 40% indicates a high risk of perioperative complications including death. PpoFEV$_1$ and ppoDLCO are independent of each other and should be measured in all patients undergoing lung resection. Another index, which is used for risk stratification is the predicted postoperative product (PPP), which is a product of ppoFEV$_1$% and ppoDLCO%. A PPP less than 1650 has been shown to predict postoperative complications; however, further studies are needed to validate this.

Why is cardiopulmonary exercise testing important in these patients?

Patients presenting for lung resection usually have a long-standing history of smoking. This predisposes them to atherosclerosis and ischemic heart disease, and COPD. This in turn may lead to left and right ventricular dysfunction. These patients therefore, may be at higher risk for perioperative myocardial ischemia. In addition, the pulmonary vasculature becomes rigid due to chronic lung disease. Further reduction in the pulmonary vascular bed after lung resection puts the patient at risk for postoperative increases in pulmonary vascular pressure and right heart failure.

What cardiovascular work-up is needed?

Formal exercise testing where maximal oxygen consumption (VO2max) can be measured is the gold standard for evaluating the cardiopulmonary interaction. VO2max greater than 15 mL/kg/min is considered acceptable and these patients should tolerate pneumonectomy; less than 10 mL/kg/min deems the patient inoperable; and those with 10–15 mL/kg/min are considered high risk and require careful evaluation before surgery. However, formal exercise stress testing is expensive and time-consuming, and is only carried out in patients who have clinical or ECG features, which are suggestive of IHD.

Several alternatives have been developed as substitutes to exercise testing for pre-thoracotomy assessment.

Stair climbing: The patient is asked to climb stairs at his own pace without stopping. A flight of stairs is taken as 20 steps at 6 inches/step. Patients who can climb five flights of stairs have a VO2max over 20 mL/kg/min; those who manage less than one flight equate to less than 10 mL/kg/min. The ability to climb fewer than two flights is considered high risk.

Shuttle walk test: Patients walk between two markers, 10 meters apart. Their speed is gradually increased every minute. The inability to complete 25 shuttles suggests a VO2max less than 15 mL/kg/min.

6-minute walk test: The patient is made to walk on level ground at the maximum pace possible without discomfort. The distance covered and oxygen saturation is monitored. A 6-minute walk test distance of less than 2,000 feet correlates to a VO2max less than I5 mL/kg/min. Desaturation of over 4% from the resting value during the test is associated with an increased risk of postoperative complications. Dyspnea and fatigue scores are also assessed

This patient's pulmonary functions are as follows:

FEV_1 = 1.04 liters (51% predicted) [6% change after bronchodilator]
FVC = 1.21 liters (55% predicted) [5% change after bronchodilator]
FEV_1/FVC = 87% predicted
MVV = 54% predicted [8% change after bronchodilator]
DLCO = 50% predicted

Arterial blood gas on room air shows PO_2 of 83 mm Hg, PCO_2 of 37 mm Hg and saturation 97%, with normal pH.

Interpret these PFTs and calculate ppoFEV$_1$ and ppoDLCO after left pneumonectomy. Is this patient suitable for a left pneumonectomy?

Table 8.2 describes the changes in PFT in obstructive, restrictive and mixed defects. For more details see the chapter on Pulmonary Function Tests.

Table 8.2 Changes in pulmonary function tests

	FVC	FEV_1/FVC
Obstructive	Normal	Low
Restrictive	Low	Normal
Mixed	Low	Low

This patient's PFTs show moderate restriction with insignificant response to bronchodilator therapy. There is a decrease in diffusion capacity. ABG shows mild hypoxemia with no CO_2 retention.

After left pneumonectomy, the patient will be left with 22 out of 42 lung sub-segments.

ppoFEV$_{1\%}$ after left pneumonectomy = 51% × 22/42 = 26% which is less than the cut-off of 40%

ppoDLCO% after left pneumonectomy = 50% × 22/42 = 26% which is less than the cut-off of 40%

Based on these predicted postoperative values, the patient appears unfit for a left pneumonectomy.

What are the limitations and alternatives to this method of calculating postoperative lung function?

Calculation of ppo lung functions using the above formula assumes that the entire lung is contributing to ventilation and perfusion. However, diseased lung may be non-functional with very little blood flow. When any of the whole-lung pulmonary function values are worse than the cut-off limits, the function of each lung needs to be assessed separately. This consists of measurement of the ventilation and perfusion of each lung using radioactive isotope scanning. V/Q scanning allows detailed assessment of the functional capacity of the lung and accurate determination of which lobes or segments contribute proportionally to gas exchange before their resection.

A perfusion scan shows 13% perfusion to the left lung and 87% perfusion to the right lung. Based on these findings, is the patient suitable for a left pneumonectomy?

This patient's FEV_1 is 51%. The right lung has 87% perfusion.

The ppoFEV_1% after left pneumonectomy is

$$\frac{87}{100} \times 51 = 44\%$$

Similarly, ppoDLCO% after left pneumonectomy is 87% × 50 = 44%. Both these values are above the cut-off of 40%; therefore, this patient appears suitable for a left pneumonectomy.

What further tests can be done?

If second-phase criteria are not met, then tests are conducted to mimic postoperative conditions by temporary occlusion of the corresponding main stem bronchus or pulmonary artery. A rise in pulmonary artery pressure above 45 mm Hg, $PaCO_2$ above 60 mm Hg or a fall in PaO_2 below 45 mm Hg indicates inability to tolerate lung resection. However, these tests are rarely carried out.

Why lobectomy is considered a functional pneumonectomy?

Lobectomy should be considered a functional pneumonectomy for the following reasons:

1. During the immediate postoperative period, atelectasis and infection can worsen the function of the lung tissue remaining on the operative side.
2. Intraoperative reassessment of the disease may change the staging and may change the surgical plan from lobectomy to pneumonectomy.
3. The non-operated lung may develop postoperative functional impairment due to contamination or infection.
4. Compensatory hyperinflation of the remaining lobes of the lung, which has been resected occurs in the postoperative period. This results in ventilation-perfusion abnormalities.

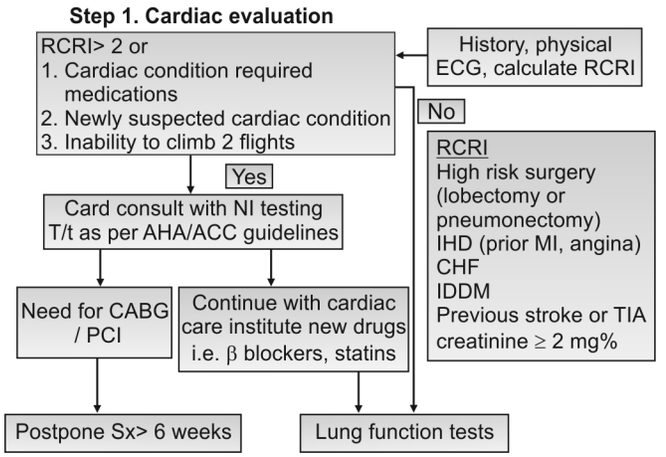
Hence, patients scheduled for lobectomy must be assessed for ability to tolerate pneumonectomy as well.

Summarize the preoperative cardiorespiratory assessment of a patient presenting for pneumonectomy.

No single test of respiratory function can be used as a method of preoperative assessment. An estimate of respiratory function in all three areas (testing of lung mechanics, parenchymal function and cardiopulmonary reserve)—the so-called "three-legged approach" should be made for each patient. Transport of oxygen to the lungs correlates with respiratory mechanics; transport into the blood depends on with parenchymal function; and transport to the tissues relies on cardiopulmonary interaction.

All patients undergoing lung resection should have spirometry and DLCO testing done to calculate predicted postoperative lung function. Assessment of effort tolerance should be done to evaluate cardiac function. Patients with borderline ppo lung functions should be considered for ventilation-perfusion scanning. Patients with a history of cardiorespiratory disease, patients with limited effort tolerance and patients with borderline ppo lung functions should undergo arterial blood gas analysis and cardiopulmonary exercise testing. A Stepwise algorithm for evaluation for lung resection is as per guidelines of European Respiratory Society (2009) is outlined in Flow chart 8.1. Step one is to evaluate cardiac function, step 2 for evaluating pulmonary function.

Flow chart 8.1 A stepwise algorithm for evaluation for lung resection



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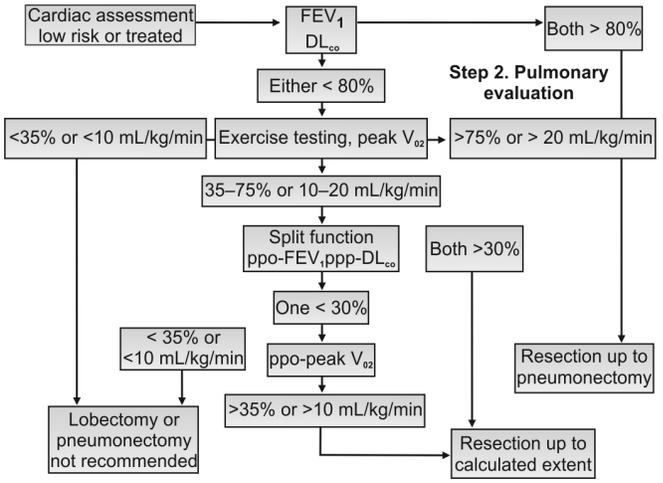

Why is preoperative respiratory optimization important in this patient?

Patients presenting for lung resection are at high risk for perioperative respiratory complications like atelectasis and pneumonia because of 3 reasons:

1. Most of these patients are smokers and have underlying pre-existing respiratory disease.
2. Handling of the lung during surgery may cause dysfunction of surrounding normal tissue.
3. If a thoracotomy approach is used, postoperative pain may interfere with the patient's ability to cough and perform spirometry.

Therefore, it is important to maximally optimize pre-operative respiratory function and minimize complications.

What is the preoperative optimization of this patient?

The five elements of the preoperative regimen are stopping of smoking, dilating airways, loosening and removing secretions, and taking measures to increase motivation and education and to facilitate postoperative care.

Cessation of smoking is the single most important intervention in the preoperative period. A study by Warner found that the incidence of postoperative pulmonary complications was highest (57%) in patients who quit smoking for less than 8 weeks; it decreased to 14.5% if cessation was more than 8 weeks. It was least in (11.9%) in those who never smoked. Current smokers (33%) also had a high risk of postoperative complications. It is hypothesized that the absence of the irritant effect of cigarette smoke in the postoperative period inhibits coughing and leads to retention of secretions and small airway obstruction, in patients who stopped smoking less than 8 weeks. Within 12 hours of cessation, the carboxyhemoglobin levels drop. Long-term cessation (more than 8 weeks) leads to improvement in mucociliary function with increased sputum clearance and also reduced airway reactivity and sputum production. The risk for patients who quit more than 6 months is the same as those who never smoked. Smoking has also been shown to delay wound healing.

Treat chest infection with appropriate antibiotics.

Bronchodilator therapy will benefit not only these patients but also those without bronchospasm; even if no improvement in FEV_1 is seen.

Steroids: Inhaled corticosteroids improve symptoms, lung function; and reduce exacerbations when FEV_1 is less than 60% of predicted value.

Many of these patients might be receiving steroids. Recommendations for perioperative management of patients on steroid are given in Table 8.3.

Table 8.3 Recommendations for perioperative management of patients on steroids

Dose	Surgery	Recommended dose
< 10 mg/day	Minor/ Moderate/ Major	Additional steroid cover not required (assume normal HPA response)
> 10 mg/day	Minor surgery	25 mg of hydrocortisone at induction and normal medications post-op
> 10 mg/day	Moderate surgery	Usual dose preoperative and 25 mg hydrocortisone IV at induction then 25 mg IV TDS for 1 then recommence preoperative dosage
> 10 mg/day	Major surgery	Usual dose preoperative and 100 mg hydrocortisone at induction then 100 mg IV TDS for 2–3 days

Patients with cor pulmonale may be receiving *diuretics and/or digitalis*. This will need to be continued perioperatively.

Hydration and mucolytic therapy will help in loosening of secretions and along with chest physiotherapy will help in clearing the chest.

Preoperative *incentive spirometry* has always been recommended for reducing POPCs though there is no supportive evidence.

What anesthetic technique would you use?

General anesthesia with controlled ventilation with thoracic epidural analgesia should be used. Intravenous induction with propofol or thiopentone can be performed. Propofol is preferred since many of these patients will have reactive airways and use of thiopentone and tracheal instrumentation in light plane can lead to bronchospasm. Since this patient has a normal airway, a non-depolarizing neuromuscular

blocking agent can be used to facilitate intubation and IPPV. Anesthesia will be maintained with Isoflurane delivered in an oxygen/air or oxygen/nitrous oxide mix. During one-lung ventilation, anesthesia can be maintained intravenously with propofol and an air/oxygen mix.

What monitors would you like to use for this patient?

Monitoring will include ECG, pulse oximetry, capnography, noninvasive blood pressure and temperature probe. Urinary catheterization is not indicated in straight-forward cases but may be needed in complicated cases, which are expected to take a longer time. An arterial cannula allows frequent monitoring of blood gases during one-lung ventilation. Invasive arterial pressure monitoring is useful in patients who are at higher cardiopulmonary risk and in those in whom the surgery is expected to be complicated. Central venous pressure monitoring might be indicated in patients with serious comorbidities for intravascular volume assessment as well as administration of vasoactive drugs.

When is a PA catheter indicated? What are the problems with PA catheter in left lateral decubitus position?

Pulmonary artery catheters usually tend to locate in the right lung. Hemodynamic measurements are most accurate when the tip of the PA catheter is situated in West zone 3 area. During a right thoracotomy with OLV, the catheter tip is usually in the nondependent lung. This lung is collapsed and not ventilated, and therefore the hemodynamic measurements may be inaccurate.

What is the plan for ventilation intraoperatively?

The patient will need lung isolation and selective ventilation of the left lung to allow collapse of the right lung and access to the bronchovascular structures.

What are the indications for one-lung ventilation?

The indications for one-lung ventilation can be classified as:

1. Isolation of one lung from other to prevent spillage or contamination
   a. Unilateral bronchopulmonary lavage in patients with pulmonary alveolar proteinosis
   b. Lung abscess
   c. Bronchiectasis
   d. Pulmonary hemorrhage.
2. Control of distribution of ventilation
   a. Bronchopleural fistula
   b. Surgical opening of major bronchus
   c. Giant unilateral cyst or bulla.
3. Surgical exposure
   a. Lung, esophageal, vascular, mediastinal, spine surgeries in the thoracic cavity.

Describe various techniques for lung isolation with advantages and disadvantages of each.

Three types of devices are available for providing one-lung ventilation during anesthesia: double-lumen endotracheal tubes (DLTs), bronchial blockers and endobronchial tubes (Table 8.4).

Table 8.4 Advantages and disadvantages of various techniques for lung isolation

	Advantages	Disadvantages
Double lumen tube	Can suction lungs independently Quality of suctioning better Can apply CPAP to non-ventilated lung more easily	Difficult to insert in distorted airway and in patients at risk of aspiration Needs change of tube if postoperative ventilation is considered – which may be risky Needs determination of appropriate size Potential for tracheo-bronchial injury
Bronchial blocker	Can be used for selective lobar blockade Can be used in tracheostomized patients Can be used in critically ill patients who are already intubated with a single-lumen tube Can be used in children and small adults, in whom the smallest DLT may be too big	Because of small lumen, lung inflates and deflates very slowly More difficult to apply CPAP to non-dependent lung Cannot be used if main stem bronchus on operative side is involved by disease
Endobronchial tube	Useful in emergencies like massive bleeding Useful in children and very small adults	Inability to ventilate or suction opposite lung If on right side, high risk of obstructing right upper lobe bronchus Difficult to negotiate into left side

How is the size of the DLT determined for each patient?

Single-use polyvinyl chloride DLTs are available in sizes 26 F, 28 F, 32 F, 35 F, 37 F, 39 F, 41 F.

An ideally placed DLT should pass easily through the glottis and should enter the intended main bronchus without causing trauma. Excessive pressures should not be needed for inflating the bronchial cuff; at the same time an air leak should be present when the bronchial cuff is deflated.

Generally 35 F and 37 F are used for small and large females respectively, and 39 F and 41 F for small and large males respectively.

Measurement of the tracheal diameter on the preoperative CT scan has been used to predict the correct size of the DLT needed for a patient (Table 8.5).

Table 8.5 Ratio of DLT size with measurement of tracheal width

Measured tracheal width (mm)	DLT size (Fr)
≥ 18 mm	41
≥ 16 mm	39
≥ 15 mm	37
≥ 14 mm	35

It has also been found that tracheobronchial dimensions correlate with height. Therefore, the height of the patient can be used to calculate the size of DLT (Table 8.6).

Table 8.6 Ratio of DLT size with height of the patient

Sex	Height (cm)	DLT size (Fr)
Male	> 170	41
Male	< 170	39
Female	> 160	37
Female	< 160	35

For females < 152 cm, consider 32 F. For males < 160 cm, consider 37 Fr.

Why left-sided DLTs are preferable to right sided ones?

The right upper lobe bronchus takes off from the right main bronchus 0.5 to 1 cm below the carina. Therefore, when a right sided DLT is placed, there are high chances that the right upper lobe bronchus may be occluded, and care has to be taken to align the slot for the right upper lobe bronchus, with the opening of the bronchus.

The left mainstem bronchus is much longer than the right one (50 mm as compared to 20 mm). Therefore, the margin of safety while positioning a left sided DLT is more.

Are there any specific indications for right sided DLTs?

Specific conditions where a right-sided DLT would be indicated are

- Exophytic tumor that compresses the entrance of left bronchus
- Intraluminal tumor near entrance of left bronchus
- Left-sided tracheo-bronchial disruption
- Left bronchus stent
- Descending thoracic aortic aneurysm compressing the entrance of left bronchus
- Sharp angle of the entrance of left bronchus.

You decide to insert a 39 F right DLT in this patient. Describe the technique of insertion and confirmation.

The DLT is passed with the distal curvature initially concave anteriorly. After the tube tip passes the larynx the stylet is removed and the tube is rotated 90 degrees to the right (so that the distal curve is now concave toward the right side and the proximal curve is concave anteriorly) to allow endobronchial intubation on the right side. After rotation, the tube is advanced until most of it is inserted and resistance to further insertion is felt. The average depth of insertion for both male and female patients 170 cm tall is 29 cm, and for each 10-cm increase or decrease in height, the average placement depth is increased or decreased by 1 cm.

After insertion, the tracheal cuff should be inflated, and both lungs should be auscultated for equal ventilation. If breath sounds are not equal, the tube is probably too far in, and the tracheal lumen opening is in a main-stem bronchus. The tube should then be withdrawn until breath sounds are bilaterally equal.

The second step is to clamp the tracheal lumen (left side in the case of the right-sided tube) and open the tracheal cap. Then, the bronchial cuff is slowly inflated to prevent an air leak from the bronchial lumen around the bronchial cuff into the tracheal lumen. This ensures that the bronchial cuff is not excessively inflated. Inflation of the bronchial cuff usually requires 2–3 mL of air.

The third step is to remove the clamp and check that both lungs are ventilated with both cuffs inflated. This ensures the bronchial cuff is not obstructing the contralateral hemithorax, either totally or partially.

The final step is to clamp each side selectively and watch for absence of movement and breath sounds on the ipsilateral (clamped) side; the ventilated side should have clear breath sounds, chest movement that feels compliant and peak airway pressures should not be excessively high.

The pediatric flexible fiber optic bronchoscope is the gold standard for confirmation of DLT position. When using a right-sided DLT, the bronchoscope is usually first introduced through the tracheal lumen. The carina is visualized, but no bronchial cuff herniation should be seen. The upper surface of the blue endobronchial cuff should be just below the tracheal carina. The bronchoscope should then be passed through the bronchial lumen, and the right middle and lower lobe bronchial orifices should be identified. The orifice of the right upper lobe bronchus should be identified when the bronchoscope is passed through the right upper lobe ventilating slot of the DLT.

Can a bronchial blocker be used in this patient? What precautions are needed during surgery?

This patient will need a left sided bronchial blocker. Since the left main bronchus is involved by disease, it will not be able to place the blocker. There are high chances of malposition, trauma to the bronchus, and dislodgement and distal embolization of tumor.

When a bronchial blocker is used for a pneumonectomy, care should be taken to withdraw the blocker prior to stapling the bronchus.

The surgery will be done in lateral decubitus position. Describe how the lateral decubitus position is given and what are the problems with this position.

Following confirmation of the side of surgery, the patient is turned into the lateral position. The lower shoulder is pulled through anteriorly. The lower arm is flexed at the elbow and tucked under the pillow supporting the head. The upper arm is extended and placed over the head on an arm rest.

The lower leg is slightly flexed at the hip and knee while the upper leg is kept relatively straight. A pillow is placed between the legs. Chest and pelvic supports are used for further stability. All vulnerable areas padded. A small roll is placed under the dependent thorax to prevent pressure on the dependent arm neurovascular bundle. This padding should not be allowed to migrate into the axilla where it can worsen the pressure on the neurovascular bundle. The cervical spine should be aligned with the thoracolumbar spine, and there should be no pressure on the lower eye.

The common problems with this position are:

1. Ischemia, nerve damage, or compartment syndrome to the dependent arm
2. Postoperative shoulder discomfort
3. Lateral angulation of the neck leading to jugular venous obstruction
4. Hyperextension of the non-dependent arm leading to traction or compression of the brachial and axillary neurovascular bundles.

Describe the physiology of ventilation and perfusion in the lateral decubitus position.

The effect of gravity, along with re-distribution of ventilation and perfusion leads to an increase in pulmonary arterio-venous shunting of blood from 5% in the supine position to 10 to 15% in the lateral position. This is offset to some extent by the development of hypoxic pulmonary vasoconstriction.

Describe the management of one-lung ventilation[2]

Before Instituting OLV

Non-dependent lung:

The composition of the gas mixture in the non-dependent lung will affect the rate, at which the lung collapses. The presence of nitrogen, which has low blood gas solubility will delay collapse. This can be avoided by ventilating the lung with 100% oxygen prior to instituting OLV.

Dependent lung:

To prevent atelectasis of the dependent lung, a recruitment maneuver should be applied immediately before starting OLV.

During OLV

- Maintain tidal volume 5 to 6 mL/kg
  - Maintain peak airway pressure < 35 cm H_2O
  - Maintain plateau pressure < 25 cm H_2O
- Positive end-expiratory pressure of 5 cm H_2O to ventilated lung
  - Avoid in patients with COPD
  - Respiratory rate of to maintain normal $PaCO_2$
- Volume or pressure controlled mode.

After switching to one-lung ventilation, the patient's saturation drops to 90%. What is the management of hypoxemia during one-lung ventilation?

If hypoxemia occurs during OLV, the following protocol should be followed:

- For sudden or severe desaturation:
  - Convert to two-lung ventilation
- For gradual desaturation:
  1. Increase FiO_2 to 1.0
  2. The position of the DLT should be rechecked using a fiberoptic bronchoscope. If a left thoracotomy is being performed using a right-sided DLT, ventilation to the right upper lobe should be ensured
  3. The hemodynamic status of the patient should be optimized
  4. Recruitment of the ventilated lung
  5. PEEP of 5 to 10 cm H_2O can be applied to the dependent lung
  6. CPAP of 1 to 2 cm H_2O should be applied to the non-dependent lung, after a recruitment maneuver.
  7. Intermittent two-lung ventilation can be re-instituted after discussion with the surgeon
  8. Partial ventilation of the non-ventilated lung using either low flow oxygen insufflations or high frequency ventilation
  9. If a pneumonectomy is being performed, ligation of the pulmonary artery can be carried out to completely eliminate the shunt.

What is the plan for fluid management and why is this so important in this patient?

Fluid restriction is generally advocated in lung resections. Maintenance fluid should be given at a rate of 2 mL/kg/hr. This is particularly important in right pneumonectomies where a significant portion of the pulmonary vascular bed is resected. The reasons for this are:

1. Third spacing is not excessive in lung surgeries
2. The dependent lung has a tendency for high capillary hydrostatic pressures
3. Postoperative pulmonary edema may occur if the remaining pulmonary vasculature cannot tolerate the entire cardiac output. This is true, especially in patients with long-standing respiratory disease whose pulmonary vasculature is less distensible
4. Intraoperative lung manipulation and collapse may impair lymphatic drainage.

It is recommended that the total positive fluid balance in the first 24 hours should not exceed 20 mL/kg.[2]

Will this patient need ventilation post-operatively?

If the patient is awake, warm and pain-free, it is advisable to extubate the patient in the operation theater. Positive pressure ventilation is best avoided after lung resection to prevent tension on the suture lines and chances of a stump blow-out. If ventilatory support is needed, use of pressure support ventilation is more appropriate

What are the available techniques for pain relief in this patient?

Thoracic epidural analgesia is considered the gold standard for post-thoracotomy analgesia. The epidural is most effective when placed at the vertebral level corresponding with the dermatomes of the surgical incision. Local anesthetic solutions may be infused continuously or via a patient-controlled device. Side effects of the local anesthetic may be reduced by adding opioids to the solution. Other options can be considered when thoracic epidural analgesia is not feasible or is contraindicated. These include:

Parenteral opioids: Can provide adequate analgesia. The side effects include respiratory depression, sedation and loss of cough. Patient-controlled analgesia (PCA) devices are used to deliver opioids in a more patient-specific way, and are superior to intravenous boluses or intramuscular injections.

Paravertebral blocks: A catheter may be placed in the paravertebral space under direct vision by the surgeon, or percutaneously by the anesthetist, and plain local anesthetic solution infused continuously, blocking intercostal nerves above and below as they pass extrapleurally through the space. The main advantage over thoracic epidurals is that the block is unilateral, thus reducing the extent of related side effects. However, large volumes of local anesthetic solution are required, and the risk of local anesthetic toxicity is increased.

Intrathecal opioids: Preservative-free morphine may be injected via a spinal needle into the subarachnoid space at lumbar spine level. As it is less lipid soluble than fentanyl or diamorphine there is rostral spread in the CSF, and a single-shot dose of 5–20 µg/kg may provide analgesia for thoracic surgery for up to 24 hours postoperatively. Delayed respiratory depression has been reported.

Intercostal nerve blocks: Local anesthetic solutions may be injected into the intercostal spaces above and below the surgical incision. The main disadvantage is the short-acting nature of this technique. It may be used to supplement other techniques or provide rapid-onset relief until another technique has been established.

A summary of the various techniques available for post-thoracotomy pain relief is available from the PROSPECT (procedure specific postoperative pain management) guidelines.

What postoperative complications can occur after pneumonectomy?

1. Cardiovascular:
   a. Arrhythmias
   b. Right ventricular failure
   c. Cardiac herniation
   d. Hemorrhage
2. Pulmonary
   a. Pulmonary edema
   b. Respiratory insufficiency
   c. Pulmonary torsion
3. Pneumonectomy space
   a. Bronchopleural fistula
   b. Empyema
4. Neurological
   a. Recurrent laryngeal, vagus or phrenic nerve injury.

One week after surgery, this patient presents with dyspnea, subcutaneous emphysema, contralateral deviation of the trachea and persistent air leak from the chest tube. Bronchoscopy shows a bronchopleural fistula. This patient is posted for surgical closure of the fistula with flap cover. What are the goals of anesthetic management?

The goals of anesthetic management include maintenance of adequate ventilation and oxygenation and protection of the contralateral lung from spillage of purulent material.

Anesthetic strategies for induction of anesthesia depend on the severity of the bronchopleural fistula and the presence of infection.

1. A chest drain is necessary to prevent accumulation of escaping air leading to a tension pneumothorax and to drain purulent contents to minimize contamination
2. Positive pressure ventilation may be ineffective because of loss of tidal volume through the fistula
3. In addition, positive pressure ventilation carries the risk of contaminating the noninfected lung.

The options for induction of anesthesia include:

- Spontaneous ventilation and induction using a volatile anesthetic (sevoflurane), and intubation with a double lumen tube. The patient should be positioned head up with the bad side down to minimize contamination of the good lung.
- Rapid sequence induction to minimize the time before tracheal intubation and lung isolation.
- Awake sedated intubation in patients at increased risk for aspiration of gastric contents caused by full stomach or bronchopleuroenteric fistula.

Once the tube has been positioned, the healthy lung should be isolated immediately and the head rose slightly to decrease the likelihood of contamination. In patients with a small chronic bronchopleural fistula with minimal air leak and no associated infection or empyema, a standard endotracheal tube can be used safely. Anesthesia is maintained by IPPV delivered to the healthy lung. If the affected lung needs to be ventilated, low tidal volume ventilation or CPAP or high-frequency jet ventilation may be tried. Emergence should be smooth with avoidance of high airway pressures. Pressure support ventilation may be preferred if the patient needs postoperative ventilatory support.

Describe your anesthesia plan if this patient with bronchoplueral fistula requires anesthesia for insertion of an intercostal drain.

Usually these patients will be unstable (hypotensive, hypoxic), uncooperative and/or combative. It is important to maintain spontaneous ventilation at all times as positive pressure ventilation is associated with volume loss during tidal ventilation. If possible, patient should be positioned with back-up and procedure carried out under local anesthesia. If this is not possible patient may be given small doses of ketamine (1–1.5 mg/kg) while preserving spontaneous breathing. This will ensure hemodynamic stability and preservation of reflexes.

Suggested Reading

1. American Thoracic Society. ATS statement: guidelines for the six-minute walk test. Am J Respir Crit Care Med. 2002;166: 111–7.
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2. Walker C. Analgesia after thoracotomy. Anaesthesia and Intensive Care Medicine. 2005;6:418–20.

9 Bronchiectasis with Lung Abscess

P Ranganathan, A Kothekar

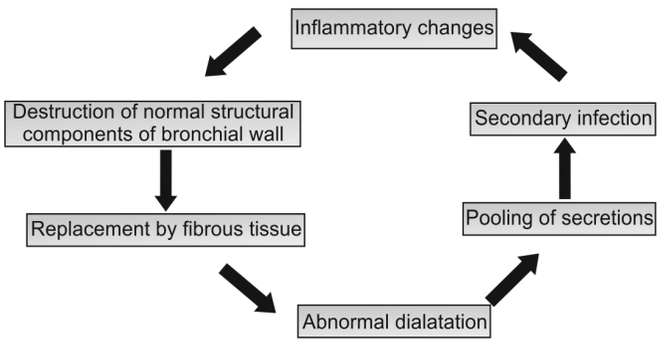
A 32-year-old woman presented with the history of fever, cough with purulent sputum and progressively worsening dyspnea since 2 months. She has history of pulmonary tuberculosis 6 years ago, for which anti-tubercular therapy was taken. Subsequently, she was diagnosed to have left lower lobe bronchiectasis. She was treated for left lower lobe pneumonia 2 months ago. She has no other medical illnesses. On examination, her height was 150 cm and weight was 56 kg. She has clubbing. There are bronchial breath sounds on the left side with basal coarse crepitations. This patient is posted for left lower lobectomy.

What is bronchiectasis?

Bronchiectasis is defined as abnormal and irreversible dilatation of part of the bronchial tree, usually at the level of the segmental or subsegmental bronchi.

What are the pathological changes in bronchiectasis?

Flow chart 9.1 Pathological changes in bronchiectasis



What is the etiology of bronchiectasis?

Bronchiectasis could be due to:

1. Infectious causes:
   Pneumonia due to
   a. Bacterial infections, e.g. *Haemophilus influenzae, Mycobacterium tuberculosis, Mycobacterium avium* complex, *Staphylococcus* aureus and anaerobes
   b. Viral infections, e.g. Adenovirus and influenza virus

Predisposing factors include:

- Immunosuppression, e.g. HIV infection or steroid therapy
- Disorders of ciliary motility—Kartagener's syndrome
- Disorders of mucus secretion—cystic fibrosis

2. Non-infectious causes:
   a. Aspiration pneumonia
   b. Airway obstruction
   c. Immune reactions—especially after allergic broncho-pulmonary aspergillosis.

What are clinical features of bronchiectasis?

Symptoms

- Persistent or recurrent productive cough
- Purulent sputum
- Frequent respiratory tract infections
- Hemoptysis
- Nonspecific symptoms like fatigue, weight loss.

Signs

- Variable auscultatory findings like crepitations, wheeze, and bronchial breath sounds
- Clubbing
- Long-standing cases may have signs of cor pulmonale and right ventricular failure.

What investigations would you like to order for this patient?

CBC: There may be raised WBC count. Neutrophil predominance if bacterial infection, lymphocytic predominance if tuberculous. Anemia may be present in view of chronic illness.

LFT, RFT: In view of major surgery and chronic illness.

CXR: It is done to check extent of bronchiectasis, rule out cavitary lesion, rule out relapsed TB, rule out pleural effusion, check position of trachea and bronchi in view of possible fibrosis with shift of trachea—since patient is presenting for lobectomy and will need lung isolation. Not needed if CT thorax done.

CT Thorax: To check the extent of bronchiectasis and lung abscess. When CT chest is done, chest X-ray is not required since both convey the same information. Measuring diameter of trachea and bronchi may be useful to decide size of double-lumen tube.

ECG: To check for signs of pulmonary hypertension, which include presence of tall peaked P wave (P pulmonale), right ventricular hypertrophy, RV strain pattern and RBBB.

PFT (Spirometry and DLCO): To document severity of dysfunction, to check for response to bronchodilator therapy and to assess suitability for lung resection.

Room air SPO_2: To check for baseline hypoxemia.

Arterial Blood Gas: For hypoxia. High PCO_2 along with raised HCO_3 and normal pH suggest coexisting COPD.

2 D Echocardiography: To measure pulmonary artery pressure to diagnose pulmonary hypertension and right ventricular size (hypertrophy in long standing pulmonary hypertension)
Sputum, gram stain, culture and sensitivity: To identify infectious organisms and to guide appropriate antibiotic therapy.

All blood investigations and a 12-lead ECG were within normal limits. CT of the chest revealed fibrocavitary and bronchiectatic changes in the left lower lobe with suspicion of a left lower lobe abscess. The right lung was relatively normal. Spirometry showed the following values. Interpret these values. Will this patient benefit from preoperative bronchodilator (BD) therapy?

Table 9.1 Spirometry values of the patient

Parameter	Predicted	Pre -BD measured	Pre BD % predicted	Post BD measured	Post BD % predicted	% Change
FVC (L)	3.57	2.96	83	3.38	95	14
FEV1 (L)	2.76	1.41	51	1.74	63	12
FEV1/FVC	77		48		46	
FEF 25–75% (L/sec)	2.67	0.31	11	0.27	10	-12
MVV (L)	110	55	50			

Spirometry shows a reduction in FEV_1, though FVC is normal. FEV_1/FVC is reduced. This suggests moderate obstructive pattern. Post-bronchodilator, there is an improvement in FEV1 by 330 ml, which is about 12% of the baseline value. Changes in FEV_1 by more than 12% or 200 mL are taken as indicators of good reversibility. Therefore, this patient will benefit from preoperative bronchodilator therapy. Since spirometry values depend on patient performance, they may not be representative of true underlying state when patient's efforts are suboptimal. Therefore, patients who do not appear to have good reversibility on spirometry may also benefit from perioperative bronchodilator therapy, if it is clinically indicated.

What are the preoperative preparations of a patient with bronchiectasis presenting for surgery?

The preoperative preparations consists of:

- Cessation of smoking (if patient is smoker). Refer to chapter on COPD for further discussion on this issue.
- Culture specific (IV or oral) antibiotics. *P. aeruginosa* infection can cause rapid deterioration of pulmonary function. The role of inhaled antibiotics is not well established.
- Bronchodilators in individual cases.
- Inhalational corticosteroid therapy is hypothesized to benefit the inflammatory component of bronchiectasis.
- Mucolytic agents for patients with inspissated secretions- The role of acetylcysteine as part of preoperative therapy is controversial; though it thins secretions, it does not appear to improve clinical end points.
- Systemic and topical hydration plays a very important role.
- Preoperative physiotherapy—postural chest drainage, percussion, vibration.
- Intravenous immunoglobulin (IVIG) for hypogammaglobulinemia.
- Vaccination schedule as in COPD patients, e.g. influenza vaccine, pneumococcal vaccine.

Describe the role of preoperative physiotherapy.

Preoperative chest physiotherapy has been shown to increase the amount of expectorated sputum and benefit patients who produce over 20 to 30 mL of mucus daily, although there is no significant effect on FEV_1.

Bronchiectasis is associated with damage of bronchial structure as a result of which there is retention and stagnation of secretions, which get infected subsequently. Bronchiectasis in the lower lobes of the lung leads to the formation of a pool of infected secretions, which can spill over and infect the other healthy parts of the lungs. The aim of postural drainage is to empty this pool of secretions by gravity-assisted drainage. The secretions are drained into the larger airways from where they can be coughed out by the patient. There are various positions for postural drainage depending on the site of the affected bronchus shown in Figure 9.1.

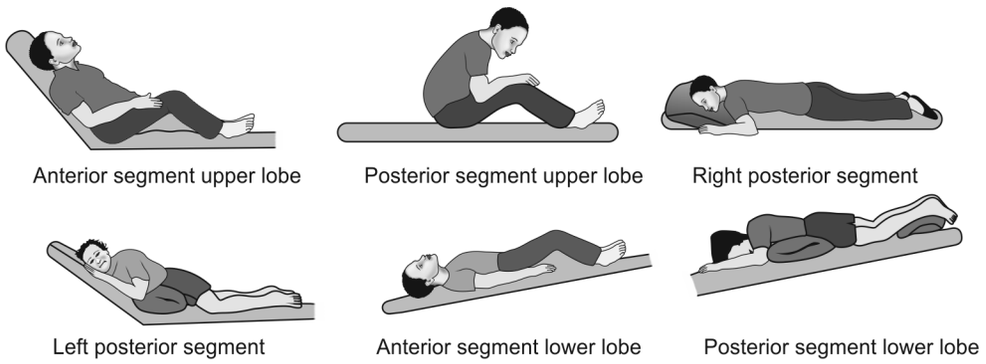


Fig. 9.1 Different positions for postural drainage

At what stage of optimization can the patient be considered for surgery?

Patients can be considered for surgery if:

- Unilateral or localized disease
- The volume of sputum decreases to less than 40 mL per day
- The secretions are no longer purulent
- The secretions are not foul-smelling.

What premedication would you like for this patient?

Anticholinergics cause drying of secretions and are best avoided. Sedatives and respiratory depressants should be used cautiously as loss of cough reflex might lead to aspiration of infected contents from affected lobe to other healthy lobe/lung.

Describe the technique of anesthesia you would use for this patient.

Preferred technique of anesthesia would be general anesthesia with thoracic epidural analgesia for perioperative analgesia. Lung isolation can be achieved with the use of either a bronchial blocker or a double-lumen tube.

Why is postoperative analgesia important?

Adequate postoperative analgesia is important to allow early physiotherapy and ambulation, so that the patient can perform spirometry and cough out secretions. Epidural analgesia will help to avoid complications of systemic opioids like cough suppression and respiratory depression

Describe the technique of general anesthesia in this patient.

It is important to avoid spillage of pus into the contralateral lung, which can occur during induction of anaesthesia.

To prevent, this one can induce these patients in secretion retaining positions so that infected secretions remain in affected lobes (lobe containing the abscess is in the most dependent position relative to the rest of the tracheobronchial tree). In this case, with a left lower lobe abscess, induction should be carried out in the left lateral semi-sitting position. A rapid sequence induction is important to avoid prolonged positive pressure ventilation, which can again increase chances of spillage (there is no need for cricoid pressure here as we are not trying to prevent gastric aspiration but a translobar/transpulmonary aspiration of secretions). Adequate preoxygenation is necessary to prevent hypoxemia during induction. One can intubate using a single lumen tube followed by lung isolation with a bronchial blocker (BB). Alternatively, lung separation can be achieved using a double-lumen tube. The airway and pre-operative imaging should be carefully assessed to rule out anomalies, which can interfere with placement of a double lumen tube (DLT) or BB. Alternatively lung isolation can be achieved while patient is awake, especially in those situations where amount of secretion continues to be high in spite of all efforts. Bronchial blockers may be preferable because they can be used (under FOB guidance) for selective lobe isolation when patient is awake and thus will prevent contamination

of the remaining healthy lobes of the affected lung during induction. Similarly placement of DLT can be done in awaken patient after giving topical anesthesia to upper airway.

Although ventilating the healthy lung care should be taken to use low tidal volume to prevent volutrauma. Oxygen supplementation via CPAP may be applied to the infected lung to decrease shunt in case of hypoxemia.

Closed circuit gas delivery system provides humidified gases but care should be taken to use HME filters while using semi-open or open circuits to prevent drying of secretions. Frequent suctioning should be carried out, using separate catheters for infected and non-infected areas. Both rigid and fiber-optic bronchoscopes should be readily available for suctioning of the airway. Intraoperative sputum samples should be collected for culture and antibiotic sensitivity.

What monitors would you like to use for this patient?

Monitoring should include ECG, pulse oximetry, non-invasive blood pressure monitoring, capnography, urinary catheter (optional) and temperature probe. An invasive arterial line may be useful for frequent blood gas monitoring during one-lung ventilation.

What is the postoperative management of this patient?

The patient can be extubated on table if she is warm, awake and comfortable. Early physiotherapy should be instituted. Bronchodilator therapy should be continued in the immediate postoperative period. Supplemental humidified oxygen should be continued for up to 48 hours after surgery.

What are postoperative complications in these patients?

- Bronchopleural fistula and prolonged air leak
- Difficulty in weaning from ventilator
- Empyema
- Arrhythmias
- Atelectasis
- Pneumonia.

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10 Chronic Obstructive Pulmonary Disease

A Kulkarni, M Desai, A Chatterjee

A 62-year-old, 65 kg male patient, with a peri-ampullary carcinoma pancreas, is posted for Pancreatico-duodenectomy. He presents with cough and yellowish mucoid expectoration. He can climb 2 flights of stairs with difficulty. He is a chronic smoker who has smoked 20 packs/day for 30 years, still smokes around 10 cigarettes a day since diagnosis. One year back, he was admitted to a nursing home with dyspnea and cough with expectoration. He was treated with Salbutamol and Ipratropium puffs, Budesonide inhaler and was given oxygen therapy. His examination reveals grade II clubbing, occasional wheeze and cardiovascular system examination is unremarkable.

What is chronic obstructive pulmonary disease?

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease, which incorporates three disorders: emphysema, peripheral airway disease, and chronic bronchitis. The patient may have one or all of these conditions but the dominant clinical feature is airflow limitation that is not fully reversible.

The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles. Poorly reversible airflow limitation associated with bronchiectasis, cystic fibrosis and fibrosis due to tuberculosis are not included in the definition of COPD.

Chronic bronchitis is defined clinically as chronic productive cough for 3 months in each of 2 successive years in a patient; in whom other causes of productive chronic cough have been excluded.

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

The important causes of COPD are cigarette smoking, air pollution, occupational exposure to dusts especially in coal mining, gold mining, and the textile industry; and alpha-1 antitrypsin deficiency. In rural India, cooking on coal fire is the most common cause of COPD in females.

How do you diagnose COPD?

Clinical diagnosis of COPD is considered in a patient who has dyspnea, chronic cough or sputum production, and/or history of exposure to risk factor for disease. Presence of post-bronchodilator $FEV_1/FVC < 0.7$ on spirometry; indicating persistent airflow limitation; is required to confirm the diagnosis in this clinical context.

What are the mechanisms underlying airflow limitation in COPD?

The hallmark of COPD is chronic airflow limitation. This is caused by a combination of inflammation and narrowing (obstructive bronchiolitis) of the small airways and destruction of the lung parenchyma. There is loss of alveolar attachments to the small airways due to inflammation with consequent decrease in the elastic recoil of the lungs. This leads to airway collapse during early expiration and thus airflow limitation. It occurs particularly in emphysematous patients, which results in marked dyspnea on exertion.

How do you differentiate between COPD and Asthma?

Asthma is a disease characterized by chronic airway inflammation, reversible expiratory airflow obstruction in response to various stimuli, and bronchial hyperreactivity.

Asthma is an episodic disease with acute exacerbations interspersed with symptom-free periods. It is characterized by mucus production and bronchoconstriction.

Table 10.1 Differences between chronic obstructive pulmonary disease and asthma

COPD	Asthma
COPD occurs later in life (middle age)	Asthma patients present early in life (often in childhood)
Associated with prolonged smoking, exposure to certain chemical dust or biomass fuel	Often a history of allergy, rhinitis and/or eczema is present
It is slowly progressive	Symptoms vary from day to day and peaking in the night or early morning
Airways as well as parenchyma are affected in COPD	Only the airways are affected in asthma
Airflow limitation in these patients is largely irreversible as it is structural in nature	Airflow limitation is largely reversible. Airway hyperresponsiveness is a hallmark of asthma
Inflammation is neutrophilic and CD8-driven in COPD, therefore, inhaled steroids are largely ineffective	Inflammation is predominantly eosinophilic and CD-4 driven, therefore, inhaled steroids are effective

How is dyspnea graded in COPD patients?

According to Medical Research Council (MRC) questionnaire, this patient has grade 1 dyspnea. The MRC grades dyspnea is shown in Table 10.2.

Table 10.2 The Medical Research Council grading for dyspnea

Description	Grade	Degree
Only gets breathless on strenuous exercise	0	None
Shortness of breath when hurrying on the level/walking up slight hill	1	Mild
Walks more slowly than people of the same age on the level because of breathlessness or has to stop for breath walking at own pace on level	2	Moderate
Stops for breath after walking about 100 m after few minutes on the level	3	Severe
Too breathlessness to leave the house, to dress and undress	4	Very severe

How will you investigate this patient? When will you ask for echocardiographic evaluation of a COPD patients?

A patient with COPD, commonly has various extra-pulmonary comorbidities, such as nutritional abnormalities resulting in weight loss and skeletal muscle dysfunction. They are at an increased risk of myocardial infarction, angina, osteoporosis, bone fractures, respiratory infection, depression, diabetes, sleep-disorders, anemia and glaucoma. The long standing COPD ultimately results in pulmonary hypertension and right ventricular hypertrophy leading to cor pulmonale. The investigations are directed at detecting presence of these comorbidities, which will help in preoperative optimization of the patient.

Complete blood count may reveal either anemia (nutritional deficiency) or high hemoglobin (carboxyhemoglobin may be present) and raised white cell count in presence of active infection.

Depending on severity and duration of COPD, chest X-ray may show flattening of diaphragm, horizontal ribs and hyperinflated lungs and tear drop or tubular heart. Spirometry frequently reveals reduced FEV_1 and FEV_1/FVC ratio with minimal or no improvement after bronchodilators. Electrocardiogram may show signs of right ventricular hypertrophy with right axis deviation and/or and right atrial hypertrophy and dilatation (P Pulmonale).

Nocturnal desaturation and frequent exacerbations of COPD lead to chronic hypoxia, which can result in right ventricular dysfunction. In addition to increasing severity of COPD, chronic hypoxia causing polycythemia together contribute to pulmonary hypertension (pulmonary artery pressure > 20 mm Hg) and right ventricular dysfunction. Routine echocardiographic and Doppler screening for pulmonary hypertension or right ventricular dysfunction in patients with COPD is not currently recommended by the American Thoracic Society. Echocardiography will be indicated in patients with COPD who have fatigue, dyspnea that is disproportionate to the severity of COPD and history of paroxysmal nocturnal dyspnea. A 2-D echocardiography will reveal increased pulmonary artery pressures, dilation and/or hypertrophy of right ventricle. Left ventricular dysfunction may be evident if associated ischemic heart disease is present. Arterial blood gas analysis will show a normal or near normal pH, borderline PO_2 and normal or raised CO_2 and HCO_3.

What is the spirometric classification of COPD?

According to Global initiative for chronic obstructive lung disease, COPD severity grading is shown in Table 10.3.

Table 10.3 The global initiative for chronic obstructive lung disease grading for COPD severity

Stage	Severity	Spirometry	
		FEV_1/FVC*	FEV_1 % predicted
Stage I	Mild	< 0.70	> 80
Stage II	Moderate	< 0.70	50–80
Stage III	Severe	< 0.70	30–50
Stage IV	Very Severe	< 0.70	< 30

*Postbronchodilator

Can we predict postoperative pulmonary complications (POPC)? When is patient deemed unfit for anesthesia and surgery?

Unlike in pulmonary resection, there is no cut-off value of FEV_1 or any other spirometric index to consider these patients unsuitable for surgery. Many studies have attempted to predict POPC, though not specifically in COPD patients.

The utility of spirometry and blood gas analysis in predicting risk of (POPC) is questionable. Various studies and systematic reviews have shown that; age > 65 years, smoking 40 pack/year or more , FEV_1 < 1 L/min, FVC < 1.5 L/min, PO_2 < 75 mm Hg and PCO_2 > 45 mm Hg indicate increased risk of POPCs. But the most significant factors are abnormal clinical findings on pulmonary examination, presence of preoperative respiratory symptoms (cough, dyspnea, excessive sputum production, chest pain and wheezing) and type of surgery (upper abdominal). Different authors suggest different criteria to predict risks of POPCs. Commonly used are:

A. Nunn and Milledge criteria:
   a. FEV_1 < 1L, normal PaO_2 $PaCO_2$. Low risk of POPC
   b. FEV_1 < 1L, low PaO_2 and normal $PaCO_2$ Patient will need prolonged O_2 supplementation
   c. FEV_1 < 1L, low PaO_2, and high $PaCO_2$ Suggests patient may need postoperative ventilation.

B. Based on spirometry:
   a. Predicted FVC < 50%
   b. Predicted FEV_1 < 50% or < 2 L
   c. Predicted MVV < 50% or < 50 L/min
   d. Predicted DLCO < 50% predicted
   e. Predicted RV/TLC > 50%

C. Based on patient criteria and type of surgery:
   a. Patient dependent factors: Current smoker, reduced health status (ASA grade > 2), old age (> 70 years in COPD patients), COPD with exercise intolerance
   b. Surgery dependent factors: Abdominal surgery (open > minimal invasive), thoracic surgery, long duration of anesthesia (> 4 hrs), general anesthesia (vs regional anesthesia).

Also it is necessary to keep in mind that these patients are also more prone to develop exacerbation of right ventricular dysfunction and failure due to intraoperative events, such as acidosis, hypercarbia, hypoxia and fluid overload.

A recent study found that these patients were more likely to develop POPC if they:

- Underwent upper abdominal (vs lower abdominal) surgery
- Had emergency (vs elective) surgery
- Had nasogastric tube present
- Underwent reoperation during same admission.

Describe the PFTs commonly done at the bedside and their implications.

The cough test: It is performed by asking the patient to take a deep inspiration and cough once. Test is positive if the first cough leads to recurrent coughing, which is suggestive of underlying bronchitis.

The wheeze test: The patient is asked to take five deep inspirations/expirations; the patient is then auscultated between the shoulder blades posteriorly to determine the presence or absence of wheezing.

Maximum laryngeal height: The distance between the top of the thyroid cartilage and the suprasternal notch at the end of expiration. If it is less than 4 cm, it is abnormal. It is an accurate sign of obstructive airways disease compared to pulmonary function tests.

Forced expiratory time (FET): Place the bell of the stethoscope over the trachea in the suprasternal notch and set the stopwatch to zero. Instruct the patient to take in the deepest breath possible and then to blow it all out as fast as possible. Start the stopwatch as the patient begins to exhale, stop it as soon as audible expiration is no longer heard. An FET more than 6 seconds indicates severe expiratory airflow obstruction with $\%FEV_1$ < 50%. Three trials are done and the results are averaged. This clinically measured forced expiratory time correlates well with the forced expiratory time measured by spirometry.

Sabrasez breath-holding time (BHT): Ask patient to take a deep breath and hold his breath as long as possible. Place a stethoscope over the trachea to identify early expiration. A BHT more than 40 seconds is normal. A BHT between 20 to 30 seconds indicates compromised cardiopulmonary reserve. BHT less than 20 seconds indicates very poor cardiopulmonary reserve.

Snider's match test: The test is used to measure the patient's maximum breathing capacity (MBC). Place a lighted match stick at varying distances from the patient's mouth. Instruct the patient to sit, keep his mouth open and blow (without pursing the lips) the candle off. Ability to blow the candle off at 22 cm from the mouth indicates MBC more than 150 L/min (MBC less than 100 L/min at 15 cm and MBC less than 50 L/min at 7.5 cm). Patients with moderate to severe COPD have great difficulty with this test. Some patients require multiple trials to huff out the match at 15 cm, and some with severe disease are unable to accomplish this. Need for use of therapeutic oxygen in patients with COPD is a contraindication to this test.

Single breath count test: The patient is asked to count out loud numbers from 1 onwards after a maximal inspiration.

Individuals with normal respiratory function can count to 50 or more. A single-breath count of less than 15 indicates severe impairment of vital capacity (VC).

Peak expiratory flow rate measurement: The use of peak expiratory flow (PEF) rate measurements has been advocated in the management of asthma but has no utility in managing COPD. In COPD, there is a poor relationship between PEF and forced expiratory volume in 1 second (FEV_1), and it is impossible to predict FEV_1 from the PEF or vice versa. Peak expiratory flow may underestimate the degree of airway obstruction in COPD and is relatively insensitive to obstruction of the small airways (mild or early obstruction). Moreover, PEF is very dependent on patient effort.

Discuss the effects of smoking on various systems.

Cigarette smoke consists of a gaseous phase (80–90%) and a particulate phase. Gaseous phase consists of mainly nitrogen, oxygen, carbon dioxide, carcinogens (hydrocyanic acid and hydrazine), ciliotoxins, irritants (hydrocyanic acid), carbon monoxide (CO) and some others. Nicotine is the main component of particulate phase.

Cardiovascular system: Nicotine stimulates the adrenal medulla to secrete adrenaline, resets the carotid body and aortic receptors to maintain a higher blood pressure, and stimulates autonomic ganglia, increasing sympathetic tone. The result is an increase in systolic and diastolic blood pressure, heart rate, and peripheral vascular resistance; leading to an increase in oxygen demand by the myocardium. An increase in coronary vascular resistance decreases the myocardial oxygen supply: demand ratio. The half-life of nicotine is 30–60 minutes. Following the smoking of 1 cigarette, the pressor response lasts for about 30 minutes. Three to four hours of abstinence results in insignificant side effects due to nicotine and a significant improvement of the myocardial oxygen supply: demand ratio.

Effects of carboxyhemoglobin: Cigarette smoke contains 400 parts per million (ppm) carbon monoxide. In the blood, CO combines with hemoglobin to form carboxyhemoglobin (COHb). In smokers, the amount of COHb in the blood ranges from 5 to 15%. The affinity of CO for hemoglobin is 200 times that of oxygen. It shifts the oxyhemoglobin curve to the left due to; (i) its high affinity for Hb, (ii) a change in shape of the oxyhemoglobin curve from a sigmoidal to a more hyperbolic curve by carboxyhemoglobin, and (iii) depletion of 2, 3-diphosphoglycerate by CO. These mechanisms lead to chronic tissue hypoxia. CO by itself may cause arrhythmias. The half-life of carboxyhemoglobin depends chiefly on pulmonary ventilation: at rest 4–6 hours, with strenuous exercise 1 hour, during sleep 10–12 hours. With 100% oxygen, the half-life is reduced to 40–80 minutes. Thus, on advising patients before anesthesia, these variations should be noted. During the day time, abstinence for 12 hours is sufficient to get rid of CO. If an operation is scheduled for the next morning, the patient should not smoke the previous evening.

Hemostatic system: Smoking increases the production of Hb, red blood cells, white blood cells, and platelets, fibrinogen and increases platelet reactivity. This results in an increase in the hematocrit and the blood viscosity, leading to an increased thrombotic tendency. Therefore, incidence of arterial thromboembolic disease is more in smokers; however incidence of DVT has not been shown to be increased. Chronic hypoxia to the cardiac muscle and the increase in incidence of thromboembolic disease causes smokers to be at a 70% greater risk of coronary artery disease compared with nonsmokers, and the postoperative mortality in smokers is higher than in nonsmokers.

Respiratory system: Irritants in smoke increase mucus secretions. The mucus becomes hyper viscous. Cilia become inactive and are destroyed by ciliotoxins, which leads to decreased tracheobronchial clearance. Cigarette smoke disrupts the epithelial lining of the lung, causing an increase in pulmonary epithelial permeability. This loss of epithelial integrity allows irritants to penetrate the epithelium more easily and stimulate the subepithelial irritant receptors, resulting in increased reactivity. Smoking leads to small-airway narrowing, causing an increased closing volume. Pulmonary surfactant is also decreased. These lead to small-airway disease. An increase in pulmonary proteolytic enzymes or elastolytic enzymes causes loss of elastic lung recoil and emphysema. 25% of smokers suffer from chronic bronchitis.

Gastrointestinal system: Smoking makes the gastro-esophageal sphincter incompetent, which allows reflux, with accompanying risks of pulmonary aspiration. The incompetence in the gastroesophageal sphincter begins within 4 minutes of beginning to smoke and returns to normal within 8 minutes after the end of smoking.

Discuss the preoperative measures undertaken to optimize a COPD patient.

Cessation of smoking is the single most important intervention in the preoperative period. A study by Warner found that the incidence of postoperative pulmonary complications was highest (57%) in patients who quit smoking for less than 8 weeks; it decreased to 14.5% if cessation was more than 8 weeks. It was least in (11.9%) in those who never smoked. Current smokers (33%) also had a high risk of postoperative

complications. It is hypothesized that the absence of the irritant effect of cigarette smoke in the postoperative period inhibits coughing and leads to retention of secretions and small airway obstruction, in patients who stopped smoking less than 8 weeks. Within 12 hours of cessation, the carboxyhemoglobin levels drop. Long-term cessation (more than 8 weeks) leads to improvement in mucociliary function with increased sputum clearance and also reduced airway reactivity and sputum production. The risk for patients who quit more than 6 months is the same as those who never smoked. Smoking has also been shown to delay wound healing.

Treat chest infection with appropriate antibiotics.

Bronchodilator therapy will benefit not only these patients but also those without bronchospasm; even if no improvement in FEV_1 is seen.

Steroids: Inhaled corticosteroids improve symptoms, lung function; and reduce exacerbations when FEV_1 is less than 60% of predicted value.

Many of these patients might be receiving steroids. Recommendations for perioperative management of patients on steroid are given in Table 10.4.

Table 10.4 Recommendations for steroids in perioperative patient

Dose	Surgery	Recommended dose
< 10 mg/day	Minor/ Moderate/ Major	Additional steroid cover not required (assume normal HPA response)
> 10 mg/day	Minor surgery	25 mg of hydrocortisone at induction and normal medications postoperative
> 10 mg/day	Moderate surgery	Usual dose preoperative and 25 mg hydrocortisone IV at induction then 25 mg IV TDS for 1 day then recommence preoperative dosage
> 10 mg/day	Major surgery	Usual dose preoperative and 100 mg hydrocortisone at induction then 100 mg IV TDS for 2–3 days.

Patients with cor pulmonale may be receiving diuretics and/or digitalis. These should be continued perioperatively.

Hydration and mucolytic therapy will help in loosening of secretions and along with chest physiotherapy will help in clearing the chest.

Preoperative *incentive spirometry* has always been recommended for reducing POPCs, though there is no supportive evidence.

How will you premedicate this patient?

Anxiolysis is important in COPD patients. In absence of hypercapnea on a baseline arterial blood gas (ABG), it is safe to premedicate these patients with a small dose of benzodiazepine as it is unlikely to produce central depression. Benzodiazepines do not alter bronchial tone. In other patients, it may be safer to administer premedication in the preoperative holding area in monitored environment. There is no role of routine anticholinergic premedication as most of modern anesthetic agents have minimum respiratory irritant properties as compared to the old ones.

What anesthetic technique would you choose in this patient? Why?

Regional anesthesia (RA) has the advantage of avoiding airway instrumentation and bronchospasm. The disadvantages of a regional block include inadequate muscle relaxation, coughing, bucking, need for high levels of spinal or epidural block and increased parasympathetic tone, which may cause bronchospasm. RA can decrease ERV by ~50%, which may be detrimental if the patient is dependent on active expiration. Hypotension due to RA, patient discomfort due to prolonged procedure and shivering, need for heavy sedation with RA (which may be worse than light GA) are other considerations. Also it is important to remember that hypoxemia or hypercarbia that can develop in high levels of motor blockade may adversely affect right ventricular function.

General anesthesia offers the advantages of control of ventilation with good muscle relaxation. This ensures oxygenation and CO_2 elimination and overcomes the decrease in lung compliance, increased resistance and decreased FRC.

A combination of GA with epidural analgesia would be ideal in our patient. Apart from all benefits of GA, epidural anesthesia (EA) adds the benefit of excellent analgesia, reduced requirement of muscle relaxants, and lower risk of hypotension. Added advantages include postoperative analgesia that would avoid use of narcotics, facilitation of early ambulation, better performance of respiratory therapy maneuvers and possible reduction in postoperative pulmonary complications and deep venous thrombosis.

What monitoring would you institute in this patient?

The standard monitoring will include electrocardiogram, heart rate, end-tidal CO_2, pulse oximetry, temperature, invasive blood pressure monitoring (in view of major surgery with fluid shifts) and hourly urine output monitoring. Invasive arterial pressure monitoring offers the advantage of beat to beat pressure monitoring as well as sampling for ABG analysis. Central venous monitoring will help in optimizing fluid therapy.

What are the options for induction of anesthesia?

It is best to avoid thiopentone as thiobarbiturates may cause histamine release. If barbiturates have to be used,

it is preferable to use oxybarbiturates (methohexitone). It is important to remember that airway instrumentation or stimulation under light thiopentone anesthesia may provoke a bronchospasm. In cardiostable patients, propofol would be agent of choice for anesthesia induction as it offers marked protection against bronchospasm; however hypotension needs to be guarded against. Though many anesthesiologists prefer ketamine for its bronchodilator effect, it is dose related and not predictable. In addition, it can cause tachycardia and hypertension and an increase in pulmonary vascular resistance via indirect adrenergic stimulation. It also leads to an increase in airway secretions.

How will you secure the airway and conduct anesthesia?

Laryngeal mask airway (LMA) has been associated with a lower incidence of bronchospasm due to less airway stimulation; we obviously cannot use it in the present case as this patient is undergoing upper abdominal surgery and may need postoperative ventilation. Also the lung compliance may be poor needing high airway pressure for ventilation, which may be a problem with LMA. This is because airway pressures more than 25 cm H_2O are known to be associated with gastric insufflation; which may lead to regurgitation and aspiration of gastric contents.

It is best to use a non-depolarizing muscle relaxant (NDMR), narcotic and adequate plane of anesthesia before intubating the patient. It is known that NDMRs, which affect the M_2 (muscarinic type 2) receptors more than the M_3 receptors cause and enhance bronchoconstriction. These include gallamine, pipecuronium, which are not in much use now. Vecuronium, rocuronium, and pancuronium affect M_3 receptors and can be used safely. Benzyl quinolones like atracurium, mivacurium cause dose-dependent histamine release and increased bronchial tone and should be avoided. This histamine release is not seen with cis-atracurium and it can be used.

Opioids induce cough suppression and are one of the components of balanced anesthesia. Fentanyl in large doses, alfentanil and sufentanil can cause chest rigidity, mimicking bronchospasm. This effect is common with faster injection and increasing age of the patient. Morphine and pethidine are known to cause histamine release.

Among the inhalational agents, halothane is the most potent bronchodilator. It suppresses airway reflexes, causes direct relaxation of airway smooth muscle in a dose related manner due to Beta-adrenergic receptor stimulation, which is decreased by beta-blocking agents. Sevoflurane and isoflurane at higher MAC's are equally good, though isoflurane due to its irritant smell can provoke bronchospasm.

Administration of topical lignocaine for intubation or before endotracheal suctioning is no more recommended as it itself can provoke bronchospasm.

How will you diagnose and treat intraoperative bronchospasm?

Increased peak and plateau airway pressures, decreasing slope of expired CO_2 and wheeze or absence of breath sounds are signs suggestive of intraoperative bronchospasm. This may lead to hypoxemia, hypercarbia and hypotension due to dynamic hyperinflation and development of auto PEEP (PEEPi). Causes of intraoperative increase in peak airway pressure are:

- Light anesthesia, coughing, bucking
- Obstruction in the circuit
- Blocked/kinked tube
- Endobronchial intubation
- Bronchospasm
- Pneumothorax
- Major atelectasis
- Pulmonary edema
- Aspiration pneumonia
- Head down position, bowel packing.

On noticing raised airway pressures, rule out various causes mentioned above. Once bronchospasm is diagnosed, increase the FiO_2 to 1.0. As the most common cause of bronchospasm is surgical stimulation under light anesthesia, deepen plane of anesthesia. Perform thorough endotracheal suctioning after ensuring adequate depth of anesthesia. The pharmacological options to treat bronchospasm are use of inhaled (or nebulized, or MDI) β_2- agonists, use either nebulized β_2- agonists, or in severe case use subcutaneous terbutaline or intravenous adrenaline. Inhaled terbutaline and albuterol have similar efficacy via the intravenous route. Often more than 2 puffs may be needed to counter the acute bronchospasm. Intravenous $MgSO_4$ (1–2 g over 20 minutes) can also be used. Intravenous corticosteroids are indicated in severe bronchospasm. Aminophylline bolus (6 mg/kg) followed by an infusion (1.0 mg/kg/h for smokers, 0.5 mg/kg/h for nonsmokers and 0.3 mg/kg/h for severely ill patients) can be used. However, it has low therapeutic window and may lead to intractable arrhythmias and seizures (more caution should be exerted if using halothane).

What is auto PEEP? What is pathogenesis of auto PEEP? What are other situations that can lead to generation of auto PEEP?

In a patient with COPD, during a stage of bronchospasm, increased bronchial smooth muscle tone leads to resistance to air movement. Since inspiration is an active process, this

phase is not much affected however expiration being a passive process that totally depends on caliber of airway and elastic recoil of lungs gets prolonged. If next breath is delivered to the lungs (spontaneous or controlled) before complete exhalation of previous breath, there will be accumulation of air in alveoli (breath stacking). This "trapped air" generates Positive airway Pressure at the End of Expiration, and this is known as "auto PEEP" (self-generated PEEP) or "intrinsic PEEP". This cumulative increase leads to increasing amount of air being trapped in the lungs and results in over expansion of lungs leading to "dynamic hyperinflation" of the lungs. Other causes of auto PEEP are mucus plugging of the airways, large minute ventilation, block in the expiratory circuit of the ventilator (such as blocked HME filter) and ventilating with small endotracheal tube with inadequate expiratory time that leads to incomplete expiration.

How do you confirm development of auto PEEP? How do you treat it?

Most modern ICU ventilators will allow direct measurement of auto PEEP. For intraoperative diagnosis, apnea test can be carried. Hypotension, if it develops over time, is almost always due to excessive hyperinflation and auto PEEP in the absence of hypovolemia. Disconnect the patient from the ventilator for 30 second (apnea). If this leads to normalization of blood pressure (due to improved expiration and thus venous return), this confirms the diagnosis of auto PEEP. Auto PEEP can be eliminated by increasing expiratory time; this can be achieved with reduction in tidal volume, respiratory rate, and increasing the peak inspiratory flow rate.

Do you anticipate any problems during reversal? When will you extubate this patient?

Reversal of muscle relaxation with neostigmine may provoke bronchospasm; this can be countered with administration of atropine 1.2–1.8 mg or glycopyrrolate 0.6 mg before neostigmine. Extubation under deeper plane of anesthesia may reduce incidence of bronchospasm, in this case delayed extubation may be preferred. Actually this patient may require a period of postoperative ventilation due to prolonged surgery and hypothermia. The patient can then be extubated once fully awake and obeying commands, has sustained head lift and adequate gas exchange.

Discuss postoperative management of this patient.

Postoperative analgesia plays a pivotal role in the outcome of these patients. Continued utilization of the epidural infusion (local anesthetic alone or combined with small dose of narcotic) will ensure adequate analgesia. This may be further improved upon by addition of systemic NSAIDs and or paracetamol. Adequate analgesia will allow administration of postoperative respiratory therapy in the form of incentive spirometry and deep breathing exercise, will prevent development of basal atelectasis, and improve gas exchange. Deep venous thrombosis prophylaxis with either unfractionated or low molecular weight heparin should be given. Early mobilization and enteral feeding (via nasojejunal tube or jejunostomy) are also important.

Enumerate likely postoperative pulmonary complications in this patient.

1. *Decreased FRC:* The large incision causes decrease in FRC due to postoperative pain, Splinting of the diaphragm and conversion from diaphragmatic to rib-cage breathing pattern, decreased sputum clearance.
2. *Bibasal atelectasis* with retained secretions can lead to postoperative pneumonia. This can be prevented with incentive spirometry, chest physiotherapy and early ambulation.
3. Need for mechanical ventilation, prolonged ICU/hospital stay.
4. Delayed ambulation.
5. *Deep venous thrombosis (DVT), pulmonary embolism (PE):* Poor preoperative mobility, development of polycythemia and intraoperative hypothermia makes these patients particularly vulnerable to development of deep venous thrombosis. Ideally such patients should receive one dose of low molecular weight heparin preoperatively. In the presence of absolute or relative contraindications, mechanical prophylaxis (in the form of graduated compression stockings and or sequential calf compression devices) should be resorted to intraoperatively as well. As soon as possible postoperatively, DVT prophylaxis should be commenced.
6. Right ventricular dysfunction/failure.

Discuss the causes and management of hypoxia in a postoperative patient.

Hypoxia is the most common postoperative complication. Use of pulse oximetry has improved the detection and management of postoperative hypoxia. In elderly patients, those with shivering or hemodynamic instability, the degree of hypoxia is increased manifold.

The common causes of postoperative hypoxia are:

1. *Diffusion hypoxia:* Nitrous oxide (N_2O) is much more soluble (more than 40 times) in blood than nitrogen. Once N_2O is discontinued at the end of anesthesia, it diffuses out into the alveoli. If the patient is breathing air at this time, FiO_2 in the alveoli is less than 0.21, thus reducing alveolar PO_2. This can be prevented by giving 100 O_2 by mask for at least 5 minutes after discontinuation of N_2O.

2. *Low inspired concentration of oxygen:* This may occur in rare instances, such as crossing of N_2O and O_2 pipelines during hospital construction, low barometric pressure, switching off of adapters, nitrogen flooding of the gas pipeline during repairs.
3. *Alveolar hypoventilation:*
   a. Residual effect of neuromuscular blockers (NMB) opiates, pain—use analgesia, adequate reversal of NMB, administration of supplemental O_2
   b. Airway obstruction: Insertion of oro-or nasopharyngeal airways, supplemental O_2
   c. Bronchospasm: Treatment of cause and supplemental O_2
4. *Intrapulmonary right-to-left shunt:* The most common cause of postoperative hypoxia.
   a. Atelectasis (collapse of part or whole lung) can occur due to a variety of etiologies in the postoperative period.
      - Bronchial obstruction from secretion or blood
      - Endobronchial intubation with collapse of the opposite lung
      - Pneumothorax
      - Diffuse airway collapse. When the closing capacity exceeds FRC, airways collapse during tidal ventilation resulting in right to left shunting.
5. *Pulmonary edema* can occur due to left ventricular failure as a result of sudden increase in afterload (commonly seen as increase in the systolic blood pressure) in the postoperative period. Pulmonary edema of the noncardiac origin (ARDS) due to increased pulmonary capillary permeability may occur in aspiration, postsurgical sepsis, trauma, and acute severe pancreatitis. Treatment will depend on cause and ventilation with high FiO_2 and PEEP, diuresis, vasopressors and inotropes (in case of shock) may be needed.
6. *Pulmonary embolism:* It is rare but may be life-threatening in the immediate postoperative period. Accurate diagnosis is essential. Bedside 2-D echocardiography, pulmonary angiography or spiral CT scan are the diagnostic modalities. Anticoagulation with continuous intravenous infusion of heparin and /or surgical embolectomy; are the treatment options. Thrombolysis may be contraindicated in most of the postoperative cases but may be given weighing the risks and benefit in surface surgeries.

Discuss oxygen therapy in COPD patients.

Regardless of whether patient has COPD or not, all hypoxic patients must be given oxygen. COPD patients often remain stable even in presence of what would be alarmingly low PO_2 levels for normal patients. Hypoxia in COPD patients can lead to a sudden cardiorespiratory arrest. It is difficult to predict the PO_2 level, which can lead to catastrophe but most COPD patients will remain oxygenated if PO_2 is less than 50 mm Hg. Episodes of acute exacerbation will cause further drop in the PO_2 levels. Aim of oxygen therapy in COPD patients is to achieve PO_2 less than 50 mm Hg. It is difficult to judge beforehand the PO_2 levels that may be achieved in COPD patients for a particular FiO_2. Hypercarbia may occur due to respiratory depression with oxygen therapy. As long as one monitors the PCO_2 and in particular pH, so as to intervene (generally at PH < 7.25), patient can be safely given low FiO_2 with a ventimask (0.24–0.36) and kept under observation.

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11 Intercostal Drain

P Ranganathan, B Trivedi

A 55-year-old male patient presented with history of dull left sided chest pain and progressively increasing breathlessness since 3 months. He gives history of cough with occasional hemoptysis, low grade fever and weight loss. On examination, he is malnourished. There is generalized lymphadenopathy. Examination of the respiratory system reveals grossly decreased air entry over the left hemithorax. The patient is breathless at rest and cannot walk more than a few steps. Chest X-ray and ultrasonography of the chest reveal a massive left pleural effusion. This patient is posted for cervical lymph node biopsy under general anesthesia.

How would you manage this patient's pleural effusion?

Since this patient has a large pleural effusion and is symptomatic, he needs drainage of the effusion by inserting a chest tube or intercostal drain (ICD).

What are the common indications for chest drain insertion?

Asymptomatic patients with a minimal pneumothorax (< 15–20% of hemithorax) can be managed conservatively. Symptomatic patients or patients with a larger pneumothorax need aspiration or drainage.

Chest tube drainage is usually indicated for the following conditions:

1. Pneumothorax
   a. In a mechanically ventilated patient
   b. Tension pneumothorax after initial decompression by inserting a needle
   c. Persistent or recurrent pneumothorax after simple aspiration.
2. Large or symptomatic pleural effusions
3. Other pleural collections
   a. Pus (empyema)
   b. Blood (hemothorax)
   c. Chyle (chylothorax).
4. Postoperative—after thoracotomy or thoracoscopy.

What is the function of a chest drain?

Chest drains are inserted to remove air or fluid that collects in the pleural space to allow restoration of negative intrathoracic pressures, and to allow the lung to re-expand.

How does a chest drain function?

Chest drainage systems work by a combination of the following:

1. When the patient breathes out deeply or coughs, the expiratory positive pressure, which is generated helps to push air and fluid out of the chest.
2. Positioning the collection chamber of the chest drain below the level of the chest allows fluid drainage by gravity.
3. Application of suction to the collection chamber can hasten drainage from the chest; however, suction must be applied cautiously.

What are the components of a chest drainage system?

Chest drainage systems are made of the following components:

1. A chest tube is inserted into the pleural cavity or mediastinal cavity to allow contents (air, fluid) to leave the chest.
2. This tube is connected by a length of flexible tubing to the drainage system.

3. The drainage system consists of a chamber that collects fluid. There is a one-way seal (fluid level or mechanical valve) that prevents outside air from entering the thoracic cavity during inspiration.

What is the importance of the underwater seal?

The underwater seal acts as a one-way valve through which air is expelled from the pleural space during expiration and prevented from re-entering during the next inspiration.

Describe the single-bottle drainage system.

The drainage tube is submerged to a depth of 1–2 cm in a collection chamber of approximately 20 cm diameter. When the patient inspires, water is drawn up the tube to the height equal to the negative intrathoracic pressure (usually up to 20 cm of water). Therefore, the collection chamber must be kept far enough below the patient to prevent water from being sucked up into the chest. Usually a height of 100 cm is sufficient, to allow for negative pressures as high as 80 cm H_2O. The portion of the tubing that is underwater offers resistance to expiration; this should be no more than 2 to 3 cm. The end of the tube in the underwater seal bottle must remain covered with water at all times. Collected air escapes through a side vent in the chamber.

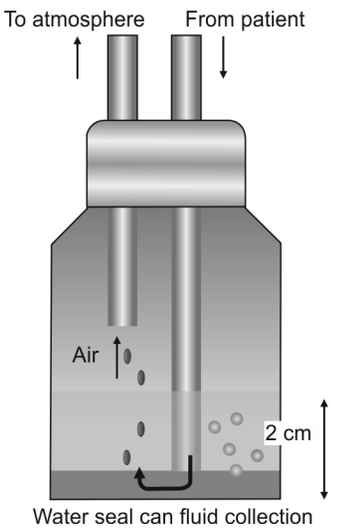

Fig. 11.1 Single-bottle drainage system

What is the disadvantage of this single-bottle drainage system?

As fluid drains from the chest, the level of fluid in the underwater seal starts rising. This increases the resistance against which the patient has to breathe and can obstruct further outflow of fluid from the chest.

Describe the two-bottle drainage system.

To overcome the disadvantage of the single bottle system, a second bottle is introduced between the chest tube and the underwater seal. This trap bottle collects the fluid draining out of the chest, while the air passes on to the second bottle and is vented to the atmosphere. This keeps the underwater seal at a constant level.

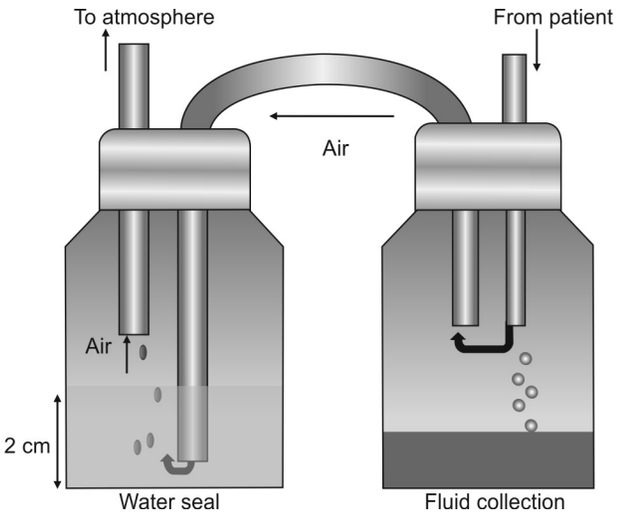

Fig. 11.2 Two-bottle drainage system

Describe the three-bottle drainage system.

When a negative pressure is desired, a third bottle is added between the underwater seal and the suction device. It has an inlet, connected to the vent of the underwater seal chamber of the two-bottle system, an outlet connected to the suction device, and a control tube, which is open to the atmosphere on one side and submerged 20 cm underwater on the other.

When suction is applied, air is drawn down the atmospheric vent in this bottle, equal to the pressure inside the bottle that is decreased by the vacuum. This low pressure suction is transmitted to the underwater seal bottle and then into the pleural cavity, thus aiding evacuation of contents and allowing quicker re-expansion of the underlying lung. The maximum force of suction is determined by the depth of the

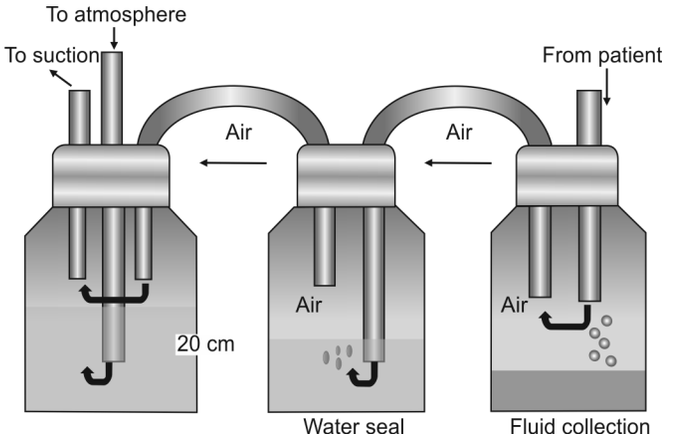

Fig. 11.3 Three-bottle drainage system

atmospheric vent underwater in the suction regulation bottle. For example, to obtain a suction of 20 cm of water, the tip of the tube should be 20 cm below the surface of the fluid. Turning up the suction does not increase negative pressure within the pleural drainage system.

The three-bottle system provides a fairly stable water-seal level, allows for accurate documentation of the drainage and also controlled suction. Unfortunately, it is bulky and does not allow for easy transport or ambulation of the patient.

How much suction can be applied to the ICD bottle? When should suction be avoided?

Continuous low-pressure suction (recommended level of suction 5 to 20 cm of water) may be applied to the ICD bottle. Suction pressures higher than 20 cm of water have the potential to damage lung tissue. Suction should be avoided if there is an on-going air leak (the air leak may worsen) and in post-pneumonectomy patients.

What is the multifunction chest drainage system?

The 2 and 3-bottle drainage systems are bulky and can result in accidental disconnections. Commercially available multi-function drainage systems incorporate all the components of the three-bottle system into a single unit. Examples of multifunction chest drain systems include Pleurovac and Redivac systems.

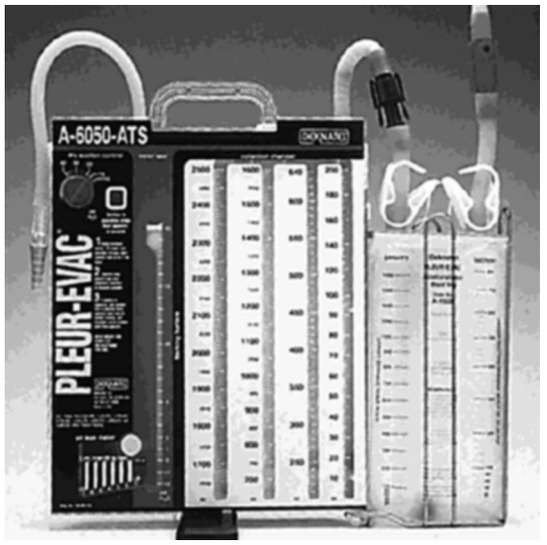

Fig. 11.4 Multifunction chest drainage system

Describe the management of the chest tube and precautions that need to be taken during anesthesia.

1. A bubbling chest tube should never be clamped. Clamping a chest drain in the presence of a continuing air leak may lead to tension pneumothorax.
2. Drainage of a large pleural effusion should be controlled to prevent the potential complication of re-expansion pulmonary edema. No more than about 1.5 liters should be drained at one time, or drainage should be slowed to about 500 mL per hour.
3. The drainage bottle should always be placed below the level of the chest to prevent fluid from re-entering into the thoracic cavity.
4. In the presence of a large air leak, loss of tidal volume should be anticipated. These patients should be maintained on spontaneous breathing if possible, until lung isolation can be achieved.

Three days after insertion of the chest tube, it is found that the fluid column is not moving. What are the possible causes?

Absence of fluid oscillations can occur when:

1. The tubing is clamped or kinked.
2. The patient is lying on the tubing.
3. There is a dependent, fluid-filled loop in the tubing.
4. Lung tissue or adhesions are blocking the catheter eyelets inside the pleural cavity.
5. Blockage of the tube due to a blood clot.
6. No more air is leaking into the pleural space and the lung has re-expanded completely.

When can an ICD be removed?

An ICD can be removed when:

1. Drainage diminishes to little or nothing.
2. Air leak has stopped.
3. Fluctuations in the water seal chamber stops.
4. The patient is breathing normally without any signs of respiratory distress.
5. Breath sounds are equal and at baseline for the patient.
6. Chest X-ray shows the lung is re-expanded completely and there is no residual air or fluid in the pleural space.

What are the complications of chest drains?

1. Hemorrhage due to vascular injury while inserting
2. Blocked tube due to poor positioning
3. Cardiac dysrhythmia
4. Infections
5. Re-expansion pulmonary edema.

Suggested Reading

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3. Laws D, Neville E, Duffy J; Pleural Diseases Group, Standards of Care Committee, British Thoracic Society. BTS guidelines for the insertion of a chest drain. Thorax. 2003;58(2):ii53–9.

12 Hypertensive Disorders in Pregnancy

S Bakshi, R Ambulkar, S Bhosale

A 24-year-old primigravida, diagnosed with pregnancy-induced hypertension (PIH) on medication was admitted at 36 weeks for elective section. She has high blood pressure and headache. Discuss perioperative management.

Define pre-eclampsia, severe pre-eclampsia and eclampsia.

According to the International Society for the Study of Hypertension in Pregnancy (ISSPH 2001), hypertensive disorder during pregnancy is defined as a diastolic blood pressure of more than 90 mm Hg taken on two occasions more than 4 hours apart or a single diastolic blood pressure above 110 mm Hg.

Hypertensive disorders of pregnancy can be classified as:

1. Gestational hypertension (formerly PIH or transient hypertension)
2. Pre-eclampsia and eclampsia
3. Pre-eclampsia superimposed on chronic hypertension
4. Chronic hypertension.

Gestational hypertension: It is said to be present when BP > 140/90 mm Hg for first time during pregnancy after 20 weeks, but no proteinuria. This is transient hypertension and blood pressure returns to normal by 12 weeks postpartum.

Pre-eclampsia: It is defined as new hypertension presenting after 20 weeks with significant proteinuria [more than 300 mg per 24 hours, or persistent 30 mg/dL (1+ on dipstick)] in random urine samples.

Severe pre-eclampsia is pre-eclampsia with any of the following.

- BP more than 160/110 mm Hg
- Proteinuria 2.0 g per 24 hours or more than 2+ on dipstick
- Serum creatinine more than 1.2 mg/dL unless known to be previously elevated
- Platelets less than 100,000/mm^3 (sign of worsening pre-eclampsia caused by platelet activation and aggregation and microangiopathic hemolysis induced by vasospasm)
- Microangiopathic hemolysis
- Elevated SGOT/SGPT levels
- Persistent headache and or visual disturbances
- Persistent epigastric pain (Results from hepatocellular necrosis, ischemia, and edema leading to stretch in Glisson's capsule)

Severity of Pre-eclampsia

The differentiation between mild and severe pre-eclampsia can be misleading because mild disease may progress rapidly to severe disease.

Table 12.1 Differences between mild and severe pre-eclampsia

	Mild pre-eclampsia	Severe pre-eclampsia
Diastolic BP	< 100 mm Hg	110 mm Hg or higher
Proteinuria	Trace or 1 +	Persistent 2 + or more
Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Convulsion	Absent	Present (eclampsia)
Fetal growth restriction	Absent	Obvious
Pulmonary edema	Absent	Present
Serum creatinine	Normal	Elevated
Thrombocytopenia	Absent	Present
Liver enzyme elevation	Minimal	Marked

Chronic hypertension: BP > 140/90 mm Hg before pregnancy or diagnosed before 20 weeks gestation or hypertension first diagnosed after 20 weeks of gestation and persistent after 12 weeks postpartum.

Superimposed pre-eclampsia (on chronic hypertension): All chronic hypertensive disorders regardless of their cause predispose to development of superimposed pre-eclampsia or eclampsia. Pre-eclampsia is accompanied by proteinuria.

Eclampsia: Eclampsia is the occurrence of seizures in a woman with pre-eclampsia when the seizures cannot be attributed to other causes. The seizures are grand mal and may appear before, during, or after labor. It may develop more than 48 hours after delivery and may be encountered up to 10 days postpartum.

Discuss the pathogenesis of pre-eclampsia.

Risk factors: Increased incidence is found in primigravida, multiple pregnancies, history of chronic hypertension, maternal age more than 35 years, obesity. Decreased incidence is found in women who smoke and in patients with placenta previa.

Theories for development of pre-eclampsia: Vasospasm is basic to the pathophysiology of pre-eclampsia and eclampsia. Vascular constriction causes resistance to blood flow and accounts for the development of arterial hypertension. It is likely that vasospasm itself causes damaging effect on vessels. The vascular changes together with local hypoxia of the surrounding tissues, presumably lead to hemorrhage, necrosis and other end organ disturbances. Some theories put forward include:

1. *Increased pressor responses:* Women with PIH have been found to have increased vascular sensitivity to pressors, and increased sensitivity to Angiotensin II preceding the onset of PIH.
2. *Prostaglandins:* In PIH, there is decreased prostacyclin production and increased thromboxane A2; resulting in vasoconstriction and sensitivity to infused Angiotensin II.
3. *Nitric oxide* is also known as endothelial derived relaxing factor (EDRF) is a potent vasodilator. Decreased levels are found in PIH patients.
4. *Vascular endothelial growth factor (VEGF):* It is important in vasculogenesis and control of microvascular permeability. VEGF has been reported to be increased in serum from women with pre-eclampsia. Increased level in PIH may represent compensatory mechanism to restore uteroplacental blood flow towards normal.
5. *Genetic predisposition:* The tendency for pre-eclampsia-eclampsia is inherited.
6. *Immunological factors:* PIH is probably an immune response to antigenic sites on placenta. This may arise where effective immunization by a previous pregnancy is lacking, as in first pregnancies or where the number of antigenic sites provided by the placenta is unusually great (multiple pregnancies).
7. *Inflammatory factors:* Pre-eclampsia is considered a disease due to extreme state of activated leukocytes in the maternal circulation.
8. *Endothelial cell activation:* Pre-eclampsia is an immunologically mediated deficiency in trophoblastic invasion of spiral arteries leading to fetoplacental hypoperfusion. These changes in turn provoke activation of the vascular endothelium with the clinical syndrome of pre-eclampsia.

Can Pre-eclampsia be predicted and prevented?

Early predictors of PIH: A variety of biochemical and biophysical markers have been proposed for the purpose of predicting the development of pre-eclampsia later in pregnancy. All the strategies have low sensitivity for the prediction of pre-eclampsia. Some of the tests are as follows:

1. *Rollover test:* The patient is asked to assume supine position after lying laterally recumbent. A positive test is an elevation of 20 mm Hg or more in systolic blood pressure. This test has a very good correlation with the angiotensin sensitivity test.
2. *Elevated uric acid* levels > 5.9 mg/dL.
3. *Calcium metabolism:* Hypocalciuria seen with pre-eclampsia.
4. *Angiotensin II infusion test:* It is difficult to perform and hence not used in routine clinical practice.
5. *Decreased urinary Kallikreins excretion:* Might precede development of pre-eclampsia.
6. *Increased cellular plasma fibronectin:* these generally precede the development of pre-eclampsia.
7. *Coagulation activation:* Thrombocytopenia and abnormal platelet aggregation appear to be an integral feature of pre-eclampsia. Excessive platelet activation has been linked to maternal vasoconstriction, endothelial cell injury, placental infarction (atherosis and fetal growth restriction) and transient renal dysfunction. Thromboxane A2 is released promoting vasospasm, further platelet aggregation and endothelial cell injury.
8. *Immunological factors*: Levels of TNF-α, growth factors and interleukins increased.
9. *Placental peptides*: Increased corticotropin—releasing hormone, Activin A and Inhibin A.
10. *Doppler velocimetry studies of the uterine arteries*: It is an unreliable screening test. Sensitivity 78% with PPV 28%.

Risk Factors for Pre-eclampsia

As per NICE guidelines 2010, the following have been described to be risk factors for pre-eclampsia.

High risk factors (any one)

1. Hypertension during last pregnancy
2. Chronic renal disease
3. Autoimmune disorders like SLE
4. Diabetes
5. Chronic hypertension.

Moderate risk factors (more than one)

- First pregnancy
- Age 40 years or older
- Pregnancy interval of more than 10 years
- Body mass index (BMI) of 35 kg/m^2 or more at first visit
- Family history of pre-eclampsia
- Multiple pregnancies.

Preventive measures

Aspirin should be started at 75 mg daily from 12 weeks of gestation till delivery for patients having any one high-risk factor or more than one moderate-risk factor. Nitric oxide donors, LMWH, diuretics and progesterone have no role to play and there are no recommendations for its use. Nutritional supplementation in the form of fish oil, garlic, antioxidants, vitamins C and E, folic acid, salt restriction have not been shown to prevent PIH. There is inconclusive evidence regarding calcium supplementation in prevention of PIH.

What are the pathological alterations that occur due to pre-eclampsia?

Pathological Manifestations of Pre-eclampsia

1. *Cardiovascular changes* are basically related to increase in cardiac afterload due to hypertension.
2. *Hematological changes:* Thrombocytopenia (defined as platelet count less than 1,00,000/mm^3) is likely due to platelet activation and consumption and reduced platelet production. The other changes include: microangiopathic hemolysis (due to intense vasospasm), decreased clotting factors, increased erythrocyte destruction, increased fibrin degradation products and deficiency of antithrombin III.
3. *Endocrine changes:* Increase in deoxycorticosterone in third trimester due to conversion from plasma progesterone causes retention of sodium. Sodium retention leads to inhibition of juxtaglomerular apparatus resulting in decreased plasma levels of renin, angiotensin II and aldosterone as compared with normotensive pregnancy.
4. *Fluid and electrolyte changes:* In pre-eclampsia there is decreased plasma oncotic pressure, which leads to increased extracellular fluid with associated decrease in intravascular volume.
5. *Kidney:* Oliguria is a result of reduced renal perfusion and glomerular filtration. Plasma uric acid and serum creatinine are elevated. There is proteinuria > 300 mg per 24 hours or more than 1 + on urinary dipstick test. Acute renal failure and renal cortical necrosis may develop in severe cases.
6. *Liver:* Pre-eclampsia is often associated with increased liver enzymes. Epigastric and right hypochondriac pain when present; is due to periportal hemorrhagic necrosis, which causes hepatic rupture or subcapsular hematoma.
7. *HELLP syndrome:* Triad of Hemolysis, elevated liver enzymes and low platelets.
8. *Nervous system:* Cerebral edema, hyperemia, focal thrombosis and hemorrhage may be present in severe cases. Retinal artery vasospasm may cause visual disturbances. Increased cerebral perfusion pressure may cause severe headaches.
9. *Uterus and placenta:* Incomplete trophoblastic invasion of spiral arteries causes reduction in diameter of vessels as compared to normal. In addition, vasospasm decreases placental perfusion leading to Intra Uterine Growth Retardation (IUGR).

Describe medical management of PIH.

Medical Management of Pre-eclampsia

1. Early prenatal detection and prompt institution of therapy and corrective measures. The timing of prenatal examinations needs to be scheduled at intervals of 4 weeks until 28 weeks, and then every 2 weeks until 36 weeks and weekly thereafter.
2. Hospitalization is considered for women with new-onset hypertension, which is persistent or worsening or development of proteinuria. A systematic evaluation should include:
   a. Detailed examination followed by daily scrutiny of headache, visual disturbance, epigastric pain and rapid weight gain.
   b. Weight on admission and every day thereafter.
   c. Analysis for proteinuria on admission and at least every 2 days thereafter.
   d. Four hourly blood pressures monitoring in sitting position.
   e. Measurement of serum creatinine, complete blood count (CBC), liver enzymes, platelets—frequency determined by the severity of hypertension.
   f. Frequent fetal monitoring clinically or by USG.
3. Ample but not excessive proteins and calories; and no salt or fluid restriction in the diet.

Anti-hypertensive Drug Therapy

Hypertensive women who take ACE inhibitors, ARBs and chlorothiazides have to stop these drugs and change over to other drugs before they become pregnant as these drugs can cause congenital malformations. Blood pressure should be controlled to below 160/110 mm Hg to prevent maternal morbidity, particularly from intracranial hemorrhage, encephalopathy, myocardial ischemia and failure. Oral antihypertensive include the following drugs:

a. *Beta blockers:* Labetalol (300 mg to 2400 mg/day). It is combined α and β blocker. It is preferred to pure β blocker. Important to note: Prolonged beta blockers may reduce fetal growth.
b. *Methyldopa:* It is α methyl analogue of dopa, the precursor of dopamine (DA) and NA. The α methyl-NA is a selective α2 agonist formed in brain from methyldopa and acts on central $\alpha 2$ receptors to decrease efferent sympathetic activity. Antihypertensive effect develops over 4–6 hours and lasts 12–24 hours. Dose: 0.25–0.5 g BD–QID oral (max 4 g/day).
c. *Nifedipine:* Calcium channel blocker, which lowers BP by decreasing peripheral vascular resistance without compromising cardiac output. Usual dose is 10 to 20 mg every 4 to 6 hourly.

The target of blood pressure control in uncomplicated chronic hypertension is to keep blood pressure less than 150/100 mm Hg. Do not lower diastolic blood pressure below 80 mm Hg. In pregnant women with target-organ damage secondary to chronic hypertension; aim is to keep blood pressure lower than 140/90 mm Hg.

What are the complications associated with PIH?

Maternal Complications

- Intracranial hemorrhage (leading cause of maternal death)
- Disseminated intravascular coagulation (DIC)
- Pulmonary edema
- Congestive heart failure (CHF)
- Placental abruption
- Postpartam hemorrhage (PPH)
- Acute renal failure (ARF)
- Liver rupture
- Cerebrovascular accident
- Septic shock.

Fetal Complications

- Prematurity with respiratory distress
- Intrauterine growth retardation
- Oligohydramnios
- Intracranial hemorrhage
- Small for age
- Meconium aspiration.

What conditions mandate immediate delivery?

- Severe hypertension that persists after 24 to 48 hours of treatment
- Progressive thrombocytopenia
- Liver dysfunction
- Progressive renal dysfunction
- Premonitory signs of eclampsia
- Evidence of fetal jeopardy
- Persistent headache or other neurologic sequelae of pre-eclampsia.

Describe management of uncomplicated pre-eclampsia.

Management of pre-eclampsia depends upon:

- Severity of pre-eclampsia
- Duration of gestation
- Condition of cervix

When the fetus is known or suspected to be preterm, the tendency is to temporize in the hope that a few more weeks in utero will reduce the risk of neonatal death or serious morbidity. Assessment of fetal wellbeing is done using non-stress test and biophysical profile. Measurement of lecithin-sphingomyelin ratio in amniotic fluid may provide evidence of lung maturity. Glucocorticoids may be tried for fetal lung maturity before 34 weeks of gestation. If birth is likely within seven days, give 2 doses of betamethasone 12 mg IM 24 hours apart in women between 24–34 weeks. For a woman near term, if pre-eclampsia is mild but cervix is firm and closed, the pregnancy is allowed to continue under close observation until cervix is more suitable for induction.

For a woman near term with soft, partially effaced cervix, and even with milder degrees of pre-eclampsia, labor is induced by carefully monitored oxytocin induction.

In the past, both spinal and epidural were avoided in women with pre-eclampsia and eclampsia due to concerns of severe hypotension induced by sympathetic blockade and dangers from pressor agents or large volumes of intravenous fluids used to treat hypotension. However, with improvement in the techniques, and increased usage of epidural analgesia, many clinicians feel that epidural analgesia should be the preferred method. Advantages of epidural analgesia are:

- Good pain relief.
- Attenuation of the exaggerated hypertensive response to pain of pre-eclamptic women.
- Reduced circulating levels of catecholamines and stress related hormones.
- Improved inter-villous blood flow in pre-eclamptic women.

Pre-eclamptic women are at increased risk of cesarean delivery compared with normal parturients, and an early administration of epidural analgesia for labor facilitates the subsequent administration of epidural anesthesia for emergent cesarean delivery.

What are the adverse effects of labor pain and what various techniques are available for pain relief during labor?

Adverse Effects of Painful Labor

- Painful labor causes maternal hyperventilation. The resulting alkalosis shifts the oxyhemoglobin dissociation curve to the left in the mother. This results in increased binding of maternal hemoglobin to oxygen and reduced oxygen delivery to the fetus.
- Hyperventilation at the time of uterine contractions is followed by hypoventilation in between the contractions. This can result in fetal hypoxemia.
- Catecholamine release in response to labor pain causes reduction in uterine blood flow.
- The hypertensive response to sympathetic stimulation is detrimental in the hypertensive mother.
- Sympathetic stimulation causes maternal acidosis because of lactic acid production from skeletal muscle activity and free fatty acid activation. Lactic acidosis can cause incoordinate uterine action and prolonged labor.

Techniques of Labor Analgesia

Nonpharmacological Techniques

- *Psychoprophylactic approach of Lamaze:* This is based on gate control theory of pain relief. It hypothesizes that through stimulus-response conditioning, women can learn to use controlled breathing to reduce the pain in the labor. So, the mother is taught to concentrate and is conditioned to respond to contractions with relaxation using breathing techniques to reduce pain.
- *Leboyer's technique:* In this method baby is allowed to be born in a quiet dimly lit room, with minimal noise to reduce the trauma and stress for the newborn.
- Transcutaneous electrical nerve stimulation (TENS).
- Aromatherapy.
- Hypnosis.
- Acupuncture.
- Reflexology.

Pharmacological techniques

Entonox: This is a 50% mixture of nitrous oxide in oxygen. It is delivered to the patient via a demand valve through a low resistance breathing system from the cylinder after pressure reduction.

  - The advantages are that it has rapid onset and elimination.
  - The disadvantages are that, this technique is not adequate for forceps delivery, episiotomy, perineal suturing or removal of retained placenta.
  - There is also a concern about the pollution of the labor suite, though scavenging devices can be used.
  - Below 6°C the nitrous oxide may remain in the liquid phase allowing the oxygen to be exhausted first after which the cylinder will deliver only 100% nitrous oxide . Guidelines laid down for storage and handling of these cylinders should be followed.
- *Sevox:* Patient-controlled inhalation analgesia. Sevoflurane is a volatile inhalational agent commonly used during general anesthesia. Because of its short onset and offset of action, it appears to be the best-suited inhalational agent for labor analgesia and can be administered as patient-controlled inhalation analgesia. It is used in the concentration of 0.8% with oxygen and needs specialized equipment. Further, there is a concern for environmental pollution and maternal amnesia and loss of protective airway reflexes. Larger studies are needed to assess the incidence of maternal compromise.
- *Opioids:* In the UK, pethidine is commonly used for labor analgesia and can be prescribed by the midwives. It is less depressant for the baby than morphine. It is used in the dose of 100 to 150 mg IM. Pethidine 25 mg IV and Nalbuphine 10 mg IV are also used. The side effects are:
  - *Maternal:* Opioids produce nausea, vomiting, decrease in gastric motility, respiratory depression, disorientation and dis-inhibition in the mother. Gastric emptying can be a problem if the mother subsequently needs a GA for an emergency surgery.
  - *Neonatal:* The newborn can have respiratory depression, CNS depression and impaired temperature regulation. The respiratory depression has to be reversed with Naloxone 0.01mg/kg (20 mcg injected into the umbilical vein). Maximal neonatal depression is seen when the baby delivers 3 hours after the dose of pethidine is given. The neurological depression can be demonstrated up to 3 days after birth. The metabolite of pethidine (norpethidine), which is produced in the fetoplacental unit may contribute to this delayed action. Elimination half life of pethidine in the neonate is 18 hours and that of norpethidine is 60 hours. Recently short acting opioids like fentanyl and remifentanyl are being used as continuous IV infusion or as patient controlled infusion under medical supervision. These techniques are useful for patients

in whom regional blocks are contraindicated due to various reasons like bleeding diathesis, infection or allergy to local anesthetics.

- *Epidural analgesia:* This is the most effective method of producing pain relief in labor. Maternal satisfaction and neonatal outcome (as assessed by APGAR scores, umbilical cord pH, need for naloxone) are better in patients receiving epidural analgesia.
- *Subarachnoid block:* This is not a suitable technique to produce pain relief during labor as the duration of block is too short. CSE is preferred as it has the advantage of immediate pain relief and prolongation of action by using the epidural catheter.

How does the epidural analgesia (EA) work? What are the indications and contraindications to EA?

During the first stage of labor, pain is felt due to uterine contractions and dilatation of cervix. It is felt in the cutaneous distribution of T10-L1 dermatomes, i.e. lower abdomen, groins and lower back. During the second stage of labor, stretching, distension and tearing of fascia, skin and subcutaneous tissues as well as the pressure of the presenting part on the perineum cause pain (S2-S4).

These pain fibers are blocked using local anesthetic in dilute concentration so as to cause sensory block and not motor blockade.

Indications

Maternal

- Maternal request.
- Expectation of operative delivery (e.g. multiple pregnancy, malpresentation).
- Any condition where the stress response to labor is to be avoided, e.g. hypertensive, diabetic, pre-eclampsia.
- Maternal anxiety and pain leading to prolonged labor, incoordinate uterine action.
- Conditions in which general anesthesia may be life-threatening particularly if rapid regional anesthesia may be difficult to institute, (e.g. morbid obesity).

Contraindications

- Maternal refusal
- Uncorrected hypovolemia
- Local and systemic sepsis
- Bleeding diathesis
- Fetal distress.

Relative contraindications

- Expectation of significant hemorrhage
- Untreated systemic infections
- Previous back surgery.

Describe techniques of labor analgesia.

Prerequisites

- There should be adequate number of trained anesthetists, midwives and obstetricians to care for the mother's safety.
- Resuscitation equipment should be available in every labor room.
- A large bore IV line should be secured to preload the patient with fluids and to manage complications.
- Patient should be in active labour with regular painful contractions.

Studies have shown that there is no need to wait for cervical dilatation to reach to an arbitrary 4–5 cm and maternal request is a sufficient indication for initiation of labor analgesia.

1. Informed consent is obtained, and the obstetrician consulted.
2. Monitoring includes:
   a. Blood pressure every 1–2 min for 15 minutes after giving a bolus of LA.
   b. Continuous maternal heart rate during procedure.
   c. Fetal heart rate monitoring.
3. Hydration with 500–1000 mL of RL (co load).
4. The woman assumes a lateral decubitus or sitting position.
5. The epidural space is identified with a loss of resistance technique.
6. The epidural catheter is threaded 3 cm into the epidural space.
7. A test dose of 3 mL of 1.5% lidocaine or 0.25% bupivacaine with 1:200,000 epinephrine is injected as test dose. There are reservations to use epinephrine in the test dose. Another method for ruling out intravascular placement is using 100 µg of fentanyl epidurally and watch for drowsiness and euphoria, which is seen within 5–10 minutes in case the catheter is accidently placed in the intravascular space (sensitivity 92.4% and specificity 92%).
8. If test dose is negative, a bolus dose of 0.065–0.125% bupivacaine is injected to achieve a cephalad sensory level.
9. After 15–20 minutes, the block is assessed using loss of sensation to cold or pinprick. If no block is evident, the catheter should be reinserted. If the block is asymmetrical, the epidural catheter should be withdrawn 0.5–1.0 cm and an additional 3–5 mL of bupivacaine injected. If the block remains inadequate, the catheter should be reinserted.
10. Caval compression should be strictly avoided throughout labor, as the hypotension due to epidural may be aggravated due to aortocaval compression. The patient has to be nursed in sitting or lateral position.

11. Maternal blood pressure is recorded every 5–15 minutes. The fetal heart rate is monitored continuously.
12. The level of analgesia and intensity of motor block should be assessed hourly.

It is important to remember that the operator should use the technique that he/she is most familiar with, i.e. position of the patient—sitting/lateral, loss of resistance to saline or air. In a patient in labor, maternal heart rate variability from the pain of uterine contraction may confuse interpretation of heat rate response, so it is important to inject between uterine contractions. The oral intake of modest amounts of clear liquids may be allowed for uncomplicated patients in labor. The volume of liquid ingested is less important than the type of liquid ingested. However, patients with additional risk factors for aspiration (e.g. morbidly obese, diabetic, difficult airway) or patients at increased risk for operative delivery (e.g. non-reassuring fetal heart rate pattern) may have further restrictions of oral intake determined on a case-by-case basis.

Epidural block to T10 is needed for labor and to level T4 for cesarean section. The American Society of Regional Anesthesia and Pain Medicine recommendations for neuraxial anesthesia in the presence of LMWH should be followed. Needle placement should occur at least 12 hours after the LMWH dose, whereas patients receiving higher doses of LMWH (e.g. enoxaparin 1 mg/kg twice daily) will require longer delays (24 hours). If LMWH is to be given after a spinal or epidural anesthetic, then the first dose of LMWH should be administered no earlier than 24 hours postoperatively. Catheter removal should be delayed for at least 10–12 hours after a dose of LMWH. Again, subsequent dosing should not occur for at least 2 hours after catheter removal.

Discuss the role of ultrasound in epidural analgesia.

Ultrasound may be used to locate the epidural space in the obese patients and in patients with abnormal spines. It helps in guiding the needle depth and minimizes failure rates.

Discuss the drugs used for epidural analgesia and monitoring that is employed.

Bupivacaine is the local anesthetic of choice. It is long acting lipid soluble weak base. It is variably bound to alpha-1 acid glycoprotein and therefore the fetal transfer is not significant after epidural administration. Bupivacaine can be used as repeated boluses at interval of 60 to 90 minutes or as continuous infusion. 0.5% bupivacaine causes a fairly dense block and is not used for labor analgesia. It is used for interventions like forceps delivery, perineal tear suturing or for Cesarean section. 0.065–0.125% bupivacaine has been used to produce labor analgesia. The tone and power of abdominal and pelvic muscles is better preserved when dilute solutions are used. The quality of analgesia can be improved by combining bupivacaine with small dose of opioid. Maintenance of the block with a continuous infusion, or patient-controlled epidural analgesia with a background continuous infusion, provides more stable analgesia and the fluctuation in blood pressure is also reduced.

Newer drugs include the following:

Ropivacaine: It is less cardiotoxic, has a wider safety margin. The protein binding is 94%. For ambulatory analgesia, 0.08% ropivacaine is used with 2 µg of fentanyl. However, clinical studies have failed to show any advantage over bupivacaine.

Levobupivacaine: It is an S-enantiomer of bupivacaine, hence it is less cardiotoxic. More evidence is needed for this drug.

Patient-controlled epidural analgesia (PCEA): The quality of analgesia is similar to that obtained with other forms of epidural administration. PCEA is a form of epidural and, as such, has the same safety requirements. PCEA has not shown to reduce the workload of the anesthetic team, but involves additional cost of the PCA pump.

Computer-integrated PCA has been developed where the computer controls the background infusion rates depending on the previous hour's demand boluses.

Monitoring During Labor in a Patient with Epidural

- Patient's position—should be sitting, lateral or supine with a wedge under pelvis to avoid aortocaval compression.
- Arterial blood pressure—should be monitored every 5 minutes for the first 20 minutes after every bolus. Thereafter the BP should be monitored every 30 minutes.
- IV infusion should be running satisfactorily.
- FHS (Fetal Heart Sounds) to be monitored continuously.
- Mother must never be left unattended.

What do we mean by walking or ambulatory epidural?

Ambulatory or mobile epidural: This technique basically involves the use of low dose of local anesthetic in combination with a small dose of opioid. It could be a CSE technique or only epidural technique. Bupivacaine 1–2.5 mg with fentanyl 10–25 microgram has a rapid onset and lasts for 90–120 minutes. This is followed by epidural infusion. Morphine 100 microgram can be used intrathecally; but it is associated with higher incidence of nausea and vomiting. The maternal satisfaction is better in this technique because the motor power is preserved and the mother can ambulate during labor. Some studies have claimed that ambulation may help

with cervical dilatation and engagement of presenting part. However, other studies found that walking or sitting did not shorten labor duration from the time of epidural insertion to complete cervical dilatation. Though the power, coordination and reflexes in lower limb are preserved, distal proprioception and vibration sense may be affected. Hence, whenever the mother is ambulating, she should be accompanied by a responsible adult.

Enumerate the complications of epidural analgesia.

Mild Complications

- Shivering
- Intrapartum fever
- Mild itching with the use of opioids
- Backache—it is often not attributable to epidural
- Missed segment or unilateral block
- Urinary retention
- Local anesthetic toxicity.

Major Complications

- Prolonged labor: It has been shown that the mean duration of labor is longer by an hour in patients who have epidural as compared to those who do not have one. Oxytocin is used twice as frequently in the epidural group than in patients receiving IV analgesia.
- Hypotension
- Dural tap (0–2.6%)
- High block
- Bloody tap
- Neurological damage
- Assisted delivery: Increase in the rate of instrumental deliveries (forceps/vacuum) has been shown in patients receiving epidural analgesia. However, no relationship has been shown between epidural analgesia and the rates of Cesarean section.

How will you treat hypotension that occurs after epidural analgesia or anesthesia is instituted?

- Administration of oxygen with face mask.
- Left uterine displacement.
- Increased hydration.
- Elevation of lower extremities to facilitate venous return.
- Administration of small increments of 2.5 to 5 mg ephedrine, or phenylephrine 50 to 100 μg IV, if blood pressure falls more than 20% of baseline.

How would you manage an accidental dural tap?

Immediate management: Pass the epidural catheter into intrathecal space and label, and use as intrathecal catheter. OR. Reinsert the epidural one inter space higher. Run epidural as usual but beware of the possibility of intrathecal spread of local anesthetic.

Prophylactic Treatment

Bed rest: Because of risk of DVT and thromboembolism, bed rest should not be routinely encouraged in asymptomatic women.

Epidural infusion of saline will compress the dural sac and alleviate symptoms. After continuous infusion, the incidence of PDPH is marginally reduced. However, there is no strong evidence to support use of epidural infusions.

Prophylactic epidural blood patch has a lower success rate and as bacteremia is common after delivery it has to be used cautiously.

Symptomatic Treatment PDPH.

- Simple analgesics should be started round the clock.
- Plenty or oral fluids.
- Caffeine or theophyllines: These act by reducing intracranial vasodilatation. Both oral and IV caffeine are efficacious. A suitable regimen is 600 mg caffeine/day in divided doses.
- Sumatriptan (5HT agonist): This leads to cerebral vasoconstriction, and hence provides relief from symptoms. It is given 6 mg subcutaneously. However, it is expensive and can cause coronary artery spasm.
- ACTH acts by increasing concentration of β endorphin and intravascular volume.
- Abdominal binders: Relieve symptoms of PDPH by increasing epidural vein blood flow, which in turn compresses the dural sac.
- Blood patch: Epidural blood patch performed around 48 hours postpartum has a 60–90% cure rate at the first attempt. Mechanism of action: Injected blood compresses the dural sac and raises the intracranial pressure. This produces an almost instantaneous improvement in pain. Later the injected blood forms a clot over the site of the dural tear and this seals the CSF leak.

Technique of Epidural Blood Patch

Consent must be obtained. Two operators are required. Procedure should be done in aseptic technique both at epidural site and the site of bloodletting, usually the antecubital fossa. An epidural should be performed at the same or a lower space as the dural puncture with the patient in lateral position. Once the space is identified, 20–30 mL of blood is injected slowly through the epidural needle until

patient complains of pain (lancinating pain of dermatomal origin) or to a maximum of 20–25 mL. To allow the clot to form, maintain bed rest for 2 hours after procedure. A simultaneous blood sample for culture should be obtained while collecting blood for the blood patch. Post-procedure backaches are common. Other complications of epidural blood patch include neurological deficits, epilepti-form fits and cranial nerve damage.

How will you diagnose and treat bupivacaine toxicity?

Systemic and localized toxic reactions can occur due to accidental intravascular or intrathecal injection or administration of large dose of bupivacaine.

Local toxicity: Local anesthetic drugs employed rarely produce localized nerve damage.

Systemic toxicity: Systemic reactions to local anesthetics involve primarily the CNS and CVS. Bupivacaine is reported to cause rapid and profound cardiovascular depression. The cardiovascular collapse/CNS ratio is lower for bupivacaine compared to lignocaine.

Cardiovascular signs:

- Direct cardiac effects
  - Myocardial depression, cardiac dysrhythmias, and cardiotoxicity in pregnancy.
- Peripheral effects
  - Vasoconstriction at low doses
  - Vasodilatation at higher doses (hypotension)
- The range of signs and symptoms of cardiovascular toxicity include the following:
  - Chest pain
  - Shortness of breath
  - Palpitations
  - Lightheadedness
  - Diaphoresis
  - Hypotension
  - Syncope.

Central Nervous System Signs

- Initial sign and symptoms:
  - Lightheadedness
  - Dizziness
  - Visual and auditory disturbances (difficulty focusing and tinnitus)
  - Muscular twitching and tremors initially involving muscles of face and distal parts of the extremities
  - Disorientation
  - Drowsiness.
- Higher-dose symptoms
  - Often occur after an initial CNS excitation followed by a rapid CNS depression
  - Convulsions.

Allergic manifestations: Allergic manifestations of local anesthetics include rash and urticaria. Anaphylaxis due to local anesthetics is very rare but should be considered if the patient is wheezing or in respiratory distress following administration.

Management of toxicity:

- Airway management
- Cardiovascular resuscitation
- Suppression of seizure activity
- Administration of lipid emulsion: Bolus 1.5 mL/kg IV over 1 minute. Continuous infusion 0.25 mL/kg/min (500 mL over 30 minutes). The bolus can be repeated after 5 minutes and infusion doubled if cardiovascular system remains collapsed. The infusion should be continued for 30 minutes.

What are the signs of impending eclampsia?

- Headache
- Visual disturbance, such as blurring or flashing before the eyes
- Epigastric pain
- Oliguria
- Vomiting
- Sudden swelling of the face, hands or feet.

What measures can be taken to prevent seizures?

It has been proved that magnesium sulfate is superior to phenytoin in preventing eclamptic seizures. There is little evidence to support its prophylactic administration to women with mild disease. Unmeasured fetal effects of maternal eclampsia justify attempts to prevent as well as treat eclampsia in women at high risk for convulsions.

$MgSO_4$ dosage schedule for severe pre-eclampsia is same as for eclampsia.

Use the Collaborative Eclampsia Trial regimen for administration of magnesium sulphate, i.e. loading dose of 4 g should be given intravenously over 5 minutes, followed by an infusion of 1 g/hour maintained for 24 hours. Recurrent seizures should be treated with a further dose of 2–4 g given over 5 minutes. Measure Mg level at 4–6 h and adjust infusion to maintain levels between 4–7 mEq/L. $MgSO_4$ is discontinued 24 h after delivery. It is recommended not to use diazepam, phenytoin or lytic cocktail as an alternative to magnesium sulfate in eclampsia.

How would you monitor and manage $MgSO_4$ toxicity?

In therapeutic doses magnesium sulfate is associated with muscle weakness. This is as a result of decrease in the amount of acetylcholine released at motor nerve endings, decreased action of acetylcholine on the motor end plate and reduction of excitability of motor fiber membrane. Therefore, it increases the sensitivity of the mother to both the depolarizing as well as non-depolarizing muscle relaxants. When a patient is started on $MgSO_4$ it is necessary to monitor:

- Patellar reflex (disappears when plasma magnesium level reaches 10 mEq/L; warning impending Mg toxicity)
- Rate and depth of respiration (depressed at level above 10 mq/L, respiratory paralysis and arrest occurs above 12 mEq/L)
- Urine output (Mg is cleared totally by renal excretion, when there is renal insufficiency, plasma magnesium level needs to be checked periodically and dosage adjusted accordingly).

Table 12.2 Plasma magnesium levels in mEq/L and clinical effects

Sr magnesium (mEq/L)	Clinical manifestation
1.5 – 2.0	Normal plasma level
4 – 8	Therapeutic level
5 – 10	ECG changes (wide QRS, > PQ) loss of deep tendon reflexes SA and AV nodal block Respiratory paralysis
25	Cardiac arrest

Other Effects

Uterine effects: In high concentration $MgSO_4$ depresses myometrial contractility secondary to inhibition of intracellular free Ca^{++} concentration in the uterine cell (seen at serum Mg level of 8–10 mEq/L).

Neonatal effects: Magnesium crosses the placenta to achieve equilibrium in fetal serum. Magnesium sulfate is associated with a minimal risk of intracranial bleed and irregular bone deposits. It can cause a transient reversible change in fetal heart rate and should not be considered as a sign of fetal distress. The neonate may be depressed only if there is severe hypermagnesemia at delivery.

Treatment

- Withhold $MgSO_4$.
- Administer Calcium gluconate, 1 g intravenously.
- For severe respiratory depression and arrest, prompt tracheal intubation and mechanical ventilation is life-saving.

How would you demonstrate the patellar reflex?

Make the patient sit on edge of table or with legs crossed so that leg swings freely, or support the limb by placing a hand beneath the thigh so the leg hangs freely. Locate the tibial tuberosity and the lower edge of patella. With a percussion hammer, strike with the pointed end in the center of the soft space between these two hard landmarks. This stimulates the stretch receptors that trigger the local reflex arc; that triggers the quadriceps femoral muscle to contract; giving rise to sudden extension of the leg called knee jerk.

How will you treat eclampsia?

Previous regimens like lytic cocktail (IV pethidine 100 mg + phenergan 50 mg + largactil 50 mg), are no longer recommended.

Use the Collaborative Eclampsia Trial regimen for administration of magnesium sulfate, i.e. loading dose of 4 g should be given intravenously over 5 minutes, followed by an infusion of 1 g/hour maintained for 24 hours. Recurrent seizures should be treated with a further dose of 2–4 g given over 5 minutes. General care includes protection of airway and support of ventilation, intermittent IV or oral administration of antihypertensive medications to lower the blood pressure, judicious fluid management and delivery of the baby.

How will you manage patient with eclampsia; on $MgSO_4$; with fetal distress who is posted for emergency LSCS?

- Aspiration prophylaxis.
  - Pre-induction administration of nonparticulate antacid such as 0.3 M sodium citrate, 30 mL.
  - Intravenous administration of histamine receptor blocking agent 40 minutes before induction.
  - Administration of metoclopramide to decrease gastric volume 30–60 minutes before induction.
- Pre-oxygenation and denitrogenation with 100% oxygen over 3 minutes is preferred technique; however, if not feasible; patient can be advised to take 8 vital capacity breaths of 100% oxygen in one minute.
- If blood pressure is high, rapid control of blood pressure can be achieved by:
  - Hydralazine (5 mg IV aliquots up to a maximum of 20 mg)
  - Labetalol (5–10 mg IV every 10 min)
  - Oral nifedipine (sublingual nifedipine should be used with caution because of associated rapid changes in placental circulation; which may compromise fetal condition).

- In resistant cases, infusion of sodium nitroprusside and glyceryl trinitrate may be needed. But in these cases continuous arterial pressures monitoring should to be instituted.
- Consider arterial line placement before induction in patients with severe pre-eclampsia.
- Not every patient needs central venous access. It is indicated in patients with persistent oliguria and presence of pulmonary edema for fluid management in antepartum and postpartum period.
- Anticipate difficult airway, keep small size tubes, gum elastic boogie, LMA and difficult airway set ready. Plan for attenuation of response to laryngoscopy.
- Crash induction using thiopentone sodium or propofol (if blood pressure is high) in titrated doses and succinylcholine after checking ability to ventilate.
- Magnesium sulfate potentiates the effects of both depolarizing and non-depolarizing muscle relaxants. One may consider use of non-depolarizing muscle relaxant, atracurium, for maintenance of neuromuscular block once the patient has demonstrated recovery from succinylcholine or small intermittent doses of succinylcholine can be used. It is preferable to use a peripheral nerve stimulator in the intraoperative period.
- Use two-thirds of the MAC of inhalation agent to ensure adequate depth of anesthesia (MAC requirement decreases in pregnancy).
- Watch for hemodynamic changes during delivery and removal of placenta. Fluid boluses should be given if hypotension develops.
- Chance of decreased uterine contractility causing increased bleeding in view of magnesium sulfate therapy.
- Continue $MgSO_4$ during intraoperative and postoperative period.

How will you attenuate pressor response during laryngoscopy and endotracheal intubation?

You can use a wide variety of drugs and techniques to counteract the cardiovascular responses to laryngoscopy and endotracheal intubation. The ideal anesthetic drug and method should have a rapid onset of action, safe and convenient to prepare and use, and have duration of action to fit the particular situation.

Lignocaine: IV lignocaine occasionally blunts the BP response and almost never limits the HR response during intubation. If administered, lignocaine should be given at a dose of 1.5 mg/kg IV 3 minutes in young healthy patients and 4 minutes in geriatric patients prior to laryngoscopy.

Intravenous induction agents: Propofol may be a better IV induction drug than thiopental, etomidate, methohexital, or ketamine; to attenuate the BP effects of laryngoscopy and intubation, especially if the patient is hypertensive at the time of induction. Concomitant use of a low-dose opioid (fentanyl 1.5 to 3.0 µg/kg) in conjunction with an IV induction drug helps to attenuate the hemodynamic response.

Fentanyl: The recommended IV doses for attenuating the BP and HR are 5 to 6 µg/kg in young healthy patients and fentanyl 1.5 µg/kg to 3.0 µg/kg in elderly patients administered 3–4 minutes prior to laryngoscopy and endotracheal intubation. These doses may produce respiratory depression and delayed awakening in short surgical cases.

Sufentanil: 0.5–1 µg/kg given intravenously 2 minutes prior to laryngoscopy and intubation.

Alpha-agonists: Clonidine is the preferred alpha agonist to administer at a dose of 5 mg/kg (up to a maximum dose of 300 mg) orally 90 minutes prior to anesthesia induction. The drawback is that it has to be administered orally, necessitating adequate preoperative preparation time, a prolonged duration of action, possible postoperative sedation, and an increased incidence of perioperative bradycardia and hypotension.

Vasodilators: The vasodilators as a class, attenuate the BP but not HR effects of laryngoscopy and intubation. A low dose of a vasodilator combined with a short-acting beta blocker may provide a possible solution to control both BP and HR. Nitroprusside 1µg/kg to 3 µg/kg IV or Nitroglycerin 1.5 µg/kg to 2.5 µg/kg IV can be started 15 seconds prior to laryngoscopy and intubation.

Magnesium sulfate: Magnesium sulfate 40 mg/kg IV 1 minute prior to laryngoscopy attenuates the BP but not the HR response to laryngoscopy and intubation.

Calcium channel blockers as a class attenuate the BP but not HR response to laryngoscopy and intubation. Diltiazem 0.2 mg/kg to 0.3 mg/kg or verapamil 0.1 mg/kg may be given 1 minute prior to intubation.

Beta-Blockers: Labetalol 0.4 to 0.75 mg/kg IV 2 to 5 minutes or esmolol 1.5 to 2.0 mg/kg IV 2 minutes prior to laryngoscopy and intubation are recommended to attenuate the HR and BP effects.

Should Ergot alkaloids be given to the mother after delivery of the baby?

Pre-eclampsia is a relative contraindication to use of ergot alkaloids because of the risk of hypertensive crisis. In pre-eclamptic patients, uterine atony unresponsive to

oxytocin may be treated with 15-methyl prostaglandin F2 α (misoprostol) given directly in the uterus.

Are there any concerns with respect to fetal resuscitation in these cases?

Neonatal resuscitation equipment needs to be kept ready.

- Prior information to NICU and neonatologist standby is preferred.
- The neonate may be hypotonic (if there is severe hypermagnesemia at delivery).
- Problems related to preterm baby.
- Respiratory depression due to high dose of opioids given to mother during general anesthesia (Naloxone needs to be kept ready).
- In absence of a neonatologist, in case of a single anesthesiologist, it is the duty of the anesthetist to first attend to the mother and then to the neonate.

Can spinal be considered in the above case?

It is now widely recognized that epidurals provide relatively smooth control of blood pressure, maintain or improve utero-placental perfusion, optimizing fetal outcome, and eliminate the airway and hemodynamic problems associated with general anesthesia. Epidural anesthesia is the current technique of choice among most obstetric anesthetists for cesarean section (CS) in severe pre-eclampsia. Good communication between anesthetist and obstetrician would help to anticipate CS need early, and the epidural catheter inserted and topped-up in good time. However, in the case of urgent CS where it is not appropriate to wait for the time required to produce effective epidural blockade, the choice of technique lies between spinal and general anesthesia.

There has been a concern of bleeding in view of low platelet count in these patients and also an increase incidence of hypotension following spinal anesthesia. However, it is seen that the incidence of hypotension during spinal anesthesia for cesarean delivery is quite less in pre-eclamptic patients, and that this is primarily attributable to pre-eclampsia associated factors. The bleeding risk during epidural anesthesia is greater than that of lumbar puncture.

The most important factor to be considered before regional analgesia is the trend in platelet count.

As per the NICE guidelines:

- If the platelet count is < 1, 00,000/mm^3 then a clotting screen is required.
- If platelet count is > 80,000/mm^3 and clotting screen is normal—regional techniques can be used.
- If counts are < 80,000/mm^3 then potential risk and benefits should be assessed.

What is HELLP syndrome?

HELLP syndrome is a severe form of pre-eclampsia characterized by:

- **H**-Hemolysis (abnormal peripheral blood smear, and an increased bilirubin level of 1.2 mg/dL or greater)
- **EL**- elevated liver enzymes (SGOT > 70 U/L, LDH > 600U/L)
- **LP**- low platelets (< 1, 00,000/mm^3)

It can occur antepartum or postpartum. Delivery represents the only definitive treatment of HELLP syndrome. Patient with HELLP syndrome have a high incidence of serious maternal complications including disseminated intravascular coagulation, placental abruption, need for blood transfusion, pleural effusion, acute renal failure, and wound infection. After delivery of placenta, recovery from HELLP syndrome can be expected to start within 24–48 hours. These patients should be monitored in the ICU or HDU. Steroids have no role in management of HELLP syndrome.

What are the concerns in postpartum period?

Provide adequate analgesia using titrated doses of opioids like tramadol. NSAIDs should be avoided in view of renal dysfunction and low platelets. Paracetamol should be used carefully in patients with deranged liver enzymes. Monitor urine output. $MgSO_4$ should be continued for at least 24 hours postpartum. Maintain hemodynamic control with antihypertensives.

What points would you remember while giving CPCR to a pregnant lady?

- Modifications to BLS and ACLS Guidelines
- Primary ABCD Survey
- Airway
  - Apply continuous cricoid pressure during positive pressure ventilation for any unconscious pregnant woman.
- Breathing
  - Perform chest compressions higher on the sternum, mid-sternal level. This will adjust for the elevation of the diaphragm and abdominal contents caused by the gravid uterus.
- Circulation
  - Place the woman on her left side with her back angled 15° to 30° back from the left lateral position. Then start chest compressions. Or
  - Place a wedge under the woman's right side (so that she tilts toward her left side). Or
  - Have one rescuer kneel next to the woman's left side and pull the gravid uterus laterally. This maneuver will relieve pressure on the inferior vena cava.

- Defibrillation
  - No modifications in dose or pad position.
  - Defibrillation shocks transfer no significant current to the fetus.
  - Remove any fetal or uterine monitors before shock delivery.
- Secondary ABCD Survey
- Airway
  - Insert an advanced airway early in resuscitation to reduce the risk of regurgitation and aspiration.
  - Airway edema and swelling may reduce the diameter of the trachea. Be prepared to use a tracheal tube that is slightly smaller than the one you would use for a non-pregnant woman of similar size.
  - Monitor for excessive bleeding following insertion of any tube into the oropharynx or nasopharynx.
  - No modifications to intubation techniques. A provider experienced in intubation should insert the tracheal tube.
  - Effective preoxygenation is critical because hypoxia can develop quickly.
  - Rapid sequence intubation with continuous cricoid pressure is the preferred technique.
  - Agents for anesthesia or deep sedation should be selected to minimize hypotension.
- Breathing
  - No modifications in confirmation of tube placement.
  - The gravid uterus elevates the diaphragm:
    - Patients can develop hypoxemia if either oxygen demand or pulmonary function is compromised. They have less reserve because functional residual capacity and functional residual volume are decreased. Minute ventilation and tidal volume are increased.
    - Tailor ventilatory support to produce effective oxygenation and ventilation.
- Circulation
  - Follow standard ACLS recommendations for administration of all resuscitation medications.
  - Do not use the femoral vein or other lower extremity sites for venous access. Drugs administered through these sites may not reach the maternal heart unless until the fetus is delivered.
- Differential Diagnosis and Decisions
  - Identify and treat reversible causes of arrest using the acronym (BEAU-CHOPS)
    - **B**leeding/DIC
    - **E**mbolism: coronary/pulmonary/amniotic fluid embolism
    - **A**nesthetic complications
    - **U**terine atony
    - **C**ardiac disease (MI/ischemia/aortic dissection/cardiomyopathy)
    - **H**ypertension/pre-eclampsia/eclampsia
    - **O**ther differential diagnosis of standard ACLS guidelines (6 Hs and 6 Ts)
    - **P**lacenta abruptio/previa
    - **S**epsis
  - Decide whether to perform emergency hysterotomy. The use of abdominal ultrasound by a skilled operator should be considered in confirming fetal wellbeing and possible causes of the cardiac arrest, but this should not delay other treatments.
  - Emergency hysterotomy is an aggressive procedure. It may seem counterintuitive given that the key to salvage a potentially viable infant is resuscitation of the mother. But the mother cannot be resuscitated until venous return and aortic output are restored. Delivery of the baby empties the uterus, relieving both the venous obstruction and the aortic compression. The hysterotomy also allows access to the infant so that newborn resuscitation can begin.

Rescuers should take the decision of emergency hysterotomy within 4–5 minutes if BLS and ACLS interventions are ineffective.

Decision making for emergency hysterotomy

The following factors should be considered in determining the need for an emergency hysterotomy.

Consider gestational age: Although the gravid uterus reaches a size that will begin to compromise aortocaval blood flow at approximately 20 weeks of gestation, fetal viability begins at approximately 24 to 25 weeks. Portable ultrasonography may aid in determination of gestational age in experienced hands and positioning. However, one should not wait for ultrasound for the decision to perform emergency hysterotomy. For gestational age of 20 weeks or less urgent cesarean delivery is not needed because a gravid uterus of this size is unlikely to compromise maternal cardiac output significantly.

For gestational age of approximately 20 to 23 weeks, emergency hysterotomy should be performed for successful resuscitation of the mother, not the survival of the delivered infant.

For gestational age of approximately 24 to 25 weeks, emergency hysterotomy should be performed to save the life of both the mother and the infant.

What factors influence chances of infant's survival?

- Short interval between the mother's arrest and the infant's delivery.

- No sustained pre-arrest hypoxia in the mother.
- Minimal or no signs of fetal distress before the mother's cardiac arrest.
- Aggressive and effective resuscitative efforts for the mother.
- The hysterotomy is performed in a medical center with a neonatal intensive care unit.

Suggested Reading

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13 Pregnancy: Physiological Changes and Anemia

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What cardiovascular changes occur during pregnancy? What are the implications of these changes?

Cardiovascular changes begin as early as from 4th week of gestation and these occur to maximize oxygen transport to placenta. These changes are caused by increased levels of circulating estrogen and progesterone, which cause vasodilatation and a consequent fall in peripheral vascular resistance by 20%. This causes a fall in systolic and diastolic blood pressure and a reflex increase in heart rate by 15%. Stroke volume is increased by 25% and together with heart rate causes an increase of cardiac output by 50% by the third trimester. Myocardial thickness as well as volume of chambers increases resulting in left heart enlargement. Flow murmurs are quite common due to increased plasma volume and cardiac output. Cardiac output may further increase during labor and in immediate post-delivery period on account of autotransfusion. Even during this period; there may not be substantial increase in central venous pressure (CVP) or pulmonary artery occlusion pressure (PAOP) due to the vasodilatory effect of progesterone on arterioles and veins. The increased blood volume serves two purposes. First, it facilitates maternal and fetal exchange of respiratory gases, nutrients and metabolites. Second, it reduces the impact of maternal blood loss at delivery. Anatomically the heart is displaced upward and to the left by the gravid uterus. Table 13.1 summarizes the differences in cardiovascular parameters between a pregnant and a non-pregnant patient.

Table 13.1 Cardiovascular parameters in pregnant and non-pregnant patient

	Pregnant patient	Non-pregnant patient
Heart rate (bpm)	> 80	70
Cardiac output (L/min)	6.0	4.5
SVR (dyne.s.cm-5)	1200	1400
PVR (dyne.s.cm-5)	70–80	120
CVP (cm H_2O)	5–6	5-6
Colloid oncotic pressure (mm)	16–18	22

Clinical Implications

- In pregnant patient with heart disease and low cardiac reserve, the increase in the work of the heart may cause ventricular failure and pulmonary edema. In these women, further increase in cardiac workload during labor must be prevented by effective pain relief, provided by extradural analgesia. Since cardiac output is highest in the immediate postpartum period, sympathetic blockade should be maintained for few hours after delivery and then weaned off slowly.
- *Heart size and displacement:* The ECG reflects these changes including left axis deviation, ST segment depression and T wave flattening.

What is aortocaval compression? What is it's significance?

From mid-pregnancy, the enlarged uterus compresses both the inferior vena cava and the lower aorta when the patient lies supine. Obstruction of the inferior vena cava reduces venous return to the heart leading to a fall in cardiac output by as much as 20% towards term. When awake, most women are capable of compensating for the resultant decrease in stroke volume, by increasing systemic vascular resistance and heart rate. There are also alternative venous pathways: the paravertebral and azygos systems. During anesthesia, however, these compensatory mechanisms are reduced or abolished so that significant hypotension may develop rapidly. Obstruction of the lower aorta and its branches causes diminished blood flow to kidneys, uteroplacental unit and lower extremities.

Clinical Implications

No woman in late pregnancy or labor should lie supine without shifting the uterus off the great abdominopelvic vessels. During cesarean section and for other indications demanding the supine position, the uterus should be

displaced, usually to the left, by placing a rigid wedge under the right hip and or tilting the table left side down.

During regional anesthesia, the effects of aortocaval compression will be exaggerated due to a lack of compensatory reflexes subsequent to the sympathetic blockade. This can lead to profound hypotension. In extreme hypotension (or fetal compromise, such as a bradycardia), the patient can be turned to the full left lateral position.

Engorgement of veins in the epidural space due to caval compression results in a reduction in volume of the epidural and subarachnoid spaces, and reduced volumes are required to produce adequate levels of block.

What respiratory system changes occur during pregnancy? What is their significance to the anesthetist?

Respiratory changes occurring during pregnancy are to meet the increased requirement of oxygen as the oxygen consumption nearly doubles at term. There is an increase in the respiratory rate as well as the tidal volume. This also results in low $PaCO_2$ and higher PaO_2. There is reduction in FRC. Chest wall compliance and lung compliance decrease by about 25–30%.

Anatomical dead space may decrease due to airway edema especially in patients with PIH. Table 13.2 differentiates the respiratory system in a pregnant and non-pregnant woman.

Table 13.2 Respiratory system changes in pregnant and non-pregnant patient

Parameter	Pregnant patients	Non-pregnant patients
RR (per min)	16	12–14
Tidal volume (mL)	650	450
Vital capacity (mL)	3200	3200
Expiratory Reserve volume (mL)	500	700
Inspiratory reserve volume (mL)	2050	2050
FRC (mL)	1300	1600
Residual volume (mL)	800	1000
pH	7.44	7.4
$PaCO_2$ (mm Hg)	30	35
PaO_2 (mm Hg)	100-110	90–100

Clinical Implications

- Pregnant patients are more prone to hypoxia. Increased oxygen consumption and the decreased reserve due to the reduced functional residual capacity may result in rapid fall in arterial oxygen tension despite careful maternal positioning. This is more marked in obese patients and with multiple pregnancies. Further the reduced functional residual capacity causes airway closure in 50% of parturients at term in the supine position making pre-oxygenation less effective.
- Preoxygenation is essential to prevent rapid desaturation during periods of apnea.
- Rapid induction with inhalation agents: The increased minute ventilation combined with decreased functional residual capacity hastens inhalation induction or changes in depth of anesthesia. This is due to increased tidal volume, which results in large volume of inspired gas mixing with smaller volume of alveolar gas.
- Reduced compliance necessitates higher airway pressures to maintain adequate ventilation.

Why is gravida considered to be a "full stomach" patient?

Heartburn during pregnancy is very common and as many as 80% suffer from reflux at term, which is aggravated by the supine position. The gastrointestinal system is considered to return to normal 24–48 hours after delivery. The effect of progesterone results in reduction of lower esophageal sphincter pressure. Later in pregnancy the mechanical effects of gravid uterus cause an increase in intragastric pressure and a decrease in gastroesophageal angle. Placental gastrin secretion is increased, which worsens gastric acidity. All these changes put the mother at increased risk of regurgitation and aspiration of gastric contents.

Prevention: Prophylaxis in the form of a H_2 blocking drug, non-particulate sodium citrate and metoclopramide to all pregnant patients for surgery from 2nd trimester onwards is a must. The danger of aspiration is almost eliminated when regional anesthesia is used. During general anesthesia airway protection by means of a cuffed endotracheal tube is mandatory; so is rapid sequence induction from 2nd trimester of pregnancy till 48 hours postpartum. Extubation should be done with the patient awake and on their side to reduce the risk of aspiration of gastric contents.

Why is a pregnant woman said to be living in a state of "accelerated starvation"?

Metabolic functions are increased during pregnancy to provide for the demands of fetus, placenta and uterus as well as for the mother's increased basal metabolic rate and oxygen consumption. Protein metabolism is enhanced to supply substrate for maternal and fetal growth. Fat metabolism increases as evidenced by elevation in all lipid fractions in the blood. Carbohydrate metabolism, however, demonstrates the most dramatic change to meet the growing demands of the fetus. Hence, metabolically speaking, pregnant women live in a state of "accelerated starvation".

What are the hematological and coagulation changes seen in pregnancy?

During pregnancy the plasma volume increases by 45%. This increase is mediated by a direct action of progesterone and estrogen on the kidney causing the release of renin and thus an activation of the aldosterone-renin-angiotensin axis. This leads to renal sodium retention and an increase in total body water. Through an increase in renal erythropoietin production, red cell mass increases by 20%. As the increase in red cell mass is relatively smaller than that of plasma volume, the hemoglobin falls from 14 g/dL pre-pregnancy to 12 g/dL during the third trimester. The blood viscosity is reduced which may slightly decrease cardiac work. At 2-weeks post partum, the blood volume returns to pre-pregnancy levels. Table 13.3 summarizes these changes.

Table 13.3 Hematological changes in pregnant and non-pregnant patient

Parameter	Pregnant patients	Non-pregnant patients
Hematocrit	0.30–0.34	0.4
Hemoglobin (g/dL)	11–12	13–14
RBC (X 10^{12}/L)	3.8	4.2
ESR	58–70	10
WBC (X 10^{9}/L)	9	6
Plasma volume (mL/kg)	80-85	65

The white cell count rises throughout pregnancy and peaks after delivery. Pregnancy is a hypercoagulable state. Plasma concentrations of fibrinogen and all clotting factors, except XI and XIII gradually increase. Although there is an increase in platelet production, the platelet count falls because of increased platelet activity and consumption. Platelet function remains normal in pregnancy. The changes in the coagulation system are detailed in Table 13.4.

Table 13.4 Changes in coagulation system

Coagulation factor/Parameter	Changes
Fibrinogen	Increased from 2.5 g/L to 5 g/L
Factor II	Slightly increased
Factor V	Slightly increased
Factor VII	Increased 10 folds
Factor VIII	Increased 2 fold
Factor IX and X	Increased
Factor XI	Decreased 70%
Factor XII	Increased 40%
Factor XIII	Decreased 40%
Bleeding time	Unchanged
PT	Unchanged
PTT	Unchanged

Clinical Implications

- The increased circulating volume offers protection for mother and fetus from the effects of hemorrhage at delivery.
- Raised WBC count makes a diagnosis of infection more difficult. There is also an increase in ESR.
- Thromboembolic complications remain a common source of morbidity and mortality associated with pregnancy.
- In pre-eclampsia there is an increase in thromboxane A2 produced by platelets, which causes vasoconstriction and decreases uteroplacental blood flow.

Why is failed intubation more common during pregnancy?

- During pregnancy, there is capillary engorgement and edema of the upper airway down to the pharynx, false cords, glottis and arytenoids. Edema of the airway makes upper airway obstruction and bleeding more likely and may make tracheal intubation more difficult.
- The increase in chest diameter and enlarged breasts can make laryngoscopy with a standard Macintosh blade difficult. Failure to intubate the trachea is 7 times more common in the term parturient compared to non-pregnant patients.
- Anticipate difficult intubation and a need for a range of different ETT sizes. Also airway adjuncts should be readily available. A smaller diameter endotracheal tube may be required for intubation especially in cases of pre-eclampsia.

Which other systems are affected during pregnancy and what are the implications?

Renal: Renal plasma flow and glomerular filtration rate increase (about 50%) during pregnancy. Blood urea and serum creatinine are reduced by 40%. 24 hours creatinine clearance is also increased. Renin-angiotensin, aldosterone and progesterone are increased leading to water retention and a decreased plasma osmolality. The tubular re-absorption of glucose falls and glycosuria is present in 40% of parturients. Progesterone-mediated ureteric smooth muscle relaxation can lead to urinary stasis making pregnant women prone to urinary tract infections.

Clinical Implications

There is an increase in the volume of distribution for drugs and may have to be given in higher than normal dosages.

Hepatic: Liver blood flow does not increase. Plasma cholinesterase falls by 25%. The levels of ALT, AST and LDH are slightly elevated. Plasma concentrations of alkaline phosphatase are increased 3-fold as a result of placental production. Serum albumin is decreased reducing the plasma oncotic pressure.

Clinical Implications

There may be a prolongation of neuromuscular blockade after administration of succinylcholine. This is not usually clinically significant.

The total concentration of plasma protein is reduced due to the increase in plasma volume. This results in a drop in the colloid oncotic pressure, and may account for the edema seen in pregnancy.

Also the pharmacokinetics of protein bound drugs will be affected.

Endocrine: There is an increased insulin resistance during pregnancy. The placenta produces human placental lactogen, which has similar effects to growth hormone and may be the cause of maternal insulin resistance. Any carbohydrate load will cause a greater than normal increase in plasma glucose concentrations.

The thyroid gland undergoes hypertrophy during pregnancy. Increased production of thyroxin and triiodothyronine are normally balanced by increased production of thyroid binding globulin so the levels of the free hormones remain the same. Levels of parathyroid hormone tend to fall during pregnancy as does the level of serum calcium, although the level of ionized calcium remains constant.

Musculoskeletal system: The placenta produces relaxin, which causes relaxation of ligaments. This results in widening and increased mobility of the pubic and sacroiliac joints to allow passage of the fetus through the birth canal. Due to the enlarging uterus, there is a compensatory increase in the lumbar lordosis. As a result, backache is a common complaint during pregnancy.

Clinical Implication

- Due to increased lumbar lordosis, subarachnoid block or epidural become more challenging.

Others: Blood flow to nasal mucosa and peripheries is increased.

Clinical Implications

- Nasal intubation is frequently associated with epistaxis.
- Blood flow to the skin is increased resulting in warm and clammy feet. This is to dissipate heat from the metabolically active uteroplacental unit.

What are the neurological changes in a pregnant woman? Discuss the implications to the anesthetist.

The minimal alveolar concentration (MAC) of volatile anesthetics decreases during pregnancy. This may be secondary to the high levels of progesterone; and possibly an increase in β endorphin levels. There is a similar increase in sensitivity to opioids, sedatives and local anesthetics. The effects of local anesthetic drugs when used for neuraxial anesthesia and analgesia are also enhanced secondary to mechanical factors within the epidural and subarachnoid space. Compression of the inferior vena cava results in diversion of blood through the vertebral venous plexus that lies within the epidural space. This causes the epidural veins to engorge and the volume of the epidural and subarachnoid space to decrease. The pressure of the CSF is increased due to compression from the epidural veins in the epidural space. Between contractions; the pressure may be around 28 mm Hg; but during painful contractions it may rise to as much as 70 mm Hg. This altered response subsides progressively in the early postpartum period.

Clinical Implications

- As the volume of the epidural and subarachnoid space decreases, an identical volume of local anesthetic will spread more extensively in the pregnant than in the non pregnant state. Increased pressure in space facilitates diffusion of drug into dural space. Venous congestion of lateral foramina decreases loss of local anesthetic along the dural nerves. During the last months of gestation, approximately two-thirds of the normal dose is adequate.
- As the CSF pressure rises during contraction, it is probably safer not to advance an epidural or spinal needle during contractions for risk of puncturing the dura and expulsion of CSF at high pressure.
- Epidural veins are dilated by the action of progesterone and cannulation of an epidural vein when performing epidural insertion ('a bloody tap') is also more common.

What are the functions of placenta? What factors affect the transfer of drugs from maternal to fetal side?

The placenta is a vital barrier and link between maternal and fetal circulations. The barrier between fetal and maternal circulations is two cells thick: the fetal capillary endothelium and its covering syncytial trophoblast. It is an imperfect barrier and substances do cross from the mother to fetus.

Functions

- *Gas exchange:* O_2 and CO_2 exchange.
- *Hormonal synthesis:* Placenta synthesizes and releases estrogen, progesterone, chorionic gonadotropin, prolactin, prostaglandins.
- *Nutrient supply:* Water, electrolytes, minerals.
- Detoxification of drugs and substances.
- Transfer of immunity from mother to fetus by way of immunoglobulins.

Placental transport mechanisms are:

- *Simple diffusion:* O_2 and CO_2 transport occurs due to the difference between partial pressures on both sides. Fatty acids are also transported by means of simple diffusion.
- *Secondary active transport:* Amino acids are transferred mostly as linked carriers. Sodium down its concentration gradient drags amino acids with it.
- *Pinocytosis:* Placenta is impermeable to proteins, only IgG is transported.
- *Facilitated diffusion:* Glucose.
- *Bulk transport:* Water and electrolytes.

Factors affecting placental transfer of drugs are:

- *Lipid solubility:* The placental membrane is freely permeable to lipid soluble substances, which undergo flow dependent transfer. Higher the lipid solubility, higher the transfer of drugs.
- *Molecular weight:* Drugs with smaller molecular weight diffuse easily (< 600 Da).
- *Degree of ionization:* Ionized form will not cross the barrier easily. The degree of ionization of acidic drugs is greater on the maternal side and lower on the fetal side. The converse applies to basic drugs.
- *Protein binding:* Protein bound drugs will not diffuse easily, only free drug would cross the placental barrier easily. Acidosis reduces the protein binding of local anesthetic agents. Reduced albumin concentration increases the proportion of unbound drug.
- *Maternal fetal concentration gradient:* The rate of transfer is governed by Fick's law of diffusion, when the transfer is by simple diffusion. Note: Fetal maternal concentration is used as an index for transfer of drugs.

Anesthetic Drugs

Opioids: All opioids cross the placenta in significant amounts. They are weak bases, bound to α1-glycoprotein.

Pethidine: Longer half-life is due to its active metabolite norpethidine, which may lead to respiratory depression in the neonate.

Morphine: It is poorly lipid soluble but readily crosses the placenta due to low protein binding.

Fentanyl: It is highly lipid soluble and albumin bound, so crosses the placental barrier easily.

IV induction agents: Sodium thiopentone is highly lipid soluble, weakly acidic, 75% protein bound and less than 50% ionized at physiological pH. It crosses the placenta easily with an F/M ratio of 0.4–1.1. Doses of > 8 mg/kg produce neonatal depression, whereas lower doses produce no significant neonatal effects providing induction to delivery time is < 5 minutes.

Propofol: It is highly protein bound and lipophilic. F/M ratio of 0.6–0.8

Inhalational agents: These agents are highly lipid soluble with low molecular weights.

Muscle relaxants: These are quaternary ammonium compounds and fully ionized. These drugs are fully ionized as well as have low lipid solubility; hence they do not cross the placenta.

Local anesthetics: These drugs have low molecular weights (< 600 Da) and also are lipid soluble. Different drugs have different protein binding.

Bupivacaine: Because of its high protein binding (96%) crosses the placenta in lesser concentration then lignocaine (65%). Transfer to the fetus also is dependent on the site of administration (paracervical route concentration will be higher than epidural).

Anticholinergics: Atropine passes through the placental barrier unlike glycopyrrolate.

Benzodiazepines: Diazepam crosses the placenta easily (highly lipid soluble and nonionized). F/M ratio is 1, as compared to midazolam with F/M ratio 0.76.

Anemia in Pregnancy

What are the causes of anemia of pregnancy?

In India, incidence of anemia in pregnancy is as high as 40–80%. The common causes are discussed below:

Physiological anaemia of pregnancy: Blood volume begins to increase in the 6th week of gestation and by the end of pregnancy it reaches approximately 40–50% more than in the pre-pregnant state. Red cell mass increases as much as 20–30% above pre-pregnancy levels. The plasma volume increase is greater than the increase in red blood cell mass, leading to hemodilution, resulting in 'physiological anemia of pregnancy'.

Acquired causes: The most common cause of anaemia in India is nutritional deficiency. Nutritional deficiency may be of iron or folic acid/vitamin B 12.

Iron deficiency anemia (60%): Most common type of anemia in pregnancy. Iron stores are not adequate to meet the demands of pregnancy. The body is unable to keep up with the blood volume expansion in the 2nd and 3rd trimester resulting in iron deficiency anemia. This can be prevented by encouraging pregnant women to supplement their diet with 60 mg/day of elemental iron.

Macrocytic anemia (10%): This is due to deficiency of folic acid and or vitamin B12. Patients with vitamin B12 deficiency need further workup in addition to serum levels of vitamin B12, to determine the level of intrinsic factor to exclude pernicious anemia. Folic acid requirements are increased during pregnancy. Folate deficiency in pregnancy is often associated with iron deficiency, though it is less common than iron deficiency. Folic acid is transported to the placenta by active transport. An increase in MCV can be suggestive of folate deficiency.

Infections: Although rare, anemia can be caused by infections such as malaria, parvovirus, cytomegalovirus, HIV, hepatitis viruses, Epstein-Barr virus. If the patient's history suggests exposure to any of these infectious agents, appropriate laboratory studies should be performed.

Aplastic or hypoplastic anemia: Pregnancy induces erythroid hypoplasia and in such cases termination of pregnancy may be indicated.

Anemia of chronic disease: Anemia can be a feature associated with chronic disease, such as neoplasm, chronic infection (tuberculosis), chronic renal failure, cancer, chemotherapy and HIV infection. During pregnancy anemia worsens due to the associated medical condition.

HELLP syndrome: Discussed in the section on anesthetic considerations in patients with pregnancy, induced hypertension.

Hereditary: Inherited disorders, such as thalassemias, sickle cell anemia, and hereditary spherocytosis, can also be the cause of anemia.

Why is the incidence of anaemia in pregnancy in India very high?

In India, many people are vegetarian and therefore have Vitamin B12 deficiency. In addition to it, the Indian subcontinent has a high content of phytates in their staple diet. Chapatti contains high amounts of inositol hexaphosphate (phytate) and inositol pentaphosphate; these are known inhibitors of iron absorption.

How can one prevent anemia in pregnancy?

India was the first developing country to take up a National Program to prevent anemia among pregnant women and children. The National Anemia Prophylaxis Program of iron and folic acid distribution to all pregnant women in India through the primary health care system was evolved and implemented from 1972.

Ensuring good nutrition, balanced diet and supplementation can prevent anemia in pregnant women.

The diet and supplementation should include the following:
Iron rich foods: Pulses, cereals, jaggery, beet root, green leafy vegetables, meat, liver, egg, fish, legumes, dry beans, and iron enriched white breads.

Iron supplements: National nutritional anemia prophylaxis program advices 60–120 mg of elemental iron.

Folic acid supplements: Women who are pregnant and breast-feeding should take 500 mcg – 1 mg of folic acid a day. It is one of the most important nutrients to have during pregnancy and before conception, as women deficient in folic acid have a greater risk of having a baby with a neural tube defect, such as spina bifida.

Folic acid rich foods: Green leafy vegetables, citrus and kiwi fruit, peas, beans, cauliflower, fortified breakfast cereals, fortified bread. Several observational and controlled trials have shown that neural tube defects can be reduced by 80% or more when folic acid supplementation is started before conception

Vitamin B12 supplements: Vitamin B12 supplement required is 3 mcg/day. Treatment of vitamin B12 deficiency includes 0.1 mg/day for 1 week, followed by 6 weeks of continued therapy to reach a total administration of 2 mg.

Vitamin B12 rich food: Meat, egg, milk.

Severity of Anemia

Anemia is defined as a qualitative or quantitative deficiency of hemoglobin. It can be classified as follows:

- Mild anemia — Hemoglobin 9–11 g %
- Moderate anemia — Hemoglobin 7–9 g %
- Severe anemia — Hemoglobin 4–7 g %
- Very severe anemia — Hemoglobin < 4 g %

What are the compensatory mechanisms in anemia?

1. Increase in cardiac output
2. Rightward shift of oxygen dissociation curve
3. Decrease in blood viscosity
4. Increase in 2,3 DPG concentration in red blood cells
5. Release of renal erythropoietin to stimulate erythroid precursors in bone marrow.

The tissue oxygenation is not impaired in physiological and chronic anemia due to compensatory mechanisms but sudden onset severe anemia may precipitate cardiac failure and pulmonary edema.

What are the causes of concern in anemia associated with pregnancy?

Anemia is a known risk factor for many maternal and fetal complications.

Maternal: Severe anemia is associated with a higher mortality rate and may be related to a poor ability to withstand the adverse effects of excessive blood loss. Also it leads to an increased risk of infection.

- Preterm labor
- Poor weight gain
- Dysfunctional labor
- Intranatal hemorrhage
- Anesthesia risk
- Cardiac failure
- Postnatal sepsis.

Fetal: There is an increased risk of low birth weight due to poor fetal growth and preterm birth.

- Poor Apgar score
- Fetal distress requiring prolonged resuscitation.

Neonatal and infant

- Anemia due to poor reserve
- Higher prevalence of failure to thrive
- Poorer intellectual developmental milestones
- Cardiovascular morbidities.

What are the risk factors for anemia in pregnancy?

Risk factors for anemia in pregnancy are:

Sociodemographic factors: Age, level of formal education, marital status, areas of residence.

Obstetrical factors: Gravidity, parity, multiple pregnancies.

Behavioral factors: Smoking, tobacco, alcohol usage.

Medical conditions: Diabetes, renal or cardiorespiratory diseases, TB.

How would a patient with anemia in pregnancy present?

- Asymptomatic
- Vague complaints of ill health
- Fatigue
- Loss of appetite
- Dyspnea
- Palpitation

Clinical examination may reveal pallor, pale nails, koilonychia, and pale tongue.

How would you investigate a patient presenting with anemia?

Hemoglobin estimation: Despite limitation, it is a useful method for detecting anemia.

Study of peripheral smear:

- *Iron deficiency anemia:* The peripheral smear looks pale; there is hypochromia (large central vacuoles), and microcytosis (small red cells), and anisocytosis (varying size of RBCs).
- *Megaloblastic anemia:* There is macrocytosis (MCV is greater than > 95 femtoliters), and fully hemoglobinized red blood cells. In addition to macrocytes, oval macrocytes and hypersegmented neutrophils are seen in various types of macrocytic anemia. When the anemia is more severe, there may be marked poikilocytosis, with teardrop poikilocytes and red-cell fragments.
- *Normochromic normocytic anemia* is seen in patients with chronic diseases, such as long standing infections (TB), malignancy and renal failure. In hemolytic anemia there would be polychromatic cells, stippled cells and target cells. In patients with sickle cell disease, peripheral smear will reveal sickle cells and large number of reticulocytes.
- Microspherocytes (cells that are both hyperchromic and significantly reduced in size and therefore in diameter) may be present in low numbers in patients with a spherocytic hemolytic anemia but are also characteristic of burns and of microangiopathic hemolytic anemia.

Following special laboratory investigations may be required for establishing the cause of anemia:

- Total iron binding capacity (TIBC)
- Serum ferritin concentration (SF)
- Serum folic acid levels
- Serum iron concentration
- Bone marrow studies
- Sickle test
- Hb electrophoresis

The serum iron, total iron-binding capacity, and serum ferritin are the best indicators of iron available for erythropoiesis.

How do you treat iron deficiency anemia?

A variety of options are available to treat iron deficiency Anemia. Depending on the need of the patient one of the following may be chosen:

Oral iron is safe, inexpensive and effective way to administer iron. Oral route should be the route of choice in routine cases. Ferrous sulfate is least expensive and allows more elemental iron absorbed per gram administered. Oral iron must be continued for 3–6 months after hemoglobin levels have become normal.

Parenteral route of iron therapy: The indications for parenteral iron therapy are:

- Patient unable to tolerate oral iron.
- Patients suffering from inflammatory bowel disease.
- Non-compliant patient.

- Patient near term.
- Failure of oral therapy.

Parenteral iron has the advantage of causing less gastrointestinal discomfort and inconvenience. Intramuscular iron should be administered in upper outer quadrant of buttock using a Z-track technique. Intravenous iron is expensive and has greater morbidity than oral preparations of iron. Intravenous route should be reserved for those who do not wish to have frequent intramuscular injections.

Iron-sorbitol-citric acid complex (75 mg) is used for intramuscular route only.

Iron-dextran (Each mL contains 50 mg of elemental iron) can be used both by intramuscular and intravenous route. It is available as 50 mg/mL and in 1 mL and 2 mL vials. The administration is done as follows:

- Test dose: A test dose (25 mg Fe) is required for 1st exposure. This should be diluted in 50–100 ml of Normal Saline and infused over 15–20 minutes. Monitor HR, RR, and BP every 15 minutes for 1–2 hours after test dose is started.
- Subsequently it can either be administered as one large dose (diluted in 500–1000 mL NS and infused over 4 to 8 hours) or divided into many smaller doses given 1–3 times per week.
- Each 100 mg dose may be administered undiluted as IV push.
- Alternatively each 100 mg dose is diluted in 250 ml NS and infused over 30–60 minutes.

Iron sucrose is given as an infusion. It is available as 5 ml vials of 20 mg/mL strength.

- Each 5 mL contains 100 mg (20 mg/mL) of elemental iron as iron sucrose.
- No test dose is required.
- Each 5 mL vial may be diluted in a maximum 100 mL Normal Saline and administered over at least 20 minutes.

Total dose infusion (TDI): Iron can be given intravenously at one shot as total dose infusion (TDI). Utmost caution is needed for total dose iron therapy via intravenous route because of severe anaphylactic reaction that may occur. Therefore, total dose of iron therapy by intravenous route should only be given in a hospital setting where facilities are available to manage severe reaction.

The total dose is calculated as follows:
Total dose of iron in mg (for TDI) = (15–patients Hb %) × body weight in Kg × 3.

Nephritis, cardiorespiratory disease, allergy are contraindications to parenteral iron therapy.

Blood/Packed cell transfusions: Transfusion of packed RBC is reserved for patients with either significant acute bleeding or patients in danger of hypoxia and or coronary insufficiency or anemia discovered at term.

Response to iron therapy can be documented by an increase in reticulocytes, 5–10 days after the initiation of therapy. The hemoglobin concentration increases about 1 g/dL weekly.

What are the side effects and complications of parenteral iron therapy?

- Intramuscular injections can cause pain and staining of the skin at injection site, myalgia, arthralgia and may cause injection abscess.
- Intravenous administration can cause pain in the vein, flushing, and metallic taste in mouth.
- *Allergic reactions:* With intravenous iron therapy reactions can range from rash to anaphylaxis and death. These are more common with iron dextran preparation.
- Abdominal pain with nausea and vomiting.
- Headache, dizziness, disorientation, seizures.
- Wheezing, dyspnea, respiratory arrest.
- Chest pain, tachycardia, vascular collapse.
- Iron toxicity presents with nausea, dizziness, and a sudden drop in blood pressure.

What are the important principles while anesthetizing an anemic patient?

Tissue oxygenation in anemia is hindered by the low oxygen-carrying capacity of the blood. However in most cases, compensatory changes occur, which tend to restore the balance. (Available oxygen = Cardiac output × Arterial oxygen content). In pregnant patient as the cardiovascular system is already overworked due to elevated cardiac output, heart rate and increased myocardial work. Presence of anemia further exacerbates the workload and can land the patient in high output cardiac failure.

In these patients, severe desaturation due to reduced oxygen carrying capacity is very likely; due to excess demand and decreased supply. Since maternal oxygenation directly affects fetal oxygenation, it is important that meticulous attention is paid during the conduct of labor analgesia/regional or general anesthesia to prevent any harm to the baby. Neonatologist skilled in resuscitation forms an important part of the team during delivery.

How does anemia affect oxygen carrying capacity of the blood?

Oxygen carrying capacity of blood is calculated as: $CaO_2 = (Hb) \times 1.34 \times (\%SaO_2/100) + (0.003 \times PaO_2)$.

1 gram of Hb binds to 1.34 mL O_2. In blood 97% of oxygen is bound to hemoglobin, and only 3% is dissolved in plasma

which decides the PaO_2. 3 mL of oxygen is dissolved in 1 liter of plasma. Normally, if Hb is considered as 15 g/dL, then oxygen carrying capacity is 19–20 mL/100 mL of blood at SaO_2 97–100%, of which 5 mL/100mL is extracted by the tissues, leaving a large reserve. But if the Hb is 5 g/dL, then the oxygen content of blood will only be 6.7 mL/100 mL of blood. Such patients cannot compensate for further reductions in cardiac output, hemoglobin concentration or oxygen saturation. Pregnant women have higher oxygen consumption, hence tolerate severe anemia badly.

What is the preoperative transfusion trigger in anemic patient?

Healthy myocardium can compensate for low Hb (7–8 gm/dL) and hematocrit (21–24%). But transfusion is definitely indicated for Hb < 6 g/dL and rarely indicated for Hb > 10 g/dL in a stable patient. For a woman in labor or in postpartum period, the decision of transfusion is based on patient's clinical condition, ongoing blood loss, existing comorbidities if the Hb ranges from 7–9 g/dL.

Discuss preoperative considerations in an anemic pregnant patient.

In the assessment of the pregnant anemic patient for anesthesia and surgery, the cause of the anemia is of prime importance. Unexpected anemia should be investigated and treated. Appropriate oral or parenteral therapy with iron, vitamin B12 or folic acid takes some time and is thus practical only in patients requiring purely elective surgery, which can be deferred to a reasonable time period. Recombinant erythropoietin can also be considered in the treatment but its role is yet to be established.

In contrast, when emergency surgery is required, there is little time to diagnose or treat the anemia beforehand. Transfusion of whole blood or packed red cells would raise the total circulating volume but it should be done at least 24 hours prior to surgery to allow restoration of 2,3 DPG levels.

What are the anesthetic goals in anemic pregnant patient?

The aim of anesthesia is to minimize blood loss while maintaining tissue oxygenation.

- Good IV access for maintenance of intravascular volume.
- Careful positioning to reduce venous pressure
- Scrupulous surgical technique.
- Maintain cardiac output, avoid myocardial depression and hypotension
- Adequate FiO_2 to maintain SpO_2.
- Maintenance of adequate oxygen carrying capacity by using blood transfusion if needed.

Discuss induction and maintenance of anesthesia in an anemic pregnant patient.

- *Adequate pre-oxygenation is essential:* FRC is decreased, which may result in rapid and significant fall inPaO_2 during a period of apnea. This may not be well tolerated by a severely anemic patient.
- *Minimize the reduction in cardiac output:* Titrated doses of induction agents known to have little effect on myocardial contractility.
- Decreases in cardiac output due to the high levels of spinal anesthesia required for cesarian section may be detrimental in patients with severe anemia. General anesthesia with careful titration of induction agent may be preferred.
- Avoid hyperventilation, hypovolemia, hypothermia, acidosis and of peripheral pooling of blood due to adverse posturing.
- Optimal blood replacement with goals of transfusion to achieve Hb > 7–8 g/dL (or HbA > 40% of the total Hb in case of abnormal Hb).
- Avoid or vigorously treat the conditions, which increase oxygen demand, such as shivering or fever.
- Nitrous oxide should be used with caution in patients with folate and vitamin B12 deficiency.

Discuss postoperative care in anemic pregnant patient.

The aim is again to maximize oxygen delivery and minimize blood loss. Patients should be closely monitored (hemodynamics) in a high-dependency unit. Provide oxygen supplementation, maintain euvolemia, good pain relief and treat any infections aggressively.

Discuss in brief the management of Elective Cesarean Section in a patient with a normal pregnancy.

Obstetric anesthesia is a unique situation of providing care for the mother and the unborn baby.

Premedication

Aspiration prophylaxis: Although the risk of aspiration is greater during general anesthesia than during regional anesthesia, a patient may still vomit and aspirate gastric contents while receiving regional anesthesia. Therefore, pharmacologic prophylaxis is administered to all patients regardless of type of anesthesia planned. The aim of the prophylaxis is to decrease gastric volume and increase gastric pH. Suspension antacids; i.e. those which contain particulate matter, e.g. Gelusil; may cause pulmonary injury similar to that caused by the aspiration of gastric acid; and are not recommended. A clear antacid, such as 0.3 M non-particulate sodium citrate (30 mL) is preferred. The administration of an H2-receptor antagonist increases gastric pH; but it will not alter the pH of existing gastric contents. Therefore, a combination of therapies, which will

increase pH as well as alter pH of preexisting gastric content is more useful. Intravenous administration of ranitidine 50 mg and the oral administration of sodium citrate 30 mL results in a greater increase in gastric pH than the administration of sodium citrate alone provided both are administered at least 30 minutes prior to intubation.

Anxiolysis: Small dose of intravenous benzodiazepine (e.g. midazolam 0.5 to 2 mg) and or an opioid (e.g. fentanyl 25 to 50 µg) result in minimal fetal and neonatal depression and may be safely given. During regional anesthesia, major disadvantage of the benzodiazepines is their potential for amnesia; and it should be avoided if the mother wants to remember her childbirth experience.

Antisialogogues: Routine use of glycopyrrolate is not recommended.

Choice of Anesthesia

Regional

Regional anesthesia is preferred over general anesthesia for elective cesarian unless contraindicated.

Advantages: Avoidance of multiple drugs, mother remains awake to witness the childbirth, early breastfeeding can be started.

Regional technique can be safely performed, if the platelet count is more than 70–75000/mm^3 in a parturient, in absence of coexisting coagulopathy.

Single shot spinal anesthesia can be given using 25 or 24 gauge Sprotte needle or 27 or 25 gauge Whitacre needle. It can be performed in lateral or sitting position to ensure sensory blockade up to T4-T6.

Table 13.5 Anesthetic drugs, their doses and effects

Drugs Local anesthetics	Dose in mg for SAB	Duration of action	Remark
Lidocaine	60–75	45–75 minutes	Faster onset, avoided for TEN (Transient neurological syndrome)
Bupivacaine	7.5–15.0	60–120 minutes	Most widely used
Ropivacaine	15-25	120 minutes	Less motor blockade
Levo-bupivacaine	4-12	60–120 minutes	S enantiomer of bupivacaine, less cardiotoxic than racemic bupivacaine
Adjuvant drugs			
Epinephrine	0.1–0.2		
Morphine	0.1–0.25	6-18 hours	
Fentanyl	0.010–0.025	3–4 hours	

Hypotension Prophylaxis

Hypotension in obstetric patients is defined as a decrease in systolic blood pressure of at least 25% or systolic blood pressure less than 100 mm Hg. It is perhaps the most common complication of regional anesthesia in obstetric patients. Hypotension results from increased venous capacitance and pooling of a major portion of the blood volume in the lower extremities and the splanchnic bed; and decreased systemic vascular resistance.

Measures to prevent hypotension include:

- Administration of fluids before the administration of regional anesthesia
- Left uterine displacement
- Administration of a prophylactic vasopressor.

It is recommended to administer Ringer's lactate or Normal saline 15–20 mL/kg (1000–1500 mL of crystalloid or 500–1000 ml of colloid) half an hour prior to regional anesthesia. In patients undergoing elective cesarean delivery under spinal anesthesia, the timing of fluid loading (before: preload or during induction of spinal anesthesia: coload) does not have an impact on the incidence of hypotension. Therefore, the surgery should not be delayed in order to deliver a preload of fluid. Regardless of the fluid loading strategy, either prophylactic or therapeutic vasopressors may be required in a significant proportion of patients.

Vasopressors: Phenylephrine bolus 50–100 µg intravenously may be associated with a lower incidence of intraoperative nausea and vomiting, and higher umbilical artery pH and base excess; compared with ephedrine. However, the difference in pH is small and unlikely to be clinically relevant in low-risk deliveries. It may be more useful in patients undergoing emergency LSCS for fetal distress.

Epidural Anesthesia: It is possible to extend the block by giving 'top-up' dose of local anesthetic agents to a patient having indwelling epidural catheter inserted for labor analgesia. Additional (5-mL boluses of 2% lidocaine + 1:400,000 epinephrine or 5-mL boluses of 0.5% bupivacaine or 0.5% ropivacaine may be given to attain motor blockade and sensory level of T4, for surgical procedures.

Combined spinal-epidural anesthesia: Subarachnoid injection allows quick and dense block and then it is possible to extend the block through epidural catheter. This also allows postoperative epidural analgesia to be administered.

General Anesthesia (GA)

Indications: Maternal refusal of regional anesthesia, fetal distress in the absence of preexisting epidural catheter, failed regional anesthesia, significant coagulopathy, acute maternal hypovolemia.

Conduct of GA includes:

- H2 receptor antagonist or proton pump inhibitor and/or metoclopramide intravenously.
- Clear antacid orally.
- Left uterine displacement.
- Application of monitors.
- Denitrogenation (administration of 100% oxygen)
- Intravenous induction: Thiobarbiturate, 5–7 mg/kg, propofol 2 mg/kg and succinylcholine 1–2 mg/kg OR rocuronium 0.6–1.2 mg/kg.
- Intubation with a 6.0- to 7.0-mm cuffed endotracheal tube.
- Preparedness for difficult intubation is always mandatory.
- Nondepolarizing neuromuscular blocking agent.
- intermittent positive pressure ventilation using 30 to 50% nitrous oxide in oxygen and a low concentration (e.g. 0.5 minimum alveolar concentration [MAC]) of a volatile halogenated agent.
- After delivery of the fetus, concentration of nitrous oxide can be increased, with or without a low concentration of a volatile halogenated agent; and opioids (Fentanyl 2 mcg/kg) can be administered.
- Patient should be extubated once wide awake.

Continuous spinal anesthesia: This technique lost popularity because of post-dural puncture headache. Also the microcatheters are associated with large incidence of kinking and breakage.

Intraoperative Monitoring:

- NIBP, pulse oximetry, capnometry are basic standard monitoring; and are mandatory.
- The ASA requires the continuous electrocardiogram during the administration of anesthesia for surgery. ST-segment depression in the lateral leads may be seen in 25 to 65% of patients after the delivery during cesarean section. The possible causes are acute hypervolemia, tachycardia, venous air embolism, coronary vasospasm, vasopressor administration, and or amniotic fluid embolism.
- *Other (optional) monitoring includes:* Fetal Heart Rate monitoring using scalp ECG electrodes, Doppler ultrasound for air embolism in patients with intracardiac shunts, noninvasive cardiac output monitoring in indicated cases.

Replacement of blood loss: In normal vaginal delivery, about 500 mL of blood loss is normally expected. This corresponds to the average amount of blood conserved during pregnancy; as about 50 mL (10–80 mL) of blood is lost during normal menstrual cycle. During cesarean section, the accepted average blood loss is one liter in a non-anemic patient.

Pediatrician or neonatologist should attend the cesarean section for care of the neonate. If anesthesiologist is the only physician providing care for mother and the neonate, then maternal safety is considered essential.

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14 Emergency Lower Segment Cesarean Section (LSCS)

S Bakshi, R Ambulkar, J Doctor

A 22-year-old female with 39 weeks amenorrhea is admitted to labor ward with labor pains. On examination, fetal heart rate is 100/min. Consent for emergency LSCS has been taken, patient is to be shifted to theater. Discuss management.

Discuss fetal monitoring during labor?

Fetal monitoring begins in the antepartum period. The important consideration in deciding when to begin antepartum testing is the prognosis for neonatal survival and the severity of maternal disease. The biophysical profile is used for fetal evaluation, which includes the non-stress test, fetal breathing, fetal movement, fetal tone, amniotic fluid volume (above variables are derived during sonography). Fetal well-being is dependent on an intact uteroplacental unit. Simultaneous monitoring of fetal heart rate (FHR) and uterine contraction helps in detection of fetal distress. The normal FHR is 120 to 160 b/min with beat to beat variability. If the baseline fetal heart rate is less than 110 bpm, it is termed bradycardia. If the baseline rate is greater than 160 bpm it is termed tachycardia.

Beat to beat variability is an index of cardiovascular function and is regulated by the cardiovascular system, such irregularity is defined as baseline variability.

Abnormalities in FHR, such as bradycardia, tachycardia, dysrhythmias, decreased or absent beat to beat variability or transient decelerations can be a sign of fetal asphyxia. Transient or periodic decelerations in FHR fall into the following three categories depending upon their time with respect to maternal contractions.

Early, or type 1, decelerations: Decrease in fetal heart rate, which occurs simultaneously with the onset, peak and end of contraction. This is due to the vagal stimulus caused by the fetal head compression and usually not associated with fetal hypoxia.

Late, or type 2, decelerations: A gradual decrease in FHR is seen which begins after the onset of a contraction and lasts beyond the end of a contraction. This is suggestive of uteroplacental insufficiency and requires prompt attention.

Variable, or type 3, decelerations: Changes in FHR, which is unrelated to uterine contractions. They usually result from umbilical cord compression. They are further classified as;

Mild: FHR 80 bpm lasting less than 30 seconds, usually insignificant

Moderate: FHR less than 70 bpm for 30 to 60 seconds, signify mild hypoxia

Severe: FHR less than 70 bpm lasting more than 60 seconds, indicate fetal acidosis.

If the FHR tracing is normal, 99% of the time the fetus is not depressed. In contrast, if FHR tracing is abnormal in 50% cases, the infants are normal with no umbilical cord acidosis. Fetal scalp capillary blood pH, FHR accelerations and fetal pulse oximetry are additional methods to assess fetal wellbeing.

How do you diagnose fetal distress and what are the causes?

The term fetal distress is too broad and vague and based on the fetal heart rate patterns. Normal labor is a process of repeated fetal hypoxic events, which results in acidemia. Normal parturition is an asphyxiating event for the fetus and identification of fetal distress based upon fetal heart rate is imprecise and controversial. The NICHD fetal monitoring workshop classifies the fetal heart rate patterns as follows:

- Normal
  - Baseline 110–160 bpm
  - Variability 6–25 bpm
  - Accelerations present
  - No decelerations
- Intermediate
  - No consensus
- Severely abnormal
  - Recurrent late or variable decelerations with zero variability
  - Substantial bradycardia with zero variability.

There has been consensus about the definitions of fetal heart rate at the extremes of normal and severely abnormal patterns.

Causes of fetal distress

- Maternal hypotension
- Placental abruption
- Umbilical cord compression (secondary to cord prolapse/ oligohydramnios)
- Pregnancy-induced hypertension
- Uterine hypertonus.

What are the implications of meconium stained amniotic fluid?

Three theories have been suggested to explain fetal passage of meconium:

1. The pathological explanation proposes that the fetus passes meconium in response to hypoxia, and that meconium signals fetal compromise.
2. In utero passage of meconium may represent normal gastrointestinal maturation under neural control.
3. Meconium passage could also follow vagal stimulation from common but transient umbilical cord entrapment and resultant increased peristalsis.

In conclusion, high incidence of meconium observed in the amniotic fluid during labor often represents fetal passage of gastrointestinal contents in conjunction with normal physiological processes.

When fetal acidemia supervenes, in cases of fetal hypercarbia, the fetal respiration is stimulated leading to aspiration of meconium into the alveoli. This leads to secondary alveolar damage. Thus meconium in amniotic fluid is an environmental hazard for the fetus.

What preoperative medications would you give this patient?

Pregnant women are more prone to aspiration of gastric contents after induction of general anesthesia than non-pregnant patients. All obstetric patients are at increased risk of pulmonary aspiration and should receive aspiration pharmacoprophylaxis, which includes non-particulate antacid, such as 0.3 M sodium citrate, 30 milliliters. This has duration of action of 40 to 60 minutes. Intravenous administration of histamine receptor (H2) blocking agents 40 minutes before airway management is a useful adjuncts. Omeprazole, a proton pump inhibitor and antisecretory agent require 40 minutes to reduce gastric acidity. Ranitidine is preferred.

Administration of metoclopramide decreases gastric volume as a prokinetic agent within 30–60 minutes. It crosses the placenta but has not been shown to have any lasting adverse neurobehavioral effects on the newborn.

Would you insert a nasogastric or orogastric tube preoperatively?

The use of a nasogastric or orogastric tube prior to induction of anesthesia, to enable physical emptying of the stomach has been suggested; particularly if a parturient has had opiates in labor and is therefore most likely to have a full stomach.

Physical removal of gastric contents would reduce the volume by a greater degree than pharmacological methods, but the remaining volume may still make the pregnant woman vulnerable to aspiration.

The efficacy of cricoid pressure in presence of gastric tube is controversial. Hence, the gastric tube could be pulled out prior to induction. There exists another school of thought that in a parturient at high risk of aspiration, it may be more practical to insert a nasogastric or orogastric tube after induction of anesthesia, once the airway is secured. As the risk of aspiration continues into the recovery period, this may be a logical approach and one that is more acceptable to the mother.

What do we understand by Mendelson's syndrome? How will you prevent it?

Mendelson was the first who described acute chemical aspiration pneumonitis. Patients are considered at risk for aspiration pneumonitis if gastric volume exceeds 25 mL (0.4 mL/kg) and pH is less than 2.5.

Early signs include:

- Cyanosis
- Tachycardia
- Massive pulmonary edema
- Bronchospasm occurs often (unlike with amniotic fluid embolism)
- Hypotension
- Hypovolemia with hemoconcentration (the reactive transudation of fluid into the lungs contributes to this).

Later cardiac failure may develop and accompanying this. There may be:

- Increased pulmonary artery pressure
- Reduced static lung compliance
- Falling arterial oxygen
- Severe metabolic acidosis (usually develops later)
- Infection is not usually a feature
- Chest X-ray shows pulmonary edema and patchy atelectasis (but there is often poor correlation between the extent of pulmonary damage and the radiological appearance).

Differential diagnosis

- Amniotic fluid embolism
- Pulmonary embolus (see Amniotic Fluid Embolism)
- Other causes of shock and circulatory collapse include:
  - Abruptio placentae
  - Heart disease
  - Other pulmonary diseases including asthma, pneumothorax
  - Subarachnoid hemorrhage
  - Malignant hyperthermia—very rare.

Prevention of Mendelson's syndrome: Preventive measure may be applied in labor (particularly in patients at risk of having a cesarean section), before cesarean section and postpartum (for example with anesthesia for retained placenta) and include:

- Avoidance of general anesthesia where possible, for example by use of regional anesthesia, epidurals, etc.
- Oral alkalis in labor to reduce pH of stomach contents. Different drugs and preparations have been used alone or in combination with the aim of raising pH above 2.5 and reducing volume of gastric contents below 25 mL. It is assumed that this will reduce the risk of aspiration. Drugs used include:
  - Sodium citrate is effective at elevating gastric pH but not at reducing gastric volume.
  - Ranitidine, orally or intravenously. Used intravenously at induction or as a pre-medication orally is effective. It is also effectively used in combination with sodium citrate.
  - Cimetidine orally, intramuscularly or intravenously is effective.
  - Metoclopramide used intravenously with oral sodium citrate reduced volume of gastric contents and pH.
  - Omeprazole.
- Good anesthetic technique including:
  - Presence of experienced obstetric anesthetist at every cesarean section or anesthetic for retained placenta.
  - Use of Sellick's maneuver (cricoid pressure) to prevent regurgitation.
  - Preoxygenation of the patient before intubation.
  - Identifying patients likely to be difficult to intubate. Patients can be identified according to certain characteristics (short neck, etc.) but also if intubation has been difficult with previous anesthetics.
- Compliance with and training in a 'failed intubation procedure'.

What do you understand by Sellick's maneuver (cricoid pressure)?

Cricoids pressure, sometimes called Sellick's maneuver (or even 'The Sellick's), is the application of backward pressure on the cricoids cartilage to occlude the esophagus.

Cricoids pressure should be performed during induction of anesthesia for both emergency surgery (full stomach) and for elective surgery when lower esophageal sphincter is likely to be incompetent (e.g. last half of pregnancy or gastro-esophageal reflux disease), and in patients with delayed gastric emptying (e.g. diabetic autonomic neuropathy).

How do you perform Sellick's maneuver?

Locate the most prominent protuberance on the front of the neck in the midline (the thyroid prominence). Find this point then run your finger towards the patients feet (staying in the midline) until you feel your finger drop into the cricothyroid notch or membrane. The next horizontal bar is the cricoid cartilage. Place the thumb and index finger on either side of the cricoid cartilage and press directly backwards at a force of 20–30 newtons against the cervical vertebrae, which is about 2–3 kilograms (10 N = ~ 1 kg). Maintain pressure until directed to release. The application of cricoid pressure requires a dedicated rescuer as it must be maintained until the airway is secured by endotracheal intubation. This may be difficult where there are limited rescuers or when prolonged application is required. Whilst too little force is ineffective, overzealous efforts can restrict ventilation and may worsen laryngoscopic view.

Cricoid pressure should not be confused with optimal external laryngeal manipulation (OELM) or backward upward right pressure (BURP) on the thyroid cartilage, which is used to improve visualization of the vocal cords when intubating. BURP is performed by an assistant and moves the larynx to the right whilst the tongue is displaced to left by the laryngoscope blade. These are techniques employed to improve visualization of the cords, and do not protect the lungs from regurgitation.

Cricoid pressure trainer: It encompasses realistic patient anatomy with real time feedback on the technique.

Cricoid pressure and the laryngeal mask airway: Cricoid pressure has been shown to prevent gastric insufflations when used with laryngeal mask airway (LMA).

Although the LMA can be successfully inserted whilst cricoid pressure is being applied, some have stated it makes insertion difficult, and others found ventilation may be impeded. If insertion of LMA fails, cricoid pressure may be momentarily released and re-applied after successful insertion.

Cricoid Pressure and Pediatrics

The application of cricoid pressure in the pediatric population has been shown to prevent insufflations of gas into stomach during face mask ventilation.

How would you manage if aspiration occurs?

- Tilt the operating table to a 30 degree head low to prevent further aspiration and allow gastric contents to drain to outside
- Maintain cricoid pressure
- Suck the mouth and pharynx as rapidly as possible.
- Intubate the patient with immediate inflation of endotracheal cuff to prevent aspiration
- Suction through endotracheal tube before administering 100% oxygen
- Insert orogastric tube to empty the stomach, measure pH of gastric contents
- Tracheobronchial aspirate collected for culture and sensitivity
- Electively ventilate
- Treat bronchospasm
- Serial ABG, watch for hypoxia
- Early application of PEEP.

Is there any role of prophylactic antibiotic and steroids in aspiration?

There is no role of prophylactic antibiotic; excluding feculent aspirate, which can occur in obstructed cases—antimicrobial therapy for anaerobic and gram-negative organisms may be warranted. Super infection can occur. Cultures should be collected and treat as clinically indicated.

The role of steroid is controversial. Pneumonia caused by gram-negative bacteria was more frequent after aspiration in patients treated with corticosteroids than in those who were not. Two multicenter, randomized, controlled trials failed to demonstrate benefit of high dose corticosteroid in patients with ARDS, the administration of steroids cannot be recommended.

In acid aspiration pneumonitis, pulmonary lesions were aggravated by irrigation of bronchial tree as the large volume of fluid helped to push the acid deeper and mixing of acid and treatment solution.

Bronchial irrigation is indicated only in obstructive type of aspiration. 5–10 mL of normal saline is instilled into the tracheobronchial tree, followed immediately by suction. This is to be repeated till aspirate fluid is clear.

What anesthetic technique would you use for emergency LSCS?

The choice depends on:

- Indications for the surgery
- The degree of urgency
- Maternal status
- Desire of the patient.

Spinal Anesthesia

Advantages:

- Rapid onset
- Provides dense block
- Small doses of local anesthetic is used, so there is minimal transfer of drug to fetus
- Failures are very infrequent with block
- Decreased risk of failed intubation and aspiration of gastric contents
- Avoidance of depressant agents
- Ability of mother to remain awake and enjoy the birthing experience
- It has been suggested that blood loss is reduced under regional anesthesia.

Disadvantages:

- Higher incidence of hypotension.
- Finite duration of anesthesia.
- Despite achieving an adequate (T4) block, some women under spinal anesthesia will experience some degree of visceral discomfort during section (particularly in situation in which the obstetrician exteriorizes the uterus). The quality can be improved by adding opioids.
- PDPH with larger needles.
- Total spinal block—a rare and serious complication that occurs after excessive cephalad spread of the local anesthetic.
- *Hematoma:* The incidence of neurologic injury resulting from hematoma associated with spinal is very low with estimates of 1 in 220,000.

Epidural Anesthesia

Advantages:

- It provides flexibility, when catheter is placed for labor analgesia and can be used for section
- The volume of local anesthetic drug can be titrated
- Incidence of hypotension is less
- All advantages of regional block as mentioned above.

Disadvantages:

- Takes a little longer time for insertion and institution of analgesia

- Large doses of local anesthetic are used to achieve adequate levels
- Failures including incomplete or patchy block are more frequent than spinal.

General Anesthesia

It is necessary when

- Life-threatening fetal compromise is present
- Cases with overt coagulopathy
- Maternal hemorrhage.

Advantage

- Speed of induction
- Control of airway
- Superior hemodynamics.

How would you treat spinal hypotension?

- Co-loading with crystalloids.
- Left uterine displacement with the help of a wedge beneath the right buttock and thus reducing aortocaval compression by the gravid uterus.
- *Use of vasopressor:* Ephedrine had been considered the safest vasopressors because it increases mean arterial pressure and uterine artery blood flow without a concomitant decrease in uteroplacental perfusion. The overall increase in cardiac output from the adrenergic stimulation of ephedrine will maintain uterine artery perfusion and compensate for its mild adrenergic vasoconstriction. Currently a number of studies show that phenylephrine did not adversely affect neonatal outcome and may even decrease the incidence of fetal acidosis.

What is rapid sequence spinal anesthesia? Discuss.

It is a new approach of spinal anesthesia for emergency LSCS. It aims in reducing the induction time for spinal anesthesia, which has been the deciding factor in choosing between spinal and general anesthesia in emergency cases. The aim of rapid sequence is to establish spinal anesthesia with the bare essentials and to abandon the same in case of failure of the block, thus also limiting the number of attempts. For rapid spinal anesthesia it is suggested to use sterile gloves but no gown. The position for spinal anesthesia would be based on obstetric factors and anesthetist preference. Lateral position is preferred in most places as it is best for the uterine blood flow in cases of fetal compromise. In a series of rapid sequence, spinal anesthesia reported from Bristol, UK, the total time to induce spinal anesthesia was 8 minutes. Kinsella et al advices starting of surgery before a standard block is achieved. The risk of pain or conversion to general anesthetic, in case of failure, is much lower than the risk associated by giving all these patients straight away a general anesthetic. If needed, surgery can be paused after delivery, to allow anesthesia to intensify. This technique, however, has raised concerns with respect to sterility and feasibility of a team work in out- of- hour's periods.

If you had unexpectedly been unable to intubate this patient, how would you manage the airway?

Obstetrics is one of the few areas of medicine where there is a "true" emergency. There are two lives at risk, that of the mother and the baby.

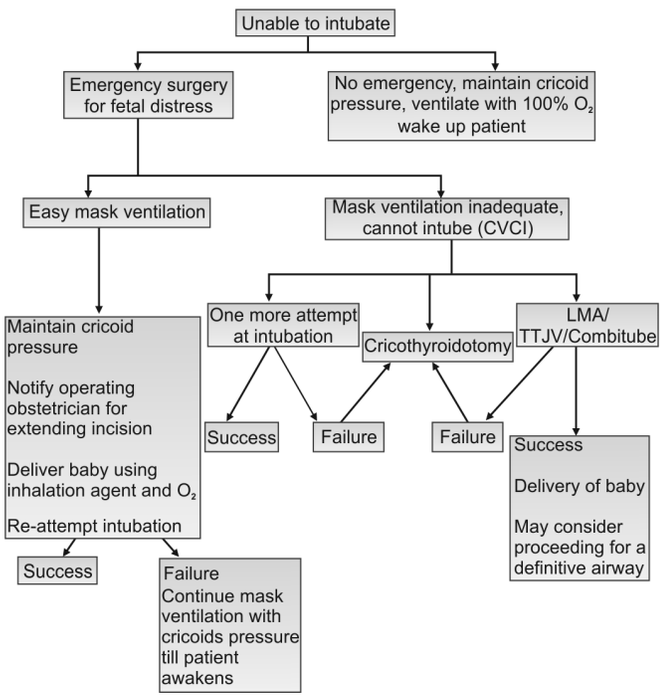
In the same manner as for the ASA difficult airway algorithm, initial assessments are required and subsequent decisions are made based on these assessments. The initial assessments include evaluation of the maternal airway and fetal status. The clinician needs to decide whether the airway is difficult, as an expected difficult airway is easier to manage than an unexpected difficult airway.

Although "conventional wisdom" endorses a regional technique in the expected difficult airway, complications or failure of the regional technique may make it necessary to intubate the trachea. Thus, a backup plan is necessary, with the availability of appropriate equipment.

The difficult and failed obstetric airway is a problem for all involved in the care of the pregnant patient in the labor and delivery room. All anesthesia providers must be trained in the assessment and care of the obstetric airway.

It is necessary to have a difficult airway chart, which should include: proseal LMA, Intubating LMA, combitube, fibreoptic flexible bronchoscope, cricothyrotomy set with jet ventilation.

Flow chart 14.1 Management of the non-intubatable patient



What is the effect of Inhalation agent on uterine contractility?

If less than one MAC of a potent volatile inhalation agent, such as isoflurane or sevoflurane is used, there is no increase in blood loss or decrease in uterine contractility. At these low concentrations, the uterus still responds to oxytocin. At higher concentration uterine contractility is decreased and blood loss in increased.

What is Apgar scoring system? What is its significance?

Apgar score is a useful aid to evaluate the need for infant resuscitation applied at 1 and 5 minutes after birth. The score was devised in 1952 by Dr Virginia Apgar. An Apgar score of 10 is in practice rarely assigned. Most infants are in excellent condition at birth and have Apgar score of 7 to 10.

A low 1 minute Apgar score does not correlate with the infant's future outcome. Change in score between 1 and 5 minutes is a useful index of effectiveness of resuscitation efforts. Apgar scores at 1 and 5 minutes correlate poorly with either cause or outcome. Correlation of the Apgar score with future neurological outcome increases when the score remains 0 to 3 at 10, 15 and 20 minutes.

Table 14.1 Apgar scoring system

Mnemonic	Sign	Score		
		0	1	2
A	Appearance (color)	Blue, Pale	Body pink, Extremities blue	Pink
P	Pulse	Absent	< 100/min	>100/min
G	Grimace (reflex irritability; response to catheter in nose)	Absent	Grimace	Cough, sneeze
A	Activity (muscle tone)	Limp	Some extremity flexion	Active motion
R	Respiratory effort (Breathing)	Absent	Weak or Irregular	Strong efforts with a good cry

Discuss the immediate resuscitation of the newborn it neonatologist arrives?

Those newly born infants who do not require resuscitation can generally be identified by a rapid assessment of the following four characteristics:

- Was the baby born after a full-term gestation?
- Is the amniotic fluid clear of meconium and evidence of infection?
- Is the baby breathing or crying?
- Does the baby have good muscle tone?

If the answer to all four of these questions is "yes," the baby does not need resuscitation and should not be separated from the mother. The baby can be dried, placed directly on the mother's chest, and covered with dry linen to maintain temperature. Observation of breathing, activity, and color should be ongoing. If the answer to any of these assessment questions is "no," there is general agreement that the infant should receive one or more of the following four categories of action in sequence:

A. Initial steps stabilization (provide warmth, position, clear airway, dry, stimulate, reposition)
B. Ventilation
C. Chest compressions
D. Administration of epinephrine and/or volume expansion. For additional details refer to CPCR guidelines.

How would you manage postoperative pain?

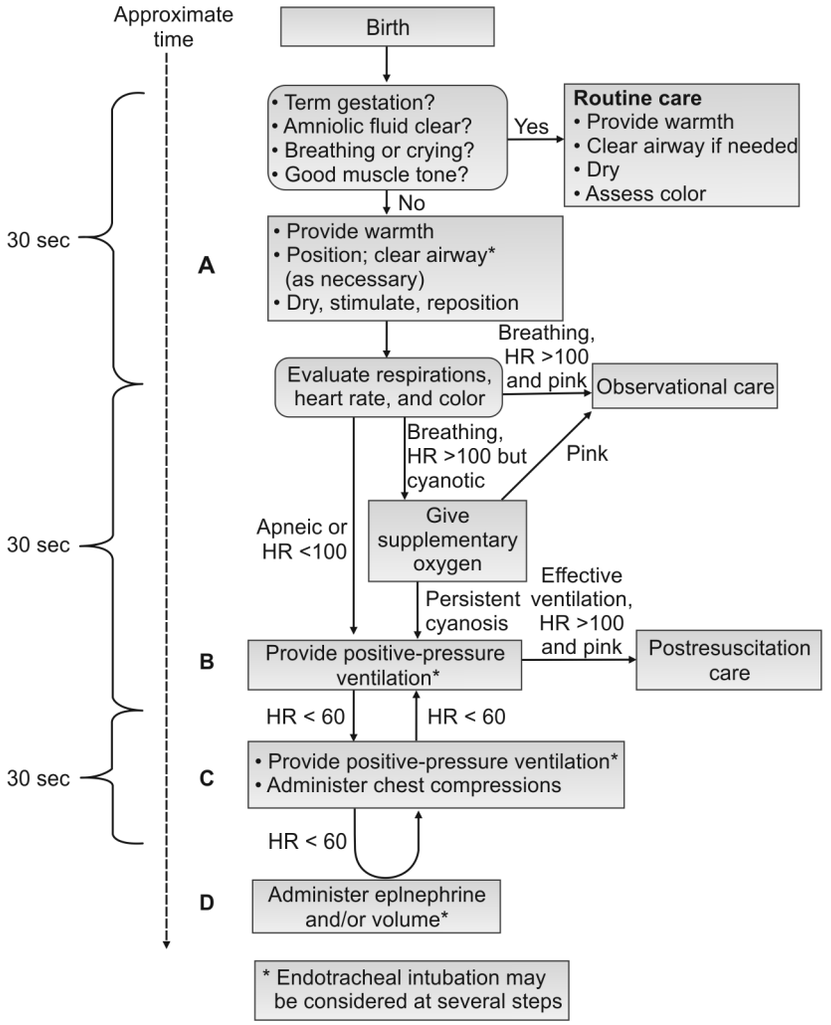
As per guidelines laid by breastfeeding network.

- Paracetamol is safe and can be given in dose of 1 g four times a day.
- Transfer of NSAIDs is small and can be used for pain relief.
- Aspirin should preferably be avoided as a painkiller, as the small amounts that are secreted in the milk can cause increased risk of Reyes syndrome in pediatric viral infections. There are reports of neonatal apnea following codeine given to the mother. Hence, the neonate needs to be watched, if the mother is on opioids for pain relief.

Is there any protocol for neonatal resuscitation?

Yes, the protocol for the neonatal resuscitation is shown in Flow chart 14.2

Flow chart 14.2 Protocol for the neonatal resuscitation



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15 Non-obstetric Surgery in a Pregnant Patient

S Bakshi, R Ambulkar, M Joshi

A 29-year-old female with previous bad obstetric history is admitted for threatened abortion. Cervical cerclage is planned. Discuss the anesthesia plan.

What is the incidence of surgery during pregnancy?

About 2% of pregnant women undergo non-obstetric surgery annually.

The operations include:

- Those directly related to pregnancy, such as cervical cerclage.
- Those indirectly related to pregnancy, such as ovarian cystectomy.
- Unrelated to gestation, such as appendectomy.

What is the etiology of early pregnancy loss? What is cervical cerclage?

The etiology of early pregnancy loss is varied and often controversial. More than one etiologic factor is often present. The most common causes of recurrent miscarriages are as follows:

- Genetic causes
  - Mendelian disorders
  - Multifactorial disorders
  - Chromosomal inversions
- Autoimmune causes
- Anatomical variations
  - Uterine Müllerian anomaly
  - Incompetent cervix
  - Leiomyomas
  - Uterine polyps
- Infectious causes
- Environmental causes
  - Smoking
  - Excessive alcohol consumption
- Endocrine factors
  - Diabetes mellitus
  - Antithyroid antibodies
  - Luteal-phase deficiency
- Hematologic disorders.

The gestational age at the time of the spontaneous abortions can provide clues about the cause. For instance, nearly 70% of spontaneous abortions in the first 12 weeks are due to chromosomal anomalies. However, losses due to cervical incompetence tend to occur after the first trimester.

The cervix normally stays tightly closed during pregnancy. Occasionally, it starts to open early, leading to miscarriage. For some women, this recurs in subsequent pregnancies. This may be due to cervical weakness (incompetence) if the miscarriage occurs in the second or early third trimester. One option is cervical cerclage—surgery to insert a suture (stitch) to keep the cervix closed.

What are the main concerns of surgery in the pregnant patient?

- Maternal concerns
- Effect on developing fetus
- Surgical problem itself.

Maternal safety: During pregnancy maternal anatomic and physiologic changes occur, which have implication on the anesthetic management. Pregnant woman are more prone to hypoxia and hypercapnia. Airway management can be technically difficult. Physiologic compensation for aortocaval compression can be compromised by anesthetic techniques that interfere with sympathetic tone and can result in

profound hypotension. Pregnant women are at increased risk for gastric acid aspiration during anesthetic induction or unconscious sedation.

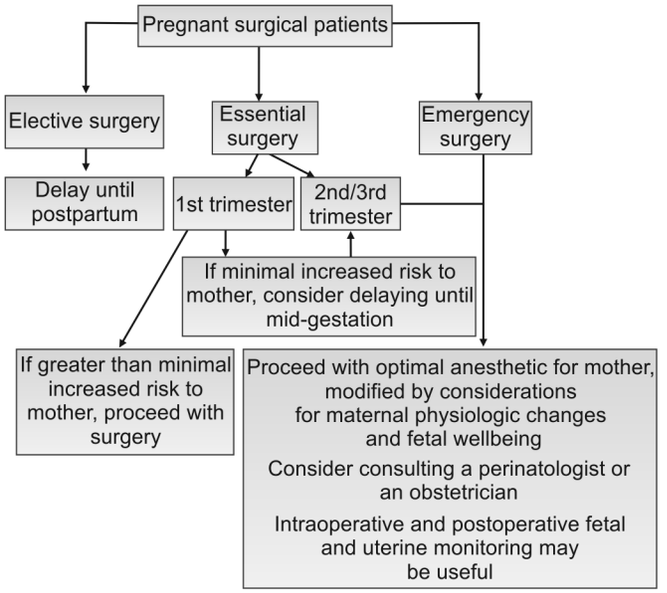
Effect on developing fetus: Fetal loss, fetal asphyxia, teratogenic effects of the anesthetic agents, premature rupture of membranes and premature labor.

Surgical problem itself, especially in non-obstetric surgery.

What will you discuss with the patient preoperatively?

The maternal and fetal risk associated with surgery needs to be explained and included in the written consent (Flow chart 15.1).

Flow chart 15.1 Maternal and fetal risk associated with surgery



What are the concerns regarding fetal malformations?

The most susceptible time for the developing fetus is between 15th and 30th day after conception; susceptibility declines thereafter to the 50th day. Virtually every drug and every inhalation anesthetic is teratogenic to some species under certain conditions. None has as yet been identified as a definite human teratogen. However, we cannot assume that some potential for teratogenicity does not exist. It is therefore most prudent to postpone elective surgical procedures until after pregnancy. If this is not possible then the first trimester should be avoided.

As per US FDA fetal risk categories for therapeutic agents are:

Category A: Controlled studies have shown no risk to the fetus during the first trimester and later trimesters as well.

Category B: Animal studies have demonstrated no fetal risk, but no controlled studies have been performed in humans.

Category C: Studies have shown fetal risk in animals (teratogenic or embryocidal) but no controlled human studies.

Category D: Confirmed evidence exists for human fetal risk, but benefits are acceptable despite known risk.

Category X: Agents of this class are contraindicated in pregnant patients for any reason because animal or human studies have displayed teratogenicity or there is evidence of fetal risk from prior human experience.

Most of the anesthetic drugs belong to either category C or B. Following drugs belong to category B: Methohexital, propofol, enflurane, isoflurane, desflurane, sevoflurane, lidocaine, ropivacaine, fentanyl.

Is nitrous oxide a known teratogen?

Nitrous oxide oxidizes cobalamin and inhibits methionine synthase activity. This is a key link in the synthesis of S-adenosyl methionine and in the tetrahydrofolic acid cycle. DNA production, myelin deposition, and folate and methylation process-dependent reactions might be affected. Though teratogenicity has not been demonstrated in humans, nitrous oxide is best avoided during the first trimester and if used at other stages of pregnancy, pre-treatment of the patient with folic or folinic acid could offer some additional benefit.

How will you premedicate this patient?

Aspiration prophylaxis should be administered to all patients over 14 weeks gestation because physiologic changes at lower esophageal sphincter enhance the risk of aspiration. An H_2 antagonist should be given the night prior and 1 hour before surgery if possible and a non-particulate antacid, such as sodium citrate just before induction of anesthesia. Prokinetic agent, such as metoclopramide 10 mg intravenously enhances gastric emptying. The long-standing relative contraindication and concern about benzodiazepine use, particularly in the first trimester has been dispelled. Appropriate use of benzodiazepine to treat anxiety is essential as catecholamine increased by pain or anxiety may adversely affect uterine blood flow.

How will you decide on the anesthetic technique?

Anesthetic technique depends upon the clinical status, surgical procedure and patient preferences. Whenever possible, regional anesthesia should be preferred.

Spinal anesthesia: The advantages are that spinal anesthesia offers the least drug transfer, and airway manipulations are not involved. The disadvantages are hypotension, aortocaval compression and possibility of maternal hypoxia and acidosis (in case of high spinal).

Epidural anesthesia/Regional block yields higher local anesthetic blood levels and hence more placental transfer, however, no teratogenic effect have been seen in humans.

General anesthesia: Though there is better control of airway and hemodynamics there is maximum fetal exposure to anesthetic drugs.

Describe the technique for general anesthesia.

- Secure intravenous line.
- Preoxygenate for at least 3 minutes (or 8 vital capacity breaths in one minute with oxygen at 15 L/min) to ensure denitrogenation and to avoid maternal and fetal hypoxemia during induction and intubation.
- Carry out induction using a rapid sequence technique using thiopentone and succinylcholine.
- Avoid supine position; displace the uterus laterally, a wedge beneath the right buttock may suffice.
- Maintenance of anesthesia using safe drugs like opioids, non-depolarizing muscle relaxant, inhalational agents. Nitrous oxide can be avoided and higher concentration of inhalational agents can be used.
- Avoid hyperventilation and hypoventilation.
- Reversal agents should be administered slowly to avoid acute increase in acetylcholine, which might stimulate uterine contractions.
- Extubate when patient is awake and has regained laryngeal reflexes.

How will you monitor this patient intraoperatively?

Monitoring should include:

- Electrocardiogram
- Pulse oximetry
- Intermittent blood pressure
- Temperature monitoring
- Capnometer (if general anesthesia planned)
- Urine output.

Fetal monitoring should include external FHR monitoring from 18 week onward but whether monitoring can affect outcome is controversial. It is advisable to document FHR before and after institution of both regional and general anesthesia and on completion of surgery. The decision to perform fetal monitoring should be individualized and may be based on gestational age, type of surgery and facilities available.

What is the role of tocolytic therapy in pregnant patients undergoing non-obstetric surgery? Describe the mechanism of action, side effects in brief.

β-agonists are routinely used for tocolysis. β stimulation leads to relaxation of the uterine muscle. Fenoterol and Ritodrine are the most commonly used β-agonists. Onset of action for Ritodrine is rapid and its half-life is short but after prolonged use tachyphylaxis may be seen. In contrast Fenoterol has longer duration of action and it more selective for β-2 receptors. The side effects match the pharmacologic effects, i.e. tachycardia, arrhythmias, hypotension, pulmonary edema, etc.

Calcium channel blockers reduce the inflow of Ca^{++} in to the cell by inhibiting the slow calcium channels. One study found oral nifedipine to be better than β-agonists in delaying labor. Side effects include tachycardia and mild hypotension. The effect of prostaglandin synthesis inhibitors, such as indomethacin for tocolysis was long known. Recently COX-2 inhibitors have been advocated for tocolysis. Since the degree of tocolysis is not great and the experience is limited they are not very commonly used. Other worrying factors are fetal or neonatal effects, such as premature closure of the ductus arteriosus, vasoconstriction and pulmonary hypertension. Some people advocate use of magnesium sulfate; however it has not been shown to be a very effective tocolytic agent.

Describe postoperative care.

- Provide adequate hydration
- Continue left uterine displacement
- Administer supplemental oxygen. Consult the obstetrician if tocolytics are needed in the postoperative period, and USG may be repeated to ascertain fetal wellbeing
- Opioids preferred for postoperative pain relief
- NSAIDs avoided after first trimester because some of agents may close the fetal ductus arteriosus.

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16 Amniotic Fluid Embolism

R Ambulkar, S Bakshi, A Kothekar

A woman in second stage of labor gets breathless and then collapses. She is cyanosed. There is no evidence of bleeding.

What is your differential diagnosis?

Obstetric Causes

- Acute hemorrhage
- Amniotic fluid embolism
- Placental abruption
- Uterine rupture
- Uterine atony
- Eclampsia
- Peripartum cardiomyopathy

Anesthetic Causes

- High spinal anesthesia
- Aspiration
- Local anesthetic toxicity

Nonobstetric Causes

- Pulmonary embolism
- Air embolism
- Anaphylaxis
- Sepsis/septic shock
- Intracerebral bleed
- Drug toxicity
- Acute myocardial infarction

What do you understand by amniotic fluid embolism?

Amniotic fluid embolism (AFE) is a life-threatening obstetric emergency. It is associated with amniotic fluid, fetal cells, hair, or other debris entering the maternal circulation. It usually presents as sudden, profound, and unexpected maternal collapse associated with hypotension, hypoxemia, and disseminated intravascular coagulation (DIC).

What are the risk factors for amniotic fluid embolism?

There are no clinical factor that can consistently predict AFE. However, it might be associated with following conditions:

- Advanced maternal age
- Multiparity
- Meconium stained liquor
- Obstructed labor
- Intrauterine fetal death
- Polyhydramnios
- Tetanic uterine contractions
- Maternal history of allergy or atopy
- Uterine rupture
- Placenta accreta
- Trauma
- Diabetes mellitus
- Operative delivery including cesarean section.

What is the pathophysiology of amniotic fluid embolism?

The pathophysiology of AFE still seems unclear. It is uncommonly known as Anaphylactoid Syndrome of Pregnancy. Maternal circulation gets exposed to amniotic fluid or debris via ruptured membranes or ruptured cervical or uterine vessels as a result of a pressure gradient from the uterus to veins. This may result in hypoxemia, pulmonary vasospasm, cardiac failure or even death.

This may result in activation of the complement cascade stimulating endogenous immunomediators, producing a reaction similar to anaphylaxis. Amniotic fluid also contains procoagulant tissue factor. In 1995, Clarke described a biphasic response to AFE:

Phase 1: There is a release of biochemical mediators once amniotic fluid and fetal cells gain entry into the maternal circulation that results in pulmonary artery vasospasm leading to pulmonary hypertension. This results in hypoxemia and hypotension due to elevated right ventricular pressures and right ventricular dysfunction. This phase may last up to 30 minutes.

Phase 2: Left ventricular failure and pulmonary edema may occur in patients who survive the initial insult. Biochemical mediators are known to trigger DIC that eventually leads to massive hemorrhage and uterine atony.

What are the clinical features of amniotic fluid embolism?

Table 16.1 Clinical features of amniotic fluid embolism

Symptoms	Signs
Dyspnea	Hypotension
Cough	Fetal distress
Headache	Pulmonary edema/ARDS
Chest pain	Cyanosis
	Coagulopathy
	Seizures
	Bronchospasm
	Cardiopulmonary arrest
	Uterine atony

How would you treat a patient of amniotic fluid embolism?

Amniotic fluid embolism is a diagnosis of exclusion. High index of suspicion is necessary to diagnose this life-threatening condition. It should be suspected in sudden onset of dyspnea, cardiovascular collapse and DIC. Ongoing resuscitation and early delivery of fetus should be the plan. The management is supportive and symptomatic with the goal of maintaining oxygenation, hemodynamic support and correction of coagulopathy.

Team approach: Anesthetists, obstetricians, hematologists and intensivists.

Immediate Management

Resuscitation—ABC approach.

Airway and breathing

- Administer 100% oxygen via a non-rebreathing reservoir face mask
- Prompt assessment, with control of the airway and ventilation of the lungs with tracheal intubation may be essential.

Circulation

- Establish IV access with 2 large bore cannulae. Take blood at the same time for: Cross-match 6 units, full blood count and coagulation studies.
- Left lateral tilt/Manual uterine displacement.
- Hemodynamic support would include preload optimization and vasopressors.
- Commence fluid resuscitation with crystalloid/colloid to optimize filling.
- Infusion of an inotrope may be required to maintain a mean arterial blood pressure and achieve an adequate urine output.
- An arterial line for continuous blood pressure monitoring is essential, and the use of a non-invasive cardiac output monitor may be helpful.
- Continuously monitor the fetus and early consideration should be given to delivery of baby.
- *Uterine tone:* Maintainance of uterine tone is important and is achieved by pharmacological agents, by using oxytocin, ergometrine, and prostaglandins such as carboprost and misoprostol.
- *Coagulation:* The development of consumptive coagulopathy may warrant the use of plasma, cryoprecipitate, and platelets that is guided by clinical condition of the patient and laboratory investigations. Recombinant factor VII may be used, but one should be careful as this can cause thrombotic complications (Thrombosis in major organs can lead to permanent disability, multiorgan failure and death) in pregnant patients. It should be considered only if bleeding is not controlled with aggressive blood component therapy.
- Antifibrinolytics, like e-aminocaproic acid and tranexamic acid, might be helpful but evidence is lacking.

What investigations would be useful?

Investigations

- *Complete blood count:* Hemoglobin may be within normal limit unless there is excessive blood loss due to uterine atony and DIC. Thromobocytopenia can occur
- *Coagulation profile:* AFE is associated with DIC in >80% cases
- Electrocardiogram shows tachycardia, ST segment and T-wave changes, and findings consistent with right ventricle strain
- *Arterial blood gases:* changes consistent with hypoxia
- *Chest X-ray:* consistent with pulmonary edema
- Echocardiogram
- Serum tryptase
- V/Q scan.

What are the main problems you will encounter while treating this patient?

- DIC
- Uterine atony

How would you manage this lady after delivery of the fetus?

- She will need intensive care monitoring
- One should be aware that there is high-risk at developing: ARDS, heart failure, DIC
- Supportive treatment: Ventilation, inotropic support, Hematological support
- Steroids may be useful

Potential Interventions for Severe Life Threatening Cases of AFE

- Inhaled nitric oxide for pulmonary hypertension leading to right-sided heart failure
- ECMO for severe hypoxia and left heart failure.

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17 Obstetric Hemorrhage

R Ambulkar, S Bakshi, A Chatterjee

You are called to the labor ward to see a 25-year-old lady (G_3P_2) in labor, who has lost 700 mL of blood per vaginally. She looks pale and sweaty, her pulse is 140/min, regular but thready and her BP is 100/50 mm Hg.

Would you manage this patient?

- Administer 100% oxygen via a non-rebreathing reservoir face mask.
- Establish IV access with 2 large bore cannulae and take blood at the same time for: cross-match 6 units, complete blood count and coagulation studies.
- Place the patient in a head down and left lateral tilt position.
- Start fluid resuscitation with either crystalloids or colloids.
- Use group-specific or O Rh-negative blood for resuscitation till cross-matched blood is available.
- Set up a fluid warmer, rapid infusion device and (if available) a cell saver.
- Monitor hematocrit, hemoglobin and coagulation at regular intervals.
- Communication is vital: Notify theater staff, alert the blood bank and hematologist.
- Joint assessment with the obstetrician and decision on further plan of action.
- Continuous maternal and fetal monitoring.

If the Massive Bleeding Continues

- Give FFP and cryoprecipitate and platelets according to the coagulation results.
- Invasive arterial blood pressure monitoring.
- Consider rFVIIa (*Novo Seven*).

How would you assess the degree of blood loss?

Blood loss can be notoriously difficult to assess in obstetric bleeds. There could be problems detecting the loss accurately since the early signs of bleeding mimic the normal physiological changes of pregnancy, e.g. dilutional anemia, increased pulse and respiratory rate. Bleeding may sometimes be concealed and the presence of amniotic fluid makes accurate estimation difficult. Physical signs that would aid the diagnosis are pallor, tachycardia, blood pressure (beware: blood pressure of healthy women may not drop until significant amount of blood has been lost), capillary refill, skin color, level of consciousness and oliguria.

What are the likely causes of her bleeding?

- Placenta previa
- Placental abruption
- Uterine rupture
- Cervical or vaginal tear
- Coagulation disorders.

Her previous surgery was a LSCS, is it relevant?

Previous cesarian section increases the possibility of placenta previa and uterine rupture in future pregnancies.

Classify causes of hemorrhage related to pregnancy

Early Pregnancy

- Incomplete abortion
- Septic abortion
- Ruptured ectopic.

Antepartum Hemorrhage

- Placenta previa
- Placental abruption

- Uterine rupture
- Trauma.

Postpartum Hemorrhage

- Primary
  - Uterine atony
  - Retained products of conception
  - Genital tract trauma
  - Abnormally adherent placenta
  - Clotting defects
  - Acute uterine inversion
- Secondary
  - Puerperal sepsis
  - Retained products of conception.

Define massive obstetric hemorrhage.

Massive obstetric hemorrhage is defined as: loss of more than 1500 mL of blood; a drop in hemoglobin > 4 g/dL; or acute transfusion requirement of more than 4 units of blood. Obstetric hemorrhage is divided in to antepartum (APH) and postpartum (PPH). Hemorrhage is termed antepartum when bleeding occurs any time after 24 weeks gestation but before delivery. Postpartum hemorrhage is when bleeding occurs after delivery of baby and can be primary (within 24 hours of delivery) or secondary (24 hours to six weeks after delivery).

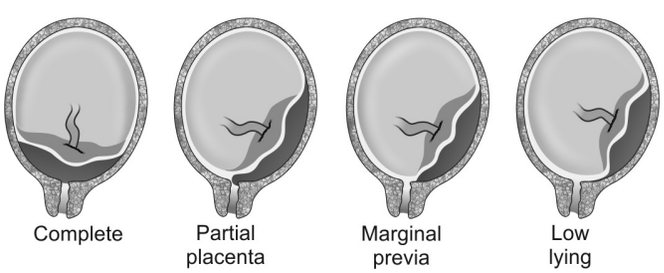
Discuss the causes of obstetric hemorrhage in detail.

Antepartum Hemorrhage

Placenta previa and abruption are major causes of significant hemorrhage in the third trimester.

Placenta praevia: It is the encroachment of the placenta upon the cervical os.

- Grade 1: Low-lying placenta (in lower uterine segment) that does not reach the os
- Grade 2: Placenta reaches the os (marginal)
- Grade 3: Placenta covers os but is positioned to one side (partial)
- Grade 4: Placenta is positioned squarely over the os (complete).



Diagnosed during routine antenatal ultrasound screening and is classically associated with painless vaginal bleeding. Placenta previa can occur in up to 10% full-term pregnancies. If placenta previa is suspected then avoid PV examination as it can result in massive blood loss.

Risk Factors

- Previous cesarean section
- Multiple pregnancy
- Multipara
- Previous myomectomy

Placental abruption: It is the premature separation of a normally implanted placenta with bleeding that can be either concealed or per vagina. Usually presents with painful vaginal bleeding and is associated with uterine tenderness. Much of the bleeding is concealed. It can be diagnosed with USG or MRI. This presents a significant risk to both mother and fetus.

Risk Factors

- Maternal hypertension
- Uterine overdistension
- Previous abruption
- Abdominal trauma
- Smoking
- Post-amniocentesis
- High parity.

Complications

- Anemia
- Coagulopathy including DIC
- Acute renal failure
- Uterine atony
- Fetal distress—fetal death.

Uterine rupture is an uncommon but potentially fatal to both mother and baby.

Risk Factors

- Previous uterine incision
- Use of prostaglandins to stimulate labor following a previous LSCS
- Uterine trauma
- Uterine anomalies
- Tumors
- Fetal malposition.

Rupture may be painless, with the early signs being limited to slow progress in labor and deterioration in the cardiotocogram (CTG). A high-degree of suspicion is required in a patient in labor after previous cesarean section.

Hemorrhage can be torrential, accompanied by hypotension, abdominal pain, a change in uterine contour.

Treatment

A general anesthetic is the choice as there is rapid decompensation of the patient.

The management is directed at:
- Resuscitation of mother
- Emergency delivery of baby
- Repair of uterus or hysterectomy
- Preparation for the management of massive blood loss
- Invasive monitoring
- Intraoperative serial investigation-CBC, Coagulation profile, electrolytes
- HDU/ICU care postoperative.

Postpartum hemorrhage (PPH): Defined as greater than 500 mL of blood lost in the first 24 hours after delivery. Incidence: 5–10% of deliveries.

Uterine atony accounts for 80% of primary PPH.

Risk Factors
- Prolonged, augmentated or precipitant labor
- Uterine overdistension
- Placenta previa
- Increasing parity
- Advanced maternal age.

Treatment
- Physical compression of uterus by bimanual compression or uterine massage.
- Drugs:
  - IV infusion of oxytocin
  - IM ergometrine
  - Prostaglandins.

Trauma: Trauma to the perineum, cervix and vagina are common after normal vaginal delivery, the risk further increased by instrumentation (forceps delivery or vacuum extraction). In a well-contracted uterus with ongoing bleeding, genital tract trauma must definitely be ruled out.

Retained placenta is another common cause of both primary and secondary PPH.

Uterine inversion is a rare but serious condition. Early reduction of the uterus is vital and this may require uterine relaxation; nitrates—50 mcg bolus, β2-agonists, and magnesium have all been successfully used.

Risk Factors
- Uterine atony
- Uterine anomalies
- Inappropriate fundal pressure
- Excessive umbilical cord traction.

Coagulopathy: Both congenital and acquired, contributes to massive obstetric hemorrhage.

What would be your management plan for anticipated massive obstetric hemorrhage such as a lady posted for cesarean section with a low-lying anterior placenta and previous uterine scar?

Pregnant women are often young, healthy and have an increased blood volume of up to 20% at term and therefore are likely to compensate well in case of hemorrhage until the circulating blood volume is very low.

Goals of Management
- Rapid access to circulation and infusion of fluid in the first instance with rapid availability and administration of blood
- Avoid complications of massive blood transfusion, i.e. acid/base disturbance, transfusion related acute lung injury (TRALI), hypocalcemia, hyperkalemia, hypothermia and thrombocytopenia
- Efficient team work involving senior members including anesthetist and obstetrician.

Describe management plan in case of massive obstetric hemorrhage.

- 2 large bore IV cannulae
- Rapid infusion device or pressure bags in theatre
- Blood warmer and warming blanket
- Blood cross-matched and available
- Institute preoperative invasive monitoring
- Consider cell salvage if available
- Consider interventional radiological procedures if available

Communication and teamwork are essential in cases of both anticipated and unanticipated maternal hemorrhage:
- Alert blood transfusion service and the hematologist
- Allocate roles to team members
- Ensure departmental guidelines exist for the management of massive obstetric hemorrhage.

Enumerate the causes of DIC related to obstetrics.

- Intrauterine death
- Amniotic fluid embolus
- Sepsis

- Pre-eclampsia
- Placental abruption
- Retained products of conception
- Induced abortion
- Excessive bleeding
- Acute fatty liver.

What are the surgical and radiological treatment options for massive obstetric hemorrhage?

Surgical Options

- B-Lynch suture (brace suture) is a continuous suture used to encircle and mechanically compress the uterus, with the aim of avoiding a hysterectomy.
- Uterine tamponade by using a Rusch urological balloon or Sengstaken-Blakemore tube.
- Compression/clamping aorta as a temporary measure to minimize blood loss while preparing for a permanent option.
- Ligation of uterine and internal iliac arteries.
- Hysterectomy.
- Uterine replacement in case of a uterine inversion.

Radiological Options

- Bilateral iliac artery balloons may be placed electively and inflated at cesarean section or should bleeding occur.
- Selective uterine artery embolization.

Is intraoperative cell salvaging an option in this scenario?

There is increasing evidence that cell salvage in obstetrics is safe unlike as previously thought of since the likely risks of amniotic fluid embolism, contamination with fetal debris and isoimmunization of a rhesus negative mother by fetal red cells have not been proven nor is there any evidence to suggest the same. However, a separate suction should be used for the amniotic fluid to reduce contamination risk and an appropriate leucocyte depletion filter used to administer the salvaged blood.

What are placenta accreta, increta and percreta?

Placenta accreta is an abnormal attachment of the placenta to the uterine wall such that the chorionic villi invade abnormally into the myometrium. Three grades have been identified, based on histopathologic assessment of myometrial invasion by the chorionic villi:

Placenta accreta (75–78%) is when the placenta is attached directly to the muscle of the uterus wall.

Placenta increta (17%) the placenta extends into the uterine muscle and is termed placenta increta.

Placenta percreta (5–7%) the placenta extends through the entire wall of the uterus and is termed placenta percreta.

Risk Factors

- Prior cesarean section
- Uterine instrumentation or surgery
- Placenta previa.

Diagnosis

Transvaginal ultrasound.

Complications

- Prematurity
- Hemorrhage
- Transfusion related
- Increased incidence of hysterectomy.

Management of Placenta Accreta

- Conservative management: Curettage and/or oversewing of the placental bed and occluding the blood vessels that supply the pelvis.
- *Majority of cases:* Hysterectomy remains the procedure of choice.

A 24-year-old lady has a retained placenta. What precautions would you take while anesthetizing this lady?

- If the placenta has not been delivered within 30–60 minutes following the birth it is considered a retained placenta
- The retroplacental myometrium contracts following delivery of the baby that leads the placenta to shear away from its bed and be expelled. Retained placenta complicates 2% of deliveries worldwide.

Risk Factors

- Previous retained placenta
- Previous injury to uterus
- Preterm delivery
- Induced labor
- Multipara.

Potential Complications

- Primary postpartum hemorrhage
- Secondary (delayed) postpartum hemorrhage—due to retained placental tissue
- Uterine inversion
- Puerperal sepsis

Active Versus Expectant Management of the Third Stage of Labor

Active management involves the administration of oxytocin after delivery followed by early clamping and cutting of the umbilical cord. Controlled cord traction is applied with simultaneous suprapubic pressure to prevent inversion of the uterus.

Expectant (also known as conservative or physiological) *management* involves waiting for signs of spontaneous separation and delivery of the placenta.

Signs of Placental Separation

- Uterus rises in maternal abdomen
- Uterine shape changes from discoid to globular
- Umbilical cord lengthens
- Vaginal blood loss.

Management

Observe the patient for signs of blood loss e.g. pallor, tachycardia and hypotension. Blood loss may be concealed and difficult to estimate. Choice of anesthesia will depend on severity of bleeding and hemodynamic stability of the patient.

- 2 large bore IV cannulae
- Fluid resuscitation with crystalloid/colloid
- Blood cross-matched and available
- Aspiration prophylaxis.

Anesthetic Technique

Comparison of general anesthesia, regional anesthesia and sedation

Technique	Advantages	Disadvantages
GA	Dose-dependent uterine relaxation by volatile agents	Risks of general anesthesia e.g. airway compromise, failed intubation, aspiration, anaphylaxis
Spinal	Rapid establishment of profound analgesia. Avoids risks of GA	Potential for sudden hypotension if extent of hemorrhage is underestimated
Epidural	Good if already *in situ*	Takes time to establish one in case of emergency as well as to achieve surgical anesthesia
Sedation	Quick and easy	Poor uterine relaxation Unprotected airway: risk of aspiration if overdose

General Anesthesia

Adequate pre-oxygenation followed by rapid sequence induction with an endotracheal intubation is the gold standard. The choice of induction agent would vary according to the hemodynamic stability of the patient. In this scenario, IV induction agents like etomidate and ketamine should be considered. Volatile agents depress uterine contractility in a dose-dependent manner.

Regional Anesthesia

Spinal anesthesia eliminates the risks associated with general anesthesia. Approximately 1.5–2.0 mL of hyperbaric 0.5% bupivacaine would be adequate to attain the desirable level of surgical anesthesia and maternal intraoperative comfort. A low-dose spinal anesthetic regimen comprizing, 1.2 mL of 0.5% bupivacaine and 25 micrograms fentanyl can also be considered as another option.

Hypotension secondary to regional anesthesia is likely to be related to maternal blood loss rather than block height.

Note: IV NTG 50–100 mcg has been used effectively to relax the uterus under close hemodynamic monitoring.

What drugs are useful (main or as adjuncts) in the management of obstetric haemorrhage?

Oxytocin: A synthetically produced hormone causes uterine contraction. The dose is 5–10 IU bolus followed by an infusion at 5–10 IU/hr. Used in:

- Induction and acceleration of labor
- Missed and incomplete abortion
- Postpartum hemorrhage (atonic uterus).

Side-effects

- Hypotension and reflex tachycardia due to peripheral vasodilatation
- Antidiuretic properties in high doses
- May prolong the Q-T interval and cause T-wave flattening.

Ergometrine: An ergot alkaloid derivative that stimulates contractions of uterine and vascular smooth muscle. Like other ergot alkaloids, ergometrine produces arterial vasoconstriction by stimulation of alpha-adrenergic and serotonin receptors and inhibition of endothelial-derived relaxation factor releaze. Dose: 0.5 mgIM or 0.1 mg slow IV injection over 10 minutes. Action: Produces effective uterine constriction.

Side-effects

- Severe nausea and vomiting
- Systemic vasoconstriction can produce hypertension
- Diarrhea
- Dizziness
- Hallucinations
- Vertigo and tinnitus.

Contraindications: Ergometrine is contraindicated in patients with:

- Eclampsia, preeclampsia or a history of hypertension
- Peripheral vascular disease or heart disease
- If there is any suspicion of retained placenta.

Carboprost: Carboprost (hemabate) is a prostaglandin (PGF2a) and potentiates the uterotonic effect of oxytocin. It is used to treat postpartum hemorrhage due to uterine atony in patients unresponsive to ergometrine and oxytocin
Dose: 0.25 mg into the myometrium or IM every 10–15 minutes till a maximum of 2 mg. Action: Uterine constriction

Caution:

- It can lead to severe nausea and vomiting
- May cause bronchospasm (avoid in asthmatics)
- Excessive dosage may cause uterine rupture.

Contraindications:

- History of glaucoma or raised intraocular pressure
- Uterine scars.

Misoprostol: Misoprostol is a synthetic prostaglandin E1 (PGE1) analog. It is used to treat postpartum hemorrhage due to uterine atony in patients unresponsive to ergometrine and oxytocin.

Dose: 400 – 800 mcg (2–4 × 200 mcg tablets). The sublingual, oral, rectal and intrauterine routes have all been used successfully.

Side-effects: Nausea, vomiting, diarrhea, abdominal pain, dyspepsia, flatulence, rashes and dizziness.

Suggested Reading

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18 Hydrocephalus

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A 3-year-old male (14 kgs) child presents with history of:

• Frequent headaches and blurring of vision	since 2 months
• Altered mental status	since 3 days
• Vomiting 8–10 episodes	since 2 days
• Fever 102°F	since 1 day

CT scan reveals an obstruction within the ventricular system and he is planned for ventricular peritoneal shunt procedure.

What is Hydrocephalus? What is the incidence of this condition?

Hydrocephalus, one of the most common pediatric and adult neurosurgical disorders, is an abnormal increase in the amount of cerebrospinal fluid (CSF) resulting from a disturbance of formation, flow or absorption of CSF thus resulting in enlarged cerebral ventricles. This may be congenital or acquired. Patients with hydrocephalus usually present with an increase of intracranial pressure (ICP).

Hydrocephalus can occur at any age depending on the etiology. Incidence of congenital hydrocephalus is 3 per 1,000 live births, whereas the incidence of acquired hydrocephalus is not known exactly.

Where is CSF produced and how does it flow? What is the normal CSF pressure and what are the common causes of CSF blockade?

CSF is produced by the choroid plexus of the ventricles and circulates in one direction from the lateral ventricles to the third ventricle and through the aqueduct of Sylvius to the fourth ventricle. From the fourth ventricle, CSF exits the brain through 3 separate openings, one in the midline (foramen Magendie) and one on either side (foramina Luschka). It enters the subarachnoid space at the foramen magnum, circulates down to the spine, and then circulates up again to the surface of the brain where it is absorbed at the arachnoid granulations. These are sieve-like structures where the CSF enters the venous circulation, leading to the sagittal sinus.

In the adult human CSF is formed at a rate of about 0.5 ml/min. The total volume is about 200 mL, of which 30 mL is in the ventricles and the remainder in the subarachnoid space. The circulation of CSF leads to the fluid being completely replaced about every 4 hours.

Normal CSF pressure when one is lying down is about 130 mm of H_2O (10 mm Hg) with a range of 70–180 mm H_2O. In vertical position the CSF pressure is higher (375–550 mm H_2O).

Common Causes of CSF Fluid Blockade

a. *Infectious:* Meningitis, encephalitis, abscess
b. *Neoplastic:* Astrocytoma, meningioma, choroids plexus papilloma, meduloblastoma
c. *Vascular:* Arteriovenous malformations, aneurysm
d. *Congenital:* Chiari malformation, Arachnoid cysts, colloid cysts, encephalocoele.

Give the etiological classification of Hydrocephalus? What is the pathophysiology of this condition?

1. Overproduction of CSF—choroid plexus papilloma.
2. Blockage of the normal flow of CSF
   a. Communicating or Absorptive—blockage of the resorption of CSF in the arachnoid villi, basal cisterns,

or subarachnoid space. The ventricles are patent and all 4 are enlarged.

- Inflammatory – Infectious ventriculitis, chemical ventriculitis, intraventricular hemorrhage. In these cases, the arachnoid granulations are occluded by protein or blood degradation products rendering CSF absorption relatively ineffective
- Impaired CSF absorption in spina bifida

b. *Non-communicating:* Obstruction proximal to the foramina of Lushka and Magendie at the outlet of the 4th ventricle.

i. Cysts
ii. Tumors – Tumors of the posterior cranial fossa
iii. Infection and hemorrhage
iv. Congenital malformations – Aqueduct stenosis, Arnold-Chiari malformation, Dandy Walker cyst
v. Aqueductal stenosis.

The classical distinction between obstructive and communicating hydrocephalus is less useful clinically as the cause of reduced absorption of CSF in communicating hydrocephalus is usually functional obstruction at the arachnoid villi (e.g. by blood or protein).

Due to the above factors CSF accumulation leads to ventricular enlargement and rise in intraventricular (and hence intracranial) pressure. In infants with open fontanelles, some of this rise in pressure is counteracted with enlargement of the head. When the maximum capacity for head enlargement has been used, rapid deterioration follows because of raised intracranial pressure.

In neonates hydrocephalus may exist despite normal ICP an increase in CSF volume is compensated by a decrease in brain mass until the compliance limit of immature brain tissue is reached.

Older children with closed fontanelles develop clinical signs of intracranial hypertension without progressive head enlargement.

Enumerate some of the common syndromes and anomalies associated with this condition?

- *Meningomyelocele:* Myelomeningocele is protrusion of the meninges through a midline bony defect of the spine, forming a sac containing cerebrospinal fluid and neural tissue.
- *Aqueduct stenosis:* In newborn children common cause of hydrocephalus is obstruction of the aqueduct of Sylvius. Obstruction here prevents the free passage of cerebrospinal fluid from the lateral and third ventricles to the fourth ventricle, and from thereon to the subarachnoid space.
- *Arnold-Chiari Syndrome:* Malformation of the brain. It consists of a downward displacement of the cerebellar tonsils and the medulla through the foramen magnum. Symptoms include cerebellar symptoms, headache, visual disturbances, nausea, extreme muscle soreness, facial pain, hearing problems, insomnia, pain in the neck/upper arm,tingling ,burning. Other conditions sometimes associated with Chiari Malformation include hydrocephalus, spina bifida, and connective tissue disorders such as Ehlers-Danlos syndrome and Marfan Syndrome.
- *Dandy-Walker Syndrome:* Dandy-Walker complex is a congenital brain malformation involving the cerebellum and the fluid filled spaces around it. Symptoms of increased intracranial pressure and signs of cerebellar dysfunction such as unsteadiness, lack of muscle coordination or jerky movements of the eyes may occur. Other symptoms include increased head circumference, bulging at the back of the skull, problems with the nerves that control the eyes, face and neck, and abnormal breathing patterns.
- Intracranial tumor
- Mucopolysaccharidoses which obliterate the subarachnoid space – Group of metabolic disorders caused by the absence or malfunctioning of lysosomal enzymes needed to break down molecules called glycosaminoglycans.
- Achondroplastic disorders (which alter occipital bone growth and impede venous outflow).
- Arachnoid cyst.
- Porencephaly.
- X-linked hydrocephalus.
- In utero intraventricular hemorrhage.
- Maroteaux-Lamy syndrome.

What is the clinical presentation of hydrocephalus both in an infant and in older child?

- Infant and newborn
- Cranium enlarges at rate > facial growth
- Fontanelle full and bulging
- Poor head control
- Irritability, lethargy
- Delayed milestones
- Nausea/Vomiting
- Enlargement and engorgement of the scalp veins—due to reversal of flow from the intracerebral sinuses due to increased ICP
- Macewen's sign cracked pot sound on percussing over-dilated ventricles
- Setting sun sign (upward gaze palsy): Parinaud's syndrome from pressure on the region of suprapineal recess
- Hyperactive reflexes

- Cranial nerve palsies
  - Lateral gaze palsy (6th nerve—abducens palsy): the long intracranial course is postulated to render this nerve sensitive to pressure. (More common with Arnold-Chiari malformation)
  - Stridor due to bilateral vocal cord palsy due to stretching of the Vagus N over the lip of the jugular foramen
  - Cortical blindness (Optic N)
  - Visual backing disorder (Oculomotor N)
- Irregular respiration and apneic spells
- Splaying of cranial sutures (seen on plain X-ray skull).

Older child/adults with rigid cranial vault

Symptoms of increased ICP including papilledema, headache, nausea vomiting, gait changes, upward gaze and abducens palsy. Slowly enhancing ventricles may initially be asymptomatic.

Chronic Hydrocephalus

- Beaten copper cranium (beaten silver appearance) on plain X-ray
- 3rd ventricle herniating into the sella (on CT, MRI)
- Erosion of sella turcica
- Macrocrania
- Atrophy of corpus callosum
- In infants—sutural diastasis, delayed closure of fontanelles, developmental delay and failure to thrive.

Signs and symptoms of hydrocephalus are due to the acute or chronic rise in intracranial pressure. Acute hydrocephalus may be caused by intraventricular hemorrhage in the premature infant, hemorrhage into a tumor, or expansion of a colloid cyst of the third ventricle. The symptoms may be unrecognized in non-verbal and developmentally delayed child. Vomiting, dehydration, lethargy, neurogenic pulmonary edema, and coma are indications of emergency decompression to prevent herniation of the brain stem, respiratory and cardiac arrest and possibly death.

Chronic hydrocephalus can be due to congenital aqueductal stenosis, meningitis, and spinal cord tumors.

Slowly progressive signs like irritable behavior, poor school performance, intermittent headaches, rambling speech, altered behavior, confusion lethargy, unsteady gait, seizures, incontinence and papilledema may be present.

In neonates, nonfusion of the cranial sutures cause widening of the sutures and enlargement of the head circumference causing airway obstruction.

In infants and small children, vomiting and decreased oral intake is common presentation and can be misdiagnosed as viral illness or gastroenteritis. Acute obstructive hydrocephalus in undiagnosed intracranial neoplasm may lead to sudden and unexpected death due to cerebral herniation.

What is Normal Pressure Hydrocephalus and what are its characteristics?

Normal pressure hydrocephalus (NPH) is a chronic type of communicating hydrocephalus whereby the increase in intracranial pressure (ICP) due to accumulation of cerebrospinal fluid (CSF) becomes stable and that the formation of CSF equilibrates with absorption. The ICP gradually falls but still maintains a slightly elevated level and the CSF pressure reaches a high normal level of 150 to 200 mm H_2O. Because of this equilibration, patients do not exhibit the classic signs of increased intracranial pressure such as headache, nausea, vomiting, or altered consciousness. However, patients do exhibit the classic Hakim triad of symptoms includes gait difficulties, urinary incontinence, and mental decline (dementia) as first described by Hakim and Adams in 1965. It is often misdiagnosed as Parkinson's disease, Alzheimer's disease, and senility due to its chronic nature and its presenting symptoms.

Although the exact mechanism is unknown, normal-pressure hydrocephalus is thought to be a form of communicating hydrocephalus with impaired CSF reabsorption at the arachnoid villi.

How will you diagnose this condition?

1. History and clinical findings.
2. Serial head circumference measurements.
3. Skull radiographs.
4. CT scan confirms diagnosis.
5. Shunt scan (in patients with a shunt to determine the site of malformation).
6. Fundus examination.
7. Venous ophthalmodynamometry: This technique may be used in the differential diagnosis of malfunction of ventricular shunts, gastrointestinal disorders, hypertensive hydrocephalus, and brain atrophy.
8. Transcranial Doppler: Transcranial Doppler is a non-invasive method of evaluating hydrocephalus. Decrease in diastolic velocity and increase in pulsatility index (systolic velocity-diastolic velocity/mean velocity) is diagnostic.

What is the medical treatment of this condition?

Hydrocephalus remains a surgically treated condition.

Diuretic therapy: May be tried in premature infants with bloody CSF (as long as there is no evidence of active hydrocephalus) while waiting to see if there is resumption of normal CSF absorption. However, this is considered as an adjunct to definitive treatment or as a temporizing measure.

Satisfactory control has been seen in 50% of children <1 year who had stable vital signs and no symptoms of elevated ICP using the following:

- Acetazolamide (a carbonic anhydrase inhibitor): 25mg/kg/day PO divided TID increase by 25mg/kg/day until 100 mg/kg/day is reached
- Simultaneously start Furosemide: 1mg/kg/day PO divided TID
- Osmotic diuretic—not preferred because of rebound phenomenon
- Correct acidosis
- Watch for and correct electrolyte abnormalities
- Watch for acetazolamide side-effects—lethargy, tachypnea, diarrhea.
- Perform weekly ultrasound or CT scan and insert ventricular shunt if ventriculomegaly occurs.

Spinal Taps: Hydrocephalus after intraventricular hemorrhage may be only transient. Serial taps (ventricular or LP) may temporize until resorption resumes, but LPs can only be performed in communicating hydrocephalus. If spontaneous resorption does not occur shunts will be required.

Describe in detail the common neurosurgical procedures for this condition?

An emergency ventriculostomy or extracranial ventricular drainage may be performed before the direct surgical introduction of a ventricular shunt.

For ventriculostomy, right frontal region is preferred because it is rarely the dominant hemisphere and this can be done bedside in ICU. Ventriculostomy also allows for monitoring of intracranial pressure in the postoperative period.

Choroid plexectomy: For communicating hydrocephalus, open surgery is associated with a higher mortality.

Eliminating the obstruction: (e.g. opening the stenosed sylvian aqueduct). Often has higher morbidity and lower success rate than simple CSF diversion with shunts, except perhaps in case of a tumor.

Third ventriculostomy: There is a resurgence of interest in this procedure due to the increased ventriculoscopic surgery. Developments in optics, image processing and improvements in anesthesia have enabled Endoscopic Third Ventriculostomy to be used safely with comparable results to VP shunts. It is usually done in patients with obstructive HCP. May be an option for managing shunt infection as a means to remove hardware without subjecting the patient to raised ICP. This has also been proposed as an option for patients who develop subdural haematomas after shunting.

Shunting: Different types of shunt procedures can be performed:

- Ventriculoperitoneal shunt
- Ventriculoatrial shunt
- Ventriculopleural shunt
- Torkildsen shunt
- Lumboperitoneal shunt.

Describe the ventricular shunts available for this condition?

In most cases, surgical treatment of hydrocephalus consists of insertion of a ventricular shunt. The shunt is an artificial device designed to maintain normal intracranial pressure.

Shunts usually consist of three parts:

1. Proximal end is radio-opaque and is placed into the ventricle. This end has multiple small perforations.
2. Valve allows for unidirectional flow. Can adjust various opening pressures. Usually has a reservoir that allows for checking shunt pressure and sampling CSF
3. Distal end is placed into the peritoneum or another absorptive surface by tracking the tubing subcutaneously.

Essentially two types of shunts are available, the pressure regulating shunt and the flow regulating shunt. The pressure-regulating shunts are designed to maintain a difference of pressure between their inlet and outlet, and they allow flow of CSF once that preset pressure has been reached. The flow-regulating shunts are designed to allow a constant flow of CSF, simulating the normal flow of CSF. The differential pressure valves are more prone to cause overdrainage complications, whereas the flow regulating valves are more prone to valve obstruction. To avoid overdrainage, the differential pressure valves are further divided into low pressure (20–40 mm water), medium pressure (40–70 mm water) and high pressure (80–100 mm water).

Shunt insertion is done through parieto-occipital burr hole on the non-dominant side so as to place the ventricular catheter in the frontal horn of the lateral ventricle. With this approach, the shunt valve is situated behind the ear and is easily palpable by the parents and patients through the skin. The distal catheter then drains the CSF from one-way valve to one of the extracranial locations in the body from where it gets absorbed.

The Ventriculo-Peritoneal (VP) shunt is small tubing that is placed inside the brain's ventricle and tunneled underneath the skin to the peritoneum. The purpose of the VP shunt is to reduce the amount of cerebral spinal fluid (CSF) in the brain by draining it into the abdominal (peritoneal) space. The peritoneum is the usual, safe site and also the most preferred to place the end of the ventricular shunt.

Certain circumstances may require placing the end of the shunt tubing into a blood vessel leading to the heart (Ventriclo-Vascular or Atrial, VA) or into the pleural space of the chest (Ventriculo-Pleural, VP). In VA shunt, the distal tube is put under the fluoroscopic guidance. CT scan is done before discharge to verify the position of the ventricular catheter and serves as future reference. Other options may be the gall bladder, ureter and urinary bladder.

What are the common complications of shunt procedures?

Common to all Types of Shunts:

- Malfunction—because of obstruction, disconnection, breakage, migration
- Infection
- Overdrainage—early over drainage can cause subdural hematoma. Chronic overdrainage can lead to intermittent shunt obstruction due to slit ventricle symptom. It can also give rise to secondary craniosynostosis and thick skull (hyperostosis cranii ex-vacuo)
- Pneumocephalus
- Seizures—5.5% risk of seizures in the first year
- Hardware erosion through skin
- Act as a conduit for extraneural metastasis of certain tumors
- Silicone allergy.

Complications Unique to Ventricular Peritoneal Shunts

- 17% incidence of inguinal hernia
- Obstruction of peritoneal catheter – due to occlusion by omentum, by peritoneal cyst, severe peritoneal adhesions.
- Peritonitis from shunt infection
- Hydrocoele
- CSF ascites
- Tip migration—into scrotum, through diaphragm, perforation of viscus
- Intestinal obstruction, volvulus
- Overshunting.

Complications Unique to Ventricular Atrial Shunts

- Requires repeated lengthening in a growing child
- Higher risk of infection
- Possible retrograde flow of blood into the ventricles if valve malformation
- Shunt embolism
- Vascular complications—thrombophlebitis, pulmonary microemboli, pulmonary hypertension.

What mechanisms regulate Cerebral Blood Flow (CBF)?

Cerebral perfusion pressure is defined as the difference between the mean arterial pressure (MAP) and the sum of intracranial pressure and the central venous pressure (CVP).

$$CPP = MAP - (ICP + CVP)$$

The cranial contents consist of brain tissue (80%), blood (10%) and cerebrospinal fluid (10%). The volume of the cranial vault is fixed once the sutures of the skull have become fused, normally by 2 years of age. In the neonate, even though the volume of the vault can expand if the increase in intracranial contents occurs gradually, such as in congenital hydrocephalus, this accommodation has a maximum limit which once exceeded will lead to an increase in ICP. Any changes in the volume of the individual cranial contents can lead to an alteration in ICP and this has both pathological and therapeutic implications.

Cerebral blood flow (CBF) determines cerebral blood volume and is higher in children compared to adults (100 vs 50 mL/100g/min). It is coupled to the metabolic demands of the normal brain and regulated via oxygen requirement, $PaCO_2$ and intracerebral acidosis. Autoregulation allows for a relatively constant blood flow across a wide range of arterial pressures which can be as low as 40 mm Hg in small children. This mechanism is not available to premature neonates or during some pathological processes (e.g. infection) where the relationship between CBF and arterial pressure is more linear. Reducing $PaCO_2$ causes vasoconstriction of the cerebral vessels, reducing CBF, blood volume and ICP but at the expense of oxygen delivery. Hyperventilation should only be used as a short-term measure when ICP is high.

Cerebrospinal fluid (CSF) is continuously produced by the choroid plexus, and after circulating through the ventricles is absorbed at the arachnoid villi. In children the rate of production of CSF is 0.2–0.4 mL/min with around 250 mL produced and absorbed per day and at any given time around 70 mL is present in the head. Any interruption to normal flow, increased production or decreased reabsorption of CSF can manifest itself as hydrocephalus.

What investigations will you ask for this child?

1. Complete blood count
2. ESR
3. Renal function
4. Serum electrolytes
5. X-ray skull
6. CT scan
7. Investigations related to coexisting medical condition or syndrome.

What are the important points to be considered while taking preoperative history and examination?

The history and examination is an essential component of the pre-operative assessment and should include:

- Standard anesthetic history
- Birth history (prematurity)
- Immunization history
- Family history
- History of any allergies
- History of problems with previous anesthesia/surgery
- Drug History: past medications, current medication (e.g. anticonvulsants, acetazolamide, furosemide)
- Fasting status
- Associated anomalies
- Possibility of co-morbidities
- Systemic:
  - *CNS:* Level of consciousness, cranial nerve palsies, Signs of raised ICP, Brain stem involvement (reduced level of consciousness and therefore increased risk of pulmonary aspiration).
  - *RS:* Assessment of the airway, bronchopulmonary dysplasia, kyphoscoliosis, recurrent respiratory infections secondary to neurological dysfunction, If the preoperative assessment reveals a patient with multiple co-morbidities, such as an ex-premature infant with chronic lung disease, the need for additional respiratory support in the postoperative period should be considered and organized in advance.
  - *CVS:* congenital cardiac disease.
  - *Volume status* should be determined as prolonged vomiting and dehydration may necessitate intravenous fluid preoperatively.

How will you optimize this patient prior to surgery?

This child will require immediate surgery in view of the clinical findings.

Antibiotics need to be started since the patient is febrile and shows clinical signs of infection.

Ensure adequate hydration and correction of any dyselectrolytemia if present in view of the persistent vomiting. Medical treatment may be started while awaiting surgery.

What are the factors to be considered while premedicating the child for VP Shunt?

- Sedative premedication should be considered carefully as it may exacerbate or mask signs of neurological dysfunction. Narcotics and sedatives may depress ventilation, resulting in further rise in ICP. As a general rule preoperative sedation should not be used. However, an anxious combative patient may have a detrimental rise in ICP during induction. Hence, sedation should be given only if required in the above situation keeping in mind the factors stated. A titrated dose of oral or intranasal midazolam may be considered, however, the child should be carefully monitored whilst awaiting surgery for signs of neurological deterioration as this can occur rapidly.
- Prophylactic antibiotics should be given to lower shunt infection rate and continued if already started in a child with signs of infection or if shunt revision is planned

What are the anesthetic considerations for Ventriculo-Peritoneal Shunt?

- The child should be carefully monitored whilst awaiting surgery for signs of neurological deterioration as this can occur rapidly. If patient suffers respiratory arrest or sudden deterioration in neurological status due to ↑ ICP and tonsillar herniation, endotracheal intubation and hyperventilation with 100% oxygen should be carried out and child should be prepared for urgent ventricular tap.
- Latex precautions are recommended for patients with myelomeningocele undergoing shunt placement.
- The method of induction is determined by the circumstances of the case and the preference of the patient and the anesthetist. The goal should be to avoid any further rise in ICP and avoid hypotension which will decrease the CPP during this period. IV or inhalational induction can be used in a child with normal ICP.
- Intravenous induction allows for rapid control of the airway in emergency situations (raised ICP) if the patient is not fasted and if no difficulties with the airway have been anticipated. This may be achieved using a suitable induction agent in a carefully titrated sleep dose after preoxygenation (propofol 2–4 mg/kg, thiopentone 3–5 mg/kg). Ketamine should not be used as this will increase the ICP.
- Gaseous induction is an acceptable alternative with a non-irritant volatile anaesthetic agent such as sevoflurane or halothane especially when IV cannula is not in place. However, intravenous induction should be preferred if child has signs of raised ICP, stupor or delayed gastric emptying.
- Muscle relaxation can be achieved with the use of a neuromuscular blocking agent (NMB). Suxamethonium can be used if the risk of aspiration outweighs the problems of transient increases in ICP, otherwise non-depolarizing NMB are preferable.

- Endotracheal Intubation—In case of a very large head, intubation can become technically difficult. Proper positioning using a pad or bolster below the shoulder can help overcome this problem. Lateral position may also be tried for intubation. Flexometallic tube may be used to avoid kinking during head rotation. The tube should be firmly secured.
- Anesthesia can be maintained with a volatile agent and a mixture of oxygen and air. The aim is to maintain CPP until the raised ICP is relieved by positioning of the VP shunt. To do this hypotension should be avoided and minute ventilation controlled to maintain normocarbia to optimize CPP and avoid increases in ICP. Positive end-expiratory pressure (PEEP) should be minimized to avoid venous congestion in the head but may be used if there are difficulties in maintaining oxygenation.
- *Ventilation:* Mild hypocapnia (32 -35 mmHg) following tracheal intubation may prevent further elevation of PaCO2, however, aggressively decreasing the $PaCO_2$ increases the risk of brain ischemia and is not currently recommended. Normocapnia should be maintained in patients with normal ICP.
- *Positioning during surgery:* The head is turned away from the surgeon with a pad/bolster below the operative site. Extreme rotation of the head can impede venous return through the jugular veins and lead to impaired cerebral perfusion, ↑ ICP and cerebral venous bleeding. All anaesthetic equipments and IV lines must be on the side of the anesthetist. The patient should be positioned to allow good access for the surgeon and to avoid any undue pressure on vulnerable areas.
- Measures to maintain CPP and prevent rises in ICP.
- Consideration should be given to optimizing venous drainage to increase CPP, reduce venous bleeding and improve the surgical field.
- *Temperature Control:* The core temperature of the patient should be monitored (rectal or esophageal) and warming devices (e.g. warm air blankets) used as required to maintain normothermia.
- On cannulating the ventricle, BP may drop abruptly as brain-stem pressure is suddenly releazed.
- The most stimulating parts of the surgery include the initial incision and tunneling under the skin. A short acting opioid, such as fentanyl (1–3 mcg/kg) or remifentanil (1 mcg/kg), or increased depth of anesthesia, can be used to attenuate the increase in heart rate and ICP.
- Normal Saline or Ringer Lactate can be used as maintenance fluid as they are mildly hyperosmolar and theoretically attenuates cerebral edema.5% Dextrose should not be used as this is hypoosmolar and can increase cerebral edema.
- Prophylactic antiemetic before 30–60 minutes of extubation.
- At the end of the procedure neuromuscular blockade can be antagonized using neostigmine combined with an anticholinergic (e.g. atropine). Most patients can be extubated once awake, avoiding hypercarbia, and with a technique which minimizes the risk of aspiration. Patients with severe neurological deficits may have poor airway control and can be more prone to postoperative respiratory problems.

What is the relationship between Cerebral Perfusion Pressure (CPP) and the Intracranial Pressure (ICP) and what measures can we take to manipulate them in the intraoperative period?

$$CPP = MAP - (ICP + CVP)$$

CPP can increased by either increasing MAP (with fluids, vasoconstrictors or inotropes) or by reducing ICP (hyperventilation, mannitol, sedation and analgesia) and reducing CVP (head up position, head midline, prevent obstruction of venous drainage of neck with by avoiding extreme neck rotation.

Most anesthetic agents cause a reduction in MAP but this potentially deleterious effect is usually offset by the corresponding reduction in the cerebral metabolic demand for oxygen ($CMRO_2$).

What are the indications for rapid sequence intubation in these children?

Child with a full stomach for emergency surgery, delayed gastric emptying and vomiting are indications for rapid sequence intubation. Child with severe neurological compromise may have a gastrostomy tube preoperatively which should be aspirated before induction and left open to prevent gastric distension and regurgitation.

How is the child positioned during surgery and in the postoperative period?

- *During Surgery:* The head is turned away from the surgeon with a pad/bolster below the operative site. Extreme rotation of the head can impede venous return through the jugular veins and lead to impaired cerebral perfusion, raised ICP and cerebral venous bleeding. All anesthetic equipments and IV lines must be on the side of the anesthetist. The patient should be positioned to allow good access for the surgeon and to avoid any undue pressure on vulnerable areas.
- *Postoperatively:* Patient should be nursed flat so as to avoid subdural hemorrhage which may occur because of rapid collapse of ventricles in head high position.

Why is the use of Nitrous oxide not recommended for this procedure?

The use of nitrous oxide is not recommended as it significantly increases the cerebral blood flow and volume which can contribute to an elevation in ICP and also since it is associated with a strong emetic effect it may confuse the evaluation of the patient postoperatively.

Why is intraoperative spontaneous ventilation not recommended during this surgery?

Intraoperative spontaneous ventilation is not recommended due to the risk of air embolism during craniotomy and ventriculoatrial shunt placement and also due to the risks of pneumothorax during ventriculopleural shunt placement.

What are the current recommendations for maintaining the intraoperative $PaCO_2$?

Mild hypocapnia ($PaCO_2$ 32–35 mm Hg) following tracheal intubation may prevent further elevation of $PaCO_2$, however aggressively decreasing the $PaCO_2$ increases the risk of brain ischemia and is not currently recommended. Normocapnia ($PaCO_2$ 35–45 mm Hg) should be maintained by in patients with normal ICP.

How will you monitor the child during the intraoperative period?

This procedure does not usually require invasive monitoring. Intraoperative monitoring should include:

- ECG
- Pulse oximeter
- NIBP monitor
- Capnometer
- Precordial stethoscope
- Temperature monitor
- Arterial catheterization—reserved for patients with uncontrolled ICP and hemodynamic instability.

How will you manage postoperative pain in this child?

Postoperative analgesia can be provided by a combination of infiltration of local anesthetic such as bupivicaine 0.25% (0.5–0.75 mL/kg) and with a combination of paracetamol (15 mg/kg) either intravenous or per rectum and non-steroidal anti-inflammatory drugs (NSAIDs) if not contra-indicated with additional oral opioid for breakthrough pain.

High doses of long-acting opioids should be avoided because of potential detrimental effects on conscious level. Patients with severe neurological deficits can be more prone to postoperative respiratory problems, hence analgesics should be used judiciously and under close supervision in these patients.

What are the signs and symptoms of shunt malfunction? What is the incidence of revision shunt/shunt removal and what problems can you anticipate during these procedures?

In a child with a ventriculoperitoneal shunt, the shunt is statistically unlikely to be the cause of any specific problem. However, if family members suspect shunt malfunction or no other cause for fever, malaise, behavioral change, etc. can be found (i.e. ear infection), careful and diligent evaluation of the shunt is mandatory.

Median survival of a shunt (before need for revision) in a child < 2 years of age, is 2 years; > two years of age is 8–10 years.

The common problems of shunt malfunction may be due may be due to obstruction or infection. Disconnections and breakage of tubing are another cause of malfunction, though less common than occlusion. Migration into the scrotum, perforation of the bowel wall, and intussusception are all rare complications in the peritoneum.

Common Signs and Symptoms of Shunt Malformation Due to Obstruction and Infection

Obstruction: Headache, malaise, general not feeling well, vomiting, mental status alterations, increased blood pressure, head circumference increase, Cushing's triad, bulging fontanelle, sixth nerve palsy signs, Macewen's sign, changes in gait, and personality changes. There may also be an increase of seizures and a complaint of neck pain. The parents often recognize signs of shunt block. Teachers may state that there has been a change of school performance.

Infection

- Fever, meningeal signs, vomiting, signs and symptoms of shunt malfunction, abdominal pain, and peritonitis
- There may be evidence of purulent material around the shunt insertion site and redness along the shunt tract
- Most common organisms are *S. epidermidis and S. aureus* and also gram-negative organisms
  External ventricular drainage may be tried in such cases.

If shunt revision/removal is planned, the following points need to be kept in mind:

- Psychological trauma of repeat surgery for parents
- Intravenous cannulation may be difficult due to repeat procedures
- Antibiotics will need to be started if infection is suspected
- Medical measures to decrease ICP until shunt removal

- In cases of shunt revision or shunt removal surgery, retrieval of the ventricular catheter may cause rupture of the choroids plexus which has grown into the shunt lumen. This can lead to life-threatening hemorrhage.

Suggested Reading

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19 Meningomyelocele

S Bakshi, S Myatra

What is meningomyelocele?

Meningomyelocele (MMC) is a congenital defect in vertebral arches with cystic dilatation of meninges and structural or functional abnormality of spinal cord or cauda equina. Meningocele on the other hand is a congenital defect in vertebral arches with cystic distension of meninges, but no abnormality of neural tissue but neurologic deficit may be present in a third of patients. Meningomyelocele is a progressive neurologic disease that eventually produces orthopedic, neurologic and genitourinary complications. Kyphoscoliosis is common in patients with thoracic lesions, and it occurs in 20% of patients with lumbosacral defect.

What is the pathophysiology of this condition? Discuss the embryologic basis of these defects.

The nervous system develops in two stages:

Primary neurulation: The neural folds elevate, approximate each other, and start closing, thus forming the neural tube. The point of initial closure occurs at the caudal rhombencephalon or cranial spinal cord. The cutaneous ectoderm fuses first, followed by the neuroectoderm. The caudal neuropore closes between T11 and S2. Parallel to this process, the cutaneous ectoderm separates from the neuroectoderm to form the overlying skin, while the lateral mesoderm migrates between the 2 ectodermal layers to form the posterior vertebral arches.

Secondary neurulation: This comprises further neural development occurring caudal to the caudal neuropore after the termination of primary neurulation. This process includes formation of the filum terminale and conus medullaris from a poorly-differentiated cell mass of the medial eminence. Because of differential growth between the vertebral column and the spinal cord, the conus becomes more rostral during later development.

Meningomyelocele constitute primary neural tube defect, which is due to primary failure of closure of neural tube or possibly disruption of already closed neural tube between 18 – 28 days of gestation. The other primary neural defects include

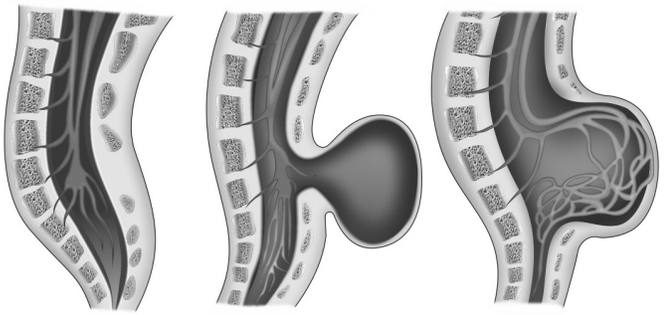
Encephalocele which is an out-pouching of dura with or without brain, noted in occipital region commonly.

Anencephaly is a severe form of defect not compatible with life. Cranial vault and posterior occipital bone are defective, and derivatives of the neural tubes are exposed, including both brain and body tissues.

Meningocele is the herniated protrusion of only the meninges and constitutes secondary neural tube defects. It results from abnormal development of lower sacral or coccygeal segments during secondary neurulation. These lesions rarely are associated with hydrocephalus. The skin is typically intact over the defect. Other secondary neural tube defects include: Sacral agenesis, dysgenesis, diastematomyelia (split cord), and myelocystocele.

Spina Bifida

Spina bifida occulta is defined as failed fusion of the neural arch without herniation of the meninges or neural elements. Superficial sign of this lesion may include tuft of hair, cutaneous angiomas, lipoma or a skin dimple.



What are the causes and incidence of this condition? How is the intrauterine and postnatal diagnosis made?

MMC is more common in low socioeconomic class. Incidence is highest in Hispanics and lowest in Asians and Pacific islanders. Female: Male: 1.2:1. Risk increases to 2–3% if there is one previous birth with meningomyelocele and 6–8% after two affected children

Etiology in MMC is mutlifactorial.

- Specific etiologic factors are:
- Nutritional deficiency of folic acid in mothers
- Hypervitaminosis A
- Associated chromosomal abnormalities – trisomies 13 and 18, single-gene mutations
- Maternal insulin-dependent diabetes mellitus (IDDM), maternal obesity
- Maternal hypothermia
- Intrauterine drug exposure to valproate, carbamazepine and ovulation inducing drugs.

Intrauterine Diagnosis

Laboratory Studies:

- Elevated maternal serum α fetoprotein levels in second trimester
- Amniotic fluid α fetoprotein assay
- Presence of acetyl cholinesterase (a nerve-specific enzyme) in amniotic fluid.

Imaging studies:

- Fetal ultrasound at about 18 weeks gestational age.

Postnatal diagnosis: Meningomyelocele is immediately obvious at birth.

After delivery MRI of spine helps in accurate assessment. Serial cranial ultrasounds, CT scan, and MRI of brain is often needed in cases of concomitant hydrocephalus.

What syndromes and anomalies are associated with this condition?

Neural tube defects are often associated with anomalies of central structures which include the heart, esophagus, kidneys, brain, limbs, and anal canal. Associated anomalies which may be present:

1. Club feet (neurogenic)—unilateral/bilateral (commonest).
2. Arnold-Chiari malformation due to associated abnormality of the cephalic anterior neural tube (80–90%). This is the abnormality of neural tube characterized by caudal displacement of posterior fossa structures, kinking of cervicomedullary junction, the disruption of the brainstem vascular supply and lower cervical nerves. It is often associated with meningomyelocele, hydrocephalus and syringomyelia.
3. Hydrocephalus (80%).
4. Neurogenic bladder (90%).
5. Musculoskeletal defects (absent ribs, scoliosis).
6. Urogenital anomalies- Extrophy bladder, undescended testis, hydronephrosis, solitary kidney or malformed ureters.
7. Hip dislocation (neurogenic).
8. Facial clefts.
9. Anorectal malformations.
10. Congenital heart disease—ASD,VSD.
11. Umbilical hernia.
12. *VATER-L:* Vertebral, anal, TE – fistula, renal (genitourinary) and limb anomalies. These should be diagnosed prior to the repair, because they may preclude a long-term outcome.

What are the common clinical findings in this condition?

- Paraplegia
- Hydrocephalus
- Cranial nerve dysfunctions
- Seizures
- Neurogenic bladder and bowel
- Renal failure
- Progressive bony, spine and joint deformities,
- Pathological fractures.

What specific history will you ask for in patient with MMC? How will you proceed with the examination in these patients prior to corrective surgery?

Specific history includes:

- Antenatal history of drug ingestion
- Similar defect in siblings
- Birth history—prolonged labor and fetal hypoxia can occur in babies with large head due to hydrocephalus
- History to rule out associated symptoms and signs like seizures
- Increased precordial activity, found in assoc congenital heart disease
- Limb movements to rule out neurological deficits.

General Assessment of the Newborn

- For overall development and nutrition
- Look for deformities, evidence of muscle weakness, contractures.

Back Examination Includes

- Inspect the defect—its location, size and shape and any cerebrospinal fluid leak
- Note the curvature of spine
- Palpate for deformity like spina bifida which is generally associated with MMC. One should exercise due caution while palpating
- Latex allergy is known and it is preferable to use a sterile non-latex gloves while examining the sac.

Neurological Examination

- Observe for spontaneous activity to sensory stimuli in all extremities
- Tendon reflexes could often be absent in associated neurological deficits
- Signs of associated hydrocephalus
  - Increase in the head circumference
  - Bulging of the anterior fontanel
  - Cranial nerve involvement abducent nerve paresis leading to conjugate movement abnormalities.

Airway Assessment

- Rule out associated facial cleft
- In children with Arnold-Chiari syndrome—inspiratory stridor, and apneic episodes is common.

Associated congenital anomalies (to be ruled out)

- Kidney enlargement
- Club feet
- Hip dislocation
- Cardiomegaly.

What investigations will you ask for in this patient?

Investigations needed include the routine baseline and specific (neurological) investigations. These include:

- Hemoglobin, platelet count.
- White cell count could be elevated in cases of infection which could be cause of an infected sac, at times UTI are also seen.
- Serum creatinine.
- Urine routine and microscopy- important in this case as various associated anomalies make the child susceptible to repeated episodes of urinary tract infection.
- X-ray chest may be asked if child is symptomatic. Also useful to rule out cardiomegaly.
- Blood grouping and cross-matching: With large defect, blood loss may be significant and may warrant transfusion.
- CT Brain and MRI—to evaluate the presence of hydrocephalus. With hydrocephalus MRI shows characteristic appearance, i.e. occipital horns are more dilated than the frontal horns, and the long-axis of left ventricle tends to be parallel.
- As meningomyelocele may be associated with involvement of other organ systems, additional investigations will be warranted as per the presence of this involvement.
  - 2D echocardiography: to rule out congenital heart disease.
  - Ultrasound of urinary tract: To detect possible hydronephrosis and structural abnormalities of urinary tract.
  - Urodynamic studies—such as voiding cystourethrogram.

Describe the management of child with meningomyelocele? What are the surgical options for this condition?

Management of a child with MMC includes the following three aspects:

1. Assessment and management of lesion
   a. If lesion has ruptured start antibiotics
   b. Cover the lesion with sponges soaked in NS or RL to prevent desiccation
   c. Trendelenberg position, with child on stomach (keeps pressure of lesion)
   d. Monitor infant closely for signs of meningitis.
2. Neurological assessment
   a. Assess and document presence of neurodeficit.
3. Ancillary assessment and management
   a. Evaluation by neonatologist to assess other abnormalities
   b. Urological consultation
   c. Orthopedic consultation for severe kyphotic or scoliotic spine deformities and hip or knee deformities.

What are the surgical considerations in MMC patients?

Surgery in utero is now available at many centers. The benefit of early correction is being evaluated at many centers.

In case an early diagnosis is made and in utero correction has not been done, then the pediatric team should be present for delivery, which should almost always be by cesarean section. Since these patients are at a high-risk of developing latex allergies; which can be quite severe and life-threatening. Therefore, attempts should be made to avoid latex exposure and sensitization.

Timing of closure: Early closure of MMC defect is not associated with improvement of neurological function, but leads to lower infection rates. The defect should be closed within 24 hours whether or not membrane is intact.

Simultaneous repair and VP shunting: In patients with clinically overt hydrocephalus at birth, MMC repair and VP shunt may be done in the same sitting without increased risk of infection.

If VP shunt is not performed, an increased risk of MMC repair breakdown is present.

Outcomes: With modern treatment 85% of infants survive. Mortality is mainly related to associated conditions such as Arnold-Chiari syndrome, shunt malfunction and infections. 80% patients will have normal IQ.

What are the problems anticipated in the perioperative period?

- Problems related to age
  - Mainly a neonate/infant
- Problems related to airway
  - Large head (if assoc hydrocephalus)
  - Associated facial clefts
  - Paediatric airway and its implications
- Problems related to other associated syndromes/ anomalies
  - Hydrocephalus
  - Arnold-Chiari syndrome, often presents with stridor
  - Congenital heart disease
  - Renal problems
  - Musculoskeletal disorders, succinyl choline should be avoided in cases of muscle weakness
- Problems related to surgery
  - Surgery in prone position
    - Extreme head flexion in Chiari malformation may cause brain stem compression
    - Improper positioning may lead to venous congestion of face, tongue and neck, reduce lung compliance
    - Increase abdominal pressure causes vena caval compression that can cause increase bleeding through engorged epidural veins
    - Often surgery dissection could be difficult in case of tethered cord
    - Hypothermia
    - Blood loss can be difficult to assess in view of simultaneous CSF loss
    - Nerve studies may be needed intraoperatively to help in identification in few cases
    - At dural closure Valsalva maneuver is needed to test the integrity of closure
- Problems in the postoperative period
  - Due to nursing in prone position
- Acute hydrocephalus
    - If not present preoperatively, can occur due to closure of defect
    - May occur due to shunt malfunction.

How will you premedicate this child? How will you induce anesthesia?

The need to premedicate will depend upon the age of the child. Since defect correction is done early, the neonate will not need anxiolytics for premedication. Antisialogogue may be given as the patient will be placed prone. Blood sugar monitoring would be needed. In case if the parents bring the child later in infancy, which is not uncommon in our country—premedication, to allay separation anxiety, will be needed. The choice will depend upon presence of associated raised ICT. In such cases ketamine (oral/intramuscular) is best avoided and oral midazolam is preferred.

In 1995, Viscomi et al reported a series of 14 neonates will lumbar or sacral meningomyelocele which were done under spinal anesthesia. However, general anesthesia remains the technique of choice as the surgery is in prone and airway remains secure.

Induction of General Anesthesia

- In syndromic babies and abnormal facies, a difficult airway is anticipated. The induction agent of choice would be inhalational anesthetic. Sevoflurane is preferred. Intubation with difficult airway kit should be ready and spontaneous ventilation is safest.
- In patients with neurological deficit succinyl choline should be avoided.
- In babies with a huge defect placement of the child during intubation is important.
- The MMC should be placed with adequate padding and the rest of upper part of the body could rest on an adequate size pillow.
- Alternatively the patient can be administered anesthetics and trachea intubated in the lateral position.

How is the child positioned? How will you maintain anesthesia and monitor this child during surgery?

The child is positioned prone on adequate sized bolster and head well-supported all bony points and eyes are padded and abdomen should be free. Anesthesia is maintained with inhalational agents and opioids for pain relief. Use of relaxant should be timed properly in case if nerve studies are needed for nerve identification intraoperatively.

Monitoring: Routine monitoring that is used includes ECG, pulse oximetry, capnometry, non-invasive blood pressure, invasive BP may be needed in patients with large defects and difficult anatomy. Temperature monitoring hourly urine output, careful and accurate measurement of blood loss.

What are the anesthetic implications for subsequent surgery in this child?

Due to multi-system involvement the patient may need repeated surgeries for the following:

Hydrocephalus: Child may need VP shunt, if shunt is done already it may require revision procedure.

If the patient is undergoing tethered cord correction, muscle relaxation may have to be withheld at the time of motor function assessment. The child may need orthopedic intervention later for correction of skeletal deformities. Scoliosis deteriorates in the presence of untreated hydrocephalus and improves following successful shunting in patients with MMC. Neurogenic bladder which does not respond to clean intermittent catheterization (CIC) and/or medications may require surgical intervention for augmentation cystoplasty and urinary diversion to prevent renal failure. The number of surgical interventions and atopic disposition lead to development of latex allergy in these children. Establishment of latex-free environment for surgery is a must. Children with meningomyelocele often also have nerve problems and dysfunction of the bowels, known as neurogenic bowel. They will often have poor or weak bowel peristalsis and motility. Malnutrition is commonly seen in such children.

What is the role of fetal surgery in these patients? What are the considerations for the mother during fetal surgery?

Recently patients with severe meningomyelocele and other anomalies have been treated inutero. The neurological outcomes are better than surgeries done during neonatal period. There are excellent reviews on this subject (cited below). Essentially fetal surgery can be of the following types:

1. Minimally invasive (e.g. insertion of shunt).
2. Fetoscopic surgery (e.g. surgery for congenital diaphragmatic hernia called FETO—fetal endoscopic tracheal occlusion).
3. Open surgery involving hysterotomy, often called ex utero intrapartum treatment (EXIT) procedure is done in second trimester or near term. This generally involves securing the airway while the fetus is still being oxygenated by the placental circulation. This is generally performed for obstructive lesions of the airways such as cystic hygroma or congenital goiter.

The basics remain the same for a pregnant patient undergoing non-obstetric surgery where aim is to prevent premature labor. (See chapter on Anesthesia for a pregnant patient undergoing non-obstetric surgery).

Additional factors include consideration for foetal analgesia as it has been shown that fetus also experiences pain and develops stress in response to the operative procedure. IV remifentanil infusion given to mother crosses placenta and can be used to titrate to fetal pain relief. Fetal analgesia and muscle relaxation can also be obtained by direct placental or IM injections of opioids and muscle relaxants. Tocolysis is an important consideration in fetal surgery since even minor interventions can result in placental separation or strong uterine contractions. Ritodrine and fenoterol are two commonly used β-agonists for tocolysis. Some people also advocate use of calcium channel blockers. Nitroglycerine has also been used as a tocolytic agent successfully.

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20 Traumatic Brain Injury

A Kulkarni, M Joshi

Case history
A 35-year male patient presents to the casualty with history of vehicular accident. He was conscious at the site of accident and became progressively drowsy during transport. At presentation in the casualty he has become unresponsive and has no purposeful activity. He has multiple bruises over the right side of chest and abdomen and he appears to be in respiratory distress. His pulse rate is 130/min, blood pressure 96/68 mm Hg and saturation is 93% on room air. How will you assess this patient?

Initial management of a trauma patient consists of rapid initial assessment, simultaneous institution of treatment along with necessary imaging. Protocolized management is extremely important as it prevents oversight and allows treatment to proceed in an orderly manner. The ATLS guidelines scheme is easy to follow:

ATLS scheme to assess trauma patient

I. Overview
   A. Perform visual scan of patient for obvious injuries
   B. Obtain history from prehospital personnel and patient
II. Primary survey (ascertain "ABCDEs")
   A. Airway maintenance with (cervical spine control)
      1. Look for chest wall movements, retraction, and nasal flaring
      2. Listen for breath sounds, stridor, and obstructed ventilation
      3. Feel for air movements
   B. Breathing (give supplemental oxygen)
      1. Determine whether ventilation is adequate
      2. Inspect chest to exclude open pneumothorax, sucking chest wound or flail segment
      3. Auscultate for bilateral chest sounds
      4. Provide assisted ventilation for ventilatory failure
   C. Circulation (establish venous access)
      1. Check peripheral pulses
      2. Grade shock according to vital signs
      3. Correct hypovolemia and obtain blood samples
   D. Disability (determine neurologic status)
      1. Evaluate central function (AVPU)
         A. Alert
         V. Responds to vocal stimulus
         P. Responds to painful stimulus
         U. Unresponsive
      2. Evaluate pupillary response to light
   E. Expose patient for complete examination
III. Resuscitation phase
IV. Secondary survey
V. Definitive care phase.

Check the airway, breathing, and adequacy of circulation. Check hemodynamic parameters such as blood pressure and whether peripheries appear warm and well-perfused. Evaluate for presence of neurological problems. Assess for presence of other injuries. Neurological dysfunction is assessed by checking level of consciousness, pupils and posture.

How is the CNS assessed? Does this patient need to be intubated? How will you intubate him?

CNS is assessed using the Glasgow Coma Score (GCS scale) or the AVPU system. Glasgow Coma Score is a tool for sequential assessment of the central nervous system. It has been well validated in the management of the trauma patients. Glasgow Coma score range is 3–15, if it is less than 8; the patient has

serious traumatic brain injury with raised intracranial pressure (ICP) more than 20 mm Hg. The GCS scale is given below:

Table 20.1 Glasgow coma scale (3–15)

Eye Opening (1–4)	
Spontaneous	4
To speech (not necessarily a request for eye opening)	3
To pain (stimulus should not be applied to face)	2
None	1
Best motor response (1–6)	
Obeys commands	6
Localizes purposeful movement towards the stimulus	5
Normal flexion (withdraws from painful stimulus)	4
Abnormal flexion (decorticate posture)	3
Extension (decerebrate posture)	2
No movement	1
Verbal response (1–5)	
Oriented (knows name, age)	5
Confused (still answers questions)	4
Inappropriate (recognizable words produced)	3
Incomprehensible sounds (grunts/groans, no actual words)	2
None	1
Severe head injury < 8 Moderate head injury 9–12 Mild head injury 13–15	

The **AVPU** is simple to carry out and offers a rapid method of assessment.

Table 20.2 APVU assessment method

Alert	Yes/No
Verbal—response to verbal command	Yes/No
Pain—response to painful stimulus	Yes/No
Unresponsive	Yes/No

Patients who are not alert and are not responding to command (P or worse) are equivalent to a GCS of around 8 which indicates a severe injury.

This patient appears to be maintaining his airway and is maintaining his saturation. However, the ATLS and Brain Trauma Foundation guidelines recommend that at GCS < 8; patients should be intubated to protect the airway. Therefore this patient should be intubated. Apart from a GCS < 8; rapidly worsening GCS, presence of hemorrhagic shock, need to transport patients for imaging or surgery should also prompt intubation and ventilation.

The most important consideration during intubation of the trauma patient is cervical spine protection. Patient is assumed to have cervical spine fracture at all times unless the spine is cleared radiologically. If excluding C-spine injury is not possible (due to unconsciousness, need for urgent surgical intervention for head or other injuries), cervical spine injury must always be assumed to be present and patient's neck protected with hard cervical collar.

Orotracheal intubation using direct laryngoscopy facilitated by muscle relaxant with manual in-line mobilization (performed by an assistant crouching at the head of table) is the method of choice for endotracheal intubation in trauma patients. Manual in-line traction is performed by grasping the mastoid processes and the front part of the collar is removed to allow adequate mouth opening. Excessive traction should not be applied as this can cause further damage to the cervical spine. The usual drugs for induction include an intravenous narcotic (fentanyl 100–150 µg, pethidine 50 mg or morphine 5 mg) followed by a slightly reduced dose of thiopentone 150–200 mg to ensure anesthesia, without causing hypotension. A reduced dose of propofol 90-100 mg can also be used for induction. Intubation technique will need to be changed in case of maxillofacial trauma or in presence of fracture of the mandible. In such circumstances, surgical airway; preferably surgical cricothyroidotomy (in desperate situations) or a surgical tracheostomy (without giving neck extension) may be carried out. During any procedure, cervical spine protection is of vital importance. The Ryle's tube should also be placed via the orogastric route in such patients. In anticipated difficult intubation, it is preferable to electively opt for a surgical airway rather than struggle with intubation; to avoid chances of exacerbating possible cervical spine injury.

Other important consideration during intubation is prevention of aspiration of gastric contents. All trauma patients must be assumed to be full stomach and a modified rapid sequence intubation is carried out. It has been shown that correctly applied cricoid pressure prevents gastric insufflation during mask ventilation as well as preventing aspiration of gastric contents. Cricoid pressure, when applied correctly, does not interfere with cervical spine protection. It is recommended that intravenous lignocaine may be administered before intubation to prevent rise in the intracranial pressure (ICP), however, there is no evidence to support this practice. Basilar skull fracture may be associated with CSF leak, cranial nerve injuries (vestibulocochlear, olfactory) and intracranial air. It is important to avoid nasal endotracheal tubes in these patients to avoid cerebral infection.

How will you assess and replace the blood loss in this patient?

The blood loss can be assessed from the vital signs of the patients. Blood loss up to 30% of total volume can be replaced

Table 20.3 Changes in vital signs with percent blood volume lost by hemorrhage

Vital signs	< 15%	15–30 %	30–40%	> 40%
Heart rate	< 100	> 120	> 120	> 140
SBP	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Capillary refill	Normal	Delayed	Delayed to absent	Absent
Respiratory rate	14–20	20–30	30–40	> 35
Mental status	Anxious	More anxious	Anxious and confused	Confused and lethargic

by crystalloids or colloids (in adults) provided further or ongoing losses are not suspected. Loss more than 30% needs to be replaced with uncrossed-matched O-Rh negative blood when urgent and then appropriately cross-matched blood once the patient's blood group is obtained.

What is primary brain injury? What are secondary injuries? Why are these important?

Primary brain injuries result immediately from the initial trauma. Primary injury occurs at the moment of trauma and includes contusion, damage to blood vessels and axonal shearing in which the axons are stretched and torn. The blood brain barrier and meninges may be damaged in the primary injury and neuronal death occurs. Secondary injury results in further brain damage from ischemia (so called because it is not due to initial trauma) caused by low blood pressure and cerebral swelling. This results from factors such as hypoxia, high carbon dioxide levels and venous congestion. Prolonged hypotension and hypoxia will increase mortality of a patient with severe head injury by 70–80%. A few examples of primary and secondary injuries are given below.

Table 20.4 Types of injuries

Primary brain injury	Secondary brain injury
Intracerebral hemorrhage	Edema
Subdural hemorrhage	Impaired metabolism
Subarachnoid hemorrhage	Altered cerebral blood flow
Epidural hemorrhage	Free radical formation
Cerebral contusion	Excitotoxicity
Cerebral laceration	
Axonal stretch injury	

What is excitotoxicity?

Head injury leads to releaze of glutamate from neuronal cells and which causes increased concentration of glutamate in the (CSF). This increased glutamate levels and glutamate receptor stimulation leads to increase in intracellular calcium ion. This further causes activation of protein kinase, phospholipase, nitric oxide synthase, proteases, and other enzymes. This leads to proteolysis, free radical formation, lipid peroxidation, deoxyribonucleic acid (DNA) damage, and ultimately neuronal death.

How is the head injury patient resuscitated?

Initially the following should be taken care of:

- Airway
- Ventilation
- Cardiovascular stability
- Lower ICP
- Maintain cerebral perfusion pressure (CPP = MAP – ICP). If required vasopressors should be added to increase MAP.

What is Cerebral Perfusion Pressure (CPP)? What are the factors affecting CPP?

The cranium, a rigid box, contains brain (80%), CSF (8%) and blood (12%). Increase in the amount of any of these will lead to increased intracranial pressure since the cranium can not expand. This in turn will affect the cerebral blood flow (CBF)

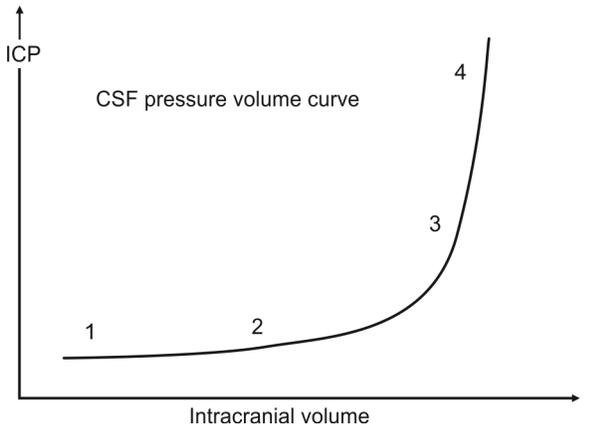

Fig. 20.1 Stage 1–2 are compensation phase, stage 3–4 are decompensated phase

as the cerebral perfusion pressure (CPP) is dependent on the mean arterial pressure (MAP) and the intracranial pressure (ICP). Normal ICP is about 5–13 mm Hg.

When there is an increase in the volume of either the brain or blood, the CSF is forced out into the spinal sac maintaining the ICP. With further increase in intracranial volume, venous

blood and more CSF is forced out of the skull. When these compensatory mechanisms fail, there is an abrupt increase in ICP. Increase in the ICP and or a fall in MAP or a combination of both will ultimately lead to a situation where CPP becomes very low; leading to progressive decrease in CBF ultimately causing brain death.

Assessment of CPP is vital and possible either by measurement of both ICP and MAP or by measuring MAP and making a reasonable estimate of ICP. During anesthesia therefore; if ICP is raised, a fall in blood pressure must be avoided or treated quickly by volume replacement or vasopressors. When CPP is inadequate the oxygen saturation of jugular venous blood falls (normal range 65–75%) because of increased oxygen extraction. Inadequate CPP (less than 70 mm Hg) has been shown to be a major factor in the poor outcome of patients with raised ICP.

Discuss in brief about Cerebral Blood Flow (CBF) and factors affecting CBF.

Cerebral Blood Flow: The normal cerebral blood flow is 45–50 mL/100 g/min ranging from 20 mL/100g/min in white matter to 70 mL/100 g/min in gray matter. There are two essential facts to understand about cerebral blood flow. Firstly in normal circumstances when the flow falls to less than 18–20 mL/100 g/min, physiological electrical function of the neuronal cell begins to fail. Secondly an increase or decrease in CBF will cause an increase or decrease in cerebral arterial blood volume because of arterial dilatation or constriction. Thus in a brain which is decompensated as a result of major intracranial pathology, increases or decreases in CBF will in turn lead to a significant rise or fall in ICP.

Autoregulation: CBF is maintained at a constant level in normal brain in the face of the usual fluctuations in blood pressure by the process of autoregulation. It is a poorly understood local vascular mechanism. Normally autoregulation maintains a constant blood flow between MAP 50 and 150 mm Hg. However, in traumatized or ischemic brain, or following vasodilator agents (volatile agents and sodium nitroprusside); CBF may become blood pressure dependent. Thus as arterial pressure rises; CBF will rise; causing an increase in cerebral volume. Similarly as pressure falls the CBF will also fall, reducing ICP, but also inducing an uncontrolled reduction in CBF. If CPP falls below the critical value of 70 mm Hg, the patient will have inadequate cerebral perfusion. Autoregulation will cause cerebral vasodilatation leading to a rise in brain volume. This in turn will lead to a further rise in ICP and induce the vicious circle described by the vasodilatation cascade resulting in cerebral ischemia.

Carbon dioxide causes cerebral vasodilatation. As the arterial tension of CO_2 increases, CBF increases and when CO_2 levels fall, vasoconstriction is induced. Hyperventilation can lead to a mean reduction in intracranial pressure of about 50% within 2–30 minutes, however, when $PaCO_2$ is < 25 mm Hg (3.3kPa) there is no further reduction in CBF. Hence there is no advantage in inducing further hypocapnea; as this will only shift the oxygen dissociation curve further to the left, making oxygen less available to the tissues. Acute hypocapnic vasoconstriction will only last for a relatively short time (5 hours). While hypocapnea is maintained, there is a gradual increase in CBF towards control values leading to cerebral hyperemia (over-perfusion) if the $PaCO_2$ is returned rapidly to normal levels. When long-term ventilation is required, only mild hypocapnea (34–38 mm Hg: 4.5–5.1 kPa) should be induced.

What is the triphasic response to head injury?

The triphasic response to head injury is sequential derangement of water balance. This may also be seen post-neurosurgery, particularly pituitary surgery patients. Patients display polyuria (due to diabetes insipidus with rising sodium), followed by Oliguria (due to SIADH and low sodium) followed lastly by a return to normalcy or a polyuric response again. This triphasic response may be due to hypothalamic dysfunction, later releaze of vasopressin from the damaged pituitary and, lastly due to exhaustion of vasopressin.

The patient was intubated in the casualty and mechanically ventilated. A CT scan of the head showed right temporo-parietal extradural hematoma. Patient is now posted for evacuation of extradural hematoma. Discuss the intraoperative management of this patient. How will you manage hemodynamics of this patient? What monitoring will you need?

Hemodynamic management: This patient is already intubated in the casualty. However, if the patient is intubated in the OT, it is important to remember that a combination of drugs given for intubation, institution of positive pressure ventilation and depression of the cardiovascular system with inability to compensate for a reduction in blood volume can often lead to hypotension. Hypotension (BP < 90 mm Hg is an independent predictor of poor outcome after head trauma. This should be prevented by preloading with saline or by rapid fluid infusion after induction of anesthesia. It is best to ensure adequate fluid/blood resuscitation before induction; however, this may not always be possible. Small doses of vasopressor may be given to support blood pressure till fluid can be infused. It is important to avoid hypotension because this will lead to reduced blood flow through the

brain, reducing oxygenation of the brain and cause ischemia leading to major neurological damage (stroke, paralysis, death). Also autoregulation will lead to arterial dilatation in an attempt to reduce the resistance to increased flow. This will lead to further increase in brain volume and intracranial pressure, making the situation worse. If the MAP is low such that the CPP is < 70 mm Hg, vasopressor (noradrenaline) infusion should be started and titrated to maintain CPP of at least 70 mm Hg. On the other hand, hypertension might be undesirable if autoregulation is impaired, as it will lead to either increased CBF or worsen bleeding.

Monitoring: Monitoring should include electrocardiography, pulse oximetry, temperature and end-tidal CO_2 monitoring. Continuous invasive arterial blood pressure monitoring is useful for monitoring CPP, titrating vasopressor infusion and fluid therapy. A central venous access will help in assessing volume status as well as allow infusion of vasopressor. Surgery should not be delayed by taking time to institute invasive monitoring. ICP monitoring is extremely useful and an intraventricular drain placed during the surgery can be used postoperatively for monitoring as well as CSF drainage as a temporizing measure to decrease ICP. Urine output needs to be monitored as a surrogate of perfusion and since mannitol is used to control ICP.

Maintenance of anesthesia: Anesthesia can be maintained using narcotics (i.v. fentanyl 150 mcg or morphine 10 mg given slowly), vecuronium 10 mg and the patient ventilated with inhalational agents (isoflurane as the first choice) in oxygen enriched air. Most intravenous drugs decreases metabolism and maintains coupling excluding ketamine. While using inhalational agents it is important to remember that high inspired concentrations of inhalational agents (>1MAC) will cause cerebral vasodilatation with increased cerebral blood volume, cerebral edema and consequent rise in ICP. It decreases metabolism and changes metabolic coupling. At the same time, inhalational agents will cause a fall in blood pressure. This combination of raised ICP and low BP is extremely dangerous as it leads to a reduction in CPP, worsening neuronal damage and may lead to adverse outcome. Nitrous oxide is contraindicated in patients with raised ICP.

Ventilatory strategy: Aim of ventilation in a head injured patient is to provide adequate oxygenation to avoid hypoxemia and thus prevent sudden increase in CBF and ICP. Intermittent positive pressure ventilation is given with aim to achieve low normal arterial CO_2 ($PaCO_2$ 35 mm Hg) which will help to reduce cerebral swelling and hence intracranial pressure. Severe hyperventilation is not recommended. Moderate hyperventilation to reduce intracranial hypotension is likely to work only for a short period of time (< 5 hours) and it just gives time before more definitive management can be undertaken.

How will you manage fluid therapy in a head injured patient?

The brain is surrounded by a membrane separating it from the vascular space (the blood-brain barrier), which allows only water to pass through it. It is necessary therefore not to give hypotonic fluids such as dextrose solutions in water (5% Dextrose) as dextrose metabolism will leave just the water or a very dilute saline solution. This will be taken up by the brain leading to further cerebral edema and increase in ICP. Normal saline (0.9%) has a similar concentration of sodium and therefore is the fluid of choice. Colloids, blood and blood products can be given to treat hypovolemia due by major blood loss.

What are the predictors of outcome in head injury patients?

- Age
- CT scan diagnosis
- GCS score at admission
- Pupillary activity
- Hypotension

If he had liver laceration with significant intra-abdominal bleeding as well, what should be done first evacuation of cranial blood or laparotomy?

Both would be evacuated at the same sitting, however evacuation of cranial blood is the foremost priority as longer we wait, higher the chances of secondary injury due to increasing intracranial volume of blood, higher ICP, thus lower CPP and thus cerebral hypoperfusion, ischemia and neuronal damage. Then laparotomy with suturing of liver laceration can be carried out. The timing of surgery for non-life threatening orthopaedic surgery is controversial. Hypotension in the first 24–48 hours of head injury is associated with a worse outcome. If surgery is necessary within these hours, then it should proceed under ICP control with careful maintenance of CPP, oxygenation, $PaCO_2$ and avoidance of anemia.

What are the physiological effects of raised ICP? Discuss management of Intracranial Hypertension (ICH).

The manifestations of physiological effects of raised ICP are hypertension, bradycardia, wide pulse pressure—the Cushing's Triad and ventricular dysrrthymias.

ICH must be aggressively treated with immediate interventions aimed at lowering ICP and increasing CPP. Sustained ICP ≥ 20 mm

Hg for more than 10 minutes is associated with poor outcomes and immediate treatment is necessary.

Airway management: Patients with a GCS ≤ 8 require intubation for airway protection. Use of therapeutic hyperventilation reduces ICP through vasoconstriction. However, Hyperventilation is not a good long-term strategy. It is recommended to maintain the $PaCO_2$ at the low end of normal while more definitive strategies for reducing ICP are attempted.

Maintain CPP: This is done by reducing the ICP and ensuring adequate MAP to maintain CPP > 60–70 mm Hg with fluid resuscitation and vasopressor support.

Vasodilators such as diazoxide, hydralazine, nitroglycerine, or nitroprusside should not be used.

Osmotic diuresis: Mannitol is given as a 0.5–1g/kg bolus and repeated every 6–8 hours to maintain serum osmolarity at > 310 mOsm/L. Mannitol results in an osmotic diuresis. The altered osmolar gradient facilitates fluid shifts and reduces cerebral edema. Furosemide increases intravascular oncotic pressure via hypoosmolar diuresis, which reduces cerebral edema and CSF production. These agents should be used with caution because hypovolemia and hypotension may reduce CPP. Urine output should be matched with crystalloid replacement to maintain intravascular volume.

Hypertonic saline: Hyponatremia indicates worse outcomes, and serum sodium should be maintained at the upper limits of normal. Sodium chloride is the fluid of choice for resuscitation. Hypertonic saline solution increases serum osmolarity, which both expands intravascular volume improving CBF and facilitates removal of fluid in cerebral edema. It also has an effect on microcirculation.

Hypothermia: Hyperthermia increases ICP and should be prevented. Hypothermia reduces ICP and may mediate apoptosis, coagulopathy and electrolyte abnormalities. Shivering may occur, which increases ICP. Though animal data is interesting; the current evidence in humans does not support use of therapeutic hypothermia.

Corticosteroids: Previously steroids were thought to be of help in reducing cerebral edema but recent studies have shown increased mortality in patients receiving steroids when compared to placebo. Steroids also increase blood glucose levels, which can adversely affect the injured brain. So according to current evidence there is no role of steroids in the head injured patient and should not be used.

Glycemic control: Hyperglycemia is likely to lead to poor outcomes in severely head-injured patients and if present should be aggressively treated.

Postural drainage: 15–30 degree head elevation and neck maintained in a midline position facilitate venous return. Jugular central venous lines and improperly positioned cervical collars may obstruct venous drainage and should be avoided.

Sedation and analgesia: Pain and agitation increase ICP and should be avoided and treated if present. Benzodiazepines and propofol have the added benefit of increasing the seizure threshold. Excessive sedation and analgesia may mask examination findings and prolong the course of mechanical ventilation. Vasodilators such as diazoxide, hydralazine, nitroglycerine, or nitroprusside may be catastrophic in this situation.

Neuromuscular blockade: Therapeutic paralysis reduces ICP and prevents shivering if induced hypothermia is attempted. However, it may mask seizure activity, negates the physical examination; potentially delaying early signs of deterioration, prolongs ventilation, and increases the risk for pressure ulcers and venous thromboembolism. Deep sedation is safer while providing similar benefits.

Barbiturate coma: They may be considered in patients with refractory intracranial hypertension or in those with uncontrolled seizures. Continuous bedside EEG monitoring is necessary to monitor adequate burst suppression with barbiturate infusions.

CSF drainage: Removal of CSF through an intraventricular catheter reduces ICP. Drainage should be continued to maintain the ICP between 5 and 15 mm Hg; however, the chances of infection are high.

Decompression Craniectomy: Bone and brain removal are extremely effective in reducing ICP. Craniectomy greatly facilitates ICP management and may improve outcomes and is recommended if other therapies are not effective. Wide Decompressive craniectomy with duraplasty is an effective procedure for decreasing raised ICP in patients with intraparenchymal contusions. A recent meta-analysis showed that ICP was lower and CPP higher in patients who had undergone decompressive craniectomy. However, studies so far have failed to show an improvement in long-term neurological outcomes.

Describe surgical management for various lesions in head injured patient.

Subdural hematoma (SDH): Subdural hematoma with thickness more 10 mm or midline shift more than 5 mm on CT scan should be evacuated irrespective of GCS score. All patients with acute SDH; who are comatose (GCS less than

9) should have intracranial pressure monitoring instituted. A comatose patient (GCS less than 9) with hematoma less than 10 mm thick or midline shift < 5 mm should undergo surgery if GCS Score decreases by 2 points from admission and patient presents with asymmetric or fixed pupils, or if the ICP is more than 20 mm Hg. A majority of patients with subdural hematoma may have concomitant intraparenchymal lesions. For high volume lesions (more than 50 cms^3), surgery is the only option. Low volume lesions (less than 25 cm^3) are not operated.

Extradural hematomas (EDH): Surgical management for epidural hematomas (more than 30 cm^3) should be surgically evacuated irrespective of GCS status. An EDH of volume less than 30 cm^3 and thickness less than15 mm with less than 5mm midline shift with GCS score more than 8; without neurological deficit; can be managed conservatively with CT scanning and neurologic monitoring. Patients with GCS less than 9 with anisocoria should be operated immediately.

Traumatic parenchymal lesions: Patients with traumatic parenchymal lesions, signs of neurological deterioration, medically refractory raised ICP, signs of mass effect on CT scan should be treated operatively. Patients with GCS score of 6–8 with frontal and temporal contusions greater than 20 cm^3 with midline shift of a least 5 mm and cisternal compression on CT scan and patients with lesions greater than 50 cm^3 should be treated operatively.

Patients with parenchymal mass lesions, who do not show evidence of neurologic compromise, have controlled ICP, and no signs of mass effect on CT scan can be managed non-operatively with intense monitoring and serial imaging. Bi-frontal decompressive craniotomy is recommended within 48 hours of traumatic injury in patients with severe diffuse axonal injury with intracranial hypertension which is refractory to usual medical management. Decompressive procedures including subtemporal decompression, hemispheric decompressive craniectomy are treatment options for patients with refractory intracranial hypertension and diffuse parenchymal injury with clinical and radiological evidence of transtentorial herniation.

Posterior fossa lesions: Patients with mass effect on CT scan or neurological dysfunction or deterioration due to the lesion should undergo operative intervention which should be done as early as possible since they are likely to deteriorate rapidly. Suboccipital craniectomy is the recommended method for evacuation of hematoma. Patients with no significant mass effect on CT scan and signs of neurological dysfunction should be managed with close observation. Long-term anticonvulsants have no role in head trauma but some patients may have seizures in first week which responds to phenytoin. The BTF guidelines recommend short term prophylaxis with phenytoin for head-injured patients.

Discuss postoperative care of a head injured patient. How would you give prophylaxis against DVT? Should this patient be given antiepileptic and for how long?

If the patient has undergone simply an evacuation of extradural hematoma, he can be extubated once wide awake. If additional injuries such as diffuse axonal injuries are present, then ventilation for a longer period may be required. It is important to remember that peak brain edema can take 24–72 hours to occur. The care in PACU aims at (1) prevention of secondary injuries due to physiological factors which may cause cerebral edema, (2) observation to detect any deterioration, and (3) providing adequate analgesia. Any further insult can be detected by changes in level of consciousness, development of focal neurodeficit or unequal pupils.

Use of unfractionated or low molecular weight heparin for postoperative DVT prophylaxis in patients with intracranial hemorrhage is not advocated. Mechanical prophylaxis using graduated stockings or sequential calf compression devices is generally used in the postoperative period.

Prophylactic treatment with phenytoin, beginning with an intravenous (IV) loading dose, should be initiated as soon as possible after injury to decrease the risk of post-traumatic seizures and then stopped after 7 days.

Summary [as per the Brain Trauma Recommendations 3rd Edition (2007)]

- Initial Management
- ABC physiologic resuscitation
- Sedation and neuromuscular blockade
- Avoid BP < 90 mm Hg
- Avoid O_2 saturation < 90%, PAO_2 < 60 mm Hg
- ICP Monitoring appropriate with severe Head Injury and CT abnormalities
- Treat ICP > 20–25 mm Hg
- CPP maintained >70 mm Hg
- Hyperventilation
- Brief $PaCO_2$ < 25 mm Hg to control ICP
- Avoid prophylactic $PaCO_2$ < 25 mm Hg
- Mannitol is effective for control ICP. 0.25–1 gm/Kg if volume resuscitated. Avoid hypotension.
- Mannitol boluses are better than infusion. Aim to keep serum Osm < 320 mosm.

- Barbiturates: High-dose may be considered in salvageable severe head injury with refractory ICP.
- Steroids are not recommended.
- Nutrition: Replace metabolic requirements with enteral or parenteral feedings.
- Antiseizure prophylaxis: Prophylaxis of late seizures not recommended. Prophylaxis of early seizures with phenytoin and carbamezipine is recommended.

Conclusion

Initial assessment and resuscitation is vital in head-injured patient as it will prevent secondary injuries and adverse outcomes. Anesthetic management should be based on the understanding of physiological and pharmacological principles. Prompt and appropriate management of ICH will improve the outcome.

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21 Managing Difficult Airway

V Patil, J Doctor

Induction of general anesthesia followed by direct laryngoscopy and oral intubation can be difficult, if not impossible, in several situations. To determine the optimal intubation technique, the anesthesiologist must elicit an airway history and carefully examine the patient's head and neck. Any available prior anesthesia records should be reviewed for previous problems in airway management. If a facial deformity is severe enough to preclude a good mask seal, positive-pressure ventilation may be impossible. Furthermore, patients with hypopharyngeal disease are more dependent on muscle tone (while awake) to maintain airway patency. These two groups of patients should not be allowed to become apneic for any reason—including induction of anesthesia, sedation, or muscle paralysis—until their airway is secured. In other patients in spite of thorough airway evaluation one has to remember surprises can crop up and anesthetist should be ready with options to manage difficult airway at any time. In short, easy to intubate on history taking and clinical examination does not necessarily mean easy intubation.

What are some of the common screening tests employed to assess the airway?

Visual inspection and examination: Face, teeth, incisors, nose, palate, tongue, mandible, tonsils, oral cavity, neck.

Single Parameter Tests

1. *Assessment of TMJ function*
   a. Interincisor gap/mouth opening—> 5 cm or normally >3 fingers
   b. Jaw protrusion
   c. Upper lip bite test:
      - Class I lower incisors can bite upper lip above the vermilion line
      - Class II lower incisors can bite upper lip below vermilion line
      - Class III lower incisors cannot bite upper lip.
   d. Movement of condyle of mandible in front of the tragus
2. ***Assessment of mandibular space and floor of mouth for tongue displacement***
   a. Thyromental distance—Normal > 6.5 cm (4 fingers)
   b. Hyomental distance—Normal > 6 cm (3 fingers)
   c. Mandibular angle to mental symphysis distance or horizontal length of mandible—at least 9 cm.
3. *Assessment of adequacy of the oropharynx for laryngoscopy and intubation*
   a. Mallampati classification—(Samsoon and Young modification) pharynx to tongue ratio. Examiner sits opposite the patient at eye level and the patient opens the mouth as wide as possible and protrudes the tongue without phonating.
      - Grade 1—Soft palate, uvula, tonsillar fauces and posterior pharyngeal wall visible
      - Grade 2—Soft palate, uvula and tonsillar fauces visible
      - Grade 3—Soft palate and base of uvula seen
      - Grade 4—Only hard palate visualized
      - Grade 0—Epiglottis seen.
4. *Assessment of cervical and atlanto occipital joint function:* Optimal position for direct laryngoscopy involves flexion of cervical spine and extension of AO joint (Sniffing position aligns oropharyngeal and laryngeal axes to a favorable line of sight). If the patient can touch his manubrium sterni with his chin this assures neck flexion of 25–30 degrees. The patient is now asked to open his mouth wide such that the occlusal surface of the upper incisor teeth is parallel to the ground. On extending the

AO joint the angle created by movement of the occlusal surfaces is estimated. It should be at least 80–85 degrees.

5. *Thyroid to floor of mouth distance:* It indicates the position of the larynx in the neck. It is normally placed if the patient can place 2 fingers between the top of the thyroid cartilage and the floor of the mouth.
6. *Sternomental distance:* This is measured with the head in full extension and the mouth closed. Normal distance is > 12.5 cm.
7. *Quality of glottic view during laryngoscopy:* Not usually done in an OPD setting. Useful in cases of recorded previous difficult intubation.
   a. Indirect mirror laryngoscopy or Hopkins examination.
   b. Direct laryngoscopy: Done under topical anesthesia (awake look) or with the patient under inhalational or intravenous induction but not apnoeic or paralysed or patient completely under anaesthesia and paralysed. Cook's modification of Cormack and Lehane's grading.
      - Grade I – Visualization of entire vocal cords.
      - Grade IIa – Visualization of posterior part of vocal cords
      - Grade IIb – Visualization of arytenoids only
      - Grade IIIa – Epiglottis liftable
      - Grade IIIb – Epiglottis adherent or only tip visible
      - Grade IV – No glottic structures seen.
   - POGO Scoring – Discussed in detail later

Group Indices: Multiple Parameter Tests

1. *Wilsons scoring system:* Based on weight of patient, Head and neck movement, inter-incisor gap presence of buck teeth and receding mandible. It has a total score of 10. Score < 5 = Easy laryngoscopy, 6–7 moderate difficulty, 8–10 severe difficulty.
2. *Benumof's 11 parameter analysis:* (first 4 parameters for teeth) Length of upper incisors, Inter-inscisor gap, involuntary buck teeth override, voluntary protrusion of mandibular teeth in relation to maxillary teeth, (next 2 parameters for intraoral structures) MPC, Palatal configuration and narrowness, (next 2 for mandibular space) Thyromental distance, compliance of mandibular space, (last 3 parameters for neck) neck length, neck thickness, Head and neck movement.

Radiological Assessment

1. X-ray neck cervical spine (AP/Lat)
   a. Atlanto-occipital gap < 5 mm
   b. C1-C2 gap < 5 mm
   c. Airway compression/Deviation
   d. Mandibular length/Depth ratio.
2. CT Scan—if available
3. MRI—If available.

Advanced Assessment Tests

1. Flow volume loops—if available
2. Acoustic Response measurement and USG.

Assessment for difficult bag mask ventilation: BONES

1. Bearded individuals
2. Obese individuals
3. No teeth
4. Elderly
5. Snorers.

Assessment for Difficult Supraglottic Airway Device Placement: RODS

1. Restricted mouth opening
2. Obstructed upper airway
3. Disrupted upper airway following trauma and burns
4. Stiff lungs (poor lung or thoracic compliance).

Assessment for Difficult Surgical Airway: BANG

1. Bleeding tendency
2. Agitated patient
3. Neck scarring or flexion deformity
4. Growth or Vascular abnormalities in the region of the surgical airway.

In addition to the above if the patient is to be posted for emergency surgery the risk of aspiration along with a difficult airway should also be borne in mind.

Why do we get unanticipated difficult intubations? Why do predictive tests fail?

Four terms provide the information needed to analyze the usefulness of a predictive test.

Sensitivity: Test sensitivity is a measure of whether it identifies correctly the difficult patients as being difficult. A test sensitivity of 80% indicates that 80% of the difficult patients will be identified correctly as difficult, and 20% will be missed and classified as not—difficult or normal. A test sensitivity of 100% is ideal.

Specificity: Specificity of a test identifies that a normal patient is normal. A specificity of 80% indicates that 80% of normal patients will be correctly identified as normal, but 20% of normal patients will be identified incorrectly as difficult. A test specificity of 100% is ideal.

Positive predictive value (PPV): The positive predictive value is the percentage that is true difficult intubations out of all those predicted by the test to be difficult.

Likelihood ratio: The likelihood ratio (LR) is an extremely useful term and can be calculated within seconds using only the sensitivity and specificity. The LR is the chance of a positive test if the person is difficult, divided by the chance of a positive test if the patient is normal.

From the following table it is clear why prediction tests fail.

Predictive ability of common tests

Test	Sensitivity (%)	Specificity (%)	PPV (%)
Mallampati (original)	42–60	81–89	4–21
Mallampati (modified)	65–81	66–82	8–9
Thyromental	65–91	81–82	8–15
Sternomental	82	89	27
Wilson score	42–55	86–92	6–9
Mouth opening	26–47	94–95	7–25
Jaw protrusion	17–26	95–96	5–21

Enumerate basic principles of difficult airway management.

1. Preparation of team (anesthetists, surgeon, nurse, technician/OT attendant): Equipments, right frame of mind, well-defined primary and back-up plans.
2. Good airway preparation with antisialogogues and local anesthetics.
3. Obtaining maximum cooperation from patient.
4. Maintenance of spontaneous breathing till airway is secured.

What is Intubation difficulty scale?

The intubation difficulty scale (IDS) was proposed in 1997 and incorporates seven variables (given below) to describe the ease or difficulty of a particular intubation by direct laryngoscopy. The sum of each variable produces the IDS score allowing a gradation of intubation from easy through to impossible, rather than a simple difficult/not difficult definition. The first three (N1-3) variables have no upper limit; the fourth variable (N4) is glottic exposure according to the Cormack and Lehane four grades minus one. A Grade 1 view is awarded zero points and a Grade 4 view three points. Successful blind nasotracheal intubation scores 0. The glottic exposure is evaluated during the first attempt by the first operator. The last three variables (N5-7) are scored either 0 or 1. An IDS of 0 indicates intubation without difficulty and there is no upper limit. The cut-off to define difficult intubation is arbitrary but a value of five has been used in more than one study. In a prospective study of 1171 patients an IDS of 0 was found in 55% patients and IDS > 5 occurring in 8% patients. It may be very useful in communicating the total intubating difficulty for a given patient to the next care-giver. It should be noted that a particular score may be obtained through differing problems and any score, on its own, is not diagnostic. It is ideal to transmit information in case of difficult/failed intubation and is only of practical use if the score for each of the seven variables is recorded and transmitted to the next anesthetist.

N1- Number of attempts >1
N2- Number of operators >1
N3- Number of alternative techniques
N4-Cormack Lehane grade-1
N5- Lifting force required (normal 0 or increased 1)
N6- Laryngeal pressure (not applied 0 or applied 1)
N7- Vocal cord mobility (abduction 0 or adduction 1)

What are predictors of difficult mask ventilation?

Predictors include:

- BMI > 30 kg/m2
- Beard
- Age > 57
- Snoring
- Limited jaw protrusion
- Abnormal neck anatomy
- Sleep apnea
- Thyromental distance < 6 cm

- **O** – obese
- **B** – beard
- **E** – elderly
- **S** – sleep apnea, secretions
- **E** – edentulous.

What is POGO score?

The percentage of glottic opening (POGO) score represents the portion of the glottis visualized. Glottis is defined anteriorly by the anterior commissure and posteriorly by the interarytenoid notch. The score ranges from 0% when none of the glottis is seen to 100% when the entire glottis including the anterior commissure is seen. The POGO score does not differentiate

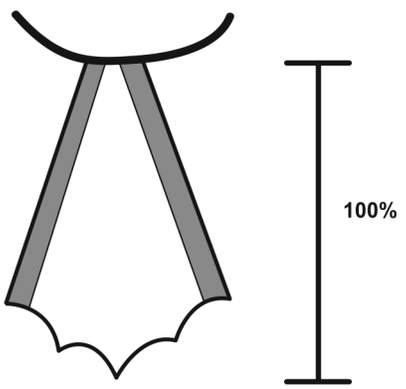


Fig. 21.1 POGO score

between visualization of the epiglottis and the tongue. In each case the POGO score would be 0. The POGO score is a simple, easy way to categorize laryngeal view. Studies have found that it has better inter-physician reliability than Cormack-Lehane grading.

What is RAMP position and where is it useful?

It is also called as Head-Elevated Laryngoscopy Position (HELP) position. If the patient is obese, it may be necessary to make a "ramp" of pillows or blankets under her head, shoulders and upper back to achieve the proper airway angle and a sniffing position. Dr Richard Levitan, inventor of the Airway Cam, calls this the head-elevated laryngoscopy position (HELP), originally introduced by Chevalier Jackson at the beginning of the 20th century. The shoulders and head should be raised enough so that an imaginary horizontal line drawn through the ear canal intersects the xiphoid process. Rescue ventilation techniques (oral airway/bag and mask), are facilitated by the HELP position. Since head and neck are elevated above the chest and abdomen, the airway is more isolated and easier to work with. Further, the weight of the abdomen falls away from the diaphragm allowing better ventilation. Positioning the morbidly obese patient has shown that this position improves the laryngoscopy view and also eases ventilation and improves oxygenation allowing more apneic period. Stacking with blankets can create the HELP position, but this may cause head and neck to be unstable. A pre-cut foam positioner designed to quickly achieve the HELP position is commercially available and preferred.

What are the contents of airway cart/difficult intubation trolley?

Anesthetists should be ready to deal with difficulties in airway management at any time. The correct equipment must be immediately available. This should include:

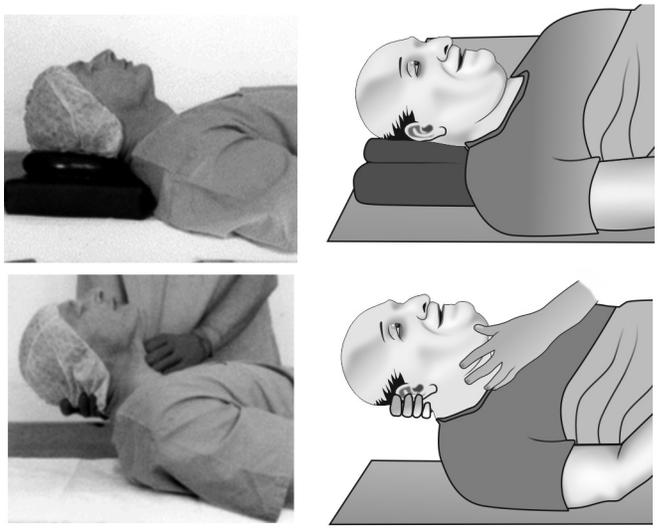


Fig. 21.2 Ramp position

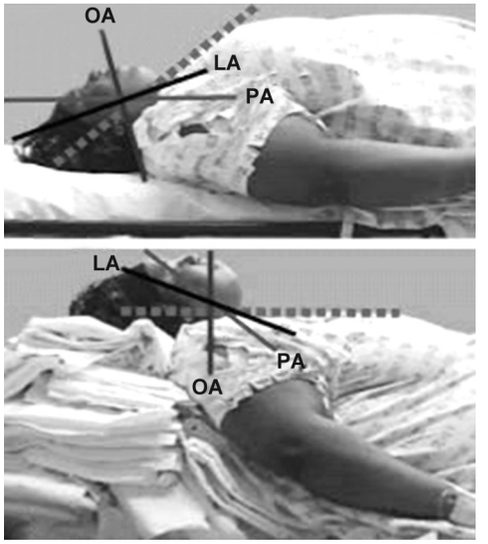


Fig. 21.3 Alignment of airway axes

Suggested portable storage unit, ASA 2003

Alternative laryngoscope blade (McCoy, Miller, Bullard)
Endotracheal tubes of assorted sizes
Endotracheal tube guide (semi-rigid stylets, tube changer, lighted stylet, Magill forceps)
Laryngeal mask airways of assorted sizes and types
Flexible fibreoptic intubation equipment
Retrograde intubation equipment
Noninvasive ventilation devices (combitube, hollow jet ventilation stylet, transtracheal jet ventilation)
Emergency invasive airway access (cricothyrotomy equipments)
Exhales carbon dioxide detectors.
Also one should have all sizes of oral and nasal airways, Reliable suction equipment, and of course a trained assistant.

Case Scenarios

Case No. 1. A 32-year-old, 58 kg male patient diagnosed to have ankylosing spondylitis was referred for the spine surgery. He gives history of gradual onset, progressive painless deformity since 4 years, resulting in thoraco-lumbar kyphosis. He cannot look up or straighten himself while walking. He cannot lie down supine, and needs two to three pillows, beneath his head to support it while sleeping.

Ankylosing spondylitis leads to fibrosis, ossification, and ankylosis along the spinal column and sacroiliac articulations. Cervical column and atlanto-occipital articulation mobility are reduced and in severe cases the cervical vertebrae become fixed in a flexed position. This portion of the spine is also the most susceptible to fracture, particularly in hyperextension, and can damage the cervical spinal cord during maneuvers to

manage the airway. Patients with this condition may also have temporomandibular joint involvement, further complicating airway management. Preferably they should be managed along the awake limb of difficult airway algorithm.

How will you prepare a patient for awake intubation? What premedication will you use?

Adequate preparation is must for success. An antisialogogue, administered in sufficient time to work, is imperative. IV scopolamine, atropine or glycopyrrolate may be used. We routinely use glycopyrrolate because it is non-sedative and results in fewer hemodynamic and central side-effects than atropine. The nasal passage (if the nasal route is chosen) and the oropharynx, cords, and trachea should be anesthetized with local anaesthetic agent, the most commonly used agent being lignocaine. The appropriate level of analgesia and sedation is challenging because overdose of medication resulting in the loss of spontaneous ventilation, can be disastrous in a difficult airway, and insufficient medication may result in patient discomfort, pain, coughing, tachycardia, making the tracheal intubation technically more difficult to even impossible. Use titrated amount of short acting sedatives and opioids like midazolam and fentanyl to a Ramsay scale 2–3. Adequate topical anesthesia in conjunction with light sedation usually produces very satisfactory intubating conditions. The nasal passage should be anesthetized with 2% lignocaine jelly in case nasal route is selected. Vasoconstriction of nasal mucosa is very important and can be accomplished by using phenylephrine/adrenaline soaked pellets or nasal decongestants like metazolines.

How would you anesthetize the airway for awake intubation?

Translaryngeal block: Placement of local anesthetic within the larynx can provide anesthesia of the larynx and trachea below the vocal cords. Sensory supply to larynx below vocal cords is by the recurrent laryngeal nerve, and above vocal cords is by superior laryngeal nerve both being branches of vagus nerve. This block should be avoided when patient coughing is undesirable (e.g. raised ICP/IOP).

To perform the block, identify the cricothyroid membrane, which can be very easily identified by running the finger in the midline of neck in extension. Cricothyroid membrane will be felt as a soft depression just below Adams apple. Place a 22 gauge needle through this membrane (using smaller gauge needle prevents rapid injection of LA in trachea. Needle can get displaced with coughing of patient when LA is being injected slowly and prevent instillation of full volume of LA in trachea when finer needles are used. This may lead to unsatisfactory anesthesia). Feeling of sudden give way marks entry into trachea. Aspirate air from the trachea to confirm the position. Inject 3–5 mL of local anesthetic (4% lignocaine) rapidly and withdraw the needle. The patient will immediately cough and spread the local anesthetic up and down the larynx and trachea.

Superior Laryngeal Nerve Block: The superior laryngeal nerve is a branch of the vagus nerve. It travels just inferior to the greater cornu of the hyoid bone and divides into internal and external branches to provide sensation to the anterior and posterior epiglottis and laryngeal mucosa to the level of the vocal cords.

To perform the block, gently displace the hyoid bone toward the side to be blocked. Place a 24/25 gauge needle just inferior to the greater cornu of the hyoid bone and superior to the thyroid cartilage and inject 2–3 mL of local anesthetic. 1% lignocaine may be used. Repeat the procedure on the other side.

Nebulisation: Same can be achieved by using nebulisation with 4% lignocaine before procedure.

While using all these techniques one must take care to calculate total dose of LA used and restrict amount to safe maximum dose (3–5 mg/kg for lignocaine)

What are the different ways of securing the airway in an awake patient?

Blind nasal intubation: This technique is not entirely blind as name suggests but it is guided by breath sounds. Here a nasal endotracheal tube is gently passed through the nose towards the larynx. One should continuously hear breath sounds and guide the tube in the direction of the loudest breath sounds by moving the patient's head until the larynx is entered. This technique can be employed for patients with trismus where mouth opening is limited but the oropharyngeal and laryngeal anatomy is normal. This technique requires a great deal of skill and expertise of operator and cooperation of patient and is not feasible if the head and neck cannot be moved.

Retrograde intubation is a technique first described in Nigeria for intubation of patients with cancrum oris. A soft wire or epidural catheter is passed through the cricothyroid or cricotracheal membrane in a cephalad direction (towards the head) until it comes out of the nose or mouth. (In some patients it is necessary to grasp the catheter in the mouth using a pair of Magills forceps). An endotracheal tube is then inserted over this wire into the trachea from either the nasal or oral route. Ensure oxygenation is maintained throughout. During the technique, if a nasal intubation is required but the

wire comes out of the mouth, place another catheter through the nose, pull it out through the mouth, tie it to the wire, and then pull the wire up through the nose. Due to discrepancy between thin wire/catheter and large endotracheal tube many times it becomes difficult to guide tube into trachea. To overcome this problem, some researchers advocate doing this in two stages—use of bougie or airway exchange catheter over wire, and then guiding tube over bougie or airway exchange catheter.

Fibreoptic intubation: Fibreoptic intubation is simple in principle, but requires practice to learn. If one has to master the technique one has to use the scope frequently. Familiarity with the scope features and practice in manipulation should be obtained before clinical use to decrease damages of costly equipment. Tip control is quite simple, up/down motion is adjusted with the lever and side to side motion is accomplished by clockwise/counter-clockwise rotation of the entire scope-handle and shaft. Trying to achieve this rotation by turning the handle alone (without simultaneous rotation of the shaft with the other hand) produces torque which can damage the scopes especially if it is passing through a snug fitting endotracheal tube and should be avoided at any cost as this twisting is one of the most common causes of scope damage. Nasal approach is usually the preferred route compared to the oral one, as it is easier to navigate the scope.

The light wand: This consists of a handle, a wand and a stylet. On the handle, a locking clamp accepts and secures a standard ETT connector. The distal end of the wand has a bright light bulb. To accommodate ETT of different lengths, the length of the wand can be adjusted by sliding the connector along the handle. The stiff, retractable stylet can be used to shape wand in a "hockey stick" shape. ETT-light wand and stylet are used together as one unit. When the tip of the ETT is in the glottic opening, a hockey stick bend allows the maximum light intensity to point to the surface of the skin. In a sniffing position, the epiglottis is almost in contact with the posterior pharyngeal wall, making it difficult for this assembly to pass through vocal cords. Therefore, it is recommended that the patient's head and neck be placed in a neutral or relatively extended position. In patients where neck extension cannot be given, the epiglottis can be lifted off the posterior pharyngeal wall with a simple jaw lift maneuver.

LMA/ILMA: Introduced in1981, it is a supraglottic device that shifts the mask seal from the face to a supraglottic location. There is plenty of literature describing successful insertion and use of this device by even non-medical personnel. ILMA was developed from MRI studies in non-obese patients for airway management, and it is easier to insert because of its curvature and handle and can be manipulated to achieve optimum seal.

Reported success rate of blind tracheal intubation via ILMA varies between 89.51–100%. Various methods used for intubation through ILMA are:

1. Specialized flexometallic tubes that are provided with ILMA.
2. Standard short bevel flexometallic tubes (armoured tubes).
3. Standard PVC endotracheal tubes.
4. Gum elastic bougie through ILMA.
5. Airway exchanger – Cook or Patils.
6. Fiberoptic bronchoscope through ILMA.

Awake tracheostomy performed under local anaesthesia is the best solution when a patient is impossible to intubate, and regional anesthesia is not a practical option. This is a straightforward technique, except in children, when sedation with ketamine can be used to facilitate this approach.

A 17-year-old girl presents for emergency drainage of a submandibular abscess. How will you manage her airway?

Induction of GA followed by direct laryngoscopy and oral intubation is dangerous in these situations. To determine the optimal intubation technique, elicit a proper history and carefully examine patients head and neck. If hypopharynx is involved to the level of hyoid bone (evident from edema and redness), intubation will be very difficult. Also look for signs of airway obstruction like stridor/chest retraction and signs of hypoxia like agitation, restlessness, anxiety, lethargy etc. Aspiration pneumonia is likely if the patient has eaten recently or if pus is draining in mouth and in both the cases abolition of reflexes should be avoided.

In the above case, on physical examination, there is extensive facial edema, with limited mouth opening due to pain, and frank pus is observed in mouth.

In this case awake intubation is the only option. The blind nasal approach to awake intubation is a relative contraindication here, as this patient will have extensive intraoral edema, a distorted anatomy that can lead to more trauma and worsen situation further. Thus FOB-guided intubation is the technique of choice.

If the patient has adequate NBM status and there is no rupture of abscess cavity intraorally, one can try anesthetizing the patient maintaining spontaneous breathing and performing direct laryngoscopy with an attempt to visualize glottis and intubation. Regardless of technique, one has to be ready for a tracheostomy with an experienced surgeon in OT. Give antisialogogues, such as glycopyrrolate injection

as a premedication to decrease secretions. Sedative premedication should be avoided if possible or given very carefully in titrated doses. Explain the need for awake intubation with stepwise approach to allay patients' anxiety and secure better cooperation.

Case No. 3. A 4-year-old malnourished child presents with huge adamantinoma for resection and reconstruction. On airway examination we find mouth opening of 1.5 cm. Mallampati class II, normal neck movements.

Anesthetic management of children with anticipated difficult intubation should always include a full and frank discussion of risks with parents (and child if appropriate). The possibility of tracheostomy and, of failure to secure the airway should always be mentioned.

In adult anesthetic practice, an awake technique is often employed, but this cannot be done in children since cooperation is required to gain good bronchoscopic views. Antisialogogue premedication should be administered and child should be anaesthetized maintaining spontaneous ventilation. Here due a big disfiguring mass, achieving proper mask fit may be difficult. Oxygen should be given with either sevoflurane or halothane. Intubation should be performed under deep inhalational anesthesia. Alternately, anaesthesia may be induced *using propofol taking care to preserve spontaneous breathing*. Use of a muscle relaxant during induction of anesthesia should be avoided as it may result in a situation of CVCI, necessitating establishment of surgical airway rapidly.

Merits and demerits of inhalational induction versus intravenous induction in a patient with difficult airway

Inhalational induction assures maintenance of spontaneous breathing which is crucial in difficult airway situations. Also patient regulates his/her own depth of anesthesia—in case the obstruction worsens with deepening plane, less inhalational agent will be delivered and patients depth of anaesthesia will be reduced bringing back muscle tone and establishing patency of airway. However, inhalational induction is slow, may be irritant to airway, increase secretions, difficult to induce when proper mask fit cannot be achieved, will lead to OT pollution, patient might become very agitated and difficult to manage in lighter plane and more importantly gives you very less time for laryngoscopy. As against this IV induction with propofol, is smooth, easily accepted by patient, one can easily control both the speed of induction and the depth of anesthesia and thus give more time for laryngoscopy. However, major problem is that there are chances of apnea, which can prove life-threatening.

For our case:

Plan A: Inhalational induction, laryngoscopy and intubation.

Plan B: FOB guided intubation under inhalational anesthesia: The smallest size of fibreoptic bronchoscope available has an outer diameter of 2.5 mm and thus can take a size 3.0–3.5 endotracheal tube loaded onto them which can be used even in infants. However, these scopes have no suction channel and secretions must be aspirated with a normal suction catheter. It is also a delicate piece of equipment, and can be easily damaged. The nasal route is favoured in adult practice and can be used successfully in children. Walker et al. have described the technique for difficult paediatric intubation using LMA. Inhalation induction with spontaneous ventilation is followed by the use of a standard LM to maintain depth of anesthesia and provide a conduit for the fibreoptic bronchoscope. Guide the bronchoscope into the trachea through LMA. Pass a guide wire through the suction channel of the bronchoscope, remove the bronchoscope pass an airway exchange catheter over the guide-wire. Correct positioning of the catheter is confirmed by capnography and visually via the bronchoscope. Finally, remove the LM and pass tracheal tube over the catheter into the trachea.

In the above mentioned case insertion of LMA may be difficult due to big intraoral extension of mass. Either performing FOB-guided intubation or surgical tracheotomy in spontaneously breathing anaesthetized patient; are the two options that can be followed.

Plan C: Tracheostomy under inhalation anesthesia.

Case No. 4: A 70-year-old man, chronic bidi smoker (60 pack years) presents with hoarseness and dysphagia of six months duration. Since last 2 months he has difficulty in breathing and at present has mild inspiratory stridor. On flexible fibreoptic laryngoscopy he has a large exophytic left pyriform fossa mass. He has been posted for direct laryngoscopy to evaluate the extent of the lesion and biopsy to make a tissue diagnosis so that further plan of management can be decided. On airway examination we find mouth opening 5 cm, Mallampati class I. and he has artificial dentures. Thyromental distance is 3 fingerbreadths and has normal range of neck movements. How will you manage airway?

Lesions arising in the hypopharynx are most frequently located in the pyriform fossa and can commonly grow to a size sufficient to cause airway obstruction. Though not very vascular, they are friable and tend to break in pieces with instrumentation. Large tumors also distort the anatomy in hypopharynx making identification of structures difficult, especially in the presence of blood. A narrowed airway then becomes completely occluded making mask ventilation also difficult.

Such patients who already have signs of stridor would not cooperate for awake fibreoptic laryngoscopy. Trying any airway manipulation in an uncooperative patient will further add to trauma, bleeding and chances of dangerous airway obstruction.

Since this patient has partially obstructed airway, keep small sized endotracheal tubes ready. Keep a tracheostomy standby and have low threshold to perform tracheostomy.

In this case choice of plans can be:

Plan A: Preoxygenate well, anaesthetize with inhalational agents, perform gentle laryngoscopy and intubate if laryngoscopic view is good.

Plan B: Preoxygenate well, anesthetize with inhalational agents, perform gentle laryngoscopy and if you fail to have good laryngoscopy view, ask surgeons to perform DLscopy. As surgeons DL scope is channeled and is longer in length visualization of glottis and intubation is much easier.

Plan C: Upfront tracheostomy and then proceed with procedure.

Alternatively these patients can be induced with IV propofol and follow above plans.

Remember that airway resistance is directly proportional to flow rates and inversely proportional to size of glottic opening. In the presence of anxiety patient tries to hyperventilate, thus increasing inspiratory flow rate and getting feeling of more breathless. With induction of anesthesia, flow rates fall and one might find that glottis is not that badly narrow.

All blind intubation techniques are contraindicated in this setting with distorted anatomy, friable tumor and potential for trauma.

Case No. 5: 53-years-old female patient weighing 55 kg, presents with history of neck swelling gradually increasing over last 5 months with change in voice since 4 months and dysphagia to liquids and solids since 1 month. She also gives history of choking sensation intermittently. CT scan showed Enlarged thyroid, with tracheal compression and mediastinal extension, Hopkins mirror examination showed left vocal cord fixed. Her thyroid function Hematology and Biochemistry investigations are normal. How will you manage her airway?

Large goiters or cancers of the thyroid can be a threat to the airway by external airway compression, deviation, or distortion. Such lesions can cause upper airway obstruction by local invasion of the trachea, softening of the trachea (tracheomalacia), mass effect causing distortion of the trachea, and through involvement of the recurrent laryngeal nerve. Involvement of this nerve may jeopardize an already compromised airway by causing additional narrowing of the glottic opening as in our case.

Routine preoperative indirect laryngoscopy to evaluate the vocal cords of all patients presenting for thyroid surgery is important for both medical and medicolegal reasons. Medically it is important to detect nerve palsy which may not be manifest by voice changes because of compensation by the nerve on the other side. This would emphasize the importance of special caution in terms of preservation of the contralateral nerve. Medicolegally, such evaluation demonstrates the presence or absence of nerve involvement preoperatively, a fact which is of importance in the evaluation of voice disturbance postoperatively.

All patients with an anterior mediastinal mass should have a chest radiograph and a CT scan prior to any surgical procedure and the anesthesiologist must look at the imaging to plan the airway management. The CT scan will show the site, the severity, and the extent of the airway compromise. **Most important in such situations is not to allow loss of muscle tone, as that may be what is keeping tumor off the airway.**

Most of the times visualization of glottis on direct laryngoscopy is not a problem as problem lies below vocal cord level but correct placement of distal end of tube is difficult. Awake fibreoptic intubation is a safe and expeditious approach to the airway in a patient of this type. The major advantage of FOB-guided intubation is that one can place distal end of tube beyond the level of obstruction.

Patients with an anterior mediastinal mass who require general anesthesia need a step-by-step induction of anesthesia with continuous monitoring of gas exchange and hemodynamics. Maintaining spontaneous ventilation until the airway is definitively secured is a safest and popular strategy. Anesthetic induction can be inhalational with a volatile agent such as sevoflurane, or by intravenous titration of propofol, with or without ketamine. If muscle relaxants are required, assisted ventilation should first be gradually taken over manually to assure that positive-pressure ventilation is possible and only then can a short-acting muscle relaxant be administered. Development of airway or vascular compression requires that the patient be awakened as rapidly as possible and then other options for surgery can be explored. Perioperative life-threatening airway compression usually responds to one of two therapies: either repositioning of the patient (it should be determined before induction if there is one side or position that causes less symptomatic compression) or rigid bronchoscopy to open the obstruction for which an experienced bronchoscopist and rigid bronchoscopy equipment must always be immediately available in the operating room during these cases. For patients with life-threatening cardiovascular compression after induction that does not respond to

lightening the anesthetic the only therapy is immediate sternotomy and surgical elevation of the mass off the great vessels.

There are important differences in the management of airway in mediastinal tumors in children versus adults. Anesthetic deaths have mainly been reported in children. The deaths may be the result of the more compressible cartilaginous structure of the airway in children or because of underestimation of the severity of the airway compression in children due to the difficulty in obtaining a clear history of positional symptoms. Even with proper management, children with tracheo-bronchial compression greater than 50% cannot safely be given general anesthesia. Also, securing the distal airway with awake fibreoptic intubation and placement of an endotracheal tube distal to a tracheal obstruction, which is an option for some adults with masses compressing the mid-trachea, is not an option in most children.

What is the role of CP bypass in cases of mediastinal tumors with distal airway compression?

Many authors and textbooks advocate cardiopulmonary bypass as a 'standby' during induction of anesthesia for patients with large mediastinal tumor. The establishment of cardiopulmonary bypass prior to induction of anesthesia can be safely performed in adult patients. But once airway or cardiovascular collapse occurs, it will require at least 5–10 min to cannulate and establish adequate circulation and oxygenation, even with a primed pump and a prepared team. In such a scenario it is probable that a young patient can be resuscitated but will suffer neurological injury. Patients with severe symptoms due to large mass and airway or cardiovascular compression should not be given general anesthesia, even with maintenance of spontaneous ventilation. In these patients cardiopulmonary bypass or ECMO should be established prior to induction of anesthesia to maintain oxygenation or circulation.

Case No. 6: A 72-year man presented for CABG for triple vessel disease. Airway Evaluation showed Mouth opening 4–5 cm, Mallampati II and Prominent upper front teeth and incisors. Thyromental distance is 3 finger breadths. He has normal neck movements. Induction of anesthesia was achieved with thiopental, and vecuronium and patient could be adequately mask ventilated with oxygen, and isoflurane. On direct laryngoscopy only posterior arytenoids could be visualized but even after four attempts, could not be intubated. How will you tackle this situation?

This is a situation of Unanticipated difficult intubation: Elective Situation: Unanticipated, failed intubation following induction of general anesthesia is one of the most common airway emergencies anesthesiologists encounter in the operating room. What should one do when initial attempts at intubation are unsuccessful? The initial management following the first failed attempt is most important because the manner in which the initial problem is handled may greatly influence the subsequent outcome of this potentially life-threatening emergency.

Failed First Attempt

- Ventilate with 100% O_2
- Call for help
- Note time
- Maintain adequate anesthesia—use volatile inhalational agents with oxygen as that will still help in using high FiO_2 and maintain adequate depth of anesthesia. Downside is this might lead to OT pollution. Alternating one can use repeated boluses/infusion of IV hypnotic agents.
- Check vital signs
- Reassess head and neck position
- Define the problem
- Reassess equipment selection: change the technique and equipment with which you are familiar. There is no point in insisting on the same technique with which you have failed. One can use gum elastic bougie, different blades, LMA, ILMA, etc.
- Get the most skilled person available.

The next attempt has to be the `best' attempt (you had already four unsuccessful attempts, actually one has to stop at 2 attempts and not get tempted to have one more go), i.e. optimal neck position, optimal laryngoscope blade, gum elastic bougie and the most experienced `intubator'. Until all these are ready it is best to keep oxygenating and ventilating the patient rather than multiple attempts at intubation.

If the further attempt fails:

- In an elective situation like this one should prefer to wake the patient and cancel surgery. Subsequently patient can undergo FOB-guided intubation, or retrograde intubation
- In an emergency situation one can use an LMA/ILMA, and proceed with surgery with cricoid pressure or perform surgical tracheostomy.

ASA guidelines advocate only 2 attempts per operator. This may seem a drastic step, allowing only 2 attempts at visualization; however, a little reflection will reveal that multiple unsuccessful attempts at intubation can change a controlled situation into an uncontrolled one. Airway structures including the tongue, pharynx and the epiglottis are delicate structures, which are very prone to injury, bleeding and swelling, all of which impair visualization, making the laryngoscopic view more difficult and worse,

impairing mask ventilation, leading to the life-threatening CVCI situation.

Case No. 7. 80 kg 62-year-old male patient presents for cholecystectomy. On airway evaluation mouth opening is 6 cm. Mallampati I. Absent left lateral incisor and canine. Thyromental distance-three and half-finger breadths and has normal neck movements. No airway problems were anticipated preoperatively by either the surgeon or anesthesiologist. Patient was induced with fentanyl, thiopental and succinylcholine. Oxygenation and ventilation was maintained by mask and two attempts were made to intubate the patient, neither successful. There was dislodgement of tooth and considerable bleeding after the first attempt. Patient could be ventilated with mask. First operator again tried twice but was unsuccessful. A second anesthesiologist attempted to intubate the patient but failed on further two attempts to visualize even the arytenoids. Mask ventilation after this could not be established and there was significant stomach distension, there was considerable bleeding, the oropharynx suctioned but patient could not be ventilated and the saturation begins to fall: 70....60....50....40%.

This is a situation of unanticipated difficult intubation: Emergency situation

Remember patients usually do not die from our failure to intubate; they die because we can't stop trying till we land up in situation like this. In this case with bleeding at first attempt the operator should have maintained mask ventilation and reassess the situation. Change of technique or change of operator is dictum for failed first attempt. Bleeding and edema are progressive with each attempted intubation and a patent airway can be lost as happened here. In the life threatening cannot intubate/cannot ventilate situation, three methods to ventilate and oxygenate the patient have been described: transtracheal jet ventilation (TTJV), the esophageal tracheal combitube (ETC), and the laryngeal mask airway (LMA).

Role of LMA and Combitube in CVCI situation: In most situations of CVCI these two supralaryngeal devices will be adequate for managing the patients. It is seldom that transtracheal jet ventilation will be needed.

Classic LMA is a reusable device made of medical grade silicone. It is to be discarded after 40 autoclaving. Combitube is a disposable double lumen tube that combines the features of a conventional tracheal tube and that of an esophageal obturator airway. (Details are given in instrument section). Parmet et al have reported of rescuing 16 of 17 CVCI patients with use of combitude. The only patient who could not be rescued by them was a patient with intratracheal blood clots. Davis et al have shown 97–99% success rate of using Combitube in prehospital airway rescue when patients could not be intubated.

Transtracheal Jet Ventilation (TJV) in CVCI situation: A technique to establish TJV should be part of every anesthetist's clinical skill and the equipment necessary to do so should be included in every difficult airway cart. The technique should be considered early in all situations where non-surgical techniques have failed to establish adequate ventilation. The TJV technique, using needle cricothyrotomy, is perhaps the fastest method to establish oxygenation in a patient of CVCI. In adults this is performed by placing a large-caliber cannula 12- to 14-gauge, through the cricothyroid membrane into the trachea. After confirming correct placement by aspirating air in a saline filled syringe, the needle is removed and the cannula fully inserted into the trachea in a caudal direction and firmly held. The cannula is then connected to a Jet Injector. Alternately attach 2 mL syringe to the cannula, fix 8/8.5 noseworthy connection to cannula-syringe assembly and then attach AMBU unit to ventilate the patient. Remember that now exhalation is occurring through very narrow lumen and you need to give adequate time for expiration otherwise patient can develop barotrauma. This is only a rescue airway and a definite airway has to be established in form of tracheostomy.

Today, several ready-to-use commercial kits are available in the market for performing cricothyrotomy. The Seldinger technique is associated with maximum success and least morbidity. This kit includes 4.0 mm ID tube, a needle, syringe, flexible wire and number 10 scalpel blade.

Case No. 8. 73-year-old man underwent complex facial surgery with radial forearm free flap reconstruction, and shifted to recovery room with nasal ETT in situ to be extubated coming morning. 4 hours after shifting he self-extubated and has partial airway obstruction. How will you manage?

The moment you realize that your patient has self-extubated, call surgeons for your help as situation can turn tricky. Oxygenate the patient. Here patient has fresh surgery with intraoral reconstruction which has changed the anatomy. There will be intraoral edema, secretions and some amount of blood. Patient might be having airway obstruction due to secretions which he is unable to clear. Perform a thorough intraoral suction. Try and oxygenate the patient with mask. Any attempts of direct laryngoscopy in uncontrolled environment can be disastrous. Possibility of a retained gauze piece or throat pack should be considered and rechecked with surgeons. Doing emergency tracheostomy is not an easy option here as patient will be uncooperative, achieving proper position will be difficult, and *using sedation to get patients cooperation will be faced with danger of loosing the existing airway*. It would be preferable to shift this patient back to OT, Upper airway can be nebulized with 4% lignocaine and try and perform gentle FOB and intubate.

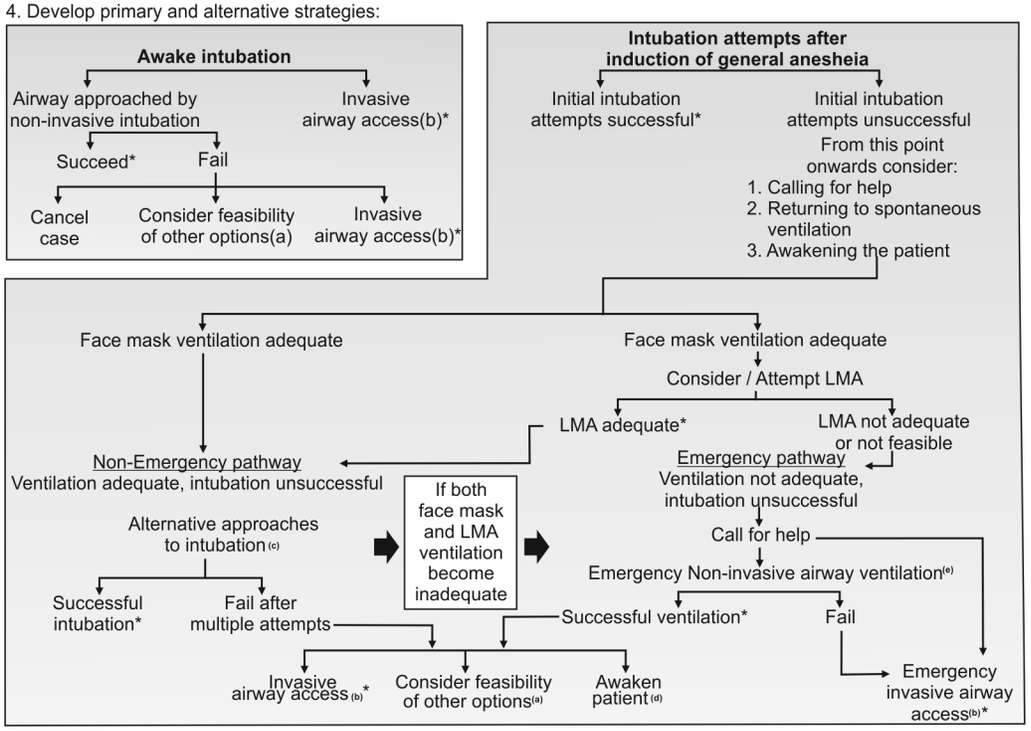
AMERICAN SOCIETY OF ANESTHESIOLOGISTS

DIFFICULT AIRWAY ALGORITHM

1. Assess the likelihood and clinical impact of basic management problems:
   - A. Difficult ventilation
   - B. Difficult intubation
   - C. Difficulty with patient cooperation or consent
   - D. Difficult tracheostomy
2. Actively pursue opportunities to deliver supplemental oxygen throughout thr process of difficult airway management
3. Consider the relative merits and feasibility of basic management choices;

Awake intubation	vs.	Intubation attempts after induction of general anesthesia
Non-invasive technique for intial approach to intubation	vs.	Invasive technique for intial approach to intubation
Preservation of spontaneous ventilation	vs.	Ablation of spontaneous ventilation

4. Develop primary and alternative strategies:



Suggested Reading

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22 Burns

N Amin, V Patil, B Trivedi

A 30-years-old male patient is brought to the casualty after being rescued from a house fire. He has burns over his face, hands and trunk.

How will you assess the severity of burns?

Severity of burns can be assessed in several ways.

There are 3 commonly used methods for measuring a burn.

a. Wallace rule of nines is used to assess the percentage of body surface area burnt. The total body surface area is divided into multiples of nine and is used as a rule of thumb to quickly calculate the total body surface area burnt. Though it tends to overestimate the burned area, it is a good method for measuring medium to large burns.

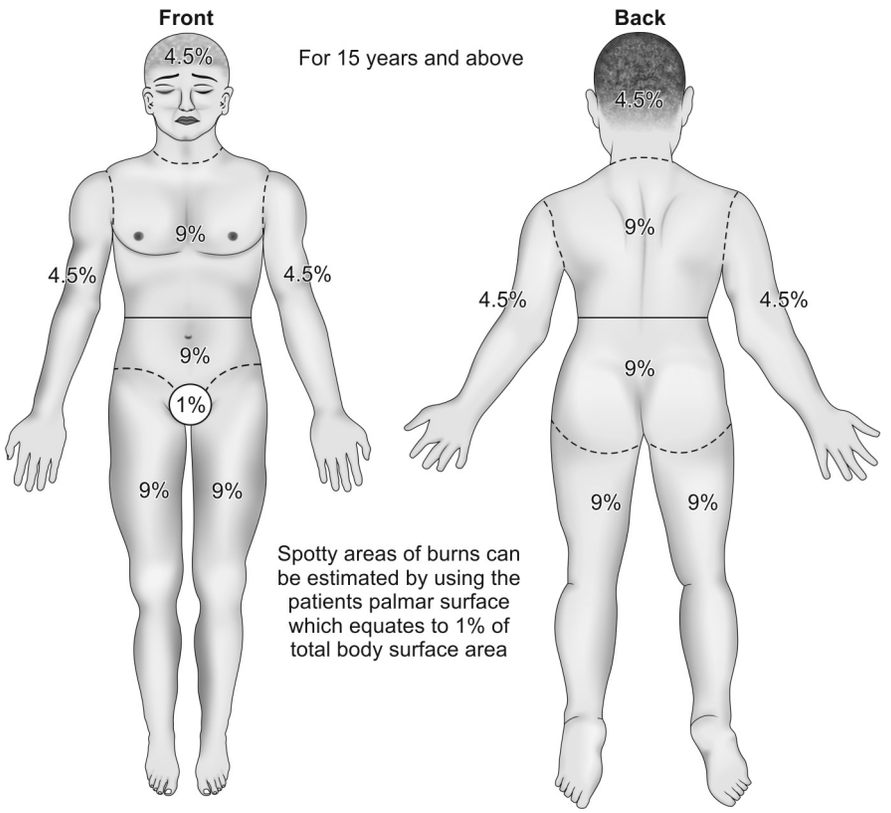

Fig. 22.1 Calculation of body surface area in adults

Head and neck	9%
Upper extremities	9% each
Chest (anterior and posterior)	9% each
Abdomen	9%
Lower back	9%
Lower extremities	18% each
Perineum	1%

b. *Palmar surface:* The palmar surface of the patient's hand, with fingers very slightly spread, equates to approximately 1% of the patient's TBSA. This is a quick and reliable method of assessing patchy areas of burns up to 15% of TBSA. It is not very accurate in estimating moderate burns. If extent of burns is greater than 85% then, burns can be estimated by simply measuring the unburned areas.

c. Lund and Browder chart (described later).

Burns can also be classified according to the depth of the injury. Assessment of the depth of the burn is important for planning wound care and predicting functional and cosmetic results.

- *Superficial:* Only epidermis is damaged. Heals spontaneously within 7 days with no scarring. There is erythema, no blisters, it blanches on pressure and is painful.
- *Superficial partial thickness (superficial dermal):* Epidermis completely destroyed, together with approximately the upper one-third of the dermis, but the adnexal skin structures (hair follicles, sebaceous glands, sweat glands) survive. However, the epithelium regenerates rapidly and the wound heals within 10–14 days without any scarring. It has a red and mottled appearance with swelling and blanches on pressure. There is blister formation and is painful.
- *Deep partial thickness (deep dermal):* Epidermis and substantial part of dermis with adnexal structures are destroyed. Healing takes several weeks or months as epithelialization is slow. There is scarring. It is pale in appearance with no blanching. There is no pain only pressure sensation is felt. These burns require excision and grafting for rapid return of function.
- *Full thickness:* All skin elements destroyed. Damage may extend to involve deep structures such as muscle, bone and major neurovascular bundles. It appears dark and leathery. The surface is dry and painless and does not blanch on pressure. It requires excision and grafting. Heals very slowly by wound contracture with minimal epithelialization from edges.

Is the "rule of nines" also used for assessing the severity of burns in children?

Children have relatively larger heads and smaller limbs as compared to adults. Due to the different body proportions the "rule of nines" is inaccurate in children. Age-adjusted nomograms are a better method because they reflect the gradual change in the relative body surface areas with age. Currently the Lund and Browder chart is the most accurate method for assessing the burn area in children.

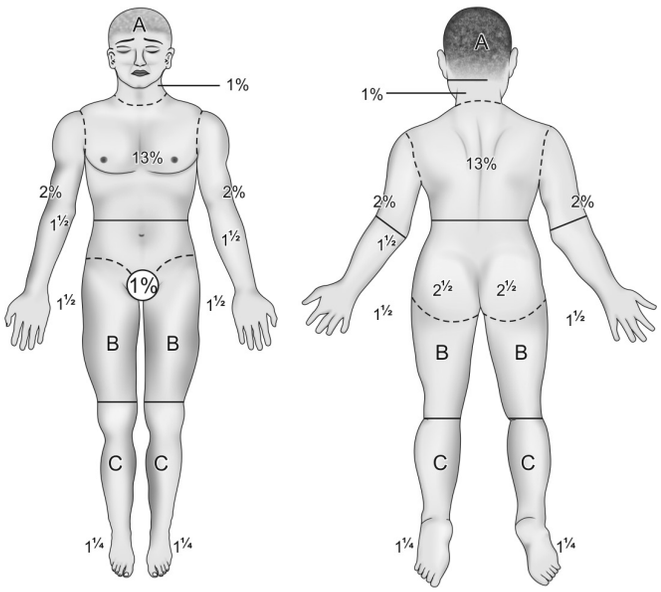

Fig. 22.2 Calculation of body surface area in children

Chart for calculation of percentage of BSA burn in children (Age in years)					
Area	0	1–4	5–9	10–14	15
A: half of head	9½	8½	6½	5½	4½
B: half of one thigh	2¾	3¼	4	4¼	4¼
C: half of one lower leg	2½	2½	2½	3	3¼

Briefly describe the three major layers that make up healthy skin.

Epidermis: This layer is composed of keratinized, stratified, squamous epithelium. It provides a thick, water-proof, protective covering over the underlying layers of skin.

Dermis: This layer is composed primarily of dense, irregular, fibrous connective tissue that is rich in collagen (for strength) and elastin (for elasticity). The dermis contains blood vessels, nerve endings, and epidermally derived cutaneous organs such as sweat glands, sebaceous glands, and hair follicles.

Hypodermis (subcutaneous layer): This layer is composed primarily of loose, areolar connective tissue and adipose tissue. This fat layer provides both mechanical cushioning as well as a thermal insulation for the underlying organs.

What are the functions of skin?

Skin is the largest organ in the body. In adults the surface area ranges from 1.5–2 m^2. It has various important functions:

- It protects the body against invasion from microorganism
- Temperature regulation
- Fluid and electrolyte homeostasis
- Touch, pain and temperature sensations
- Vitamin D metabolism
- UV protection.

How would you determine the prognosis in a patient with burns?

Survival of a patient after burn injuries depends on the patient's age and percentage of body surface area burnt. As a rough guide %mortality = BSA of burn + age (Baux score). Younger patients are more likely to survive. Other factors which influence the outcome are associated inhalational injury (doubles the mortality rate), depth of the burn, other injuries sustained during burns, and associated medical conditions.

How would you define major burns?

Definitions of major burns are based on body surface area burned and the area of the body burned:

- Third degree (full thickness) burn injuries involving > 10% of TBSA
- Second degree (partial thickness) burn injuries involving > 20% TBSA at extremes of age and > 25% TBSA in adults
- Burns involving face, hands, feet, genitalia, perineum, major joints
- Inhalational injuries
- Chemical burn injuries
- Electrical burn injuries
- Burn injuries in patients with co-existing medical disease
- Burns associated with trauma.

What is the role of an anesthetist during the management of a burns patient?

1. Anesthetist may be involved in the initial resuscitation of a burns patient. Especially if there is hypoxemia and airway compromise which is life-threatening.
2. Intensive care management would be needed if the patient develops sepsis and multi-organ failure.
3. A burns patient would need general anesthesia for various surgical procedures
   - Early excision of damaged tissues
   - Excision of granulation tissue and subsequent skin grafting
   - Change of dressings
   - Reconstructive plastic surgical procedures to relieve contractures, correct deformities and restore limb function.

What are the pathophysiologic changes seen in burn injuries?

Following burns there is local and systemic inflammatory reaction, which results in the releaze of various inflammatory mediators (oxygen radicals, histamine, prostaglandin, bradykinin, nitric oxide, serotonin and substance P and complement). In minor burns, the inflammatory process is limited to the wound itself. However, in major burns, local injury triggers the releaze of circulating mediators which results in a systemic response. Interleukins 1 and 6 and TNF are the primary mediators of systemic inflammation after burns. Various studies have shown that endotoxin concentrations correlate with burn size and predict the development of multiple-organ failure and death. After burn injury there is increased nitric oxide levels which may contribute to hemodynamic and immunologic alterations.

Hemodyanamic response: There is increased capillary permeability due to systemic inflammatory response which results in loss of large amounts of protein-rich fluids from the intravascular compartment to interstitial compartment. As a result of this there is hypoalbuminemia which is further exacerbated by decreased hepatic albumin production in favor of acute phase protein synthesis. The loss of fluid into the interstitial space results in decreased circulating blood volume. Cell membrane adenosine triphosphatase activity is reduced which results in accumulation of intracellular sodium and water, further depleting the intravascular compartment. Fluid loss also continues by evaporation and exudation from the wound site further causing intravascular depletion. During the first 24 hours of burns there is releaze of catecholamines, vasopressin, and angiotensin which causes peripheral and splanchnic vasoconstriction and decreased cardiac contractility. All this can result in hypoperfusion of the end organs. This phase is known as burn shock. It is due to a combination of hypovolemic, cardiogenic and distributive element. Excessive resuscitation at this stage leads to generalized edema, which further compromises tissue oxygenation. Reperfusion of the ischemic zone releazes oxygen-free radicals further causing local cell membrane damage and immune response. After the patients are adequately fluid-resuscitated, there will be a phase of increased cardiac output and a marked reduction in systemic vascular resistance.

Respiratory responses: Releaze of inflammatory mediators can cause bronchoconstriction in the absence of inhalation

injury. In patients with inhalational injury the releaze of inflammatory mediators can lead to the development of the acute respiratory distress syndrome (ARDS).

Metabolic responses: After major burns there is a hypermetabolic response which lasts for up to 1 year after the burns injury. There is decreased protein synthesis and increased muscle breakdown. Increased circulating levels of catabolic hormones lead to increased gluconeogenesis resistant to insulin infusion. Increased peripheral lipolysis leads to rapid depletion of Glycogen stores. All these changes lead to impaired wound healing exposing patients to increased infection risk. Rehabilitation is often difficult in these patients.

Thermoregulation: Due to the hypermetabolic responses, the metabolic thermostat is reset. Burns patients increase their skin and core temperatures somewhat above normal regardless of the environmental temperatures. Following thermal injury, thermoregulatory functions of the skin like vasoactivity, sweating, piloerection and insulation are either abolished or diminished. Daily evaporative water loss is 2500 mL/m^2 in adults and 4000 mL/m^2 of burn surface in children.

Immune system responses: Following severe burns there is loss of protective skin barrier and suppression of the immune system. Also these patients need invasive lines and urinary catheter for monitoring. As a result of all this, burns patients have a significantly increased risk of repeated episodes of infection.

Hematologic system: Burns patients are usually anemic due to loss of erythrocytes from the burn wound, bleeding during surgeries, haemolysis of erythrocytes due to thermal injury, suppression of erythrocyte production and decreased erythrocyte survival after burns. Also the hematocrit is increased for at least 48 hours after burns due to hemoconcentration and may not be an appropriate guide for need for transfusion. Platelet counts and coagulation factors are decreased due to dilution and consumption. There is activation of thrombotic and fibrinolytic mechanisms.

Renal and Electrolytes: Decrease in renal blood flow secondary to hypovolemia and decreased cardiac output and systemic vasoconstriction due to circulating catecholamines can lead to renal impairment. Other mechanisms for renal failure include the nephrotoxic effects of drugs, myoglobin, and sepsis. Renin angiotensin aldosterone system is activated due to decreased renal blood flow resulting in the releaze of antidiuretic hormone. The net effect on the renal function is retention of sodium and water and exaggerated losses of potassium, calcium and magnesium. Hyperkalemia may occur due to tissue necrosis and hemolysis.

Gastrointestinal tract: Ileus is common in burns patients; hence early decompression of the stomach through a nasogastric tube is indicated. Curlings ulcers are acute ulcerations of the stomach or duodenum seen in patients with sepsis or extensive burn injuries. The exact etiology is unknown. Endotoxemia can result from disruption of the gastric mucosal barrier. Gastric mucosal ulcerations can be minimized by proper hemodyanamic resuscitation, early enteral feeding and the use of antacids and H2 receptor blockers.

You are called to the casualty to see a patient with burns. What would make you suspect the presence of inhalational injury?

The diagnosis of inhalational injury depends on a high degree of suspicion, clinical examination and laboratory testing. Inhalational injury should be suspected if there is a history of explosion, impaired mentation and/or confinement in a burning environment, inhalation of toxic fumes and associated alcohol intoxication.

On examination there would be darkened or reddened oral and/or nasal mucosa, burns to the face, lips or nares, singed eyebrows or nasal hairs. The presence of carbon or soot on teeth, tongue or throat, hoarse voice or productive cough would indicate inhalational injury. Tracheal tug, inspiratory stridor or inability to clear secretions may indicate impending airway occlusion. There may be swelling of the tissues around the airway due to circumferential burns to neck. Therefore early intubation is indicated in this situation. Arterial blood gases (showing high lactate levels in hemodyanamically-stable patient) and carbon monoxide levels (measured using co-oximetry) may be useful in determining the degree of insult.

What is the pathophysiology of inhalational injury?

Inhalation injury occurs as a result of direct thermal injury to the mucosa of the respiratory tract or from chemical tracheobronchitis due to the inhalation of incomplete products of combustion. The pathophysiological process leading to inhalational injury can be remembered using the mnemonic HOTT: Heat, Oxygen deficiency, Toxins—local, Toxins—systemic.

Airway injury above the larynx: Direct thermal injury primarily occurs in the upper respiratory tract only. This is because there is reflex closure of the glottis and also because heat dissipation through the bronchial tree is very efficient. Usually there is

upper airway edema, which may progress to complete airway obstruction. The temperature required to produce such injury will depend on the heat capacity characteristics of the gas or vapor. Dry gases have less injurious potential than a similar exposure to saturated vapors. Upper airway edema usually peaks at 24 hours following injury and resolves within the first week. Stridor and respiratory distress indicate inhalation injury. If airway obstruction is suspected intubation should be performed immediately as the pharyngeal mucosa swells rapidly particularly after fluid resuscitation. Even patients with a mild upper airway injury are at risk for development of progressive airway obstruction as tissue edema develops.

Airway injury below the larynx: The lower airway is rarely burned by exposure to heat unless substances with a very high specific heat capacity are inhaled, such as superheated steam.

Chemical tracheobronchitis: The inhalation of incomplete products of combustion leads to a pulmonary inflammatory response similar to that seen after gastric acid aspiration. Water-soluble gases such as ammonia, nitrogen dioxide, sulphur dioxide, and chlorine combine with water in the respiratory tract, producing strong acids and alkalis. For example, sulphur dioxide forms sulphuric acid. Lipid-soluble gases such as aldehydes and hydrogen chloride are carried into the lower airways on carbon particles. Aldehydes such as acrolein, which are produced by the combustion of cotton, wood, and various synthetic fibers, impair ciliary function and damage mucosal surfaces. These chemical products act as direct irritants causing bronchospasm, edema, and mucous membrane ulceration. Necrotic cell debris, particulate matter and proteinaceous fluid accumulate in the bronchial tree, leading to cast formation and atelectasis. The production of surfactant is impaired and a ventilation/perfusion mismatch develops as the alveolar injury evolves. Smaller the size of the smoke particle, deeper is the penetration into the lung.

Systemic Intoxication Injuries: Toxins and chemicals like carbon monoxide (CO), hydrogen cyanide, ammonia, hydrofluoric acid and phosgene that reach the alveoli can be absorbed into the systemic circulation. Severe systemic intoxication often results in metabolic acidosis. CO interferes with tissue oxygenation by binding avidly to hemoglobin, preventing oxygen from binding to hemoglobin. It also binds to cytochrome oxidase and inhibits mitochondrial function at a cellular level. Hypoxic encephalopathy secondary to CO poisoning is thought to result from a reperfusion injury in which the products of lipid peroxidation and free radical formation contribute to morbidity and mortality. Combustion of plastics, polyurethane, wool, silk, nylon, nitriles, rubber, and paper products can all lead to the production of cyanide gas. Cyanide interferes with cellular metabolism by binding to the ferric ion in cytochrome A_3. Due to decreased oxygen utilization there is anaerobic metabolism with development of lactic acidosis.

Oxygen deficiency: During inhalation injury hypoxia is multifactorial and may be immediate or delayed.

- Utilization of oxygen during combustion can cause significant decrease in ambient oxygen concentrations.
- Loss of consciousness or airway edema can lead to airway obstruction.
- Inhalation of CO or hydrogen cyanide can result in cytotoxic hypoxia.

Lung injury develops in 24 and 48 hours resulting in delayed hypoxemia. This is significant as it usually coincides with the period of maximal tissue edema. The combination of low capillary oxygen tension and reduced tissue oxygen diffusion will compound tissue hypoxia and subsequent 'reperfusion' may result in worsening of the injury.

What are the other components of smoke and their effects?

1. *Oxygen free radicals:* They cause peroxidation of the outer lipid layer of the cell leading to cell damage and cell death.
2. *Ammonia:* It is an airway irritant causing cough, increased secretions and bronchoconstriction. It forms ammonium hydroxide, a potent alkali leading to tissue necrosis especially of the lower airways.
3. *Sulphur dioxide:* It is also an irritant to the airway and the eyes. It is oxidized to sulfurous acid and sulphuric acid. It is associated with lower-airways injury and lung edema.
4. *Nitrogen dioxide:* It is a gas with limited solubility in water and can be carried by particles. The injury is mainly to lower airways and is delayed in onset up to 72 hours. Lipid solubility leads to damage to all membranes and cell death.
5. *Chlorine:* It is an irritant compound causing bronchospasm. When dissolved in water on the mucosal surface, it forms hydrochloride and hydrochlorus acid. It is a potent oxidizing agent causing cell necrosis.

How would you diagnose and treat inhalational injuries?

High-degree of suspicion based on the history, a good clinical examination and laboratory testing will help in the diagnosis of inhalational injury (already discussed above).

The presence of inhalational injury is currently diagnosed by bronchoscopy or xenon lung scan. Fibreoptic bronchoscopy (FOB) is performed for diagnosis and treatment. Mild injury gives rise to erythema and edema of the mucosa. In severe

inhalational injury FOB may show grey colored mucosa, erosions, ulcerations and/or desquamation. However there is no method available for assessing the severity of the injury. Studies in sheep have shown that CT scan is potentially useful in assessing the severity of inhalational injury non-invasively. However, human trials are needed to establish the clinical utility of this method. A chest X-ray at presentation may be normal because radiographic changes often do not appear until 24 hours or more after the insult.

Management is primarily supportive. Early tracheal intubation, ventilatory support, aggressive pulmonary toilet, bronchodilator therapy and bronchoscopic lavage all play an important part in the treatment of inhalation injury.

The ABC (Airway, Breathing, and Circulation) approach should be used for managing patients with inhalational injury. There should be a low threshold for intubating patients with inhalational injury. Consider early intubation if the there is stridor, GCS < 8, hypoxia or hypercapnia, full thickness neck burns, deep facial burns and oropharyngeal edema. A head up position may delay swelling. With an abnormal airway or upper airway obstruction, the safest way to secure the airway is with the patient awake. Key prerequisites include effective topical anesthesia, proper patient positioning, and supplemental oxygen administration. Alert patients can be given intravenous opioid for pain relief, but sedatives should be used cautiously as it may worsen airway obstruction. The patient should be anesthetised using inhalational induction. Choice of intubation technique depends on the expertise of the operator. Fibreoptic intubation techniques are not useful in patients with swollen upper airways as it is impossible to anaesthetise the airway and airway instrumentation may cause further trauma and swelling. Alternatives include direct laryngoscopy, the laryngeal mask airway, and use of Bullard laryngoscope. General anesthesia with inhalational induction may be needed in uncooperative patients. Oral intubation should be done and cutting the tube should be avoided.

When the upper airway is badly damaged and endotracheal intubation is not possible, a direct surgical approach to the airway is indicated. Options include a needle cricothyroidotomy, surgical cricothyroidotomy, or tracheostomy; however, because of the high incidence of complications a surgical airway should be considered only as a last resort.

Mild smoke inhalation can be managed supportively with high flow oxygen, humidified inspired gases, bronchodilators and physiotherapy. Patients with more severe injury will need ventilator support for deteriorating lung function. FOB should be done in all intubated patients with suspected inhalational injury as it allows better diagnosis and enables aggressive pulmonary toilet.

Fluid restriction does not prevent pulmonary edema. Actually under resuscitation makes pulmonary damage worse. Ventilatory management should include the low tidal volumes (6 mL/kg) and low pressures (plateau pressures less than 30 cm H_2O) currently recommended for the management of the acute respiratory distress syndrome (ARDS). Bronchodilators may be helpful in the treatment of bronchospasm following inhalation injury. Mucolytics like N-acetylcysteine may help in the airway clearance of mucus plugs and may help in improving the PaO_2/FiO_2 ratios. Steroids and prophylactic antibiotics are not indicated. Management of CO poisoning and cyanide toxicity is discussed later.

How would you resuscitate a patient with burns?

Treat according to CPR protocol (ABC's). If there are any signs of airway obstruction or any suspicion of inhalation injury then it is an indication for immediate endotracheal intubation. All burns patient should receive 100% oxygen with a reservoir mask.

First aid: In the initial first aid one should try and stop the burning process and cool the burn wound. Use blankets to extinguish flame burns. Remove the heat source like clothing, embers, chemical etc. Synthetic fabrics burn at high temperatures and melt into a hot residue that continues to burn the patient. Chemical powders should be brushed from the wound. The burnt area should be rinsed with tepid water (at least 15°C for 20–30 minutes). This helps to limit the eventual depth and area of the resulting burn. Ice should never be used because it causes vasoconstriction leading to further tissue damage and hypothermia. The patient should then be covered with warm, clean, and dry linens to prevent hypothermia.

Primary Survey

A. Airway maintenance with cervical spine control
B. Breathing and Ventilation
C. Circulation with Hemorrhage control
D. Disability – neurological status
E. Exposure and environmental control
F. Fluid resuscitation proportional to burn size.

Primary Survey

Airway and control of cervical spine: The first step is to identify inhalational injury in burns patient. A burns history can give an idea about the possibility of inhalational injury. Initial compromise of the airway is always due to low GCS.

Coma may be due to trauma, drugs and alcohol, as well as effects of CO and smoke inhalation. If there is any history of trauma, cervical spine should be immobilized. Airway can be supported with simple manual techniques or use of basic airway adjuncts. After clearing the airway; administer 100% oxygen via a reservoir mask; this would also help to treat CO poisoning. Elevate the head of bed to 30–90° to reduce the facial or airway edema provided there is no cervical spine injury. Endotracheal intubation and mechanical ventilation is needed in patients who are unconscious from co-existing trauma or from inhalation of toxic substances, patients who develop acute respiratory failure due to smoke inhalation and in patients with major burns.

Breathing: The chest should be exposed to ensure that chest expansion is adequate and equal. Look for signs of respiratory distress (increased respiratory rate, tracheal tug and excessive use of accessary muscles). Circumferential burns to the chest may restrict breathing and impair gaseous exchange. An escharotomy may be required in circumferential burns that compromise breathing. Blast injuries related to the burn can cause lung contusions and alveolar trauma, potentially leading to ARDS. CO poisoning should be ruled out by measuring the HbCO level. An arterial blood gas should be obtained to determine the carboxyhemoglobin level. Pulse oximeter will give spuriously high readings in the presence of significant levels of COHb. High flow oxygen should be administered via a non-rebreathing mask at 100%. Check for additional injuries that may have occurred when the patient sustained a burn injury (e.g. pneumothorax, hemothorax, tension pneumothorax or a flail chest).

Circulation: Rapid assessment of the volume status should be done. Monitor the blood pressure, pulse and capillary refill in both burned and unburned limbs. Escharotomies may be required if there is isolated poor perfusion in a limb with circumferential full thickness burns. Use of a blood pressure cuff may be difficult in a burned patient and invasive arterial monitoring may be needed. Stop any bleeding with direct pressure. If the burned patient is hypotensive then look out for other causes of hypotension.

Disability (Neurological status): Establish the level of consciousness and examine the papillary response to light. Restlessness and decreased level of consciousness may be due to hypoxemia, hypovolemia as well as concurrent head trauma. While taking history and assessing the airway a simultaneous assessment of baseline Glasgow Coma Score should be done in all patients.

Exposure and environment control: Remove any jewellery and clothing and keep the patient warm. Burns patients rapidly become hypothermic due to loss of their protective thermoregulatory skin and evaporation of fluid from the exposed tissue.

Fluid resuscitation: Following tissue burns there is large fluid shift due to increased capillary permeability. This results in edema formation particularly during the first 36 hours. The resultant fluid depletion is greatest in the first few hours and early fluid resuscitation is essential to avoid hypovolemic shock and ARF. Adults with greater than 15% total body surface area (TBSA) burns, and children with greater than 10% TBSA burns will need fluid resuscitation. While resuscitating burns patient insert urinary catheter in order to monitor urine output as a function of organ perfusion. One should aim for a urine output of at least 0.5 mL/kg/hr in adults and 1 mL/kg/hr in children who weigh less than 30 kg.

After establishing airway patency and identifying and treating immediately life threatening injuries, 2 large bore IV cannulae should be inserted, preferably in a non burned area. Upper extremities are preferable to lower extremities for venous access because of high incidence of phlebitis in the saphenous veins. Other options are saphenous cut down, femoral cut down and intra-osseous needles in children less than 6 years.

Warmed ringer lactate solution should be used for treating hypovolemic shock. Parkland formula is widely used for burns resuscitation. Fluid requirement is 4 mL/kg/% BSA of burns; give half in first 8 hours and the remaining over next 16 hours. In children it is necessary to administer maintenance IV fluids containing glucose in addition to the burn formula. These formulae are only a guide and assessment of resuscitation should be based clinically on heart rate, BP, capillary refill, CVP, urine output, peripheral and core temperature and mental state of the patient.

Blood should be collected for grouping and cross-matching, CBC, blood glucose, serum electrolytes, serum urea and creatinine, COHb levels and arterial blood gas analysis. Urine should be tested for myoglobin/hemoglobin. Chest X-ray should be done. Other X-rays may be indicated if there are associated injuries.

Pain management: A severely burned patient may be restless and anxious due to hypoxemia and hypovolemia rather than pain. Hence, these patients respond better to oxygen or increased fluid administration rather than narcotic analgesics or sedatives which may mask the signs of hypoxemia or hypovolemia. Narcotic analgesics and sedatives should be administered in small frequent doses by intravenous route only. Intramuscular and subcutaneous injections should be avoided as absorption will be erratic and analgesia delayed.

Secondary survey: Once the patient has been stabilised he should be thoroughly examined for any concomitant injuries. After assessing the size and depth of burns, wash the burnt area and do the dressing. Use dressing (preferably transparent to facilitate wound inspection) to cover the burnt area as it protects the wound, reduces heat and evaporative losses.

Insert nasogastric tube and decompress the stomach if nausea and vomiting are present, in patients with burns more than 20% TBSA and all intubated patients. As patient with burns are in a highly catabolic state establishing enteral feeding within 4 hrs of admission helps to improve the outcome. Enteral feeding should be commenced as soon as possible after burn injury to promote normal gut function and to decrease the potential for bacterial translocation across the gastric mucosa.

Prophylactic antibiotics are not indicated in the early post burn period. Antibiotics should be reserved for treatment of infection.

Tetanus prophylaxis should be given.

Early referral to a burns centre would be appropriate.

What are the various formulas for fluid resuscitation in patients with burns. Which is the most commonly used formula?

The main aim of resuscitation is to support the perfusion of vital organs and also to maintain adequate tissue perfusion to the burn area itself and so prevent it from deepening. historical overview of various burns resuscitation formulas is given in the Table 22.2.

The Parkland formula: It is the most widely used resuscitation formula in the world. Only the initial 24 hours crystalloid portion of the formula is followed closely with little attention focussed on the subsequent 24 hours colloid recommendations. Children also require maintenance fluid at an hourly rate of 4 mL/kg for the 1st 10 kg + 2 mL/kg for the 2nd 10 kg + 1 mL/kg for every kg thereafter. This can be given orally (preferably as a nutritious fluid such as milk), or intravenously using 5% dextrose and 0.45% normal saline solution, but not both together. The amount of fluid given should be constantly adjusted to maintain a urine output of 0.5–1 mL/kg/h in adults and 1–2 mL/kg/h in children. The fluids have to be calculated from the time of injury and not from the time of admission. The older fluid resuscitation formulas are confusing and difficult to recall. As a result of this the US army institute of surgical research (USAISR) have developed a simplified resuscitation formula named 'The Rule of Ten' which is currently being implemented. It falls within American Burn Association (ABA) acceptable guidelines for fluid therapy. Initial fluid rate in mL/h = %TBSA × 10. For every 10 kg above 80 kg, 100ml is added to this rate. When compared directly to the Parkland formula, the fluid rate would be overestimated for patients under 40 kg and underestimated for patients over 140kg. Further adjustments to the fluid rate can be made based on clinical observations of effect.

What are the complications of fluid resuscitation?

Compartment Syndrome: It occurs when the pressure within a compartment of the arm or leg or in the abdomen

Table 22.2 Formulas for fluid resuscitation

Historical overview of various burn resuscitation formulas		
1942	**Harkins formula**	Any patient with at least 10% burn: administer 1000 mL plasma for each 10% TBSA burn over first 24 hours
1947	**Body-weight Burn budget**	First 24 h: 1-4 lit RL+ 1200 ml 0.5 NS + 7.5% body weight colloid+ 1.5-5 lit 5%D. Second 24 h: same formulation except change colloid to 2.5% body weight
1952	**Evans formula**	First 24 h: NS at 1ml/kg/% burn + colloids at 1ml/kg/% burn +2lit glucose in water Second 24 h: one half the first 24 h crystalloid and colloid requirement + the same amount of glucose in water as in the first 24 hours.
1953	**Brooke formula**	First 24 h: RL at 1.5 mL/kg/% TBSA burn + colloid at 0.5 mL/kg/% TBSA burn Second 24 h: 2 lit 5% D
1968	**Parkland Formula**	First 24 h: RL at 4 mL/kg/% TBSA; give half in first 8 h and the remaining over next 16 h Second 24 h: colloid at 20-60% of calculated plasma volume (55% of blood volume) to maintain adequate urinary output
1974	**Muir and Barclay**	Colloids (mls) = % TBSA burns × body weight in kg/ 2 Time blocks- 4hrly for 1st 12 hrs, 6 hrly for next 12 hrs, after 12 hrs. A total of 36 hrs post injury. Crystalloid: (1.5ml x body weight) mL/hr Blood: 50ml/% BSA
1979	**Modified Brooke**	First 24 h: RL at 2 ml/kg/% TBSA burn, one half in the first 8 h and half in the remaining 16 h Second 24 h: colloid at 0.3-0.5 ml/kg/% TBSA burn + 5% D to maintain urine output.
1984	**Monafo formula**	First 24 h: saline or ringers lactate to target urine output of 30 mL/hr Second 24 h: one third of isotonic salt administered orally

increases. Due to the increase in pressure there is a decrease in blood supply resulting in damage to the muscles and nerves. This occurs with third spacing or extravasation of intravenous fluids. It also occurs with circumferential burns on the limbs or body. Children are at a higher risk for the compartment syndrome.

During resuscitation, careful monitoring should be maintained. Early recognition of development of compartmental syndrome is the key to management. Vigilant clinical monitoring is the most important factor for diagnosing any compartment syndrome. Limb compartment syndrome is treated by escharotomy or fasciotomy. Abdominal compartment syndrome is treated by surgical decompression and "leaving the abdomen open" (some form of protective dressing is used).

How are burn wounds taken care of?

Typically a burn wound has 3 layers. The centre is the zone of coagulation where irreversible damage has occurred. Around this is the zone of stasis, characterized by impaired blood flow. This is the area which can be salvaged by timely resuscitation. The outermost is the zone of hyperemia due to vasodilation and recovery of tissue in this zone is usually good.

It is important to remove the necrotic tissue by debridement; as the necrotic tissue acts as a culture medium for bacterial growth giving rise to infections. The wound after debridement should be covered with anti-bacterial ointments and emulsifications to keep it moist and resistant to bacterial invasion. Many biological (potato peel, amniotic membrane, etc.) and commercial dressing materials are available. This also prevents fluid loss from the wound and helps in early recovery.

What are the causes of Carbon monoxide (CO) poisoning?

CO is a colorless, odourless, toxic gas that is a product of incomplete combustion of carbon containing compounds like wood, coal and gasoline. The commonest cause of fire related deaths are due to smoke inhalation and CO poisoning. CO comes from both natural and manufactured sources. CO is produced in the human body as a by product of hemoglobin degradation. Baseline carboxyhemoglobin saturation is 1–3% in non-smokers and it increases to 10–15% in heavy smokers. Inhalation of combustible fumes produced by small gasoline engines, stoves, generators, lanterns, gas ranges or by burning charcoal and wood in the household environment is the largest source of CO poisoning. Tobacco smoke, automotive exhaust (drivers stuck in traffic), and occupational or industrial sources (steel foundries, paper mills) can lead to chronic CO poisoning. Other sources could be furnaces, gas powered engines, home water heaters, paint removers containing methylene chloride, pool heaters and smoke from all types of fire. Exposure to any fossil fuel can result in acute CO poisoning.

What is the mechanism of CO poisoning?

Carbon monoxide reduces the oxygen carrying capacity of blood resulting in tissue hypoxia. The PaO_2 is normal. CO binds avidly to other haem containing compounds, especially the cytochrome system. Possible mechanisms of toxicity include:

- Decrease in the oxygen carrying capacity of blood.
- Leftward shift of the oxyhemoglobin dissociation curve, further decreases oxygen delivery to the tissues.
- Binding with cytochrome A_3 results in decrease in cellular respiration.
- Binding to myoglobin, potentially causing myocardial and skeletal muscle dysfunction.

Incapacitation of the cellular mechanisms to utilise oxygen is one of the most important toxic action of CO which results in chemical asphyxiation and hypoxia.

Hemoglobin is a tetramer with 4 oxygen binding sites. CO binds with the oxygen binding sites as it has 250 times more affinity for hemoglobin than oxygen. As a result of this oxygen cannot bind with the red cells which results in histotoxic hypoxia. Carboxyhemoglobin causes a leftward shift of oxyhemoglobin dissociation curve due to which oxygen is not readily released to the tissues. CO also competes with oxygen for other haem proteins, for example myoglobin, peroxidases, catalases and cytochromes. It also binds with cytochrome oxidase in the cells due to which cells are unable to utilize the oxygen.

CO binds to cardiac and skeletal myoglobin as well. CO binds to Cardiac myoglobin three times more than skeletal myoglobin. Carboxymyoglobin dissociation is slower than COHb due to the increased affinity of CO for myoglobin. Fetal hemoglobin binds CO more avidly than hemoglobin A, and with slow transplacental transport, fetal levels decrease much more slowly than in the mother. This accounts for the occurrence of fetal death in nonfatal maternal exposures.

What are the symptoms of CO poisoning?

To function normally oxygen is required by the cells in the body; therefore all body systems are affected by CO poisoning. The heart, brain, and lungs are most susceptible to the effects of CO poisoning as they need a continuous supply of large amounts of oxygen for normal function. Early signs and symptoms are due to impairment of respiratory, cardiovascular, and neurologic systems. CO poisoning often mimics a viral illness with flu-like symptoms such as fatigue, lethargy, somnolence, malaise, nausea, vomiting,

and dizziness. Headache, nausea, and dizziness are the most common symptoms. Cardiac contractility is decreased due to binding of CO to cardiac myoglobin which further decreases tissue oxygen delivery (low-flow hypoxia). Due to persistent changes in the redox state of cytochrome systems, the clinical signs of CO poisoning may persist long after blood COHb has returned to normal.

Various other clinical effects are seen due to widespread ischemia (rhabdomyolysis, pulmonary edema, multiorgan failure, disseminated intravascular coagulation, dermal injury, and renal failure). Carbon monoxide exposure can also be made worse by comorbidities such as congestive heart failure, anaemia, drug ingestion, burns, trauma, and smoke inhalation.

The severity of symptoms depends on the COHb levels. Patient is usually asymptomatic if the levels are < 10%. As COHb increases above 20%, the patient may develop headache, dizziness, confusion and nausea. Coma and seizures due to cerebral edema are common with levels greater than 40%, and death is likely above 60%.

COHb%	Symptoms
10%	Asymptomatic or may have headaches
20%	Dizziness, nausea and vomiting, syncope
30%	Visual disturbances, fatigue
40%	Hallucinations and ataxia
50%	Seizures and coma
60%	Cardiopulmonary dysfunction and death

How would you treat a patient with CO poisoning?

Acute CO poisoning is treated by terminating the exposure by removing the patient from the exposed environment and by administering supportive care. The binding of CO with haemoglobin is competitive and reversible. Hence the mainstay of therapy for CO poisoning is supplemental O_2, ventilatory support and monitoring for cardiac arrhythmias. All patients should be given 15 L/min of oxygen via a non rebreather mask. The half-life of COHb is 250 mins at atmospheric pressure, 40–80 minutes while breathing 100% O_2 and 22 minutes in a hyperbaric chamber at 2.5 atm. Oxygen therapy should be continued because secondary peak of COHb occurs after 24 hrs due to the washout of CO from the cytochrome.

What is the role of hyperbaric oxygen therapy in CO poisoning.

- Hyperbaric oxygen (HBO) therapy reduces the half life of COHb to 20–30 minutes.
- It also increases the amount of oxygen dissolved in the blood from 0.3 mL/dL with isobaric therapy (FiO_2 100%) to 5.5–6.4 mL/dL (2.4–2.8 atm)
- HBO induces cerebral vasoconstriction, which may reduce intracranial pressure and cerebral edema, HBO result in more rapid dissociation of CO from respiratory cytochromes
- HBO may antagonize the oxidative injury that occurs after CO poisoning.

However the precise role of hyperbaric oxygen therapy is controversial and depends on the availability of hyperbaric facilities. Indications for considering hyperbaric oxygen therapy would be:

- Any neurological abnormality or cognitive impairment
- Seizure
- Hypotension
- Chest pain, abnormal ECG or elevated cardiac enzymes, arrhythmias
- COHb levels more than 40%
- Pregnancy with COHb level more than15%
- History of loss of consciousness or syncope
- Comatose patients.

Relative indications for HBO therapy include:

- Persistent neurologic symptoms (headache or dizziness) after 4 hours of high flow oxygen therapy via non-rebreathing mask.
- Persistent acidosis
- Concurrent chemical or thermal burns
- Pregnancy with history of any CO exposure.

Why do you get cyanide poisoning in burns patient?

Hydrogen cyanide is releazed as a product during the combustion of plastics and other polymers, silks, wool, cotton, and many other nitrogen-containing substances. Plastics and foams are increasingly being used in households as a result of which cyanide poisoning is likely in burns patients.

What are the causes of cyanide poisoning?

Cyanides come from a wide range of natural and manmade sources. Both gaseous and solid forms of cyanide is used extensively, often in very large quantities, in industries including metallurgy, electroplating, and metal cleaning. Cyanide also is used in the recovery of gold and silver from mineral ores and of silver from photographic materials; in the production of plastics, pigments, and dyes; and as a pesticide. Other sources of cyanide include emissions from production of iron and steel, coal burning, vehicle exhaust, and cigarette smoke. Combustion of substances like polyurethane, acrylonitrile, nylon, wool, and cotton can result in the production of hydrogen cyanide. Nitriles are used as solvents in the manufacturing of plastics. They can produce hydrogen cyanide when they are metabolized in the body after absorption through the skin or gastrointestinal tract.

Some drugs (e.g. sodium nitroprusside, benzylisothiocyanate and amygdalin [a fraudulent cancer remedy]) and plant constituents (e.g. those in cassava or Puerto Rican lima beans) can generate cyanide in the body when they are metabolized.

What is the pathophysiology of cyanide poisoning?

Cyanide causes tissue asphyxia by inhibiting intracellular cytochrome oxidase activity, the final step in oxidative phosphorylation, thus preventing mitochondrial oxygen consumption. Tricarboxylic acid cycle is also inhibited by cyanide poisoning. Affected cells can only generate adenosine triphosphate (ATP) via anaerobic metabolism. As a result there is lactic acidosis due to anaerobic conversion of pyruvate to lactate. Cyanide poisoning should be suspected in a burns patient if there is persistent lactic acidosis in spite of fluid resuscitation.

Cyanide poisoning can be difficult to diagnose. With a concentration of 50 parts per million (ppm), symptoms include headache, dizziness, tachycardia, and tachypnea. Above 100 ppm, lethargy, seizures, and respiratory failure occur. Possibility of cyanide poisoning should be suspected whenever there is a history of smoke inhalation. These patients have high anion gap acidosis which does not respond to O_2 administration. Plasma lactate levels are useful alternative diagnostic tool as they correlate with cyanide levels. In patients with cyanide poisoning the mixed venous O_2 saturation is high.

Cyanide is normally metabolized by hepatic rhodanase to thiocyanate, with thiosulphate as a substrate, but this process is slow. Administration of additional thiosulphate will speed up the hepatic metabolism of cyanide to produce non toxic thiocyanate which is the excreted in urine. Non-hepatic metabolic pathways to remove cyanide include the combination of cyanide with methemoglobin and hydroxycobalamin. Administration of Amyl sodium nitrite results in oxidation of hemoglobin to methemoglobin which combines with cyanide to form cyanomethemoglobin. Although methemoglobin does not transport oxygen, patients can tolerate methemoglobin levels of up to 40%, and this can be used to guide therapy.

What are the problems with pulse oximetry in patients with burns?

Burns patients can have CO poisoning which in turn increases the carboxyhemoglobin levels in the blood. Pulse oximeter may not be reliable indicator of tissue oxygenation these patients in the presence of carboxyhemoglobin, as it overestimates the oxygen saturation. A co-oximeter, which measures the percentage of hemoglobin, oxyhemoglobin, carboxyhemoglobin, and methemoglobin, is needed to obtain accurate oxygen saturation.

What are anesthetic considerations for excision and grafting of major burn injuries?

Preoperative Evaluation

History: Burns patient have a 48 hours period of initial stabilisation after which they are posted for burn wound excision and skin grafting. A complete medical and surgical history should be taken and a thorough physical examination should be done. A burns history would tell you about the possibility of inhalational injury.

Examination: In the pre anaesthetic evaluation the age and the % TBSA of burns will give an idea about the patient's physiologic condition. The adequacy of resuscitation should be assessed. In the airway assessment one should check the Mallampatti class, thyromental distance and head and neck mobility. The presence of facial burns can make mask ventilation difficult. Edema, scarring, or contracture formation may limit mouth opening and neck mobility. Traditional IV access sites may be unavailable.

Investigation: Blood grouping and cross-matching, complete blood count (CBC), blood glucose, serum electrolytes, serum urea and creatinine and arterial blood gas analysis to know COHb levels should be done. Chest X-ray should be done. Adequate blood should be kept ready as blood loss has been estimated to range from 200–300 mL/% of body surface excised and grafted. ECG has to be done in patients above 40 years of age or if they have associated medical conditions.

Preoperative optimisation/preparation: In major burns patients it is extremely difficult to achieve adequate nutritional status as they are in a hypercatabolic state. Therefore avoiding prolonged periods of fasting before the surgery would be beneficial. These patients are many times on analgesics for pain relief and should be continued through perioperative period. Depending on the extent of surgical excision adequate sized IV access should be taken.

Location of burns and donor skin sites indicate the need for special positioning, for repositioning the patient during operation, or both.

Monitoring: Intraoperative monitoring should include ECG, blood pressure, $ETCO_2$, pulse oximetry, core temperature, neuromuscular and urine output monitoring. Cutaneous burns may make placement of conventional monitoring devices such as ECG electrodes or blood pressure cuff difficult. Gel electrodes may not pick up ECG through damaged skin. Skin staples or subcutaneous needles attached to crocodile clips will give good signal. Invasive blood pressure monitoring would be preferable in patients requiring extensive debridement. It may be difficult to get the pulse oximetry trace due to peripheral burns or vasoconstriction.

Alternative sites like ear, nose or tongue can be used. Carboxyhemoglobin can give spurious results hence arterial gas analysis with carboxyhemoglobin levels should be done if there is any cause for concern. Because there is increased dead-space in inhalation injury, end-tidal CO_2 may not reflect $PaCO_2$. Arterial gas analysis should be used to monitor the ventilator settings. Central venous catheter may be needed in patients with extensive burns.

Temperature monitoring is essential because hypothermia is common and difficult to prevent. Patients with burns lose heat due to evaporation. Also the cutaneous vessels in the burnt area cannot constrict and prevent heat loss by radiation.

The operating room ambient temperature should be 28°C–32°C and all topical and intravenous fluids should be warmed. When possible, non-operative sites should be covered and forced air warming devices used.

Induction of anesthesia: General anaesthesia with the combination of an opioid, muscle relaxant, and a volatile agent is the most widely used technique for burn excision and grafting. Laryngeal mask airway has been shown to be effective in maintaining the airway in both adults and children with burns. Consider awake fibreoptic intubation if mouth opening is restricted. Tracheostomy is generally undesirable because of the risk that infection may spread to the damaged skin.

Various IV agents can be used successfully for induction. Ketamine has a lot of advantages. It not only provides stable hemodynamics, it also has analgesic properties. Hence, it has been used extensively as the primary agent for both general anesthesia and analgesia for burn dressing changes. Its major drawback is its tendency to produce dysphoric reactions. An antisialagogue premedication is a must. Diazepam or midazolam may be used to control the emergence hallucinations in some patients who receive ketamine. Etomidate can be an alternative to ketamine in hemodynamically-unstable patient. Thiopentone can be used as an induction agent in patients who are adequately volume resuscitated and hemodyanamically stable. Succinylcholine should be avoided 24 hours after burns injury.

Maintenance of anaesthesia: If laryngeal mask is used, the patient can be kept spontaneously breathing with O_2, nitrous and inhalational agent. The choice of volatile agent does not appear to influence outcome from anaesthesia for burn surgery, however, it must be kept in mind that these patients may require repeated anesthetics over a short period of time, hence use of halothane should be avoided to reduce the risk of halothane hepatitis. If patient has been intubated then non-depolarising muscle relaxants (atracurium or vecuronium) can be used for controlled ventilation.

As these patients routinely receive opioids for various procedures or routine pain management, they develop tolerance and may need large doses of opioids in perioperative period for pain relief.

They have increased minute ventilation requirements due to increased metabolic rate and parenteral hyperalimentation. In patients with more than 40% burns, the metabolic rate is doubled, resulting in increased oxygen consumption and CO_2 production. Patients with ARDS due to inhalational injury may be PEEP dependant.

Regional anaesthesia is useful in minor burns. However in major burns it is not very beneficial because skin may have to be harvested from extensive areas which may not be effectively covered by regional blockade. Also regional techniques should not be performed through burned tissue because of the potential for infection to spread. Epidural local anesthetic should be used cautiously in extensive debridements as there is a potential for massive blood loss and hypotension. However, epidural opioids can be safely used.

Blood loss during excision and grafting procedures can be minimised by using topical epinephrine. Application of bandages soaked in 1:10,000 adrenaline after excision of burned skin is effective in producing a bloodless surface for placement of skin grafts. After burn injury, cardiovascular responses to catecholamines are attenuated because of a reduced affinity of the B-adrenergic receptor for ligands and decreased second messenger production. This may explain the minimal changes in blood pressure and heart rate observed after topical administration of such high concentrations of epinephrine.

Patients would need large amounts of fluids intraoperatively which can cause considerable soft tissue edema. If the patient is prone or if there is significant facial edema after the operation then extubation should be delayed till the swelling subsides.

Postoperative care

Patient controlled analgesia can be used in postoperative period using various opioids. Non-opioid analgesics like NSAIDs or paracetamol can be considered depending on presence or absence of renal or liver dysfunction.

Can suxamethonium be used for muscle relaxation in a patient with burns?

Suxamethonium can be used in the first 24 hours to facilitate endotracheal intubation. After burns there is an increase

in the number of post-junctional receptors which causes prolonged depolarization and marked releaze of potassium. This process of receptor proliferation takes several days to develop, with an initial window of safety of unknown duration. The most dangerous period is thought to be between 4 days and 10 weeks after thermal injury. How long the hyperkalemic response to suxamethonium persists is also unclear. However, it is advisable that suxamethonium should be avoided for up to 2 years post burns. It should also be avoided in patients with extensive muscle damage as it can cause the releaze of large amounts of potassium into the circulation sufficient to cause cardiac arrest. Rocuronium may be an alternative to suxamethonium.

Discuss the problems with the use of non-depolarizing muscle relaxants in patients with burns.

Patients with thermal injury are resistant to the action of non-depolarizing muscle relaxants (NDMR). This resistance develops by 1 week and usually persists for 8 weeks. Marked resistance to NDMRs only occurs when the burn is >30% TBSA. Proliferation of the acetylcholine receptors is responsible for the resistance to NDMR. The resistance to NDMRs implies that the burned patient will require larger than normal doses of NDMR to achieve a desired effect and that the duration of action will be shorter than normal. Hence neuromuscular junction monitoring should be used to assess the adequacy of neuromuscular blockade as well as reversal with anticholinesterases.

How would you give anesthesia for burns dressing?

This entails administration of anesthesia/analgesia away from the well-equipped operating room. IV ketamine and titrated doses of opiates are good options. It is necessary to ensure empty stomach. Limb burns dressing may be managed under epidural analgesia or peripheral nerve blocks as appropriate.

A 25-years-old female patient with a history of burn injury to the face, hands and upper part of the body 1 year back is posted for release of neck contracture. She has limited neck mobility due to contractures in the neck. She also has microstomia due to facial scarring and there is severe scarring in both the upper limbs. How would you anesthetise this patient?

A complete medical and surgical history including a thorough examination of the patient would be required. Main concerns would be difficult airway in view of the limited neck mobility and microstomia. Due to the facial scarring it may not be possible to get a good mask seal, making mask ventilation difficult. Insertion of an LMA would be difficult due to microstomia. Blind nasotracheal intubation is unlikely to succeed if the neck movements are restricted or it is fixed in flexed position. Microstomia and distortion of the oral cavity make use of the LMA and oral fibreoptic intubation difficult. Retrograde intubation may not be possible because extensive scarring on the anterior surface of the neck which distorts the surface anatomy of larynx. So technique of choice would be an awake fibreoptic intubation, however, one has to check for patency of external nares.

IV access in the upper limbs will be difficult due to scarring. Since surgery does not require muscle relaxation, patient can be maintained on spontaneous ventilation using inhalational agents.

What are other options for releaze of neck contracture?

Tumescent local anesthesia can be used for release of neck contracture. It is a type of infiltration anesthesia where a large volume of local anesthetic with adrenaline is infiltrated into the subcutaneous tissue and after waiting period of 20 mins the neck contracture can be releazed. Some surgeons use it as a sole means of anesthesia. One formula uses 25 ml of 2% lidocaine and 1 ml of 1:1000 adrenaline for each liter of sodium lactate intravenous infusion. Dose of the local anaesthetic depends on the site and indication and large volumes ranging from 70–200 mL may be required depending on extent of contracture. Lignocaine doses of up to 35 mg/kg have been used without any side-effects. This is because dilution of lidocaine and epinephrine induced vasoconstriction diminishes and delays the peak plasma lidocaine concentrations, thereby reducing potential toxicity. Infiltrating a large volume of dilute epinephrine assures diffusion throughout the entire targeted area while avoiding tachycardia and hypertension. Clinical local anaesthesia can last as long as 18 hours, obviating the need for postoperative analgesia. If required analgesic doses of ketamine 0.5 mg/kg can be given. In children anesthetic doses of ketamine would have to be given at the beginning and the surgery can be carried out under ketamine and tumescent anaesthesia. Ketamine is preferred over thiopentone or propofol because it preserves the hypoxic ventilation responses and airway reflexes. O_2 can be supplemented with nasal prongs or face mask.

How is the pharmacology of drugs affected in burns?

Pharmacokinetic Effects: The pathophysiologic changes occurring after thermal injury alter pharmacokinetic parameters such as absorption, bioavailability, protein binding, volume of distribution, and clearance. The extent of these changes depends on the magnitude of injury and the time between injury and drug administration.

In the acute phase after a burn, organ blood flow is reduced because of hypovolemia and decreased cardiac output. Drugs administered by routes other than IV route are likely to show delayed absorption.

Plasma albumin concentrations decrease and α-1 acid glycoprotein levels increase. Plasma protein binding of albumin-bound drugs such as benzodiazepines is decreased, resulting in an increase in the free fraction and thus a larger volume of distribution for the drug. Raised fibrinogen and α_1-acid glycoprotein will reduce the free fraction of basic drugs (local anesthetics, propranolol, and muscle relaxants). The changes in the non protein bound fraction of the drug can alter the pharmacological response.

However, because most anesthetic drugs are not highly protein bound, and because the hemodynamic changes with burns are so marked, the effect of protein binding on the pharmacologic effects of anaesthetics is minimal. In addition, fluid loss to the burn wound and edema elsewhere can decrease plasma concentrations of many drugs below those expected in the unburned patient.

After the initial resuscitation phase, cardiac output increases as the hypermetabolic state develops. This increases blood flow to the kidneys and liver with increased drug clearance. However, there is wide patient-to-patient variability in renal and hepatic function after burns, so drug therapy must be tailored to each patient.

The volume of distribution of the drug may be altered by changes in protein binding and in extracellular fluid volume. Changes in the loading dose of the drug may be required if the drug has a small volume of distribution or a narrow therapeutic range. Total plasma clearance must be considered when considering drug maintenance doses and dosage intervals.

Pharmacodynamic changes: Changes in the drug-receptor interaction are common after burns and appear to account for many of the clinically important alterations in anesthetic pharmacology.

Burn injury causes proliferation of the extra-junctional receptors leading to resistance to nondepolarizing muscle relaxants and hypersensitivity to depolarising muscle relaxants. This effect may occur within a week of injury, persist for up to a year and is proportional to the total burns surface area.

Dose requirements for all anesthetic agents are increased due to hyperdynamic circulation and hypermetabolic state. MAC values are increased and duration of action is decreased.

These patients also develop tolerance to the effects of sedatives, analgesics and inotropic medications.

What is the mechanism of burn pain?

The skin consists of 3 layers namely the outer epidermis, inner dermis and hypodermis. The sensory structures are contained within the dermis and consist of free nerve endings (for pain, temperature and touch), Meissner's corpuscles (light discriminatory touch) and Pacinian corpuscles (pressure). Fibres from the free nerve endings travel in Aδ fibres (fast pain) and unmyelinated C fibres ('slow' and chronic pain) to synapse in the substantia gelatinosa of the dorsal horn of the spinal cord. Fibres then cross the midline of the spinal cord to ascend to the thalamus in the lateral spinothalamic tracts and from there to the post-central gyrus, where conscious perception of the stimulus may occur.

The instant pain that follows a burn injury is due to stimulation of skin nociceptors that respond to heat (thermoreceptors) and mechanical distortion (mechanoreceptors). Exogenous (e.g. hydrofluoric acid) and endogenous (e.g. inflammatory mediators, notably histamine, serotonin, bradykinin, leukotrienes and prostaglandins) chemical stimuli are also responsible for pain after burn injury. Nerve endings that are entirely destroyed will not transmit pain, but those that remain undamaged and exposed will generate pain. The immediate pain sensations are carried by the unmyelinated C and thinly myelinated Aδ fibres.

Subsequently, there is primary and secondary hyperalgesia.

Primary hyperalgesia: Following burns there is a massive inflammatory response resulting in release of inflammatory mediators which sensitizes the active nociceptors at the site of injury. Due to this the wound and the skin immediately adjacent to it becomes sensitive to mechanical stimuli such as touch, rubbing or debridement, and chemical stimuli such as antiseptics or other topical applications.

Secondary hyperalgesia: It is the increased sensitivity in the surrounding unburned areas resulting from continuous or repeated peripheral stimulation of nociceptive afferent fibres. This is mediated by the spinal cord and exacerbated by the mechanical stimulation that occurs as a result of frequent dressings changes.

Different sizes and degrees of burn can result in differing amounts of pain. Usually larger the burnt area more the pain and deeper the burn for a given area less is the pain (because there is greater destruction of nerve endings).

Discuss pain management in patients with burns? What analgesics are used?

Pain management is a problem in these patients. Intravenous morphine remains the gold standard for management of

pain in patients with burns who are not at a risk of airway obstruction. These patients often need multiple dressing changes and various operations as a result of which they develop a tolerance to opioids very rapidly and therefore may require large doses of opioids and use of other analgesics.

In the initial first aid cooling with tepid water reduces the pain. On admission to the hospital pain can be treated with titrated doses of IV morphine. Non-steroidal anti-inflammatory drugs (NSAIDs) can be used in patients with minor burns. They provide pain relief by inhibiting the formation of inflammatory mediators like prostaglandins releazed during burns. However, NSAIDs should be used with caution in patients with shock.

For postoperative analgesia multimodal analgesic technique combining paracetamol, NSAIDs, local anesthesia and opioids (IV morphine PCA) can be used. Local anesthetic solutions can be directly applied to skin donor sites. Although there is a theoretical risk of local anesthetic toxicity following unpredictable absorption over a large area studies have not demonstrated this to date.

Regional blocks may be used but there may be practical difficulties like infection close to the site of insertion, generalized sepsis and coagulation abnormalities.

How would you manage chronic pain after burn injury?

Neuropathic pain develops secondary to nerve damage, abnormalities in nerve regeneration and central nervous system reprogramming. This can destroy sleep pattern, result in depression and impair rehabilitation. There may be hyperalgesia (increased response to painful stimulus) and allodynia (painful response to normally innocuous stimulus) which can start early in the post-injury course and persist for many years after the initial injury. The severity of pain is related to the size of burn and the number of skin grafts performed.

This type of pain is very difficult to treat with conventional analgesics. Treatment includes antidepressants (amitriptyline), anticonvulsants (gabapentin and sodium valproate), regional nerve blocks and cognitive behavioural therapy.

How are electrical burns different from thermal burns?

Low voltage causes local contact burn. Domestic supply is 50Hz A.C., 240V which can cause cardiac arrest. Muscle spasm may prevent releaze of the electrical source. There is no associated deep tissue damage.

High voltage (more than 500V) causes flash burns or deep tissue damage due to current transmission. When electric current comes in contact with the patient's body the body acts as a conductor. The conversion of high voltage electric energy to thermal energy results in burns. The amount of thermal energy transferred depends on the voltage of the electrical source, skin resistance of the victim, duration of contact with the source of electrical current. The entry and exit burns are usually very small; however, there is massive underlying muscle damage. This is because the deeper tissues cannot dissipate the thermal energy as rapidly as more superficial tissues.

Patients with electrical burns need continuous ECG monitoring for at least the first 48 hours after burns as cardiac dysrhythmias are very common.

Respiratory arrest can also occur and may be due to respiratory muscle paralysis or tetanic contractions or some indirect trauma post injury.

Rhabdomyolysis can cause releaze of myoglobin which can cause acute renal failure. These patients should receive intravenous fluids to maintain a urine output of at least 1.5–2ml/kg/hour. Mannitol is recommended to preserve renal function once adequate fluid resuscitation has been achieved.

The affected limbs should be observed carefully as compartment syndrome may develop.

Neurologic complications are very common. They may result from direct injury or may occur later due to perineural scarring or neural ischemia.

Cataract formation is another sequel of electrical burn injury.

What indicates high-risk for arrhythmias after electrical injury?

The high-risk criteria for arrhythmias after electrical injury are:

- Abnormal ECG on presentation
- Loss of consciousness at time of electrical injury
- Exposure to high voltage (>240 volts)
- Past cardiac history
- Unwitnessed event
- Increased skin conduction, e.g. wet skin, high humidity
- Tetany at time of electrocution.

What is lightning electrical burn?

Lightning is an electrical burn of extremely high voltage (approx.10–120 million volts). Lightning strikes are rare and only 3–5% of these are direct injuries. Most of the times the lightning strikes inanimate objects like the ground, tree, or other objects that, once hit, transmit this energy to the victim.

Type of injuries: Only 1/3 of injured people have signs of burns and they too are usually superficial. The injuries are primarily neurological and affect all 3 components of the

nervous system: central, autonomic, and peripheral. Cardiac and respiratory arrests may also occur. Indirect injuries from falling, being thrown by muscle contractions, or barotrauma from the explosive force of a nearby lightning strike may occur.

How is chemical burn different from a flame burn?

Chemical burns can result from exposure to acids, alkalies or petroleum products. Industrial or household alkalis and acids are commonly used chemicals, e.g. bleach, washing powder, disinfectants, drain cleaner. Immersion in complex hydrocarbons (petrol, diesel) without ignition may cause systemic toxicity. Phosphorous burns can result from fireworks and military applications.

Hands and upper limbs are most frequently affected areas. Management is same as for flame burns with the exception that the burnt area should be irrigated with water for several hours. Irrigation helps to fully remove the corrosive substance because residual chemicals on the skin and clothing will allow the burning to continue.

Chemical burns are influenced by duration of contact, concentration of chemical and amount of the agent. Alkali burns are more serious than acid burns because alkalies penetrate more deeply. Contaminated clothing should be removed at the earliest. The chemical should be immediately flushed with large amounts of water using a shower or hose if available. Any dry powder on the skin should be brushed away before irrigation with water. Irrigation should be carried out for at least 20–30 minutes. Alkali burns to the eye requires continuous irrigation during first 8 hrs after burns. Irrigation should be continued till the pain stops.

Neutralizing agents do not have any advantage over water lavage, because reaction with the neutralizing agent may itself produce heat and cause further tissue damage.

Hydrofluoric acid used for etching in glass industry is highly toxic. Burns of 2% TBSA can be fatal as it can cause significant and life-threatening systemic hypocalcemia. First remove contaminated clothing and irrigate with water. Treatment includes topical 2.5% calcium gluconate burn gel.

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23 Cleft Lip and Cleft Palate

V Agarwal, A Chatterjee

A 2-year-old child comes with history of nasal regurgitation of fluid and food since birth. The patient has a nasal twang, delayed development of speech and has recurrent respiratory infections. The child was full term at birth and doesn't have any other significant medical history. This patient is posted for cleft lip and palate surgery.

Cleft lip and palate (CLP) is one of the most common congenital malformations, associated with difficulty in feeding, speech development and facial disfigurement. Surgery not only restores the function but also results in the defects being unrecognizable. Depending on the extent of the defect, different surgical manipulations may be required in various stages such as early orthopaedic manipulation of the dental arch followed by cleft lip repair, palate repair surgery for velopharyngeal incompetence and maxillary advancement at a later stage. These cases pose a challenge to the anaesthetist as they present at a very young age, may have associated congenital anomalies, varying degrees of difficult airway, need repeated corrective surgeries, and lastly the airway has to be shared with the surgeons.

What are the types of cleft lip (CL)?

The cleft lip can be classified anatomically by inspection.

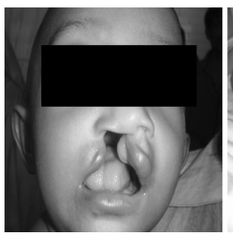
- **The cleft can be unilateral or bilateral.** Bilateral clefts are frequently associated with a deficiency of central columella and elongation of the vomer, which causes protrusion of the anterior aspect of the premaxillary process. In some cases, the premaxilla is completely absent.
- **It can be complete or incomplete.**
  - Complete CL – involves the entire lip going into the nostril
  - Incomplete CL – ranges from small to large defects- may be just a small gap or cleft in the vermillion, may slightly extend into the skin above the lip or extend almost to the nostril

What are the types of cleft palate?

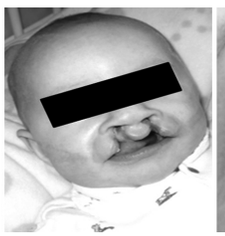
Cleft palate (CP) can be unilateral or bilateral fissure within the soft palate which may extend into the hard palate. CP may occur along with CL when the lip defect extends beyond the incisive foramen and includes the sutura palatina. Defects anterior to the incisive foramen are primary (prepalatal) clefts and posteriorly are secondary (postpalatal) clefts.

- **The primary cleft palate (prepalatal)** involves anterior palate, alveolus, lip, nostril floor, and ala nasi. They can be complete or incomplete.
- **Secondary cleft palate (postpalatal cleft)** is posterior to incisive foramen. They can be complete or incomplete depending on whether or not they extend all the way through the soft and hard palates to the incisive foramen.
- **Last type of palatal cleft is the submucosal cleft** in which a bone defect exists without a mucosal defect.

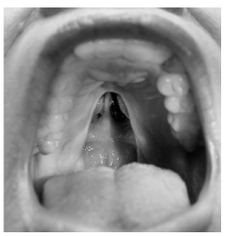
The most common cleft of the palate is a left complete cleft of prepalatal and postpalatal structures. The second most common is midline cleft involving the entire soft palate



Complete right-sided cleft lip and palate



Premaxilla



palatal cleft

and part of the hard palate without involving the prepalatal structures.

Embryology

During the first trimester, defects in the palatal growth leads to cleft lip and palate. The palate grows inwards to fuse in the midline in two stages. Initially by about 6 weeks the primary palate which forms the alveolus and lip fuses, weeks later to be followed by the secondary palate which is posterior to the incisive foramen. The suggested theories for failure of fusion include mechanical obstruction by tongue position, structural hypoplasia or primary breakdown.

What are the pathophysiological features of CLP?

- Cleft lip/palate results in difficulty in feeding since birth, suckling, swallowing may be impaired. The neonate/ infant may fatigue during feeding leading to inadequate weight gain and nutritional deficiencies and anaemia. Recent literature suggests that perioperative complications do not increase with mild degrees of anaemia (Hb 8–10 g/dL).
- Chronic upper respiratory tract infections are common in these patients secondary to regurgitation of fluids and food predisposing them to lower respiratory tract and ear infections. If the child at the time of surgery has acute upper respiratory tract infection it is wise to postpone the surgery for 2–3 weeks as the postoperative respiratory complications increase particularly with general endotracheal anesthesia.
- CLP may be associated with other congenital anomalies/ syndromes which may have their own symptomatology (see next question for details)
- CP leads to problems with phonation of the palatal consonants such as P, R, T and D, etc.

What other problems are associated with cleft lip and palate?

Up to 70% of patients with cleft lip/ palate have an isolated anomaly and are not associated with any syndrome. However isolated cleft palate may be associated with syndromes (see table 1 for some of the syndromes).

Anesthetic implications: Patients with syndromes listed in the table 1 may invariably have difficult airway (mask ventilation and/or intubation). Pierre Robin Sequence and Treacher Collins Syndrome and Down's syndrome children in addition have predisposition to postoperative respiratory obstruction. Syndromes like Down syndrome, Stickler Syndrome, Klippel-Feil syndrome that have high incidence of cardiac involvement will also require infective endocarditis prophylaxis.

Table 23.1 Other syndromes associated with cleft lip and palate

Syndrome	Associated anomalies
Pierre Robin Sequence	80% associated with cleft palate Micrognathia Glossoptosis Underlying syndrome/anomalies
Treacher Collins Syndrome	Around 1/4th children have associated cleft palate Micrognathia and maxillary hypoplasia Eye and ear malformations Choanal atresia
Hemifacial Microsomia (Goldenhar syndrome)	Hemifacial and mandibular hypoplasia Ear and eye abnormalities Cervical spine abnormalities
Velocardiofacial Syndrome	Microcephaly and microstomia Flat nasal bridge, small ears, short stature Immune deficiency Congenital cardiac disease, Laryngeal and tracheal anomalies Velopharyngeal incompetence with/ without cleft palate 22q 11 deletion (FISH test)
Stickler Syndrome	Micrognathia and flat face Eye, ear and joint abnormalities Congenital cardiac disease Progressive connective tissue disorder
Down Syndrome	Microstomia and relative macroglossia Atlantoaxial subluxation and instability Epicanthic folds, simian crease Congenital cardiac disease
Fetal Alcohol Syndrome	Smooth philtrum, thin vermillion Small palpebral fissures Growth retardation CNS abnormalities
Klippel-Feil syndrome	15% associated with cleft palate Congenital cardiac disease Short, webbed neck and fused cervical vertebrae Renal anomalies Scoliosis

What is VATER or VACTREL association?

It is a group of anomalies which often occur together. To qualify a VACTERL child they must have at least 3 of the following anomalies. In addition to these anomalies they may have other characteristics which occur more frequently in affected children than the rest of the population. These are ear abnormalities, genital anomalies, cleft lip and/or palate, thumb abnormalities and presence of a single artery in the umbilical cord

- V – Vertebral anomalies
- A – Anal atresia
- C – Cardiovascular anomalies
- T – Tracheoesophageal fistula
- E – Esophageal atresia

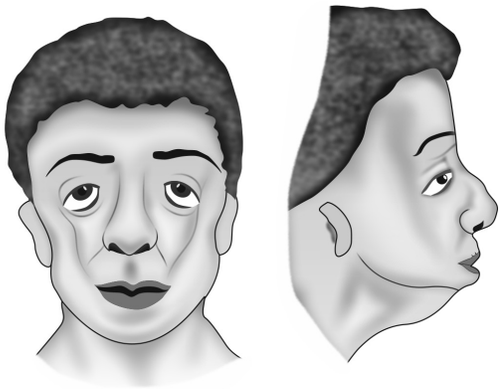
- R - Renal (Kidney) and/or radial anomalies
- L - Limb anomalies (in front of or above the central axis of the limb).

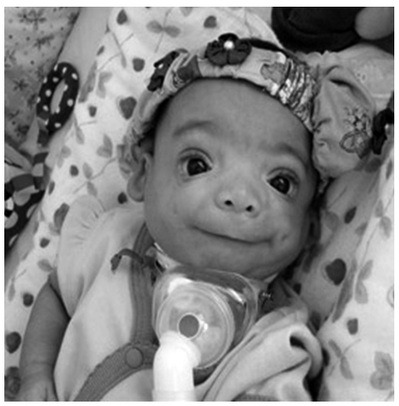
When is the appropriate time to undergo surgery?

- The common time for surgical repair of a cleft lip is 3 months after birth; however, isolated cleft lip without any other associated conditions can be closed as early as the first week of life. This makes suckling easier.
- The hard palate can be repaired up 4 to 5 years of age in various stages but soft palate should be closed prior to speech development, i.e. between 12 to 15 months of age.
- These children may require a secondary procedure in later years for cosmetic purposes. If child has a cleft lip and palate, the lip is repaired first because the repaired lip helps to decrease the width of palate defect.

Fetal surgery to correct the cleft lip with minimal or no scarring is in the experimental stages.

What is Treacher Collin's syndrome?



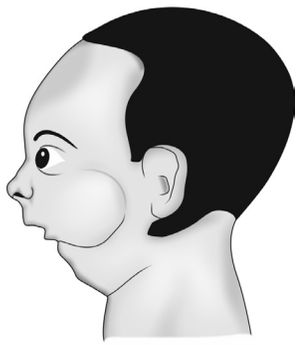


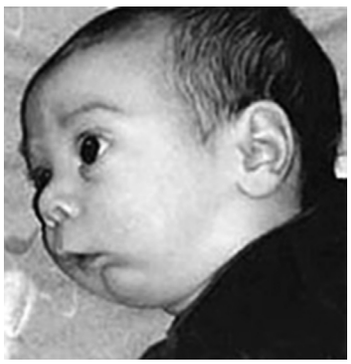
Treacher Collins Syndrome, also known as mandibulofacial dysostosis, causes bilateral and symmetric deformities of head and face. It is an autosomal dominant disorder and has a prevalence of 1 per 25,000 to 1 in 50,000 live births. Both sexes are equally affected. It occurs due to genetic mutation in chromosome 5, which affects facial development.

Characteristics include:

- Palpebral fissures – slanting downwards
- Notched lower eyelids
- Underdevelopment or absence of maxilla and lateral wall and floor of the orbit
- Retrognathia and posteriorly positioned tongue (creating an impression of a large tongue due to reduced pharyngeal space) – difficult airway and predisposing to postoperative airway obstruction. Relief of obstruction may need lateral or prone positioning to maintain airway. Rarely a tracheostomy may be needed
- Ear abnormalities – hearing loss
- Cleft palate in one-third of the patients, palatopharyngeal incompetence may be present in additional one-third of the patients leading to difficulty in feeding and swallowing.

What is difference between Pierre Robin Sequence and Syndrome?





A 'syndrome' describes a condition for which the etiology is the same between individuals (e.g. Down Syndrome is always caused by trisomy 21), whereas 'sequence' is used to describe a clinical pattern resulting from a single initiating anomaly (e.g. micrognathia) of multiple causes.

The triad of micrognathia, cleft palate, and retroglossoptosis was known as **Pierre Robin syndrome** until 1974. The term **sequence** was recently introduced to include any condition that consists of a sequence of events initiated by a single malformation. Since the most accepted theory is the mechanical theory which states that initiating event is mandibular hypoplasia which keeps the tongue high within the oral cavity; this in turn causes a cleft palate by preventing the closure of the palatal shelves, this syndrome is now called

as **Pierre Robin sequence.** Another proposed theory is that, mandibular growth may be restricted mechanically in utero by oligohydramnios or due to abnormal development of the temperomandibular joint. It has a prevalence of approximately 1 per 8500 live births. Equally affects both sexes, except in the X-linked form. An autosomal recessive inheritance is possible.

Characteristics include:

- Micrognathia
- Glossoptosis—both may cause severe breathing feeding difficulty in the newborn. Also, obstructive sleep apnea may occur
- Cleft palate
- Otitis media occurs most commonly. There may be conductive hearing loss
- Speech development may be affected. Velopharyngeal insufficiency is usually more pronounced in these patients than in those with isolated
- Cardiac defects may include benign murmurs, pulmonary stenosis, patent ductus arteriosus, patent foramen ovale, atrial septal defect
- Musculoskeletal anomalies
- Other associated syndromes and conditions include Stickler syndrome, trisomy 18 syndrome, Velocardiofacial syndrome.

When managing a patient with CLP, even the ones diagnosed as 'Pierre Robin Syndrome or Sequence', it is essential to have a high index of suspicion for other underlying problems or syndromes.

What are the anesthetic considerations for this child with left-sided CLP undergoing surgery?

Key concerns dominating the preoperative assessment of an infant posted for cleft lip/palate surgery are:

1. Pediatric patient.
2. Assessment and evaluation of associated congenital birth defects or a syndrome if present.
3. Possibility of a difficult intubation.
4. Shared airway.

Preoperative assessment

History

In addition to the usual information elicited (developmental milestones and vaccination history) during the preoperative interview particular attention should be given to the following:

- Patients with a cleft lip and palate are at risk of recurrent upper airway and ear infection secondary to regurgitation of fluid and food through the cleft, it is essential to rule out any upper respiratory infection to avoid postoperative complications. The risk of postoperative respiratory tract infection increases particularly in patients with greater severity of defect (e.g. a child with bilateral CLP has a significantly higher risk compared to another with unilateral CLP, 9% versus 2% respectively) and requiring general anesthesia. In some cases, it is advisable to defer the surgery for 2–3 weeks until symptoms improve and the airway reactivity decreases. It is also important to note that following surgery the risks of postoperative respiratory tract infection decreases, so the case should be individually assessed regarding the risks of postoperative respiratory infection and benefits of surgery.
- These children may have associated congenital heart defects (ASD, VSD, Mitral valve prolapse, some may have cyanotic congenital heart disease). Most of them would have undergone cardiology evaluation, and necessary information can be sought. If the child has not undergone cardiology evaluation a through evaluation of the cardiovascular system is essential looking for murmurs.
- If there are associated syndromes, the pathophysiology of the syndromes should be thoroughly understood before administering anesthesia.
- Enquire about breathing or feeding difficulties since birth, how they were managed and whether they have resolved.

Examination

- They should be examined for signs of retrognathia.
- The methods to evaluate airway in adults are not useful or have several limitations when applied to these cases. The infant/child may not co-operate, no specific thyromental distance or any other tests have been validated as a predictor for difficult intubation.

Investigations

- Full blood count – to look for anaemia, and evidence of infection.
- Echocardiogram – if there is a murmur detected on clinical examination or if there is associated anomaly to assess the structure of the heart.

Preoperative preparation

- Blood should be grouped and cross matched as palatal surgery may bleed, particularly so in patients with anemia.
- Antibiotic prophylaxis administered if there is co-existing congenital heart disease.
- Sedative premedication should be avoided if difficult airway is suspected or anticipated. Premedication with oral atropine may be done to decrease secretions and the possibility of laryngeal spasm. Atropine administered

orally in a dose of 0.02 mg/kg (maximum dose 0.6 mg) 45 minutes prior to induction is effective.
- Pediatric difficult airway kit should be made available if anticipating difficult airway.

Anaesthetic management

Induction of Anesthesia

- Monitoring include ECG, NIBP, pulse oximetry, end tidal capnometry, temperature and precordial stethoscope. Inhalational induction with sevoflurane is the choice in patients with anticipated difficult airway. Nitrous oxide may be administered in addition to sevoflurane to speed the induction.
- In patients without any airway problems/difficulty, intravenous induction with either thiopentone or propofol and muscle relaxant can be done.
- During laryngoscopy if a large cleft is present on the left, a lubricated dental role or gauze can be placed within the gap to prevent trauma to the underlying tissue. Alternatively the laryngoscope can be used to approach from the side rather than the midline.
- The trachea is preferably intubated with preformed oral endotracheal tube (RAE tube, South Pole tube). Alternatively an oxford tube can be used. The endotracheal tube rests in the midline on the tongue underneath the tongue plate of the Dingman mouth gag. It is secured in the midline over the chin.
- A throat pack is usually inserted by the surgeon to prevent aspiration of blood.
- The endotracheal tube position should be checked for any misplacement and obstruction after the patient is positioned and following the insertion and opening of a mouth gag. The ventilation should be monitored carefully during the placement of the mouth gag for any compression or displacement leading to increased airway pressure or leak.
- Once the airway is secured ventilation is controlled to maintain normocapnea.
- Antibiotic prophylaxis against staphylococcus should be given following induction.
- Surgeons will often use adrenaline infiltration to reduce bleeding, the anesthetist must make sure that the concentration used is 1:2,00,000 and the appropriate amount is used (rule of the thumb 1 mL/kg).

Maintenance of Anesthesia

- Anesthesia can be maintained with nitrous oxide, inhalational agent such as isoflurane, fentanyl and muscle relaxant such as vecuronium or atracurium.
- Blood loss should be assessed and blood should be transfused if necessary.
- Fluid warmer and *convective* warming blanket should be used to prevent hypothermia.
- At the end of the procedure, the throat pack should be removed and oral cavity should be examined for swelling of the tongue, uvula and any evidence of bleeding. A gentle laryngoscopic examination and suction should be done. If there is any evidence of swelling of the tongue or uvula, extubation should be postponed and these patients should be electively ventilated till the swelling resolves, especially following a prolonged surgery.
- Neuromuscular blockade should be reversed with neostigmine and glycopyrrolate.
- The child should be extubated when awake and has full control of airway reflexes.
- Postoperative analgesia can be achieved by using infraorbital nerve block and administering systemic analgesics, before the effect of block wears off. Diclofenac suppository (1 mg/kg, 12 hourly) is commonly used. A loading dose of paracetamol 30 mg/kg rectally may be administered post induction. Infraorbital nerve block can be administered following induction of anaesthesia or after CL surgery.

Postoperative Care

In patients with CP following surgery the size of the airway decreases, this can predispose them to postoperative airway obstruction at the time of extubation.

What are the causes of postoperative airway obstruction?

Causes include:

1. After palatal surgery, reduction in the size of the airway
2. Edema of the tongue due to pressure from the mouth gag.
3. Residual effect of anesthesia and/or opioids.
4. Laryngeal edema following endotracheal intubation.

All patients at risk of delayed airway obstruction should monitored closely in the postoperative period.

What are preoperative fasting guidelines for paediatric patients?

Children can safely be allowed clear fluids 2 hours before surgery without increasing the risk of aspiration. Food should normally be withheld for 6 hours prior to surgery in children aged 6 months or older. In children under 6 months of age it is probably safe to allow breast milk but not formula or non-human milk up to 4 hours before surgery.

Fig. 23.2 American Society of Anesthesiologists Fasting Guidelines[7].

Clear liquids (E.g. include but are not limited to water, fruit juices without pulp, carbonated beverages, clear tea and black coffee. Does not include alcohol)	2 hr
Breast milk	4 hr
Infant formula	6 hr
Non-human milk	6 hr
Light meal (Toast without butter and clear liquid)	6 hr
Full meal (Fried/fatty meal)	> 8 hr

How do you replace perioperative fluids in pediatric patients?

- Hypovolemia should be corrected rapidly to maintain cardiac output and organ perfusion
- In a child, the fall in blood pressure is a late sign of hypovolemia
- Maintenance fluid requirements should be calculated using the formula of Holliday and Segar
- Body weight daily fluid requirement
- 0–10kg: 4 mL/kg/hr
- 10–20 kg: 40 mL/hr + 2 mL/kg/hr above 10 kg
- >20 kg: 60 mL/hr + 1 mL/kg/hr above 20 kg
- Fluid and dextrose management during surgery: A fluid management plan for any child should address 3 key issues
  - Any fluid deficit which is present
  - Maintenance fluid requirements
  - Any losses due to surgery, e.g. blood loss, 3rd space losses.

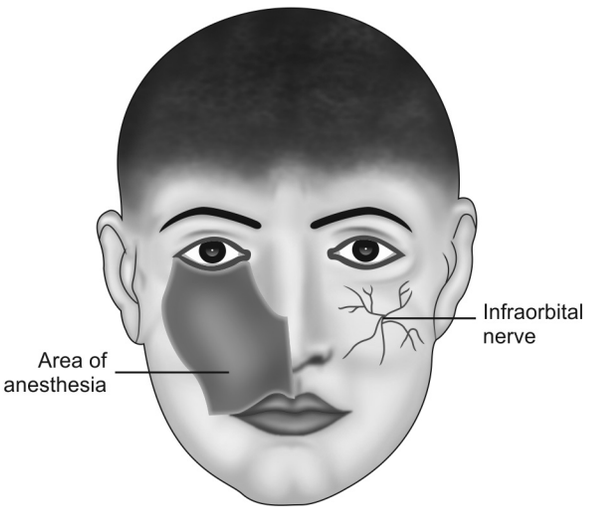
During surgery all these requirements should be managed by giving isotonic fluid – such as 0.9% sodium chloride or Ringer lactate/Hartmann's solution, in all children over 1 month of age.

1. During surgery the majority of children may be given fluids without dextrose. Blood glucose should be monitored if no dextrose is given.
2. Neonates in the first 48 hours of life should be given dextrose during surgery. Preterm and term infants already receiving dextrose containing solutions should continue with them during surgery.
3. Infants and children on parenteral nutrition preoperatively should continue to receive parenteral nutrition during surgery or change to a dextrose containing maintenance fluid and blood glucose monitored during surgery.
4. Children of low body weight (less than 3rd centile) or having prolonged surgery should receive a dextrose containing maintenance fluid (1–2.5% dextrose) or have their blood glucose monitored during surgery.
5. Children having extensive regional anesthesia with a reduced stress response should receive a dextrose containing maintenance fluid (1–2.5% dextrose) or have their blood glucose monitored during surgery.
   - Blood loss during surgery should be replaced initially with crystalloid or colloid, and then with blood once the haematocrit has fallen to 25%. Children with cyanotic congenital heart disease and neonates may need a higher hematocrit to maintain oxygenation.
   - Dehydration without signs of hypovolaemia should be corrected slowly.
   - In the postoperative period ongoing losses from drains or nasogastric tubes should be replaced with an isotonic fluid such as 0.9% sodium chloride with or without potassium chloride.
   - Fluid therapy should be monitored by daily electrolyte estimation, use of a fluid input/output chart and daily weighing if feasible.

(APA Consensus Guideline on Perioperative Fluid Management in Children[8])

How will you manage the pain postoperatively in patients with cleft lip repair? or Can you describe the infraorbital nerve block?

The infraorbital nerve is 1 cm inferior to the infraorbital ridge and in the midpupillary line (vertical line passing through the supra orbital foramen, pupil, the infraorbital and the mental foramina). Infraorbital nerve is a branch of the maxillary division of the trigeminal nerve. The infraorbital nerve innervates the upper lip, medial side of the cheek, lateral aspect of nose and lower eyelid.



Infraorbital nerve block

It can be performed in 2 different ways:

- Direct cutaneous injection
- The infraorbital foramen should be palpated, and approximately 1–2 mL of 0.25 or 0.5% bupivacaine is injected near to surround the nerve, but not in the foramen.
- Intraoral injection.
- During palpation of the foramen with the non dominant hand, the needle is inserted into the superior labial sulcus at the apex of the canine fossa. Approximately 1–2 mL of 0.25 or 0.5% bupivacaine is injected around the infraorbital foramen.

  Supplemental systemic analgesics as mentioned earlier.

If this patient with repaired CLP presents for a non cleft surgery (tonsillectomy), what are the anesthetic implications?

Detailed evaluation should be done as mentioned earlier. Important considerations relevant to this case are mentioned below:

- The child may have received multiple anesthetics before, it is important to go through the notes and retrieve any relevant information with regards to difficult airway, co-existing conditions, any problems faced in the postoperative period.
- It is important to elicit the nature of surgery which was performed for, e.g. pharyngoplasty. This operation reduces the size of the space between the soft palate and pharyngeal wall. This predisposes them to obstructive sleep apnea. One must enquire about snoring.
- Nasotracheal intubation should be avoided in these cases. If a nasotracheal intubation is anticipated, it is safer to do nasophaaryngoscopy to evaluate the nasopharynx.

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24 Geriatric Patient

R Gehdoo, A Kothekar

Medical advances in the recent years have increased the life expectancy of the people over the past century. As the population ages, more surgical procedures will be performed on these patients. Although many older adults are quite active and continue to work, they have limited physiological reserve whereas many others have multiple disease processes and are at an increased risk over the general population. This makes it essential to develop a better understanding and an increased expertise in caring for the geriatric surgical patient.

What are the physiological changes due to Aging?

I. Cardiovascular and Autonomic Nervous System:

Aging produces a decrease in arterial elasticity. There is hyperplasia of intima, collagenization of media and accumulation of calcium and phosphate in elastic fibers of vessels leading to narrowing of arteries and veins. Decrease in arterial compliance results in increased systemic vascular resistance, increase in afterload and elevated systolic blood pressure.

- The systolic blood pressure rises roughly 6 to 7 mm Hg per decade, whereas the diastolic pressure changes very little with aging and may actually fall.
- Calcification of valves (Aortic valve sclerosis and mitral annular calcification)—Presence of aortic valve sclerosis in elderly increases risk of cardiovascular death by 50%. In fact aortic valve sclerosis has been suggested as an indicator of coronary artery disease.
- Increased interstitial fibrosis of myocardium leads to diastolic dysfunctions and impaired diastolic filling.
  - Greater dependence on LA contraction for LV filling leads to high atrial pressures which in turn gives rise to pulmonary congestion.
- Geriatric patients experience decreases in maximal heart rate, stroke volume, cardiac output, ejection fraction, and oxygen consumption in response to stress.
- There is decrease in compliance of venous system that hinders response to changes in the intravascular volume as experienced during position changes, third space losses, or hemorrhage.
- With advanced age, there is decrease in heart's inotropic and chronotropic response to endogenous and exogenous catecholamines. In addition, the cardiac conduction system undergoes fibrotic changes, causing a loss of sinoatrial node cells, making them more prone to dysrhythmias.
- Cardiac output linearly decreases after 3rd decade of life at the rate of 1% per year in normal subjects who do not have underlying cardiac disease. Thus 80-year-old will have cardiac output half that of 20-year-old

All above factors cause the elderly patient less capable of defending his or her cardiac output and blood pressure against the usual perioperative challenges. In addition, atherosclerosis may convert a moderate degree of hypotension into an intolerable reduction in cardiac, cerebral or renal blood flow.

II. Respiratory System:

- Decline in function of pharyngeal and laryngeal structures due to loss of muscular pharyngeal support predisposing the elderly to upper airway obstruction.
- Increased incidence of periodic breathing during sleep (Obstructive Sleep Apnea), which makes them more likely to have apnea and airway obstruction in the recovery room.

- Protective reflexes involved in the regulation of coughing and swallowing are diminished. Also there is loss of ciliary function. Both these factors combined result in repeated aspirations and chronic pulmonary inflammation.
- The thorax changes shape with age. Kyphosis of the thoracic spine is the first change, beginning around the age of 50 in females and a decade later in males, and is due principally to osteoporotic vertebral collapse. This leads to increase in antero-posterior diameter of the chest at the expense of the lateral diameter, and eventually leads to the 'barrel-chest' deformity.
- There is flattening of diaphragm due to barrel-chest deformation. Such flattened diaphragm becomes less efficient mechanically. Loss of muscle mass reduces this efficiency further.
- Costochondral joints get calcified making thorax less compliant and thus increase work of breathing.
- Since airways lose elastic support, they become more collapsible and larger numbers of airways close during expiration, particularly in the lung bases. This probably accounts for the very low flows seen in older subjects at the end of expiration. This premature airway closure leads to mismatch of ventilation and perfusion, and eventually to arterial desaturation.
- The volume of the pulmonary capillary bed also decline with age resulting in a marked increase in the mean pulmonary artery pressure by 30%, and an increase of the pulmonary vascular resistance by up to 80%.
- There is a small but measurable increase in dead space, decrease in tidal volume, and increase respiratory rate.
- Vital capacity declines progressively with age by 20 to 30 mL and Residual volume (RV) increases by 10 to 20 mL/yr from age 20.
- Ratio of RV to total lung capacity (TLC) increases from 25% at 20 years of age to about 40% in a 70-year-old man. Also the ratio of FRC to TLC increases with age due to loss in TLC, and very modest increase in FRC.
- There is an increase in the closing volume (CV) and closing capacity (CC).
- The reduction in motor power of the accessory muscles of breathing as well as the decreased expansion of the chest wall causes the dynamic lung volumes and capacities to decrease progressively with age, e.g. fall in FEV_1 by 30 mL per year. With increasing age the FEV1/FVC ratio may be as low as 65 to 55% in apparently healthy individuals.
- The increased V/Q mismatch plus the increased alveolar dead space adversely affect the aged patient's blood gas values. Mean PaO_2 declines from 95 ± 2 mm Hg at age 20 to 73 ± 5 at age 75 years. This decline in arterial oxygen tension is modest; approximately 0.4 mm Hg/year. After age 75, however, PaO_2 stays relatively constant at approximately 73 mm Hg.
- The increasingly rigid pulmonary vasculature blunts the hypoxic pulmonary vasoconstrictor (HPV) reflex. Thus the ability of the aged lung to respond to altered ventilation/perfusion ratio is compromised. This effect is more highlighted in one lung anesthesia.
- The ventilatory response to hypercapnia and hypoxia is blunted in the elderly patient due to reduced neuronal output to respiratory muscles (ventilatory drive) making them sensitive to respiratory depressant effects of opioids and benzodiazepines.
- Overall, the age-related changes of the respiratory system essentially consist of a combination of restrictive and obstructive lung diseases.

III. Central Nervous System:

- The brain size decreases by about 20% beyond 80 years of age.
- The cerebral blood flow and cerebral oxygen consumption ($CMRO_2$) decreases in proportion to the decrease in brain mass.
- However, the cerebral autoregulation is well maintained in geriatric patients without prior neurological disease.
- There is a continual loss of neuronal substance. The majority of the neural tissue losses occur among those that synthesize neurotransmitters, causing depletions of dopamine, norepinephrine, tyrosine, serotonin, and perhaps other neurotransmitters. However, there is increased activity of catabolic neurotransmitter enzymes such as monoamine oxidase and catechol o-methyltransferase.
- The association of serotonergic, cholinergic and dopaminergic systems, respectively with mood, memory, and motor function, may partially account for depression, loss of memory and motor dysfunction in the elderly.
- *Sleep:* Increased latency to sleep onset is often present, as well as increased awakenings and periods of wakefulness during the night.
- *Memory:* Memory and reasoning performance decline linearly with advancing age.
- *Age-related CNS diseases:* Age-related diseases such as cerebral arteriosclerosis, Alzheimer's and Parkinson's disease are all more common with advancing age.

IV. Peripheral Nervous System and Neuromuscular Function:

Aging produces a generalized increase in the thresholds for virtually all forms of perception, including vision, hearing,

touch, sense of joint position, smell, and peripheral pain and temperature responses. These are due to:

- Simultaneous attrition of afferent conduction pathways in the peripheral nervous system and spinal cord
- Reduced velocity and amplitude of electrical transmission
- Deterioration of electrical conduction along motor pathways
- Decreased velocity of peripheral motor nerve conduction.

Motor neuron degeneration produces the disseminated neurogenic atrophy responsible for age-related changes at the neuromuscular junction. Thickening of the post-junctional membrane and its spread beyond the usual end-plate areas because of age-related deficiency of cyclic GMP generate atypical 'extrajunctional' cholinergic receptors. The increase in the number of cholinergic receptors at the end plate and surrounding areas might compensate for the age-related decline in the number and density of motor end-plate units. Therefore, despite loss of skeletal muscle mass, dose requirements for competitive neuromuscular blocking drugs are not reduced, and, in fact, are frequently slightly increased. The increased sensitivity to succinylcholine seen in some elderly men is due to reduced plasma cholinesterase enzyme concentrations and not to changes at the neuromuscular junction itself.

V. Autonomic nervous system:

- Decreased sensitivity of baroreceptor leading to orthostatic hypotension and syncope.
- Marked reduction in autonomic end-organ responsiveness associated with aging, like ability of β-adrenergic agonists to enhance the velocity and force of cardiac contraction or to increase the rate of electrical discharge of excitable tissues (endogenous β-blockade of aging) due to decreased number of receptors, reduced affinity for agonist molecules, and impairment of adenylate cyclase.
- Thermoregulation is affected by autonomic impairment, as well as changes to the skin and blood vessels leading to increased heat loss and reduced heat tolerance. As a consequence, the elderly are vulnerable to heat stroke and hypothermia.

VI. Renal System

- There is a progressive reduction of renal mass with ageing (30% by 80 years of age) due to glomerulosclerosis, paralleled by thickening of the vascular intima, and chronic infiltration by inflammatory cells. This results in a decline in renal plasma flow and glomerular filtration rate (GFR).
- Microscopically there is a reduction in the number of functional glomeruli.
- Serum creatinine levels remain relatively normal due to a concomitant decrease in muscle mass. Therefore, serum creatinine levels are poor indicators of glomerular filtration rates in the geriatric population.
- Cockroft Gout formula can be used to estimate GFR.
- GFR = (140-age) × Wt/72 × S. Creatinine
- Thus for a 68-year-old weighing 60 kg with serum creatinine 1.2, estimated GFR is 50 mL.
- Geriatric patients have reduced abilities to concentrate urine or conserve sodium.
- Loss of lean body mass also reduces total body stores of exchangeable potassium and predisposes to iatrogenic hypokalemia. Elderly tolerate hypokalemia poorly owing to age related myocardial and conduction tissue dysfunctions. Thus, fluid and electrolyte status should be carefully monitored in the elderly patient.
- This reduced blood flow and loss of the total available mass of nephron units in the elderly also delay the drug clearance and prolong the clinical effects of drugs, which require final elimination by renal pathways.
- Renal vascularity is reduced and the cardiac output is redistributed, predisposing the geriatric population to renal ischemia in the perianesthetic period. One fifth of geriatric perianesthetic surgical mortality is due to acute renal failure.

VII. Gastrointestinal System:

The gastrointestinal system of the geriatric patient may be characterized by gastric acidity, decreased colon motility and anal function, constipation, fecal impaction, fecal incontinence, osteoporosis, or vitamin B_{12} deficiency. Albumin level, a marker of nutritional status, has been identified as a predictor of postoperative mortality in the elderly surgical patient.

VIII. Hepatic System:

- Liver tissue decreases approximately 40% by 80 years of age which leads to decreased hepatic function.
- There is a lack of correlation between structural and functional data concerning the ageing liver, as a decline in organ volume does not necessarily reflect impaired metabolic function.
- Hepatic tissue loss leads to delayed drug metabolism and earlier saturation of metabolic pathways.

IX. Protein Binding:

- The circulating level of serum protein, especially albumin decreases, reducing available protein-binding sites for a variety of drugs.
- Qualitative changes may occur in circulating protein that reduces the binding effectiveness of the available protein.
- Thus, drugs that are highly protein bound should be delivered to an elderly individual with the expectation

that reduced protein binding will lead to higher free drug levels.

X. Body Compartments:

- Loss of skeletal muscle (decrease in lean body mass).
- Decrease in total body water mainly due to decrease in intracellular water.
- Plasma volume, red cell mass and extracellular fluid volumes are well-maintained in non-hypertensive elderly individuals with adequate levels of physical activity.
- Increase in percentage of body fat
  - The increase in percentage of body fat acts as a reservoir for deposition of lipid-soluble anaesthetic drugs, increasing the elimination time and prolonged recovery time.

XI. Musculoskeletal System:

Decreased muscle mass, bone density and lubrication of the joints cause stiffness of the joints, osteoporosis, frequent fractures of the hip and functional impairment of bones and joints. Extra care and attention must be given to positioning and padding the patient because of fragile skin, decreased subcutaneous fat, and poor skin turgor. In addition, age-related changes in the musculoskeletal system and limitations often imposed by chronic pain in the elderly make patient positioning an extremely important aspect of care.

XII. Endocrine System:

- Glucose intolerance secondary to a progressive impairment of insulin function or subtle antagonism of its effect on the target tissues.
- Decreased activity of the endocrine system, causing impaired glucose homeostasis, decreased thyroxin clearance or production, decreased production of renin, aldosterone and testosterone, decreased vitamin D absorption and activation and increased plasma concentration of antidiuretic hormone.
- The consequences of all of these changes include the development of diabetes mellitus, thyroid dysfunction, decreased sodium retention, increased potassium absorption, and osteoporosis which causes bone fractures.

XIII. Metabolism:

- Basal resting metabolic activity decreases in direct proportion to the age-related decrease in lean tissue mass
- Impaired thermoregulation. Also time required for spontaneous rewarming postoperatively appears to increase in direct proportion to patient's age. Therefore, maintaining normothermia is of special concern in the geriatric patient.
- The age-related progressive impairment of the ability to handle an intravenous glucose challenge may also be in large part due to decrease in lean body mass tissue as this tissue normally provides storage for carbohydrates. The renal threshold for glucose is notoriously unpredictable in the elderly and urine testing is not a reliable sign of presence or absence of hypo/hyperglycemia.

XIV. Hematological and Immune System:

- Reduction in bone marrow production and spleen size, reduces the haematopoietic response to imposed anemia.
- Depression of the selectivity and the effectiveness of immune response making them susceptible to life-threatening infection.
- Anatomic involution of thymus gland and altered function of T lymphocytes.
- Elderly patients are known to have atypical presentation of diseases. Typical response to infection like fever, rise in WBC count might be less pronounced or may be absent in Elderly patients.

Describe the Alterations in the pharmacokinetics and pharmacodynamics of various anesthetic drugs due to aging.

There are multiple physiological changes that alter the ability of elderly to handle pharmacologic agents (Table 24.1). The apparent age-related increase in potency in the anesthetic drugs may be due to a pharmacodynamic phenomenon (e.g. change in the beta-receptor sensitivity resulting in a reduced chronotropic and ionotropic response to beta-adrenergic drugs thus requiring increased doses) or it may just be a result of the pharmacokinetic changes (continual loss of neuronal substance accompanied by a parallel reduction in the cerebral blood flow and oxygen consumption $CMRO_2$) necessitating an alteration in drug doses.

Other factors that affect the pharmacokinetics and pharmacodynamics are:

Table 24.1 Changes in body composition

Change	Change in compartment	Effect
Decrease in lean body mass	Smaller central compartment	Higher peak concentrations
Decrease in total body water	Small 'rapidly equilibrating peripheral compartment'	Decreased dose requirement
Increase in body fat	Very large 'slowly equilibrating peripheral compartment'	Longer duration of drug effect
	Decrease in rate of inter-compartmental clearance (i.e., constant-K)	

Changes in Cardiovascular System: Elderly individual, are more susceptible to the hypotensive effects of anesthetic drugs. There is delayed induction of anesthesia following administration of intravenous anesthetic agents due to reduction in cardiac output. In contrast, inhalational inductions are faster due to the rapid rise of the alveolar partial pressure, which would in turn, help in achieving higher brain partial pressures of inhalational agents.

Renal and Hepatic Effects of Ageing: The renal clearance is reduced in the elderly subjects even though they maintain normal serum creatinine values. Thus the elderly will have a delay in the offset of drugs and their metabolites excreted by the kidney, e.g. pancuronium, pethidine.

Most of the intravenous drugs used in the practice of anesthesia are metabolised in the liver. Aging is associated with decrease in the liver volume, liver blood flow and the intrinsic hepatic capacity, all leading to a reduced total drug clearance and a higher steady state plasma concentration in the elderly.

IV. Protein binding: Albumin and alpha 1 acid glycoprotein are the primary sites of plasma protein binding. There is a reduction in the plasma albumin concentration even in the well-nourished healthy elderly individual. This change is exaggerated in the poorly-nourished and those with advanced illness. As a result a greater quantity of free drug is available for diffusion to the site of action. Therefore acid drugs bound to albumin such as pethidine and diazepam have reduced dose requirements in the elderly. The increased alpha 1 acid glycoprotein in the elderly reduces the free fraction of lignocaine and retards clearance in the elderly. Therefore these changes in protein binding have an impact on the free fraction, clearance and the volume of distribution of drugs.

Change in the Pharmacology of Specific Drugs

Thiopentone sodium: Decrease in the lean body mass → Reduction in the volume of distribution → High plasma concentrations → Increased sensitivity

- Thiopental induction doses should be about 85% of the younger counterparts
- Prolonged infusion of Thiopentone will lead to accumulation of drug.

Propofol:

- Elderly brain is more sensitive to propofol
- Smaller central compartment and a reduced volume of distribution
- Reduced clearance
  - Induction dose-reduced (around 1.7 mg/kg)
  - Maintenance dose- reduced by 30–50%

Benzodiazepines:

- Midazolam, lorazepam, diazepam have comparable protein binding and volume of distribution.
- However, the high clearance rate of midazolam makes it an attractive alternative.
- A reduction of about 75% in the Midazolam dose has been suggested due to increased sensitivity of the brain and a reduced clearance.

Opioids:

- Twice as potent in the elderly subjects.
- 50% reduction in doses
- Shorter acting opiods, i.e. fentanyl, alfentanil, sufentanil, remifentanil are better choices.

Muscle Relaxants: The number of acetylcholine receptors at the neuromuscular junction and their sensitivity to non-depolarizing muscle relaxants is not altered by advancing age. Therefore the dosage of these drugs required to block the neuromuscular junction is unchanged or slightly increased. But the decreased hepatic and renal blood flow and a reduced hepatic function are responsible for the prolonged neuromuscular blockade in the elderly. Muscle relaxants such as Atracurium and Cis-atracurium, which do not depend on the hepatic clearance, are recommended especially in the compromised elderly population.

However the reversal of neuromuscular blockade by neostigmine is unchanged though elderly patients are more prone to the adverse cardiac effect of this drug.

Inhalational Agents: Age-related changes in the cerebral metabolism reduce the minimum alveolar concentration (MAC) of the inhaled anesthetics. The decreased cardiac output in the elderly as mentioned earlier along with a decrease in the MAC values results in rapid induction rates.

Recovery from inhalational agents is prolonged and has a pharmacokinetic basis. The decrease in the lean body mass and increase in the body fat, increases the volume of distribution especially for fat-soluble agents. In addition decreased hepatic function together with a decreased pulmonary gas exchange may decrease the anesthetic clearance with age.

Other Drugs:

The dosage of atropine required to increase the heart rate in the event of bradycardia may be increased in the elderly. This response is due to a decrease in the vagal outflow that occurs with aging. As central cholinergic syndrome due to the excessive use of atropine is more common in the elderly, Glycopyrrolate, which does not cross the blood brain barrier, may be used alternatively.

Adrenaline, isoprenaline and other adrenergic drugs used in an event of crisis require an increase dosing due to reduced beta-receptor sensitivity.

Adverse Drug Interaction:

Balanced Anesthesia requires the administration of various drugs. The possibility of adverse drug reaction increases with polypharmacy used for balanced anesthesia in the elderly due to the decreased physiological reserve as well as the concomitant drug therapy that they commonly take.

Dramatic hypotension following induction of general anesthesia is seen after the combination of opioids, hypnotics and inhalational agents. This is seen even in healthy elderly individuals and is more profound and hazardous in patients with coronary artery disease and poor left ventricular function.

Propofol-opioid combinations cause more hypotension as compared to barbiturate-opioid and benzodiazepine-opioid combination. Fluid loading prior to anaesthesia as well as significant reduction in the induction doses of the individual agents is mandatory.

Volatile anesthetics are frequently combined in balanced anesthesia. With the exception of halothane low concentrations of volatile anesthetics in combination with opioids are well-tolerated even in compromised patients. The newer volatile anesthetics desflurane and sevoflurane in combination with opioids do not produce exaggerated response as compared to the intravenous agents.

Hypotension after induction may be compounded if the patient is on beta-blockers, calcium entry blockers, nitrates or ACE inhibitors. Vecuronium which normally is cardio-stable can cause severe negative ionotropic and chronotropic effect when used in the combination with the above drugs.

Hemodynamic changes thus induced can prolong the metabolism and excretion by the liver leading to exaggerated responses and prolonged recovery. Substances that alter the function of the liver enzymes can alter metabolism of the anesthetic drugs. Enzyme induction by alcohol can markedly alter the metabolism of ansthetic agents. Drug such as H_2 receptor antagonist can suppress the liver enzymes and cause prolonged action of drugs such as midazolam, propofol, alfentanil, etc.

Describe the Perioperative risks involved in respect to old age.

It is the high incidence of existing medical conditions that causes elderly patients to have a greater risk of perioperative morbidity and mortality. Multiple diseases are the rule rather than exception in elderly patients. A variety of chronic disorders, such as hypertension, diabetes mellitus, atherosclerosis, myocardial infarction, cerebrovascular accidents, pulmonary diseases, osteoporosis, osteoarthritis, anemia, Parkinsonism, dementia and even malignancy are observed with increasing frequency in this patient group. In addition to all these, the physiologic changes that accompany the process of aging contribute to an increased perioperative risk for elder persons who have to undergo anaesthesia and surgery.

Ischemic Heart Disease: Elderly patients invariably have atheromatous plaques in their vessels and there are high chances of having them in their coronary vessels. It is well-known that surgery imposes a certain definite threat for the development of postoperative MI in patients with IHD whether diabetic or non-diabetic. One can use ASA risk stratification and other risk stratification scores given in Chapter on IHD. The overall perioperative risk should take in to account the physical status of the patient, and also considers the skill of the surgeon, anesthesiologist, and site and duration of the procedure.

Hypertension: It is the most common disease with serious potential consequences. The details that need attention are the degree (mild, moderate or severe), the duration (recent or chronic), whether primary (essential) or secondary, end-organ changes, whether the patient is at increased perioperative risk, will lowering the blood pressure preoperatively lower risk and how long should the patient be treated before surgery. For patients who are recently diagnosed to be hypertensive or in whom control is not good, if the surgical condition permits, treatment should be optimized until the patient is normotensive on follow-up for 3–4 weeks, to allow for the normalization of some of the hypertensive vascular changes, including LV hypertrophy.

Diabetes: The preoperative evaluation can be distinctly divided into two parts, viz. the evaluation of the diabetic status *per se* and associated end organ disease in a diabetic which includes the cardiac status, hypertension, the renal status, peripheral vascular disease, autonomic neuropathy and ocular changes.

The principal involvement of the heart in diabetes is in the form of: atherosclerotic CAD, cardiomyopathy, autonomic nervous system dysfunction, silent MI – (incidence 25%), increased risk of CHF, blunted response to stress all of which increase risk of sudden unexplained death.

Cerebrovascular Accidents/Insufficiency (CVA): Higher Systolic BP and LVH are the strongest predictors of stroke in the elderly. Cardiac impairment like Chronic heart disease, cardiac failure, atrial fibrillation, valvular problems increase stroke risk

at any level of BP. Intraoperative hypotension, hypertension and arrhythmias can increase the risk of stroke. CVA can present as strokes, focal neurologic deficit or transient ischemic attacks (TIA).

Cognitive Dysfunction: Impaired memory and concentration, mild personality changes, and emotional instability, are commonly referred to as postoperative cognitive dysfunction (POCD). Reported incidence in literature ranges from 10–60% in elderly population. Several etiologic mechanisms behind POCD have been suggested which include cerebral hypoxia caused by arterial hypoxemia or low flow, residual concentrations of drugs such as benzodiazepines that have also active metabolites, long-lasting effect of general anesthestics on cholinergic or glutaminergic neurotransmission and psychological factors related to illness and environment during hospitalization. Anesthetic techniques does not influence incidence of POCD, and studies comparing regional versus general anesthesia haven't demonstrated a significant effect on the incidence of this complication.

What factors influence Regional anesthesia techniques in the old age?

For neuraxial blocks:

- Size of epidural space is reduced
- Permeability of the dura is increased
- Volume of CSF decreased
- There is decrease in size and number of myelinated fibers in the dorsal and ventral nerve roots.
- Narrowing of intervertebral spaces and osteophyte growth which decrease transforaminal escape of the local anesthetics injected during epidural anesthesia, producing an increased level of block.
- The onset of analgesia with epidural anesthesia is more rapid. This is believed to result from an increased permeability of extraneural tissues to local anesthetics.

For local anaesthesia/peripheral nerve blocks:

- There is decrease in conduction velocity of peripheral nerves owing to decrease in inter–Schwann cell distance.
- Decreased number of axons in the peripheral nerve.

All above mentioned factors suggest a generalized reduction in the requirements for local anesthetic drugs in a variety of regional techniques. Higher plasma concentrations of local anesthetics after epidural anesthesia have been observed in elderly people. This may reflect prolonged elimination half-life resulting in reduced plasma clearance. Because of reduced clearance, toxic plasma concentrations may result from lower doses of local anesthetic drugs in elderly patients.

How will you evaluate and assess a geriatric patient preoperatively?

An elderly with significant coronary disease, smoking history and diabetes may already consume most of his "reserve" just to maintain his vital signs and saturation of 90%, but trauma of surgery can precipitate widespread decompensation. Development of palpitations, cardiac arrhythmia, precordial pain, severe dyspnea, or extreme fatigue while performing simple exercise such as walking or climbing one flight of stairs indicates a lack of ability to compensate for mild stress.

The aim of preanesthetic assessment is to establish the functional status of each major organ system including heart, lung, central and peripheral nervous systems, hepatic and renal systems. Individual organ systems functional reserve should be established by history, physical examination, review of medications and by appropriate laboratory tests.

A preoperative assessment should include medication details, medical problem evaluation, detection of sensory perceptual deficits, mental preparation before surgery, and neuropsychological testing, if possible as this age group is prone to develop cognitive dysfunction. Explain need for postoperative ventilation, ICU stay, lines/tubes, etc. Also note presence of artificial dentures, hearing aids, pacemaker, AICD, etc.

Preoperative Testing:

- Should be clinically directed as indicated by both physical status of patient and site and invasiveness of surgical procedure.
- Should provide the information needed to determine the severity of pre-existing disease and the adequacy of a remaining organ system reserve. This would help in risk stratification as well as optimization of patient before surgery.
- Elderly patients with multiple cardiac risk factors require more extensive evaluation to define perioperative risk for myocardial ischemia. If needed, 2–D echocardiography and/stress test should be performed.
- Since pulmonary morbidity is common in elderly patients undergoing major surgery, preoperative pulmonary function tests can be ordered so as to optimize respiratory function preoperatively, however, there is no evidence showing change in outcome.

Describe important issues in Postoperative period in the old age

All elderly patients after completion of surgery should be transferred to a recovery room, a high dependency unit or an intensive care unit depending on their condition. Just like other patients, details of the preoperative status, the

anesthetic, drugs, doses, and timing of drug administered during surgery, details of monitoring, other procedures performed and complications must be noted.

Oxygenation: In elderly patients, the increase in cardiac output and ventilation to satisfy oxygen demand does not occur readily. Diffusion hypoxia may be more serious and prolonged in the elderly after GA and hence all patients should receive supplemental oxygen.

Postoperative analgesia: Poorly controlled pain in the postoperative period can lead to slow recovery and life-threatening complications. Also one of the risk factor suggested for POCD is poor pain control. Hence, adequate analgesia in postoperative period is a mandate.

NSAIDS and paracetamol may be used by intravenous, intramuscular, oral or rectal route. NSAIDs should be avoided in those over 70 years of age, those with pre-existing renal dysfunction, and in those who have suffered hemodynamic instability or major blood loss.

Peripheral blocks when feasible should be used. Intra articular injection of LA or narcotic can provide effective analgesia in joint surgeries. Epidural analgesia should be used for surgeries involving thorax, abdomen, pelvis and lower extremities.

Hemodynamics: The heart rate may not be a reliable index of hypovolemia in the elderly patient. Due to reduced numbers of adrenergic receptors, decreased efficacy of baroreceptor reflexes, and the administration of concomitant beta-blockade and hypothermia, hypovolemia may exist without tachycardia, and may manifest with profound hypotension. Patients may have atypical signs of impending hemodynamic instability such as nausea, mental obtundation and tachypnea.

In all elderly patients, ECG, hourly urine output, and blood pressure must be monitored. Direct intra-arterial pressure and CVP monitoring or other form of advanced hemodyanamic monitoring must be instituted early in patients at high-risk or those who have developed hemodynamic instability. Warm peripheries (toes), coupled with an hourly urine output > 0.5 mL/kg/hr usually imply a satisfactory hemodynamic status.

Hypotension is a common problem. Patients are often hypovolemic from the preoperative period itself. They may be hypovolemic due to decreased thirst and inadequate fluid intake, prolonged periods of fasting, and concomitant diuretic therapy. This may get exacerbated perioperatively due to reduction in autonomic activity and insufficient compensation. The volume of fluid that may be necessary to support the circulation during the anesthetic may prove excessive once the anesthetic is eliminated and sympathetic tone returns, exposing the patient to a risk of volume overload and pulmonary edema. It is safer to administer volume in small intermittent boluses, watching the response of the CVP, blood pressure and urine output.

Administration of hypotonic fluids (5% dextrose, 0.33% NS, etc.) may result in hyponatremia and low serum osmolality (< 235 osmol/L) with resultant risk of cerebral edema.

Arrhythmias are common occurrence in elderly patients. In patients without pre-existing cardiac disease, they may represent disturbances due to hyperventilation (most common cause is pain), hypokalemia, hypomagnesemia, hypocalcemia, hypoxia or hypercarbia. These metabolic abnormalities can be easily diagnosed and treated. Arrhythmias may indicate myocardial ischemia (especially VPCs > 5/min, bigeminy, ventricular tachycardia, various degrees of heart block other than first degree). Atrial fibrillation (AF) is not uncommon, and should be treated if the ventricular response is rapid or there is hemodynamic deterioration. AF with low blood pressure may require electric cardioversion. When the blood pressure is well-maintained, calcium channel blockers like diltiazam or beta blockers can effectively reduce the ventricular response. Minor arrhythmias may be significant in elderly patients with ischemic or valvular disease such as aortic stenosis. Hemodynamic implications include loss of atrial kick in AF; bradycardia worsening a fixed low cardiac output; tachycardia decreasing ventricular filling and increasing oxygen consumption, and VPCs resulting in less effective contraction.

Hypothermia: Hypothermia manifests as altered mental status, delayed recovery from anesthesia, sluggish deep tendon reflexes, and a slow or shallow respiratory pattern. Cardiac rhythm disturbance include SVT, AF, atrial and ventricular ectopics, various degrees of heart block and conduction disorders, and suppression of SA node, associated with hypotension and a falling cardiac output. Hypothermia also lead to metabolic disturbances, reduce liver and renal perfusion, induce coagulaopathy, and also make non-depolarizing muscle relaxants more difficult to reverse with anticholinesterase drugs.

Mild hypothermia is best corrected with gradual spontaneous rewarming with a blanket, in a comfortably warm room but severe hypothermia would need active warming methods, such as use of warm IV fluids and surface warming in postoperative room along with continuous core temperature monitoring.

Postoperative Confusion/Mental Dysfunction: Postoperative confusion and mental dysfunction in the elderly patients after surgery is a well-known. Surgery has a significantly

decompensating impact on the mental status of older persons. The use of anticholinergic drugs, antihistaminics, phenothiazines and tricyclic antidepressants have been shown to correlate more closely with postoperative confusion than the technique of anesthesia. Elderly patients often react to benzodiazepines with confusion. Elderly patients with mental depression are known to have lower levels of neurotransmitter substances, which might predispose to confusion. Occasionally an acute confusional state may be due to neurological problems, or to metabolic disturbances such as hypoxia, hypercarbia, acidosis, hypoglycemia, hyperglycemia, hyponatremia, renal and liver dysfunction. All these potentially treatable conditions should be ruled out when confronted with an acutely confused patient.

Sepsis is another potentially dangerous cause of an acute confusional state. One must keep in mind that elderly patients may not demonstrate high fever, tachycardia or leukocytosis as a response to sepsis. The only manifestation of serious sepsis may be mental obtundation or confusion.

Postoperative delirium: Postoperatively elderly patients often think and speak incoherently, are disoriented and show impairment of memory and attention in postoperative period.

Postoperative CVA/TIA: Strokes may present as mono/hemi/para paresis/plegia. Patients may be obtunded or confused. Any mental change persisting or occurring beyond the normal expected period of anesthetic recovery should be viewed suspiciously, and needs a thorough clinical examination. Radiological imaging in form of CT scan or MRI should be performed, and if there is no evidence of intracerebral hemorrhage, aspirin or heparin may be considered.

Sleep disordered breathing: The reason for the increased episodes of disordered breathing during sleep with age is unclear. As high as 2/3rd of the elderly have frequent episodes of desaturation and apnea during sleep with diminished response both to hypercapnia and hypoxia. Patients receiving narcotics and sedatives should be closely monitored by pulse oximetry.

Renal system: Elderly patients are at risk for renal failure in the postoperative period following major surgery. The reasons are multifactorial, and include prerenal causes such as hypovolemia, and cardiac dysfunction. Drugs are important causes of renal dysfunction, and aminoglycosides, ACE inhibitors, NSAIDs, and radio-contrast media are frequently associated with renal failure. Renal failure may be one of the major manifestations of sepsis.

Miscellaneous: Care must also be given to make the patient as comfortable as possible, and to provide him with eye glasses, hearing aids, prosthesis, etc. Bowel and bladder function must be monitored. Elderly patients may not pass urine even after a minor operation performed under a neuraxial block.

Higher incidence of benign prostate hyperplasia (BPH) should be kept in mind.

DVT: Elderly patients are more prone for deep vein thrombosis (DVT) and pulmonary embolism (PE). Advanced age, obesity, prior thromboembolism, malignancy, immobility, pelvic, hip, and orthopedic surgery, central lines and congenital thrombophilic disorders are other important risk factors. Though more commonly it occurs in postoperative period, sometimes it can complicate preoperative or intraoperative period. Depending on patient's mobility, clinical condition, baseline risk factors and nature of surgery, appropriate DVT prophylaxis should be considered.

Suggested Readings

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25 Transurethral Resection of Prostate

J Divatia, R Gehdoo, S Bhosale

A 67-year-old male patient who presented with history of dysuria, urgency and hesitancy is posted for TURP.

What is Transurethral resection of the prostate (TURP)?

Transurethral resection of the prostate (TURP) involves the resection of benign hypertrophic prostatic tissue by means of a movable electrocautery cutting wire loop located at the end of a urethroscope. The urethroscope is passed through a sheath that has been positioned within the patient's urethra. As the surgical field is visualized through the urethroscope, the cutting wire loop is moved back and forth, carving away a small piece of prostatic tissue each time the loop is withdrawn toward the surgeon. Simultaneously, an irrigating solution flows into the surgical site via a channel in the urethroscope to distend the bladder and to bathe the surgical site, washing away blood and tissue debris removed by the wire loop. So a clear operative field is maintained for the surgeon.

Describe the anatomy of the prostate gland.

The prostate gland approximately weighs 20 g and underlies the apex of the male bladder and surrounds the prostatic portion of the urethra. The prostate is formed by enlargement of the urethral glands. A fibrous sheath surrounds the prostate, and the body of the gland consists of a fibromuscular stroma that envelops the glandular tissue. Venous drainage is via the thin-walled veins, or sinuses, of the prostatic plexus.

Although developmentally divisible into two lobes, the prostate gland is anatomically divisible into five lobes. The median and lateral two lobes of the prostate gland most frequently undergo benign prostatic hypertrophy. The nerve supply to the prostate derives from the prostatic plexus, which originates from the inferior hypogastric (pelvic) plexus. Afferent pain fibers of the prostate, urethra, and mucosa of the bladder originate primarily from sacral nerves 2, 3, and 4 (S2, S3, and S4). Pain impulses from an over stretched bladder travel with sympathetic fibers that have their origin in the twelfth thoracic and first and second lumbar nerves (T12, L1, and L2). Proprioceptive impulses from the muscular wall of the bladder, which are activated by stretching of the muscular wall as the bladder fills, are carried by the parasympathetic fibers of S2, S3, and S4.

How would you assess this patient for anesthesia?

TURP is generally performed on elderly men. Hence, one must also take into account multiple co-morbidities to which the elderly are prone like, i.e. IHD, HT, DM, renal dysfunction, etc. In addition, they may be on treatment for such conditions with medications like aspirin, oral anti-coagulants, etc. For details about assessment of geriatric patients, refer to the chapter "Anesthetic Considerations in Geriatric patients". A brief discussion of age-related organ changes is given here.

Central nervous system: Reduced functional tissue in the CNS is probably one of the factors that reduces the anesthetic dose needed in older patients. Old patients often have sluggish, impaired or absent reflex responses (e.g. pupillary light reflex) which may complicate monitoring during anesthesia. Loss of function of the special senses such as sight and hearing may lead to apprehension in strange environments, and sometimes sedation is needed to reduce preoperative stress which otherwise can significantly increase sympathetic stimulation. Geriatric population has reduced ability to generate body temperature and is susceptible to develop

hypothermia, particularly during prolonged surgery or the postoperative recovery period. In this context it is important to remember that core body temperature may differ from peripheral measurements, and the use of esophageal thermometers or infra-red thermometers (applied in the aural canal) may be preferable to rectal temperature recording.

Peripheral nervous system: Supersensitivity of postsynaptic receptors may prolong the action of muscle relaxants. Interpretation of the significance of poor reflex responses during anesthesia is more difficult in older patients than in the young.

Cardiovascular system: Subclinical and clinical cardiac disease is common in elderly, and impaired cardiovascular function should be expected in elderly patients. Baroreceptor function may be impaired, particularly in patients with chronic congestive heart failure, and the cardiovascular system's ability to compensate for surgical hemorrhage, or for the vasodilatory effects of anesthetic agents may be inadequate resulting in severe hypotension. Existing cardiovascular conditions such as congestive heart failure, cor pulmonale, sick sinus syndrome and the cardiac signs associated with hyperthyroidism should be stabilized before general anesthesia. Anesthetic agents depress cardiac function and cardiac arrest can be precipitated if cardiac arrhythmias are present, particularly ventricular arrhythmias that are refractory to therapy, right bundle branch block and bradycardia. Monitoring heart rate and systolic and diastolic blood pressures by direct or indirect methods is desirable throughout anesthesia as is ECG recording, monitoring pulse character and regular examination of visible mucous membranes for capillary refill time.

Respiratory system: Age-related degenerative changes progressively decrease pulmonary function and physical changes occur in the lungs and chest wall. There is reduced alveolar surface area and diffusion capacity, pulmonary fibrosis, reduced lung elasticity, and reduced mechanical ventilation reserve. Chronic obstructive lung disease is common. All of these changes impair gaseous exchange during anaesthesia hence oxygen supplementation is beneficial, and in some cases the use of bronchodilators may be indicated. Pulmonary embolism is a common postoperative complication in old people. The incidence of deep vein thrombosis is reported to be as high as 45% in those aged over 40, and 65% in patients over 71.

Renal: Impaired renal function prolongs the plasma half-life of drugs eliminated via the kidney and may alter fluid, electrolyte and acid-base balance, so screening for renal function is important before anesthesia. It is good practice to administer a balanced solution intravenously before and during anaesthesia to facilitate control of fluid, electrolyte and acid-base balance and to maintain renal perfusion. If hypotension occurs during anesthesia tubular ischemia may result leading to acute tubular necrosis and renal failure. Advancing age and general anesthesia are both important risk factors for the development of acute renal failure. Other risk factors include the administration of non-steroidal anti-inflammatory drugs (NSAIDs), aminoglycosides, tetracyclines and other nephrotoxic drugs.

Liver: Most liver function tests usually remain normal in geriatric patients and this probably reflects the huge reserve capacity of this organ, however, in humans bromsulphalein (BSP) retention does increase with age. In the presence of impaired hepatic function the plasma clearance rate for drugs may be decreased resulting in increased duration of action. At the same time drugs and nutrients that need to be converted to an active form by the liver may exhibit reduced activity.

What is the position of the patient for TURP? Describe the complications associated with that position?

Patient Position during TURP: An important aspect during TURP is the position given to the patient. For optimal surgical exposure, the patient is put in lithotomy position. The first consideration is to fit the equipment to the patient and not the patient to the equipment.

How to give lithotomy position: Patient is positioned supine on the table, anaesthesia instituted and then patients' legs are elevated TOGETHER, flexed TOGETHER and only then put in stirrups. Hence, the thighs are flexed at about 90 degrees to the abdomen and outwardly rotated. For TURP- Lithotomy with obtuse angle between the thigh and abdomen is desirable.

Variations of lithotomy position

1. Lithotomy with Trendelenberg position.
2. Walcher position.
3. Exaggerated lithotomy position (Trendelenberg position with sheets/towels beneath the sacrum to tilt it up).

Complications of the lithotomy position

1. *Venous stasis:* Occurs in prolonged lithotomy. Stasis occurs at points of compression by equipment or at the groin due to thigh flexion. Patients with varicose veins are at high risk.

Preventive measure: If lithotomy position is for more than 15 minutes then the legs have to be protected by elastic stockings.

2. *Peripheral nerve injury:* It occurs either due to direct trauma or usually due to compression of blood vessels supplying the nerve.
   a. Obturator nerve (L2, 3, 4): It leaves the pelvis through the obturator foramen and starts dividing in the upper thigh. Thus, acute flexion of the thigh to the groin causes nerve compression and trauma. This leads to weakness or paralysis of adductors of thigh. This leads to difficulty in walking and will manifest as an outward swing of the leg.
   b. Saphenous nerve (L 2, 3, 4): Compression of the medial aspect of the leg against the knee brace results in sensory loss to the medial side of the thigh. Femoral nerve is injured by acute thigh flexion with angulation against the undersurface of the pubic ramus.
   c. Common peroneal nerve (L 4, 5-S1, 2): It courses around the head of the fibula. It becomes flattened here and is prone to pressure injury to its nutrient vessel. Any prolonged compression of the lateral aspect of the knee can result in nerve injury and leads to foot drop.
3. *Cardiorespiratory compromise:* The lithotomy position results in an 18% reduction in the vital capacity, which is further increased by Trendelenberg tilt (up to 14.5% with a 20^0 tilt). Spinal anaesthesia causes pooling of blood in the lower limbs, and this may be abruptly returned to the central circulation when the legs are raised to give the lithotomy position. All this may result in precipitation of respiratory and / or cardiac failure in elderly patients with pre-existing disease and diminished cardiopulmonary reserve.

What is the primary concern and complication associated with TURP?

The primary concern associated specifically with TURP is intravascular absorption of large volumes of irrigating fluid during the procedure. The absorption occurs predominantly through exposed venous sinuses of the surgical capsule.

Complications can be categorized according to the time of occurrence.

Intraoperative complications:

- TURP syndrome.
- Hypothermia: this is due to use of cold irrigation fluids.
- Myocardial infarction: the incidence of perioperative myocardial infarction ranges from 1–3%.
- Bleeding and bladder perforation are also common occurrences during TURP.

Postoperative complications:

- TURP syndrome.
- Clot Retention.
- Bleeding.
- Postoperative cognitive dysfunction.

What are the first signs and symptoms of TURP syndrome?

Approximately 1–8% of TURP surgeries may have TURP syndrome. Recent larger studies have demonstrated lower incidence rates of between 0.78% and 1.4%, with mortality rates of 0.2–0.8%. The development of TURP syndrome can be from 15 mins of procedure to 24 hours post-procedure. The clinical manifestations brought about by intravascular fluid absorption are referred to as the TURP syndrome, and the degree of symptoms depends on the type, magnitude, and extent of absorbed fluid. The anesthesiologist must recognize signs and symptoms of developing TURP syndrome. For the patient undergoing TURP under regional anesthesia (subarachnoid block or epidural block), the first sign has been described classically as restlessness and mental confusion. However, presentation is variable, and the syndrome may manifest initially with nausea, vomiting, dizziness, headache, unresponsiveness, or transient visual changes. Hemodynamic instability (hypertension, hypotension, heart rate changes, cardiac arrhythmias, pulmonary edema, or cyanosis) may be the first indication of a development of TURP syndrome.

Which is the best irrigating fluid for TURP? Give a brief description of all irrigating fluid used for TURP?

Several types of fluids are available for use during a TURP procedure. But the ideal irrigant should be:

1. Iso-osmolar and non-hemolytic
2. Non-electrolytic
3. Non-toxic
4. Transparent
5. Non-metabolizable
6. Rapidly excretable
7. Inexpensive and sterile.

Irrigating solutions available are:

Saline and Lactated Ringer's solution: It conduct the electrical current but not the thermal properties of the ESU. They disperse the electrical current, and no cutting or coagulation occurs. Electrolyte-based solutions are therefore not used as an irrigating solution for TURP procedures.

Water: Water may be used for short TURP procedures, but it is not used commonly because it is hypotonic and eventually

will result in hemolysis; releasing free serum hemoglobin and potassium. Although a serum hemoglobin level is not toxic until it reaches 600 mg, there is a risk of serum hemoglobin combining with abnormal serum proteins, rendering it nephrotoxic .Release of potassium can lead to hyperkalemia, resulting in cardiac arrhythmias and muscular weakness. Severe hyponatremia and hypo-osmolality can occur, with CNS symptoms including convulsions and coma.

Glucose: Glucose 5.4% has been used in the past but is no longer used because it causes tissue charring and also leaves the surgical site and instruments very sticky. This makes the procedure surgically difficult. Additionally, elevated serum glucose levels can occur as the fluid is absorbed into the blood stream. This poses an unnecessary risk for patients with diabetes.

Urea: Urea 1.8% also has been used in the past but is no longer used as it crystallizes on the instrments. Urea is permeable to the intracellular and extracellular spaces, which results in elevated serum urea levels even when only small amounts are absorbed. Elevated serum urea levels result in nausea, vomiting, headache, tachycardia, elevated blood pressure, diminished vision, convulsions, and coma. Urea also has an osmotic diuretic effect, so dehydration can occur.

Sorbitol: Sorbitol 3.3% (osmlality: 165 mosm) is used infrequently as an irrigation solution. It is metabolized into carbon dioxide, glucose, and water. The kidneys excrete all three substances as by-products. Water intoxication remains a problem when large quantities of sorbitol are absorbed. Additionally, the resultant elevated serum glucose levels can pose problems, especially for patients with diabetes. It may also cause lactic acidosis.

Mannitol: Even though mannitol 3% is an osmotic diuretic, it has been used for irrigation. Dehydration, hyperosmolality and acidosis with resultant neurological disturbances can occur.

Cytal: It is a combination of sorbitol 2.7% and mannitol 0.54%. It is not widely used. Sorbitol is metabolised to fructose, which may present problems to patients with hypersensitivity to fructose.

Glycine: Currently, the most commonly used irrigant is 1.5% glycine (osmlality: 200osm). The liver metabolizes glycine into ammonia, water, and glycolic acid, resulting in hyperammonemia. When large amounts of glycine are absorbed, hyperammonemia and water intoxication can lead to cerebral edema and seizures. Increased absorption can also cause hyper-oxaluria.

What is TUR syndrome? Describe the physiology and management of TUR syndrome?

Transurethral resection syndrome is a constellation of symptoms and signs caused primarily by absorption of irrigating fluid. The factors leading to this syndrome have been discussed above. The features of the syndrome include:

1. CNS manifestations are disorientation, restlessness, confusion, agitation, drowsiness, convulsions and coma. This is usually due to water intoxication, dilutional hyponatremia and hypo-osmolality.
   Blindness, delayed recovery from anaesthesia, drowsiness may be due to glycine toxicity.
2. CVS manifestations are dyspnea, pulmonary congestion, pulmonary edema, cardiac arrest. This is due to fluid overload and negative inotropic effect of hyponatremia.
3. Hemolysis.
4. Hypothermia—bladder irrigation with room temperature irrigation fluids can drop body temperature > 2°C.

Factors affecting intra-vascular absorption and methods to reduce the risk of the TURP Syndrome

1. Number and size of the venous sinuses opened.
2. Duration of exposure: Reduction of the operative time to less than 1 hr decreases the chance of TUR Syndrome. The rate of absorption of fluid is 10–30 mL/min.
3. Venous pressure at irrigant-blood interface.

The irrigating fluid bag should be at a maximum height of 60 cm (2 feet) above the level of the pubic symphysis. This leads to a hydrostatic pressure of 60 cm of water which is maximum allowable.

Intravascular absorption of irrigating fluid can lead to:
1. *Over-hydration:* It was 1st described as a syndrome in 1973 when 1.2% glycine was used.
The syndrome of over-hydration consists of a triad of signs:

a. Bradycardia.
b. Elevated systolic and diastolic BP with increased pulse pressure.
c. Cerebral agitation and depression.

Cerebral signs may be variable but generally commence with headache, dizziness, restlessness, agitation and confusion graduating to progressive obtundation, stupor and coma. Neuromuscular disturbances ranging from twitches to seizures can occur. Later dyspnea, cyanosis, refractory hypotension and cardiac arrest may follow.

The absorption of large amounts of electrolyte free irrigating solution results in increased intra-vascular volume, hemodilution, hyponatremia and development of left heart failure, pulmonary and cerebral oedema and ultimately

cardio-vascular collapse. It is seen that faster the fall of serum sodium levels, more are the toxic symptoms likely to develop. A drop of 20–30 meq/lit or an absolute value of 120 meq/lit indicates a severe reaction.

Table 25.1 Serum sodium systemic effects

S. Sodium levels	CNS effects	ECG effects
120 meq/L	Confusion, restlessness	Wide QRS
115 meq/L	Somnolence, nausea	Wide QRS, ST elevation
110 meq/L	Seizures, coma	Vent. Tachycardia/Fibrillation

In less florid cases; hypothermia, weight gain and post-operative diuresis is seen. Unusual symptoms of fluid absorption include visual disturbances ranging from blurred vision and papillary dilatation to transient edema. These symptoms were ascribed to cerebral and spinal oedema respectively.

When over-hydration syndrome is suspected immediate measurement of serum sodium and serum osmolality should be done. Those patients with isolated alteration of sodium concentration may not develop serious sequelae and spontaneously diurese and need no further treatment.

When patient develops neurological signs prompt intervention is required. Treatment is directed towards reversing the flow of water into the cells and correcting hypotonicity. Immediately the surgery should be stopped, patient to be oxygenated and diuretic like Furosemide to be administered with restriction of fluid intake. Then correction of hyponatremia is best done by hypertonic saline in which case sodium requirement is calculated based on total body water. Aim should be to bring the sodium level > 120 meq/lit. As administration of hypertonic saline is not without risk of fluid overload, simultaneous furosemide should be administered. Therapy should be monitored by assessing clinical and biochemical improvement.

Enumerate complications of TURP? Give brief description of each?

Apart from TURP syndrome (described above), other complications of TURP are described below:

1. *Glycine toxicity:* Intravascular absorption of glycine from the irrigant can cause direct neuro-toxic effect. It is a major inhibitory neurotransmitter in the spinal cord and midbrain. Glycine may lead to encephalopathy and seizure via NMDA receptor activity (an excitatory neurotransmitter). NMDA receptor activity is markedly potentiated by glycine and, along with its role as an inhibitory transmitter, may facilitate excitatory transmission in the brain through an allosteric activation of the NMDA receptor. Signs of glycine toxicity are nausea, vomiting, headache, malaise, weakness and visual symptoms which range from blurred vision to complete blindness. (Glycine is now thought to be the most likely cause of visual disturbances). Glycine also causes renal toxicity. It is broken down by the liver (oxidative deamination) to ammonia and oxalate. This oxalate excretion continues long enough postoperatively for calcium oxalate to deposit in the kidneys and cause renal failure. Glycine toxicity can be treated with arginine which increases ammonia metabolism via the urea cycle. Magnesium exerts negative control on the NMDA receptor. Therefore, a trial of magnesium therapy for seizure control deserves consideration, especially if osmolality is near normal. The normal value of serum glycine is 13–17 mg/L. Vision approaches normal within 24 hrs as glycine levels return to normal. This is predictable as the half-life as glycine is 85 mins. Thus, reassurance may be the best treatment.
2. *Hemolysis:* Danger of hemolysis exists when hypotonic fluids are used. It leads to hemoglobinaemia, hemoglobinuria, anemia and hyperkalemia. Acute renal failure can occur due to free hemoglobin and other breakdown products. Patient develops chills, clamminess, chest tightness, rising BP and bradycardia. The treatment is aimed at promoting diuresis and correction of biochemical and hematological anomalies.
3. *Blood loss:* It is difficult to quantify. Visual estimation is difficult due to dilution of blood with the irrigating fluid. Also the warning signs like tachycardia and hypotension are masked by bradycardia and hypertension of over-hydration syndrome. A rough estimate is 15 ml/gm of resected prostatic tissue or 2.6–4.6 mL/min of resection time. Methods of quantifying are:
   - Radioactive labeling of RBC's or albumin.
   - Alteration in electrical conductivity.
   - Calometric method.

The way to calculate true blood loss is to collect all of the irrigant-blood mixture and to measure its hematocrit:

$$\text{Blood loss (mL)} = \frac{\text{Hb of irrigating fluid (gm/mL)}}{\text{Patient's Hb (gm/lit)}} \times \text{Amount of irrigating fluid}$$

Causes of increased blood loss:

- *Circulatory overload:* Due to fluid absorption or iatrogenic over infusion leading to increased venous pressure and thus bleeding.
- *Vasodilatation:* Due to Spinal anesthesia, lithotomy position, dependant position of operative area favours blood pooling and raised venous pressure in the pelvis and prostatic sinuses. Shivering, coughing and straining can also increase venous pressure.

- *Infection:* Indwelling catheter will cause local tissue congestion and hyperemia leading to increased bleeding.
- Excessive bleeding may result from activation of plasminogen by urokinase (an euglobin of plasma protein fraction liberated from prostatic tissue) to plasmin causing fibrinolysis. This leads to generalized oozing from the prostatic fossa, delayed post-operative bleeding, bleeding from puncture sites, etc. This is treated with EACA, Tranexamic acid, fibrinogen or blood.

Bleeding can be decreased by using cooled irrigants and by selective cooling of pelvic organs. However, the risk of hypothermia should be considered in the elderly.

5. *Perforation:* This can be:
   - Urethral
   - Intra-peritoneal (rupture of dome)
   - Extra-peritoneal (rupture at neck)
   - Capsular or peri-prostatic extravasation.

Causes: It may be due to Instrumentation or over-distension of bladder.

Signs and symptoms are:
- Abdominal distension.
- Patient may complain of abdominal pain which may be generalized, suprapubic or radiating to the shoulder. In case of spinal anasthesia (SA) (level < T 10) capsular tear will cause supra-pubic pain.
- Rigidity.
- Autonomic signs, such as pallor, nausea, vomiting and diaphoresis.
- Bradycardia, hypertension and shock like picture with large extravasation.
- Non-return of irrigating fluid.

Treatment: Localised, extra-peritoneal, peri-prostatic or urethral tears are treated with catheterization and antibiotics. More extensive tears require exploration and repair of perforation and drainage of peri-vesical space. Intraperitoneal rupture requires surgical treatment and extrapritoneal rupture is treated conservatively.

1. *Hypothermia:* The common causes are illustrated below:
   - In regional anesthesia there is redistribution of body heat from deep to superficial tissues in the area of the block.
   - In the area of block ability to shiver is lost and metabolic response to trauma is obtunded.
   - There is reduced thermoregulatory capacity in elderly patients.
   - Frequent use of cold IVF and irrigating fluids.
   - Patient are on cold OT table with cool preparatory solutions.
   - Cold, dry operating room.
2. *Bacteremia:* During the procedure bacteria from the infected urinary tract may enter blood stream. Patient presents with rigors and hypotension.

Describe anesthetic management of TURP? What is the preferable anesthetic technique and why?

Anesthesia Management

Ideal anesthetic should provide adequate operative conditions and analgesia. Physiological disturbance should be minimal and compensatory mechanisms should not be compromised. Bleeding must be minimized. There should be adequate muscle relaxation to provide good irrigant flow into the bladder and to allow good surgical access. Doses of the drugs must be carefully adjusted to patient's requirement. The technique must allow early detection of over-hydration, perforation and hemolysis.

Sub-arachnoid block is the method of choice as:
1. Little anesthetic is required.
2. Patient remains awake and mentation can be assessed regularly.
3. Physiologic disturbance is minimal.
4. Respiration is affected to the minimum.
5. Early recognition of complications (Level to be restricted to T 10).
6. Peripheral vasodilatation helps reduce circulatory overload.
7. Morbidity and mortality is low.

Note the nerve supply of the prostate gland and bladder neck is:

1. Sympathetic:	T11 – L2,3
2. Parasympathetic	S2 – S4
3. Spinal levels of pain conduction	T11 – L2, S2-S4

When is TURP syndrome likely to occur?

The time to onset of TURP syndrome depends on numerous factors, including the experience of the surgeon, the surgeon's aggressiveness with the electrocuting loop, the pathology and size of the gland, the amount of tissue removed, and the amount of irrigation used. The incidence of morbidity increases as resection time exceeds 60 minutes, and for many years it was believed that TURP syndrome was unlikely during the first hour of resection. However, TURP syndrome can also develop more rapidly.

The patient is not free of risk once the resection is completed. If the integrity of the prostatic capsule or wall of the bladder is violated during surgery, irrigating fluid may be sequestered in the intraperitoneal and extraperitoneal space during resection.

The fluid may be absorbed into the intravascular space during the postoperative period and result in intravascular fluid overload and symptoms of TURP syndrome.

What is the treatment for TURP syndrome?

Treatment of TURP syndrome should begin the moment the problem is recognized.

- Terminate surgery as quickly as possible and switch to normal saline for continuous bladder irrigation. Be sure that the irrigation is warm, as the bladder irrigation now should be to prevent the development of hypothermia.
- Support ventilation as needed and obtain the following baseline laboratory tests: complete blood count, platelet count, electrolytes, and clotting studies if a bleeding problem is suspected. Prothrombin time and/or international normalized ratio (INR), partial thromboplastin time, and fibrinogen level should be included in the coagulopathy work-up.
- Administration of intravenous normal saline and diuretics may be all that is needed to correct the problem. Administer injection furosemide, 20 mg, intravenously. If the patient is on chronic diuretics, a dose of 40 mg or more may be required, but dosing should be based on the diuresis obtained initially from 20 mg bolus. 15% Mannitol can also be used as an alternative to furosemide. Maintain intravascular volume with normal saline as diuresis progresses.
- If the patient demonstrates significant effects from hyponatremia, intravenous administration of hypertonic saline may be appropriate. Restrict the use of hypertonic saline to patients who have developed central seizures or cardiac dysfunction.
- Consider placement of a central venous catheter to guide fluid replacement during the immediate postoperative period.
- If hemodynamic instability develops, consider placement of an arterial catheter and pulmonary artery catheter to aid in resuscitation.
- Monitor the serum potassium level. Patients frequently become hypokalemic as diuresis occurs.
- Reassure patients that any symptoms, especially visual changes, are only temporary and that their symptoms will dissipate as their condition improves.

Why not replace sodium deficit with hypertonic saline in patients suffering from TURP syndrome?

The use of hypertonic saline for correction of hyponatremia associated with TURP syndrome should be restricted to patients demonstrating significant symptoms, namely, central seizures or cardiac dysfunction due to electrolyte imbalance. If hypertonic saline is chosen for fluid replacement, close attention must be paid to the patient's electrolyte and intravascular fluid status. Remember that the patient has not lost sodium, but has gained water. Excessive administration of hypertonic saline results in additional fluid overload and complicates an already difficult management problem. Hypertonic saline should be administered through a central line at a rate no greater than 100 mL/h.

Is it possible to calculate how much irrigating fluid has been absorbed?

The amount of irrigating solution absorbed can also be estimated by comparing sodium levels at any time during the procedure with levels at the start of the procedure.

$$\textit{Volume absorbed} = \frac{\text{Preoperative S. Na}}{\text{Postoperative S. Na}} \times \text{ECF} - \text{ECF}$$

Example:

A 70 kg man undergoes TURP under subarachnoid block (spinal anesthetic). After 50 minutes of resection, he complains of headache and appears somewhat disoriented. The procedure is immediately terminated, and a blood sample is sent to the laboratory for electrolyte analysis. The patient's preoperative serum sodium concentration was 142 mEq/L compared with the immediate postoperative value of 106 mEq/L. If the patient has an ECF compartment of approximately 20% of body weight, his ECF volume at the start of the procedure was about 14 L (0.20 x 70 = 14 L). Using the above formula, (142/106) x 14 L = 18.75 L. Subtracting his initial extracellular volume of 14 L from the postoperative extracellular volume of 18.75 L yields an absorption of 4.75 L. This figure represents a minimal volume of absorption, because any fluid that has shifted into the intracellular space is lost in the calculation.

A more accurate technique for calculating fluid absorption is to add a 1% ethanol to the irrigating solution and then to monitor and quantify the amount of ethanol that the patient expires. This method is sensitive enough to detect approximately 75 mL of fluid absorption per 10 minutes of surgery.

One litre uptake of fluid within one hour roughly corresponds to decrease in the serum sodium concentration of 5–8 mmols/L. Risk of circulatory overload occurs when the weight of the gland is more than 45 grams.

What can be done to minimize the risk of developing TURP syndrome?

- The patient must be prepared properly for surgery. Preparation should include adequate hydration, electrolyte

analysis, and coagulation profile. Patients who are debilitated and demonstrate poor reserve benefit from the placement of hemodynamic monitors for preoperative assessment and treatment as well as for intraoperative monitoring.

- The most important step in minimizing the risk of TURP syndrome is to limit the duration of surgery. Because fluid can be absorbed at a rate greater than 50 mL/min, it is possible to place nearly 3 L of fluid into the intravascular and interstitial spaces within 1 hour of resection time. Limit resection time to 1 hour or less.
- The hydrostatic pressure created by the fluid irrigating the surgical site must be minimized. Because the irrigating fluid flows by gravity, the bag of irrigation should not hang higher than 60 cm above the operative field.
- The surgeon should limit the extent of bladder distention created by the irrigant. Frequent drainage of the bladder by the surgeon reduces the amount of irrigant absorbed.
- Careful surgical resection minimizes exposure of the venous sinuses and preserves the capsule of the prostate. Hence operator experience plays major role in prevention of TURP syndrome.
- Blood pressure must be stable. A decrease in pressure lowers the periprostatic venous pressure and allows increased absorption of fluid.
- Intraprostratic vasopressin injection shown to reduce bleeding and absorbtion of irrigating fluid
- Use of Newer techniques like bipolar electrode resection and laser excision of prostrate (Holmium:yttrium-aluminum-garnet and potassium-titanyl-phosphate lasers) can minimize the risk of bleeding and also absorption of irrigating fluid, practically eliminating the risk of TURP syndrome.

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26 Proximal Fracture Femur

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72-years-old male with history of controlled Hypertension (since 12-years) and Diabetes Mellitus (since 15-years) taking Atenolol 25 mg BD, Glimepiride 2 mg HS, Ecosprin 75 mg presents to the ED. After lunch, he slipped in the bathroom, which resulted in swelling of the right hip area with excruciating pain. He was rushed to the casualty by his son at 10 pm (within 1 hour of fall).

Enumerate the goals of management of proximal fracture femur.

Transfer of the patient to the hospital by the ambulance should be after immobilization at the site of injury. Strategies for analgesia, fluid therapy, patient warming and pressure care therapy during emergency room stay should be in place. Early fracture fixation after appropriate medical optimization should be done. Regional anesthesia is preferred. Postoperative care and rehabilitation programme extending in the post-hospital discharge period should be the norm. There should be a "Fast-track" system for the management of proximal fracture femur cases coming into the emergency department. It has been found that streamlining flow through the emergency department of these elderly patients with hip fracture; who are at risk of significant morbidity and mortality, leads to prompt surgical fixation of the fracture and effective rehabilitation. A protocol-driven multidisciplinary approach leads to faster onset of definitive management. This is of importance as surgery is the best analgesic for these patients.

How would you assess a patient with proximal fracture femur preoperatively?

Preoperative assessment by the anesthetist is mandatory, though now more and more ortho-geriatricians are also involved in the assessment. This allows for preoperative optimization of the patients along with anesthetic planning and risk assessment.

Co-morbidities: Due to preponderance of elderly patients; there is a high chance of multiple co-morbidities which need to be evaluated in the preoperative period. (Refer to the chapter on Geriatrics).

It should also be noted that the elderly patients are on multiple drugs and this may lead to increase chance of adverse drug reaction. Hence, it is necessary to review the medication list and identify pharmacokinetic and pharmacodynamic interactions.

What investigations are necessary?

- *Complete Blood Count:* Preoperative anemia is common due to fracture-related haemorrhage, hemodilution, poor nutrition and/or chronic disease. It has been suggested that older patients require a higher blood transfusion trigger than is generally applicable for patients undergoing elective surgery, to the extent that preoperative transfusion should be considered if Hb is less than 9 g/dL, or if Hb is less than 10 g/dL if the patient has a history of ischemic heart disease.
  Leucocytosis is often noted and is usually due to response to trauma rather than infection.
  A platelet count of more than 100,000/L is safe for neuraxial blockade.
- *Blood grouping and cross-match:* Revision surgery or periprosthetic fractures incur greater blood loss and hence adequate blood units must be cross-matched. Cell salvage should be considered if facilities are available. If massive

blood transfusion is anticipated than blood components may be required.

- *Serum electrolytes:* Hyperkalemia may indicate rhabdomyolysis. Hypokalemia with hypovolemia may precipitate atrial fibrillation. Hyponatremia is a common finding in elderly and may result from treatment with thiazide diuretics.
- *ECG:* Should be done in all elderly patients.
- *Chest X-ray:* Information in chest X-ray may provide useful with new onset heart failure and pneumonia or chest injuries.
- *2D-Echocadigraphy:* In an elective setting, echocardiography has a role but in the emergent setting; it may lead to delay in surgery. If patient has good effort tolerance, patient may be taken up for surgery in emergency setting. Any known cardiac murmur or a new onset murmur warrants a 2DECHO. Departmental guidelines for expediting echocardiography in hip fracture patients with cardiac murmur should be made in liaison with the cardiology department.

How will you optimize the patient preoperatively?

As soon as the patient arrives in the emergency department simultaneous assessment and optimization should be initiated. Monitoring should be instituted with pulse-oximetry, respiratory rate, ECG, NIBP, temperature, static and dynamic pain scores. This should be followed by:

1. *Preoperative traction:* Routine use of preoperative skin and skeletal traction is not recommended.
2. *Oxygen therapy:* Pulse oximetry is mandatory from the time of emergency admission to 48 hours after surgery, and oxygen administered as necessary. There is some evidence supporting routine use of oxygen therapy for the first 72 hours after surgery. Hypoxemia is common after proximal femur fractures and may be due to the pathophysiology of the fracture, or the effect of bed rest, or decreased reserve in the elderly, or coexisting diseases. Any pre-existing pulmonary compromise may get magnified after patient suffers from a fracture (e.g. fat embolism or atelectasis).
3. *Prevention of pressure sores:* A pressure-relieving mattress should be used for all patients. Patients at very high risk of pressure sores should ideally be nursed on a large-cell, alternating-pressure air mattress or similar device[2].
4. *Thromboprophylaxis:* Hip fracture patients should receive low molecular weight heparin. Mechanical devices should be used for patients in whom anticoagulants and antiplatelet agents are contraindicated.
5. *Pressure gradient stockings:* Patients should be wearing pressure gradient stockings as soon as possible after admission.

What is the ideal time of Surgery?

Surgery should be performed within 48 hours of hospital admission after hip fracture. Delaying surgery beyond 48 hours from admission is associated with prolonged hospital stay, increased morbidity (pressure sores, pneumonia, thromboembolic complications) and increased mortality.

What is the anesthetic technique of choice?

Anesthetists tend to use a technique with which they are familiar, roughly half administering regional anesthesia and the remainder general anesthesia. It is important to remember the limited reserves and co-morbidities of the elderly patients.

According to a 2004 Cochrane database review, regional anesthesia decreases the incidence of postoperative confusion. Based on this, the Scottish Intercollegiate Guidelines Network has recommended that spinal or epidural anaesthesia should be considered for all patients undergoing hip fracture repair, unless contraindicated. A recent meta-analysis suggests that regional anaesthesia is the technique of choice (although) the limited evidence available does not permit a definitive conclusion to be drawn with regard to mortality or other outcomes. The other advantages of regional anesthesia are decreased intraoperative blood loss, early mobilization, decreased respiratory complications, decreased incidence of deep vein thrombosis, less postoperative nausea and vomiting and delirium.

What are the options available when you plan regional anesthesia? Describe advantages and disadvantages.

1. *Subarachnoid Anesthesia:* This is quick to administer and hence beneficial in the patient with pain. It is commonly given with low-doses of intrathecal bupivacaine (< 10 mg) to decrease the incidence of associated hypotension. Also, to reduce the risk of hypotension; unilateral sub-arachnoid block is preferred (using lower volume of hyperbaric solution with the affected side down). When it is difficult to position the patient with the affected side down, it may be useful to use an isobaric solution (preservative-free) to achieve sensory-motor block. Addition of opioids also helps in prolonging postoperative analgesia. Sedation should be used with caution in the elderly and supplemental oxygen should be provided.
2. *Epidural Anesthesia:* It might be difficult to position the patient for inserting the epidural catheter due to the acute pain. The advantage of this technique is that the duration of anesthesia can be prolonged. Also, local anesthetic

dose can be administered in increments and hence may be better tolerated in the hypovolemic and morbid elderly patients.

3. *Combined Spinal Epidural Anesthesia:* This combination is of help as there is faster onset of action due to the subarachnoid block. The duration of block can be extended by using the epidural catheter. The downside is the time required for the procedure. This can be shortened with expertise.

How would you administer general anesthesia?

Graduated doses of anesthetics need to be given to maintain hemodynamics. Inhalational induction is better tolerated and advocated by many. Intraoperative higher inspired oxygen concentration should be administered as hypoxemia is common.

Describe perioperative analgesia.

Surgery is the best analgesic for fracture femur. Preoperative analgesia is required so that the patient is comfortable and co-operative for positioning for anesthesia. Blockade of the femoral, obturator and lateral cutaneous nerve of thigh (3-in-1 femoral nerve block/Psoas compartment block) is an effective method of providing analgesia to patients with hip fracture in the emergency department and is useful for reducing postoperative pain.

Femoral nerve block or the Fascia Iliaca Compartment Block (FICB) may not block all the three nerves but causes adequate analgesia for appropriate positioning for the neuraxial blockade. This block can be achieved by the anterior approach under ultrasound guidance as well. Also, a catheter can be placed for continuous local anesthetic infusions in the postoperative period.

What intraoperative monitoring will you use?

Basic monitoring includes the continual presence of the anesthetist, pulse oximetry, capnography, ECG and non-invasive blood pressure monitoring. Core temperature monitoring should be used routinely; as elderly patients are at the risk of hypothermia, which is increased if blood transfusions are given. It is also recommended to have Point-of-care Hemoglobin analyzers to assess anemia and guide blood transfusion. As these patients have high incidence of significant co-morbidities; further monitoring may be established, which may include:

- *Invasive arterial blood pressure monitoring:* In patients with limited left ventricular function or valvular heart disease and also in patients with massive blood loss with hypotension. It can also be used for multiple sample collection.
- *Central venous pressure (CVP) monitoring:* It may be indicated in patients with limited left ventricular function or undergoing revision of periprosthetic fracture surgery.
- *Cardiac output monitoring:* Transoesophageal Doppler may be used to guide fluid therapy under anesthesia. Transthoracic Doppler probes are becoming available for use in sedated or awake patients. Dilution techniques (e.g. LiDCO) are increasingly accurate, and may be used in conjunction with invasive blood pressure monitoring. Use of Stroke Volume Variation (FloTrac™/Vigileo™ and the PiCCOplus™ system) for fluid responsiveness is associated with better outcomes.
- *Bispectral index (BIS):* May be considered to optimise the depth of anesthesia and avoid potential cardiovascular depression.
- *Cerebral oxygen saturation:* The homeostatic regulation of cerebral blood flow is poor in older patients and depressed further by anesthesia. Thus monitoring cerebral oxygen saturation may help in reduction of postoperative cognitive dysfunction.

What are the causes of intraoperative hypotension?

1. *Hemorrhage:* Patients with fracture femur can lose considerable amount of blood even before reaching the hospital. Moreover, blood loss also occurs during surgery from the raw bone and muscles which may be more than from the identifiable bleeders. Thus, one should be careful while assessing blood loss during the surgery. Special attention should be given to the concealed blood loss in the drapes and the peri-incisional area. It is prudent to do point-of-care haemoglobin estimation to estimate blood loss and assess the requirement of blood transfusion.
2. *Fat embolism syndrome (FES):* Fat embolism as a subclinical event occurs in majority of the cases of long bone fracture. The gravity of situation depends on the amount of fat embolized, concurrent medical disease and aggravating factors. During the intramedullary reaming of femur there is alteration of the medullary blood supply. As the intramedullary pressure rises, there is reversal of the direction of blood flow (becomes centrifugal instead of centripetal). This leads to intravasation of bone marrow or fat into the vascular system. Arterial hypoxemia may be the only clinical manifestation of subclinical FES whereas hypoxemia, tachycardia, hypotension and fever are early signs preceding the development of FES. Chest radiograph finding are inconsistent, ECG may show ST-segment changes consistent with ischemia. Signs of right heart strain may be present. Once the syndrome has developed, it is associated with high mortality (10–15%). Venting medullary cavity and induced hypotension can reduce the

amount of embolism. Laboratory findings of the FES are–a rapid fall in hematocrit, thrombocytopenia, elevation of fibrinogen degradation products, prolonged PT and PTT and raised serum lipids- free fatty acid and triglycerides.

3. *Bone Cement Implantation Syndrome (BCIS):* Cardiovascular reactions to acrylic bone cement in patients with fracture neck femur and total hip replacement are a common complication. Elderly patients with fractures of the femoral neck constitute a special risk group. Methylmethacrylate (MMA) cement interdigitates within the interstices of cancellous bone, and strongly binds the prosthetic device to the patient's bone. Mixing polymerized MMA (PMMA) powder with liquid MMA monomer causes polymerization and cross-linking of polymer chains. This exothermic reaction leads to cement hardening and expansion against prosthetic components. The resultant intramedullary hypertension can cause embolization of fat, bone marrow, cement, and air into the femoral venous channels. The residual monomer can also cause vasodilatation and a decrease in systemic vascular resistance, thought to be the cause for the transient hypotension often seen with cement insertion. This bone cement implantation is associated with histamine release. In the elderly patients with pre-existing cardiac diseases and/or hypovolemia; even moderate histamine release can cause serious cardiovascular complications. Also the release of tissue thromboplastin may trigger platelet aggregation, microthrombus formation in the lungs, and cardiovascular instability as a result of circulation of vasoactive substances. The endogenous cannabinoids, anandamide (ANA) and 2-arachidonylglycerol (2-AG) are also reported to be strong vasodilators and play a role in the hypotension associated with hemorrhagic and septic shock. The clinical manifestations of this syndrome are hypoxemia (increased pulmonary shunt), hypotension, dysrhythmias (including heart block and sinus arrest), pulmonary hypertension, and decreased cardiac output. Occasionally, it can be fatal.

   Treatment: BCIS may be reversible with prompt basic life support, combined with treatment to maintain both coronary perfusion pressure and right heart function. An anaesthesiologist ordinarily manages this intervention of supporting the cardiovascular system, 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. If this syndrome does not result in sudden cardiac death, it may persist for several hours. BCIS is a time-limited process. BCIS is generally reversible even in elderly patients, if their hemodynamic stability is maintained by supportive therapy.

   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.

4. **Deep Vein Thrombosis and Thromboembolism:** Venogram and ventilation/perfusion studies have shown a prevalence of 37% for deep vein thrombosis (DVT) and 6%for PE (pulmonary embolism), although clinical symptoms are only seen in 1–3% of DVTs and 0.5–3% of PEs in patients with hip fracture.

   Factors responsible for this are:

   – Pre as well as postoperative immobilization – more so, if it is a prolonged one
   – Decreased blood flow to the limb e.g. kinking of femoral vessels
   – Postoperative hyper-coagulable state.

   DVT prophylaxis must be given to all elderly patients undergoing orthopedic or pelvic operations who are likely to be confined to bed for more than a day, especially those having malignancy as they are at high risk of DVT and pulmonary embolism. Either standard unfractionated heparin (5000 units SC BD or TDS) or low molecular weight heparin should be given. Respiratory distress, unexplained bronchospasm, hypotension or hypoxia occurring during or a few days after surgery should raise the suspicion of pulmonary embolism. Any atypical pneumonia not responding to antibiotics, tachycardia or dry cough with the classic triad of dyspnea, hemoptysis and pleural pain should warn an anesthetist of a possible pulmonary embolism. ABG may show hypoxia and hypocapnea. The ECG may show a $S_1Q_2T_3$ pattern (very rarely), and the X-ray chest may show a cut-off of the pulmonary vasculature, pulmonary infarction or a wedge shaped opacity. However, both ECG and X-ray are commonly normal in most patients with PE.

Intravenous heparin should be started if PE is suspected, while investigations such as V/Q scan, echocardiography (to rule out right-sided dysfunction), spiral CT scan or pulmonary angiography are carried out. Treatment consists of heparin infusion. Hemodynamically unstable patients may be considered for thrombolysis up to two weeks after the onset of PE, but you may not be able to give it in immediate postoperative period. In severe cases, in appropriately equipped centers, surgical embolectomy can be performed.

What other precautions should to be taken intra-operatively?

Thromboprophylaxis: Low molecular weight heparin or unfractionated heparin or Fondaparinux are commonly prescribed to these patients. The timing of administration of heparin should be adjusted so that it does not interfere with neuraxial blocks. Thromboembolism stockings or intermittent compression devices should be used in the perioperative period as well. Early surgery, regional anaesthesia and mobilisation reduce the risk of DVT.

Antibiotics: Appropriate antibiotic proplylaexis according to the local hospital protocols should be used.

Pressure care: Elderly are prone to damage due to pressure. They should be positioned for surgery carefully and pressure points be padded adequately to avoid pressure sores and neuropraxia. The skin is thin, fragile and liable to be damaged by minimal trauma like removal of dressings, ECG electrodes, diathermy plates, and moving of the patient; hence utmost care should be taken.

Thermoregulation: Elderly patients are prone to hypothermia in the perioperative period especially in the intraoperative period. Adequate warming strategies should be employed to maintain temperature. Fluid warmers, convective warming blankets should be routinely used.

Intravenous fluids: These patients are at high risk of hypovolemia due to blood loss. Active fluid management starting in the pre-operative period is essential to maintain blood pressure and urine output. Ringer lactate solution is usually used however, saline may be used to maintain preload. Plasma expanders (starch etc.) may be required when blood products are still to be given. CVP often fails in providing information regarding cardiac preload and it does not predict fluid responsiveness. In this situation assessing fluid responsiveness with Stroke Volume Variation (SVV) is helpful.

What care should be taken in the postoperative period?

As most of the patients are elderly and with multiple co-morbidities, they are at relatively high risk of complications and hence may require a prolonged period of monitoring in the postoperative period.

- *Analgesia:* Analgesia requirements vary from patient to patient especially during remobilization. Single shot peripheral nerve blockade does not last long. In addition 1 gm acetaminophen IV or oral as applicable, administration should continue. Continuous infusion of local anesthetics helps in relieving pain during the postoperative period. NSAIDS and opioids; intravenous or oral, should be used with caution. Pain scores should be regularly charted to evaluate the treatment.
- *Oxygen:* Oximetry assessment should guide oxygen therapy. Some studies suggest oxygen therapy for 72 hours in the postoperative phase. Mobilization helps in improving oxygenation and respiratory function.
- *Fluid balance:* Early oral fluid intake should be encouraged as soon as possible. Urinary catheters should be removed at the earliest to decrease the risk of urinary tract infection.
- *Wound care:* Avoiding staplers for skin closure is suggested to decrease the rate of infection.
- *Postoperative cognitive dysfunction/acute confusional state/ delirium:* This is common and interrupts rehabilitation of these patients. Prevention is the best treatment of postoperative cognitive dysfunction. Optimising analgesia, nutrition, hydration, electrolyte balance, appropriate medication, bowel habit and mobilisation, in conjunction with identifying and treating complications such as chest infection, silent myocardial ischaemia and urinary tract infection helps in preventing delirium. Drugs such as haloperidol or lorazepam may be used to tide over the crisis cautiously.
- *Nutrition:* Most patients are malnourished. The calorie requirement should be calculated for individual patients and supplemented accordingly.
- *Rehabilitation:* This starts post-surgery and continues even after discharge from the hospital. It should be aimed at getting the patients to their pre-injury status. Multidisciplinary approach is required for adequate rehabilitation and recovery and should include including physiotherapists, occupational therapists, social workers, nursing staff and their own relatives.

What preventive measures may be advised to the patients?

1. *Osteoporosis treatment:* Vitamin D and Calcium supplementation are recommended for osteoporosis in the elderly. Vitamin D supplementation, injected or given orally, suppresses parathyroid hormone, increases bone mineral density. It is shown that older people in institutions may sustain fewer hip and other non-vertebral fractures if given vitamin D with calcium supplements.

2. An annual IV infusion of 4 mg zoledronic acid is associated with a reduction in rate of new clinical vertebral and non-vertebral fractures and may improve survival after a low-trauma hip fracture. Oral alendronate and oral risedronate are associated with reductions in rates of vertebral and non-vertebral fractures.
3. *Hip protectors:* They may reduce the risk of hip fracture in institutionalized patients, but not in community-dwelling older people. Patient acceptance of hip protectors and adherence to their use remain poor due to discomfort and practicality.

What ethical issues should be considered?

Ethical issues arising while treating elderly patients with hip fracture are:

- *Consent:* Elderly patients may have mild to severe cognitive impairment. Also, the ability to assimilate information and communicate decisions may be impaired by poor vision, hearing or speech. Thus, if the patient is not in the capacity to consent for surgery, next of kin or the legal guardian should be consulted about treatment options and decision made.

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27 Cataract

S Bakshi, R Gehdoo

A 75-year-old lady is posted for a cataract surgery with an Intraocular lens implant.

How would you evaluate this patient preoperatively?

This patient is an elderly female and expected to have old age related illness which should be sought for accordingly. This aspect has been dealt with in detail, in a separate chapter "Anesthetic considerations in a geriatric patient". In short, in elderly patients ask for the following comorbidities:

1. Hypertension: duration, medication, effort tolerance, h/o ischemic heart disease (IHD), any anticoagulant therapy.
2. Diabetes: duration, medications, any end organ damage or autonomic dysfunction if present.
3. Respiratory problem: symptoms, duration, medication, dyspnea on lying down would make the patient uncomfortable under regional.
4. Orthopedic problem: degenerative spine (cervical), joint pain, back pain, which prevents patient from lying down for a long time.
5. Central nervous system (CNS) and psychiatric illness: In elderly there is increased incidence of functional illness and emotional disturbances. Alzheimer disease, titubation (involuntary head movements) could pose a problem during surgery under regional anesthesia.

Also note: if patient has deafness, dentures, language difficulties, dizziness or claustrophobia.

What preoperative investigations you would ask for this procedure?

Cataract surgery is a relatively low risk surgery in a high risk patient. There exists a controversy in the extent of investigations to be done for a patient undergoing this surgery under regional anesthesia or monitored anesthesia care (MAC). As per the 2002 American Society of Anesthesiologists (ASA) preoperative testing advisory no tests are required for minimally invasive surgery, if the patient is clinically fit. Hence, the number of investigation would depend upon anesthesia technique, and institutional guidelines.

In cases of planned general anesthesia a detailed work up as per associated medical illness would be needed. In pediatric cases, echocardiography to look for cardiac involvement and X-ray neck lateral to look for vertebral anomalies should be performed in addition to a complete hemogram. In general, cataract surgery is a planned case. However, it is of urgent nature when;

1. Intraocular tension (lens induced glaucoma) is raised: These patients are generally started on tab. acetazolamide or inj mannitol to reduce intraocular pressure (IOP). Both drugs cause electrolyte imbalance.
2. In infants, to prevent suppression amblyopia.

What is the difference between preoperative evaluation of this patient as against a pediatric patient coming for cataract surgery?

In cases of congenital Cataract:

One should note forget the details of ANC (maternal rubella), antenatal period e.g. birth history. Often, the eye finding is a part of a syndrome. History and investigations to rule out other system involvement is a must.
Common syndromes include:

- Down's syndrome: to rule out heart defects, thyroid problem, seizures, difficult airway in presence of macroglossia is common.
- Galactosemia: Galactose intolerance affects liver, kidney, brain and eyes.
- Other syndromes are congenital rubella syndrome, Pierre Robin Syndrome, Chondrodysplasia syndrome, Lowe syndrome, Trisomy 13.

What are the various anesthetic techniques that may be used in these patients?

Choice of Anesthesia depends upon:

- Surgical choice
  - need for complete akinesis
  - duration of surgery
  - extent of surgery
- Patient factors
  - Age and patient's co-operation for the procedure
  - Associated medical conditions.

Anesthetic techniques available are:

- Regional
  - Retrobulbar block +/– Facial block
  - Peribulbar block
  - Subtenon's block
  - Topical anaesthesia
- Regional + Monitored anesthesia care
- General anesthesia +/– Regional

What are the advantages of local blocks over general anesthesia?

1. Patients can be safely discharged home on same day
2. Produce good akinesia and anesthesia
3. Minimal influence on intraocular pressure
4. Require minimum equipment.

What are the contra-indications to blocks?

1 Procedures lasting significantly more than 90 minutes
2 Bleeding or coagulation disorders
3 Perforated globe
4 Allergy to local anesthetics
5 Disorientation or mental impairment
6 Inability to lie flat.

Describe relevant anatomy with nerve supply of the eye ball?

Anatomy: Each orbit has the shape of an irregular pyramid with its base at the front of the skull and its axis pointing poster medially towards the apex. The depth of the orbit measured from the rear surface of the eyeball to the apex is about 25 mm (range 12–35 mm). The axial length (AL) of the globe (eyeball) is the distance from the corneal surface to the retina and is often measured preoperatively. An axial length of 26 mm or more denotes a large eye, indicating that great caution is necessary as globes longer than this are easier to perforate during regional anesthesia.

The orbital fat is divided into central (retrobulbar/intracone) and peripheral (peribulbar/pericone) compartments by the cone of the recti muscles. The central space contains the optic, oculomotor, abducent and nasociliary nerves. The peripheral space contains the trochlear, lacrimal, frontal and infraorbital nerves. All the motor and sensory nerves can be blocked by an injection into the orbital fat.

Nerve supply to the eyes: The motor nerve supply to the extra-ocular muscles is easy to remember using the mnemonic $LR_6(SO_4)_3$—lateral rectus by the sixth (abducent) cranial nerve, superior oblique by the fourth (trochlear) and the remainder by branches of the third (oculomotor) nerve.

Table 27.1 Summary of sensory nerve supply

Sclera and Cornea		Short ciliary nerves Long ciliary nerves
Conjunctiva	Superior	Supraorbital nerve Supratrochlear nerve Infratrochlear nerve
	Inferior	Infraorbital nerve
	Lateral	Lacrimal nerve (with contribution from zygomaticofacial nerve)
	Circumcorneal	Long ciliary nerves
Periorbital skin		Supraorbital Supratrochlear Infraorbital Lacrimal

The parasympathetic supply is from the Edinger -Westphal nucleus accompanying the 3rd nerve to synapse with the short ciliary nerves in the ciliary ganglion. The sympathetic fibers are from T_1 (the first thoracic sympathetic outflow) and synapse in the superior cervical ganglion before joining the long and short ciliary nerves.

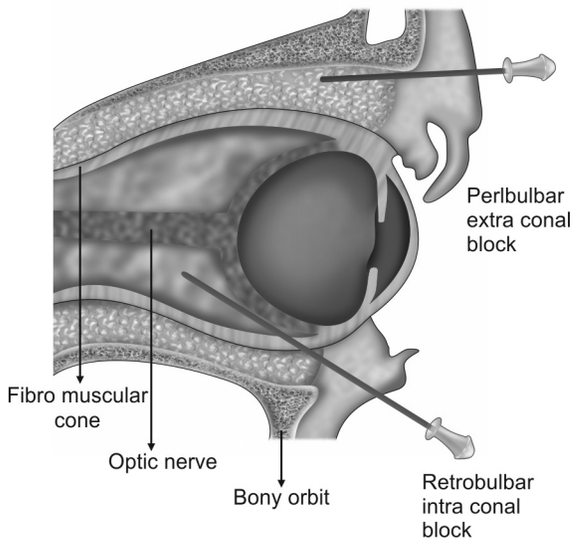

Fig. 27.1 Peribulbar and retrobulbar needle position

Discuss peribulbar Block (Peri-Cone).

Advantages

Less incidence of optic nerve damage. The incidence of retrobulbar hemorrhage and perforation is the same. Facial block is not needed.

Disadvantages

Since the local anesthetic is placed outside the muscle cone the concentration around the optic nerve may not be sufficient to abolish vision completely. Some light perception will therefore remain; however the patient is not able to see the operation. Periorbital ecchymosis is common. Larger volumes of LA needed.

Procedure

The block is administered by two injections, first through upper lid (at junction of medial one third and lateral 2/3rd) and second through the lower lid (at junction of lateral one third and medial two third). After orbital injection Honan pressure cuff ball can be applied on eyeball for sustained pressure. Assessment of the block is usually judged after an interval of 10 minutes. The signs of a successful block are:

- Ptosis (drooping of the upper lid with inability to open the eyes)
- Either no eye movement or minimal movement in any direction (akinesia)
- Inability to fully close the eye once open.

Compare retrobulbar and peribulbar block with regards to technique, advantage, disadvantage and associated complications.

Retrobulbar block (intra-cone)

The local anesthetic is delivered within the muscle cone itself, behind the globe. It is aimed at blocking the ciliary ganglion, ciliary nerves, cranial nerves II, III, VI. Cranial nerve IV is not affected since it lies outside the muscle cone. In the accompanying figure, A shows the direction for peribulbar block, while B shows the direction for retrobulbar block.

Procedure: A 24 gauge 3.5 cm long needle is used. Entry: Transcutaneously at junction of middle and lateral third of lower orbital margin. Once you feel the first pop through the orbital septum (around 15 mm), angle 45° medially and 45° degrees superiorly towards the apex of the orbit until the second pop through the muscle cone is felt.

Facial Block

It is necessary to block the facial nerve which supplies orbicularis occuli muscle, so that patient cannot squeeze the eyelids during surgery. It is needed in most cases with retrobulbar block. A variety of techniques have been described for this. These are:

- *Van Lint Method:* Nerve block close to outer canthus , a wheal is raised 1 cm below and behind the lateral canthus. The needle is passed through the wheal upward towards the temporal fossa and drug injected while withdrawing, the needle is turned medially and downwards towards the

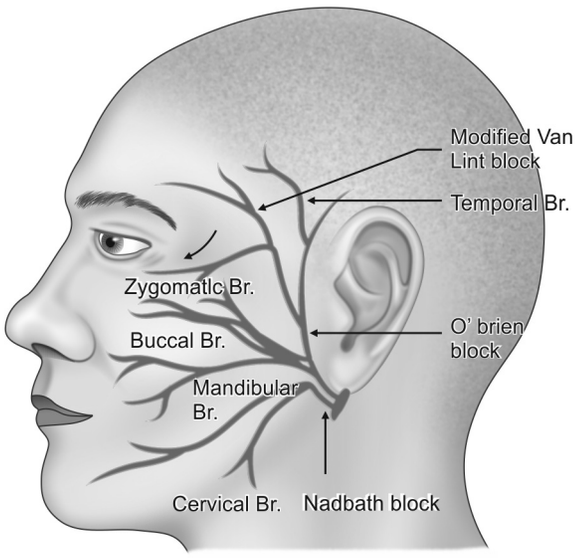

Fig. 27.2 Major approaches to blocking the facial nerve

infraorbital foramen and LA injected. Lastly LA is injected along the lower margin of zygoma

- *O'Brien method:* Paralysis of VII nerve, anterior to the condyloid process of the mandible. The needle is inserted directly to hit the bone and local anesthetic is injected as the needle is withdrawn.
- *Nadbath block:* The facial nerve is blocked at the stylomastoid foramen.

What are the complications of the retrobulbar Block?

- **Retrobulbar Hemorrhage** is the most common complication seen and is due to inadvertent puncture of vessels within the retrobulbar space. Signs are proptosis and a palpable increase in intraocular pressure. Subconjunctival blood and eyelid ecchymosis may be seen as the hemorrhage extends anteriorly. Retrobulbar hemorrhage can lead to other complications such as central retinal artery occlusion and stimulation of the oculocardiac reflex. It is best to postpone surgery for 2–4 days if this complication occurs.
- *Bradycardia* due to oculocardiac reflex can occur several hours later in the event of an expanding hemorrhage. Intravenous atropine is the treatment of choice (0.07 mg/kg)
- *Central Retinal Artery Occlusion* can result from retrobulbar hemorrhage or injection of LA into optic sheath and may result in total loss of vision if not treated. A deep lateral canthotomy or an anterior chamber paracentesis to decompress the orbit may be needed.
- *Puncture of the Posterior Globe:* More likely in patients with a severely myopic eye ("long eye") and in patients with staphyloma. Repeated anesthetic injections using long, sharp needles increases the risk of globe penetration. Perforation usually occurs in inferolateral quadrant. Retinal detachment and vitreous hemorrhage are common sequelae with potential for visual loss. Accidental forceful local anesthetic injection into the globe is more hazardous and can cause rupture of globe. Pain at time of block, hypotonia, sudden loss of vision, should alert the anesthesiologist.
- *Others*
  - Shivering: Because of absorption of LA along optic nerve sheath into CNS
  - Penetration of the optic nerve
  - Inadvertent brain stem anesthesia: Accidental injection into the CSF due to perforation of the meningeal sheaths that surround the optic nerve.
  - Injection into muscle leading to muscle dysfunction
  - Postoperative ptosis (13%)
  - Allergic reactions.

What are the precautions that can taken to prevent globe perforation?

- Excise caution in myopics and patient with staphyloma
- Repeated injections to be avoided.
- In such cases peribulbar block is to be preferred.
- *Wiggle test:* After retrobulbar needle is in orbit, before any anesthetic is injected the needle is moved from side to side. Any rotation of eye suggest-either the sclera, optic nerve, extraocular muscle has been penetrated. This test will prevent forceful injection of local anesthetic drug into vital areas, though it does not prevent globe perforation in the true sense.

What is a Subtenon's block?

Subtenon or a parabulbar block is block given in the subtenon's capsule or space. A conjunctival incision 2–3 mm in size is made halfway between the inferior limbus and fornix to open into subtenon's space. A blunt cannula is then used to inject anesthetic into posterior subtenon space.

Advantages: Lesser volume is needed. There are less chances of vascular, optic nerve injury.

Is topical anesthesia a suitable alternative?

The terminal branches of cornea and conjunctiva form an intraepithelial plexus. These nerve endings are superficial and vulnerable to the effect of local anesthetics.

Advantages

- Quick
- noninvasive
- Well tolerated by patients
- No complications of other blocks
- Patient has an immediate improvement in vision and eye shield is not needed.

Disadvantages

- No akinesia
- Patient should be motivated and co-operate
- Only a simple uncomplicated cataract can be included
- Expertise surgical hand needed.

What is oculocardiac reflex (OCR)?

Oculocardiac reflex, also known as *Ascher-Dagnini Reflex* is bradycardia due to traction on the extraocular muscles, conjunctiva, orbital structures, and pressure on the globe, retrobulbar block, and ocular trauma. The afferent is carried by trigeminal nerve while vagus nerve brings the efferent arm of the reflex. The afferent pathway follows the long and short ciliary nerves to the ciliary ganglion and then to gasserian ganglion along the ophthalmic division of trigeminal nerve.

These afferent pathways terminate in the main trigeminal sensory nucleus in the floor of the fourth ventricle. The efferent impulses start in the muscles of the vagal cardiac depressor nerve, causing negative inotropic and conduction defects. Oculocardiac reflex can also lead to arrhythmia including ventricular tachycardia and rarely asystole. Hypotension and increased arterial carbon dioxide partial pressure significantly increase incidence of bradycardia during surgery. Incidence of OCR is highest in children if no atropine pretreatment is used. In children, administration of atropine 0.02 mg/kg or glycopyrrolate 0.01 mg/kg prior to commencing surgery is indicated. In adults prophylaxis is not usually indicated.

Treatment

1. Removal of stimulus.
2. IV anticholinergics.
3. Check and deepen depth of anesthesia.
4. Lignocaine if VPCs are a problem.

What is Monitored Anesthesia Care (MAC)?

Monitored anesthesia care (MAC) is a comprehensive physician service that includes all aspects of anesthetic care—pre-procedural visit, intraoperative care, and post procedure anesthetic management. The American Society of Anesthesiologists (ASA) practice guidelines for MAC have been established. These guidelines frequently are consistent with those for general or regional anesthesia and should be followed accordingly. Although MAC is safe when administered by well-trained anesthesia personnel, ASA closed claims data demonstrate that rare and severe injuries or death can occur during MAC. Clinicians must understand the pharmacokinetic and pharmacodynamic principles of drug administration, (e.g. context-sensitive halftime, effect-site half-time) so that cognitive errors can be avoided. Proper drug dosing can optimize patient safety and recovery and discharge times. Therapeutic windows for drugs administered during MAC are much smaller than those for general anesthesia. Sedation with propofol is classified as deep sedation by the ASA. When used in doses consistent with MAC, significant changes occur in airway anatomy and cardiopulmonary physiology. These changes occur even in healthy patients and are amplified in the elderly and in patients with pre-existing systemic disease. Propofol should be administered only by clinicians qualified to rescue patients from any level of sedation, including general anesthesia.

Discuss management of a restless patient undergoing cataract surgery under LA.

- Ask surgeon to stop.
- Recheck vitals including saturation
- Discuss with patient the cause for restlessness and reassure.
- Often patient "feels out of breath" under drapes—in such cases starting of oxygen through mask or cannula is of help. This should be ideally done for all patients. Just lifting the drapes of the face also helps in allaying anxiety and at times preventing rebreathing of CO_2 which is highly probable with the drapes.
- In case patient complains of pain, LA should be repeated in the form of a repeat block or topical drops.
- Intravenous analgesics like fentanyl and anxiolytics can be considered. One has to titrate these drugs by giving small boluses and observing for the desired effect. Also one should be watchful for airway obstruction and respiratory depression.
- At times if patient does not cooperate, propofol can be used. Oropharyngeal airway or LMA should be ready for insertion.
- In a few rare instances, if above interventions do not help, administration of general anesthetic and relaxant with full control of airway with endotracheal tube *in situ* may be necessary.

Recommended Reading:

1. Miller's Anesthesia. 6th edition.
2. Ophthalmic Anaesthesia By Chandra M.Kumar.
3. Parson's diseases of the eye.

28 Morbid Obesity

V Patil, M Joshi

A 53-year-old female patient weighing 105 kg with height of 157 cm presents with history of neck swelling. The swelling has gradually increased over last 5 months and her voice has changed since 4 months. She has developed dysphagia to liquids and solids since 1 month. She also gives history of choking sensation intermittently. She always sleeps in semi-recumbent position. She was seen by surgeons and FNAC of lesion was performed which showed poorly differentiated thyroid carcinoma.

How do you define obesity?

Obesity is defined as a body weight that exceeds the expected or ideal weight by more than 10%, taking into account height, age, body built, and sex. An alternative definition is based on fat content of the body: patient is said to be obese when more than 25% of the body weight in males or 30% in females is attributable to fat (normally 15–18% body weight is fat). Morbid obesity is defined as actual weight exceeding twice the ideal body weight. The table below gives division of patients according to body mass index (BMI) or Quetelet index. It was described by Lambert Adolphe Jacques Quetelet in 1835. It is calculated by dividing the patient's weight in kilograms by his height in meters square (Table 28.1).

BMI = weight (kg)/[Height (m)]^2.

Table 28.1 Categories of obesity

BMI (kg/m^2)	Category
< 18.5	Underweight
18.5–24.99	Normal
25–26.9	Overweight
27–30	Mild obesity
> 30	Moderate obesity
> 35	Severe obesity
> 40	Morbid obesity
> 50	Super obesity

How will you proceed with the preanesthesia check up in this patient? What are the associated diseases linked to obesity which can alter anesthesia management?

This patient's BMI is 42.6 kg/m^2 and she falls in the category of morbid obesity. Patients with morbid obesity have long list of comorbidities which are of concern to the anesthesiologist (Table 28.2). Careful history should be taken to ascertain or rule out presence of comorbidities. When necessary, leading questions can be asked. The medication history also can be a guide to know patients comorbidities. Always take detailed history to find out or rule out conditions that may not have been diagnosed; before morbidly obese patients are subjected to surgery and anesthesia. Most important among these are:

1. History of symptoms of DM: Ask for polyuria, polydipsia, nocturia, frequent fungal skin infections or recurring conjunctival, gum or urinary tract infections, irritability and mood changes, blurred visions, tingling and numbness in legs, slow healing of cuts and wounds, etc.
2. History of airway involvement: habitual snoring, frequent interruptions in breathing during sleep as reported by others, excessive day time sleepiness, hypertension, nocturnal choking, waking unrefreshed, and morning headaches.

Table 28.2 Diseases linked to obesity

System	Disease
Cardiac	Hypertension
	Atherosclerosis
Respiratory	Sleep apnea
	Asthma
	Pulmonary embolism
	Pulmonary hypertension
	Obesity hypoventilation syndrome
Endocrine	Diabetes, gout, glomerulosclerosis
Hepatic	Fatty liver
	Cholelithiasis
Vascular	Varicose veins
	DVT
Skeletal	Degenerative arthritis, carpel tunnel syndrome
Oncology	Carcinoma endometrium
	Carcinoma breast
	Carcinoma prostate
	Carcinoma colon

3. Sleeping habits: Many patients are unable to lie flat for several years, and may routinely sleep sitting up in an armchair. An assessment of the ability to tolerate the supine position may reveal profound oxygen desaturation, airway obstruction, or respiratory insufficiency.
4. Dietary habits: It is also important to recognize patients with nutritional deficiency in the face of obesity as most of the times these patients are on some sort of weight reduction diet which may give rise to various nutritional deficiencies.
5. The drug history: Ask if patient is consuming amphetamine-based appetite suppressants as these contribute to increased perioperative cardiac risk.

Since morbidly obese patients have restricted physical activity, one cannot rely on exercise tolerance as measure of cardiorespiratory reserve. For cardiac assessment follow AHA guidelines on perioperative cardiac evaluation for noncardiac surgery. When it comes to type of testing there is no consensus on which test is best. The first decision would be whether or not patient can exercise sufficiently to get 85% of predicted maximal heart rate. If they cannot; then a pharmacological stress test like dobutamine stress echocardiography or adenosine dipyridamole scan would be indicated. Studies have shown that non invasive testing is less sensitive and even less specific in obese compared to nonobese individuals. If the noninvasive test is positive then invasive testing in the form of a cardiac catheterization is indicated.

How is cardiovascular system function altered with increasing weight?

Ischemic heart disease, hypertension, and cardiac failure are the most common occurrences with obesity. The adipose tissue has a resting blood flow of 2 to 3 mL/100 g/min, but it can increase up to 10-fold after meals. However, with increasing obesity the perfusion per unit mass decreases. It falls from 2.36 mL/min to 1.53 mL/min when the percentage of fat increases from 20% to 36% of the body weight, and so the increase in cardiac output is not directly proportional to the total fat. Thus each kilogram of fat acquired above IBW requires an increase in cardiac output of 20–30 mL. This increased cardiac output is mainly achieved through increase in stroke volume and not through increase in heart rate. The left ventricle dilates in response to this volume overload; and over time develops an eccentric type of hypertrophy to keep the wall stress normal. The left atrium also enlarges in obese individuals and this is initially caused by the increased blood volume and venous return. Later, other factors like left ventricular hypertrophy and diastolic dysfunction may also be responsible for increased left atrial size. Right ventricular structure and function may be similarly affected by the above mentioned morphologic and hemodynamic alterations and by pulmonary hypertension related to the sleep apnea or obesity hypoventilation syndrome.

Arrhythmia: There is an increase in the incidence of sudden cardiac death and arrhythmias in obesity. This may be due to increased levels of catecholamine. In addition, increased free fatty acid levels in the obese may also affect repolarization.

Coronary Artery Disease: Obesity is an independent predictor of coronary artery disease, as observed in many studies and is due to accelerated atherosclerosis.

Hypertension: Blood pressure is the product of cardiac output and systemic vascular resistance, and cardiac output is increased in obese patients because of increased blood flow to the adipose tissue. Blood pressure increases by 6.5 mm Hg for every 10% increase in body weight. Generally, systemic vascular resistance should be low in obese individuals because of the increased cross-sectional area of the vascular bed. However in addition to store fat, adipose tissue also acts as an endocrine organ and synthesizes variety of peptides and nonpeptide compounds like interleukin-6 (IL-6), plasminogen activator inhibitor-1, resistin, lipoprotein lipase, etc. which lead to low-grade inflammation and over-activity of the sympathetic nervous system. In addition a disordered sleep pattern may also increase the systemic vascular resistance. Thus, with increasing severity of obesity, hypertension becomes more prevalent.

What is obesity cardiomyopathy?

Cardiomyopathy in obese individuals is caused by a direct effect of obesity on the heart. Initially, various tissues of heart, like the sinus node, atrioventricular node, right bundle branch, and the myocardium, are infiltrated by fat cells. These can cause conduction defects like sinoatrial block, bundle branch block, and, rarely, atrioventricular block. Subsequently, such infiltration may cause pressure-induced myocyte degeneration. Accumulation of triglycerides in myocytes can directly cause cell dysfunction because of lipotoxicity. Adipose cells also secrete locally active molecules like adipokines, which indirectly damage adjacent myocytes. In addition increased cardiac output and blood volume leads to ventricular dilatation and subsequently hypertrophy of left ventricle which then can lead to diastolic dysfunction (failure to relax properly). If the wall thickening fails to keep pace with chamber dilatation systolic dysfunction (failure to contact properly) can occur as well. This disorder is called as obesity cardiomyopathy and can occur in absence of systemic hypertension and underlying organic heart disease. This is the reason why preoperative echocardiography is indicated in morbidly obese. The predominant causes of death in those with obesity cardiomyopathy are progressive congestive heart failure and sudden cardiac death.

Describe types of pulmonary involvements in morbid obesity. What is the difference between Obesity Hypoventilation Syndrome (OHA) and Obstructive Sleep Apnea (OSA)?

Along with cardiac disorders, various pulmonary disorders are of major concern to anesthetists. Most amongst these are OHA, OSA and cor pulmonale. In addition; patients with morbid obesity usually have decreased pulmonary reserve even if they do not have specific pulmonary disorder. These patients also have an increased incidence of restrictive pulmonary disorder. Even if consequences of pulmonary dysfunction are less severe than cardiac ones they are more common. Morbidly obese patients have reduced FVC, FRC and TLC with decreased expiratory reserve volume, and increased respiratory resistance. Due to these reasons; some amount of pulmonary insufficiency is always present in morbidly obese. These changes are of particular interest to the anesthetist; as added decrease in these values due to anesthesia, supine position and postoperative pain; can worsen respiratory function in the postoperative period. Also these patients have decreased chest wall compliance due to fat deposition on chest wall. All these changes lead to decreased respiratory reserve, difficulty in ventilation and faster desaturation.

OSA: The presence of OSA indicates high chance of having other sequel like hypertension, LVH and RVH. Diagnosis is made based on Polysomnography (PSG), commonly known as sleep study. OSA can be suspected from clinical symptoms and signs. History such as habitual snoring, interrupted nocturnal breathing during sleep as reported by others, excessive day time sleepiness, arterial hypertension, nocturnal choking, waking unrefreshed, morning headaches, are frequently seen with OSA. Abnormal anatomy of the pharynx, increased collapsibility of the upper airway and defective airway reflexes all lead to development of OSA. Obesity is usually associated with OSA due to the deposition of fat in the lateral pharyngeal wall; resulting in decreased patency of the pharynx and increased the risk of airway collapse; particularly during sleep, when there is relaxation of the pharyngeal dilator muscles. Decreased upper airway activity with REM sleep also leads to pharyngeal narrowing. Vibration of upper airway structures due to turbulent flow results in snoring. Also the pharynx tends to collapse due to Bernoulli effect; with partial or complete obstruction. Hypercapnia and hypoxia which follows leads to awakening and opening of pharynx due to increased upper airway muscle activity. The improved breathing decreases carbon dioxide tension and improves oxygen saturation. The cycle repeats with sleep onset. These patients are very sensitive to narcotics and sedatives. While many anesthetists are worried about induction and intubation in these patients, truly challenging period is extubation and postoperative period. It may be very difficult to balance postoperative pain relief and apnea. Patients given more narcotics during intraoperative period have high incidence of postoperative complications. These patients commonly have low baseline PO_2 but oxygen supplementation does not improve PO_2 as they have longer periods of apnea and respiratory center is stimulated by hypoxia and not hypercarbia.

OHS: Also known as Pickwickian syndrome is distinct from OSA and was originally described as combination of morbid obesity, hypersomnolence, plethora and edema. While in OSA patient breaths normally while awake, in OHS patient is hypercapneic at rest as well as while awake. The cause of OHS is unknown but likely involves combination of disorder of respiratory center and decreased chest wall compliance. It is usually diagnosed when patient's BMI is more than 30 kg/m^2, and they have resting hypercapnea (PCO_2 more than 45 mm Hg) in absence of other causes of hypoventilation; such as airway obstruction, drug overdose, neuromuscular disorders, etc.

How does one diagnose OSA? What is polysomnography (sleep study)?

Polysomnography (PSG) is indicated for the diagnosis of sleep related disorders. Standard polysomnography is performed in a sleep laboratory, hospital, or other dedicated unit under the supervision of a sleep technician. It includes measurements of O_2 saturation, electrocardiography (ECG), electroencephalography (EEG), electromyography (EMG), electrooculography (EOG), airflow, and respiratory effort measurements. Nasal and oral airflow can be measured using pressure transducers, or a thermocouple, fitted in or near the nostrils. The study identifies sleep architecture, number and degree of arousals, number and type of apneic episodes, episodes of oxygen desaturation and severity, cardiac arrhythmias, limb movements, disorders associated with REM sleep, and seizure activity.

Indications for sleep studies

1. Witnessed apnea during sleep greater than 10 seconds in duration; OR
2. Any combination of two or more of the following:
   - Excessive daytime sleepiness as evidenced by one or more of the following:
     - Inappropriate daytime napping (e.g. during driving, conversation, or eating);
     - Sleepiness that interferes with daily activities; (The following should be ruled out as a cause for these symptoms: poor sleep hygiene, medication, drugs, alcohol, hypothyroidism, other medical diagnoses, psychiatric, or psychological disorders, social or work schedule changes.)
     - An Epworth Sleepiness Scale score (normal 0–9, maximum 24) greater than 10;
   - Persistent or frequent socially disruptive snoring;
   - Obesity (BMI greater than 30 kg/m^2) or hypertension;
   - Choking or gasping episodes associated with awakenings. OR
3. Symptoms suggesting narcolepsy, e.g. sleep paralysis, hypnagogic hallucinations, cataplexy. OR
4. Violent or injurious behavior during sleep; OR
5. Other situations (if nocturnal pulse oximetry suggests nocturnal oxygen desaturation) such as:
   - Unexplained right heart failure;
   - Unexplained polycythemia;
   - Presence of, or increase in cardiac arrhythmias during sleep;
   - Unexplained pulmonary hypertension. OR
6. Excessive daytime sleepiness together with witnessed periodic limb movements of sleep; OR
7. Unusual or atypical parasomnias based on patient's age, frequency, or duration of behavior; OR
8. Patient's with moderate or severe congestive heart failure, stroke/TIA, coronary artery disease, or significant tachycardic or bradycardic arrhythmias who have nocturnal symptoms suggestive of a sleep related breathing disorder or otherwise suspected of having sleep apnea.

What is Apnea-Hypopnea Index (AHI) and how do you grade it?

This is an index of total number of complete cessations (apnea) and partial obstructions (hypopnea) of breathing occurring per hour of sleep. These pauses in breathing last at least for 10 seconds and are associated with a decrease in oxygenation of the blood. In general, the AHI can be used to classify the severity of disease (mild 5–15, moderate 15–30, and severe greater than 30). It is also known as respiratory disturbance index or RDI. The effects of sleep apnea are not only functional and psychological but they also modify physiology. People with sleep apnea have increased sympathetic activity and endothelial and metabolic dysfunction. All this leads to higher incidence of cardiovascular complications. These patients are at increased risk for high blood pressure, ischemic heart disease, stroke, and congestive heart failure. As the AHI score increases, the risk of cardiovascular events increase. So, the AHI is a good indicator of disease severity and the urgency to treat a patient increases with increasing AHI.

Patient gives history of hypertension for last 9 years for which she is on amlodipine 5 mg twice daily and atenolol 25 mg once daily. On direct questioning she says she is regular with her treatment and follow-up and that her blood pressure remains on higher side of normal. She is also diabetic being treated with oral hypoglycemic agents. She gives history suggestive of obstructive sleep apnea:

H/o snoring

H/o witnessed apneic spells by husband at night

H/o daily afternoon nap of 4 hours

She fell 2 years ago and suffered from Potts fractures of left leg and tibial and patellar fracture of right knee. She had undergone surgery under GA and postoperative course was uneventful. Following fracture she complains of stiff and painful right knee due to which she has restricted mobility, unsteady gait, and she walks with walker. Following this surgery she had developed swelling in both legs; with darkening of skin and tingling in her both lower legs. She was investigated for this—compression venous ultrasound was performed; which was negative for DVT. She was started on pentoxifylline orally.

How should a patient with morbid obesity be investigated for any surgery? What investigations would you advice for this patient?

Investigations should be tailored to the individual patient, depending on comorbidities and the type and urgency of surgery. A full blood count, electrolytes, renal and liver function tests, and blood glucose form a basic set of investigations. Arterial blood gas analysis will be useful in those suspected of respiratory comorbidity (OSA, OHA, large collar size, and other pulmonary disease). Many times cause of obesity is hypothyroidism and from clinical history and examination it is very difficult to rule out hypothyroidism. Hence, thyroid functions form a part of baseline investigations. As cardiac dysfunction of varying degrees is quite common in long standing morbid obesity, a preoperative ECG is essential to look for significant rhythm disturbances, left ventricular hypertrophy and cor pulmonale, and as a guide to indicate need for more extensive cardiac investigation. Studies have shown that in patients with evidence of right ventricular hypertrophy or cor pulmonale a period of elective nocturnal noninvasive ventilation before elective surgery is beneficial. Nocturnal noninvasive ventilation is effective in relieving right heart failure, day-time somnolence, and pulmonary hypertension. Chest X-ray may be used to assess cardiothoracic ratio and evidence of cardiac failure. Younger patients, those at the lower end of the BMI range, those with a good exercise tolerance, and those with a benign fat distribution need not be tested further. But some group of patients will require detailed preoperative cardiac evaluation. These patients include: patients with limited performance status, patients with hypertension, those with sleep apnea and hypoxia hypertension and those with history of edema feet/face. Patients with these features should undergo transthoracic echocardiography to estimate systolic and diastolic function, valvular status and chamber dimensions, although good images may be difficult to obtain by the transthoracic technique. In patients in whom echocardiographic window is unsatisfactory, radionuclide ventriculography may be used to assess LV and RV morphology and function. Exercise ECG testing may be impractical, but a short walk along the ward or an attempt at climbing a flight of stairs can give useful functional information.

Noting breath holding time would help in assessing combined cardiorespiratory reserve.

Pulmonary function tests may reveal a restrictive defect, but are not performed on all patients unless there is a specific indication. Morbidly obese patients have reduced FVC, FRC and TLC with decreased expiratory reserve volume, and increased respiratory resistance and hence one should expect some amount of pulmonary insufficiency in all morbidly obese. Studies have failed to show any impact of PFTs on perioperative outcome in obese patients but can be used to predict risk of postoperative pulmonary complications.

Obesity is an independent risk factor for deep venous thrombosis (DVT) and pulmonary embolism, and all the patients should be screened for presence of asymptomatic DVT using compression Doppler ultrasound.

Role of sleep studies in obese patients: In patients giving classical history of OSA; doing sleep studies may not add much to the preexisting knowledge. But in other obese patients PSG might help in identifying population at risk. From present evidence it is unclear whether routine PSG would improve safety and outcomes.

Since this patient has history of long standing hypertension, diabetes and OSA, she has high risk for underlying cardiac dysfunction and would need cardiac work up in addition to ECG in the form of a transthoracic echocardiogram. Drug history (pentoxifylline) suggests that she might be suffering from peripheral vascular disease; which makes her a strong candidate for underlying coronary artery disease as well; which needs to be kept in mind. A dobutamine stress echocardiography should be asked for.

This lady also has big neck mass and associated compressive symptoms, hence PFTs with flow volume loop would help in identifying tracheal compression. Also ABG should be done to know status of oxygenation.

This patient also gives history of compressive symptoms (dysphagia); further investigation with CT neck and chest would help to know tracheal compression, deviation and retrosternal extension.

She also has very limited mobility; hence chances of DVT are high. She should undergo lower limb compressive ultrasonography to detect presence of DVT as part of preoperative evaluation.

Potential venous and arterial access sites should be evaluated during the preoperative examination, and the possibility of invasive monitoring should be mentioned to the patient. Since this patient has such a strong history of OSA patient should be told about possible need for postoperative elective mechanical ventilation.

What are common ECG abnormalities associated with morbid obesity?

- Low voltage complexes
- LV hypertrophy with/without strain
- Prolonged QT/QTc

- Inferolateral T wave abnormalities
- Right axis deviation or RBBB
- P pulmonale.

USG neck revealed multinodular goiter with left IJV entrapment. CT scan showed enlarged thyroid, with tracheal and laryngeal compression, mediastinal extension of thyroid, compression of left common carotid artery, occluded Lt IJV, and presence of metastatic nodules in both lung fields. Hopkins mirror examination showed left vocal cord to be fixed. Her thyroid function tests, hematology and biochemistry investigations were normal. ABG on air showed PaO_2: 69 mm Hg, SpO_2: 95%, $PaCO_2$: 32 mm Hg. 2D- Echocardiography showed LVEF of 60%, no RWMA and severe PAH (PASP 65 mm Hg). Spirometry showed moderate restriction. Flow volume loop did not reveal any significant tracheal compression. Barium swallow showed decreased distensibility of left pyriform fossa with filling defect.

What is the role of preoperative weight reduction? Can this patient be put on weight reducing diet before subjecting to surgery? What are advantages and side effects of preoperative weight loss?

Even though weight reduction immediately before surgery has not been shown to reduce perioperative morbidity and mortality; it may reverse some of pathophysiological changes in the obese patients. Even mild weight reduction before surgery may reduce liver size facilitating surgical approach to diaphragmatic hiatus (for bariatric surgery). In addition decrease in intra-abdominal pressure could improve conditions for mechanical ventilation.

For preoperative weight reduction it is important that adequate proteins should be provided (1.5 g/kg ideal body weight) so that lean body mass will be maintained. Diets in preoperative period should be properly supervised as it can dangerously influence perioperative outcome. Starvation diets, low and very low calorie diets and protein deficient diets can lead to development of severe cardiac arrhythmias and even sudden death. The implementation of low fat, low calorie diet typically results in an increased intake of vegetables and low intake of milk and dairy products, cereals, fruits, meat products, and added fats. This may result in a significant decreased daily intake of phosphorous, zinc, magnesium, iron as well as vitamins B_1, B_2 and B_6. All these deficiencies; particularly B complex; should be replenished before surgery. However reduction in simple carbohydrates reduces glycemia and lipogenesis and should be routinely recommended. A particularly compromised group is patients who have had prior bariatric surgery. They must always be considered to have latent malnutrition and thus they present a greater risk for postoperative complications.

Since this patient has a large thyroid with compressive symptoms and malignancy, she is not a candidate for preoperative weight reduction and surgery should be conducted as soon as possible. However, a dietician should be involved in her perioperative management and a diet modification can be done.

How will you prepare this patient for surgery? What premedication would you use?

1. An important adjunct to dietary management is implementation of physiotherapy and exercise program. This improves glucose tolerance and respiratory capacity. Breathing exercises and incentive spirometry should be a routine practice in order to improve perioperative oxygenation and prevent postoperative atelectasis.
2. No sedatives or narcotics. Use councelling and reassurance technique. Patients with high level of anxiety can be given very light premedication but avoid narcotics and monitor them carefully.
3. This patient has very strong history of obstructive sleep apnea. Also she has a large thyroid with mediastinal extension. Both these factors make her very prone to mechanical airway obstruction in the perioperative period. These patients are extremely sensitive to sedatives and analgesics and may develop apnea or respiratory obstruction following even small doses of these drugs. Hence premedication should be avoided in this patient. She should be observed on previous night for frequency of obstruction and periods of desaturation.
4. 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/m^2 to 2.7 in patients with BMI more than 40 kg/m^2. 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 anesthesia. 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.
5. Antacid prophylaxis, as these patients are prone for aspiration. A significant residual gastric volume and acidic pH is common. Antacids, proton-pump inhibitors, H_2 receptor antagonists, and prokinetic agents are all likely to be of value in the perioperative period. Routine prophylaxis with ranitidine or a proton-pump inhibitor is advisable and can be administered orally at the time of premedication. Sodium citrate (0.3 M) may be given to

patients with significant reflux symptoms; 20–30 minutes before induction.
6. Preoperative antibiotics prophylaxis as recommended for all surgical procedures.
7. Avoid IM injections due to unpredictable absorption.

What are the principles of anesthetic management? What precautions would you take while padding and positioning?

Operation theater (OT) tables have weight limits and overweight patients can lead to table breakage. Whenever possible; use table with proper weight bearing capacity. If that is not available, two OT tables (side by side, with a board across the lower half so the back can still flex to a sitting position) should be used. 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 anesthesia 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.

Obese patients are also more prone for pressure related injuries and proper padding of buttocks and back is necessary to prevent gluteal compartment syndrome which can lead to rhabdomyolysis and renal failure. Patients with surgery longer than 4 hours are prone to develop this syndrome. Prone position is poorly tolerated by these patients and lateral decubitus is better because it keeps abdominal weight off chest. Postoperative bed should have higher weight limit than that used for normal patients.

This patient was scheduled to undergo total thyroidectomy. Clinical examination revealed morbidly obese patient with orthopnea (she couldn't lie supine in bed). She was receiving oxygen via nasal cannula at 3 L/min. Airway assessment showed short bulky neck with Mallampati class IV. Thyromental distance could not be estimated due to the tumor size and due to limited neck movements. The trachea could not be palpated anteriorly except for 1"at the cricoid level.

Why and how should one preoxygenate these patients?

There are two techniques advocated for preoxygenation: breathing 100% oxygen through well fitting face mask for 3 minutes or taking 4 vital capacity breaths with100% oxygen. The purpose of preoxygenation is to fill lungs with 100% oxygen and thus have prolonged apnea time safely. Studies have shown that breathing 100% oxygen for 3 minutes is superior to 4 vital capacity breaths. Rate of desaturation will depend on FRC and BMR. Thus obese patients would desaturate faster as they have very reduced FRC. Rate of desaturation is fastest for morbidly obese; followed by children and septic patients due to increased BMR; followed by normal adults. As incidence of difficult intubation is high with morbid obesity, one can use continuous oxygen insufflation using nasopharyngeal catheter during laryngoscopy. Some recently marketed blades have provision for continuous administration of oxygen during laryngoscopy.

Why do you prefer to induce morbidly obese patients in reclining position? Why do these patients tolerate supine position badly?

FRC is divided as ERV (the volume expired by active expiration after passive expiration) and RV (the volume left in the lungs after maximal expiratory efforts). The closing volume (CV) is the lung volume above RV at which airway collapse occurs during expiration; mainly in the lower lung zones due to gravitational effects. The relation between CV and FRC is determined by the balance between the inward elastic recoil of lungs and outward pull by thoracic cage. In severe obesity CV exceeds ERV due to a decrease in ERV. Some airways are closed during tidal breathing resulting in increase in shunt and arterial hypoxemia. The airway closure becomes more pronounced when these patients assume supine position since the increased weight of chest wall and abdomen exacerbate early airway closure and promote arterial hypoxemia.

How would you induce this patient and how will you manage her airway?

A theater table with an appropriate maximum weight allowance must be used. There must be enough trained and experienced staff in theater to assist with moving the patient quickly, should it become necessary during induction. Standard monitoring should include a correct-sized blood pressure cuff. Venous cannulation can sometimes be difficult and central venous cannulation may be necessary. Direct intra-arterial monitoring should be considered for situations where rapid hemodynamic changes are possible, surgery is prolonged and in patients with cardiorespiratory disease or if noninvasive arterial pressure monitoring is impractical.

Airway management: The choice between awake and asleep intubation is difficult and depends on anticipated difficulties along with experience of anesthetist. Obesity is associated with increased incidence of difficult mask ventilation and difficult intubation. Commonly used anesthetic agents like

propofol, thiopentone, narcotics, benzodiazepines, muscle relaxants can all lead to pharyngeal collapse. Obese patients therefore should be carefully evaluated for difficult airway. History of sleep apnea, limited neck movements, limited mouth opening, large tongue and short thyromental distance all increase risk for difficult airway.

Also in these patients gastric insufflation during ineffective mask ventilation will further increase risk of regurgitation and aspiration of stomach contents. Hence fibreoptic intubation is frequently recommended in these patients. Also alternative plan of securing the airway should be ready in case first plan fails. An emergency tracheostomy is challenging in these patients and may not be practical or safe which further favors awake intubation. Looking at history and clinical examination; this patient should undergo awake fiberoptic bronchoscopy. Patient positioning is of paramount importance before induction, particularly head position. A 'sniffing the morning air' position may be difficult to achieve due to the large soft tissue mass of the neck and chest wall, and a wedge or blanket beneath the shoulders is often beneficial ('ramped' technique).

Difficulties encountered in bag and mask ventilation can be overcome by a four-handed technique. Using anesthesia ventilator and PEEP for mask ventilation help in maintaining FRC and thus improve oxygenation.

Standard use of the 30° reverse Trendelenburg (head up) position during preoxygenation, induction, and emergence from anesthesia is useful. A polio handle and or a long blade may be helpful in intubation in case direct laryngoscopy is planned. Difficult airway management cart should be ready. Efficient help in the form of an additional trained anesthetist, operating room technician, and the surgeon is mandatory during induction and emergence.

What would be your choice of inhalational agent?

For faster induction and rapid recovery, volatile agents should be insoluble in blood with minimal or no metabolic degradation, and low lipid solubility. Rapid elimination and analgesic qualities of nitrous oxide make it a preferred agent. Isoflurane is cheaper compared to desflurane and sevoflurane, but it has a longer washout period and produces slower awakening as compared to other two agents. Desflurane has low partition coefficient (0.42) that allows for faster washout and elimination than sevoflurane and it has even been observed with BIS monitoring that desflurane produces faster awakening. However sevoflurane may be the preferred agent as it causes less tachycardia; which is advantageous in patients with underlying CAD. So one has to decide the agent of choice depending on whether your patient has strong history of OSA. If you want faster awakening, choose desflurane. If your patient has bad CAD, you want better rate control then choose sevoflurane.

How would you manage this patient intraoperatively? What are the ASA task force guidelines for patients with OSA?

Because of their propensity for airway collapse and sleep deprivation, patients with OSA are especially susceptible to the respiratory depressant and airway effects of sedatives, opioids, and inhaled anesthetics. Therefore, in selecting intraoperative medications, the potential for postoperative respiratory compromise should be considered. Select short acting agents (e.g. alfentanil, propofol, midazolam, atracurium), and avoid using long acting agents (e.g. morphine, diazepam or pancuronium)

For superficial procedures, one should consider the use of local anesthesia or peripheral nerve blocks, with or without moderate sedation. Use of ultrasound and nerve locator adds to safety and success of the procedure. If moderate sedation is used, ventilation should be continuously monitored by capnography if feasible; because of the increased risk of undetected airway obstruction in these patients. Consider administering CPAP during sedation to patients. General anesthesia with a secure airway is preferable to deep sedation without a secure airway, particularly for procedures that may mechanically compromise the airway. For all but very short surface procedures IPPV will be needed. High inflation pressures will be required due to decreased compliance and increased resistance. $ETCO_2$ monitor would guide regarding adequacy of ventilation. Dose of muscle relaxants should be guided by monitoring with peripheral nerve stimulator. Unless there is a medical or surgical contraindication, patient at increased perioperative risk from OSA should be extubated while awake. Full reversal of neuromuscular block should be verified before extubation. When possible, extubation and recovery should be carried out in the lateral, semi-upright, or other nonsupine position.

Consider central neuraxial blockade (spinal/epidural) for peripheral procedures. Postoperative shivering increases oxygen consumption, and increases cardiovascular stress. Effective temperature maintenance during perioperative period is important; it also reduces postoperative wound infection. Forced warm air blankets in combination with fluid warmers are effective in preventing perioperative hypothermia.

What intraoperative monitoring would you institute?

Other than standard intraoperative monitoring of ECG, SpO_2, temperature and $ETCO_2$; these patients require invasive

monitoring frequently. Invasive hemodynamic monitoring is indicated in patients with CHF, renal insufficiency, or severe pulmonary hypertension (PA systolic > 60–70 mm Hg) or for procedures with expected large fluid shifts perioperatively. A preoperative echocardiography will be of great help to decide on indications for invasive hemodynamic monitoring.

Appropriate sized noninvasive blood pressure cuff is important for accurate BP measurement. Usual sized BP cuffs are too small and larger cuffs (thigh cuffs) are too wide and extend down past cubital fossa in most of the patients. Also most of these patients have conical shaped arms that are difficult to keep a BP cuff on. Many clinicians prefer routine invasive arterial pressure monitoring, as non invasive pressure may be unreliable due to lack of adequate sized cuff. Intraoperative fluid balance is difficult to assess. The blood volume may be reduced from normal 70 mL/kg. CVP monitoring is helpful with large blood losses; but again one has to consider underlying cardiac dysfunction, RVH or cor pulmonale; where CVP will loose its significance as an indicator of preload. Urine output may be a satisfactory measure of adequate circulating volume when CVP can not be monitored. For major surgeries one may consider use of advanced hemodynamic monitoring techniques of measuring continuous cardiac output like PiCCO or Flotrac which also give reliable information of preload by giving stroke volume variation (SVV) or pulse pressure variation (PPV) during controlled mechanical ventilation.

What are the principles of postoperative management according to the ASA task force guidelines patients with OSA?

Where possible, those patients fit enough for extubation should be extubated wide-awake in the sitting position and transferred to an appropriate postoperative environment. Regional analgesic techniques should be considered; to reduce or eliminate the requirement for systemic opioids in patients with increased perioperative risk from OSA. If neuraxial analgesia is planned, weigh the benefits (improved analgesia, decreased need for systemic opioids) and risks (respiratory depression from cranial spread) of using an opioid or opioid-local anesthetic mixture as compared with a local anesthetic alone. If patient-controlled systemic opioids are used, continuous background infusions should be avoided. Nonsteroidal anti-inflammatory agents (NSAIDs) and other modalities like transcutaneous electrical nerve stimulation (TENS) should be considered; if appropriate to reduce opioid requirements. Avoid concurrent administration of sedative agents like benzodiazepines; as it increases the risk of respiratory depression and airway obstruction. In morbid obesity, acetaminophen (paracetamol) should be used in standard doses, as it's volume of distribution is largely confined to the central compartment. However, as its clearance is increased in obesity, consider increasing frequency of dosing when analgesia is unsatisfactory.

Supplemental oxygen should be administered continuously to all patients who are at increased perioperative risk from OSA; until they are able to maintain their baseline oxygen saturation while breathing room air. One has to keep in mind that supplemental oxygen may increase the duration of apneic episodes and may hinder detection of atelectasis, transient apnea, and hypoventilation by pulse oximetry. CPAP or NIPPV, with or without supplemental oxygen, should be continuously administered when feasible (e.g. when patients are not ambulating) to patients who are already using these modalities preoperatively, unless contraindicated by the surgical procedure. Encourage patients to bring their own equipment to the hospital as it will improve compliance.

If possible, patients at increased perioperative risk from OSA should be placed in nonsupine position throughout the recovery process. Hospitalized patients, who are at increased risk of respiratory compromise from OSA, should have continuous pulse oximetry monitoring even in ward. Continuous monitoring should be provided during entire period of hospitalization. Intermittent pulse oximetry or continuous bedside oximetry without continuous observation does not provide the same level of safety. If frequent or severe airway obstruction or hypoxemia occurs during postoperative monitoring, initiation of nasal CPAP or NIPPV should be considered. Early mobilization is encouraged where possible, as it reduces postoperative atelectasis and the risk of venous thromboembolism.

Describe role of perioperative NIV?

Patients with OSA generally are on CPAP or BiPAP. CPAP treats apneas and hypopneas by providing air under positive pressure through a nasal or facial mask, thus creating a pneumatic splint in the pharynx, which prevents collapse of the pharyngeal airway. A second theory postulates that noninvasive ventilation improves respiratory system compliance by reversing microatelectasis of the lung, thereby diminishing daytime work of breathing. BiPAP gives two levels of pressure; lower and higher alternatingly; and is sometimes better tolerated by the patients who do not tolerate CPAP. It is important to know what mode patient is on and what pressures being were being used so that same settings can be used perioperatively. Also since mask fit may be problem, ask the patient to get his own mask and head gear. Postoperative NIV allows patients to receive slightly

higher doses of analgesics; and gives a safety margin; as very frequently these patients desaturate to 70–80%, even with supplemental oxygen. This is especially true for patients with OSA and OHA, where it can lead to longer recovery stay and also hospital stay.

What are Intermittent Pneumatic Calf Compression Devices (IPCD)? What are the indications and contraindications for their use?

An IPCD consists of a pair of double-walled, vinyl pneumatic sleeves, placed around the calves and connected to a compressor that inflates and deflates the garments. Compressions last 12 seconds per minute, with inflation pressure of 40 mm Hg. The leg sleeves (12 to 16 inches long) extend distally from the inferior border of the patella. They are applied preoperatively and are worn during surgery; they are removed once the patient begins walking. This device compresses the lower leg, reproducing the action of the calf muscles, thus promoting venous return. Studies indicate that calf compression stimulates fibrinolysis, and contributes to preventing thrombus formation.

IPCDs are indicated in patients prone for DVT; hypercoagulable states like malignancies, in prolonged surgeries leading to stasis of blood and in patients with contraindications for anticoagulation, requiring short term DVT prophylaxis.

Contraindications to IPCD use include acute thrombophlebitis, suspected DVT, congestive heart failure, pulmonary edema, and leg ischemia due to severe peripheral vascular disease.

How does bariatric surgery help morbidly obese individuals?

The precursors for obesity are multifactorial. They include genetic tendency, environmental effects, education, sex, race, and socioeconomic status. Obesity is mainly due to changes in behavior pattern, including decreased physical activity and over consumption of high fat food. There are simple solutions to the problem: education, sensible long term diet, increased physical activity and exercise and in some cases medication. Unfortunately though the answers are easy, they are not always practical and hence the only treatment that is effective is bariatric surgery. Following the weight loss there is a high cure rate for diabetes and sleep apnea with significant improvement in other complications of obesity such as hypertension and osteoarthritis.

The two types of weight reduction surgeries available are: a) Gastric restrictive procedures like stapled gastroplasty, Roux-en-Y gastric bypass, and adjustable gastric banding and b) Malabsorptive procedures like biliopancreatic bypass and jejunoileal bypass. Some of these procedures have been discontinued either because of ineffective weight loss, as in horizontal gastroplasty, or because of complications, as in jejunoileal bypass. Other techniques, such as gastric bypass and laparoscopic adjustable gastric banding (LAGB) have been widely adopted as the procedures of choice in weight reduction. Cardiopulmonary and wound complications remain a major morbidity problem in morbidly obese patients following open surgery. Obese patients can benefit greatly from minimally invasive techniques that may help to reduce the incidence of these complications. Laparoscopic adjustable gastric banding, which was introduced in early 1990s, is now the most common procedure and offers the advantages of minimally invasive surgery, adjustability, and reversibility.

What is the role of regional anesthesia in morbidly obese patient?

Although the superiority of epidural anesthesia in obese patients is not yet proven, a recent study has shown less postoperative reduction in vital capacity and other spirometry values and quick recovery of lung volumes in patients receiving epidural compared with those treated with opioids. Where ever it is possible and feasible, regional anesthesia should be administered for obese patients. The advantages are reduction in the use of opioids and inhalational agents, reduced postoperative complications, and less airway related complications.

Main problem with the regional blocks is the technical challenge. Challenges include the lack of palpable bony landmarks, the depth of the space, and 'false' loss-of-resistance in fatty tissues. Special long epidural and spinal needles may be needed. Use of ultrasound for identification of the epidural space and peripheral nerves improves safety and success of regional anesthesia. Insulated needles and a nerve stimulator can be used to identify the appropriate nerves for peripheral nerve blocks. Neuraxial spread of local anesthetics is directly related to BMI. Increased abdominal pressure shifts blood from the inferior vena cava into the epidural venous system, decreasing the volume of the epidural and subarachnoid spaces. Epidural fat further reduces the capacity of the epidural space resulting in higher blocks. For epidural and spinal blocks, local anesthetic dose requirements are reduced by 20–25%. Blocks extending above T5 can cause cardiorespiratory collapse and all resuscitation equipment should be kept ready.

Enumerate the factors affecting drug doses (pharmacokinetics) in obese patients.

Classical pharmacokinetic parameters such as volume of distribution (Vd), clearance (Cl) and protein binding can change for some drugs in morbidly 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 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 α1-acid glycoprotein.

Drug clearance is affected by:
- Increased renal blood flow
- Increased GFR
- Increased tubular secretion
- Decreased hepatic blood flow in congestive cardiac failure.

Highly lipophilic drugs such as barbiturates and benzodiazepines show significant increases in Vd for obese individuals. Less lipophilic compounds have little or no change in Vd with obesity. Exception to this rule is remifentanil, which is a highly lipophilic but shows no significant change in distribution in obese individuals. Consequently, the absolute Vd remains relatively unchanged and the dosage should be calculated on the basis of IBW. Drugs with weak or moderate lipophilicity can be dosed on the basis of IBW or more accurately on lean body mass (LBM). These values are not identical because 20–40% of an obese patient's increase in TBW can be attributed to an increase in LBM. Adding 20% to the estimated IBW dose is sufficient to include the extra lean mass. Nondepolarizing neuromuscular blocking agents can be dosed in this manner.

Succinylcholine is an exception; dosage should be calculated using TBW. The majority of anesthetic drugs are strongly lipophilic. Increased Vd is expected for lipophilic substances, but this is not consistently demonstrated in pharmacological studies, because of factors such as end-organ clearance or protein binding. It has been observed that the Vd of water-soluble agents is less affected by obesity than lipophilic compounds (Table 28.3).

Table 28.3 Commonly used anesthetic agents and their doses

Drug	Recommended dosing
Propofol	Induction: IBW Maintenance: TBW or IBW + (0.4 × excess weight)
Fentanyl	TBW
Thiopental	7.5 mg /kg IBW TBW
Midazolam	TBW for initial dose IBW for continuous dose
Vecuronium	IBW
Atracurium	TBW Initial dose 0.15 mg/kg–2.3 mg/10 kg > 70 kg. Supplemental dose 0.15 mg/kg–0.7 mg/10 kg > 70 kg
Cisatracurium	TBW
Rocuronium	IBW
Succinylcholine	TBW > 140 kg. Maximum 120–140 mg
Mivacurium	TBW. Divided dosing 0.15 mg/kg + after 30 s 0.15 mg/kg
Neostigmine	TBW
Alfentanil	IBW or corrected weight Corrected weight = IBW + (0.4 × excess weight)
Sufentanil	TBW , corrected weight BMI > 40
Remifentanil	IBW
Morphine	IBW
Paracetamol	IBW

Enumerate treatment modalities for OSA

Treatment modalities available are:
- Mandibular advancement devices
- Surgery
  - Jaw advancement
  - Uvulopalatopharyngoplasty
  - Bariatric surgery
  - Tonsillectomy in children
  - Tracheostomy is curative but rarely used because of the associated morbidity.

Enumerate problems and their management in obese obstetric patients:

The problems are:
- Increased risk of chronic hypertension, pregnancy induced hypertension (preeclampsia) and diabetes (2 to 8 fold increase in incidence).
- A higher incidence of induced labor, assisted deliveries and cesarian section.
- Weight gain and maternal diabetes may increase the incidence of fetal macrosomia (a newborn with an excessive birth weight), with associated risks and difficulty in delivery.
- Possibility of greater blood loss during cesarean section, the surgery tends to be longer, and the incidence of post-operative complications tends to be higher.

- Increased risk of anesthesia related maternal morbidity and mortality during cesarean section, when compared with nonobese patients.
- Increased risk of fetal morbidity and mortality.
- Cephalad retraction of panniculus (layer of fat tissue, consisting of subcutaneous fat in the lower abdominal area) in morbidly obese during cesarean section, may lead to hypotension and fetal compromise, as well as problems in gas exchange.
- In case of central neuraxial blockade, higher levels can lead to loss of intercostal muscle function, which may create greater breathing problems in the obese parturient.
- Supine and trendelenburg positions given during cesarean section may further decrease FRC, increasing the likelihood of hypoxemia and aspiration.
- Use of PEEP to increase oxygenation may decrease cardiac output, and possibly compromise uterine blood flow.

Problems with regional anesthesia are:

- Greater cephalad spread of local anesthetics during spinal anesthesia.
- The consequences of excessive blockade can create trouble with spinal in an obese patient with a difficult airway.
- Increased surgical time of cesarean section should be a consideration when selecting an anesthetic for spinal anesthesia.
- Higher incidence of failed epidurals in the morbidly obese due to technical problems.

Advantages of selecting an epidural are:

- Slower onset: ability to titrate, less hypotension.
- Potential for less motor blockade.
- Facilitates postoperative analgesia.

Considerations for GA are

- Higher risk of aspiration combined effect of pregnancy, obesity and difficult airway.
- Anticipate a difficult bag and mask ventilation and laryngoscopy secondary to large breasts, poor neck range of motion and a decreased chin to chest distance.
- The mother's well being should be considered first: a rapid sequence induction should not be attempted if the intubation is anticipated to be difficult. Opt for awake FOB guided intubation.
- Consider use of a short handled laryngoscope.
- Anticipate a more rapid oxygen desaturation than that in nonobese and nonpregnant obese patients.
- Preoxygenate well with 100% oxygen for 3 minutes of tidal ventilation.
- Anticipate difficulty in securing emergency airway via cricothyrotomy or tracheostomy, secondary to poorly defined landmarks and difficulty in proper positioning.
- Postoperative hypoxemia is more severe in obese patients, and the incidence is increased with a vertical incision.
- Patient should be recovered in semi-recumbent position and provide supplemental oxygen administration.

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29 Cirrhosis with Portal Hypertension

A Kulkarni, J Divatia

A 40 years old male patient presents with a history of lump in the right hypochondrium. He received blood transfusion for a road trauma several years ago. He had a bout of jaundice 7 years ago and was found to be HBsAg positive. A CT scan now reveals cirrhosis of liver with a large nodule in the right lobe of the liver. He is posted for right hepatectomy.

What are the functions of the liver?

Metabolic Functions

Carbohydrate metabolism: Liver plays a vital role in both short term and long term maintenance of glucose concentration within a narrow, normal range by *glycogenesis* (generation and storage of glycogen), *glycogenolysis* (glycogen breakdown) and *gluconeogenesis* (glucose synthesis).

Fat metabolism: Triglyceride oxidization for energy production, fatty acid broken down to acetoacetate for metabolism in other tissues. Liver also undertakes synthesis of lipoproteins, cholesterol and phospholipids and conversion of carbohydrates and proteins into fatty acids and triglycerides.

Protein metabolism: This consists of deamination and transamination of amino acids, conversion of amino acids to glucose or lipids, synthesis of urea (removal of ammonia), nonessential amino acids, plasma proteins, clotting factors.

Secretory and excretory functions: Liver secretes (400 to 800 mL/day) bile which contains bilirubin, water, cholesterol, electrolytes, bile acids, and phospholipids. Waste products are also secreted in bile. The bile acids (cholic and chenodeoxycholic) emulsify the fat particles in the gut forming chylomicrons, which are important for absorption of fat soluble vitamins such as vitamin K.

Vascular functions (for reservoir function see below)

Phagocytic system: Sinusoids are lined by endothelial cells, interspersed with tissue macrophages (called Kupffer cells). These remove particulate materials and microbes by phagocytosis and prevent their entry in systemic circulation.

Formation of lymph: Through fenestrations in sinusoidal endothelial cells, lymph is secreted in the "space of Disse" (space between hepatocytes and endothelium). From here it goes along lymphatic capillaries associated with portal triads then to the systemic lymphatic system. Liver contributes 50% to total lymph production. If there is an increase in sinusoidal pressure (as in portal hypertension) lymph production is increased. Hence we get ascites in portal hypertension.

What is the blood supply to the liver? How is hepatic blood flow regulated?

The liver has dual blood supply; oxygenated blood from the hepatic artery (25%) and deoxygenated blood from the gut via the portal vein (75%). The liver is a very vascular organ and receives 30% of the resting cardiac output. It functions as a major vascular reservoir as it has ability to store or release blood. The liver can compensate for moderate hemorrhage by ejecting adequate blood. In hypervolemic states, liver can act as buffer by storing the blood.

Regulation of hepatic blood flow: Hepatic blood flow is regulated at 3 levels. First level is the systemic circulation.

Changes in cardiac output, arterial resistance and central venous pressure will lead to changes in the hepatic blood flow. Regional macrocirculation is the second regulatory level. Hepatic blood flow depends on portal venous flow and resultant changes made by hepatic artery to buffer these alterations to maintain constant blood flow (autoregulation and hepatic arterial buffer response). Hepatic microcirculation is the third level of regulation where changes occur in response to nitric oxide and endothelin concentration in the vessel walls.

What are the tests of liver function (LFTs)?

LFTs are done for detection, evaluation and monitoring of liver damage. The commonly done tests include:

Serum enzymes: Aminotransferases—alanine (ALT) and aspartate (AST): raised levels reflect hepatocellular injury.

Cholestasis: Alkaline phosphatase (ALP), 5'-nucleotidase, and -glutamyl transpeptidase (GGT)

Detoxification and excretory functions: Serum bilirubin (total and direct), serum ammonia, urine bilirubin

Biosynthetic function of the Liver: Serum albumin, serum globulin and coagulation tests (prothrombin time).

Is the albumin level or the prothrombin time a better test of acute liver dysfunction and why?

Prothrombin time, which collectively measures factors II, V, VII, and X. Biosynthesis of factors II, VII, IX, and X depends on vitamin K. Clotting factors have rapid turnover, with a half life of 12 hours, as compared to albumin (whose half life is 18–20 days with a slow 4% degradation/day). Therefore measurement of the prothrombin time is the single best acute measure of hepatic synthetic function and helpful in both the diagnosis and assessing the prognosis of acute parenchymal liver disease.

Which coagulation factors are synthesized in liver? Where are the other factors synthesized? Which factors are vitamin K dependent factors?

All coagulation factors are made in the liver, except for vonWillebrand factor (vWF) and factor VIII. vWF is produced in endothelium (in the Weibel-Palade bodies), megakaryocytes (α-granules of platelets), and subendothelial connective tissue. The vitamin K dependent factors are: Procoagulants: Factor II, VII, IX, X. Anticoagulants: Proteins C, S and Z.

How does vitamin K enter the liver?

After ingestion, vitamin K gets emulsified by action of bile salts mixed micelles are formed. These are taken up and formed into chylomicrons, which are secreted into the lacteals; within the intestinal villi; which drain into lymphatic vessels. These empty into the circulation via the thoracic duct. In muscles and adipose tissues chylomicrons get converted to chylomicron remnants, (containing vitamin K in a lipid core) which are then taken up by hepatocytes. Vitamin K is also formed in the body by the action of gut bacteria (mainly bacteroids) and the main site of absorption in solution in the bile, is the terminal ileum, Vitamin k then reaches liver by enterohepatic circulation. In the literature however, there is doubt about the bioavailability of this vitamin K (produced by bacterial action) as it is tightly bound to the bacterial membrane. The largest amount is present in the colon (not terminal ileum), which does not have bile salts to make vitamin K soluble for absorption.

Which conditions result in prolongation of the prothrombin time? Which conditions respond to vitamin K therapy?

Prothrombin time is prolonged in vitamin K deficiency, von Willebrand disease and disseminated intravascular coagulation. Conditions which respond to vitamin K therapy are:

1. Hemorrhagic disease of the newborn.
2. Fat malabsorption states such as cystic fibrosis, celiac disease, tropical sprue, Crohn's disease, ulcerative colitis, Ascaris infection, bacterial overgrowth, chronic pancreatitis, short bowel syndrome.
3. Cholestasis due to common duct obstruction due to stones and strictures, primary biliary cirrhosis, cholangiocarcinoma, and chronic cholestasis (obstructive jaundice).
4. Drug interaction: cholestyramine (binds bile salts) causes fat malabsorption.
5. Vitamin K antagonists. Coumadin (Warfarin) inhibits vitamin K epoxide reductase and vitamin K reductase, and creates an intracellular deficiency. Cefamandole, cefoperazone, salicylates, hydantoins, rifampin, isoniazid, and barbiturates are associated with vitamin K deficiency, but the mechanism is unknown.
6. Broad spectrum antibiotics that destroy vitamin K producing bacteria.
7. Diseases where coagulation inhibitors are produced: Lupus anticoagulant, antithrombins, and paraproteinemias, such as multiple myeloma.

What is jaundice? How is bilirubin formed and how is it metabolized?

Jaundice is yellow discoloration of the skin, sclera (icterus), and other tissues caused by deposition of excess circulating bilirubin.

Bilirubin formation and metabolism: Bilirubin is formed in the reticuloendothelial system, i.e. liver and spleen, when heme (70 to 80% from the breakdown of RBCs, and 20 to 30% derives from other heme proteins—prematurely destroyed erythroid cells in bone marrow and myoglobin and cytochromes in other tissues) is broken down. Unconjugated (indirect) bilirubin is water insoluble and is transported bound plasma albumin. Bilirubin is taken up by the hepatocyte (without the albumin) by carrier-mediated membrane transport. In presence of glucuronyl transferase, bilirubin is conjugated first to mono and then diglucoronide bilirubin (conjugated or water soluble bilirubin). Conjugated bilirubin is secreted into the bile canaliculus. In the intestine, bacteria metabolize bilirubin to form urobilinogen. Most of this urobilinogen is converted to stercobilins, which is excreted in the stools. Some urobilinogen is excreted unchanged in urine. Some urobilinogen is reabsorbed, extracted and excreted again by hepatocytes; this is called as enterohepatic circulation.

What are differences between hemolytic, hepatocellular and obstructive jaundice?

Hemolytic jaundice: Hemolytic jaundice results from excessive breakdown of the RBCs. In presence of normal liver function, serum bilirubin rises are modest and do not exceed 4 mg/dL. Other liver function tests are normal with purely unconjugated hyperbilirubinemia.

Obstructive or cholestatic jaundice occurs when there is physical or physiological obstruction to bile flow. The obstruction may be intrahepatic or extrahepatic. Intrahepatic causes of obstructive jaundice are hepatitis, drug intoxication, primary biliary cirrhosis, cholestasis of pregnancy, and metastatic cancer and alcoholic liver disease. Extrahepatic causes are biliary calculus, stricture of common biliary duct, carcinoma of the common bile duct carcinoma at the ampulla of vater, pancreatitis or pancreatic pseudocyst, sclerosing cholangitis and pancreatic cancer. This manifests as mixed hyperbilirubinemia. Some conjugated bilirubin reaches circulation and is excreted in urine, producing dark urine. Since most bilirubin does not reach the intestine pale or white or clay coloured stools are produced. Lack of bile salts leads to fat malabsorption and steatorhea. SGOT and SGPT are normal, serum alkaline phosphatase is raised.

Hepatocellular Jaundice is caused by hepatocyte damage, it is seen when > 80% liver is damaged. It is seen with cirrhosis or viral hepatitis. There is increase in both unconjugated and conjugated bilirubin. The liver enzymes (SGOT and SGPT) are raised. Table 29.1 summarizes the difference between the three types of jaundice.

What are types of viral hepatitis? Which is more likely to cause chronic liver disease and cirrhosis?

The following Table 29.2 details the types of viral hepatitis (Hepatitis A thorough E) and their differences.

Hepatitis B and C both have chronic infection and carrier phases and cause cirrhosis. Hepatitis B, (and HDV) when acquired during in neonatal period can lead to cancer. Hepatitis C almost always leads to chronic hepatitis. Incidence of cirrhosis and hepatocellular carcinoma is very high with hepatitis C carriers.

Hepatitis G" virus and "TT" virus, transfusion transmitted viruses have been identified but they do not cause hepatitis.

Describe standard precautions in case of needle stick injury in a patient with Hepatitis B or C?

Precautions developed for preventing exposure and transmission of disease in occupational setting, were previously called "Universal precautions" implying precautions taken "everyone, everywhere, always". This has now changed

Table 29.1 Differences between hemolytic, obstructive and hepatocellular jaundice

Features	Hemolytic Jaundice	Obstructive jaundice	Hepatocellular jaundice
Serum bilirubin	Modest rise, mostly unconjugated	High, mostly conjugated	High, both conjugated and unconjugated
Urine urobilinogen	High	Absent	Present, (absent in predominantly cholestatic phase)
Urine bilirubin	Absent	Absent	High
Urine bile salts and pigments	Absent	Absent	Present
Stool stercobilins	High	Absent	Present
Liver enzymes	Normal	Alkaline phosphatase, gGT raised	Elevated transaminases
Coagulopathy	Absent	May be Present	Present/Absent
Response to Vitamin K	NA	Yes	Yes/No

Table 29.2 Types and differences in hepatitis viruses

Hepatitis	A virus	B virus	C virus (non A- non B)	D virus (delta body)	E virus
Spread	Fecooral Contaminated food and water Oral/Anal sexual contact	Blood and body fluid contact Sex Needles Mother to baby Human bite	Blood and body fluid contact Needles Mother to baby Sex (minimal)	Coinfection or superinfection with HBV	Fecooral Contaminated food and water
Incubation	30 (15–50) days	60–90 (45–180) days	6–7 weeks (2–6 months)	60–90 (30–180) days	40 (14–60)
Symptoms of initial infection	Asymptomatic (especially HCV) jaundice, fever, myalgia nausea, vomiting, anorexia, fatigue, Dark urine and pale bowel movements				
Chronic Infection (Infection for life)	No chronic disease	1–10% chronic can cause: Liver cell damage Cirrhosis Liver cancer	85% chronic can cause: Liver cell damage Cirrhosis Liver cancer	Common	No chronic disease
Prevention	Hep A IgG up to 2 weeks after exposure	Hepatitis B IgG for PEP Recombinant vaccine	-	-	-
Treatment	Self-limiting	Interferon Lamivudine Adefovir Pegylated interferon Entecavir Telbivudine	Pegylated interferon plus ribavirin	Interferon?	None
Vaccine	Yes	Yes	No	HBV vaccine	No

to standard precautions meaning "standard of care". The components are:

1. *Use of protective barriers.* Gloves for handling of mucus membranes, body fluids, broken skin, wear goggles, gowns and masks during procedures.
2. *Sharps* (needles, scalpels, suture material, bandages, dressings), anything contaminated with any body fluid and waste: to be handled with gloves and disposed in designated puncture-resistant containers. While handling and disposing sharps do not recap needles, put containers within arms reach, use adequate light source when treating patients and wear heavy-duty gloves when transporting sharps. Incinerate used needles to a sufficient temperature to melt.
3. *Re-usable instruments* such as speculums, surgical instruments and thermometers must be thoroughly disinfected
4. *Immunizations* when available take vaccines. (Hepatitis A and B)
5. *Wound care after exposure:* Wash wounds gently (without vigorous scrubbing) with soap and water, let the wounds bleed freely. Irrigate mucosal surfaces with saline.

Compare infectivity of HIV, Hepatitis B and Hepatitis C. Discuss pre-and postexposure prophylaxis for these conditions.

Risk of transmission of infection depends on:

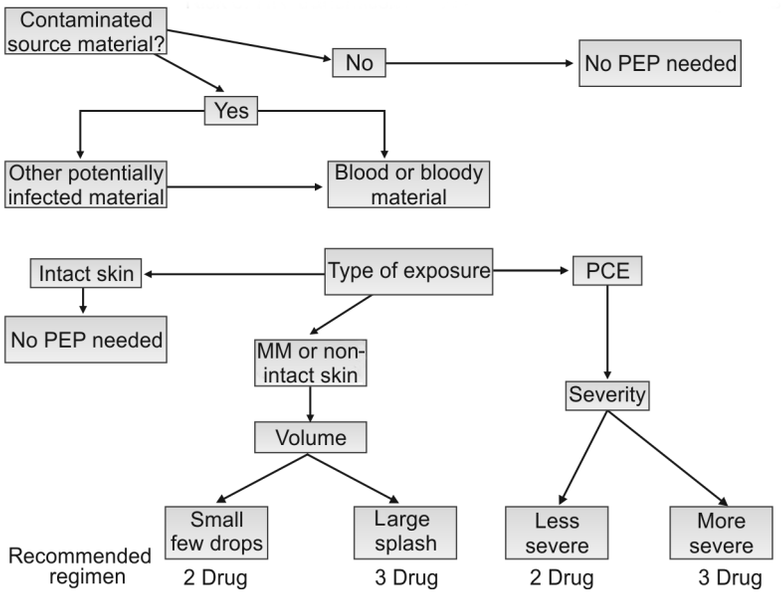
- No of exposures to infected blood /fluids.
- Prevalence of infected patients in the healthcare worker's area of practice.
- Infectivity of the pathogen.

For the HIV, risk for transmission is not very high, the reported rates of seroconversion are as follows:

Percutaneous exposure to blood 0.3%, mucocutaneous exposure 0.03%, and exposure of intact skin 0%. The seroconversion rates for hepatitis B are 40%, while those for hepatitis C are 2%. However with hepatitis C, 85% develop chronic hepatitis, 20% of these patients progress to cirrhosis, with 3% getting hepatocellular carcinoma. (For vaccines, etc. Table 29.2).

For HIV the commonly used drugs are: Zidovudine 250–300 mg BD, Lamivudine 150 mg BD and Indinavir 800 mg TDS or Efavirenz 600 mg OD. Depending on the type of exposure (Flow chart 29.1, 2 or 3 drugs). Postexposure prophylaxis (PEP) has been shown to be effective in 81%

Flow chart 29.1 Risk of HIV transmission and currently recommended regimens



patients. The flow chart below summarizes evaluation of risk of HIV transmission and current recommendations for PEP.

Hepatitis B PEP: For adults 3 (10 microgram) injections of hepatitis B recombinant vaccine are given at 0, 1–2 months and 5–6 months. A booster may be required after 5 years. Hepatitis B immunoglobulin 500 IU atleast needs to be given within 24–72 hours after exposure.

What is cirrhosis of the liver?

Cirrhosis is a pathological condition where normal architecture of liver is distorted due to development of fibrous tissue and formation of regenerative nodules. This is accompanied by decreased hepatocellular mass as well as function and alteration of blood flow.

What are the causes of cirrhosis?

- Alcoholism
- Chronic viral hepatitis (Hepatitis B, Hepatitis C)
- Inherited metabolic liver diseases
  - Hemochromatosis
  - Wilson's disease
- Autoimmune hepatitis
- Nonalcoholic steatohepatitis
- Biliary cirrhosis
  - Primary biliary cirrhosis
  - Primary sclerosing cholangitis
  - Autoimmune cholangiopathy
- Cardiac cirrhosis
- Miscellaneous
  - Cystic fibrosis
  - Cryptogenic cirrhosis
  - α-1 antitrypsin deficiency.

What are the adverse effects of alcohol?

Alcohol affects all organ systems in the body and produces a variety of adverse effects. The adverse effects also depend on type of ingestion. A summary of adverse effects is given in the Table 29.3.

What are the complications of cirrhosis?

The complications of cirrhosis are:

- Portal hypertension
  - Variceal bleeding, ascites, splenomegaly and hypersplenism
- Liver cell failure
- Hepatic encephalopathy
- Hepatorenal syndrome.
- Spontaneous bacterial peritonitis.

How is the portal vein formed? What is portal hypertension? What are the causes of portal hypertension?

Portal vein is formed by the superior mesenteric and splenic veins. Portal hypertension is defined as an increase in the hepatic venous pressure gradient to > 5 mm Hg. It is caused by increased resistance to hepatic blood flow due to cirrhosis and regenerative nodules, and increased splanchnic blood flow due to splanchnic vasodilatation.

Causes of portal hypertension are:

Prehepatic causes: splenic AV fistula, splenic or portal vein thrombosis, massive splenomegaly.

Table 29.3 Adverse effects of alcohol

System	Adverse effects
Central nervous system	Headache, cerebral atrophy, ataxia, seizures. Skull fractures subdural hematoma
Psychiatry	Anxiety, panic, sedation, euphoria, irritability, restlessness, aggressiveness, violence, depression, sleep disturbances, memory and cognitive deficits, confabulation, hallucinations, and delusions
Eye	Blurred vision, loss of vision, or color vision abnormalities
Cardiovascular system	Cardiomyopathy, congestive heart failure, arrhythmias, coronary artery disease hypertension, edema, increased risk of hemorrhagic /ischemic stroke
Respiratory system	Pneumonia
GI system	Gastritis, ulcers, bleeding, malabsorption, diarrhea or constipation, and esophageal varices
Hepatopancreatic system	Fatty liver, hepatitis, jaundice, fibrosis, cirrhosis, coagulopathies, hypoprotenemia, and ascites. Pancreatitis
Genitourinary system	electrolyte imbalances, urinary tract infections, and sexual dysfunction
Peripheral nervous system	Paresthesias, peripheral neuropathies, extrapyramidal symptoms and pain
Musculoskeletal system	Myalgias, cramps, atrophy, weakness, joint inflammation, worsening of rheumatoid arthritis, gout, bone ischemia, necrosis, and bone marrow depression
Hematological effects	Iron deficiency anemia, macrocytic anemia, leukopenia, thrombocytopenia.
Skin	Dermatitis, flushing, angiomas, urticaria, bruising, and sweating
Endocrine	Altered glucose tolerance, unstable diabetes, menstrual cycle irregularities, and gynecomastia.

Presinusoidal intrahepatic causes: Sarcoidosis, schistosomiasis, nodular regenerative hyperplasia, congenital hepatic fibrosis, idiopathic portal fibrosis, early primary biliary cirrhosis, chronic active hepatitis, myeloproliferative disorders, graft vs host disease.

Sinusoidal intrahepatic causes: Established cirrhosis, alcoholic hepatitis.

Postsinusoidal intrahepatic causes: Alcoholic terminal hyaline sclerosis, veno-occlusive disease.

Postsinusoidal posthepatic causes: Budd-Chiari syndrome, membranous IVC web, right heart failure, constrictive pericarditis.

What are the complications of portal hypertension?

Variceal bleeding, splenomegaly, hypersplenism and ascites are the main complications of portal hypertension.

Which are sites of portosystemic anastomoses?

The sites of portosystemic anastomosis are as follows (Table 29.4):

How is acute variceal bleeding managed?

Management of acute variceal bleeding consists of:

1. General resuscitation (fluid resuscitation and replacement blood and blood products, hemodynamic and respiratory support).
2. Specific measures to control the bleeding.
3. Measures to prevent hepatic encephalopathy.

The specific measures to control bleeding are:

1. Pharmacological measures which cause vasoconstriction to stop bleeding. The drugs used are vasopressin (20 units in 200 mL over 20 min (check dose) with ECG monitoring, terlipressin, glypressin, somatostatin and octreotide (50–100 mcg/h as an infusion).
2. Mechanical measures inflatable balloons for tamponade by direct pressure. These are Sengstaken-Blakemore tube (esophageal and gastric balloon), Minnesota tube

Table 29.4 Portosystemic anastomoses

Site of PS anastomosis	Portal component	Systemic component	Clinical effect
Lower esophagus	Esophageal branches of left gastric vein	Azygous veins	Submucosal gastroesophageal varices
Upper anal canal	superior rectal vein	Middle inferior rectal veins	May be confused with hemorrhoids
Umbilical (anterior abdominal wall)	Veins of ligamentum teres	Superior /inferior epigastric veins	Caput medusae
Bare area of liver	Hepatic /portal veins	Inferior phrenic veins	
Patent ductus arteriosus (rare)	left branch of portal vein	Inferior vena cava	
Retroperitoneal	Colonic veins	Body wall veins	usually none

(modification of Sengstaken-Blakemore tube) and Linton-Nachlas tube with a single large gastric balloon.
3. Surgical measures: Endoscopic variceal ligation.

Describe a Sengstaken-Blakemore tube and the procedure. How is it inserted? Which balloon is inflated first and how? How is the SB tube fixed? Which balloon is inflated next and how? Which cuff is deflated and when? What if bleeding persists?

Sengstaken-Blakemore tube has 3 components: a gastric balloon, an esophageal balloon, and a gastric suction port. The balloons are for tamponade while the gastric port is for aspiration of blood from the stomach.

Procedure for insertion: Most patients will need to be intubated as it is a large tube and very uncomfortable for the patient, however local anesthesia may be used. Patient is put in 45% head elevation. Alternatively, a left lateral decubitus position can be used. The balloons are checked by inflating and submerging under water for leaks, then ports are clamped after removing all air. Sometimes a nasogastric tube is tied about 4 cm above the esophageal balloon and used for aspiration of blood from esophagus. The tube (and balloons) is lubricated and then it is passed (either nasally or orally) in the stomach up to the 50 cm mark. First the gastric balloon is inflated with 100 mL increments and the pressure is measured. If pressure is above 15 mm Hg, the balloon may be in the esophagus, and it needs to be deflated and the tube is advanced further, till it is correctly positioned. Then the gastric balloon is inflated with 450–500 mL of air, and the ports are clamped. Proper placement can be confirmed by irrigating the gastric aspiration port with water while auscultating over the stomach. The tube is pulled back gently until resistance is felt. The tube is put on traction device (0.45–0.91 kg, generally a 0.5 L saline bottle) using a pulley.

Gastric port is aspirated to check for bleeding. If bleeding continues, the esophageal balloon is inflated to lowest pressure needed to stop bleeding (30–45 mm Hg) and then the port is clamped. A periodic watch is kept on the balloon pressure. If bleeding still continues, the traction is increased further to a maximum of 1.1 kg. Once bleeding is controlled, oesophageal balloon pressure is decreased by 5 mm Hg every 3 hours to 25 mm Hg. This pressure is maintained for 12–24 hours. The esophageal balloon should be deflated for 5 minutes every 6 hours to help prevent esophageal necrosis. If bleeding recurs, the gastric balloon and, if necessary, the esophageal balloon may be reinflated for an additional 24 hours.

If bleeding persists beyond this time, alternative therapies need to be considered, these include endoscopic sclerotherapy and banding of the varices, transjugular intrahepatic portacaval shunt (TIPS) or surgery.

Outline the management of portal hypertension in cirrhosis.

The primary treatment of portal hypertension is treatment of the cause, or removal of offending agents, if possible. Other measures include measures to lower portal venous pressure and treatment of complications.

- Non-selective β-blockers (such as propranolol) to reduce portal venous pressure.
- Low salt diet.
- Diuretics for ascites: Spironolactone to counteract sodium retention; loop diuretics can also be added.
- Vasoactive drugs—reduce hepatic venous pressure gradient (in variceal bleeding): Somatostatin or its analogues octreotide and vapreotide, vasopressin or terlipressin-splanchnic vasoconstrictors but have ischemic side-effects.
- Portosystemic shunt procedures:
  - TIPS (transjugular intrahepatic portosystemic shunt) An intrahepatic shunt is created between portal and hepatic veins. This may lead to higher risk of hepatic encephalopathy. Frequently stenosis occurs necessitating repeat procedure.
  - Surgically created portosystemic shunts: These are major procedures, not done routinely now, may be undertaken when TIPS is not possible.

What are the types of surgical shunts performed for portal hypertension?

Three types of surgical shunts for portal hypertension have been described: total, partial or selective shunts. With total shunts portal venous flow is completely diverted away from liver, achieving total decompression. The examples are end to side and side to side portocaval shunt and central splenorenal shunt. Partial shunts achieve partial decompression of the portal venous system and some portal flow is maintained, example is portocaval H graft. The selective shunts also partly decompress portal circulation the examples are distal splenorenal shunt and splenocaval shunt. The incidence of HE is high (~ 40%) with total shunts and drops (~10%) with partial and selective shunts. Surgical shunting is now rarely performed with the advent of TIPS procedure.

What is hepatic encephalopathy (HE)? Discuss pathophysiology, signs of early encephalopathy and grades of hepatic encephalopathy.

Hepatic encephalopathy (HE) or portosystemic encephalopathy is an alteration in mental status and cognitive function occurring in the presence of liver failure. HE occurs because the neurotoxins produced in the gut reach the brain due to portosystemic shunting as well as failure of liver to detoxify

them due to reduced hepatic mass and function. The commonly sited reasons for HE include raised levels of ammonia, GABA (due to altered GABA homeostatic mechanisms due to liver injury), benzodiazepine receptor ligands (which may bind to GABA receptors and produce inhibitory CNS effects). In addition, ammonia may combine with alfa ketoglutarate in CNS forming glutamate which increases GABA synthesis.

Signs of HE: The first signs of hepatic encephalopathy can be subtle and nonspecific—change in sleep patterns, change in personality, irritability, and mental dullness. Later confusion, disorientation, stupor, and eventually coma supervene. HE is exceptional in that, it has a fluctuating symptomatology which ranges form from mild neurological impairment that may progress to deep coma fast and resolve again in hours.

CNS examination: Trail-making test, where patient is asked to connect a series of 25 numbered circles as rapidly as possible using a pencil. A normal person can finish the test in 15–30 s; it is considerably delayed in patients with early hepatic encephalopathy. Micrographia may be an early sign. Patient may be asked to draw abstract objects or a fresh signature may be compared to previous signatures. More sophisticated testing includes electroencephalography and visual evoked potentials, which can detect mild forms of encephalopathy, but these are rarely clinically useful.

The following table (Table 29.5) shows grades of HE.

Some articles divide Stage 4 further as follows:

Grade 4a Coma, arousable by painful stimuli.

Grade 4b Coma without response to painful stimuli.

How can HE be prevented? What is the mechanism of action of lactulose?

Mechanism of action of lactulose is not completely understood. One hypothesis is that on ingestion lactulose reaches cecum unchanged, and is metabolized by enteric bacteria to lactate and acetate. This leads to a drop in the colonic pH. This acidification facilitates the passage of ammonia into the colon, where bacteria utilize ammonia as a metabolic substrate. This increases fecal nitrogen excretion and also reduces the amount of nitrogen reaching the portal blood with consequent reduction in serum ammonia level. Second hypothesis is that lactic acid production promotes growth of lactobacillus and other saccharolytic bacteria, which in turn suppress proteolytic and urealytic bacteria. Third proposal is that lactulose serves as an energy source, which alters the metabolism of facultative urealytic and proteolytic bacteria, reducing ammonia production.

What are the factors that precipitate HE?

Factors precipitating HE are given below:

- Increased nitrogen load
  - Gastrointestinal bleeding
  - Excess dietary protein
  - Azotemia
  - Constipation
- Disturbances of internal milieu
  - Hypovolemia
  - Metabolic alkalosis/acidosis
  - Hypoxia
  - Hyponatremia, hypokalemia
- Drugs
  - Narcotics, tranquilizers, sedatives
- Miscellaneous
  - Infection
  - Surgery
  - Superimposed acute liver disease
  - Progressive liver disease
  - Transjugular intrahepatic portal-systemic shunt (TIPS).

What is ascites? What are the causes of ascites in cirrhosis?

Ascites is pathologic accumulation of fluid in the peritoneal cavity. Etiology of ascites is mutlifactorial.

Table 29.5 Grades of hepatic encephalopathy

Grade	Level of consciousness	Intellectual function	Neurological findings	EEG
1	Lack of awareness Personality change Day/night reversal	Short attention	Incoordination Mild asterixis	Slowing (5–6 cps) Triphasic
2	Lethargic Inappropriate behavior	Disoriented	Asterixis Abnormal reflexes	Slowing Triphasic
3	Asleep Rousable	Loss of meaningful communication	Asterixis Abnormal reflex	Slowing triphasic
4	Unrousable*	Absent	Decerebrate	Very slow (2–3 cps), delta

Factors that cause generalized accumulation of fluid:

1. Decreased synthetic function of liver leads to hypoalbuminemia, which leads to reduced oncotic pressure and leaking of fluid into the peritoneal cavity.
2. Nitric oxide induced vasodilation activation of rennin-angiotensin-aldosterone system with sodium and water retention.

Factors that lead to localized fluid accumulation in the abdominal cavity:

1. Raised intrahepatic vascular resistance (with portal hypertension) along with splanchnic vasodilation leads to increase portal flow.
2. Increased splanchnic production of lymph due to raised portal venous flow, and due to seepage of lymph from lymphatic channels disrupted by nodular regeneration.

What are the complications of ascites?

Complications of ascites include development of refractory ascites and spontaneous bacterial peritonitis.

Refractory ascites is defined as ascites unresponsive to 400 mg of spironolactone or 30 mg of amiloride plus up to 120 mg of furosemide daily for two weeks.

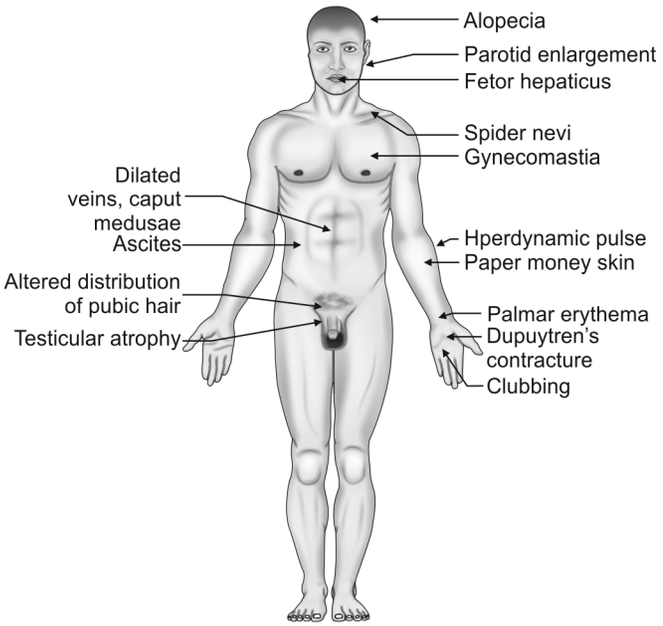
Treatment includes large-volume paracentesis (≤ 5 L) and peritoneovenous (LeVeen) shunt in selected patients with good hepatic reserve. Previous abdominal surgery, spontaneous bacterial peritonitis and large varices are relative contraindications to the shunt procedure.

Spontaneous bacterial peritonitis (SBP): This is a clinical syndrome, where ascites becomes infected in absence of a recognizable cause of peritonitis. It is more common in patients with severely decompensated cirrhosis with jaundice. Diagnosis of SBP is by paracentesis. The "gold standard" for diagnosis of SBP is a polymorphonuclear (PMN) count of >250 cells/μL. Secondary bacterial peritonitis is an important differential diagnosis of SBP and shows the following features: (1) multiple organisms vs single organism in SBP (2) ascitic fluid protein concentration > 1 g/dL; (SBP < 1 g/dL) or (3) Leukocytosis even with antibiotic therapy (SBP rapid fall in white cell count (4) culture remains positive (SBP becomes quickly sterile).

How is ascites managed?

- Salt restricted diet (water restriction only if patient is hyponatremic).
- Spironlactone 100–200 mg/d (can be increased to 400–600 mg/d) and frusemide 40–80 mg/d (can be increased to 120–160 mg/d) might be added in patients with peripheral edema.
- If ascites persists despite above refractory ascites:
  - Repeated large volume abdominal paracentesis with albumin replacement as needed
  - TIPS.

What are the clinical signs of liver cell failure?



The signs of hepatic failure include hair loss (alopecia), and flapping tremors of the body and tongue, gynecomastia, asterixis, parotid gland enlargement, digital clubbing and muscle wasting. Fetor hepaticus is the slightly sweet, ammoniacal odor that may be seen liver failure patients. Ascites, hydrothorax, prominent veins over the abdomen, and caput medusa, (veins radiating from the umbilicus) are also seen. Widened pulse pressure and signs of a hyperdynamic circulation can occur in patients with cirrhosis as a result of fluid and sodium retention, increased cardiac output, and reduced peripheral resistance. Hyperpigmentation is seen with advanced chronic cholestatic diseases. Tendon xanthomata occur as a result of retention and high serum levels of lipids and cholesterol. A slate-gray pigmentation to the skin also occurs with hemochromatosis if iron levels are high for a prolonged period. Mucocutaneous vasculitis with palpable purpura, especially on the lower extremities, is typical of cryoglobulinemia of chronic hepatitis C but can also occur in chronic hepatitis B.

Spider angiomata and palmar erythema occur in both acute and chronic liver disease. Spider angiomata are superficial, tortuous arterioles typically fill from the center outwards. Spider angiomata occur only on the arms, face, and upper torso; they can be pulsatile and may be difficult

to detect in dark-skinned individuals. Testicular atrophy may also be seen.

How is the risk assessment done in a case of chronic liver disease/cirrhosis? What is the Child-Pugh classification? What is MELD and MELD-Na?

There are various methods of staging of liver disease. Child-Turcotte-Pugh classification (more commonly called as Child-Pugh classification) is a universally used method. The score ranges from 3 to 15. The grades are class A (score of 5–6), B (7–9), or C (>10). Class C indicates decompensated cirrhosis. Grade A represents 'good' risk with 5% mortality, Grade B represents 'moderate' risk with 10% mortality, Grade C represents poor risk with mortality > 50%.

Table 29.6 Child-Turcotte-Pugh classification of cirrhosis (score range 3–15)

Factor	1	2	3
Serum bilirubin	< 2.0 mg/dL	2.0–3.0 mg/dL	> 3.0 mg/dL
Serum albumin	> 3.5 g/dL	3.0–3.5 g/dL	< 3.0 g/dL
Prothrombin time Prolongation in secs or INR	0–4 < 1.7	4–6 1.7–2.3	> 6 > 2.3
Ascites	None	Easily controlled	Poorly controlled
Hepatic encephalopathy	None	Minimal	Advanced

Model for end stage liver disease (MELD) is another system to assess severity of liver disease, was developed at Mayo Clinic to assess 3 month outcomes of patients undergoing TIPS. The calculation is done as follows:

MELD = 3.78 [Ln serum bilirubin (mg/dL)] + 11.2 [Ln INR] + 9.57 [Ln serum creatinine (mg/dL)] + 6.43. The maxium score is 40. The MELD–Na score includes serum sodium value to further refine the evaluation of the cirrhotic patient. The formula is MELD-Na = [0.025 × MELD × (140 − Na)] + 140. MELD classification may be better at predicting 90 day mortality than Child-Pugh score even in patients undergoing other surgeries. A very recent study found that in patients undergoing nontransplant surgery Child Turcotte-Pugh (CTP) predicted 30 day mortality, MELD predicted 90 day mortality and MELD-Na predicted 1 year mortality.

Discuss in brief management of hepatic encephalopathy.

Identify and correct the precipitating causes:

- Assess vital signs and volume status.
- Evaluate for gastrointestinal bleeding.
- Eliminate sedatives or tranquilizers.
- Screen for hypoxia, hypoglycemia, anemia, hypokalemia, metabolic alkalosis, and other potential metabolic or endocrine factors; correct as indicated.

Initiate ammonia-lowering therapy:

- Dietary management:
  - Previously protein restriction was advocated to reduce ammonia production
- Use nasogastric lavage, lactulose (30 to 45 mL syrup orally TDS / QID 300 mL retention enema until two to four bowel movements per day and mental status improvement) and/or other cathartics or enemas to remove source of ammonia from colon.
- Initiate treatment with lactulose or lactitol to produce two to four bowel movements per day.
- Consider oral nonabsorbable antibiotics to reduce intestinal bacterial counts.
  - Neomycin 4 to 12 g/day
  - Metronidazole and Vancomycin have also been tried for this purpose.
- Consider treatment with flumazenil or another benzodiazepine receptor antagonist.

Minimize potential complications of cirrhosis and depressed consciousness:

- Provide supportive care with attention to airway, hemodynamic, and metabolic statuses.

What are the causes of anemia in a cirrhotic patient?

Cirrhotic patients may develop anemia due to chronic GI blood loss, nutritional deficiencies, or hypersplenism related to portal hypertension (causing early destruction of RBCs) and as a direct bone marrow suppression by alcohol on the. Zieve's syndrome, a form of hemolytic anemia (with spur cells and acanthocytes) can occur in patients with severe alcoholic hepatitis. Erythropoietin though elevated as compared to normal patients are actually lower than those in patients with iron deficiency anemia, suggesting that the elevation is not enough to improve anemia.

How is cirrhosis classified? What is decompensated cirrhosis?

Cirrhosis can be classified in a variety of ways:

Morphologic classification: Macronodular, micronodular and mixed.

Histologic classification: Portal, postnecrotic, posthepatic, biliary, congestive.

Etiologic classification: Genetic, toxic, infectious, biliary, vascular, cryptogenic.

Functional classification: (Child-Pugh Classification) is the most commonly used classification. Class C indicates decompensated cirrhosis.

What factors contribute to coagulopathy in cirrhosis?

The following factors contribute to coagulopathy in cirrhosis:

- Thrombocytopenia due to sequestration, decreased thrombopoietin and DIC and qualitative platelet dysfunction.
- Impaired coagulation factor synthesis, hypofibrinogenemia.
- Coagulation factor consumption due to accelerated fibrinolysis and DIC.
- Altered activity of coagulation factors due to γ carboxylation, dysfibroginemia and vitamin k deficiency.
- Decreased breakdown of plasminogen, increased fibrinolysis.

What other investigations need to be performed in a patient with cirrhosis of liver?

Electrolytes: Electrolyte disturbances may be commonly seen in patients with peripheral edema and or ascites and renal dysfunction. In absence of these, serum electrolytes are likely to be normal.

Renal function: BUN and urea. Renal dysfunction can occur in these patients due to imaging and treatment such as invasive diagnostic procedures, imaging studies (use of nephrotoxic contrast), nephrotoxic medications, urinary tract interventions with UTI and obstruction, and therapies leading to volume depletion (diuretic therapy). IgA nephropathy is commonly seen with alcoholic liver disease. Hepatorenal syndrome (HRS) is precipitated in these patients due to bacterial infection (SBP), gastrointestinal bleed and aggressive paracentesis, and drugs. Circulatory disturbances are also common in cirrhotics and these affect renal vasculature as well. In HRS, histology of kidney is often normal and renal dysfunction is reversed after liver transplant.

Cardiovascular system: ECG and echocardiography. Cardiac dysfunction is common in cirrhotic patients (called cirrhotic cardiomyopathy) and is distinct from alcoholic cardiomyopathy. The cardiac function appears normal at rest due to reduced peripheral resistance which allows cardiac output to be maintained but function decompensates in face of exercise or pharmacological stress. Proposed mechanisms for cirrhotic cardiomyopathy are sympathetic and parasympathetic autonomic dysfunction (due to either damaged peripheral nerves or changes in endogenous neurotransmitters) impaired β adrenergic signal transduction. Decreased β-adrenergic receptor density and receptor desensitization have also been reported in cirrhotics. These patients have reduced sensitivity to catecholamines. Other problems in these patients include hypertension, cardiac failure and arrhythmias (AF, SVT, VT).

Respiratory system: Cirrhosis can induce mild hypoxemia, atelectasis, and pleural effusion. In advanced cirrhotics, hepatopulmonary syndrome (hepatic dysfunction, hypoxemia, and intrapulmonary vasodilatation) and portopulmonary hypertension may occur.

What changes in pharmacokinetics are expected in cirrhosis?

Volume of distribution: Cirrhotics are often hypervolemic and have a large volume of distribution. This means we have to give a larger initial dose of a drug to achieve an effect, however because of hepatocellular dysfunction and reduced metabolism the effect will be prolonged.

Protein composition and binding: Cirrhotics will often have lower levels of albumin; this means free fraction of drugs which are normally highly protein bound, will be higher. Presence of higher fraction of free drug (active drug), smaller doses will be needed for the clinical effect. Thiopentone is a classic example of this. When using thiopentone in cirrhotics it is titrated to effect.

Biotransformation: Rate of metabolism of drugs will be slower, prolonging the half-life of drugs, this will be most apparent when top ups or infusions of drugs are given. Additionally, drugs with active metabolites will have even more prolonged action.

What changes in anesthetic management do you expect in a patient with acute alcohol intoxication and a chronic alcoholic?

A patient with acute alcohol intoxication: Patients who present with acute intoxication for elective/emergency surgery are also likely to be chronic alcoholics, making it likely that all major organ systems are also likely to be affected (see above). Confusion, aggression, CNS depression presenting as somnolence or even coma may occur. Obtaining consent is a problem as they have to be considered as incapable of providing informed consent till the effect of alcohol wears off. Metabolic derangements include hypoglycemia and even alcoholic ketosis. Sedative drugs are less likely to be needed. They are to be treated as having full stomach, due to delayed gastric emptying, thus rapid sequence induction is needed. Alcohol induced depression means hypotension is likely after induction. One may find metabolic conditions

like hypoglycemia, alcoholic ketoacidosis. Excessive dextrose may precipitate Wernicke's encephalopathy. It is caused by thiamine (vitamin B1) deficiency and is characterized by the classic triad of ataxia, ophthalmoplegia, and acute mental confusion. Treatment includes oral or intramuscular thiamine administration (100 mg OD for 3 days) and volume restoration.

A chronic alcoholic: A chronic alcoholic is likely to have disorders of all major systems (see adverse effects of alcohol). Volatile anesthetics and induction agent's dose requirement will be higher. The initial dose requirement of most drugs will be higher (increased volume of distribution) but their effect is prolonged. Thrombocytopenia, leukopenia, anemia, coagulopathy and electrolyte derangements (hypomagnesemia, hypophosphatemia, and hypocalcemia) are common. Postoperative course may be complicated by alcohol withdrawal syndrome (AWS). This may present as tremor, gastric upset, sweating, hypertension, hyper-reflexia, anxiety, and agitation may worsen to delirium, hallucinations, and seizures, within 6 to 24 hours (up to 5 days) without alcohol.

If the patient has to undergo a lower abdominal surgery or a hernia, which methods would you prefer-regional/spinal/epidural/GA? Why?

The most important consideration in a cirrhotic patient undergoing anesthesia and surgery is maintenance of hepatic blood flow and oxygenation. A decrease in hepatic blood flow due to reduced hepatic arterial buffer response and simultaneous drop in cardiac output will lead to reduced hepatic and renal blood flow as well, making the patient susceptible to the development of hepatic decompensation and renal failure.

Herniorrhaphy can be successfully carried out under inguinal block. For lower abdominal surgery a central neuraxial blockade can be safely undertaken provided coagulopathy is tackled properly by platelet transfusion, FFP and cryoprecipitate administration. INR should be maintained below 1.4 (PT within 4 seconds of control), at the time of epidural catheter insertion and removal. Other concern is hypotension and reduced hepatic blood flow which can be managed with fluid infusion (optimizing preload) and administration of vasopressors to keep the blood pressure normal.

What anesthesia technique would you use for this patient undergoing hepatic resection?

A combination of GA and epidural anesthesia is best for this patient. The important consideration is to maintain the hepatic blood flow within normal limits by ensuring normal blood pressure.

Which agents are preferred for premedication?

In absence of hepatic encephalopathy and moderate hepatic dysfunction, small dose of benzodiazepine (midazolam) can be safely used. In patients with even early HE, benzodiazepines and opiates should both be avoided.

How are benzodiazepines metabolized? Which of these do not have active metabolites? What is the half-life of metabolites of diazepam and midazolam?

All benzodiazepines are metabolized in the liver in 2 phases, oxidation followed by conjugation. Lorazepam, oxazepam and temazepam do not have active metabolites. Diazepam is metabolized to nordiazepam (60 hours) oxazepam (8 hours) temazepam (12 hours). Midazolam is metabolized to α-hydroxymidazolam, an active metabolite with a short half life and it does not contribute to prolongation of action of midazolam.

Which induction agents are preferred? What dose adjustments may be required?

There are no preferred induction agents. Thiopentone can be used safely provided one understands that the dose needs to be reduced (in view of increased free fraction of drug due to reduced albumin levels) and titrated to sleep. Propofol has been safely used as an induction agent and as continuous infusion (for 8 h) for maintenance of anesthesia. It was observed that though the volume of distribution was significantly increased and total body clearance decreased in cirrhotic patients, this did not increase terminal elimination half life. Cirrhotic patients with moderate hepatic dysfunction still metabolize etomidate normally.

What is the impact of liver disease on pseudocholinesterase production and duration of action of succinylcholine?

In cirrhotic patients with hepatic dysfunction, the levels of pseudocholinesterase will be decreased. However, the duration of action of succinylcholine is not prolonged till pseudocholinesterase levels drop to less than 50% of normal.

What changes in the dose and dose intervals of non-depolarizing muscle relaxants are required?

In patients with cirrhosis the initial dose requirement of non-depolarizing muscle relaxants will be increased (due to increased volume of distribution), however smaller subsequent doses will be needed as duration of action of most relaxants (except atracurium) is prolonged. The best way to use a muscle relaxant is to use neuromuscular monitoring.

Which muscle relaxants are preferred? Enumerate protein binding, alternate route of excretion/ metabolism for all common muscle relaxants. Are there any concerns while using atracurium or vecuronium?

Atracurium and cis-atracurium are the preferred muscle relaxants. Atracurium is mainly metabolized by Hoffmann elimination (spontaneous degradation at physiological pH and temperature) into laudanosine and monoacrylate. Smaller amount is hydrolyzed by nonspecific plasma esterases. Since no organ is required for Hofmann elimination, duration of action is not prolonged with either renal or hepatic dysfunction. Laudanosine is eliminated by hepatic clearance and it will accumulate in presence of liver function, the concern being seizures (Also accumulates in patients with renal failure). However, doses of atracurium during anesthesia are too small to result in the levels of laudanosine that cause seizures.

Pancuronium is about 70–90% protein-bound, mainly metabolized by the liver to 3-hydroxy (50% as potent), 17-hydroxy metabolite and 3, 17-dihydroxy metabolites (both less active). About 40% is excreted unchanged by kidney. About 10% is excreted in bile. In abnormal hepatic function, the volume of distribution is increased; however, more free drug is available due to reduced plasma albumin that can increase renal elimination. Duration of action is prolonged. 60–80% of vecuronium bromide is bound to plasma protein.

Vecuronium, in the liver, is deacetylated to the active metabolites 3-hydroxy (approx 50% potent) and 17-hydroxy- and 3, 17-dihydroxyvecuronium. About 25–50% of total dose is excreted in bile in up to 42 h. About 60–80% is bound to protein. Remaining drug and metabolites are excreted via kidney. Studies in patients with hepatic dysfunction and cholestasis shown that after larger doses (0.2 mg/kg), the duration of neuromuscular blockade may be considerably prolonged.

Rocuronium is 30% protein bound. It deacetylated in the liver to 17-desacetyl-rocuronium (1/20 as active) and undergoes hepatobiliary excretion. Studies in patients with cirrhosis have shown an increased initial dose requirement (increased volume of distribution) almost doubled duration of neuromuscular blockade (reduced clearance).

How are morphine and other opiates metabolized?

Morphine is largely metabolized in liver while smaller amounts are metabolized in kidney and brain. Morphine-3-glucuronide (M3G) and morphine-6-glucuronide (M6G) are the major and active metabolites of morphine, in patients with hepatic dysfunction the duration of action of morphine is therefore prolonged. The glucuronides are then eliminated via bile and urine. In the cirrhotics with abnormal liver function morphine plasma clearance is significantly lower; the terminal elimination half-life is longer.

Pethidine is hydrolyzed to pethidinic acid and demethylated to norpethidine in the liver. Norpethidine is an active metabolite with a longer elimination half-life (8–12 hours). It is structurally similar to atropine and has anticholinergic activity leading to convulsant and hallucinogenic effects, on cumulation in patients with renal failure. Pethidine metabolites are conjugated with glucuronic acid and excreted into the urine.

Fentanyl is also metabolized in liver to norfentanyl by oxidation. Remifentanil is metabolized in the plasma by non-specific tissue and plasma esterases.

How do changes in liver blood flow and liver function affect the metabolism of drugs by the liver? What are the effects of surgical manipulations and regional anesthesia?

Hepatic drug clearance (metabolism) depends on two factors: the hepatic blood flow (greater the flow, greater the drug presented for clearance) and intrinsic enzyme metabolizing hepatic capacity for that drug. Hepatic extraction ratio is the amount of free drug taken up by the liver during one passage. When the extraction ratio is 1, all the drug reaching the liver (100% of drug amount) is taken up by the liver, and cleared from the blood. Thus the blood leaving the liver shall have very small amount left in it. Such drugs are called *high intrinsic clearance drugs.* The examples are propranolol, lignocaine, pethidine. In case of high clearance drugs the elimination is then directly proportional to the hepatic blood flow. Drugs with low intrinsic clearance are not as dependant on the hepatic blood flow, as higher blood flow will not increase their elimination further, the examples being diazepam, phenytoin, and coumadin.

Anesthesia leads to reduction in hepatic blood flow due to a reduction in blood pressure and cardiac output. Inhalational agents have a greater effect on the hepatic blood flow that the intravenous agents. Halothane causes the greatest reduction, then isoflurane, desflurane and sevoflurane show decreasing effects. Intravenous anesthetic agents propofol have no or very minimal effect on Hepatic arterial buffer response (HABR- an intrinsic regulatory mechanism to maintain total hepatic blood flow with changes in portal perfusion pressure—see above).

Upper abdominal surgical manipulations can reduce the hepatic blood flow by over 60% due to splanchnic vasoconstriction. Subarachnoid block to T4 level will reduce the blood flow by 20%.

Which inhalation agents can be used? Are all fluorinated volatile anesthetics safe? Can halothane be used? What is halothane hepatitis? Pathophysiology of halothane hepatitis (National Halothane Commission).

Isoflurane is the safest agent, i.e. with the least reduction in hepatic blood flow and with minimal (0.2%) being converted to acetylated protein adducts. Halothane hepatitis is hepatotoxicity associated with halothane administration. Two types of hepatotoxicity are seen with halothane, type I is common, benign and self limiting and occurs due to reductive biotransformation of halothane. A transient mild increase in serum transaminase and glutathione S-transferase concentrations is seen. Type I hepatotoxicity is not characterized by jaundice or clinically evident hepatocellular disease. Halothane hepatitis (type II hepatotoxicity) is probably immune mediated. Oxidative metabolism of halothane leads to formation of trifluroacetyl metabolites, which bind to liver proteins. In genetically susceptible individuals, antibodies are generated against these complexes and hepatotoxicity results. This is characterized fever, jaundice, very high serum transaminase levels and pathologically by massive centrilobular liver necrosis leading to fulminant liver failure; with a high (50%) mortality rate.. Type II hepatotoxicity appears to be immune mediated. The National Halothane study (1959–1962) was a retrospective study of possible relation between postoperative mortality due to hepatic necrosis due to use of halothane, and found that the risk of fatal hepatoxicity was 1 in 35000.

What is pharmacological preconditioning? Does it work?

Prophylactic administration of sevoflurane and isoflurane has been used in cardiac surgery to prevent cardiac dysfunction and reduce microscopic abnormalities due to reperfusion after an ischemic insult in patients undergoing cardiac surgery. A 30 minutes administration of sevoflurane before portal clamping was tried as a strategy for hepatic protection. The authors found that postoperative enzyme elevation was much less in patients who received sevoflurane as compared to those who did not. Another study in patients undergoing hepatic transplant found that sevoflurane preconditioning improved early allograft function. This has yet to become a standard of care in all centers as more studies are awaited.

What are the mechanisms of drug hepatotoxicity? Which drugs are hepatotoxic?

Mechanisms of drug hepatotoxicity: Classically these are of two types, very often it is difficult to ascribe a particular mechanism, and often these may overlap.

- *Intrinsic or predictable drug reactions:* The drug or its metabolite causes reproducible injury across species and is dose dependent. Acetaminophen and carbon tetrachloride are examples of this type of drugs.
- *Idiosyncratic drug reactions:* Idiosyncratic reaction is an adverse reaction that does not occur in most patients at normal dose of a drug and is not like the known effect of the drug. This is of two types: hypersensitivity or immune-allergic and metabolic-idiosyncratic. When hepatotoxicity is seen with fever, rash, eosinophilia and antidrug antibodies, and recurs swiftly on repeat exposure, it is said to be an immune-mediated reaction. In absence of these features it is likely to metabolic idiosyncrasy.
  - *Hypersensitivity:* The Hapten Hypothesis proposes that this reaction occurs not due to the drug itself, but by irreversible binding of the drug to a protein. This complex then acts as an allergen and initiates the reaction. Phenytoin and penicillins are drugs which cause hypersensitivity reaction.
  - *Metabolic-idiosyncratic:* It occurs due to an indirect metabolite of the drug with variable and often delayed response. Unlike intrinsic hepatotoxins, the response rate is variable and can occur within a week or up to one year later. Examples are ketokonazole, halothane.

What intraoperative monitoring will you use?

Noninvasive monitoring will consist of ECG, NIBP, $ETCO_2$, pulse oxymetry, fractional inspired concentration of volatile agent (optional), peripheral nerve stimulator for neuromuscular blockade monitoring, temperature monitoring. Invasive monitoring is extremely important in this patient. Invasive arterial pressure monitoring is extremely useful, as there are considerable fluctuations in blood pressure. This is accentuated during dissection around the inferior vena cava. Arterial line is also useful for ABG sampling to assess for metabolic disturbance, hypoxemia and hypercarbia. Intraoperative bleeding has been shown to be considerably reduced when the central venous pressure is maintained at CVP < 5 mm Hg, so a central venous catheter is very useful in these patients. Intermittent blood glucose monitoring and hourly urine output should also be monitor. Coagulation monitoring either with thrombocyte count and PT or thromboelastography is also necessary.

What are the potential problems and what precautions would you take during a prolonged surgery?

Potential problems during prolonged surgery in a cirrhotic patient include hypovolemia, hypotension and hypoxemia secondary to hemorrhage. Hemorrhage occurs because of

coagulopathy (if not completely corrected preoperatively and hypothermia) cirrhosis itself, and if the CVP is not maintained at < 5 mm Hg. Corrective measures aim at FFP, cryoprecipitate and platelet replacement. Adequate blood loss replacement with packed RBCs is also necessary.

Hypothermia frequently occurs due to convective loss of heat from the open abdomen. Active warming measures should be instituted including warming blankets and infusion of warm intravenous fluids.

How will you manage excessive bleeding?

Excessive bleeding should be managed by correcting coagulopathies, active warming and adequate replacement with blood and blood products. As mentioned earlier, reducing the central venous pressure will also help reduce bleeding.

How will you manage postoperative analgesia? What precautions should be taken if epidural analgesia is used?

In our patient, the preoperatively placed epidural catheter can be continued to be used for providing postoperative analgesia. Dilute (0.0625%) solutions of bupivacaine can be used as an infusion. Small doses of acetaminophen 325 mg in 3–4 doses/day can be added. Acetaminophen in these doses is shown to be safe in patients with reasonably preserved hepatic function. NSAIDs such as diclofenac may also be added for a synergistic effect. The catheter should be removed once the INR normalizes, which usually takes 4–6 days. If earlier removal is contemplated for some reason, then catheter should be removed after infusing fresh frozen plasma to correct INR.

What are the causes of postoperative jaundice?

Postoperative jaundice can be multifactorial in origin. The causes are as follows:

- Bilirubin overproduction
- Hepatocellular necrosis
  - Ischemic liver injury
  - Viral hepatitis
  - Drug-induced hepatitis
  - Hepatocellular necrosis after liver transplantation
- Intrahepatic cholestasis
  - Benign postoperative cholestasis
  - Infection
  - Medications
  - Intrahepatic cholestasis after liver transplantation
- Extrahepatic cholestasis
  - Upper abdominal surgery
  - Acalculous cholecystitis
  - Extrahepatic biliary obstruction after liver transplantation
- Miscellaneous causes
  - Genetic disorders of bilirubin metabolism
  - Total parenteral nutrition.

Suggested Reading

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30 Laparoscopic Cholecystectomy

P Ranganathan

A 50-year-old female patient presents with the history of jaundice 3 months ago, and pain in the right hypochondrium with anorexia and nausea since 1 month. She is diagnosed to have gallstones and is posted for a laparoscopic cholecystectomy.

What is laparoscopy?

Laparoscopy is the visualization of the abdominal cavity through an endoscope.

What are the advantages of laparoscopic surgery over open surgery?

1. Reduced stress response to surgery
2. Rapid return of GI function
3. Reduced postoperative pain and analgesic requirements
4. Improved postoperative respiratory function
5. Reduced recovery time
6. Improved cosmetic appearance
7. Less postoperative wound infection.

Describe the technique of laparoscopy.

Laparoscopic surgery involves insufflation of a gas (usually carbon dioxide) into the peritoneal cavity under pressure to separate the organs from the abdominal wall. This produces a pneumoperitoneum, which causes an increase in intra-abdominal pressure. Gas is insufflated into the peritoneal cavity at an initial rate of 4–6 liter per minute which gets cut off when the intra-abdominal pressure exceeds the pressure limits set by the clinician (usually 12–14 mm Hg). After that the pneumoperitoneum is maintained by a constant gas flow of 200–400 mL per minute.

What should be the properties of the gas used for insufflation?

The following properties are desirable in the gas used for insufflation:

1. It should not cause significant physiological changes.
2. Minimal absorption from the peritoneal cavity.
3. If absorbed, it should be rapidly excreted.
4. Should not support combustion.
5. Should be highly soluble in blood to minimize risk of gas embolism.

Table 30.1 Enumerate the advantages and disadvantages of various gases which may be used for insufflations

Gas	Advantages	Disadvantages
Air, oxygen	Easily available Inexpensive Limited physiological effects	Support combustion
Nitrogen	Does not support combustion Poorly absorbed—avoids hypercapnia	Low blood solubility—dangerous consequences if gas embolization occurs
Nitrous oxide	May be beneficial in patients undergoing procedures under regional anesthesia	Supports combustion
Helium	Poorly absorbed—avoids hypercapnia Does not support combustion	Low blood solubility—dangerous consequences if gas embolization occurs Not cost-effective
Argon	Poorly absorbed—avoids hypercapnia Does not support combustion	Low blood solubility—dangerous consequences if gas embolization occurs Not cost-effective decrease in hepatic blood flow
Carbon dioxide	Does not support combustion	Absorbed in large quantities from the peritoneal

Describe the ventilatory and respiratory changes during laparoscopy with CO_2 pneumoperitoneum

Effect of raised intra-abdominal pressure

The increase in intra-abdominal pressure results in cephalad displacement of the diaphragm, reducing FRC and compliance by as much as 30% to 50%. Peak airway pressure and plateau pressures rise by 50% and 80%, respectively, and pulmonary compliance is reduced by 47%. This results in an increase in the airway resistance and work of breathing. Hypoxemia may result from atelectasis and intrapulmonary shunting, and this is more common in obese patients and those with underlying cardiopulmonary disease.

Increase in partial pressure of CO_2

CO_2-pneumoperitoneum results in a progressive increase in $PaCO_2$ which reaches a plateau 20 to 30 min after the beginning of intraperitoneal insufflations. The increase in $PaCO_2$ depends on the intra-abdominal pressure and ranges between 15% and 30%. In patients without cardiorespiratory disease, the increased $PaCO_2$ results mainly from CO_2 absorption from pneumoperitoneum. It does not significantly modify either physiologic dead space or shunt. As a consequence, the gradient between $PaCO_2$ and end-tidal PCO_2 ($ETCO_2$) does not change and $ETCO_2$ monitoring adequately reflects $PaCO_2$ and can be used to guide adjustment of ventilation to prevent hypercapnia. In patients with cardiorespiratory disease or in cases of acute cardiopulmonary disturbances, pneumoperitoneum impairs pulmonary ventilation and perfusion, resulting in increased physiologic dead space, reduced alveolar ventilation and increased $PaCO_2$ to $ETCO_2$ gradient. This leads to a greater increase in $PaCO_2$ and more hyperventilation is required to prevent hypercapnia. In these patients, capnometry no longer provides reliable monitoring of $PaCO_2$. Carbon dioxide diffuses to the body more during extraperitoneal than intraperitoneal insufflations. Also extraperitoneal carbon dioxide insufflation leads to higher $PaCO_2$ values in the postoperative period.

Change in minute ventilation

During laparoscopy with local anesthesia, minute ventilation increases by as much as 60% to maintain normocapnea. During general anesthesia with spontaneous breathing, the compensatory hyperventilation is insufficient to avoid hypercapnia because of anesthetic-induced respiratory depression. In patients on controlled ventilation, a 15 to 20% increase in minute ventilation is needed to prevent hypercapnia.

What respiratory complications can occur during laparoscopy?

The major complications which can occur during laparoscopy are:

1. Carbon dioxide subcutaneous emphysema—due to extra-peritoneal insufflation
2. Pneumothorax, pneumomediastinum, pneumopericardium
3. Endobronchial intubation—due to cephalad movement of the diaphragm
4. Gas embolism
5. Aspiration of gastric contents.

Describe the hemodynamic changes during laparoscopy with CO_2 pneumoperitoneum

Effects of hypercapnia

Hypercapnia due to CO_2 pneumoperitoneum activates the sympathetic nervous system leading to an increase in blood pressure, heart rate, myocardial contractility, and arrhythmias. It also sensitizes the myocardium to catecholamines, particularly when volatile anesthetic agents are used.

Effects of raised intra-abdominal pressure

At IAP levels below 15 mm Hg, venous return is augmented as blood is squeezed out of the splanchnic venous bed, producing an increase in cardiac output.

At IAP levels greater than 15 mm Hg, cardiac output decreases transiently due to:

1. Decreased venous return
   a. Compression of the inferior vena cava along with the surrounding collateral vessels
   b. Pooling of blood in the legs
   c. Increased venous resistance
2. Myocardial depressant effect of anesthesia induction agents.
3. Raised systemic vascular resistance
   a. Release of neuro-humoral factors
   b. Increased vascular resistance of organs.

The increase in systemic vascular resistance causes an increase in arterial pressures. Right atrial and pulmonary artery occlusion pressures are elevated due to increased intrathoracic pressures and these cannot be considered to be reliable indices of cardiac filling pressure during pneumoperitoneum. Cardiac arrhythmias during laparoscopy are due to multiple causes—hypercapnia, peritoneal stimulation, reduced venous return, hypovolemia and venous gas embolism.

Describe the effects of pneumoperitoneum on other organ systems.

Renal effects: There is a decrease in renal blood flow and glomerular filtration rate due to the reduction in cardiac output. Renal vascular resistance may also increase due to the raised intra-abdominal pressure. This can manifest as a reduction in intra-operative urine output. Patients with pre-existing renal dysfunction are at risk of further deterioration. However, normally these changes are reversible and the urine output improves once intra-abdominal pressure comes down at the end of surgery.

Splanchnic and hepatic blood flow: The effect of pneumoperitoneum on splanchnic and hepatic blood flow is unclear. Animal studies suggest that while hypercapnia has a vasodilatory effect, this is opposed by the vasoconstricting effect of the raised intra-abdominal pressure.

Gastrointestinal effects: Raised intra-abdominal pressure can predispose the patient to regurgitation and aspiration. However, it is believed that the changes in the intra-abdominal pressure are transmitted to the lower esophageal sphincter, preventing this complication.

Neurologic effects: The combination of factors like hypercapnia, head-low position, and elevated systemic vascular resistance can lead to an increase in intra-cranial pressure with a resultant decrease in cerebral perfusion pressure. This may be exaggerated in patients with ventriculoperitoneal shunts which do not have a unidirectional valve.

Ocular effects: A slight increase in intraocular pressure has been described, though its clinical significance is not clear.

Neuroendocrine effects: Increased levels of stress hormones. Increase in ADH and aldosterone levels.

What are the alternatives to pneumoperitoneum for laparoscopy?

The gasless laparoscopic technique avoids using any gas for insufflation, relying instead on an abdominal wall lift to create an intra-abdominal space at atmospheric pressure. It can be considered for elderly patients or those with cardiopulmonary problems. Hand assisted laparoscopy is another form of laparoscopy.

Describe the preoperative management of this patient.

The patient must have a thorough preoperative assessment. Investigations like hemogram, liver and renal function tests, chest X-ray and ECG should be performed. If history or ECG is suggestive of cardiac disease, further testing in the form of 2D-Echocardiogrpaphy or stress testing is indicated. If the patient has a history of gastroesophageal reflux, she should be started on proton-pump inhibitors in the preoperative period. Deep venous thrombosis prophylaxis with heparin or low-molecular weight heparins should be considered.

What monitors will you use for this patient?

Electrocardiogram, noninvasive arterial pressure monitor, airway pressure monitor, pulse oximeter, $ETCO_2$ concentration monitor, peripheral nerve stimulator (optional) and core body temperature monitoring should be used.

In high cardiopulmonary risk patients, what additional monitors would you like?

For patients with compromised cardiopulmonary function, continuous arterial pressure, cardiac filling pressures and blood gases monitoring is indicated. In these patients, the $PaCO_2$-$ETCO_2$ gradient is increased after CO_2 pneumoperitoneum, and $ETCO_2$ may not accurately reflect the $PaCO_2$ status.

What is the anesthetic plan for this patient?

Balanced general anesthesia technique with endotracheal intubation and controlled ventilation is recommended. Nitrous oxide should be avoided. At induction, care should be taken during mask ventilation to avoid gastric insufflation, which increases the risk of trocar damage and may impede the surgical view. In cases of inadvertent gastric insufflation, the stomach must be aspirated before trocar placement to avoid gastric perforation. During pneumoperitoneum, controlled ventilation must be adjusted to maintain $ETCO_2$ at approximately 35 mm Hg.

Can this patient be managed without intubation?

General anesthesia without intubation can be performed with a laryngeal mask airway. However, the decrease in compliance during pneumoperitoneum can lead to airway pressures exceeding 20 cm H_2O. The ProSeal LMA may be a better alternative to the classic LMA because it allows an airway seal up to 30 cm H_2O.

Is it advisable to use nitrous oxide during this surgery?

It is recommended to avoid N_2O for two reasons. First, it diffuses into the abdominal cavity in concentrations sufficient to support combustion of intestinal gas; second, it will diffuse into CO_2/air bubbles, increasing their size and the potential for an obstruction of pulmonary circulation. There is some concern about N_2O diffusing into the bowel and causing

distension during laparoscopic surgery, but studies have shown no difference in operating conditions irrespective of whether N_2O was used or not.

Can this procedure be done under regional anesthesia?

Regional anesthesia offers several advantages: quicker recovery decreased PONV, less postoperative pain, shorter postoperative stay, cost effectiveness and fewer hemodynamic changes. Complications of general anesthesia such as sore throat, muscle pain, and airway trauma can be avoided. However, regional anesthesia will be suitable only if the patient is relaxed and cooperative and the surgical technique is gentle. The combined effect of pneumoperitoneum and sedation can lead to hypoventilation and arterial oxygen desaturation. Extensive sensory block is needed (T5 to L2) and shoulder tip pain due to diaphragmatic irritation may not be alleviated. This can be treated by intravenous fentanyl and by local spraying of the right diaphragmatic crus.

What position will be used for this patient? Describe the effects of the operative positions used in laparoscopic surgery.

Laparoscopic cholecystectomy is usually carried out in the reverse Trendelenberg position, with the patient's arms by the side. This position helps by displacing the abdominal contents away from the surgical site, to facilitate surgical exposure.

For laparoscopic procedures depending on site of surgery either trendelenburg (for pelvic surgeries) or reverse trendelenburg (for upper abdominal surgeries) position is used. The effects of surgical positioning are:

- *Reverse Trendelenburg (head up):* Preload is decreased, resulting in lowered pressure (MAP). Blood pooling in the lower extremities may increase the risk of venous thrombosis and pulmonary emboli. However, pulmonary function is improved.
- *Trendelenburg position (head down):* Cardiac output and central venous pressure increase. Pulmonary effects include impaired diaphragmatic function secondary to the cephalad displacement of abdominal viscera, resulting in decreased functional residual capacity, decreased total lung capacity, and decreased pulmonary compliance, predisposing the patient to developing atelectasis. Cephalad movement of the trachea may result in endobronchial intubation.

What other problems can occur during positioning?

- Nerve injuries can occur during positioning—the ulnar nerve when the upper limbs are adducted against the body, the brachial plexus when using shoulder braces, and the common peroneal nerve in the lithotomy position.
- In steep head-up position, the patient must be securely strapped to prevent the patient from slipping off the table.
- Access to intravenous lines may be a problem, and the use of extension tubing may be needed.

During the surgery, the patient has a sudden hemodynamic collapse. What is the differential diagnosis and how will you manage the patient?

There are several causes of hemodynamic collapse during laparoscopy:

1. *Hemorrhage:* Major vascular injuries can occur due to accidental insertion of the Veress needle or trocar into major vessels such as the aorta or common iliac vessels. Minor vascular injuries can involve the abdominal wall vessels.
2. *Pneumothorax:* Pneumothorax can occur with the gas traversing into the thorax either through a tear in the peritoneum or parietal pleura during dissection, or due to rupture of preexisting emphysematous bulla. There can be associated pneumomediastinum or subcutaneous emphysema. An initially asymptomatic pneumothorax can progress to a tension pneumothorax with high peak airway pressures, decreased saturation, hypotension and cardiac arrest.
3. *Gas embolism:* Gas insufflated under pressure into a tear in a vessel can lead to gas embolism. This leads to hypotension, arrhythmias, and desaturation, and can progress to cardiac arrest. A sudden fall in $ETCO_2$ should raise suspicion of gas embolism. The treatment consists of deflating the abdomen, ventilation with 100% O_2 for carbon dioxide elimination, central venous catheter(CVC) placement for aspiration of gas (inserting CVC after air embolism is generally of no use as air has already entered pulmonary circulation and cannot be aspirated from CVC), and cardiopulmonary resuscitation.

How will you manage this patient's postoperative pain?

Pain following laparoscopic cholecystectomy can arise from the incision site (incisional pain), visceral structures (abdominal pain) or referred from the subdiaphragmatic region as shoulder pain.

Shoulder pain is often mild in intensity and is due to irritation of the diaphragm by residual gas. It can be minimized by completely evacuating all gas from the abdominal cavity prior to closure.

Abdominal pain can occur due to stretching of the parietal peritoneum, release of inflammatory mediators of pain and

irritation produced by the blood. Recommended modalities of prevention and treatment include the use of suction to remove any blood and insufflated gas at the end of surgery as well as by instillation of normal saline to 'dilute' any local pain mediators.

Local infiltration of local anesthetic into the incision and trocar sites provides good pain relief. The role of intraperitoneally injected local anesthetic to provide pain relief has been debated. It should be remembered that toxicity can occur when larger doses are used.

Blocks like the transversus abdominis plane block may also be used for postoperative analgesia.

The most effective pain relief can be obtained by combining opioids, local anesthetics, and NSAIDs into balanced analgesia.

If this patient had heart disease, would laparoscopy be safe?

There are various factors which stress the cardiovascular system during laparoscopy. The reduction in preload causes the heart rate to increase to maintain cardiac output. There is increased afterload, as a result of the elevated systemic vascular resistance. The ventricular wall tension increases, reducing coronary blood flow thus leading to myocardial ischemia. Sympathetic stimulation may occur due to hypercapnia. Therefore, if the patient is intolerant of tachycardia, and cannot compensate for drop in venous return, laparoscopy may be dangerous.

What measures need to be taken during the anesthetic management of cardiac patients undergoing laparoscopy?

Patients with a profile suggesting depleted intravascular volume experience the most severe hemodynamic changes. Preload augmentation before peritoneal insufflation partially offsets hemodynamic deterioration.

- Invasive hemodynamic monitoring is essential.
- It must be remembered that central venous pressures may not be reliable in the presence of a pneumoperitoneum.
- Preload optimization is necessary to prevent hypotension.
- Gas insufflation must be slow, and intra-abdominal pressure must be kept as low as possible.
- Vasodilators can be used to correct or prevent the increase in afterload; however it should be kept in mind that this can lead to further drop in venous return and cardiac output.
- An inotropic agent such as dobutamine can be added to improve ventricular function.
- A diuretic may be needed at the end of the surgery.

Gasless laparoscopy may be a useful alternative in patients with ischemic heart disease who cannot tolerate CO_2 pneumoperitoneum.

What are the specific points in managing anesthesia for a patient with chronic obstructive pulmonary disease, who is posted for laparoscopic cholecystectomy?

Minimally invasive surgery offers advantages over open surgery in terms of less tissue handling, and less postoperative pain, leading to better respiratory function in the postoperative period. Preoperative respiratory optimization, with bronchodilator therapy in responsive patients, treatment of super-added infection, and chest physiotherapy is indicated. During ventilation of these patients, adequate expiratory time should be maintained to prevent air-trapping and development of auto-PEEP. Airway pressures may be high; it may be necessary to limit hyperventilation and accept modest levels of hypercapnia. $ETCO_2$ may not be an accurate estimate of $PaCO_2$ because the $PaCO_2$ to $ETCO_2$ gradient increases.

Is laparoscopy safe during pregnancy?

Laparoscopy can be performed during pregnancy, especially during the second trimester because organogenesis is complete, the uterine size is not large enough to interfere with the operative field, and the risk of spontaneous abortion is low.

What advantages does laparoscopy offer for the parturient?

1. Less exposure of fetus to potentially toxic agents.
2. Smaller incisions with less pain, and therefore reduced requirement for postoperative analgesics.
3. More rapid recovery and mobilization.

What are the physiological changes in pregnant patients after pneumoperitoneum?

Pregnancy is associated with physiological changes in the cardiovascular and respiratory system. These include a cephalad shift of the diaphragm leading to decreased FRC and compression of the inferior vena cava by the gravid uterus leading to hypotension. Laparoscopy can further exacerbate these changes, making the parturient more susceptible to hypoxemia and hypotension. Head low position will worsen the ventilation, though it improves venous return. Head-high position can improve ventilation, but it will further decrease venous return and cardiac output. Hyperventilation to treat hypercarbia should be controlled

as maternal respiratory alkalosis will lead to decreased uteroplacental perfusion.

What specific measures will you follow while anesthetizing a pregnant patient undergoing laparoscopy?

- Preoperative discussion with the obstetrician.
- Pneumatic compression stockings, as these patients are more prone for Deep Vein Thrombosis (DVT).
- Rapid-sequence induction with aspiration prophylaxis.
- Position with uterine displacement.
- Fetal heart rate monitoring if more than 16 weeks gestational age, in addition to other monitors.
- Orogastric tube to deflate the stomach, and improve visualization.
- Limit intra-abdominal pressure to 12–15 mm Hg.
- Routine tocolysis is not recommended—Isoxsuprine or terbutaline may be used in specific cases.

What are the contraindications for laparoscopy?

Absolute contraindications for laparoscopy include:

- Shock
- Significantly raised ICP
- Retinal detachment
- Right to left shunts.

Though laparoscopy is not absolutely contraindicated, special care must be taken in patients with bullous emphysema or patients having ventriculoperitoneal shunt, hypovolemia, congestive heart failure or other severe cardiopulmonary disease, and coagulopathy.

Can laparoscopic surgery be performed in children?

Laparoscopy has been used safely for procedures such as fundoplication, hernia repair and orchidopexy in all age groups of children, including neonates. However, to minimize the cardiorespiratory effects of raised intra-abdominal pressure, insufflations pressures should be limited to 5–10 mm Hg in neonates and infants and 10–12 mm Hg in older children.

Suggested Reading

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31 Colles' Fracture

P Jain, A Chatterjee

A sixty years old diabetic fell in the bathroom on an outstretched hand and sustained a wrist fracture.

What are the main features of Colles' Fracture?

Fracture of the distal radius as described by Abraham Colles in 1814 is known as Colles' fracture. There are several types of distal radius fractures such as: Colles fracture, Smith's fracture, Barton's fracture or Chauffeur's fracture (so called because the crank used to start old cars often kicked back and broke the chauffeur's wrists with a particular pattern). Most of these names are applied to specific patterns of distal radius fracture but confusion exists. "Colles' Fracture" is used as a generic term for distal radius fracture.

The patient usually presents with a history of fall on an outstretched hand and has a typical deformity of the wrist known as dinner-fork deformity.

What is the surgical management of Colles fracture?

A. Closed Reduction

Closed reduction of the fracture: Closed reduction means the restoration of the normal anatomical alignment of fragments by application of an external force in a direction opposite to that which produced the injury without making incision on the skin. Adequate anesthesia is of utmost importance for closed reduction of a Colle's fracture.

Maintenance of reduction: In the majority of fractures, the maintenance of the alignment of the fragments is done by immobilization with plaster of Paris cast.

Manipulation generally includes first placing the arm under traction and unlocking the fragments. The deformity is then reduced with appropriate closed manipulations (depending on the type of deformity) reduction, after which a splint or cast is placed and an X-ray is taken to ensure that the reduction was successful. The cast is usually maintained for about 6 weeks.

B. Open (surgical) Reduction and Internal Fixation

Majority of fractures can be satisfactorily managed by manipulation and closed reduction. However, in case of some fractures there is an inherent instability of the fragments or a tendency for delayed union or non-union. Such fractures are treated by open reduction of the fracture by surgical method and internal fixation.

What is the anesthetic management of Colles' fracture?

Closed reduction of a distal radius fracture can be performed under general anesthesia, intravenous regional anesthesia (Bier's block/IVRA), intravenous sedation, peripheral nerve blocks and hematoma block (done mainly in the emergency department where under sterile aseptic precautions the hematoma is aspirated using 18 G needle and 10 mL 2% plain lignocaine is injected into the area. This block acts by local action on the nerve endings at the site of fracture). The anesthesia options for patients undergoing an open surgery for reduction of a Colle's fracture would include regional (peripheral nerve block) anesthesia or general anesthesia.

The type of anesthetic technique used depends on the nature and duration of the surgery, patient's health, any medical conditions, and preferences of the patient, surgeon, and anesthetist.

What are the various regional anesthesia or nerve block techniques employed for colles' fracture?

The nerve blocks that can be employed in the increasing order of preference are: IVRA, brachial plexus block (infraclavicular or axillary) or elbow block.

Intravenous Regional Anesthesia (IVRA) Block (Bier's Block):

Described by August Bier in 1808, it involves injection of local anesthetic (Lignocaine) through an IV cannula sited on the affected hand. Two tourniquets are placed around the upper arm. The arm must be exsanguinated with an Esmarch bandage before injection of the local anesthetic. After exsanguination of the hand, forearm and upper arm to the distal tourniquet, the distal and proximal tourniquets are inflated. The distal tourniquet is deflated initially, and the first portion of the procedure is performed under tourniquet control with the proximal tourniquet.

The tourniquets should be inflated to at least 100 mm Hg above systolic blood pressure.

Onset of analgesia: 5 minutes, Complete analgesia within 10 minutes.

During the procedure, patients often become less tolerant of the proximal tourniquet at 40 minutes. At this point, the distal tourniquet is inflated over the anesthetized area. The painful proximal tourniquet can then be deflated. At the conclusion of the procedure, the tourniquet is deflated and patient is closely observed for at least 10 minutes.

In case the procedure is of a shorter duration, the tourniquet should remain inflated for a minimum of 25 minutes after local anesthetic injection even if surgery has been completed to minimize the possibility of a systemic toxic reaction. If block is to be reinstituted within 30 minutes after tourniquet release, 50% of the initial dose of the LA can be used.

However, one has to remember that it may be difficult to insert an intravenous cannula since there could be a lot of swelling on the hand. Pain due to the fracture may make IV insertion and exsanguination of the arm difficult.

Complications

- Leaking or accidentally deflated tourniquet
- Large volume of local anesthetic introduced into circulation, with symptoms and signs of toxicity

Drugs Used for IVRA

Nonadrenaline containing solutions of:

- Prilocaine 0.5%:
  - Drug of choice. Least toxic and largest therapeutic index.
  - 40 mL for upper limb, 60 mL for lower limb
  - Max dose for a 70 kg adult: 400 mg (approx. 6 mg/kg)
- Lignocaine 0.5%: alternative
  - Max dose for a 70 kg adult : 250 mg (3 mg/kg)
- Bupivacaine is contraindicated.

Advantages

- Simple, easy technique
- Rapid onset of action
- 99–100% success rate
- Low cost
- Can be used for outpatient surgery.

Disadvantages

- No postoperative analgesia
- Limited to procedures lasting < 1h
- Cannot be used where tourniquet is contraindicated , e.g. sickle cell disease
- Local anesthesia toxicity in case of tourniquet failure

Contraindications

- Patient refusal
- Raynaud's or peripheral vascular disease
- Sickle cell disease
- Crush injuries
- Children (age < 10 years): intraosseous vessels in prepubertal patients may allow the LA to bypass the tourniquet
- Uncontrolled hypertension
- Obese patient (tourniquet may not compress the arteries effectively)
- Surgery lasting more than 2 hours.

Brachial Plexus Block

The brachial plexus provides motor and sensory innervations of the upper limb. It is formed by the anterior primary rami of C5–8, together with T1. There is also some contribution from C4 and T2 at times.

It can be blocked by different approaches, namely:

- Interscalene: For surgery on the shoulder, upper arm and elbow.
- Supraclavicular: Surgery on the elbow, forearm and hand.
- Infraclavicular: Surgery on the forearm, wrist and hand,
- Axillary: Hand, wrist and lower forearm surgery.

For surgery on the distal radius-ulna (lower forearm), such as that with a colles' fracture; an infraclavicular or an axillary brachial plexus block would be technique of choice.

Infraclavicular Brachial Plexus Block

Block occurs at the level of the cords and offers the theoretical advantages of a minimal risk of pneumothorax while blocking the musculocutaneous and axillary nerves.

Three techniques have been described;

- The classical (Raj) approach

- The coracoid approach
- The vertical infraclavicular approach.

The classical approach: The needle is inserted 2 cm below the midpoint of the inferior clavicular border and advanced laterally, directed towards the axilla, away from the lung and requires three-dimensional orientation of the pyramid-shaped anatomy of the axilla to be successful. A nerve stimulator can be used to identify the plexus. Marking a line between the C6 tubercle and the axillary artery with the arm abducted is helpful in tracing the course of the plexus. Incremental injection of 20 to 30 mL of solution is sufficient after the needle is correctly placed. The success rate is improved with a distal motor response. It is associated with a minimal risk of pneumothorax and good reliability.

The coracoid approach: The needle is inserted 2 cm medial and 2 cm caudal to the coracoid process, directed posteriorly and perpendicular to the skin. However, the insertion site is quite lateral and may result in the absence of blockade of the musculocutaneous nerve, thus removing the major advantage of this approach over the simpler axillary block.

Recently, the vertical infraclavicular block has been described. The needle entry point is immediately below the clavicle at a point midway between the sternal notch and the ventral apophysis of the acromion. The needle must be advanced in a vertical direction to a maximum depth of 4 cm.

Axillary brachial plexus block: The axillary approach is the most popular because of its ease, reliability, and safety. Blockade of the musculocutaneous nerve is not always produced with this approach but, it can be supplemented at the level of the axilla or at the elbow. The injection is made at the level of the terminal branches; hence the missed segment(s) show a nerve territory distribution and not a dermatomal pattern, as is the case when the injection is placed at the level of the roots.

The patient is made to lie supine with the arm to be blocked placed at right angle to the body with the elbow flexed to 90°. The needle is introduced just superior to the pulsation of the axillary artery at the lateral border of pectoralis major muscle. Digital pressure is often applied just distal to the injection site to aid proximal spread of the local anesthetic. A typical volume of local anesthetic required is 30–40 mL. The use of a large volume (40 mL) of local anesthetic ensures blockade of the musculocutaneous nerve for the tourniquet area.

Transarterial techniques have been described; the main complication from this technique is a hematoma formation.

Researchers have compared multiple and single injection techniques. A multiple injection technique involves identifying two, three or four separate terminal nerves in the plexus and injecting a small amount of local anesthetic around each of them. It increases the success rate in blocking the musculocutaneous nerve but there is a slightly higher chance of neuropraxia after the block.

Other Brachial Plexus Block Techniques

Interscalene brachial plexus block: This is the ideal block for surgery on the shoulder. Block occurs at the level of the upper and middle trunks. Blockade of the inferior trunk is usually incomplete and the ulnar nerve may be missed and may have to be supplemented if used for forearm and hand surgery.

With the patient in supine position, arm by their side and the head turned away from the side to be blocked, the posterior border of the sternocleidomastoid muscle is identified by making the patient briefly lift his/her head. The interscalene groove can be palpated by rolling the fingers posterolaterally from this border over the belly of the anterior scalene muscle into the groove. One can also ask the patient to sniff forcefully, this tenses the scalene muscles and the interscalene groove becomes prominent. A line extended laterally from the cricoid cartilage and intersecting the interscalene groove indicates the level of the transverse process of C6.

The point of insertion of the needle is the interscalene groove, lateral to the sternomastoid at the level of the cricoid cartilage. The needle is directed medially, caudally and posteriorly, i.e. towards the transverse process of the C6 vertebra. If the needle is directed cranially, there is a risk of accidental epidural, intrathecal or intravascular injection. 30–40 mL of local anesthetic solution is injected.

Complications

- Phrenic nerve block: Avoid in patients with contralateral phrenic nerve palsy
- Recurrent laryngeal nerve block
- Horner's syndrome
- Accidental epidural or intrathecal injection
- Accidental injection into the vertebral artery.

Supraclavicular Brachial Plexus Block

Blockade is done at the distal trunk–proximal division level. At this point, the plexus is compact and a small volume of solution produces rapid onset of reliable blockade of the brachial plexus. Patient is placed supine, head turned away from the side to be blocked. The needle is inserted immediately posterior and lateral to the subclavian artery pulsations behind the mid-clavicle. The needle is then directed caudally, medially and posteriorly to the upper surface of the 1st rib and walked along the rib. About 8–10 mL of local anesthetic is injected per division. A total of 20–30 mL

of solution is required. A reliable supraclavicular blockade requires elicitation of a paresthesia or motor response. The median nerve may be missed in this block.The supraclavicular approach is best avoided if the patient is uncooperative or cannot tolerate any respiratory compromise (because risk of pneumothorax is high). Other complications include frequent phrenic nerve block (40% to 60%), Horner's syndrome, and neuropathy. Bilateral blocks should be avoided.

Block of Nerves at the Elbow (Antecubital Fossa)

Elbow blocks are rarely used alone for forearm procedures. Usually this is used to supplement patchy brachial plexus blocks. Knowledge of the territory innervated by each nerve is essential so that the appropriate nerves can be blocked for the operation.

The following six nerves can be blocked in this area:

- Ulnar
- Median
- Radial
- Medial cutaneous nerve of forearm
- Lateral cutaneous nerve of forearm
- Posterior cutaneous nerve of forearm.

Approximately 5–10 mL of local anesthetic is appropriate for blocking each of these nerves.

These blocks are easy to learn and perform but are of shorter duration.

Ulnar Nerve

The ulnar nerve is easily accessible at its subcutaneous position posterior to the medial epicondyle; blockade at this site is associated with a high incidence of neuritis. The nerve is surrounded by fibrous tissue at this point; there is a high risk of intraneural injection hence the nerve is blocked proximal to and not in the ulnar groove. Use of a very fine needle along with a small volume of solution (1 mL) diminishes the risk. The nerve can be satisfactorily blocked with 5 to 10 mL of solution at a site 3 to 5 cm proximal to the elbow. The local anesthetic should be injected in a fanlike fashion without elicitation of a paresthesia.

Median Nerve

Lies medial to the brachial artery, where it emerges medial to the biceps tendon. It can be blocked at a point 1–2 cm proximal to the flexor skin crease at the antecubital fossa. Approximately 3 to 5 mL of LA solution is injected after eliciting a paresthesia. If no paresthesia is obtained, the solution can be injected in a fanlike pattern medial to the palpated artery.

Radial Nerve

The radial nerve can be blocked at the elbow as it passes over the anterior aspect of the lateral epicondyle. The intercondylar line and lateral edge of the biceps tendon are marked. A 22-gauge, 3 to 4 cm needle is inserted at a point 2 cm lateral to the biceps tendon and advanced until bone is encountered. About 3 to 5 mL of solution is injected in a fanlike fashion.

Medial Cutaneous Nerve of Forearm

Innervates the skin on the medial side of the forearm and can be blocked at the elbow after a median nerve block, by withdrawing the needle and directing it proximally injecting the LA subcutaneously between the head of the pronator teres muscle and the medial border of the biceps tendon.

Lateral Cutaneous Nerve of Forearm

The musculocutaneous nerve terminates as the lateral cutaneous nerve of the forearm and innervates the skin of the lateral side of the forearm. It can be blocked 1 cm proximal to the intercondylar line immediately lateral to the biceps tendon. Fanlike infiltration of 3 to 5 mL of solution subcutaneously at this site provides excellent anesthesia of this nerve.

Posterior Cutaneous Nerve of Forearm

Is the proximal branch of the radial nerve and innervates the skin on the posterior aspect of the forearm. It can be blocked by injecting the local anesthetic solution subcutaneously from the lateral epicondyle of the humerus to the olecranon.

Approximately 15 minutes after giving Biers block, patient became hypotensive and restless. What is your diagnosis and how will you manage?

After Biers block or regional block, one should have a very high index of suspicion for development of local anesthetic (LA) toxicity.

Signs of LA Toxicity

- Perioral paresthesia (lips, tongue, nose).
- Hypotension may be caused by hypoxemia following central apnea, direct myocardial depression or vasodilatation.
- Transient desaturation, cyanosis.
- Arrhythmias : resistant ventricular arrhythmias (e.g. with bupivacaine).
- Dizziness, tremors.
- Loss of consciousness, convulsions.

Management of LA toxicity

- Call for help
- Reinflate cuff if it has been deflated
- Airway: Secure airway, 100% O_2, lateral position
- Breathing: Ventilate
- Circulation: IV fluids, vasopressors. CPCR if cardiac arrest
- Convulsions: IV diazepam, lorazepam, thiopentone.

How will you improve safety of peripheral nerve blocks?

Peripheral nerve blocks till recently were performed without visual guidance where the needle insertion is guided only by surface anatomical landmarks. The blind techniques have a failure rate of approximately 20%, because of incorrect needle or local anesthetic placement. Blind techniques are also associated with an increased risk of nerve injuries due to direct impalement or intraneural injection of the drug.

Use of a nerve locator increases the likelihood of placing the needle in close proximity to the nerve, however does not completely eliminate the risk of nerve impalement and post operative neuritis. Multiple attempts at needle placement can lead to a lot of pain and discomfort for the patient.

Ultrasound is being increasingly used over the last several years to assist nerve blocks. Ultrasound allows direct visualization of peripheral nerves, the block needle and local anesthetic distribution and has proven to be very useful to guide targeted drug injections and catheter placement (95–99% success rate). Proficiency with the use of ultrasound for nerve blocks however requires training, practice and familiarity with sonoanatomy.

Advantages

1. Nerves are clearly visualized on ultrasound.
2. Shows the exact location of the nerves, more so valuable in patients with anatomical variations.
3. Provides a real-time imaging, guiding needle placement. Allows continuous adjustments in direction and depth.
4. Helps identify surrounding structures like vessels and pleura, and avoid them while still positioning the needle close to the nerve.
5. Local anesthetic spread can be visualized at the time of injection, allowing repositioning in case of an incomplete spread.
6. Lesser volume of local anesthetic required for a nerve block, as it is more precise.
7. Improves the onset time, success rate and quality of the block as compared with the nerve locator techniques.
8. May reduce the number of attempts and potentially the risk of nerve injury, though it has not been proven.

Suggested Reading

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3. Yentis Steven, Hirsch Nicholas, Smith Gary. Anesthesia and Intensive care A-Z, 4th edition.

32 Kyphoscoliosis

P Jain, A Kulkarni

A 16-year-old boy is posted for surgical correction of kyphoscoliosis.

What is kyphoscoliosis?

The spine normally curves posteriorly in the thoracic (kyphosis) and anteriorly in the lumbar (lordosis) region. Scoliosis refers to the lateral curvature of the spine. Curves are structural (lack normal flexibility and do not correct with bending towards the convexity or lying supine) or nonstructural (will resolve correctly with bending towards the convexity or in the supine position) due to limb length discrepancy. Scoliosis is sometimes associated with a kyphosis (hunchback) and lordosis (swayback).

How do you classify scoliosis?

Classification of Scoliosis

Idiopathic scoliosis: Cause is obviously unknown; however genetic inheritance, central nervous system dysfunction and nutrition have all been blamed for causation. This type accounts for 70% cases. In this type progression is much faster in girls while incidence is similar in both sexes. This is further divided into infantile (0–3 years), juvenile (4–10 years) and adolescent (10 years or more).

Congenital scoliosis: It occurs due to an insult during intrauterine period and is associated with other congenital anomalies (cardiac and urinary). This is further subclassified as open (meningomyelocele) and closed congenital scoliosis. Closed congenital scoliosis may occur due to: (a) failure of vertebral development which may be partial or complete and unilateral. (b) Unilateral or bilateral failure of segmentation of vertebrae.

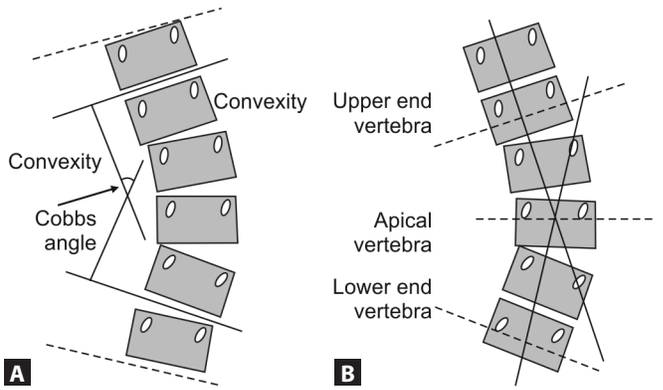
Acquired scoliosis: This may occur due to variety of reasons. This includes:

- Neuromuscular causes (muscular dystrophy, cerebral palsy)
- Mesenchymal disorders such as rheumatoid disorders, osteogenesis imperfecta.
- Neurofibromatosis
- Trauma
- Vertebral tumors.

How is the severity (degree) of scoliosis measured?

When the muscles responsible for vertebral alignment and weight transfer downward to pelvis weaken, a C-shaped scoliosis curve develops (Primary curve). The balance system tries to compensate for the misalignment. A lean to the left at the top, shifts the center of gravity to the left. To prevent tipping over, the lower spine is shifted to the right in an attempt to keep the center of gravity over the pelvis (compensatory secondary curve). The thoracic and lumbar regions are the most frequent sites of primary curve.

In the Lippman-Cobb method (Fig. 32.1), the first and last vertebrae that are most severely tilted towards the concavity are determined. Horizontal lines are drawn, one at the superior border of the upper end vertebra and at the lower border of the lower end vertebra. Perpendicular to these two lines is drawn and the intersecting angles are measured as shown. This angle is called the Lipmann-Cobb method and the angle as Cobbs' angle. Another method, is the Risser-Fergusson Method. In this method the degree of scoliotic curvature is measured from the angle formed by the intersection of lines drawn from the middle of upper and lower end vertebrae to the center of apical vertebra (Fig. 32.1). The Cobbs angle is the most commonly used method in clinical practice.



Figs 32.1A and B (A) Lippman-Cobbmethod method; (B) Risser-Fergusson method

What are the aims and indications for surgery in a patient with kyphoscoliosis?

The aim of surgery is to prevent progression of the curvature of the spine, to maintain posture, and to prevent progression of the pulmonary dysfunction. The indication for surgical intervention is documented progression of the scoliotic curves at any age. For scoliosis that is not due to neuromuscular disease, an angle of 40° may be indication for surgery, but for most of those with neuromuscular problems like Duchenne, this curvature is reached after the respiratory muscles are too weak for safe surgery. Instead, a 25° curve is taken as the cutoff point.

Nowadays, many patients have both anterior and posterior procedures performed either under a single anesthetic or in patients with a more severe or rigid curve the procedures are so major that they have to be staged as anterior followed by a subsequent posterior fusion. These sequential operations are performed within a specified time frame to optimize the results of stage 1 with that of stage 2. Anterior fusion involves thoracotomy or thoracoscopy. Thoracoscopic approach avoids cutting the chest/shoulder musculature, decreasing the morbidity of anterior spinal surgery.

Discuss preoperative respiratory assessment and optimization in patients undergoing kyphoscoliosis correction surgery. What other systemic abnormalities are likely? Which patients are likely to need elective postoperative ventilation?

Preoperative considerations for patients undergoing major reconstructive surgery of the spine needing careful attention are as follows.

These patients have extrinsic restrictive lung disease with a reduction in total lung and vital capacity. Spirometry will reveal the extent of restrictive disease and presence of obstructive element, if any. Reduction is worse with increasing spinal deformity. Respiratory mechanics are also worsened by the surgical correction spinal deformity which alters the elastic forces acting on the chest wall. Surgery also leads to fluid shifts and third spacing thus further compromising ventilatory capacity. It must be remembered that anesthesia and surgery further worsen the vital capacity. These patients also have increased pulmonary vascular resistance and ventilation and perfusion mismatch, which makes them more prone for hypoxia. All these factors must be carefully weighed to decide on the need for elective postoperative ventilation. Vital capacity below 40% almost always necessitates postoperative ventilatory support. Preoperative optimization involves teaching the patients breathing exercises, incentive spirometry and breath holding techniques. Bronchodilator therapy (if obstructive element present) and hydration may be necessary to loosen secretions. The risk of postoperative respiratory failure due to pneumonia can be minimized by ensuring proper postoperative analgesia, which will allow good cough and removal of retained secretions.

Associated congenital heart disease and mitral valve regurgitation must also be ruled out. Presence of preoperative neurodeficit must be carefully documented to prevent postoperative medicolegal issues. Presence of musculoskeletal abnormalities makes them prone to abnormal response to muscle relaxants.

Discuss preoperative counseling in patients undergoing kyphoscoliosis correction surgery

Explaining the wake-up test is most important. One must emphasize that though the patient may be awake, he/she will not feel pain, neither he/she is likely to remember anything about it postoperatively. For pediatric patients, it is important to assure the parents, as the children are likely to be affected by the anxiety of the parents.

Discuss methods for reducing blood loss and to avoid allogenic blood transfusion during kyphoscoliosis surgery.

Preoperative donation: In well-scheduled surgeries, preoperative donation may be obtained if hemoglobin is > 11g%. Subcutaneous erythropoietin injections 400U/kg may be used to induce erythropoiesis 4 weeks before scheduled date and blood may be collected every 4–7 days starting 2 weeks before surgery, last collection 3 days before surgery.

Preoperative isovolemic hemodilution: This is done on the operating table after induction of anesthesia, if the hemoglobin is >10 g%, and no comorbidities. The target hemoglobin is 7 g%. The volume may be replaced with a colloid as per institutional protocol. Starches should be used with caution in view of their effects on coagulation.

Proper positioning during surgery: Probably the most important factor, discussed below with prone position.

Induced hypotension should not be practiced as it makes the patient vulnerable to spinal ischemia particularly if there are problems with positioning.

What is your choice of anesthetic technique in this patient?

The anesthetic technique a combination of general and epidural anesthesia is preferred. Insertion of epidural catheter (placed ideally before induction of anesthesia—this may not be possible in younger patients) requires a clear understanding of the rotation of spine. Epidural insertion may have medicolegal connotations, if there are residual neurological problems postoperatively. Anesthesia is induced with standard induction agents either propofol or thiopentone in appropriate doses and neuromuscular blockade achieved with vecuronium 1mg/kg or atracurium 0.5 mg/kg. Orotracheal intubation is performed with age-specific nonkinking tube. Eyelids are taped shut and eye pads are placed over them to cushion them in prone position.

Additional intravenous access (apart from that taken prior to induction) recommended for kyphoscoliosis correction is a 16 g cannula right forearm and a 20 g arrow central venous line via right internal jugular vein. If acute isovolemic (or normovolemic) hemodilution is planned (if hemoglobin is > 12 g%, rare blood groups, etc.) 1–2 units of blood may be collected before patient is positioned for surgery. Volume loss is replaced with colloids or ringer lactate as per institutional protocol. An interpleural or paravertebral catheter may also be placed at this time to be used for postoperative analgesia.

Discuss physiological changes in prone position

Physiological changes associated with prone position are as follows:

Cardiovascular changes: Raised intrathoracic pressure causes reduction in venous return. The stroke volume is reduced with minimal changes in heart rates. Generally, the cardiac output has been shown to drop by about 20–30% of baseline value. Mean arterial pressure is maintained due to increased sympathetic activity (baroreceptor stimulation caused by reduced arterial filling).

Inferior vena cava obstruction is seen more often in patients where abdomen is compressed (not hanging free). This compression can cause engorgement of the epidural spinal vessels leading to poor surgical field and increased blood loss.

Respiratory changes: Significant increases occur in FRC and PaO_2 in prone position. In obese patients lung volumes, compliance and oxygenation have been found to be better than supine position. Contrary to earlier belief, it must be emphasized that blood flow to dorsal regions is maintained even in prone position, thus prone position is associated with a more uniform distribution of pulmonary blood flow and V/Q matching is improved in prone position.

What monitoring will you employ? Discuss monitoring used for testing spinal integrity intraoperatively.

Standard monitoring employed includes electrocardiography, pulse oximetry, end-tidal CO_2 monitoring (most important to detect circuit disconnection, leaks during prone position), noninvasive and invasive blood pressure monitoring (with a 22 g cannula in the right radial artery), urinary catheter for hourly monitoring of urine output. Airway pressure should be monitored again to detect kinking, displacement and accidental removal of the endotracheal tube. Accidental extubation can have disastrous consequences.

Spinal integrity is monitored using either the traditional "wake up test" or by using Somato-sensory evoked potentials (SSEP).

Wake-up test: During this test, anesthesia is lightened intraoperatively and patient is asked to perform certain commands. During kyphoscoliosis surgery the patient may be asked to move or wriggle his toes, once it is confirmed that he is able to follow commands. Once patient demonstrates the ability to perform these actions, patient is put back to sleep. For this not to be a frightening experience for the patient, it is imperative that proper preoperative counselling is performed. The patient needs to be assured that though he/she may be awake, there will be no pain or discomfort. It is also important to ensure complete amnesia (with midazolam or some such agent), so that patient does not have a postoperative recall of the test. A variety of techniques may be used for a prompt awakening intraoperatively. TIVA with propofol and sufentanil or remifentanil and atracurium (with reversal for wake-up test) infusions is one such technique. A N_2O/O_2/isoflurane, with switch to sevoflurane for the wake up test may also be used. The disadvantages of the wake up test are: an increased duration of surgery, a frightening experience for the patient and that the test provides information on neural damage only upto the time the test was performed. Damage occurring later is missed.

Evoked potential monitoring: An evoked potential is an electrical potential (at various loci such as cerebral cortex, brainstem, spinal cord and peripheral nerves) after application

of a stimulus, unlike the spontaneous potential detected by electroencephalogram. The stimulus is either applied or the potential recorded in the central nervous system. These can be of various types such as visual evoked potential (VEP), auditory evoked potential (AEP), somatosensory (SSEP) and motor evoked potential (MEP). During kyphoscoliosis surgery SSEP has been most commonly used, reports of transcranial MEP are also present in the literature.

Somatosensory evoked potential (SSEP) monitoring: It is used to monitor neuronal integrity during many surgeries (skull base surgeries which may require brainstem retraction, instrumentation of cervical spine, kyphoscoliosis correction surgery and surgeries for excision of spinal cord tumors, carotid endarterectomy, aneurysm repair surgery and many other surgeries on various regions of spine) particularly spinal fusion procedures. An appropriate peripheral nerve is stimulated and the responses are recorded at the somatosensory cortex. Thus a direct feedback is obtained testing posterior columns of the spinal cord. A change such as a decrease (> 50%) in amplitude or an increase (>10%) in latency indicates an interruption of the posterior column pathways. Though this sounds simple in theory, it requires particular skill and expertise as both amplitude and latency are affected by many anesthetic drugs (Table 32.1). Several other factors affect the SSEP; these are: hypoxia, anemia, hypotension, hypothermia, nerve ischemia and hypercapnia.

Advantage: SSEP provides continuous intraoperative monitoring of posterior column integrity during surgery, unlike the "wake up test".

Disadvantage: SSEP will not detect damage to the motor pathways. The effects of commonly used anesthetic agents on SSEPs are described below:

Table 32.1

Anesthetic agents	Effect on amplitude	Effect on latency
Thiopentone	Small/none	Increased
Etomidate	Increased	Increased
Fentanyl	Small/none	Modest/No increase
Diazepam	Decreased	Increased
Midazolam	Decreased	Increased
Ketamine	Increased	Increased
Propofol	None	Increased
N_2O	Decreased	No change
Volatile agents	Decreased	Increased

Motor evoked potential (MEP) monitoring: MEP is recorded after either direct stimulation of exposed cortex, or transcranial (Tc) cortical stimulation or magnetic cortical stimulation. TcMEP has been used to test neuronal integrity in kyphoscoliosis surgery.

In a large cohort of 500 patients undergoing surgical correction of idiopathic kyphoscoliosis, a combination of SSEP and TCMEP monitoring was used intraoperatively. The authors reported extremely small (0.014% n = 7) false positive rates i.e. change in monitoring data without postoperative neurodeficit rates. The identification of postoperative neurodeficit was also excellent (2 out of 2). There were no false negative results i.e. postoperative neurodeficit without intraoperative adverse change in the monitoring data. The authors concluded that combined somatosensory-evoked and neurogenic motor-evoked potentials monitoring during idiopathic scoliosis surgery represented a standard of care. It was felt that this combination obviated the need for an intraoperative wake-up test once reliable data are obtained and maintained.

What are the risk factors for intraoperative spinal cord damage? How do you minimize spinal cord damage?

The risk for spinal cord damage is related to length and type of surgical procedure, pressure and retraction and manipulation of underlying neural tissue and underlying spinal pathology. Intraoperative drop in spinal cord perfusion pressure will also lead to spinal cord damage. Careful positioning, maintaining spinal cord perfusion pressure (SCPP = MAP – CSFP). Studies in traumatic spinal cord damage suggest a doubtful neuroprotective role of methylprednisolone given within 8 hours injury. Since a hematoma in the spinal canal can compress and produce spinal cord damage, careful hemostasis along with proper adjustment of anticoagulant medications perioperatively is extremely important.

Discuss complications associated with prone position. Discuss postoperative visual loss seen after prone position.

Central nervous system complications of prone position are summarized in the Table 32.2.

Table 32.2

CNS complications			
Mechanism	**Anatomy**	**Caused by**	**Clinical consequence**
Arterial occlusion	Internal carotid artery, vertebral artery	Rotation, unrecognized cervical extension	Stroke, hemiplegia, quadriplegia, lateral medullary syndrome, brainstem and cerebellar infarcts

Contd...

Contd...

CNS complications			
Mechanism	**Anatomy**	**Caused by**	**Clinical consequence**
Venous occlusion	Abnormal veins	Poor positioning	Hemiparesis, paraparesis, quadriparesis bilateral venous infarcts
Air entrainment		Pneumorrhachis (air in spinal canal)	Transient quadriplegia
Cervical spine injury	Excessive neck flexion	Overstretching of cervical cord consequent ischemia	Transection below C5/6
	Excessive neck extension		Prolepsed intervertebral disk
Undiagnosed space-occupying lesions		Altered CSF flow dynamics epidural venous engorgement	Previously undiagnosed, lesions became symptomatic

Peripheral nerve injuries have been described frequently (0.1 to 0.14%) after prone positioning to the following nerves: supraorbital nerve (direct pressure during positioning), lingual and buccal nerves (inadvertent retraction of the jaw), brachial plexus, axillary nerve (both due to excessive abduction of arms), musculocutaneous and radial nerve, ulnar nerve, recurrent laryngeal nerve and phrenic nerve (both caused by over-extension or rotation of the neck), sciatic nerve, lateral cutaneous nerve of the thigh, dorsal nerve of penis. Direct and indirect pressure during prone position can cause a variety of injury manifestations, these include: contact dermatitis, pressure necrosis of skin at contact points, swelling of the tongue and oropharynx, compression of the trachea, swelling of the salivary glands, dislocation of shoulder, mediastinal compression, visceral ischemia, avascular necrosis of the head of femur. Limb compartment syndromes and rhabdomyolysis have also been reported after prone position.

Postoperative visual loss: It is a rare (0.0008%) but a dreaded complication of prone position. It has been reported that most (67%) cases of postoperative visual loss occur after prone positioning. A variety of opthalomological injuries have been reported: transient and permanent opthalmoplegia, orbital compartment syndrome, dislocation of intraocular lens, etc. The most commonly described injuries are ischemic optic neuropathy and central retinal artery occlusion. These two are compared in Table 32.3.

Table 32.3

	Ischemic optic neuropathy (ION)	Central retinal artery occlusion (CRAO)
Etiology	Intraoperative hypotension, prolonged surgery, excessive blood loss, large amounts of crystalloid infusion	Direct external pressure Emboli
Mechanism	Ischemia (due to factors above) Orbital edema leading to stretching compression of optic nerve	Decreased ocular perfusion pressure
Clinical features	Painless, bilateral, decreased light perception and visual fields	Painless, unilateral, periorbital swelling or ecchymosis

Discuss postoperative analgesia.

Postoperative pain control following spinal fusion is usually based on patient (or nurse) controlled analgesia (PCA or NCA) with opioids, supplemented with either nonsteroidal anti-inflammatory agents (for the pain at the site of surgery) or benzodiazepines (for muscle spasms). Children with cerebral palsy are vulnerable to undertreatment of postoperative pain due to communication barriers, concerns regarding opioid-induced respiratory depression, and individual opinion about pain perception in these children. Children with cognitive impairment , for whom behavioral pain scale was usually used, received smaller total opioid dose than those without cognitive impairment, for whom self-report was used. There is mounting evidence that epidural morphine is safe and efficient for postoperative analgesia following spine surgery. Low-dose (2–5 μg/ kg) intrathecal morphine administered in the operating room supplemented by PCA morphine provided better analgesia than PCA morphine alone, especially at rest; pain scores at coughing were similar in the morphine and placebo groups. Similarly, patients who received intrathecal morphine and sufentanil for surgery were breathing at a normal rate after surgery except for the first postoperative hour, when $PaCO_2$ was slightly elevated. No late respiratory depression was observed, but continuous monitoring of ventilation was considered necessary for at least 24 hours. There is no consensus in the literature regarding the benefits of epidural catheters after spinal surgery.

Clinical experience has indicated that the use of a continuous epidural infusion of local anesthetics with morphine delivered through a single epidural catheter provided improved analgesia when compared with intravenous opioids. However, it was noted that many

patients still had significant pain in the upper or lower portion of the surgical site. Tobias and coworkers reported placing two catheters following completion of the surgical procedure and prior to wound closure. One of them was inserted at the level T6-8 with the tip directed cephalad, to T1-4, and the other was inserted at T12 and positioned at the L1-4 levels. As the surgical wound was being closed, fentanyl and hydromorphone were administered through the catheters. This was followed by continuous infusion of 0.1% ropivacaine and hydromorphone 10 mg/mL. The mean of the median pain scores on the postoperative days 1–5 were always < 2 and the mean of the maximum pain scores < 4 on the postoperative days 1 to 5. Although the technique was effective, many of the patients still had episodes of severe pain with maximum pain scores of 6–7. No adverse effects related to epidural analgesia were noted. To avoid the possible additive respiratory depressant effects of the intravenous and epidural opioids, Tobias and coworkers used a short acting opioid (remifentanil) during the surgery. Interpleural catheters or epidural catheters can also be very useful for pain relief.

What are the complications associated with scoliosis surgery?

Aside from the complications due to intraoperative compromise of spinal integrity, which may give rise to variable neurological deficit, the complications of corrective surgery for scoliosis may be related to massive blood loss, neural deficit due to prone positioning, postoperative visual loss. Greater the degree of scoliosis, particularly above 100°, higher is the chance of postoperative complications. The rate of complications is also greater in patients with cerebral palsy, thoracoabdominal and transthoracic approaches, staged procedures, operative blood loss > 1000 mL, or previous spine surgery. Pulmonary complications are also a frequent result of scoliosis surgery. Other complications of scoliosis surgery include syndrome of inappropriate antidiuretic hormone release, pancreatitis, cholelithiasis, superior mesenteric artery syndrome, ileus, chylothorax, air and fat embolism, and tension pneumothorax.

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33 Large Thyroid Mass

M Shetmahajan

A 54-year-old female patient with a large thyroid swelling. She has no comorbidities. She is scheduled to undergo thyroidectomy.

What specific things are you seeking in the history and examination of a patient with thyroid swelling?

Size and Duration of Swelling

- Pressure effects on trachea: Dyspnea, stridor (effects of change in position)
- Pressure effects esophagus: Dysphagia
- Pressure effects on recurrent laryngeal nerve: Hoarseness
- duration: Longstanding large thyroid may be associated with tracheomalacia.

Retrosternal Extension

- Dysphagia, dyspnea
- Unable to 'get under the swelling'
- SVC obstruction/thrombosis—dilated veins in the neck and upper part of chest, facial edema.

Intratracheal Extension

- Dyspnea
- Hemoptysis.

Thyroid Hormone Status

- Signs, symptoms of hypo or hyperthyroidism.

Associated Endocrine Disorders

- MEN type 2A: Medullary thyroid carcinoma, pheochromocytoma, parathyroid hyperplasia
- MEN type 2B: Medullary thyroid cancer, pheochromocytoma, and growths around nerves (neuromas).
- MEN I: Pituitary adenoma, parathyroid hyperplasia, pancreatic tumors like insulinoma and rarely thyroid and adrenal involvement.

Thyroid Malignancies

- Usually present as thyroid nodules and are usually metabolically inactive
- Common types—Papillary, follicular, medullary, anaplastic
- Intratracheal extension may present as dyspnea and hemoptysis
- Distant metastasis to lung, bones and liver may be present especially in follicular type.

How would you present your diagnosis?

54-year-old female patient with size/side (e.g. large thyroid swelling) with/without airway compromise (e.g. with tracheal deviation and compression) with/without complications (retrosternal extension without SVC syndrome) with normal thyroid function (or controlled/uncontrolled hypothyroidism/hyperthyroidism) with/without comorbidities scheduled for subtotal thyroidectomy.

How are thyroid hormones synthesized in the body?

In the thyroid gland active uptake of iodide occurs in exchange for Na^+. Iodide uptake is stimulated by TSH. Iodide is oxidated to active iodine hydrogen peroxide (H_2O_2). The reaction is catalyzed by thyroid peroxidase (TPO). Iodine is actively transported across the apical surface of the follicular cell. Active iodine is incorporated into the tyrosine residues of thyroglobulin molecules to form mono and di-iodotyrosines (MIT and DIT).

Thyroglobulin is taken up into the colloid of the follicle where a coupling reaction between pairs of iodinated tyrosine molecules occurs. The coupling of two DIT produces tetraiodothyronine or thyroxine (T_4) while the combination of

DIT with MIT produces tri-iodothyronine (T_3). This reaction is catalyzed by TPO. Thyroid hormones are stored in this state and are only released when the thyroglobulin molecule is taken back up into the follicular cells. Thyroglobulin droplets are taken up by the follicular cells by the process of pinocytosis which is stimulated by TSH. Fusion of the droplets with lysosomes results in hydrolysis of the thyroglobulin molecules and release of T_4 and T_3. Approximately 100 µg of thyroid hormones are secreted from the gland each day, with around 90 % in the form of T_4 and the remainder as T_3. T_4 undergoes peripheral conversion to T_3 (T_3 is 3–5 times more active than T_4) in the liver and kidney or to reverse T_3 (little or no biological activity) glucocorticoids, propranolol and amiodarone block conversion of T_4 to T_3.

99.98% of T_4 and 99.8 % of T_3 is bound. It is the free fraction which is responsible for the actions and the negative feedback to the pituitary gland.

T_3 and T_4 Regulation

The production of thyroxine and triiodothyronine is regulated by thyroid-stimulating hormone (TSH) released by the anterior pituitary, which is in turn released as a result of TRH release by the hypothalamus. The thyroid and thyrotropes form a negative feedback loop. TSH production is suppressed when the free T_4 levels are high and vice versa.

What are the actions of thyroid hormone?

Thyroid hormones act on almost all cells of the body with effect on growth and development. Many of the actions of thyroid hormones are mediated by their binding to nuclear receptors that have a preferential affinity for T_3. T_3 receptors are steroid hormone receptors which regulate expression of genes coding for enzymes which regulate cell function.

CNS: Effect on neuronal function and reflexes. Reaction time of stretch reflex is shortened in hyperthyroidism. Also affects reticular activating system.

CVS: Actions of thyroid hormones and catecholamines are interrelated. Thyroid hormones have actions similar to epinephrine in stimulating cardiovascular system, nervous system and metabolic rate. Thyroid hormones increase number and affinity of beta adrenergic receptors thus increasing their sensitivity to catecholamines causing increased cardiac output and tachycardia.

Metabolism: Thyroid hormones increase metabolic rate and oxygen consumption of most tissues. They also increase carbohydrate absorption from the gut, increase protein catabolism, mobilize fatty acids and lower serum cholesterol.

Growth: Thyroid hormones are important for brain development and skeletal maturation.

How will you clinically assess thyroid function status in the patient? What are signs and symptoms of hypo and hyperthyroidism? State eye signs in hyperthyroidism.

History of taking any medicines—duration and dose, previous thyroid function test.

Symptoms and signs of hyperthyroidism: Hyperactivity, weight loss and tremor (tested in outstretched hands and tongue) are classical symptoms. Other symptoms are palpitations, diarrhea, intolerance to heat, large muscles group weakness, menstrual abnormalities, osteopenia.

Tachycardia (especially a high sleeping pulse rate), warm moist skin, irregularly irregular pulse (atrial fibrillation), mitral valve prolapse, heart failure and ischemic heart disease.

Eye Signs

Eyelid retraction ("stare") (Dalrymple sign): The eyelids are retracted upward above the superior corneoscleral limbus and the "white" of the sclera is seen.

Lid-lag (von Graefe's sign): When the patient tracks an object downward with their eyes, the eyelid fails to follow the downward moving iris, and the same type of upper globe exposure which is seen with lid retraction is seen temporarily.

Joffroy's sign: Absence of wrinkling of the forehead when looking upwards with the face tilted downwards. These ocular signs are not the same as exophthalmos (protrusion of the eyeball) which occurs specifically in Graves' disease. The forward protrusion of the eyes is due to immune mediated inflammation in the retro-orbital fat. Exophthalmos may exacerbate hyperthyroid lid-lag and stare.

Thyroid Function Tests

Low TSH, high free T_3, T_4.
Subnormal TSH response to TRH.
Other blood tests: Mild anemia, thrombocytopenia, hypercalcemia, raised alkaline phosphatase.

Symptoms and Signs of Hypothyroidism

CVS: Bradycardia, abnormal baroreceptor function, reduced plasma volume, signs of heart failure, pericardial effusion.

RS: Impaired respiratory center control mechanisms, pleural effusions.

CNS: Slow movement, mental slowing, excessive sleepiness, depressed mood.

GI: Delayed gastric emptying, constipation. Impaired clearance of free water, and impaired hepatic drug metabolism.

Fatigue, modest weight gain, cold intolerance, menorrhagia, muscle cramps, vague aches and pain.

Dry coarse hair, dry skin, large tongue, swelling of the legs.

Anemia, hypoglycemia, hyponatremia, increased cholesterol levels.

Carpal tunnel syndrome.

How will you test thyroid function?

The basic thyroid function tests are: Total T_4 and T_3, Thyroid Stimulating Hormones (TSH)

Free T_4 T_3: These are better indicators of functional thyroid status than total levels as they are the metabolically active fractions and are not affected by thyroxine binding globulin (TBG) levels. Relationship between total and free levels depends upon binding to TBG which is affected by various conditions. TBG is increased by estrogens, tamoxifen, and pregnancy and in some cases of liver disease, including hepatitis. Here there would be a high total value but normal function. Decreased TBG is found with some cases of chronic liver disease, nephrosis and systemic disease, with large amounts of glucocorticoids, androgens/anabolic steroids, and acromegaly.

TSH

It is an early indicator of hypo-and hyperthyroidism in presence of a normal pituitary thyroid axis and is a useful screening test. TSH is not useful when pituitary thyroid axis may be disturbed such as at initiation of thyroxine therapy, pregnancy and in patients with hyperthyroidism in the early stage of treatment and in rare situations such as central hypothyroidism, end-organ thyroid hormone resistance and TSH-secreting pituitary adenomas.

Table 33.1

	Hyperthyroidism	Hypothyroidism	
		Primary	Secondary
TSH	Low	High	Low
T4	High	Low	Low

Other advanced investigations include TBG level, radio iodine uptake test and anti- thyroglobulin antibodies. Thyroid function tests are advisable in patients with goiter to detect subclinical hypo or hyperthyroidism.

What is sick euthyroid syndrome?

Euthyroid sick syndrome is described as abnormal thyroid function tests that occur in the setting of acute and severe nonthyroidal illness without preexisting hypothalamic-pituitary and thyroid gland dysfunction. The most common findings are a low T_3, T_4 and TSH. The thyroid function test abnormalities are reversible after recovery from the illness.

These changes may be caused partly by cytokines or other inflammatory mediators acting at the hypothalamus, pituitary, the thyroid gland, and the hepatic deiodinase system. The degree of thyroid function disturbance generally correlates with disease severity. It is not certain whether these changes are part of an adaptive response to lower tissue energy requirements during disease or could be a maladaptive response. Administration of thyroid hormones in this situation is controversial and has not been shown to improve outcomes.

How important is it to treat hyperthyroidism/ hypothyroidism?

Thyroid hormone is highly metabolically active and has vital effects on cardiovascular physiology. So patients should be rendered euthyroid prior to surgery. Uncontrolled hyperthyroid state, when subjected to stress of surgery and anesthesia, can lead to thyroid storm which can be life-threatening.

Hypothyroidism also causes the intraoperative complications and postoperative morbidity. Myxedema coma can be life-threatening.

What are causes of hyperthyroidism?

Grave's disease, thyroiditis, toxic multinodular goiter, toxic adenomas, iodine induced, struma ovarii, thyroxine overdose, side effects of certain medications like amiodarone, irradiation thyroiditis.

How is hyperthyroidism treated?

The options are antithyroid medications, surgery and radioactive iodine therapy.

The choice has to be individualized to each patient. Antithyroid drugs e.g. methimazole, propylthiouracil (PTU) act by interfering with the organification of iodine.

PTU in large doses also blocks peripheral conversion of T_4 to T_3.

When initiating treatment, patient is investigated 4–6 weekly till control is achieved.

Doses can then be reduced. TSH is poor indicator of response at initiation of treatment.

Patients with Grave's disease in remission need to be investigated for thyroid hormone levels as relapses are known.

What are the common side effects of antithyroid drugs?

Major side effect of antithyroid drugs (so corresponding investigations should be ordered preoperatively):

Agranulocytosis: May need granulocyte colony-stimulating factor.

Hepatotoxicity: Immunoallergic hepatitis seen with PTU.

Vasculitis: Acute renal dysfunction, arthritis, skin ulcerations, vasculitic rash, and upper and lower respiratory symptoms, including sinusitis and hemoptysis. May need high-dose glucocorticoid therapy or cyclophosphamide.

Teratogenicity: PTU better than methimazole during pregnancy. Both drugs are safe for lactation.

Iodides: Iodides inhibit hormone release at high concentration, decrease synthesis and block the conversion of T_4 to T_3. The onset of action is immediate but lasts only for a few weeks. Three drops of saturated solution of potassium iodide are given 8 hourly or the radio contrast dye ipodate can be given 0.5–3 g everyday.

Describe anesthesia management in hyperthyroid patient.

Surgery is delayed until the patient is euthyroid. An endocrinologist consultation is required to titrate therapy. Only life/limb/organ threatening emergency surgery should be done in an uncontrolled hyperthyroid state.

Elective Surgery

Elective surgery should be undertaken after a minimum of 4–6 weeks therapy with methimazole or propylthiouracil. The delay is essential to achieve control as there are large stores of thyroid hormone. Estimation of TSH and Free T_4 are recommended for evaluating control of hyperthyroid state.

If surgery is semi urgent, it should be scheduled after 7–14 days. Treatment with iodide and propranolol is advisable. Antithyroid drugs should also be started even though they by themselves would not achieve control. Dexamethasone 2 mg 6 hourly IV reduces hormone release as well as inhibits the peripheral conversion of T_4 to T_3. Lugol's iodine given for 10 days preoperatively, in a dose of 0.5 mL three times a day was used to reduce bleeding, as antithyroid drugs make the thyroid more vascular. Although this practice was supported by measurements of thyroid blood flow using uptake of a radioisotope of thallium, many surgeons have abandoned this practice.

Emergency Surgery

Esmolol is titrated to control heart rate (caution in patients with CHF). Esmolol is administered in the dose of 0.5 mg/kg IV followed by an infusion of 0.03–0.3 mg/kg/min.

General anesthesia is the technique of choice for thyroidectomy. Some centers specialize in thyroid surgery under cervical plexus blocks or cervical epidural anesthesia. These should only be attempted by anesthetists skilled in the procedure for a select group of patients.

All antithyroid medications are continued on the morning of surgery. Sedative premedication is prescribed if airway is not involved. Benzodiazepines reduce anxiety and catecholamine release. Anticholinergic drugs cause tachycardia and alter heat regulating mechanisms and are best avoided.

Monitoring would include SpO_2, BP, ECG, $etco_2$ and temperature. Arterial canula and continuous BP monitoring is indicated in patients with uncontrolled thyroid condition.

Maintain deep plane of anesthesia so as to reduce the sympathetic response to surgical stimulation. Hyperthyroidism has not been seen to increase MAC in animal studies. Isoflurane and sevoflurane are inhalational agents of choice.

Thiopentone decreases peripheral conversion of T_4 to T_3 and is preferred. Ketamine is avoided due to sympathomimetic action. Clearance and distribution volume of propofol are increased in hyperthyroid patients. When total intravenous anesthesia is used, propofol infusion rates should be increased.

Other than pancuronium which has a vagolytic action, muscle relaxants in current practice e.g. vecuronium or rocuronium; have minimal hemodynamic side effects and are safe. Succinyl choline may be used in indicated cases.

Avoid indirect-acting vasopressors e.g. ephedrine. Phenylephrine, a directly acting alpha adrenergic receptor agonist is the drug of choice for hypotension. Avoid adrenaline-containing solutions. Glycopyrrolate is used with neostigmine for reversal.

Antithyroid drugs are stopped after thyroidectomy but β-blockers are continued for 7–10 days as the half-life of thyroxine is 7–8 days.

What is a thyroid storm?

It is a life-threatening condition where exacerbation of hyperthyroid state is precipitated by acute stress such as infection, surgery and trauma. Patients on amiodarone can sometimes develop this condition. Tachycardia and hyperthermia are classical signs.

Altered consciousness may be present in unanesthetized patient.

Thyroid function tests are not diagnostic because the condition is thought to be precipitated by decreased binding of thyroid hormones to TBG increasing the amount of free fraction.

DD: Malignant hyperthermia, neuroleptic malignant syndrome, pheochromocytoma.

Treatment: Treat the precipitating cause, aggressive fluid management, cooling, propylthiouracil, sodium iodide and IV Hydrocortisone 100–200 mg 8 hourly.

α and β-blockers should be given under intensive hemodynamic monitoring. Magnesium has also been used to control dysrhythmias.

What are the causes of hypothyroidism?

Hashimoto's thyroiditis, lymphocytic thyroiditis, thyroid destruction (from radioactive iodine or surgery), pituitary or hypothalamic disease, medications and severe iodine deficiency.

What is myxedema coma?

Myxedema coma is a loss of brain function as a result of severe, longstanding hypothyroidism. This condition is mostly seen in elderly patients and tends to occur more often in women. The condition carries very high mortality.

Factors which may trigger myxedema coma in a person with poorly controlled hypothyroidism include drugs (particularly sedatives, narcotics, anesthesia, lithium and amiodarone), infections, stroke, trauma, heart failure, gastrointestinal bleeding, hypothermia, and failing to take thyroid medications as prescribed

Treatment: The combination of intravenous T_3 and T_4 is recommended. Fluid resuscitation is done with normal saline solution. Glucose supplementation is given as required. Normothermia should be achieved with active warming. Mechanical ventilation and vasopressors may be required.

How will you treat hypothyroidism?

L-Thyroxine: Treatment is started with 50–100 µg (25 µg in the elderly or in patients with ischemic heart disease). Dose is titrated to clinical improvement and by monitoring TSH levels. Estimation of TSH and Free T_4 is recommended for evaluating control.

Thyroxine (T_4) has a half-life of 7 days, onset of action is at 12 hours and it takes almost 2 weeks for peak action. The half-life of tri-iodothyronine (T_3) is 1.5 days and is available in injectible form.

Thyroxine supplementation may precipitate or aggravate IHD. In patients with significant IHD, early revascularization and immediate postoperative initiation of thyroxine supplementation is advisable.

Describe anesthetic management in patients with hypothyroidism.

Patients with overt hypothyroidism should be treated prior to elective surgery. This will typically take weeks. It is advisable to give the patient's usual morning dose of thyroxine. This is especially important for T_3 which has a shorter half life. If surgery can be delayed for 24–48 hours, IV T_3 can be given. The peak action of T_3 occurs at 36–72 hours.

Because of an increased incidence of adrenocortical insufficiency and a reduced adrenocorticotropic hormone response to stress, hypothyroid patients should receive hydrocortisone cover during periods of increased surgical stress. Whether or not surgery should be postponed in a mild or subclinical hypothyroid patient is debatable.

Describe anesthesia in a hypothyroid patient (emergency surgery).

Use regional anesthesia wherever possible.

Specific Investigations

- Hb as anemia is common
- Platelet count and clotting tests as dysfunction is known
- Serum electrolytes as hyponatremia can be present
- Blood sugar monitoring to detect hypoglycemia
- ECG—bradycardia, conduction blocks.

Avoid sedative premedication in overtly hypothyroid patients as they show increased sensitivity to sedative and anesthetic drugs, probably related to low cardiac output, reduced blood volume, impaired hepatic metabolism and abnormal baroreceptor function. Chemoreceptor responses are also blunted, increasing the risk of respiratory depression. Close postoperative monitoring is advisable.

Induction: Anesthetic drugs have to be used judiciously. Ketamine has the advantage of sympathetic stimulation. Fentanyl may be used in small titrated doses. Nitrous oxide is useful in reducing requirement of volatile anesthetics as these patients are sensitive to the myocardial depressant action of the latter. Intermediate duration nondepolarizing agents e.g. vecuronium are used. Pancuronium is not preferred in spite of its cardiovascular effects because of risk of prolongation of action and residual neuromuscular blockade.

Exaggerated hypotension is common and should be treated with judicious fluid and ephedrine. Pure alpha adrenoreceptor agonists are avoided. Dopamine and epinephrine can be used for severe hypotension. Such patients should also receive additional dose of steroids.

Patients may have airway edema, large tongue and enlarged thyroid making laryngoscopy and intubation difficult.

Intraoperatively monitor temperature, fluid and electrolyte status, blood sugars. Invasive BP monitoring (and central venous canulation and cardiac output monitoring)

may be required in hypothyroid patients undergoing major surgery (fluid shifts, blood loss, etc) and in those with cardiovascular involvement (arterial cannula under local anesthesia prior to induction). Neuromuscular monitoring with peripheral nerve stimulator is advised as these patients have increased incidence of myasthenia gravis. Higher current levels may be needed for nerve stimulation in hypothyroid patients due to thick skin and hypothermia. Warming measures are instituted to prevent hypothermia. Delayed recovery is common and postoperative ventilation may be required.

What problems do you anticipate in patients with large thyroid mass? What are the complications associated with retrosternal thyroid?

1. Tracheal compression, deviation, invasion. Tracheomalacia may be present in longstanding cases which can lead to postoperative airway problems.
2. Worsening of airway compromise in supine position and with loss of muscle tone. This can cause airway obstruction at induction of anesthesia and after administration of muscle relaxant. Loss of spontaneous breathing and negative intrathoracic pressure also worsens intrathoracic airway obstruction.
3. Retrosternal extension: This can lead to SVC syndrome, airway compromise, cardiac compression, cerebral hypoperfusion as a result of arterial compression and thyrocervical steal, phrenic and recurrent laryngeal nerve palsies, Horner's syndrome, pleural effusions, chylothorax and pericardial effusions.
4. Increased intraoperative bleeding.
5. Injury to laryngeal nerves—vocal cord palsy causing stridor and impaired airway reflexes predisposing to aspiration risk.

How will you assess for tracheal deviation and compression?

History: Difficulty in breathing, stridor or stridor on lying down.

Examination

For deviation: Palpation of and auscultation on anterior neck to diagnose deviation.

For compression: Positive Kocher's test: slight push on the lateral lobes causes stridor.

Investigations

X-ray neck: AP, lateral. This is hardly required nowadays; as most patients will already have a CT-scan of the neck.

CT scan neck and upper thorax: This will accurately delineate the site and degree of airway compromise. This will help to predict the tracheal tube diameter and guide placement.

Flow volume loops: These will help in diagnosing subclinical airway obstruction. Flow volume loops will not give additional information if the patient has overt obstruction.

Flow Volume Loops

The pattern seen on flow volume loop depends on site of tracheal compression if variable obstruction. Suprasternal compression will show an extrathoracic pattern i.e. the inspiratory flow is predominantly affected whereas infrasternal compression will have intrathoracic pattern i.e. expiratory flow is limited. Large goiters and intratracheal extensions usually cause fixed obstruction where both inspiratory and expiratory flows are limited.

How will you assess for retrosternal extension of thyroid mass?

Symptoms

1. Dyspnea, choking, hoarseness, dysphagia.
2. Unable to 'get below the swelling'.
3. Percussion over the manubrium sternum (seldom followed in current practice).
4. Lateral X-ray neck.
5. CT-scan.

SVC obstruction: Patient will have edema and dilated veins on head, neck and upper part of chest. Severe cases may have respiratory distress due to airway edema.

Venous access should be secured in lower limbs or femoral vein central line inserted. Increased intraoperative bleeding is anticipated.

Pemberton's sign is the development of facial flushing, distended neck and head superficial veins, inspiratory stridor and elevation of the jugular venous pressure (JVP) upon raising of the patient's both arms above his/her head simultaneously, as high as possible. A positive sign is indicative of SVC syndrome. In current practice, with widespread use of CT-scan, this test is rarely performed as it can be dangerous in the OPD setting.

Which investigations will you prescribe and why? What is the role of flow volume loops?

Hemoglobin: Baseline. To estimate tolerable blood loss, bone marrow suppression by antithyroid medication

Thyroid function tests: To confirm euthyroid state (to detect subclinical abnormality)

ECG: Screening for IHD/dysrhythmias
Serum creatinine if patient > 60 years
X-ray chest.

CT neck: Anesthetist should always read CT scans to assess airway status.

X-ray neck AP/lat if CT neck not available—for tracheal deviation/compression.

Flow volume loop to detect subclinical airway obstruction and type and site of obstruction i.e. fixed/variable, intrathoracic /extrathoracic.

Indirect laryngoscopy to diagnose preexisting vocal cord palsy. This may also alert anesthetist to a difficult airway.

What are the principles of anesthetic management for this patient?

- Airway management at induction of anesthesia, maintenance and during recovery period.
- Management of complications of hypo/hyperthyroidism.
- Management of problems due to retrosternal extension.

What monitoring will you institute?

ECG, SpO_2, $ETCO_2$, Temp, BP.

Invasive arterial pressure monitoring if the patient has hyperthyroid state or major blood loss is expected.

How will you manage the airway in this patient?

Preparation: Preparation and planning are of utmost importance when dealing with a difficult airway. Alternate plans of action if the primary plan fails should be discussed by the anesthesia and surgical team to prevent panic and delays in management.

Patient might have airway obstruction due to external pressure, invasion into the trachea, severe deviation or vocal cord palsy.

Problems Anticipated

Deviation: Distorted anatomy—visualization of larynx may be difficult.

Compression of trachea: Patient may be unable to lie supine. A small size endotracheal tube may be required.

In a large thyroid, emergency airway by cricothyroid puncture or tracheostomy may be difficult/impossible. However in very severe obstruction, tracheostomy under local anesthesia should be done by surgeon after careful reviewing the distorted anatomy. Ultrasound may be useful in identifying vascular structures near the trachea.

Review CT scan—to assess site and extent of tracheal/bronchial obstruction and plan airway management.

A preoperative awake fiberoptic bronchoscopy may help to assess the airway involvement and help decide the induction and intubation technique. If a FOB is not available, some anesthesiologists perform laryngoscopy under topical anesthesia to assess anatomy of larynx and decide plan of anesthesia.

- Avoid sedative premedication
- Difficult airway kit should be ready
- Keep small size flexometallic tubes handy
- Rigid bronchoscope (and skilled operator) should be immediately available if required to stent the airway and allow ventilation if airway collapse occurs on induction of anesthesia. Turning the patient lateral or using forceps to lift thyroid away may also help to relieve the compression.
- Equipment for cricothyroidotomy/tracheostomy
- Experienced surgeon should be immediately available.

In case of a compromised airway, an inhalational induction with sevoflurane in oxygen is the preferred technique as it preserves spontaneous breathing. IV agents are generally avoided as they are more likely to cause apnea. Muscle relaxants are best avoided when airway difficulties are anticipated. In patients who cannot lie supine due to airway obstruction, intubation may have to be done in semi-sitting position and induction of anesthesia in this position can lead to severe hypotension.

In severe displacement of trachea, a fiberoptic guided intubation under topical anesthesia is the recommended technique. However, this technique should be used with caution in severe obstruction as the small caliber of the airways may sometimes make the passage of adult fiberoptic scope difficult and induce complete obstruction.

Controlled tracheostomy following femoro-femoral cardiopulmonary bypass that had been initiated under local anesthetic has been described in a case of airway obstruction with supraglottic edema due to a large thyroid mass. This is described as the ultimate solution to the problem of a difficult airway!!!

A flexometallic tube is preferred when there is tracheal compression and the tube is placed beyond the length of compression.

Opioids with shorter duration of action like fentanyl are preferred in order to have minimal sedative effects at recovery. Isoflurane and sevoflurane are volatile agents of choice. Muscle relaxants (e.g. vecuronium) are guided by peripheral nerve stimulator.

If recurrent laryngeal nerve monitoring is practiced, muscle relaxants are avoided.

Other Perioperative Considerations

1. Eyes should be protected especially if exophthalmos is present.

2. The patient is positioned slightly head-up to help venous drainage.
3. Neck is hyperextended and should be well-stabilized.
4. Extension tubing for IV lines and long respiratory hoses may be required.
5. Valsalva maneuver in Trendelenberg position is carried out to check hemostasis.
6. Steroids may be given if extensive tracheal handling and edema suspected.

Extubation should be smooth and the patient's coughing should be avoided to prevent bleeding. The surgeon may wish to observe the movement of the vocal cords at the end of surgery. This may prove difficult as the patient is emerging from the anesthetic. In such cases, a fiberoptic scope may be used through an LMA.

At extubation, the possibility of tracheomalacia and vocal cord palsy should be kept in mind and the patient should be observed closely for the same. Tracheomalacia should be suspected in longstanding goiters and the surgeon may palpate the trachea to assess the quality of tracheal cartilages. Absence of leak around the ETT after deflation of the ETT cuff can help suspect tracheomalacia. Leak around ETT can be detected on IPPV by palpating over the trachea or observing for difference between delivered and returning tidal volume. During spontaneous respiration, the patient should be able to breathe around the tube when the tubal lumen is plugged.

Postoperative Analgesia

1. Wound infiltration with bupivacaine.
2. Paracetamol and NSAIDs usually are sufficient.

What are the problems of positioning for thyroid surgery?

Thyroid surgery requires hyperextension of head. Care should be taken to support the head adequately to prevent strain on cervical spine ligaments and spine injury. In patients with restricted or painful neck extension or severe osteoporosis, the limit of comfortable neck extension should be assessed prior to anesthesia and the same position maintained during intubation and surgery.

Arms should be adducted by the patient's sides whenever possible. When arms are positioned at right angles, care should be taken to prevent hyperextension at shoulder joint which can stretch the brachial plexus.

What are the possible intraoperative problems? Discuss their management?

A. Increased airway resistance (causes)
   - Kinking of tube, secretions obstructing the tube, endobronchial intubation, bronchospasm
   - Surgeons manipulating the trachea during dissection
   - External compression of trachea/bronchi distal to the endotracheal tube in cases of large retrosternal thyroid.
   - Rarely a part of intratracheal tumor gets dislodged during intubation or manipulation by the surgeon and migrates distally into the tracheal or bronchial lumen causing obstruction.

B. Accidental extubation/disconnection
   - Hyperextension of head causes ETT to migrate outwards
   - Surgeons operating near the airway, so more chances of airway accidents.

C. Thyroid crisis.

Can thyroid surgery be done as day surgery?

Generally not, as postoperative airway problems are possible. May be possible in a very select group of patients with very good community medical support system (at present, does not seem feasible in India!)

What are the possible postoperative problems and their management?

1. *Respiratory Distress*

Causes

A. Specific to thyroid surgery
   a. Airway obstruction
      i. Wound hematoma—Traditionally, suture cutters were kept at the bedside to enable rapid relief of a hematoma. It is essential to open all layers to relieve obstruction. Early intubation is advisable when significant hematoma is present as airway compression and edema (hematoma obstructs venous drainage causing edema) increases with time, making intubation more difficult.
      ii. Recurrent laryngeal nerve damage (Electro physiologic monitoring of RLN has been used intraoperatively to prevent injury).
      iii. Tracheomalacia causing dynamic airway obstruction—Urgent reintubation, possibly tracheostomy and some form of tracheal support.
      iv. Laryngeal edema—uncommon—mostly related to large hematoma obstructing venous drainage. Other causes are trauma during intubation and myxedema.
      v. Hypocalcemia causing laryngospasm—hypocalcemia usually occurs at about 36 h postoperatively, usually temporary, unless there is severe damage to all the parathyroids. Treatment includes 10 mL 10% calcium gluconate. Long-term calcium supplementation is required.

B. All causes of postanesthesia respiratory distress.

2. Metabolic Disturbances

Hypothyroidism: May need thyroxine supplements.

Hypoparathyroidism: This may be due to direct trauma to the parathyroid glands, devascularization of the glands, or removal of the glands during surgery.

Hypocalcemia: The cause of transient hypocalcemia after surgery is not clearly understood. It may be attributable to temporary hypoparathyroidism caused by reversible ischemia to the parathyroid glands. Other hypotheses to account for transient hypocalcemia not caused by hypoparathyroidism include calcitonin release and hungry-bone syndrome.

Describe nerve supply of larynx.

1. Most of the muscles of the larynx receive their innervation via the recurrent laryngeal nerve (RLN) branch of the vagus nerve except the cricothyroid muscle which receives its innervation via the external laryngeal nerve. Cricothyroid muscle is important for tensing of vocal cord.
2. Sensory innervation of the larynx is via branches of the Vagus. Above the vocal folds the sensory innervation of the larynx is via the internal laryngeal nerve. Below the vocal folds it is by the branches of the RLN.
3. Parasympathetic innervation is mainly by the branches of the vagus nerve.

Describe nerve injuries common after thyroid surgery.

Causes: Contusion, traction, entrapment, actual transection and ischemia.

External branch of superior laryngeal nerve: Injury causes change of voice quality and loss of vocal stamina. Treatment involves speech therapy.

Recurrent laryngeal nerve

Unilateral injury: Unilateral cord palsy causes hoarseness and a weak cough but usually without significant airway obstruction at rest. Patient has risk of aspiration. Compensatory hyperadduction by the opposite cord may allow adequate vocal and sphincter function.

Bilateral injury: Bilateral partial injury of the abductors can cause unopposed adduction of cords causing complete glottic closure. Patient may need emergency tracheostomy. In bilateral complete denervation, both vocal cords are in cadaveric position—stridor—(predominantly inspiratory) is present and patient carries high-risk of aspiration. (extrathoracic variable obstruction pattern on a flow volume loop).

Initial treatment involves ensuring an adequate airway. If possible, endotracheal intubation is performed first. Intravenous steroids may be helpful in some cases of reversible damage.

A trial of extubation may be performed after several days. Extubate over a tube exchanger e.g. cook catheter (particularly if intubation was difficult) and in a controlled setting as reintubation may be necessary. Emergent tracheostomy may be needed.

If nerve function has not recovered after a second trial of extubation, tracheotomy is certainly warranted.

Corrective procedures should not be performed for at least 6 months after surgery as recovery is known.

Treatments of vocal cord paralysis include cordotomy (relieves stridor but increases aspiration risk), intracordal injection, laryngeal framework surgery, thyroplasty and laryngeal reinnervation.

What is the role of cervical plexus blocks and cervical epidural anesthesia in thyroid surgery?

Cervical plexus blocks and cervical epidural anesthesia in thyroid surgery are controversial.

Proponents cite avoiding airway manipulations and monitoring vocal cord function as a major advantage of the technique. Unilateral combined superficial and deep cervical plexus block have been performed for a unilateral surgery. A bilateral combined block is contraindicated because of the risk of bilateral phrenic nerve palsy. Superficial cervical plexus blocks have been tried unilaterally and bilaterally for analgesia but the reports are conflicting regarding its efficacy for analgesia. Superficial cervical plexus blocks have been used for video assisted thyroidectomy.

Cervical epidural catheters have been used in some reports. However the risk of bradycardia, hypotension and acute ventilatory failure related to respiratory muscle paralysis is high and patient needs close monitoring.

What are the implications of pregnancy in hyperthyroidism?

During pregnancy, the increase in thyroxine binding globulin causes an increase in total serum thyroid hormones which can lead to an erroneous diagnosis; so Free T_4 and T_3 levels should be used to determine thyroid status.

Both propylthiouracil and methimazole cross the placenta and can affect fetal thyroid function, especially at higher doses. Propylthiouracil is less teratogenic than methimazole. Most women with Grave's disease can be treated medically during pregnancy, with a target T_4 level at or slightly higher than the upper normal limit (and lowest effective dose) to ensure normal thyroid hormone levels in the fetus. Iodides cause fetal hypothyroidism and is contraindicated, so is radioactive iodine.

Grave's disease generally improves in the second and third trimesters of pregnancy, allowing reduction or discontinuation of antithyroid drug therapy, although it can exacerbate during the postpartum period.

During pregnancy, USG monitoring is used to check for the presence of a fetal goiter. Fetal goiter can indicate excessive antithyroid drug treatment in the mother or fetal Graves' disease. Maternal complications of Graves' disease in pregnancy include preeclampsia and preterm delivery.

Women receiving thyroxine therapy for hypothyroidism should have their dose increased by up to 50% during pregnancy. Avoiding maternal (and fetal) hypothyroidism is extremely important because of potential damage to fetal neural development, an increased incidence of miscarriage, and preterm delivery.

Suggested Reading

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34 Diabetes Mellitus

M Shetmahajan

How is diabetes classified?

According to American Diabetes Association (ADA), 2012, the etiologic classification of diabetes mellitus is as follows:

I. Type 1 diabetes (ß-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic.
II. Type 2 diabetes (could be predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance).
III. Other specific types
   A. Genetic defects of ß-cell function
   B. Genetic defects in insulin action
   C. Diseases of the exocrine pancreas
   D. Endocrinopathies
   E. Drug or chemical-induced
   F. Infections
   G. Uncommon forms of immune-mediated diabetes
   H. Other genetic syndromes sometimes associated with diabetes.
IV. Gestational diabetes mellitus (GDM).

Type 1 diabetes: Terms used previously include insulin-dependent diabetes or juvenile-onset diabetes. This results from a cellular-mediated autoimmune destruction of the ß-cells of the pancreas. Commonly presents in childhood and adolescence. The rates of ß-cell destruction are variable, rapid—mainly in children, and slow—mainly in adults. Those with complete destruction of ß-cell are dependent on insulin supplementation for survival. Ketoacidosis is a common presentation. These patients are prone to other autoimmune diseases like Graves' disease, Hashimoto's thyroiditis, Addison's disease, autoimmune hepatitis, myasthenia gravis and pernicious anemia.

Type 2 diabetes: Includes ~ 90–95% of those with diabetes. Terms used previously include non-insulin-dependent diabetes and adult or maturity onset diabetes. These patients have insulin resistance and usually have relative insulin deficiency. Most do not need insulin treatment for survival. Genetic predisposition is known in this type of diabetes. Obesity is common in this group. Ketoacidosis is unusual unless precipitated by stress such as infection.

Endocrinopathies: The abnormal secretion of certain hormones antagonises insulin action. For example, acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma.

Drug induced DM: Act by impairment of insulin action or reduction in insulin secretion. For example, β-adrenergic agonists, thiazides, glucocorticoids, thyroid hormone, diazoxide, phenytoin. α-interferon acts by developing islet cell antibodies.

Exocrine pancreatic diseases: Pancreatitis, trauma, infection, pancreatectomy and pancreatic carcinoma

'Metabolic syndrome' (also called syndrome X or insulin resistance syndrome): It is a non-causally linked cluster of symptoms which carries a high-risk of macrovascular disease. The cluster includes impaired glucose tolerance or diabetes, insulin resistance, raised arterial pressure, raised plasma triglycerides, central obesity and microalbuminuria

Why is it important to know the type of diabetes?

The risk of complications such as ketoacidosis and the perioperative management of blood sugar may differ in type I and type II diabetes.(Some observers have found a similar incidence of microvascular and neuropathic complications in types 1 and 2 diabetics when adjusted for duration of disease and quality of glycaemic control.) The type 1 patients require

basal insulin (and glucose) supplementation to prevent catabolism. They usually have less insulin resistance and tend to require lower insulin doses for same glucose load than insulin treated type 2 diabetics. However, type 1 patients tend to have more labile blood sugar concentrations during periods of stress than type 2 diabetics.

In secondary diabetes, the various endocrine abnormalities may have perioperative implications.

What other details should you seek in the clinical evaluation of a diabetic patient?

Duration of diabetes, medication history and quality of glycemic control, episodes of hypoglycemic or hyperglycemic complications, signs and symptoms of organ dysfunction associated with diabetes.

Complications of diabetes depend on the blood sugar control and duration of diabetes. Good blood sugar control is known to reduce risk of microvascular complications though the evidence for reduced risk of macrovascular complications is less robust.

What are the complications of diabetes?

The important complications for the anesthetist are **coronary heart disease, diabetic nephropathy and autonomic neuropathy** because these may have a direct effect on the development of perioperative complications.

The complications can be grouped as:

- **Macrovascular (coronary artery disease, peripheral arterial disease and stroke)**
  Mechanisms of injury:
  - Atherosclerosis
  - Increased coagulability and impaired fibrinolysis
- **Microvascular complications (diabetic nephropathy, neuropathy, and retinopathy)**
  Mechanisms of injury:
  - Osmotic stress from sorbitol accumulation.
  - Formation of advanced glycosylated end products
  - Free radical production and reactive oxygen species formation

Diabetic nephropathy

Mechanism of injury:

- Glomerular hyperfiltration
- Increase in glomerular hydrostatic pressure causes glomerular damage and microalbuminuria.
- Impaired endothelium-dependent vasodilatation.
  The earliest sign is microalbuminuria
  ACE Inhibitors and angiotensin receptor blockers have protective action

Diabetic Neuropathy

- Chronic sensorimotor distal symmetric polyneuropathy – most common symptoms are burning, tingling and numbness
- Mononeuropathies are sudden onset. Median, ulnar and radial nerves commonly affected
- Diabetic amyotrophy: Severe pain and muscle weakness and atrophy usually in thigh muscles
- Diabetic autonomic neuropathy: Autonomic dysfunction is detectable in up to 40% of type 1 and 17% of type 2 diabetic patients. Mechanisms suggested include local ischemia, tissue accumulation of sorbitol, altered function of neuronal Na^+/K^+-ATPase pump activity and immunologically mediated damage.

Diabetic retinopathy

- Dot hemorrhages, hard exudates, microaneurysms, retinal edema
- Proliferative retinopathy and retinal detachment.

Cardiac complications: The diabetic patient has an increased risk of:

- Coronary artery disease
  - Diabetic patients > 65 years of age have significant symptomatic/asymptomatic coronary artery disease. Silent ischemia may be present because of diabetic autonomic neuropathy
- Hypertension
- Peripheral arterial disease
- Systolic and diastolic dysfunction, related to:
  - Coronary artery disease
  - Hypertension
  - Left ventricular hypertrophy
  - Endothelial dysfunction
  - Obesity
  - Autonomic neuropathy.

Central nervous system

- Cerebral vascular disease related to
  - Hypertension
  - Dyslipidemia
  - Accelerated atherosclerosis
  - Abnormal endothelial proliferation
  - Increased coagulability and impaired fibrinolysis.

Renal dysfunction: These are related to hypertension, dyslipidemia and anemia. Iatrogenic exposure to nephrotoxins and cardiac surgery are responsible for precipitating renal failure.

Avoiding dehydration and nephrotoxins, and selective use of contrast-based radiographic procedures may help to prevent worsening of renal damage. Current data is not sufficient to support use of hemofiltration, renal dose dopamine, osmotic diuresis, or calcium channel blockade for renal protection. Use of N-Acetylcysteine does not have a strong evidence support but is part of contrast induced nephropathy prevention protocol in some centers.

Gastrointestinal system

- Risk of aspiration is increased because of autonomic neuropathy (decreased ability to coordinate swallowing and/or gastroparesis)

Respiratory system

Early investigations show that diabetes affects the respiratory system but evidence regarding its impact on functional status of the patient is lacking. The underlying mechanisms hypothesized include impaired lung elasticity, pulmonary microangiopathy and chronic systemic inflammation of diabetes. Some of the reported lung dysfunctions attributed to diabetes are:

- Decreased lung volume and lung diffusing capacity
- Reduced hypoxic-induced ventilatory drive (in patients with autonomic neuropathy)
- Diabetics may be prone to respiratory depression from opioids and sedative agents due to impaired chemoreceptor activity
- Airway difficulties.

What airway problems are possible and why? Which specific tests can be done to predict airway difficulties?

The "stiff joint" or 'limited joint mobility' (LJM) syndrome or diabetic cheiroarthropathy

- Manifests with joint rigidity. (Joints supporting the airway such as the temporomandibular, atlanto-occipital, and cervical spine joints are most commonly involved). Neck extension and laryngoscopy may be difficult.
- Cause: Nonenzymatic glycosylation of proteins and abnormal cross-linking of collagen in joints and other tissues.
- About one third of patients with long-term type 1 diabetes are reported to undergo a difficult laryngoscopy and intubation.

Tests

- *'Prayer' sign:* Ability to oppose the palms and fingers as in prayer. Diabetic patients with stiff joints have a positive test as they are unable to do this.
- *Palm print test:* Stiffness of the fourth and fifth interphalangeal joints causes alteration in palm print.
  - The palm and fingers of the dominant hand of the patient is painted with ink. Then the hand is firmly pressed on a white sheet of paper on a hard surface
  - Scoring:
    - Grade 0—All phalangeal areas visible
    - Grade 1—Deficiency in the interphalangeal areas of 4th and/or 5th digit
    - Grade 2—Deficiency in the interphalangeal areas of 2nd to 5th digit
    - Grade 3—Only the tips of digits seen.

How will you assess for autonomic neuropathy?

Overt signs and symptoms of autonomic disease are as follows:

Cardiovascular

- Resting tachycardia
- Exercise intolerance
- Orthostatic hypotension
- Silent myocardial ischemia.

GI

- Esophageal dysmotility (dysphagia)
- Gastroparesis
- Constipation
- Diarrhea
- Fecal incontinence.

Genitourinary

- Neurogenic bladder (diabetic cystopathy)
- Erectile dysfunction
- Retrograde ejaculation
- Female sexual dysfunction (e.g. loss of vaginal lubrication).

Metabolic

- Hypoglycemia unawareness
- Hypoglycemia-associated autonomic failure.

Sudomotor

- Anhidrosis
- Heat intolerance
- Gustatory sweating
- Dry skin.

Pupillary

- Pupillomotor function impairment (e.g. decreased diameter of dark adapted pupil)
- Argyll-Robertson pupil.

The differential diagnosis of diabetic autonomic neuropathy (DAN):

- Pure autonomic failure (Idiopathic orthostatic hypotension)
- Multiple system atrophy with autonomic failure (Shy-Drager syndrome)

- Addison's disease and hypopituitarism
- Pheochromocytoma
- Hypovolemia
- Medications with anticholinergic or sympatholytic effects (Vasodilators, sympathetic blockers)
- Peripheral autonomic neuropathies (e.g. amyloid neuropathy, idiopathic autonomic neuropathy)

Describe tests for diabetic autonomic neuropathy (DAN)

- Early stage: Abnormality of heart rate response during deep breathing alone
- Intermediate stage: An abnormality of Valsalva response
- Late stage: The presence of postural hypotension.

The tests are valid as markers of autonomic neuropathy if following factors ruled out:

1. End organ failure
2. Concomitant illness
3. Drug such as antidepressants, antihistamines, diuretics, vasodilators, sympathetic blockers, vagolytics.

The tests for parasympathetic control

Heart rate variability (HRV) in response to:

1. Deep breathing
2. Standing
3. Valsalva maneuver.

HRV methods are age-dependent but independent of the intrinsic heart rate. They are the standard screening methods for autonomic dysfunction. Valsalva maneuver is influenced by both parasympathetic and sympathetic activity.

The tests for sympathetic control

Blood pressure response to.

1. Standing or passive tilting
2. Sustained handgrip
3. Response to the Valsalva maneuver: In the standard Valsalva maneuver, the supine patient, connected to an ECG monitor, forcibly exhales for 15 s against a fixed resistance (40 mmHg) with an open glottis. This results in a sudden transient increase in intrathoracic and intra-abdominal pressures, with a characteristic hemodynamic response.
   a. *Phase I:* Transient rise in blood pressure and a fall in heart rate due to compression of the aorta and propulsion of blood into the peripheral circulation. Hemodynamic changes are due to mechanical factors.
   b. *Phase II:* Initial fall in blood pressure followed by the recovery of blood pressure later in the phase. The blood pressure changes are accompanied by an increase in heart rate. There is a fall in cardiac output due to decreased venous return which causes reflex tachycardia and increased peripheral resistance.
   c. *Phase III:* Drop in blood pressure and rise in heart rate when expiration stopped.
   d. *Phase IV:* Overshoot of blood pressure above the baseline value due to residual vasoconstriction in a setting of a now normal venous return and cardiac output. There is a reflex bradycardia.

The Valsalva ratio is calculated from the ECG waveform by dividing the longest R-R interval after the maneuver (in phase IV) to the shortest R-R interval during the maneuver. A Valsalva ratio < 1.2 is abnormal.

HRV (Heart Rate Variability)

1. Respiratory sinus arrhythmia (RSA) is a normal phenomenon due to vagal input to sinus node during expiration causing cardio deceleration. Autonomic neuropathy decreases the HRV (difference in between maximum and minimum heart rate during the respiratory cycle). Specialised monitors are available to accurately determine the HRV. A clinical but less accurate method is to ask the patient to breathe quietly and deeply at a rate of six breaths per minute (at this rate there is maximum variation in heart rate)
   a. Normal variability > 15 beats /min
   b. Abnormal result < 10 beats /min

   Aging is associated with decreased RSA due to decreased vagal tone and decreased beta receptor responsiveness and the criteria may not apply to patients above 60 years of age.
2. Response to standing up (30: 15 ratio): There is a rapid increase in heart rate in response to standing that is maximal at approximately the 15th beat after standing. This is followed by a relative bradycardia that is maximal at approximately the 30th beat after standing. The heart rate should increase by 10 %. 30:15 ratio is measured as the longest R-R interval during beats 20–40 divided by the shortest R-R interval during beats 5–25. In patients with DAN, there is only a gradual increase in heart rate.
   a. 30:15 ratio >1.04 is normal
   b. 1.01 – 1.03 is borderline
   c. < 1.01 is abnormal

Orthostatic hypotension: It is defined as a fall in blood pressure > 30 mm Hg for systolic or >10 mm Hg for diastolic blood pressure in response to postural change, from supine to standing. Normally, blood pressure is rapidly corrected by baroreflex-mediated peripheral vasoconstriction and tachycardia and the fall in SBP is < 10 mm Hg. A drop by 11–29 mm Hg is borderline and a drop > 30 mm Hg is significant.

Response to tilting: It is a more precise stimulus and may be used instead of standing. The response is similar.

Sustained hand grip: Sustained muscle contraction causes a rise in systolic and diastolic blood pressure and heart rate. Exercising muscle stimulates a reflex arc resulting in increased cardiac output and heart rate. As the peripheral vascular resistance is maintained the diastolic pressure rises by > 16 mm Hg. A response of < 10 mm Hg is considered abnormal.

Gastroparesis: The finding of retained food in the stomach after an 8–12 h fast in the absence of obstruction is diagnostic of gastroparesis.

Management of DAN: Control of blood sugar levels, ACE inhibitors and β-blockers have been tried for control of the condition.

How does autonomic neuropathy affect the perioperative course?

Autonomic neuropathy affects compensatory mechanisms of the cardiovascular system (e.g. baroreceptor reflexes) increasing risk of hemodynamic lability, particularly with change in patient position, initiation of positive pressure ventilation and institution of sympathetic blockade as in neuraxial blocks. A close monitoring is required along with meticulous maintenance of intravascular volume. Loss of HRV may be a contributory risk factor for ventricular arrhythmias and sudden death in these patients. Patients with autonomic neuropathy are likely to have silent myocardial ischemia. Respiratory arrest is also seen in patients with autonomic neuropathy.

What do you want to know about the patients' anti-diabetic treatment?

1. Which oral hypoglycemic agents
2. If insulin, type of insulin and dosing schedule.

The groups of oral hypoglycemic drugs and their perioperative implications are:

1. Sulfonylureas enhance the secretion of insulin in response to glucose and increase sensitivity to its peripheral actions.
   a. First-generation agents: For example, tolbutamide, tolazamide, chlorpropamide. Have long duration of action.
   b. Second-generation agents for example glipizide, glyburide, glimepiride, gliclazide, glibenclamide. Glibenclamide has a longer duration of action and more likely to cause hypoglycemia especially in presence of renal insufficiency.
   c. The potassium channel-blocking effect of sulfonylureas may interfere with myocardial ischemic preconditioning thereby increasing the risk of cardiac complication especially in major surgery and critical illness.
2. Meglitinides—Stimulate insulin secretion partly in a similar way to sulfonylureas but are shorter-acting and have a more rapid onset of action. e.g. repaglinide
3. Biguanides—Promote glucose utilization and reduce hepatic glucose production. Generally metformin is well tolerated and is less likely to cause hypoglycemia than sulfonylurea or insulin.

   Metformin, particularly, at high concentrations, reduces oxidative phosphorylation thereby increasing anerobic metabolism. Metformin is predominantly excreted unchanged in the urine and renal dysfunction can lead to high levels. Metformin has been associated with lactic acidosis especially in the elderly, in association with renal failure and perioperatively in association with hypotension and hypovolemia. There is no consensus on its stoppage in the perioperative period. However, it may be prudent to withdraw metformin 1–2 days prior when intraoperative tissue hypoperfusion or hypoxia is expected. In patients with low GFR at risk of contrast induced nephropathy e.g. diabetics, it is advisable to withdraw metformin on the procedural day. And restart after 48 hours after confirming stable renal function.
4. Thiazolidinediones—Reduce peripheral insulin resistance and may reduce hepatic glucose production e.g. roziglitazone, pioglitazone. There are some concerns regarding increased cardiac events in patients taking rosiglitazone.
5. α-glucosidase inhibitors—Suppress the breakdown of complex carbohydrates in the gut, delaying the postprandial rise in blood glucose concentration e.g. acarbose.

Which are the various types of insulins available? What are the problems with insulin therapy?

Insulin preparations: Extracted from beef (now rarely used) or pork pancreas—contained animal antigens which caused immunologic reactions and antibody formation. Biosynthetic human-sequence insulins are synthesized using recombinant DNA technology from *Escherichia coli*. The three types of insulin preparation are classified according to their length of action.

1. *Soluble insulins:* Have a rapid onset and short duration of action (depending upon the route of administration). When injected subcutaneously the duration of action is from 30 min up to 8 h with a peak at 2–4 h. When injected IV, it has a half-life of approximately 5 min. Insulin lispro and insulin aspart, which are recombinant human insulin analogues, have a faster and shorter action profile.
2. *Intermediate acting preparations:* Made with suspensions of insulin with either protamine ('isophane insulin') or

zinc ('crystalline insulin') salts or both together. They are administered in combination with soluble insulin to obtain rapid onset together with a long duration of action. Isophanes produce peak plasma insulin levels at variable intervals between 4 and 8 h after injection, and their glucose-lowering action wears off rapidly after 10 to 12 h.

3. *Longer-acting insulin preparations:* Peak effect occurring after 10–12 hours and its duration of action may be 16–18 hours. They are not suitable for IV use. Long-acting insulins may act for up to 36 h for animal and 24 h for human-sequence preparations.

Insulin supplementation: Physiologically, insulin secretion varies in response to feeding or starvation. A basal insulin level is maintained during the fasting state. These conditions are almost impossible to replicate with exogenous insulin administration. Consequently, insulin administered subcutaneously, even if timed optimally, will have inadequate peak concentrations and inadequate basal levels causing periods of hypoinsulinemia and hyperglycemia. Also, its entry into the systemic circulation rather than into the portal system affects its metabolic actions. Absorption of insulin injected subcutaneously is slow and unpredictable and IV administration is advised in the perioperative period.

What are the implications of surgery in a diabetic patient?

The perioperative problems posed by surgery in the diabetic patient are:

1. Stress response to surgery (magnitude depends on site of surgery, tissue injury, etc).
   a. The secretion of catecholamines, cortisol and growth hormone oppose glucose homeostasis as these hormones have 'anti-insulin' and hyperglycemic effects. Glycogenolysis and gluconeogenesis are stimulated and peripheral glucose uptake is decreased causing hyperglycemia and ketosis.
   b. Rise in blood sugar during surgery is related to magnitude of inflammation. Excessive release of inflammatory cytokines, such as tumor necrosis factor-interleukin-1, and interleukin-6 and immobility itself is associated with reduced skeletal muscle insulin sensitivity leading to hyperglycemia.
   c. In fasting patients undergoing elective intraabdominal procedures, blood glucose levels typically increase to between 126 and 180 mg/dL. During cardiac surgery the disturbance of glucose homeostasis is greater, with blood glucose values rising above 270 mg/dL in subjects without diabetes and above 360 mg/dL in subjects with diabetes. Stress may precipitate diabetic crisis (ketoacidosis or hyperglycemic hyperosmolar coma).
2. Interruption of oral intake, which may be further prolonged after gastrointestinal surgery. Starvation can lead to ketosis. Perioperative insulin administration is required to control blood sugar. The patient can be reverted to his presurgery diabetes management only after he resumes his normal oral intake.
3. Altered consciousness in perioperative period may mask the symptoms and signs of hypoglycemia.
4. Circulatory disturbance associated with anesthesia and surgery may alter the absorption of subcutaneous insulin. So intravenous administration is preferred perioperatively.
5. Hyperglycemia is associated with increased risk of complications such as wound infection and poor neurological outcomes in susceptible patients.

   The goals for the diabetic patient are minimal metabolic disruption, avoidance of untoward events, and return to stable glycemic control as soon as possible.

What are the adverse effects of hyperglycemia?

Acute consequences of untreated, or inadequately treated, diabetes mellitus include:

- Dehydration resulting from the osmotic diuretic effect of glycosuria
- Acidemia because of accumulation of ketoacids and/or lactic acid
- Electrolyte imbalance—potassium and magnesium imbalance—which can increase arrhythmia risk.
- Fatigue, weight loss and muscle wasting because of lipolysis and proteolysis in absolute insulin deficiency
- Hyperglycemia increases risk of postoperative infection and wound infection.

How will you investigate a diabetic patient for surgery?

Hemoglobin: Anemia is present with renal dysfunction, also as a baseline investigation to guide blood transfusion intraoperatively.

CBC: Look for infection (leukocytosis).

Urine routine for microalbuminuria: Alerts clinician to the presence of diabetic nephropathy and possibly other microvascular complications.

Serum creatinine: To detect renal dysfunction.

Fasting and postprandial blood sugar: To assess quality of control.

Glycosylated Hb: HbA1c of less than 7% implies good blood sugar control over the preceeding 8–12 weeks (life span of RBC is 120 days)

Serum electrolytes: To detect abnormalities in patients with history of vomiting, diarrhea, poor oral intake or tube feeding, intestinal obstruction, etc. Also in patients on insulin, ACE inhibitors, diuretics and renal dysfunction serum potassium levels can be abnormal.

ECG: To detect asymptomatic myocardial ischemia. Diabetic men are more than four times as likely, and women five times more likely to have coronary heart disease than non-diabetics.

2D echocardiography should be done (if facility available and time permits for an urgent case in a sick patient) for patients with longstanding diabetes and in those with cardiovascular symptoms.

(Noninvasive or invasive cardiac testing may be warranted preoperatively, particularly for the diabetic undergoing major noncardiac and vascular surgery)

X-ray chest: Tuberculosis is common in diabetics due to impaired immunity. Patients with abdominal distension may have pneumonia secondary to basal atelectasis or aspiration.

Morning of surgery investigations: Serum electrolytes, FBS, urine ketones.

Emergency surgery: Acute illness can cause metabolic decompensation. These patients need full clinical and biochemical assessment.

What are the principles of anesthesia management in a diabetic patient?

Timing: Diabetic patients should be placed first on the operating list. This shortens their preoperative fast and the risk of hypoglycemia and ketosis.

Fasting: Delayed gastric emptying due to diabetic autonomic neuropathy is found in up to 50% of type 1 DM patients. Undiagnosed gastroparesis may prolong retention of food in the stomach thereby increasing the risk of regurgitation and aspiration. Some studies have shown beneficial effects of preoperative oral erythromycin on gastric motility. However, other motility stimulants have not been shown to be effective. A 12-hour fast may be beneficial in diabetic patients before surgery.

IV fluids: Ringer's lactate—Lactate undergoes gluconeogenesis in the liver and may complicate blood sugar control when given in large volumes. Normal saline infusions in large volumes increase risk of hyperchloremic acidosis. Thus, there is no ideal solution and either solution may be used judiciously.

Monitoring: Frequent, rapid and accurate blood glucose measurement is essential in the anesthetized patient as the requirements for glucose and insulin in this period are unpredictable and hypoglycemia may go undetected.

Standard monitoring: ECG, SpO_2, BP, $ETCO_2$ and temperature should be instituted. Advanced monitoring is guided by presence of comorbidities.

Glycosylated hemoglobin ($HbAc_1$) measurement has no value in the intra-or postoperative period but is a valuable guide to long-term glycemic control during preoperative evaluation.

Sugar control: 'Permissive hyperglycemia' is unacceptable in current practice with availability of more accurate, rapid and easy-to-use glucose monitors. Postoperative wound healing and infection may be influenced by the adequacy of perioperative glycemic control.

Glucose supplementation: Diabetic patients receiving longer-acting insulin are at risk of hypoglycemia if glucose is not supplemented.

1. Required to safeguard against inadvertent hypoglycemia and excessive catabolism and starvation ketosis
2. Perioperative administration of glucose enhances postoperative glucose utilization rates.

Insulin supplementation:

1. Essential in patients with absolute insulin deficiency and infection to prevent lipolysis and proteolysis with resultant ketosis.
2. Some of the metabolic effects of the suppression of insulin secretion are reversed by intraoperative insulin infusion.

Anesthesia:

Induction: Choice of agent for general anesthesia depends on severity of systemic diseases, such as coronary artery disease, nephropathy, hypertension and autonomic neuropathy. Epidural analgesia may be instituted after due consideration to autonomic neuropathy, IHD and peripheral neuropathy. It should be avoided in sick patients with sepsis. Epidural analgesia may help to attenuate neurohormonal response to stress and avoid systemic analgesics like NSAIDs and opioids which may have serious side effects in a diabetic patient. A rapid sequence induction should be performed for patients with GI symptoms. Awake fiberoptic bronchoscopy would be preferred technique for an anticipated difficult airway.

A careful induction with Etomidate or high dose Fentanyl (4–5 mcg/kg) with midazolam and/or thiopentone should be performed (exaggerated hypotension due to autonomic

neuropathy is common). Succinyl choline should be avoided in patients with extensive peripheral neuropathy due to risk of increased potassium release. Atracurium and mivacurium are preferred in presence of renal dysfunction. Rocuronium may be used in rapid sequence induction.

Maintenance of anesthesia is with isoflurane or sevoflurane in an air oxygen mixture. Nitrous oxide may be used, though it should be avoided in an intestinal obstruction case due to bowel distension. Patients with perforative peritonitis and sepsis should be ventilated postoperatively to optimize their oxygen delivery. Bowel distension and a tense abdominal closure also make postoperative ventilation desirable. Airway pressures after abdominal closure should be observed to decide about postoperative ventilation. Less severe cases can be 'reversed' and extubated at the end of surgery. The patient should have adequate recovery of airway reflexes prior to extubation.

Do anesthetic drugs affect blood sugar control?

Induction agents: Ketamine may cause significant hyperglycemia. Etomidate blocks adrenal steroidogenesis and hence cortisol synthesis and decreases the hyperglycemic response to surgery by approximately 18 mg% in non-diabetic subjects. The effects on diabetic patients have not been established. The effect of propofol on insulin secretion is not known. Diabetic patients show a reduced ability to clear lipids from the circulation. This should be considered in patients receiving propofol for prolonged sedation in the intensive care unit.

Halothane, enflurane, isoflurane and sevoflurane in *in vitro* studies inhibit the insulin response to glucose in a reversible and dose-dependent manner but their effect in clinical situations is not certain.

Benzodiazepines decrease the secretion of ACTH and the production of cortisol, when used in high doses. They reduce sympathetic stimulation but stimulate growth hormone secretion and result in a decrease in the glycemic response to surgery. These effects are minimal when midazolam is given in usual sedative doses.

High-dose opiate anesthetic techniques produce hemodynamic, hormonal and metabolic stability. These techniques effectively block the entire sympathetic nervous system and the hypothalamic-pituitary axis, probably by a direct effect on the hypothalamus and higher centers. However, midazolam and fentanyl may cause hyperglycemia by reducing glucose clearance.

Ganglion-blocking agents (used for hypotensive anesthesia previously) may block sympathetically mediated hepatic gluconeogenesis with resultant hypoglycemia. The use of β-blockers is associated with slower recovery from hypoglycemia.

Describe hyperglycemic emergencies:

- Diabetic ketoacidosis (DKA)
- Hyperosmolar hyperglycemic state (HHS).

The underlying mechanism for both disorders is a reduction in the action of circulating insulin along with a rise of counter regulatory hormones, such as glucagon, catecholamines, cortisol, and growth hormone. DKA is associated with absolute deficiency of insulin whereas HHS is seen with lesser degree of insulin deficiency.

Diabetic ketoacidosis: Seen in type 1 diabetics and patients with type 2 diabetes during the catabolic stress of acute illness such as trauma, surgery, or infection. DKA is sometimes the presenting feature of type 1 diabetes. DKA is sometimes seen due to discontinuation of antidiabetic treatment or with drugs that affect carbohydrate metabolism, such as corticosteroids, thiazides, sympathomimetic agents and some antipsychotics agents. DKA is a proinflammatory state producing reactive oxygen species, proinflammatory cytokines, plasminogen activator inhibitor-1 and C-reactive protein.

The clinical features are:

1. Symptoms of hyperglycemia like polyuria and polydipsia.
2. Symptoms of acidosis and dehydration such as respiratory distress, drowsiness, coma and abdominal pain.
3. Other symptoms like vomiting, malaise, cramps and signs of infection.
4. Signs include tachycardia, dehydration, acidotic breathing, depressed consciousness and abdominal tenderness**.**

The classical triad of DKA is hyperglycemia, ketonemia, and metabolic acidosis.

Mechanism

- Increased gluconeogenesis and hepatic and renal glucose production along with impaired glucose utilization in peripheral tissues cause hyperglycemia and hyperosmolality. The increased counter regulatory hormones lead to lipolysis and hepatic fatty acid oxidation to ketone bodies (hydroxybutyrate, acetone and acetoacetatic acid) with resulting metabolic acidosis.
- Osmotic diuresis leads to water loss and electrolyte disturbance.

Hyperosmolar hyperglycemic state: HHS is seen in type 2 diabetics, more commonly in elderly people with coexisting diseases. HHS may be caused by plasma insulin concentrations that are inadequate for glucose utilization but are adequate to prevent lipolysis and ketogenesis. The hyperglycemia and volume deficit is more severe than DKA.

Clinical features are:

- Slow onset over days to weeks
- Symptoms of hyperglycemia
- Signs of dehydration
- Signs of hyperviscocity and thrombosis like delirium, coma, seizures, sensory and motor deficits
- Abdominal pain is unusual. Vomiting may be present.

Investigations for diagnosis in both DKA and HHS include CBC, ABG, serum glucose, urine analysis, electrolytes, renal and liver biochemistry, ECG, X-ray chest and appropriate cultures.

Diagnostic criteria and typical total body deficits of water and electrolytes in DKA and HHS

Table 34.1 Diagnostic criteria for DKA and HHS

Diagnostic criteria	Mild DKA	Moderate DKA	Severe DKA	HHS
Plasma glucose (mg%)	> 250	> 250	> 250	> 600
Arterial pH	7.25–7.35	7.0 to < 7.25	< 7.0	> 7.3
Serum bicarbonate (mEq/L)	15–18	10 to < 15	<10	>15
Urine ketone	+	+	+	small
Serum ketone	+	+	+	small
Serum osmolality	Variable	Variable	Variable	> 320 mosm/kg
Anion gap	> 10	> 12	> 12	< 12
Anion gap	> 10	> 12	> 12	< 12
Mental status	Alert	Alert/drowsy	Stupor/coma	Stupor/coma
Water deficit (ml/kg)	100			100–200
Na deficit (meq/kg)	7–10			5–13
Cl deficit (meq/kg)	3–5			5–15
K deficit (meq/kg)	3–5			4–6
PO_4 deficit (meq/kg)	5–7			3–7
Mg deficit (meq/kg)	1–2			1–2
Ca deficit (meq/kg)	1–2			1–2

How would you treat DKA and HHS?

Principles of treatment of DKA and HHS:

1. Correction of dehydration, hyperglycemia, and electrolyte imbalances
2. Identification of comorbid precipitating events
3. Frequent patient monitoring.

Fluid therapy: Aim: Expansion of the intravascular and extra vascular volume and restoration of renal perfusion.

15–20 mL/kg body weight of 0.9% NaCl is infused in the first hour. Further fluid resuscitation is guided by assessment of fluid status, urine output and electrolyte levels. 0.45 % or 0.9 % NaCl may be used. Total fluid deficit is corrected over 24 hours with constant monitoring to avoid fluid overload.

Patients in hemodynamic shock need fluid and vasopressor therapy with advanced hemodynamic monitoring. Patients with preexisting cardiac dysfunction need cautious fluid boluses.

Insulin therapy: IV bolus of regular insulin 0.1 unit/kg body wt, followed by a continuous infusion at a dose of 0.1 unit/kg/hr should be administered with the aim to decrease plasma glucose concentration at a rate of 50–75 mg % per hour. If the drop is not adequate, insulin dose can be doubled to achieve a steady drop. When blood sugar drops to below 200 mg% in DKA and below 300 mg% in HHS, the insulin rate is reduced and dextrose infusion can be added. Subcutaneous rapid-acting insulin has been used successfully in uncomplicated DKA. An initial injection of 0.2 units/kg is given followed by 0.1 unit/kg every hour or an initial dose of 0.3 units/kg followed by 0.2 units/kg every 2 hours until blood glucose is < 250 mg/dL. After this the insulin dose is halved and administered every 1 or 2 hourly until the resolution of DKA.

Potassium therapy: For serum potassium < 3.3 mEq/L, supplement potassium at 20–30 mEq/h before starting insulin to avoid arrythmias, cardiac arrest or respiratory muscle weakness. Potassium supplementation is avoided for levels > 5.3 mEq/L or in presence of reduced urine output

Bicarbonate: Use of bicarbonate in DKA is controversial with no universally followed threshold for administration. Bicarbonate is usually reserved for severe metabolic acidosis with pH < 7.0. 50 or100 mmoL bicarbonate can be given depending on the severity of acidosis, however bicarbonates should not be used in presence of hypokalemia till serum potassium levels are corrected. Close monitoring of blood potassium levels and aggressive correction is advised at serum potassium levels < 3.3 mEq/L.

Phosphate: Supplementation is advised only at serum phosphate concentration < 1.0 mg/dL or in the presence of anemia, cardiac dysfunction or respiratory muscle weakness. If needed, 20 –30 mEq/L potassium phosphate can be given. Routine use of phosphate is not encouraged.

Ketone bodies: The commonly used nitroprusside method measures only acetoacetic acid and acetone and not beta hydroxybuterate which is the strongest and most prevalent acid in DKA. Also during treatment, β-hydroxybuterate gets converted to acetoacetic acid making it appear that the ketosis has worsened if the nitroprusside method is used. Therefore, hydroxybutyric acid monitoring is preferred and

new bedside monitors are now available.

Monitoring: 2–4 hourly estimation of serum electrolytes, glucose, blood urea nitrogen, creatinine, osmolality and acid base status

Criteria for resolution of DKA include glucose < 200 mg/dL, serum bicarbonate > 18 mEq/L, and pH > 7.3.

Patient can be started on a new or his previous subcutaneous regime after DKA resolves.

In HHS, serum glucose is maintained between 250 and 300 mg % till the osmolality is < 315 mosm/L and the patient is mentally alert.

Complications

- Cerebral edema
  - Most dangerous complication with high mortality rate
  - Could be related to rapid drop in plasma osmolality or to increased cerebral perfusion
- Latrogenic hypoglycemia and hypokalemia
- Pulmonary edema and hypoxemia.

What are the specific things that you will consider in a diabetic patient who develops intestinal obstruction for emergency laparotomy?

1. Intestinal obstruction
   a. Fluid and electrolyte status
      i. Signs of hypovolemia/dehydration – tachycardia, low volume pulse, Low JVP, hypotension, dry tongue, low urine output—these patients will need resuscitation prior to anesthesia.
      ii. Has the patient been vomiting/have high nasogastric aspirate/severe abdominal distension? –He may have electrolyte and acid base abnormality –Evaluate ABG and serum electrolytes.
   b. Signs of peritonitis/sepsis—fever, abdominal guarding
      i. Prompt broad spectrum antibiotics
      ii. Resuscitation based on 'early goal directed therapy'.
2. Diabetes related concerns
   a. How long has the patient been unable to take orally?
   b. Has the patient been taking OHA/insulin? Dose and time.

Patient is at risk of developing DKA especially if he has been unable to take insulin or has infection, starvation ketosis if he has been not been feeding or hypoglycemia if he is on long acting antidiabetic medications.

Measurement of blood sugar, urine ketones and in some cases ABG (for metabolic acidosis) is indicated.

The surgeon has booked a diabetic patient for an emergency laparotomy for perforative peritonitis. On questioning he says the patient is stable, has a normal blood pressure and he wants to start immediately before the patient starts worsening. You find the patient has a PR 120/min, BP 100/70 mm Hg and is drowsy. What would your management plan be?

Drowsiness in this patient is most probably metabolic in origin with the following differential diagnosis:

1. Sepsis
2. DKA
3. Hypoglycemia
4. Electrolyte (sodium) imbalance
5. Hypoperfusion due to cardiac dysfunction or hypovolemia
6. Hypoxemia
7. Uremia
8. HHS
9. Sedative medications

Tachycardia is likely to be due to hypovolemia and low stroke volume (despite a 'normal' blood pressure). This patient should be optimized prior to surgery. The patient may have a cardiovascular collapse if anesthesia is induced in this condition. Optimization will include fluid resuscitation, antibiotic administration, insulin/glucose administration and/or vasoactive drugs according to the clinical condition. Invasive monitoring—intra-arterial BP and CVP—should be instituted.

Investigations for diagnosis and monitoring:

1. Immediate bedside blood sugar estimation
2. Urine ketones
3. Oxygen saturation monitoring
4. ABG with serum electrolytes and lactates
5. Detailed history (fever, etc) and drug chart examination
6. Fluid balance (intake/output) since admission
7. Serum biochemistry.

Acute abdominal pain in a diabetic patient should be evaluated in detail. Diabetes related complications such as DKA, gastroparesis and intractable vomiting can lead to a false diagnosis of acute surgical emergency. Diabetic pseudo-tabes syndrome is a rare syndrome characterized by sharp neuropathic pain along thoracolumbar dermatomes which can be confused with visceral disorders. These patients have pupillary and gait abnormalities from associated cranial and peripheral neuropathy.

How will you optimize this patient?

1. Fluid resuscitation
   a. Fluid administration should be started immediately in hypovolemic patients through wide bore canulae. Normal saline/Ringer's lactate in a volume of 5–20 mL/kg may be given depending on clinical assessment of fluid status. (In patients with cardiac dysfunction, fluid boluses should be given cautiously)
   b. In sick patients, a central line should be inserted to guide fluid therapy and to measure central venous oxygen saturation ($ScVO_2$). Postoperative $ScVO_2$ < 65% has been shown to be associated with higher complication rate. There is evidence that perioperative goal directed therapy ($ScVO_2$ endpoint of 70–75%) reduces complications in high-risk patients undergoing major surgery. If facilities available one can consider using advanced hemodynamic monitoring like pulse contour cardiac output or esophageal Doppler monitoring.
2. Continuous intra-arterial BP monitoring, with vasopressors and inotropes to optimize cardiac output can be initiated in some patients.
3. Appropriate broad spectrum antibiotics should be administered before incision.
4. Use oxygen supplementation to increase oxygen delivery. Intubation and mechanical ventilation may be required in the severely decompensated patients to reduce work of breathing and improve oxygen delivery.
5. IV insulin infusion should be started to control blood sugars. 5% dextrose in normal saline should be given as maintenance fluid at the rate of 2 mL/kg/h, apart from resuscitation fluids.

Adsorption of insulin on to the surface of syringes, IV fluid bags and IV infusion sets is an unavoidable problem. Adding albumin or a few milliliters of patients blood to the infusate was previously practiced to reduce this effect (the latter practice is not recommended due to infective risk). Flushing around 50 mL of solution is thought to be effective. In solutions with a concentration of insulin of > 10 U/L, the effect is minimal. More consistent delivery can be achieved with more concentrated solutions of lower volume administered from a syringe.

How will you manage perioperative blood sugar control?

Aims of perioperative management of diabetes are:

1. Reducing mortality and morbidity
2. Avoiding hyperglycemia
3. Avoiding ketosis
4. Avoiding hypoglycemia.

The upper limit of acceptable blood sugar level is controversial. Most problems are noted above blood sugar levels of 180 mg% but there might be advantages of tighter sugar control in some patient groups. Traditionally, blood sugars were maintained in the higher range due to the fear of hypoglycemia. Now, with increasing evidence of the risks of hyperglycemia as well as easily available bedside glucose tests, permissive hyperglycemia is no longer acceptable. Regular blood sugar measurement is extremely important to prevent catastrophic consequences of extremes of glycemia. Blood sugar should be monitored:

1. Prior to induction of anesthesia
2. At hourly intervals when insulin infusions are used intraoperatively.

The perioperative management depends on:

1. Whether patient is on diet control, OHA or insulin
2. Major surgery (requiring interruption of oral intake e.g. bowel surgery) or minor surgery.

Patients managed with diet alone: People whose diabetes is well-controlled with diet alone require no special preoperative intervention for diabetes. Fasting blood glucose should be measured on the morning of surgery, and intraoperatively if the surgical procedure lasts for more than 1 hour. If the surgery is minor, no specific therapy is required. If the surgery is major or if diabetes is poorly controlled (blood glucose >200 mg/dL), an intravenous infusion of insulin and dextrose should be considered, and hourly intraoperative glucose monitoring is recommended.

Patients treated with oral hypoglycemic agents: Sulfonylureas should be discontinued 1 day before surgery except chlopropramide, which should be stopped 2–3 days before surgery. Though there is no consensus about routine stoppage of metformin, it should be stopped 1–2 days before surgery in sick patients, and those at risk of hypoperfusion (especially renal) and hypoxia and should only be restarted after the above have resolved.

Blood glucose should be monitored before and during surgery in all patients.

Minor surgery

- Patient's usual regime continued till night prior to surgery
- Dose omitted on morning of surgery
- Dextrose containing solutions avoided
- Blood glucose monitored and treated with dextrose insulin infusion if required
- Oral intake resumed as soon as possible postoperatively. Regular or half dose of OHA is given according to intake.
- Perioperative hyperglycemia (> 200 mg/dL) in a minor surgery can be managed with a small subcutaneous dose of short-acting insulin with frequent monitoring to avoid hypoglycemia.

For major procedures: It is advisable to change over to regular insulin in the perioperative period.

Patients on Insulin: Optimal preoperative blood sugar control for elective surgeries is fasting value of 80–120 mg% and bedtime values of 100–140 mg%. Patients on long-acting insulin should be switched to a combination of short and intermediate-acting preparations 1–2 days before elective surgery. Close perioperative blood glucose monitoring is crucial to avoid extremes of glycemia. Intravenous GIK (glucose, insulin, potassium) infusion or separate insulin and dextrose infusions should be started before surgery. Blood glucose levels should be monitored hourly intraoperatively and immediately after surgery. There should be a 1-h overlap between stopping intravenous insulin and reinstituting subcutaneous insulin.

Minor surgery: GIK infusions are usually adequate in minor surgeries. The infusion should be stopped and usual insulin treatment resumed once oral intake is established.

Major surgery: Insulin-treated patients undergoing major elective surgery should preferably be admitted 2–3 days before surgery particularly if blood sugar control is suboptimal.

Standard GIK solution: 500 ml 10% dextrose solution + 15 units short-acting insulin + 10 mmol KCl.

Infuse over 5 hours (100 mL/h)
5 % dextrose solutions are used by some instead of 10% D.
Short acting insulin is added at 0.32 units/gram of dextrose.

Potassium is avoided in patients with renal dysfunction and those with hyperkalemia.

Advantages of GIK solutions

1. Inherently safe. With separate glucose and insulin infusions, one may be stopped inadvertently with potentially disastrous consequences such as severe hypoglycemia or hyperglycemia.
2. Do not need sophisticated equipment like infusion pumps.

Disadvantages

1. Insulin depends on receptor sensitivity for its action in vivo. Therefore adjustments have to be made to insulin dose to be added to 'neutralize' the dextrose which may involve making fresh solutions.
2. Fine control of sugar control more difficult. Separate infusions are easier to titrate to blood sugar levels.

Separate infusions

The obligate glucose requirement to provide for cells which can only use glucose as substrate such as neurons and RBCs is 2 mg /kg/minute. This can be provided by giving 5% or 10% solution. Dextrose solutions (5%) are given at the rate of 100 –125 mL/h. The initial insulin infusion rate can be estimated as between one-half and three-fourths of the patient's calculated hourly insulin requirement (patients total daily dose divided by 24). Insulin requirements are higher in septic, obese, critically ill patients and in those receiving steroids or undergoing cardiopulmonary bypass surgery.

How will you control blood sugars intraoperatively?

IV infusion of regular insulin is used for intraoperative blood sugar control. Blood sugar estimations are recommended hourly intraoperatively. In an awake patient, frequency of monitoring can be reduced to 2 to 4 hourly estimations when a stable dose and blood sugar level is achieved. Literature search yields various regimes practiced in different institutes. Be familiar with the regimen used in your institute.

The Vellore regimen for blood sugar control is (Table 34.2).

Table 34.2 Vellore regimen

Blood glucose	Treatment
(mg/dL)	(5% D—5% dextrose in water)
< 70	Stop insulin if on infusion. Rapid infusion of 100 mL of 5% D, measure blood glucose after 15 min
71–100	Stop insulin, infuse 5% D at 100 mL/h
101–150	1 U of insulin in 100 mL of 5%D/h
151–200	2 U of insulin in 100 mL of 5%D/h
201–250	3 U of insulin in 100 mL of 5%D/h
251–300	4 U of insulin in 100 mL of 5%D/h
>300	1 U of insulin for every 50 mg more than
	100 mg/dL in 100 mL of normal saline/h

A burette set is used to deliver the hourly dose of insulin in the 5% D/NS. This allows for hourly delivery of titrated dose of insulin without infusion pumps.

Another method of calculating hourly infusion rates of insulin is:

Insulin rate = Blood sugar in mg% divided by 150 in patients except those given below.

Insulin rate = Blood sugar in mg% divided by 100 in patients on steroids, with infection, BMI > 35.

The disadvantage of the above fixed dose regimen based on spot blood sugar levels is that they do not account for variations in insulin receptor sensitivity in individual patients and can lead to fluctuations in blood sugar levels. Fine tuning of insulin infusions rate based on dose response is better suited to achieve stable blood sugar concentrations.

Resumption of preoperative regime: May be delayed by factors such as a continuation of the stress response to surgery, persistent alterations in nutritional intake and the administration of drugs which may either reduce residual endogenous insulin secretion (in type 2 DM) or reduce insulin sensitivity. For minor surgeries under local anesthesia e.g. cataract surgery, it is acceptable to allow normal oral intake and antidiabetic therapy throughout the perioperative period. If general anesthesia becomes necessary, the surgery is scheduled at a later date.

In pancreatic diabetes, hypoglycemia is more common and shows slow recovery due to low glucagons levels. These patients need more frequent blood sugar monitoring.

What is tight sugar control?

'Tight sugar control' refers to maintenance of blood sugar within a narrow normal range, typically between 70–110 mg%. However, hypoglycemia is a significant risk in this approach. Also the threshold above which complications increase is not known. Therefore the exact limits within which to maintain blood sugar levels for the best outcomes are not well-described in literature. In some groups of patients there is better evidence on the beneficial effects of tighter blood sugar control such as cardiac surgery, neuronal injury (e.g. traumatic brain injury, acute stroke), burns, transplant surgery, critically ill patients and pregnant women.

Adverse neurologic outcome related to hyperglycemia is due to intracellular acidosis and a worsening of the hypoxic neuronal edema. Chemotaxis, phagocytosis, polymorphoneutrophil (PMN) adherence and apoptosis have been shown to be impaired by diabetes and hyperglycemia, increasing the risk of infection. Even glucose levels of 200 mg% may impair these defense mechanisms. Studies have generally shown benefit when glucose targets are in the broad range of 80–180 mg%.

In cardiac surgery, infection risk is higher in patients with poor postoperative blood sugar control, diabetic and non-diabetic. The data is less convincing for intraoperative and long-term glucose control. There is also evidence that overzealous intraoperative glucose control may be neurologically detrimental.

Which factors influence the use of regional techniques in diabetic patients?

Advantages of regional anesthesia:

- Compared to general anesthesia, epidural anesthesia has minimal effect on glucose metabolism. Circulating glucose, noradrenaline, and cortisol concentrations are not raised in patients undergoing epidural anesthesia for pelvic or lower limb surgery. Insulin response to a bolus of glucose is preserved in low spinal anesthesia. (At high spinal levels, insulin secretions may be impaired due to sympathetic blockade). However, there is no hard evidence that choice of regional anesthesia and/or general anesthesia confers any benefit in terms of mortality and major complications.
- Patients are awake, so hypoglycemia and hyperglycemic coma is detected early
- Allows early return to normal oral intake and resumption of regular treatment regime
- Regional anesthesia also avoids the potential problems of tracheal intubation
- Excellent analgesia. Spares use of systemic analgesics such as NSAIDs which can be deleterious in patients with diabetic nephropathy.

Disadvantages of regional anesthesia:

- Higher risk of nerve damage. Also ischemic nerve injuries are more likely with use of epinephrine in regional anesthesia in diabetics.
- Epidural anesthesia is less effective in reducing stress response in upper abdominal or thoracic surgery due to persistence of vagal afferent input.
- May carry higher risks in the diabetic patient with autonomic neuropathy. Severe hypotension may occur due to compromised cardiovascular compensatory mechanisms with deleterious consequences in a patient with coexisting coronary artery, cerebrovascular or renovascular insufficiency.
- The risks of infection and vascular damage may be increased with the use of regional techniques in diabetic patient.
- Epidural abscesses occur more commonly following spinal and epidural anesthesia in diabetic patients.
- Diabetic peripheral neuropathy presenting after regional anesthesia can be confused with an anesthetic complication of nerve block and can pose medicolegal problems. (A detailed preoperative neurological examination, meticulous documentation of preexisting neurological deficits and patient counseling is therefore essential.)
  (Note: Local anesthetic requirements may be lower in diabetic patients)

How does open heart surgery influence blood sugar control?

Insulin requirements are increased with cardiopulmonary bypass. The reason for the high insulin requirements in diabetics is due to insulin resistance caused by hypothermia, infusion of dead space of the bypass machine with glucose solution, and the hyperglycemic effects of adrenergic drugs.

Hyperglycemic hyperosmolar coma is known in patients undergoing cardiac surgery and carries high mortality.

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

What is gestational diabetes?

Gestational diabetes mellitus (GDM) is defined as any presence of glucose intolerance with onset or first recognition during pregnancy.

Explain pathophysiology of GDM

The physiological changes of pregnancy are aimed to provide adequate nutrients to the growing fetus. In early pregnancy, maternal estrogen and progesterone increase and promote pancreatic ß-cell hyperplasia and increased insulin release. Increases in peripheral glucose utilization and glycogen storage with a concomitant reduction in hepatic glucose production result in lower maternal fasting glucose levels. As pregnancy progresses, increased levels of human chorionic somatomammotropin, cortisol, prolactin, progesterone, and estrogen lead to insulin resistance in peripheral tissues. Cortisol has the highest diabetogenic potency and has peak effect at 26 weeks gestation. Progesterone also has relatively strong anti-insulin properties that peak at 32 weeks gestation. So tests for diagnosis are focused at these stages of pregnancy.

The mechanism of insulin resistance is likely a postreceptor defect. The pancreas releases 1.5–2.5 times more insulin in order to respond to insulin resistance. GDM results when there is delayed or insufficient insulin secretion in the presence of increasing peripheral resistance

Independent risk factors for gestational diabetes:

- Body mass index above 30 kg/m^2
- Previous macrosomic baby weighing 4.5 kg or above
- Previous gestational diabetes
- Family history of diabetes
- Ethnicity—South Asian, Black Caribbean, Middle Eastern.

What are the diagnostic criteria for GDM?

American Diabetes Association considers the following to be abnormal during the 75 or 100 g of oral glucose tolerance test

- Fasting blood glucose level ≥95 mg/dL
- 1 hour blood glucose level ≥180 mg/dL
- 2 hour blood glucose level ≥155 mg/dL
- 3 hour blood glucose level ≥140 mg/dL.

The 2-hour 75 g oral glucose tolerance test (OGTT) should be used to test for gestational diabetes. Women who have had gestational diabetes in a previous pregnancy should be investigated at 16–18 weeks and then at 28 weeks. Women with other risk factors for gestational diabetes should be investigated at 24–28 weeks.

How would you classify GDM?

White classification, named after Priscilla White who pioneered research on the effect of diabetes types on perinatal outcome, is widely used to assess maternal and fetal risk.

- Gestational diabetes (diabetes which began during pregnancy)
- Pregestational diabetes (Diabetes that existed prior to pregnancy).

There are 2 subtypes of gestational diabetes:

- Type A1: Abnormal oral glucose tolerance test (OGTT) but normal blood glucose levels during fasting and 2 hours after meals; diet modification is sufficient to control glucose levels.
- Type A2: Abnormal OGTT compounded by abnormal glucose levels during fasting and/or after meals; additional therapy with insulin or other medications is required.

These two groups are further subdivided according to their associated risks and management.

What preconception care is needed for diabetic women?

- Good glycemic control before conception (HbA_{1c} < 10%) and during pregnancy is important to reduce the risk of miscarriage, congenital malformation, stillbirth and neonatal death. Maintaining the HbA_{1c} below 6.1% help to reduce the risk of congenital malformations.
- Metformin can be continued during pregnancy, but all other oral hypoglycemic agents should be stopped. The primary concern is the drug crossing the placental barrier causing hyperinsulinemic hypoglycemia in the fetus. There were also some concerns regarding congenital malformations. Insulin is therefore preferred. (recently there is evidence for efficacy and safety of glibenclamide and glyburide)
- Rapid-acting insulin analogues (aspart and lispro) are safe for the fetus. Among the long-acting insulins, isophane insulin (NPH insulin) is preferred.
- Angiotensin-converting enzyme inhibitors, angiotensin-II receptor antagonists and statins should be discontinued in the perigestational period.

What are the risks to mother and baby due to diabetes?

Risks to mother

- An increase in incidence of preeclampsia, polyhydramnios, and operative delivery
- Risk of developing diabetes later in life
- Women with prior GDM are at greater risk for developing hypertension, hyperlipidemia and electrocardiogram abnormalities. Mortality is also higher in these patients.

Risks to baby

- Increased risk of perinatal mortality and morbidity.
- Increased risk of being large for gestational age, which increases the likelihood of birth trauma, induction of labor and cesarean section. Shoulder dystocia and brachial plexus injury is common with macrosomia
- Risk of neonatal hypoglycemia: When the umbilical cord is clamped, the fetal blood sugar level drops due to the continuing insulin secretion by the hyperplastic beta islet cells. This can be reduced by good maternal glycemic control during labor and birth (blood sugar < 90 mg%) and early feeding of the baby.
- Neurodevelopment of the fetus may be hampered.
- Risk of the baby developing obesity and/or diabetes later in life.

Why does the fetus develop macrosomia?

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

What specific antenatal care is advised?

- Most patients in late pregnancy require relatively higher doses of insulin as they become increasingly insulin resistant during the third trimester of pregnancy.
- Women with diabetes should maintain fasting blood glucose between 70 and 110 mg% and 1-hour postprandial blood glucose below 140 mg% during pregnancy.
- Women with diabetes should be followed up for glycemic control every 1–2 weeks throughout pregnancy.
- Risks of hypoglycemia and hypoglycemia unawareness is high in pregnant patients on insulin, particularly in the first trimester and close monitoring is required.
- During pregnancy, women who are suspected of having diabetic ketoacidosis should be admitted immediately in high dependency unit/monitored unit.
- Nephrology opinion should be sought in case of high serum creatinine (>1.35 mg %) or proteinuria.

Fetal monitoring

- 2D echo of the fetal heart at 18–20 weeks
- Ultrasound monitoring of fetal growth and amniotic fluid volume every 4 weeks from 28 to 36 weeks.

What are the implications of diabetes for labor and delivery?

- Pregnant women with diabetes with a normal fetus can have either an elective vaginal birth through induction of labor or by elective cesarean section if indicated, after 38 weeks. Women with macrosomic babies may need a cesarean section.
- During labor and birth, blood glucose should be monitored hourly and maintained between 70 and 110 mg%.
- Women with type 1 diabetes may need intravenous dextrose and insulin infusion from the onset of labor to maintain blood sugar control.
- If general anesthesia is induced, blood glucose should be monitored every 30 minutes throughout the anesthetic period till the woman is fully conscious.
- In preterm labor, steroids can be given for fetal lung maturation but the insulin dose may need to be modified. Beta-mimetic drugs should be avoided for tocolysis.
- Women with diabetes should be advised to give birth in hospitals with round the clock support for advanced neonatal resuscitation.

What special care is needed for babies born to diabetic mothers?

- The major concern is hypoglycemia
- Babies should be fed a soon as possible within 30 minutes and at 2–3 hours interval thereafter. The prefeed blood sugar should be above 36 mg%. Babies with low blood sugars or clinical signs of hypoglycemia should receive tube feeds or IV dextrose.
- Blood glucose testing should be carried out at 2–4 hours after birth.
- Babies should be kept in hospital for at least 24 hours to ensure adequate feeding and absence of hypoglycemia.
- Blood tests for polycythemia, hyperbilirubinemia, hypocalcemia and hypomagnesemia should be carried out for babies with clinical signs.
- An echocardiogram is performed if clinical finding are suggestive of congenital heart disease or cardiomyopathy.
- Babies can be kept with their mothers unless there are abnormal clinical signs that warrant NICU (neonatal intensive care unit) admission.

Indications for NICU admission are:

- Hypoglycemia associated with abnormal clinical signs
- Respiratory distress
- Signs of cardiac decompensation due to congenital heart disease or cardiomyopathy
- Signs of neonatal encephalopathy
- Signs of polycythemia, likely to need partial exchange transfusion
- Need for intravenous fluids
- Need for tube feeding
- Jaundice requiring intense phototherapy and frequent monitoring of bilirubinemia
- Born before 34 weeks.

How blood sugar control is managed postpartum?

- Women with gestational diabetes should discontinue hypoglycemic treatment immediately after birth. However, they need blood sugar examination at 6 weeks and annually thereafter.
- Metformin and glibenclamide can be given to lactating women with preexisting type 2 diabetes. First generation sulfonylurea are secreted in milk and should be avoided.
- Insulin dose should be reduced and blood sugar monitored frequently to achieve optimum control.
- Women are at risk of hypoglycemia postnatally especially during breastfeeding and should be advised to have a meal or snack before or during feeds.

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35 Chronic Renal Failure and Renal Transplant

V Agarwal

A 66-year-old male hypertensive patient who is currently on atenolol 50 mg and amlodepine 5 mg once daily, with history of chronic renal failure since 5 years. His serum creatinine is 5.7 mg%, serum potassium is 4 mEq/L, and ECG shows T wave inversion in inferior leads. He is posted for cholecystectomy. How will you manage this patient?

What are the stages of chronic renal failure?

Chronic renal failure (CRF) is a decline in the glomerular filtration rate (GFR) secondary to various diseases, such as diabetes, glomerulonephritis, and polycystic kidney disease.

Patients having GFR less than 60 mL/min/1.73 m^2 for more than 3 months are defined as having chronic renal disease. The stages of CRF (National Kidney Foundation KDOQI Staging System for Chronic Kidney Disease) are as follows:

- Stage 1-Kidney damage with normal or increased GFR (more than 90 mL/min/1.73 m^2)
- Stage 2 -Mild (GFR of 60–89 mL/min/1.73 m^2)
- Stage 3 -Moderate (GFR of 30–59 mL/min/1.73 m^2)
- Stage 4 -Severe (GFR of 15–29 mL/min/1.73 m^2)
- Stage 5 -End-stage renal disease (ESRD) (GFR of < 15 mL/min/1.73 m^2) or on dialysis.

Hemodialysis or peritoneal dialysis is usually initiated when the GFR decreases to less than 15 mL/min/1.73 m^2. Patients with ESRD are dependant on renal replacement therapy (RRT) to survive. Patients with CRF eventually need to undergo renal transplantation after initiation of hemodialysis or peritoneal dialysis. Following renal transplant, the 5-year survival rate is approximately 70%, whereas it is 30% in patients undergoing dialysis without renal transplant.

What pathophysiological changes occur in a patient with CRF and how these can affect the anesthetic management?

Patients with CRF may have many comorbid conditions and factors; such as advanced age, hypertension, diabetes, lipid disorders, hyper-homocysteinemia, abnormal calcium phosphate metabolism, anemia and uremic toxins; which increases their risk of perioperative morbidity. Patients with preexisting renal disease are more prone to the development of acute renal failure during their hospitalization. A small increase in serum creatinine can increase the risk morbidity by as much as 6 times. The pathophysiological changes are discussed below.

Fluid and electrolyte derangement

Sodium: CRF may be associated with sodium retention, sodium wasting or normal sodium balance. This is further influenced by factors such as diuretic use and cardiac function. There is usually a certain degree of salt and water retention however, the extracellular fluid volume remains isotonic. Patient with CRF may have impaired concentrating mechanisms and therefore they may not be able to compensate for extrarenal fluid losses for e.g. vomiting, diarrhea or pyrexia. Thus, these losses can lead to hypovolemia and hypotension.

Potassium: Potassium secretion in the distal nephron may be affected and can lead to elevated potassium levels. However, this is dependent on the factors such as diuretic use. Certain drugs such as β-blockers, potassium sparing diuretics (spironolactone), angiotensin converting enzyme (ACE) inhibitors or angiotensin antagonists, non-steroidal anti-inflammatory agents and nephrotoxins such as aminoglycosides and cyclosporins; may cause acute hyperkalemia. Extracellular acidosis causes an exchange of intracellular potassium for extracellular hydrogen ions in an

attempt to maintain electrical neutrality. In acute acidosis the serum potassium will increase by 0.5 mEq/l for each 0.1 unit decrease in pH. Therefore, hypercarbia should be avoided during general anesthesia.

Magnesium: Magnesium clearance is affected and may lead to muscle weakness and potentiation of non-depolarizing muscle relaxants.

Acidosis: Chronic metabolic acidosis is a common feature of ESRD. The clearance of hydrogen ions is decreased because of inability to secrete protons or buffers and regenerate bicarbonate. Reduced utilization of glutamine causes decrease in 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.

Calcium, phosphate, parathormone and renal osteodystrophy: Total plasma calcium concentration is reduced in CRF. Intestinal absorption of calcium decreases because of fall in production of Vitamin D_3. There is hyperphosphatemia as its excretion is impaired. Hyperphosphatemia causes calcium phosphate to be deposited in the soft tissues further lowering plasma calcium concentration. There is secondary hyperparathyroidism as a result of hypocalcemia and hyperphosphatemia; which increases the osteoclast and osteoblast activity; causing Osteitis Fibrosa Cystica.

Hematological abnormalities: Anemia: CRF is associated with normochromic normocytic anemia. Causes of anemia are multifactorial:

- Decreased renal erythropoietin production leads to reduced stem cell transformation into erythrocytes
- Reduced red cell life span due to uremia
- Dietary deficiency of iron and folate
- Chronic upper GI tract losses and those from dialysis further compound the problem.

In CRF this anemia is compensated by (1) shift of the oxyhemoglobin dissociation curve to the right because of increase 2, 3-diphosphoglycerate production, thus increasing oxygen delivery to the tissues, (2) increased cardiac output (3) decreased viscosity increasing microcirculation. These patients may be on recombinant erythropoietin (EPO) to improve the anemia associated with CRF. Target hemoglobin of 9.5 gm% is maintained to improve exercise tolerance. EPO may aggravate hypertension and can lead to accelerated thrombosis of vascular access.

Coagulopathy: There is increased tendency to bleed in the perioperative period. Standard tests of coagulation and platelet count are usually normal. The qualitative function of platelets is impaired. The platelets have decreased adhesiveness and aggregation, probably due to impaired release of von Willebrand factor/factor VIII complex, which binds to and activates platelets. Also there is increased release of β-thromboglobulin from the platelets and vascular production of PGI_2 further contributing to coagulopathy. If perioperative bleeding occurs, platelet transfusion does not correct platelet dysfunction, however dialysis may be helpful. Cryoprecipitate/DDAVP (increases release of von Willebrand factor) may be required to improve coagulation. DDAVP (0.3 mcg/kg) is effective within 1–2 hours but has a short duration (6–8 hours); also tachyphylaxis may occur. The risks of bleeding complications should be considered when planning to use regional anesthetic techniques in CRF.

Cardiovascular abnormalities

Systemic hypertension is the most common feature except in sodium-wasting nephropathies such as polycystic kidney disease or papillary necrosis. Sodium and water retention is the most frequent cause of hypertension, and may be significantly improved by dialysis. Most of these patients are on antihypertensive drugs. Altered renin and angiotensin secretion may also contribute to hypertension.

Ischemic heart disease (IHD) is a frequent cause of mortality in patients with CRF. There is accelerated atherosclerosis secondary to impaired triglyceride clearance and lipoprotein lipase activity leading to impaired lipolysis. Hypertension may be a cause or effect of renal impairment.

Hypocalcemia and hyperphosphatemia leads to increased incidence of metastatic calcific valvular heart lesions. Patients on dialysis have a higher incidence of bacterial endocarditis. Before dialysis, hemorrhagic uremic pericarditis was often seen but is now uncommon and occurs in patients on inadequate dialysis regimen. If untreated, it may progress to pericardial tamponade. Sudden death from acute cardiac arrhythmias may occur and is due to IHD and electrolyte abnormalities.

Pulmonary abnormalities

Fluid overload, malnutrition, anemia, impaired humoral and cellular immune function and decreased surfactant production predispose patients to atelectasis and infection.

Immune function

The fistula and catheter site infections are common and wound healing is poor as a result of inhibition of cell mediated immunity and humoral defence mechanisms.

The use of EPO and hepatitis B vaccine has led to a decrease in the incidence of viral hepatitis B. Universal precautions must be taken when caring for these patients.

Gastrointestinal abnormalities

Uremia is a mucosal irritant and can lead to bleeding in the gastrointestinal tract. Patients with CRF have delayed gastric emptying leading to anorexia, nausea and vomiting. They have increased risk of aspiration. Coexisting diabetes mellitus further increases the risk of difficult intubation and aspiration as a result of autonomic gastroparesis. Rapid sequence induction technique needs to be balanced against the risks of difficult intubation. Succinylcholine may increase the plasma potassium concentration by approximately 0.5 mEq/L. Rapid sequence induction is usually done in patients who are full stomach or have symptoms of gastric reflux and have a low/normal level of serum potassium.

Neurological abnormalities

Both the central (CNS) and peripheral nervous system may be affected. CNS changes range from mild alterations in personality to asterixis, myoclonus, convulsions and encephalopathy. Peripheral neuropathy occurs later as the CRF worsens. It is initially a distal "glove and stocking" sensory loss leading to motor involvement. Dialysis and renal transplantation both improve the neuropathy. The presence of a peripheral neuropathy implies autonomic neuropathy and this should alert the anesthetist, regarding delayed gastric emptying, postural hypotension and silent myocardial ischemia. Two types of neurological disturbances are unique to patients on dialysis.

- Dialysis dementia is subacute, progressive, and potentially fatal condition. It occurs due to aluminium toxicity resulting aluminium phosphate salts or aluminium in the dialysate. The incidence is low now as aluminium is removed from the dialysate. When it occurs it is a multisystem disease that includes encephalopathy, osteomalacia, proximal myopathy and anemia. The symptoms may range from dysarthria, apraxia, personality changes, psychosis, myoclonus, to convulsions and finally dementia progressing to death within 6 months.
- Dialysis disequilibrium syndrome occurs in patients receiving hemodialysis. It is a self-limiting condition caused due to a reverse urea effect. Urea is cleared at a slower rate from the brain in comparison to that from blood. This creates an osmotic gradient from the blood to the brain leading to transient cerebral edema, leading to headache, nausea, vomiting, blurred vision, disorientation, delirium, hypertension, tremors, and convulsions.

Endocrine disturbances

- Hyperparathyroidism as mentioned above
- Reduced/impaired production of EPO leads to anemia
- In CRF patients with diabetes, the requirement for exogenous insulin decreases probably due to reduced metabolism of insulin.
- The temperature regulation is altered with a reduced basal metabolic rate predisposing to hypothermia.

To summarize the main issues which need to be addressed prior to an elective procedure/surgery in a patient with CRF are :

- Progression of renal disease
- Prevention and management of fluid, electrolyte, and acid-base imbalances before and after surgery
- Need for emergency/maintenance dialysis in the perioperative period
- Prevention of further insults (iatrogenic) for e.g. radio-contrast-induced acute renal failure
- High cardiac risk due to accelerated ischaemic heart disease. Based on the American College of Cardiology/American Heart Association (ACC/AHA 2007) guidelines on perioperative cardiovascular evaluation of non-cardiac surgery, patients with a creatinine level $\geq$ 2 mg% are considered as an intermediate risk factor thereby increasing the perioperative risk. This may warrant further cardiovascular evaluation for intermediate- or high-risk surgery.
- Presence and evaluation of other comorbid conditions.

An early nephrologic evaluation is mandatory to assess renal function and the need for renal replacement therapy perioperatively. A multidisciplinary approach (anesthetist, cardiologist, nephrologist, surgeon and physician) for perioperative management of these patients is essential for reducing cardiac and renal risks for the planned procedures.

What are the causes of renal failure?

Diabetes mellitus is the most common cause of CRF in approximately 45% of the patients, followed by glomerulonephritis (20%), pyelonephritis (5%), polycystic kidney disease (5%). Other causes are hypertension, systemic lupus erythematosus, etc.

How does ESRD affect drug disposition in the body. Can you give examples of some commonly used anesthetic drugs?

CRF may alter both the pharmacokinetics and pharmacodynamics of drugs. One must understand the effects of reduced clearance and accumulation of active metabolites along with the risks of worsening existing renal disease following drug administration. Dose adjustment is usually required if the GFR

decrease below 50 mL/min/1.73m^2. Metabolic acidosis due to accumulation of organic acids may have significant effects on drug disposition and competition for active transport process in the renal tubules.

Pharmacokinetics

- *Absorption:* Absorption of drugs may be affected by delayed gastric emptying.
- *Distribution:* The volume of distribution may be increased or decreased depending on the total body water, protein binding of drugs, time since last dialysis.
- *Protein binding:*
  - Protein binding of acidic drugs is decreased leading to increased concentration of active or free fraction of the drugs bound to albumin. (e.g. salicylates, oral anticoagulants). This is due to:
    - CRF leading to hypoalbuminemia secondary to proteinuria
    - Accumulation of organic anions such as uric acid and lactic acid compete with the protein binding site on albumin
  - Alpha1 acid glycoprotein is bound to basic drugs (e.g. opioid analgesics local anesthetics) and the levels are increased in CRF thereby decreasing the unbound drug concentration.
- *Elimination:* Drugs eliminated by the kidneys will have prolonged elimination half lives, e.g. vecuronium.

Induction agents

- **Propofol**—Its pharmacokinetics is not affected by CRF, however careful administration titrated clinical effect is suggested particularly in patients postdialysis as it can precipitate exaggerated hypotension.
- **Thiopentone**—In CRF, the volume of distribution is increased along with reduced plasma protein binding. Thus, increasing the active/free fraction of the drug requiring reduction in the dosage and rate of administration.

Inhalational agents

- **Sevoflurane**—Reacts with CO_2 absorbents to form Compound A which causes dose related nephrotoxicity in rats. The metabolism of sevoflurane results in elevated fluoride levels (peak levels 0.5 mmol/L). However, exposure to > 4 MAC hours of sevoflurane was not associated with an increased risk of renal toxicity.
- **Desflurane and isoflurane** are safer in patients with CRF and are not associated with nephrotoxicity.

Neuromuscular blockers (NMBs)

The initial dose (i.e. 3 x ED 95) required to produce neuromuscular block is larger in patients with CRF in comparison to normal patients due to increased volume of distribution. Similarly, maintenance of neuromuscular block during surgery requires reduced dose as metabolism and excretion of these drugs if affected in CRF; except for atracurium and cisatracurium. One must avoid long acting NMBs in patients with CRF particularly the drugs excreted by the kidneys; and it is essential to monitor the neuromuscular block intraoperatively.

- **Succinyl choline**—when used for rapid sequence induction, may increase the plasma potassium concentration by approximately 0.5 mEq/L, therefore should be avoided in patients with serum potassium $\geq$ 5.5 mEq/L. However it can be used safely in patients with normal serum levels.
- **Atracurium**—undergoes Hoffman's elimination and hydrolysis by tissue esterases and does not depend on the kidneys for elimination. Laudanosine, an epileptogenic, by-product of atracurium metabolism is known to accumulate in CRF and may be of concern in prolonged surgery.
- **Cisatracurium** is superior to atracurium as it causes less histamine release, is more potent and produces less laudanosine and has a shorter duration of action in comparison to atracurium.
- **Vecuronium and rocuronium**—predominantly excreted in the bile but up to 30% may undergo renal excretion. Vecuronium has an active metabolite 3-hydroxyvecuronium. CRF leads to reduction in clearance, prolonged elimination half life and duration of action.

Anticholinesterases—Neostigmine clearance is reduced and its half-life is prolonged in CRF. This may result in a prolonged parasympathomimetic activity; particularly when used in combination with atropine rather than the longer-acting glycopyrrolate.

Non opioid analgesics

Acetaminophen: The use of acetaminophen is safe and does not require dose adjustment in the perioperative period.

NSAID's are contraindicated in patients with renal failure including COX-2 inhibitors. They can cause hyperkalemia, hyponatremia and can exacerbate hypertension. They can cause bleeding in the perioperative period as a result of drug induced platelet dysfunction; in addition to qualitative dysfunction caused by uremia. They inhibit the production of renal prostaglandins PGE_2 and PGI_2, which are responsible for autoregulating renal blood flow during hypovolemia and in the presence of vasoconstrictors, and thus can precipitate acute renal failure.

Opioid analgesics

Morphine is predominantly metabolised in the liver to morphine-3-glucuronide (M3G) and about 5% to morphine-6-glucuronide (M6G). M3G antagonises the analgesic effect of morphine, causes irritability and lowers the seizure

threshold. M6G has potent analgesic properties which is responsible for delayed onset of sedation and respiratory depression. M6G get eliminated by the kidneys and so in patients in CRF, it can accumulate and the half life may prolong from 2 to 27 hours. Oral morphine undergoes extensive first pass metabolism in liver and therefore the concentration of active metabolite is greater than the equi-analgesic parental does. In patients with CRF; the dose and frequency of morphine should be reduced and patient should be monitored for delayed respiratory depression postoperatively. In patients on dialysis, a significant fraction of morphine is removed.

Fentanyl undergoes extensive hepatic metabolism with no active metabolites. Approximately 7% is excreted unchanged in the urine. However, the clearance is decreased in severe uremia.

Alfentanil clearance is not affected by renal failure and it can be administered safely.

Remifentanil undergoes metabolism by nonspecific blood and tissue esterases and it does not accumulate in patients with ESRD. Its primary metabolite does accumulate but the effect is clinically insignificant; as it has low potency and pharmacokinetic simulations suggest it does not reach clinically significant concentrations even after 24 hours infusion.

Tramadol is metabolised to O-Demethyl tramadol, an active metabolite, which is excreted by the kidneys. In addition, 30% of tramadol is excreted unchanged by the kidneys. Also tramodol may be epileptogenic in these patients as uremia further lowers the seizure threshold. Extended-release tramadol should be avoided in patients with CRF. The dosing interval of tramadol (regular release) may need to be increased to every 12 hours in patients with a creatinine clearance less than 30 mL per minute. Tramadol is removed by hemodialysis.

Meperidine (pethidine) should be avoided in patients with CRF as it is metabolized to normeperidine. Normeperidine is associated with seizures, myoclonus, and altered mental state; and depends on the renal function for elimination.

Codeine and dihydrocodeine are also best avoided as their elimination half-life is significantly prolonged, and conventional doses may result in central nervous system depression.

Local anesthetics have a shorter duration of action due to altered protein binding. Maximum doses of local anesthetics should be reduced by about 25% because of altered protein binding and a lower seizure threshold.

How will you conduct the anesthesia for this patient posted for non transplant surgery such as appendicectomy?

The main anesthetic goal is to maintain renal perfusion and prevent harm to the already compromised renal function by avoiding hypoxia, hypovolemia and hypotension.

Preoperative evaluation

- *History:* Establish the cause of renal failure and duration of treatment. Also discuss with the patient the need for dialysis postoperatively. Enquire about fluid restriction if any and daily urine output. Elicit history of comorbidities (Hypertension, diabetes, IHD, connective tissue disorders) and whether controlled and on what treatment (dose, frequency).

  Enquire about exercise tolerance, anemia, LVF, electrolyte disturbance, medications (antihypertensives, diuretics, sodium bicarbonate, vitamin D3, resonium), symptoms of gastroesophageal reflux. Seek nephrology opinion regarding need for dialysis in the postoperative period.
- *Examination:* Apart from general examination, measure the patient's blood pressure in standing and in sitting position which may suggest autonomic neuropathy. They may have flow murmur secondary to anemia and pericardial rub due to uremic pericarditis. Look for ankle or sacral edema which may indicate either right ventricular failure or hypoproteinemia or both. Patient's who are fluid overloaded may have crepitations.

- **Investigations**
  - *Full blood count:* Normochromic, normocytic anemia and infection are likely.
  - *Clotting studies:* are required if the uraemia is severe.
  - *Renal function tests* (BUN, serum creatinine, Electrolytes) are mandatory prior to surgery. Creatinine is a good measure of the glomerular filtration rate (GFR). Usually, these patients have their renal functions tested periodically and looking at the trend will help in eliciting the progression of disease. Also the creatinine clearance is regularly measured and knowledge of the same will help in dose adjustments of various medications in the postoperative period.
  - *ECG:* Look for ischemia, arrhythmia, LVH, conduction blocks or hyperkalemia.
  - *Chest radiograph:* Pleural effusions, cardiomegaly, pulmonary edema
  - *ABG* to evaluate the acid base status
  - *LFT* if a major surgery is planned; as a baseline value.

Preoperative optimization/preparation

For elective surgery, it is prudent to optimize blood pressure, serum potassium level. Avoid unnecessary blood transfusion because of anemia to avoid sensitization for future transplantation. The antihypertensive should be continued perioperatively. Metoclopramide and H_2 receptor antagonists should be administered if patient has gastroesophageal reflux.

Induction of anesthesia

- Preoxygenate
- Administer induction drugs slowly to minimize hemodynamic disturbances. If hypotension occurs despite above, vasopressors can be given titrated to mean arterial pressure.
- If rapid sequence induction is necessary (inadequately fasted, delayed gastric emptying, history of gastroesophageal reflux), suxamethonium can be used if the serum potassium is < 5.5 mEq/ L, alternatively modified rapid sequence induction can be done with rocuronium (if one does not anticipate difficult airway). If difficult airway is anticipated; inhalational induction is a safer option.
- If there is any doubt regarding airway adequacy, always intubate.
- Regional anesthesia can be administered after weighing the risks and the benefits. Concern with epidural anesthesia is platelet dysfunction; increasing the risk of epidural hematoma.

Maintenance of anesthesia

- Nitrous oxide can be administered safely as it does not affect the renal function.
- Isoflurane is the inhalational agent of choice as only 0.2% undergoes metabolism and produces low levels of fluoride ions.
- Ventilation should be controlled for long procedures. If the patient has been on conservative management for renal failure, compensatory changes in the $PaCO_2$, pH, oxy-hemoglobin dissociation curve and cardiac output would have occurred to maximize oxygen delivery to the tissues, therefore ventilation should be adjusted accordingly.
- Atracurium is preferable as it undergoes Hoffman's elimination. Vecuronium and rocuronium can be used but smaller top-up doses are required less frequently. Neuromuscular blockade should be monitored using a nerve stimulator and top-up doses administered accordingly.
- Fentanyl can be administered safely; however its half life may be prolonged particularly if used as an infusion. Morphine can be used with care as clearance is reduced leading to accumulation. If morphine is administered patient should be monitored for respiratory depression in the postoperative period. It should never be used as a continuous infusion.
- Monitoring: It is essential to monitor ECG, BP, end-tidal capnometry, pulse oximetry, temperature and neuromuscular monitoring. If large fluid shifts or blood loss is anticipated; invasive monitoring (central venous pressure and invasive arterial BP) should be established to guide fluid replacement. A urinary catheter should be passed to monitor urine output hourly and should be maintained at 0.5–1 mL/kg/hr. If the urine output is low despite good hydration and BP; mannitol should be administered as first line treatment followed by frusemide.
- IV fluids should be administered cautiously, as these patients may be fluid overloaded, and if required central venous monitoring may be done to guide volume replacement.
- Forced air warmer and fluid warmer should be used mandatorily to maintain normothermia as hypothermia (< 35°C) can potentiate action of most of the drugs thus delaying recovery from anesthesia.
- Colloids and intravenous fluids: Starches should be avoided in these patients as it can accumulate and worsen the renal impairment, gelatine is safe. Ringer's lactate can be used if serum potassium is within the normal limits. Over transfusion of blood to correct anemia should be avoided as increase in hematocrit can decrease renal perfusion and further compromise the renal function.
- NSAID's are contraindicated in these patients, paracetamol can be administered safely.

Reversal of neuromuscular block

- Neuromuscular blockade can be reversed with neostigmine. Recurarization is a potential problem that can occur in the postoperative period and the recovery staff should be warned about the same. If there are any concerns about inadequate reversal, it is safer to ventilate the patient for short-term till complete neuromuscular recovery occurs.

Postoperative care

- Oxygen should be administered. ECG, BP and respiration and pulse oximetry should be monitored closely in the postoperative period. Analgesia should be administered. Early mobilization and physiotherapy are important. Dosages of medications should administered as per creatinine clearance if known alternatively it can be estimated by the following formula:
  Cockcroft–Gault equation (eCCr = estimated creatinine clearance)

$$eCCr = \frac{(140 - Age) \times Mass\ (in\ kilograms) \times [0.85\ if\ female]}{72 \times Serum\ creatinine\ (in\ mg/dL)}$$

- After minor surgery, patient should return to their preoperative regimen as soon as possible. After major surgery some patients may require dialysis for sometime in the postoperative period.

What is an arteriovenous fistula (AVF)? How will you anesthetize this patient for arteriovenous fistula surgery?

Patients with CRF may have to undergo AVF surgery, a couple of months preceding dialysis. A fistula is surgically created between radial artery and cephalic vein. Other sites include brachial and the basilic vein. The purpose of the AVF is to improve venous access for subsequent dialysis, i.e. dilates the vein sufficiently to allow easier access. This takes about 4–8 weeks to establish. Complications include infection, stenosis, thrombosis, aneurysm, and limb ischemia. This procedure may take considerable amount of time.

Various options available are general anesthesia, local infiltration anesthesia and regional anesthesia with brachial plexus block

General anesthesia (GA) is not without complications in view of significant comorbidities, but may have to be administered if patient refuses or is not willing for regional anesthesia. If GA has to administered the same considerations apply as for a CRF patient undergoing non-transplant surgery.

Local infiltration anesthesia is not ideal for an extensive procedure. Patient cooperation is very essential. Repeated injections may be required; this can increase the chances of systemic local anesthetic toxicity as mentioned earlier.

Regional anesthesia (RA) with brachial plexus block is an effective and preferred alternative to GA and infiltration anesthesia. RA has various advantages such as vasodilatation which helps in selecting the site for venous access, avoidance of hemodynamic instability and stress response with general anesthesia, excellent postoperative analgesia, and motor block. It can be administered in a patient with cardio-respiratory compromise. When conducting the case under RA, the maximum dose of the local anesthetic drug should be reduced by 25% because of their altered pharmacokinetics in CRF and lower seizure threshold; which predisposes these patients to local anesthetic toxicity.

What will be your anesthetic plan if this patient was dialysis dependent?

Dialysis [hemodialysis (HD)/ peritoneal dialysis (PD)] is usually initiated if the creatinine clearance is < 15 mL/min. An arteriovenous (AV) fistula is created usually on the non-dominant hand. It is important to establish the weekly frequency of dialysis.

Essentially the consideration and preparation is same as mentioned earlier (non dialysed CRF patient for non transplant surgery). Anesthetic considerations related to dialysis patient are mentioned below.

Preoperative evaluation

- Discuss with the nephrologists regarding fluid management of the patient. It is important to coordinate the timing of dialysis and surgery. The dialysis should finish 4–6 hours preoperatively to allow time for fluid shift, heparin to be eliminated. After dialysis, full blood count, urea, creatinine, electrolytes, clotting studies and patient's weight should be measured.
- The AV fistula site should be noted and that limb must be carefully protected during the surgery. The BP cuff should never be placed on the same arm; also IV line should be secured on the other hand or leg. Regional anesthesia can be safely administered if the clotting study is normal.
- These patients are very sensitive to induction agents and volatile agents; particularly soon after dialysis. Therefore, titrated and slow administration of induction agents will help in minimizing hemodynamic disturbances.
- Over transfusion is not a concern as the GFR does not need to be maximized. The only concern is sensitization for future transplantation, usually washed red cells should be transfused if facilities are available.

How will you manage acute hyperkalemia?

- ECG should be monitored, if there is wide QRS complex and tall peaked T wave, it should be treated urgently.
- The first line treatment is calcium chloride / gluconate (to stabilize the myocardium) and sodium bicarbonate (to reduce the acidosis). The two drugs should never be given in the same line as calcium carbonate may be precipitated.
- This should be followed by glucose insulin (a temporary measure to drive the potassium intracellularly).
- The excess potassium can be removed from the body by ion exchange resins and dialysis.

What will be your anesthetic plan for CRF patient undergoing renal transplant surgery?

The main anesthetic goal is to maintain renal perfusion and prevent harm to the donated kidney by avoiding hypoxia, hypovolemia, hypotension and nephrotoxic drugs. The shorter the duration of renal replacement therapy, better

the outcome following renal transplant. These patients undergo dialysis a day before the surgery to optimize volume (to within 0.5 kg of ideal body weight), acid base status and potassium levels. Preoperative dialysis improves graft function and decreases mortality. The anesthetic considerations are essentially the same as mentioned earlier. Certain specific concerns related to transplant surgery are mentioned below:

- These patients undergo comprehensive medical evaluation for transplant. Sometimes the transplant surgery may be done on an emergency basis; particularly in those patients undergoing cadaveric renal transplant. Preoperative history and investigations should be reviewed prior to surgery.
- Antihypertensives should be continued perioperatively.
- The AV fistula should be protected; central venous and arterial cannula should be secured on other extremities. The fistula site should be padded and palpated frequently to check patency. If the fistula patency is lost the surgeon should be informed immediately.
- Blood should be made available and transfused if required.
- IV Methylprednisolone is usually given after induction.
- Invasive monitoring (CVP and arterial) is established to guide fluid replacement.
- Post-transplantation after the renal clamps are removed, it is important to maintain perfusion to the transplanted kidney by restoration of circulatory volume (target CVP $\geq$ 10–12 mm Hg) and avoiding hypotension.
- The surgery may take hours and involve major fluid shifts, efforts should be taken to maintain normothermia.
- Anesthetists may be asked to administer diuretics to promote function of the kidney and increase the urine production. Mannitol and furosemide may be used to stimulate urine production and to restore function in the transplanted kidney after reperfusion.
- Mannitol 20–50 g (100–250 mL of 20% mannitol) is used immediately before opening the vascular anastomosis and reduces the incidence of acute renal failure. It expands the intravascular volume, increasing the tubular flow by preventing water absorption in the proximal convoluted tubule. It also enhances the release of prostaglandins in the kidney and may act as a free-radical scavenger.
- Furosemide 200–500 mg is commonly given while the vascular anastomosis is created, to stimulate diuresis. Its role in preventing renal failure is uncertain; the only indication for loop diuretics is fluid overload.
- Corticosteroids, used as a part of the immune suppression protocol, may also improve renal function.
- Hyperkalemia should be treated with insulin, glucose, hyperventilation and calcium if significant. Once the renal function is restored following transplant, hyperkalemia is controlled easily. If the kidney function is not restored, some patients may need dialysis in the postoperative period for hyperkalemia and fluid overload.
- Intraoperative hypertension may occur because of hypervolemia (preoperative dialysis is essential for hypervolemia), sympathetic stimulation secondary to surgical stress and pain should be treated by supplementing analgesia, deepening the anesthetic depth and use of short acting antihypertensive drugs. Sublingual nifedipine should be avoided as it causes a precipitous fall in BP, which can increase the perioperative morbidity.
- Hypotension occurs in the post-transplant period which can affect graft function. Therefore, blood pressure should be maintained perioperatively by restoring the intravascular volume and using vasopressors if required.
- Regional anesthesia/analgesia can be administered after weighing the risks and the benefits. Concerns with epidural anesthesia are effect of residual heparin after preoperative dialysis and platelet dysfunction; theoretically increasing the risk of epidural hematoma. Also, hemodynamic instability can adversely affect the graft function.
- Steroids and immunosuppressive agents are administered intraoperatively.

What is cold and warm ischemia time?

Ischemia time starts with clamping of the renal vessels in the donor and ends with vascular anastomosis in the recipient. Shorter the duration of ischemia time better is the organ preservation. 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.

Cold is chemia: the kidney is preserved by storing at 4°C usually less than 24 hours.

What is kidney cocktail?

The kidney cocktail is infused during the vascular anastomosis in the recipient (only for cadaveric kidneys). It consists of 600 mL of 0.45% dextrose in 0.45 % normal saline, 37.5 gm of albumin, 80 mg of frusemide and 37.5 gm of mannitol. (Some renal transplant units use the kidney cocktail; it may not be used in all units).

Postoperative care: Most patients can be extubated at the end of the surgery. Oxygen should be supplemented and all invasive monitoring should be continued. Urine output should be monitored closely. Maintaining renal perfusion is main goal in the postoperative period. The CVP should be

maintained between 10–12 mm Hg. Alternatively, advanced cardiac output monitoring can be instituted to guide fluid therapy. 5% dextrose, 0.45% normal saline or 0.9% normal saline is the recommended maintenance fluid at 30 mL/h plus previous hour's urine output. Oral hydration may be started after a couple of hours post-transplant. Immunosuppressive are started in the postoperative period. Commonly used drugs are cyclosporine A, azathioprine, monoclonal antibodies, and steroids. Newer drugs like tacrolimus and mycophenolate mofetil may replace cyclosporine A and azathioprine respectively in some immunosuppression protocols. The drug levels of cyclosporine A or tacrolimus must be maintained within the therapeutic range; therefore daily drug level monitoring is done in the perioperative period.

Postoperative analgesia: Patient controlled analgesia (PCA) with morphine or fentanyl can be used. There is no evidence to support one opioid over the other in post-transplant patients. Accumulation of metabolite may act a negative feedback and limit PCA use. Bolus dose of opioid must be reduced (0.5 mg bolus with 10–15 min lockout period) the patient should be closely monitored for respiratory depression. Epidural analgesia can be used, but hypotension should be avoided. NSAID's are contraindicated.

What postoperative complications can occur following renal transplant surgery?

The most common complications occurring in up to 70% of patient are acute renal failure and delayed graft function; requiring dialysis. Delayed graft function usually occurs because of acute tubular necrosis either due to ischemic damage prior to transplantation or hypoperfusion before and after transplant.

What are the anesthetic considerations if this patient, now post renal transplant, is posted for non transplant surgery?

The 1-year survival rate following transplant is improving, number of these patients present for elective and emergency surgery. Therefore anesthetists and surgeons may be required to manage these patients in hospitals which are not involved in transplant procedures. The main considerations for post-transplants are:

- Physiological and pharmacological problems of the grafted kidney
- Side effects of immunosuppressive drugs
- Risks of graft rejection
- Infection.

Discuss pharmacological problems seen in post-transplant patients.

Usual immunosuppressive regimen include triple therapy, consisting of a calcineurin inhibitor (cyclosporine or tacrolimus), an antiproliferative agent (azathioprine or mycophenolate mofetil), and a corticosteroid. Newer regimens include polyclonal/monoclonal antibodies and attempt to eliminate calcineurin inhibitors and corticosteroids.

Calcineurin inhibitors form the mainstay of immunosuppression therapy.

Cyclosporine: It may cause hypertension, hyperlipidemia, hyperkalemia, gum hypertrophy, and nephrotoxicity with renal fibrosis. They lower the seizure threshold so hyperventilation should be avoided. Hyperkalemia and hypomagnesemia is frequently seen with both these drugs. It potentiates neuromuscular block and may be associated with postoperative respiratory failure.

Tacrolimus is similar to cyclosporine, but may have a better cardiovascular risk profile, with less hypertension and hyperlipidemia. Although it is also nephrotoxic; it is associated with improved long-term post-transplant renal function. Important adverse effects include disturbed glucose metabolism and diabetes mellitus, tremor, diarrhea, neurotoxicity, and nephrotoxicity.

Antiproliferative agents

Mycophenolate mofetil has replaced azathioprine. It acts by inhibiting de novo purine synthesis in lymphocytes. It is not nephrotoxic. The oral bioavailability is 94%.

Azathioprine causes bone marrow suppression and can lead to pancytopenia requiring dosage adjustments. It may transiently antagonise neuromuscular block which is clinically insignificant.

Steroids are the main stay of post-transplant immunosuppression and continued to be used for prevention and treatment of graft rejection. Patients on long-term use may also have steroid-related side effects.

Conduct of anesthesia

The main anesthetic goal is to maintain renal perfusion with least alteration in the immunosuppressive regimen. Other considerations are similar to any CRF patient undergoing surgery. Important anesthetic considerations in post-transplant recipients are mentioned in this section.

Preoperative evaluation

History: One should elicit information regarding the:

- Functioning of graft and any evidence of rejection if the function is abnormal. The presence of rejection should be ruled out preoperatively. Presence of uremia, hypertension and proteinuria may indicate chronic graft rejection. Some post renal transplant patient's may require dialysis, all the necessary evaluation should be done as described earlier.
- Evidence of infection—as immunosuppressive drugs predispose these patients to bacterial, viral, fungal, or protozoal infections. They do not present with typical signs and symptoms of intra-abdominal sepsis such as fever, raised white cell count and physical signs of peritonitis.
- Other organ functions as they may be affected secondary to side effects of immunosuppressive therapy. Upper gastrointestinal bleeding may occur probably secondary to acid peptic disease, gastritis, or cytomegalovirus gastroenteritis. Hepatobiliary and pancreatic diseases are common after transplantation.
- All baseline investigations should be done including liver function test as they may be deranged secondary to immunosuppressive drugs.

Induction of anesthesia

- The choice of perioperative monitoring is determined by the nature of surgery.
- Nasal endotracheal intubation should be avoided to prevent infection caused by nasal flora. Laryngeal mask airway can be used safely.
- Regional anesthesia can be administered if the clotting studies and platelet count are normal. Azathioprine or antithymocyte globulin can cause thrombocytopenia, which increases the bleeding and hematoma risks associated with central neural blockade.
- Drugs—post renal transplant patients may have a normal serum creatinine value; however, their GFR is low in comparison to normal individuals; so the renal elimination of drugs may be prolonged and drugs should be administered cautiously.
  - NSAID's are contraindicated and can augment nephrotoxicity of cyclosporine
  - Drug interactions—cyclosporine A interacts with non depolarizing neuromuscular blockers potentiating their action, thus decreasing the dose and frequency of top-up doses is advised. Azathioprine antagonises neuromuscular blocking drugs.
  - The drug levels of cyclosporine A or tacrolimus must be maintained within the therapeutic range therefore daily drug level monitoring is done in the perioperative period.
  - Oral cyclosporine should be administered 4–7 hours prior to surgery to maintain therapeutic levels.
  - Immunosuppressive agent should not be stopped perioperatively; dose may have to be altered if route of administration is to be changed. Oral prednisone can be converted to IV methylprednisolone.
  - For azathioprine the oral and IV doses are equivalent.
- Severe perioperative airway obstruction may occur because of underlying post transplant lymphoproliferative disease.

Post renal transplant patients are on steroids, how will you supplement steroids perioperatively?

All patients who are taking less than 10 mg prednisolone have been shown to have a clinically normal response to HPA testing. These patients do not need additional steroid cover other than their usual steroid dose. This should be taken preoperatively and continued as soon as oral intake is possible.

Patients who are on long-term steroid supplementation equivalent to more than 10 mg prednisolone daily (or who have received such a dose within the last 3 months) should receive a physiological replacement regimen as mentioned in Table 35.1. Alternatively, adrenal suppression should be excluded by preoperative biochemical testing.

Table 35.1 Perioperative steroid treatment regimen

Patients who have received a regular daily dose of more than 10 mg prednisolone or equivalent in the last three months	
Minor surgery (hernias, hand surgery)	25 mg hydrocortisone at induction
Moderate surgery (hysterectomy)	Usual preoperative steroids + 25 mg hydrocortisone at induction +100 mg hydrocortisone/day
Major surgery (major trauma, prolonged surgery, or surgery where there is delayed oral intake)	Usual preoperative steroids + 25 mg hydrocortisone at induction + 100 mg hydrocortisone/day for 2–3 days Resume normal oral therapy when gastrointestinal function has returned
All other patients—no additional steroids required	

How can you prevent postoperative acute renal failure (ARF) in patients with normal renal function? How can you prevent postoperative ARF in patients with impaired renal function? What are RIFLE criteria for grading renal dysfunction?

Renal impairment following surgery is most often associated with multiorgan dysfunction, increasing postoperative morbidity, length of stay in ICU and hospital and may progress to chronic renal failure and dialysis dependent ESRD.

An international interdisciplinary collaborative group, the acute dialysis quality initiative (ADQI), recently has created a standard grading system for acute renal dysfunction. The term acute kidney injury (AKI) has recently been proposed to incorporate the entire range of abnormalities of renal function. The acronym RIFLE defines three grades of increasing severity of acute renal dysfunction (R, risk; I, injury; F, failure) and two outcome variables (L, loss; E, end-stage) that are based on the change in serum creatinine values and urine output.

The RIFLE grading system for acute renal dysfunction is shown in Table 35.2

Table 35.2 RIFLE grading system

Grade	Glomerular filtration rate criteria	Urine output (UO) criteria
R, Risk	Serum creatinine increase: 1.5-fold; GFR decrease: > 25%	UO < 0.5 mL/kg/h for 6 h
I, Injury	Serum creatinine increase: 2-fold; GFR decrease: > 50%	UO < 0.5 mL/kg/h for 12 h
F, Failure	Serum creatinine increase: 3-fold; GFR decrease: > 75%; Serum creatinine decrease: > 350 µmol/L (4 mg/dL) with acute increase > 44 µmol/L (0.5 mg/dL)	UO < 0.3 mL/kg/h for for 24 h or anuria for 12 h
L, Loss	Persistent ARF = complete loss of renal function for > 4 weeks	
E, End stage	ESRD = complete loss of renal function for > 3 months	

Patients at risk

- Undergoing cardiac/vascular surgery/major surgery
- Elderly
- Preexisting renal dysfunction
- Persistent hemodynamic instability leading to renal hypoperfusion
- Perioperative use of nephrotoxic drugs.

Prevention strategies

Nonpharmacological measures

- **Maintenance of renal blood flow and renal perfusion pressure** –
  - The first step is to restore the circulating volume and correct hypovolemia.
  - Vasopressor and inotropes should be subsequently used for management of systemic arterial hypotension and low cardiac output respectively. Norepinephrine is an excellent first-line vasopressor agent. At present there is no confirmatory evidence to prove that it decreases renal, hepatic, or gastrointestinal blood flow when used to treat arterial hypotension.
  - A target mean arterial pressure of 65–75 mm Hg is used in clinical practice however in hypertensive patients it should be higher depending on their blood pressure control as the autoregulation shifts to the right.
- **Intravascular volume expansion**
  - Both crystalloids and certain colloids can be used in patients with renal impairment.
  - Albumin and gelatin are safe in patients with normal renal function.
  - The safety of hydroxyethyl starch solutions in patients with renal failure is not established. When hydroxyethyl starch was compared to Ringer's lactate in critically ill patients with severe sepsis, it was associated with a higher incidence of bleeding and ARF.
  - Postsurgical patients who receive IV contrast will benefit from the use of isotonic IV fluids and nonionic, iso-osmolar contrast in the lowest possible volume.
- **Avoidance of nephrotoxic agents**
  - The use of once-daily aminoglycoside dosing and the use of liposomal amphotericin B have decreased the risk of nephrotoxicity associated with these drugs.
- **Early and appropriate management of postoperative complications**.
  - Early diagnosis and treatment of postoperative complications such as acute cardiac dysfunction, hemorrhage, sepsis and intra-abdominal hypertension/ compartment syndrome are essential to prevent the development of ARF.
  - Abdominal compartment syndrome caused by intra-abdominal hypertension decrease renal perfusion and may precipitate ischemic ATN. High index of suspicion and early recognition by intravesical pressure measurement helps in diagnosing this condition. If detected decompressive laparotomy is usually needed.

Pharmacological measures

Various drugs have been used as protective agents for preventing perioperative renal dysfunction such as dopamine agonists (Dopamine, Dopexamine, Fenoldopam), diuretics (Loop and Osmotic diuretics), natriuretic peptides, N-acetyl cysteine. Most of studies were in patients who underwent cardiac surgery or in critically ill patients with renal dysfunction.

- *Dopamine:* A large multicenter trial demonstrated that it does not prevent ARF. It neither avoids the need for renal replacement therapy, nor decreases the mortality.

Perioperatively, it increases the urine output but has no effect on outcome. Also systematic reviews have concluded that there is no role of low-dose (renal dose) dopamine for renal protection.

- *Fenoldopam:* It does not prevent radiocontrast-induced nephropathy in patients with preexisting renal impairment, has no effect on outcome in critically ill ICU patients with early acute renal impairment, and is not renoprotective perioperatively in high-risk cardiac surgical patients.
- *Diuretics:* Both the loop and osmotic diuretics do not demonstrate significant renal protection in the perioperative setting; however there is limited evidence and future trial has been proposed.
- *N-acetylcysteine (NAC):* There is evidence to support its prophylactic use as an antioxidant along with isotonic IV fluids in the prevention of radiocontrast induced nephropathy. Recent trials in the perioperative and critically ill patients do not demonstrate renoprotective effect.

A Cochrane review evaluating available evidence regarding pharmacological interventions (including the use of dopamine and its analogues, diuretics, calcium channel blockers, ACE inhibitors or hydration fluids) for perioperative renal protection suggested that there is no evidence that these drugs are beneficial. Also there was no difference in the morbidity or mortality.

Suggested Reading

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36 Tonsillectomy

S Myatra, S Bakshi, J Doctor

4-year-old male child, weighing 16 kg has a history of 3–4 episodes of tonsillitis in the past year. He now presents with:

Throat pain and dysphagia which has progressively increased:	Since 10 days
Reduced intake:	Since 8 days
Fever:	Since 2 days
Severe weakness:	Since 2 days

On examination the child is febrile, HR-120/min, looks dehydrated and has enlarged and inflamed palatine tonsils with cervical adenopathy. The child is posted for tonsillectomy due to repeated infections.

What are tonsils? What are adenoids? What is their function? What is tonsillitis?

The tonsils are areas of lymphoid tissue on either side of the throat. Most commonly, the term "tonsils" refers to the palatine tonsils that can be seen in the back of the throat. There are three masses of tissue: the tubal, palatine and lingual tonsil. The adenoid tissue is positioned in the midline of the posterior nasopharyngeal wall immediately inferior to the rostrum of the sphenoid. Together this set of lymphatic tissue along with the adenoid is known as Waldeyer's tonsillar ring.

The tonsils are located in the lateral oropharynx. The tonsillar branch of the facial artery forms the main arterial blood supply. The venous drainage is via a plexus surrounding the tonsil, which drains into the pharyngeal plexus. The external palatine vein enters the tonsillar bed from the soft palate. This large vein is usually responsible for the venous hemorrhage following tonsillectomy.

The sensory supply is from the glossopharyngeal and lesser palatine nerves. Important structures deep to the inferior pole of the tonsil are the glossopharyngeal nerve, the lingual artery and the internal carotid artery.

Like other organs of the lymphatic system, the tonsils act as part of the immune system to help protect against infection. In particular, they are believed to be involved in helping fight off pharyngeal and upper respiratory tract infections. Tonsillitis is the infection of the tonsils.

What are the indications and contraindications for tonsillectomy and adenoidectomy?

The indications for surgery may be absolute or relative. The indications for surgery are:

1. Upper airway obstruction, dysphagia and obstructive sleep apnea.
2. Peritonsillar abscess, not responding to adequate medical management and surgical drainage.
3. The requirement of biopsy to confirm tissue pathology in suspected neoplastic causes.
4. Recurrent tonsillitis that is unresponsive to medical treatment.
   - Sore throat secondary to tonsillitis.
   - More than 5 episodes of tonsillitis in one year.
   - Symptoms persisting for more than one year.
   - Severe disabling episodes of sore throat.
5. Persistent bad-breath and taste in mouth due to chronic tonsillitis.
6. Persistent tonsillitis in streptococcus carrier, which is unresponsive to antibiotics.

Contraindications:

- Presence of acute infection
- Abnormal coagulation profile
- Uncorrected partial/complete cleft palate since in these patients adenoids help fill the velopharyngeal gap and facilitate speech.

What is the usual clinical presentation of children with tonsillitis?

- Pain in the throat
- Dyphagia
- Mouth breathing
- Failure to thrive/repeated infection—pain fever, tachycardia.
- Cervical adenopathy.
- Visibily inflamed tonsil which may have discharge.

What are the different methods available for tonsillectomy?

- Blunt dissection and tonsilar snare
- Bipolar Radio frequency
- Electrocautery
- Ultrasonic dissection
- LASER
- Powered microdebrider.

What history and clinical examination will you perform in this child ?

History

- Routine anesthetic history and examination for a pediatric patient including milestone development and vaccination.
- History of tonsillitis: Repeat episodes of fever, throat pain, dysphagia.
- History of any easy bruising, bleeding gums, epistaxis, mennorhagia or bleeding at any other sites
- Family history of any bleeding disorders
- Recent ingestion of Aspirin, NSAIDs
- Mouth breathing
- The triad of hyponasality, snoring, and mouth breathing normally indicates enlarged, obstructing adenoids
- Other symptoms of adenoid disease include rhinorrhea, postnasal drip, chronic cough and headache
- History of possible allergies, GERD, and sinusitis.
- *Obstructive sleep apnea*. In children, adenotonsillar hypertrophy is the most common cause of obstructive sleep apnea. The signs and symptoms include chronic hypoxemia manifesting itself as polycythemia and right ventricular strain. The symptoms like snoring, apneic episodes followed by grunting and restlessness occurring during sleep. The daytime symptoms include headaches, excessive daytime somnolence and not feeling fresh in the morning.

Examination

- Routine examination in a pediatric patient
- Loose/missing teeth: Most children have fallen milk teeth at this time. This is also important for application of mouth gag
- Patency of oral and nasal cavity
- Patients may have "adenoid facies" (long face, flattened midface, open mouth) and hyponasal speech
- Enlarged (> 2 cm) or tender cervical adenopathy
- Tonsillar or pharyngeal exudates.

What investigations will you ask for?

- Hemoglobin and complete blood count
- Coagulation profile and platelets only if there is history suggestive of bleeding tendencies.

How will you optimize this child prior to surgery?

This child has clinical signs suggestive of infection and dehydration probably because of inadequate intake due to dysphagia. Hence, a course of antibiotics should be started and adequate hydration in the form of intravenous fluids should be given prior to surgery.

How will you premedicate this child?

Premedication should include an antisialogogue and possible a narcotic. However, barbiturates will be of little use in short upper airway surgery which requires quick return of protective airway reflexes. Sedatives should not be used if there is history suggestive of obstructive sleep apnea.

What are the anesthetic considerations for tonsillectomy surgery?

Basic principles of anesthesia for tonsillectomy in children remain the same as for general pediatric anesthesia. Specific points with reference to, intraoperative and postoperative management are described below:

- The goal of anesthesia for elective tonsillectomy is to maintain deep general anesthesia that prevents reflex-induced hypertension, tachycardia or arrhythmias. Muscle relaxation is required to allow placement of the mouth gag and prevent any bucking, coughing or straining. A rapid recovery of consciousness and return of protective airway reflexes is also desired.
- Inhalational induction with sevoflurane is preferred in small children especially when IV line is not inserted and in

OSA patients. If IV line is present Thiopentone or propofol along with muscle relaxant can be given.

- *Endotracheal intubation:* Intubation under deep inhalational or muscle relaxant assisted anesthesia is preferred. Regular tube/RAE tube may be passed by orotracheal route. Throat should be well packed especially when uncuffed tubes are used to prevent aspiration of blood and secretions. An oral endotracheal tube can either be fixed in the midline or fixed on one side at the angle of the mouth and the side changed once the tonsillectomy is done and hemostasis achieved for removal of the opposite tonsil. When only tonsillectomy and no adenoidectomy is planned one can also insert a nasotracheal tube. Intubation could be difficult if the tonsils are very large and approximating in the midline (kissing tonsils). Flexible LMA may be used for adenotonsilletomy surgeries and is routinely used in some centers. Advantages are that it requires lighter plane of anesthesia, and there is no need for muscle relaxants; with resultant rapid induction and smooth recovery. Here the LMA is not removed until full return of reflexes. The main disadvantage is if airway is lost during surgery, it can be difficult to rectify the situation.
- *Maintenance:* Anesthesia is maintained with inhaled anaesthetics and short-acting opioids like fentanyl using spontaneous ventilation and/or muscle relaxants with controlled ventilation. However adequate depth should be maintained to prevent any reflex-induced hypertension, tachycardia and arrhythmias and avoid bucking ,coughing or straining during surgery
- Local anesthetic plus adrenaline applied in the tonsillar fossa gives the advantages of bloodless dissection, reduced operative time and reduced postoperative pain. If large volumes are injected, it can give rise to respiratory obstruction once the patient is extubated because of bilateral glossopharyngeal nerve block.
- Since the operative field is close to the airway, one should be very vigilant about accidental extubation or aspiration of blood and secretion if the throat pack is displaced under GA when uncuffed tubes are used.
- Blood loss during tonsillectomy may be difficult to estimate and may reach up to 5 % of the blood volume. Blood transfusion may be required in some cases. IV fluids like RL or DNS can be used to maintain adequate volume status.
- Protection of the eyes is very important especially when the gag is used.
- At the end of surgery, pack removal and good pharyngeal and laryngeal suction under vision is essential.
- An antiemetic should be given prior to reversal. Patients undergoing tonsillectomy are prone to develop PONV, so an antiemetic is recommended: ondansetron (0.1 mg/kg) or dexamethasone (0.1–0.2 mg/kg) or a combination of both can be considered.
- *Reversal:* Reversal of muscle relaxant should be done using neostigmine and atropine and patient should be extubated only when awake and there is return of protective airway reflexes. Extubation should be smooth thereby preventing rise in blood pressure which can cause bleeding.
- Patient should be transported in tonsillar position with oxygen supplementation.

How is the patient positioned during surgery?

The patient's head is put in a slightly extended position, held by a sandbag/bolster, under the shoulder, while the mouth is kept open and the tongue is held out of the way by a Boyle-Davis or a similar gag. In this position, the operator has a direct view of the tonsils and there is the added advantage of the posterior part of the pharynx forming a sump into which the blood may drain, below the level of the glottis.

It is important to check that the ETT has not migrated out during extension of the neck.

How will you manage postoperative pain after tonsillectomy?

Post-tonsillectomy pain is usually mild to moderate and can be taken care of by paracetamol syrup. NSAIDs are usually avoided because of their tendency to increase bleeding and cause bronchospasm in patients with allergic diathesis.

What is the tonsillar position? What is its clinical significance?

After surgery the child is placed in the "tonsil position" which is a left lateral position, with one knee flexed and the hand under the face along with a slight head low position. This allows the blood and secretion to drain out rather than flow back onto the vocal cords.

What are the complications you can anticipate in the immediate postoperative period?

- Post-tonsillectomy bleeding
- Airway obstruction because of upper airway edema, presence of blood and secretions and laryngospasm
- Postoperative nausea and vomiting during first 24 hours (as high as 70%) because of pharyngeal mucosal irritation from surgery and swallowed blood and secretions.
- Pain and sore throat lasts for 3–4 days.

- Postoperative respiratory complications.
- Negative pressure pulmonary edema may occur due to sudden release of upper airway obstruction, but this is very rare.

How do you diagnose post-tonsillectomy bleed? How do you plan the anesthetic management?

Bleeding is seen more in older children (age > 11 years → 69%) and in patients with chronic tonsillitis (75%). Primary bleeding occurs within first 24 hours, secondary bleeding from 24 hours to 5–28 days. Bleeding from adenoidal bed (3.2%) is more common in first 4 hours, tonsillar bleeding (1.2 %) is more common in first 6–8 hours. For day care procedures, it is safer to observe the patient for 8–10 hours after surgery. The incidence of bleeding following tonsillectomy is 0.5–2% depending upon the surgical technique.

Secondary bleeding: This is associated with sloughing of the eschar (dead tissue) overlying the tonsillar bed, loosened vessel ties or infection from underlying chronic tonsillitis.

Any amount of bright red blood coming from the mouth or the nose should alert the doctor to the possibility of postoperative bleeding. Frequent swallowing, tachycardia, and coffee-ground emesis are indirect signs of bleeding. If the patient complains of intermittent bleeding but is not actively bleeding on presentation, he or she should be admitted for overnight observation and administration of IV fluids. Coagulation testing should be obtained following a bleeding episode, and if abnormal, hematology consult and additional coagulation testing should be obtained. A CBC should also be obtained to assess the degree of hemorrhage and as a baseline in case of further bleeding.

Minor bleeding may be controlled by gargling with ice water and packing. If bleeding persists and the patient is uncooperative, he will be needed to be taken up for re-exploration under anesthesia.

Important considerations while anesthetizing a child for control of bleeding after tonsillectomy are:

- Degree of blood loss may be underestimated, as it is not visible and may be swallowed.
- Child may loose large amounts of blood and become hypovolemic and even progress to shock in a short time. Child may even have altered consciousness if bleeding is severe.
- Immediate resuscitation with colloid and crystalloid while waiting for blood to become available. More blood should be kept crossmatched for surgery.
- Preoperative sedation should be avoided
- Adequate preoxygenation
- Wide bore good IV access
- IV induction agent depending on hemodynamic stability (Thiopentone sodium, propofol) may be used,
- Child should be considered as full stomach as large amount of blood and secretions may be swallowed. A rapid sequence intubation with cricoid pressure and cuffed ETT using succinyl choline is warranted. Two good working suctions should be ready at the head end in case of vomiting
- Reintubation may be difficult if bleeding is obscuring the view or due to edema from previous airway instrumentation and surgery. A smaller size ETT than the previous anesthetic should be ready. This may needed due to presence of airway edema.
- The child may become hypothermic during surgery due to large volume transfusion. The child should be kept well-covered to maintain body temperature, and if possible a warming blanket used with temperature monitoring. Hypothermia exacerbates coagulopathy.
- Decompression of stomach should be done with a nasogastric tube after intubation, to ensure that the stomach is empty prior to extubation.
- Extubation should be done in lateral position and only if the child is fully awake with normal gag, cough reflex and is stable hemodynamically.

How is the anesthetic management of tonsillectomy in an adult different from children?

This is managed exactly in the same way as in children, except that, as adenoid tissue is not present in adults the tracheal tube is more often passed nasally than through the mouth. Also the requirements of postoperative analgesics and antiemetics are greater in adults than in children.

Is there a role for performing tonsillectomy under local anesthesia?

Due to the multiple origins of the nerve supply to the tonsil, there is no single nerve trunk that can be blocked to give effective anesthesia. However, local infiltration around the capsule is quite effective. Following topical anesthesia of the mucous membrane, three separate injections of local anesthetic solutions are made: one in the region of the upper pole, one into the anterior pillar and one in the region of the lower pole. In each instance, attempt is made to deposit the solution between the capsule and the pharyngeal wall.

- This is a very vascular area and special care must be taken not to inject local anesthetic into a blood vessel.
- Tosillectomy under these circumstances is however unpleasant for the patients and surgeons alike.

- Small children will not be cooperative for local injections.
- It may also be dangerous in situations where the protective laryngeal reflexes can be obtunded by spread of the nerve block beyond the immediate area of the tonsil making even a conscious patient liable to laryngeal obstruction and aspiration of blood and debris.

For these reasons tonsillectomy is rarely performed under local anesthesia. However, the use of local infiltration anesthesia in combination with general anesthesia has been claimed to reduce the reflex response from surgical field, reduce bleeding, aid dissection and contribute to postoperative comfort.

How will you manage a case of peritonsillar abscess or Quinsy?

This is more frequently seen in older children. This occurs in proximity to the tonsil, but outside the tonsillar capsule and most often in the soft palate. It is usually limited to one side and causes severe sore throat, difficulty in swallowing, high fever and progressive difficulty in opening the mouth. The affected tonsillar area is markedly inflamed with uvula displaced to the opposite side. If it does not resolve with antibiotics, then incision and drainage into the pharynx will be necessary. Older child may allow the drainage under conscious sedation but for anxious or smaller children, GA would be needed.

Consideration while administering general anesthesia:

- Aggravation of a preexisting respiratory obstruction
- Even with relaxation, trismus may not resolve, making laryngoscopy and intubation difficult.
- Abscess may rupture at any time during induction or intubation and there is a risk of aspiration of purulent material.
- GA is induced with inhalational agent in oxygen or intravenous induction agents like propofol along with sevoflurane.
- Patient is kept in head low position with the head turned toward the affected side.
- Under deep plane of anesthesia laryngoscopy is done extremely carefully for fear of rupturing the abscess keeping two wide bore suctions ready.
- Muscle relaxant should not be given until the anesthesiologist is certain about securing and maintaing the airway. Once airway is secured with cuffed ETT, the rest of the management is straightforward.

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37 Circumcision

P Jain

A one year old child, has ballooning and pain around penis during micturition, is scheduled for circumcision (CS).

What are the preoperative considerations in circumcision?

It is a common day-case minor procedure of about 20 minutes, with minimal blood loss, and it entails removal of prepuce or preputioplasty or prepuce plasty.

What are the various anesthesia techniques used for CS?

It can be done under GA with spontaneous ventilation (with LMA), caudal or penile block or ring block.

How do you control postoperative pain?

It is a very painful day case procedure and parents are advised to apply topical lignocaine jelly regularly and diclofenac suppository (1 mg/kg) > 1 year OR paracetamol supp 30–40 mg/kg < 1 year of age for several days.

How do you give penile block?

The safest technique is to inject an adequate volume of local anesthetic bilaterally deep to the fascia into the pear shaped spaces on each side of the suspensory ligament. This avoids mid-line injection and therefore potential damage to the dorsal vessels and provides the maximum chance of diffusion into the nerves to block them. Injecting an adequate volume of local anesthetic (estimated in children at 1 mL + 0.1 mL/kg on each side) should ensure that the ventral branch is also blocked so that a satisfactory block is achieved.

The technique involves inserting the needle until it touches the pubic symphysis. This gives a guide to depth. The needle is then withdrawn and redirected to pass below the symphysis and 3–5 mm deeper depending on the size of the patient. It is preferable to direct it slightly laterally into the pear shaped space and then to reinsert in on the other side depositing equal volumes on each side. Avoiding the midline injection reduces the chance of penetrating the dorsal vessels of the penis and causing hematoma. If a short beveled needle the fascia may be felt as a slight resistance when it is penetrated, but in small children this is not always felt as it is thin and may offer little resistance.

Describe the caudal block.

Anatomy: The sacrum articulates with the fifth lumbar vertebra above and the coccyx below. The sacral hiatus is shaped by incomplete midline fusion of the posterior elements of the distal portion of the fifth or sometimes the fourth sacral vertebra. This inverted U-shaped space is covered by the posterior aspect of the sacrococcygeal membrane and is an important landmark in caudal epidural block. The remnants of the inferior articular process elongate downwards on both sides of the sacral hiatus. These two bony processes are called the sacral cornua (horns) and define important clinical landmarks during caudal epidural block. The hiatus is covered only by skin, a subcutaneous fatty layer and the sacrococcygeal membrane. The distal most portion of the dural sac and the sacral hiatus usually terminate between levels S1 and S3.

The sacral canal contains:

1. The terminal part of the dural sac, ending between S1 and S3.
2. The five sacral nerves and coccygeal nerves making up the cauda equina. The sacral epidural veins generally end

at S4, but may extend throughout the canal. They are at risk from catheter or needle puncture.
3. The filum terminale: The final part of the spinal cord which does not contain nerves. This exits through the sacral hiatus and is attached to the back of the coccyx.
4. Epidural fat, the character of which changes from a loose texture in children to a more fibrous close-meshed texture in adults. It is this difference that gives rise to the predictability of caudal local anesthetic spread in children and its unpredictability in adults.

Technique of caudal block

Aseptic technique is mandatory. Position of the patient in the left lateral position with the legs flexed at the hip. Identify the sacral hiatus as the apex of an equilateral triangle with the base formed by a line joining the posterior superior iliac spines. The sacral hiatus is covered by the sacrococcygeal membrane. Make a small nick in the skin with a needle to reduce the possibility of a dermoid. Use a 22 G or 20 G cannula at 60° to the skin from the midpoint of the line joining the sacral cornua. A small 'give' indicates penetration of this membrane. Flatten the cannula and then advance. Advancement of a cannula rather than needle may reduce the incidence of inadvertent dural or vascular puncture. Test aspiration should be gentle. Aspiration should be repeated during injection of local anesthetic. 0.25% bupivacaine is commonly administered (1 mL/kg).

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38 Acute Postoperative Pain

P Jain, M Joshi, K Sharma

The International Association for the Study of Pain has proposed a working definition for pain: Pain is an unpleasant sensory and emotional experience associated with either actual or potential tissue damage.

During the past two decades, undertreatment of pain has been recognized as an important issue in health care worldwide. Deficiencies in pain management may be due to inadequate staff training, knowledge deficits, unhelpful staff, patient attitudes, poor pain assessment, and fear of analgesic side effects and lack of accountability. Many hospitals have multidisciplinary acute pain services (APS) for management of postoperative pain, training, development of guidelines, to undertake research and audit which leads to improved care in pain. Also the requirement of a dedicated acute pain nurse for effective pain service has been emphasized. Specialized pain relief methods like patient controlled analgesia (PCA), epidural and perineural infusions, now included in APS have improved care. However, many patients still continue to suffer considerable pain. In 2005, a survey of 14 hospitals in the United Kingdom revealed that at 24 hours postsurgery, 60% of patients had a pain score of 5 or more out of 10 on movement, at 7 days postoperatively 39% had a pain score of 5 or more on movement, with 8% reporting a pain score of 8–10.

What are the causes of postoperative pain? Why is it important to treat it?

Surgical procedures invariably cause tissue damage along with presence of drains, tubes and postoperative complications resulting in pain. Postoperative pain is often underestimated and undertreated. The impact of inadequate pain relief can result in delayed mobilization, increased morbidity and mortality due to respiratory and thromboembolic complications, increased hospital stay, psychological distress and anxiety. Pain management in perioperative setting refers to actions before, during and after a procedure that is intended to reduce or eliminate postoperative pain before discharge.

Describe the pathophysiology of postoperative pain. Explain the concept of chronic postsurgical pain and how will you manage it? Why is it difficult to treat postoperative pain?

Studies have shown that incisional pain differs from inflammatory or neuropathic pain. Hyperalgesia in incisional pain may be mediated by sensitization of A fibers and C fiber nociceptors and conversion of silent A-δ nociceptors to mechanically sensitive fibers after incision. Ischemic pain mechanism may also contribute to postsurgical postoperative pain as indicated by increased lactate concentration and low pH occurring in skin and muscle wounds. Central neuronal sensitization may also be a contributory factor.

Chronic postsurgical pain is more common especially after certain types of surgery for example thoracotomy and mastectomy. It should be diagnosed when pain persists beyond the expected healing period associated with tissue injury (two months or longer after most surgical procedures). Ongoing inflammation or injury to peripheral nerves may lead to persistent pain. Predictive factors for developing continuing postoperative pain include preoperative pain, repeat surgery, prolonged surgery, severe postoperative pain, surgical approaches with a higher risk of nerve damage, chemotherapy or radiation and psychological and depressive symptoms. Preemptive analgesia in which one or more analgesic is administered prior to a noxious event to prevent

peripheral and central sensitization reduces post injury pain. There is some evidence that it prevents development of chronic pain but it is highly likely that early intervention when signs are detected is more likely to be beneficial. A preemptive analgesic effect may be influenced by multiple factors like choice of drug, nature and degree of tissue damage, duration of surgery, time, route of administration and time period of central sensitization.

Enumerate the common drugs used for post-operative pain management?

Acute pain can cause pathophysiological responses; which are initiated when nociceptors are activated after tissue injury causing local inflammatory response; and behavioral and neuroendocrine activation like tachycardia, hypertension, hyperglycemia, immunosuppression, decreased regional blood flow, venous stasis and platelet aggregation.

Pharmacologic treatment is the mainstay. Multimodal analgesia in the form of paracetamol, NSAIDs, opioids and modern techniques such as 'patient controlled analgesia' (PCA), epidural and perineural infusions are effective in treating postoperative pain.

What is the rationale for multimodal analgesia?

Multimodal analgesia uses a combination of two or more analgesics with an aim to improve analgesia through the use of analgesics; each with a different mode of action; and decrease side effects through a reduction in the doses of analgesics, particularly opioids. It can be defined as a combination of opioid and non opioid analgesic with or without a regional anesthetic block; resulting in improved analgesia with decrease in side effects. Studies have compared patients with intravenous (IV) PCA with and without additional agents and have measured outcomes such as efficacy, opioid consumption, side effects and complications. One study examined the addition of paracetamol, selective cyclo-oxygenase 2 inhibitors (COX-2 inhibitors) and nonsteroidal anti-inflammatory drugs (NSAIDS) to IV PCA revealing that all provided an opioid sparing effect. The morphine sparing effect with paracetamol was less than 20%, with COX-2 inhibitors approximately 25% and with multiple doses of NSAIDS approximately 40%. Whenever possible, anesthesiologist should use multimodal pain management therapy. Central regional blockade with local anesthetics should be considered. Unless contraindicated, patients should receive a round-the-clock regimen of COXIBs, NSAIDs, or acetaminophen. Dosing regimens should be administered to optimize efficacy while minimizing the risk of adverse events. The choice of medication, dose, route, and duration of therapy should be individualized.

What is the role of paracetamol (acetaminophen)?

Paracetamol has b**e**en widely accepted as an effective analgesic and it is used in combination with other analgesics for moderate or severe pain. Intravenous paracetamol due to its quick onset and efficacy ensures a more predictable plasma concentration and may provide considerable opioid sparing effect. Many prescriptions now include paracetamol being given regularly by the clock and often via the IV route for the immediate postoperative period due to lower side effects, better profile and tolerability. But some studies have shown that nurses often did not give paracetamol regularly, nor did they try to explain the importance of the multimodal approach to patients who themselves thought paracetamol was too weak. Therefore, the challenges also include educating the patient and staff of the benefits of multimodal approach.

What is the role of nonsteroidal anti-inflammatory drugs (NSAIDs)?

NSAIDs are most commonly used postoperative analgesic drugs including cyclo-oxygenase inhibitors (aspirin, diclofenac, and ibuprofen) and new COX-2 selective inhibitors. They are effective in postoperative pain however adverse effects and contraindications may limit their use. The main concerns are interference with platelet function, renal impairment, gastric and duodenal ulceration and the potential for bronchospasm in asthmatics sensitive to aspirin and NSAIDs. Careful patient selection is necessary. NSAIDs being peripherally acting analgesics improve analgesia significantly, attenuate hyperalgesia around the incision and reduce the incidence of nausea, vomiting and sedation when used in conjunction with PCA. There is a considerable opioid sparing effect and may benefit patients in whom low opioid doses are preferred.

What is patient controlled analgesia? What are the other modes of analgesia?

There are reports that intravenous opioid PCA provides better analgesia than conventional intramuscular (IM) or subcutaneous (SC) opioid regimes as they maintain the minimally therapeutic serum opioid concentrations better. PCA improves patient satisfaction, reduces morbidity and hastens recovery. It allows the patient to independently titrate their analgesic requirements allowing them greater control over their treatment. Although PCA is mostly indicated for postoperative analgesia, it can be also used to maintain pain relief once pain control is achieved e.g. acute pancreatitis, rib fractures or trauma awaiting surgery. It is important to remember that PCA is essentially a maintenance

therapy, therefore before starting the PCA (individually titrated doses), the patient's pain should be initially controlled by giving analgesic boluses. Other routes for PCA include oral, intranasal, subcutaneous, transdermal, regional infusions, epidural and intravenous.

Intravenous PCA has become the preferred method for delivering opioids to patients especially in those unable to take oral analgesics. Benefits includes improved patient satisfaction, reduction of pulmonary complications such as atelectasis, greater safety, consumption of less nursing time compared to other forms of analgesia. Patient controlled analgesia has been shown to be effective in both young and elderly patients who have good cognition ability. It is not indicated in elderly or confused patients or the very young who may not be suitable candidates and patients with poorly functioning hands (arthritis, hand surgery, spinal injury) who may not be able to press the demand button.

Although PCA is a relatively simple concept, its use requires dynamic interaction with both the patient and the machine; so a preoperative assessment as to the willingness of the patient to use PCA should be conducted. The patient must be counseled about its correct use, he must be comfortable to use it independently without fear of overdose and addiction. Preoperatively pain related counseling must be done by the anesthetist responsible for the patient. Subsequently, the ward and recovery nurses should reinforce the education. Errors may include programming errors and faulty equipment. Staff training and safe standardized systems, including monitoring requirements are essential.

Postoperative nausea and vomiting is a major side effect in patients. It is important to have effective communication, education and careful attention to reduce side effects. Considering a change to an alternative opioid, if patient has persistent side effects is also important.

What is the role of regional anesthesia in post-operative pain?

Regional anesthesia is now included as a valuable component of the multimodal combination. It can be used as local wound infiltration or infusions, perineural injections, spinals and epidurals. Wound infiltration with local anesthetic for postoperative analgesia has been found to be effective in reducing pain scores, decreased need for opioids, nausea and vomiting and increase in patient satisfaction. Perineural analgesic infusion was found to be effective in reducing opioid consumption.

Epidurals with local anesthetic combined with an opioid can provide superior analgesia than most other techniques. Epidurals are often used in high-risk surgeries with evidence showing lower pain scores than systemic opioids. Studies have also shown that epidurals may have reduced risk of myocardial infarction and positive effects on cardiac and pulmonary outcomes. Thoracic epidural analgesia in high-risk patients has been shown to result in earlier return of bowel function and reduced risk of postoperative pulmonary complications. However, more recently some meta-analyses have reported no differences in mortality or morbidity when compared to other techniques of analgesia suggesting that giving thromboprophylaxis and adopting less invasive surgery may decrease the risk. Lower pain sores were reported with peripheral nerve analgesia than systemic opioids; it also facilitated earlier rehabilitation and reduced the length of stay. Studies with paravertebral analgesia have shown lesser pain scores and reduced risk of pneumonia in patients undergoing thoracotomies. Continuous spinal analgesia has shown efficacy for surgeries such as total hip replacement but complications with use of spinal microcatheters have been reported. Intrathecal opioids provide good patient satisfaction and improved analgesia in the first 24 hours. However, the other techniques may be preferred due to side effects such as delayed respiratory depression. The nurses need to be educated about the incidence of side effects; to recognize and treat them since the patients would be treated in wards. Subcutaneous infiltration of local anesthetics has been used for postoperative analgesia but the analgesic efficacy is limited to duration of analgesia of local anesthetic. There has been increasing interest in the use of disposable elastomeric devices that allows infusion of local anesthetics. This allows the surgeons to directly place catheter into wounds at the end of procedure. It also provides reasonable analgesia and reduces the need for opioids and decreases side effects.

In UK, a year long audit to assess the rate of complications following central neuraxial blocks has been completed. The results showed that the incidence of major complications leading to permanent harm was less than previous reports. Most complications, however, were in perioperative adult patients with epidurals. Peripheral nerve catheters have increased in popularity in past decade. It results in early mobilization and some centers allow use on an outpatient basis facilitating early discharge and recovery. Increasing use of ultrasound for placement of nerve blocks over blind technique such as nerve stimulation or surface anatomy have led to increased block success rate and decreased neurological injuries.

What are the various adjuvants that can be used for controlling postoperative pain?

Originally used for treatment of seizures, gabapentin and pregabalin have also been used to treat neuropathic pain

and recently the postoperative pain. They bind to a subunit of voltage gated calcium channels, thus inhibiting calcium influx and preventing release of excitatory neurotransmitters.

Meta-analyses have shown a significant reduction in pain scores and opioid consumption. Side effects include an increased risk of sedation and dizziness, but decreased vomiting and pruritus.

Dexmedetomidine, an alpha-1 agonist, used as a sedative and analgesic agent in intensive care setting, may be associated with reduced opioid consumption and improved analgesia.

Ketamine; a N-methyl-D-aspartic acid (NMDA) receptor blocker; prevents central sensitization, pain sensitivity, attenuates hyperalgesia due to opioid administration, and thereby may improve opioid effectiveness. Ketamine may have preventative analgesic effects and may improve analgesia in patients who are not responding to opioids. When used in subanesthetic doses for postoperative pain management, less morphine consumption and decreased pain score and side effects were reported. Psychotomimetic effects such as hallucinations are minimal or absent in most trials.

What are the barriers to pain management?

An accurate pain assessment is important for optimal pain management. Self report is the most appropriate way of assessing pain but is heavily dependent on the patient's understanding of pain. Assessing patient's fears of taking analgesics and restrictions due to side effects is also important. Some studies have shown that nurses tended to doubt what patients say about their pain and overestimate the percentage of patients who over-report their pain. Most often they thought of the pain as 'imaginary' or 'psychological' when pain medications did not work. Also some studies show that there are differences between what nurses say and what they actually do. Therefore, careful assessment of pain is important since pain is a subjective experience and there can be a variation in practice. Patients may also not volunteer information about pain since they are reluctant to interrupt busy nurses to request analgesics. Under reporting of pain may be due to the fear of side effects.

To administer analgesia, it is important to assess pain accurately, to choose the most appropriate drug and route and be able to evaluate its effect. Studies indicate that patients do not receive all available analgesia despite patients being in pain. It may be due to knowledge deficits, attitudes or barriers including other priorities affecting effective pain management. Knowledge deficits regarding pharmacology, risk and likelihood of side effects and addiction are common. Reluctance to administer opioids based on negative attitudes including fear of addiction or side effects often leads to ineffective pain management. Also fear of side effects and addiction may be reinforced by staff reluctance.

Managing postoperative pain can be difficult with busy hospital wards, low staff numbers, limited time, inappropriate attitudes and inadequate knowledge which impede optimal postoperative pain management. This may be mitigated by producing an environment where pain management is considered a priority by introducing regular and accurate pain assessment, a multimodal treatment approach and a focus on responding to individual patient's needs.

There is varying evidence surrounding the benefits of preoperative education. It may increase patient satisfaction and participation. It is also part of quality of care to ensure that patients received the information they need to make decisions and to participate in their recovery .Therefore patient education may be considered an important part of perioperative care.

How will you assess pain in different patient groups?

Pain assessment can be more complex, in patients with dementia, learning difficulties, elderly population and patients on long-term opioids for chronic pain or for recreational use.

What is special caution in elderly patients?

The existence of coexisting diseases and complex medication regimes may contraindicate some types of analgesics. Age related pharmacodynamic and pharmacokinetic changes may affect analgesic choices. Fear of giving opioids due to respiratory depression in patients is seen.

Cognitive impairment may prevent the elderly patients from using PCA but the misconception that they cannot cope with PCA may be assumed by staff in more patients than is actually the case. There is a need for simple assessment tools such as a verbal rating scale, available in large print and objective assessment tools for patients with severe cognitive impairment. Careful choice of treatments including a multimodal approach, careful titration of opioids with regular evaluation is required.

How will you manage opioid dependent patients?

Patients who are opioid dependent require a multidisciplinary approach with appropriate teams such as drug dependency units, general practitioners and pain teams. Current recommendations include the use of local anesthetics, multimodal analgesia and appropriate doses of opioids to provide analgesia and to prevent withdrawal. It is necessary to

aim to match the baseline opioid requirement through either continuation of their current regime with additional doses to treat the surgical pain, or using a background infusion on PCA with higher bolus doses. Patients may present with fears that staff may withhold analgesia or may wish to avoid opioid use in the fear of relapsing; therefore communication with the patient is essential.

Predicting postoperative pain

Studies have focused on identification of predictors of pain so that early intervention may improve the pain management. Preoperative pain, anxiety, young age, obesity, surgical fear, type of surgery (abdominal, thoracic surgery, long duration) have all been identified as strong predictors. Preoperative depression is also thought to be associated with increased pain.

Describe the latest advances in pain management?

Analgesic drugs currently used for pain management often cause side effects, therefore driving the search for new drugs or options which are more efficacious and with fewer side effects, especially around new drugs which act on peripheral opioid receptors but do not cross the blood brain barrier and therefore have less central adverse effects.

Sustained release or extended release formulations of conventional drugs prolong the duration of analgesia to longer than 24 hours. Extended release local anesthetics have the local anesthetic drug encapsulated in biodegradable forms like liposomes, lipospheres, and polyglycolic acid microspheres. They prolong the duration of action of local anesthetics. Extended release formulation of epidural morphine which is enclosed in microscopic lipid based particles provides postoperative pain relief for 48 hours after single dose. Targinact is a combination of oxycodone and naloxone which has recently been launched. The addition of naloxone blocks the action of opioid receptors in the gastrointestinal system therefore reducing constipation, but does not reduce the analgesic effectiveness due to its extensive hepatic first pass metabolism. Methylnaltrexone bromide and alvimopan are peripherally acting μ-opioid antagonist which reverse some of the side effects of opioid drugs like postoperative ileus without affecting analgesia. Iontophoretic transdermal delivery of fentanyl uses low intensity direct current to allow rapid transfer of fentanyl from patch into skin and local circulation.

How do you ensure that the patient has adequate postoperative pain relief?

Ability for improving pain management and ensuring that pain is well-controlled is a shared responsibility. Patients should be encouraged to understand that they have a responsibility to tell health practitioners about their pain. Medical professionals should understand the difficulty of patients to communicate about their pain but having an understanding of this can encourage patients. Nurses should take responsibility to assess pain regularly and respond by giving appropriate treatments and reviewing its effects. Surgical teams should fully and accurately assess the patient's pain to ensure that no complications have arisen postoperatively and to ensure that pain is being treated adequately. Anesthetists are required to treat pain intraoperatively and ensure that adequate postoperative pain relief is prescribed. The APS team should ensure that education, guidelines, support and advice are provided for patients and staff with appropriate communication, collaboration and cooperation between healthcare professionals to optimize pain management

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39 Pharmacokinetic Principles in Anesthesia

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Introduction

Anesthesia principally consists of administration of drugs to produce desired pharmacodynamic endpoints which in turn depend on the dose of the drug administered and the pharmacokinetics of the drug. Pharmacokinetics is series of processes which determine the concentration of the drug both in blood and at the effect site at any time.

Pharmacokinetics

Pharmacokinetics was originally defined as the quantitative study and mathematical analysis of drug and its metabolite in the body. Simplistically, it means what body does to the drug, which includes processes such as drug absorption, distribution, metabolism and excretion in numerical terms. It is the change in the concentration of drug over time. The knowledge of pharmacokinetics is fundamental for every anesthetist as the pharmacokinetics constants help in determining the loading dose of the drug, rate of infusion to maintain the steady state concentration in plasma, predict the rate and extent of drug elimination, and predict modification of drug dosage in hepatic and renal impairment, which is in turn help in tailoring the anesthetic drug to a patient's requirements.

In this chapter, the following pharmacokinetic variables and principles will be discussed with examples where appropriate and their practical application in practice of anesthesia.

1. Volume of distribution
2. Compartment models
3. Half life
4. Steady state (Context sensitive half time)
5. Clearance
6. First order kinetics
7. Zero order kinetics.

Pharmacodynamics

Pharmacodynamics describes the actions produced by the drug on the body. Thus, the effects of any drug results from a combination of its pharmacokinetic and pharmacodynamic characteristics in an individual.

Pharmacokinetic Pathway

The passage of a drug through the body occurs through three distinct phases: uptake, distribution, and elimination.

Uptake: The rate of drug uptake and the amount of drug delivered to the target or effect site depends on the route of administration. Intravenous (IV) administration of a drug results in the entire dose entering the plasma immediately; it then passes through the pulmonary circulation into systemic circulation. Some drugs have significant uptake by the lungs e.g. Fentanyl.

Enteral administration requires the drug to cross the intestinal wall. The rate of absorption depends on surface area, pH, and, in some drugs, active systems. In general, unionized drugs (e.g. ethanol) are well-absorbed throughout the intestine; absorption of weak acids (e.g. aspirin) is facilitated by a low pH and weak bases (e.g. morphine) by a high pH. For drugs that remain completely ionized throughout the gut (e.g. glycopyrrolate), passive GI absorption is negligible. Even after GI absorption, a drug may not reach the systemic circulation due to the following reasons:

- Metabolism can occur in the gut mucosa (e.g. dopamine)
- First pass metabolism in the liver via the portal vein (e.g. propranolol).

The degree to which an administered drug reaches the systemic bloodstream is termed as its bioavailability. Bioavailability of drug administered intravenously is 100% shown in Fig. 39.1.

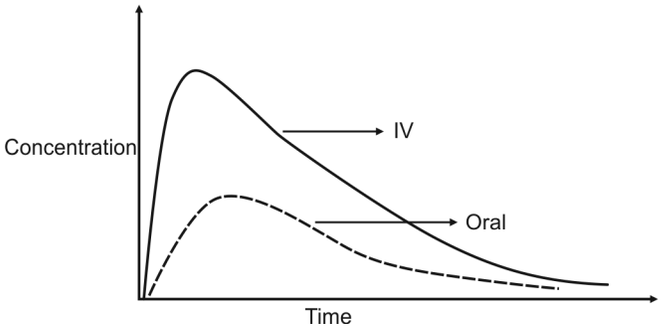

Fig. 39.1 Oral bioavailability. Same dose administered IV and orally on separate occasions. Oral bioavailability = Area under curve oral/ Area under curve IV

Uptake after intramuscular or subcutaneous administration is primarily dependent on local blood flow rather than ionization or lipid solubility. The transdermal route is used for highly lipid-soluble drugs (e.g. Nitroglycerine, Fentanyl), where slow absorption eventually produces sustained blood concentrations.

Inhaled anesthetics have a different pattern of uptake. The inhaled anesthetic crosses the alveolar membrane into the blood along its partial pressure gradient. This produces an exponential wash-in, until equilibrium (i.e. the partial pressure in blood equals that in the inspired and expired gas) when no further uptake occurs. Desired clinical effect requires adequate partial pressure in the tissues and this is achieved rapidly by:

- High fractional concentration of inhaled drug
- High minute ventilation
- Low blood gas partition coefficient, i.e. smaller number of molecules is needed in solution to exert a partial pressure.
- A low cardiac output can also accelerate the uptake. This occurs as the circulating volume is contracted with reduced perfusion to areas outside the vessel-rich group resulting in decreased dosage of drug needed to be taken up from the alveoli.

Distribution: Following uptake; based on the characteristics of the drug; it is distributed to various tissues.

Volume of distribution (Vd): It is the apparent, i.e. not real volume in which the drug is distributed to give a particular concentration. In practice, we measure the concentration of the drug in the plasma. It is an estimate of the extent of the distribution of the drug through body fluids and uptake by tissues. If the volume of distribution is more than the plasma volume, one can safely assume that the drug has left the intravascular or plasma compartment.

$$Vd = \text{dose/concentration}$$

Lower the plasma concentration higher the volume of distribution. For example, if the amount of drug administered is 10 mg to result in a concentration of 2 mg/L the volume of distribution should be 5 liters.

- If a drug has Vd around 5 L, one can assume that the drug remains in the intravascular compartment. They are highly protein bound and as a result the plasma proteins bind to the drug and retain them in the plasma compartment. For example, warfarin, phenytoin.
- If the Vd corresponds to extracellular fluid water 20 L, one can assume that the drug is probably lipid insoluble and hence cannot cross the tissue cell membrane. For example, Muscle relaxants.
- If the Vd corresponds to total body water 42 L, one can assume that the drug is lipid soluble and hence is able to cross the cell membrane to be distributed in the intracellular water. For example, digoxin, fentanyl, bupivacaine.
- If the Vd is more than the total body water, then presumably the drug is concentrated in certain tissues, e.g. fat and anesthetic agents. In this condition, greater the affinity of the tissue for the drug, more drug will be absorbed by the tissue resulting in a low concentration of the drug in the plasma. This will reflect as a very large volume of distribution. For example, propofol.

Volume of distribution at steady state: (Vdss)—One can maintain a steady plasma concentration (Cpss) if we would continuously replace the amount of drug eliminated from the body every minute i.e. input = output.

Vdss = Total amount of drug in the body (not the dose)/Cpss

Pharmacological compartments: Drugs are not distributed uniformly throughout the body. The rapidity with which a drug reaches a particular tissue is largely dependent on local blood flow, and for the purpose of analysis, similar tissue types are often grouped together into various 'compartments' depending on their blood supply. The capacity of each compartment to act as a reservoir for the drug is determined by a combination of its size and affinity for the drug.

It is important to note that pharmacokinetic compartments are mathematical models and do not correspond to actual tissues; they aid in predicting the pharmacokinetic behavior of drugs.

When performing mathematical modeling, it is likely that a lipid-soluble drug that is widely distributed is likely to have several compartments; a highly ionized drug that remains in the extracellular space is likely to be best described by assuming a one-compartment model. An example of a three-compartment model is shown in Fig. 39.4; these correspond to vessel-rich, intermediate, and vessel-poor tissues, with a central compartment (blood), through which drugs must pass during uptake or elimination.

The movement of drug between compartments is dependent on the concentration difference between them, as the process is exponential, the rate of transfer to the vessel poor tissues decreases as they accumulate more drug.

Compartment models: It is essential to understand some terminologies to make it easier to comprehend the compartment models. The following may be used in the equations of various compartment models.

e is a constant which represents mathematical interpretation of exponential changes in which the rate of change in a variable is proportional to its magnitude. Absorption, distribution and elimination of drugs result in exponential changes in drug concentration with time.

For exponential growth, i.e. increase in drug concentration the equation is $X = X_0e^{kt}$ and for exponential disappearance, i.e. decrease in drug concentration the equation is $X = X_0e^{-kt}$, where X is the concentration at any time t, X_0 is the initial value of X at zero time, **e** is the base of natural logarithms (2.718) and **k** is a constant.

The constants k12, k21, k13 and k31 represent the transfer of drug between the central and the peripheral compartments (1, 2, 3). k10 is the elimination rate constant.

One compartment model (Fig. 39.2): Assumes that the body behaves like one volume and there is one clearance.

The concentration of the drug in the plasma at any one moment may be calculated by the following equation.

$$\chi = \chi_0 * e^{-kt}$$

Where χ is the plasma concentration at the time t.

χ_0 is the initial plasma concentration or concentration at time zero.

k is the elimination rate constant and it is negative because the concentration is falling.

It is simple to understand however it has theoretical and practical limitations and is not directly applicable to most intravenous anesthetic agents.

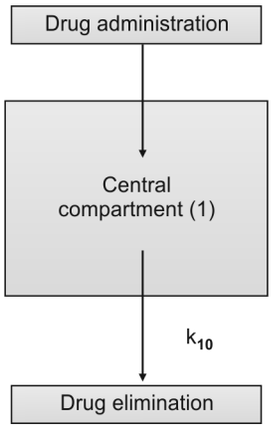

Fig. 39.2 One compartment model

Two compartment model (Fig. 39.3)

After the initial intravenous bolus injection there is a biphasic decline in plasma concentration. This initial decline is due to distribution or rapid disposition of the drug. After the distribution, equilibrium is established between the two compartments. This phase is followed by a slower decline in plasma concentration, which is due to elimination or terminal or slow disposition from the central compartment and is dependent on the rate constant k_{10}.

The plasma concentration Cp at time **t** can be expressed by the equation.

$\chi = Ae^{-\alpha t} + Be^{-\beta t}$ (where α and β are the slopes of α phase and β phase respectively i.e. elimination rate constant). Both α phase and β phase have characteristic slopes and half lives $t_{1/2\,\alpha}$ and $t_{1/2\,\beta}$. The intercepts on the Y axis (A and B) represent the amount each half life contributes to the decrease in plasma concentration after the initial IV dose. The two compartment model consists of a small central compartment into which the drug is administered and eliminated and a

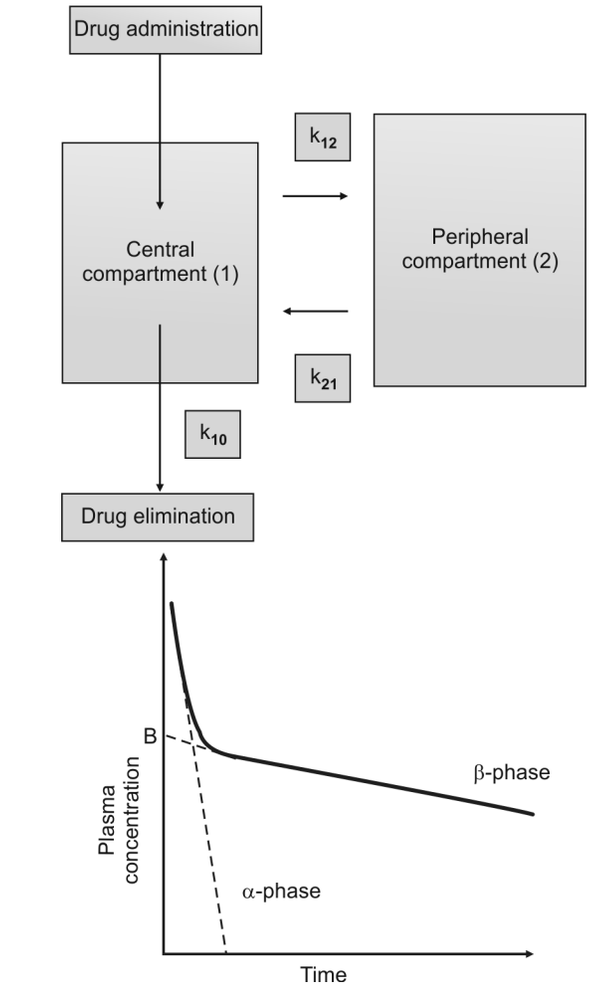

Fig. 39.3 Compartment model and its biphasic decline

larger peripheral compartment. The rate of drug transfer between compartments is governed by constants elimination is governed by the rate constant k_{12} and k_{21} and the rate of elimination is governed by the rate constant k_{10}. The central compartment in case of two compartment model consists of extracellular fluid and some intracellular fluid of highly or well-perfused organs and the peripheral compartment consists of less perfused tissues such as skeletal muscle, fat. Example of drug which follows two compartment model IV lidocaine.

Three compartment model

The three compartment model consists of a small central compartment into which the drug is administered and two peripheral compartments. One of the peripheral compartments is relatively small and represents well-perfused tissues and reflects the initial fall in plasma concentration (α phase) and the other is relatively large and represents less perfused tissues and corresponds to the slow decline in plasma concentration (β phase). The rate of drug transfer between the compartments depends on the respective rate constants (k_{12}, k_{21} and k_{13}, k_{31}) and the rate of elimination is governed by the rate constant k_{10}.

There are 3 parts of the graph in Figure 39.4:

A—The initial steep fall in the plasma concentration (Cp). This phase is explained by the uptake of the drugs by richly perfused organs—heart and brain.

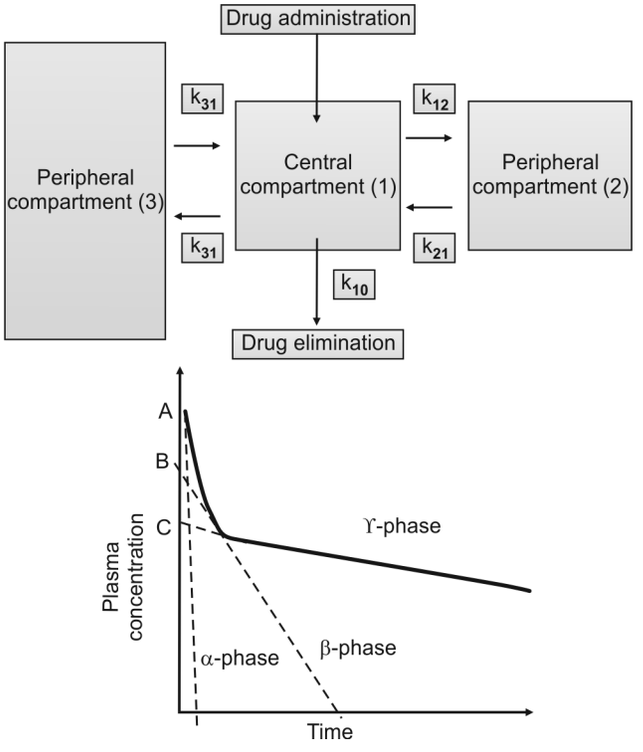

Fig. 39.4: Three compartment model and its triexponential decline

B—The sustained slow fall in the Cp. This is explained by two processes that are occurring simultaneously.

- The Cp continues to fall as the drug is taken up by intermediately perfused organs—e.g. the muscle.
- The brain and heart are fully saturated with the drug and do not take up any more drugs from the plasma. The Cp now falls, though slowly (the curve is less steep) as the muscle takes up the drug. As the Cp decreases relative to the brain and the heart concentration, the drug returns to the plasma from the brain and heart, which tends to maintain the Cp, and reduce the steepness of the curve.

C—This part is almost a straight line. This reflects that the drug is eliminated from the body at a steady rate, because of a constant half-life. The rate of fall in Cp is influenced by:

- Metabolism and elimination of the drug.
- The return of the drug into the plasma from the tissues tends to reduce the fall in Cp. As the drug is eliminated, the Cp falls below the levels in the tissues and the drug returns from the tissues due to a concentration gradient

The plasma concentration Cp at time **t** can be expressed by the equation

$X = Ae^{-\alpha t} + Be^{-\beta t} + Ce^{-\gamma t}$ (α, β and γ are the slopes of α phase, β phase and γ phase respectively, i.e. elimination rate constant). Both α phase, β phase and γ phase have characteristic slopes and half lives $t_{1/2\,\alpha}$, $t_{1/2\,\beta}$ and $t_{1/2\,\gamma}$.

Example: IV bolus dose of propofol

Elimination

Exponential process: Pharmacokinetic processes occur at a rate proportional to the concentration gradient at the time. As the process continues, the concentration gradient falls, thus progressively slowing the rate of change. This results in an exponential relationship between concentration and time and applies to most drug elimination and transfer between tissues. There are two ways in which an exponential function can be described (Fig. 39.5). If a specified time period is set, the decline is defined as the fraction by which

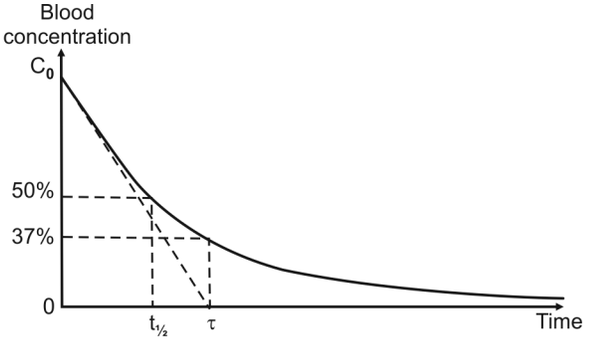

Fig. 39.5: Exponential decline of plasma concentration with time. C_0 initial concentration; $t_{1/2}$, half-life; τ, time constant

the concentration has been reduced during this interval. This is the elimination rate constant (k), expressed as time^{-1}. Alternatively, a fractional reduction in concentration is set, and the time taken to achieve this level is found.

For example, for a 50% reduction in concentration, the time taken is the half-life ($t_{1/2}$) or terminal half-life or plasma half life. It is the time taken for the plasma concentration to decrease by 50% during the terminal phase of decline. This will be constant irrespective of the starting concentration of the drug.

$$t_{1/2} \alpha \frac{Vd}{CL} = k \times \frac{Vd}{CL}$$

where Vd is the volume of distribution, CL is the clearance and k is the constant. $t_{1/2}$ depends on the Vd and CL. A prolonged elimination half life indicates a larger Vd or reduced clearance or both. A shorter half life indicates an increased clearance or low Vd or both.

Clinical application of terminal half-life

The plasma concentration starts rising once drug is administered. Sustained therapeutic effect is seen only when the Cp reaches a steady state. The Cpss is reached after 5 terminal half-lives. For example, if a drug has a half-life of 1hr, then after the fifth dose, at 5th hr, the plasma concentration would have reached a constant level (Figs 39.6, 39.7A and B).

In general, the dosing interval is a compromise between decline in drug concentration and the inconvenience of very frequent administration particularly with oral medications.

For anesthetic drugs like muscle relaxants; there is an approximate relationship between the half life and the duration of action, e.g. suxamethonium has a half life of 3–5 minutes and is a very short acting muscle relaxant whereas rocuronium has a plasma half life of 70–140 minutes and is an intermediate acting muscle relaxant.

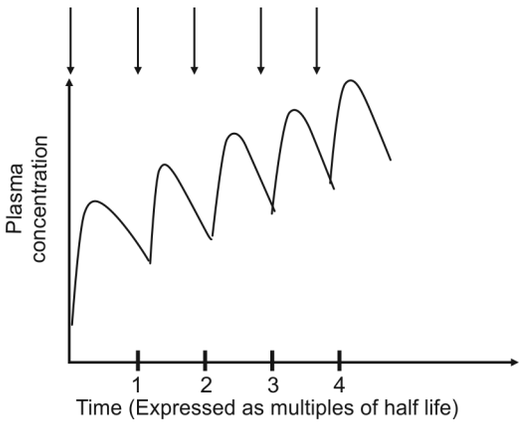

Fig. 39.6 Cumulation of drug when it is administered (red arrows) at interval equal to the half life

The relationship between terminal half life and duration of action may not be present with other drugs. For example:

1. Drugs that cross the blood brain barrier for their clinical effect. For example, opioid analgesics, morphine which has a shorter terminal half life compared to pethidine; actually has a longer duration of action.
2. Likewise for drugs when their effect is terminated by redistribution rather than elimination. For example, IV barbiturates, propofol, fentanyl, alfentanil.

The latent period before a steady state can be avoided by administration of loading dose equal to twice the normal dose for oral drugs. Similarly, when drugs are administered intravenously as continuous infusion, steady will be reached only after 4–5 terminal half lives. This steady state can be reached earlier by administration of an IV bolus followed by continuous infusion.

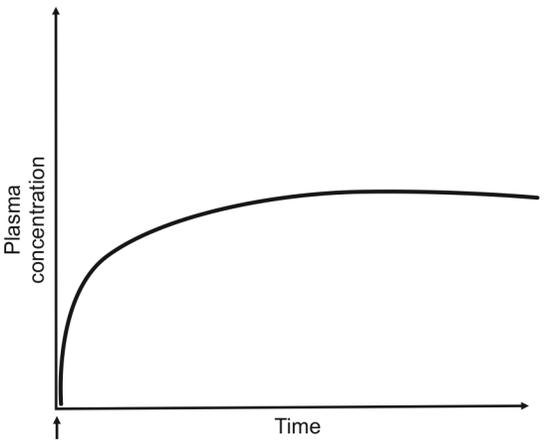

Fig. 39.7A Cumulation of drug following IV infusion (red arrows represent drug administration)

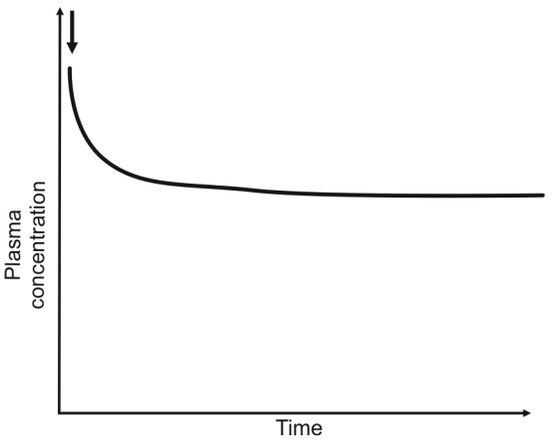

Fig. 39.7B Cumulation of drug after an IV bolus followed by IV infusion (red arrows represent drug administration)

Another time period that can be used to describe the curve is the time constant (τ). It is defined as the time taken to complete the elimination process if the initial rate of decline in concentration continued. If the initial rate of decline continued; the plasma would be cleared of 63% of the drug in one time constant. Alternatively, the plasma concentration would be 37% of the original value at the end of one time constant.

Context Sensitive Half Time (CST)

When drugs are given by infusion their terminal half lives are inaccurate indicator of drug disposition and duration of action. In these situations, context sensitive half time is used. It is defined as the time required to decrease the plasma concentration by 50% at the end of an infusion designed to maintain steady state concentration. The context here is the infusion to maintain steady state concentration. It provides a useful guide to dosage intervals for long-term drug maintenance.

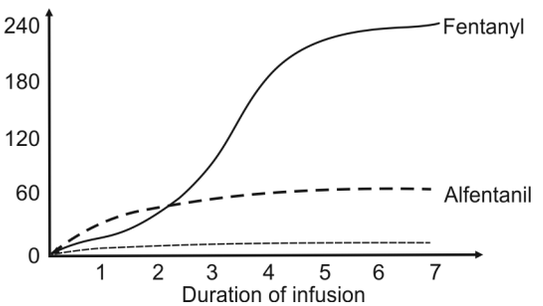

Fig. 39.8 Context sensitive half times of fentanyl, alfentanil and remifentanil

For drugs such as fentanyl, in which redistribution is the main mechanism responsible for the decline in plasma concentration after a brief infusion or bolus, the CST will initially be short (Fig. 39.8). As the duration of infusion continues, redistribution becomes progressively less important and the CST increases, until ultimately it equals the elimination half-life. For a drug with a small volume of distribution, such as remifentanil, redistribution is very limited and the CST changes little even with prolonged infusion.

The amount of drug cleared from the body is given by the equation:

$$O = Cp \times CL$$

O is the amount of drug eliminated from the body every minute. If we infuse the same amount of the drug cleared from the body every minute, then the Cp would be maintained constant, i.e. at steady state (Cpss)

$$I = (O) = Cpss \times CL$$

Cpss = I/CL where I is the amount of the drug infused every minute.

At steady state, the relationship between the drug concentration and the rate of drug delivery is independent of Vd because it is governed only by total body clearance:

Rearranging the above equation:

Rate of infusion = concentration at steady state × clearance.

To summarize, the loading dose is determined by the volume of distribution and the maintenance dose by the clearance Cl of the drug.

Nonlinear Pharmacokinetics

When drug behavior is analyzed by pharmacokinetics models, it is assumed that the distribution and elimination are first order process or kinetics.

First order kinetics implies that the rate of transfer of drug from one compartment to another is proportional to the drug concentration, i.e. the amount of drug being removed is a constant fraction in unit time rather than a constant amount. This is not always the case, as drug distribution and elimination may be dependent on carrier transport and can get saturated. Similarly, many reactions with drug metabolism are saturable and proceed at a maximal rate if the drug concentration is high. Once the relevant blood concentration is reached, elimination becomes constant, limited to a maximum amount in unit time. In these conditions, the drug transport and metabolism occur at a high but a constant rate irrespective of the concentration of the drug, i.e. a constant proportion of the drug is cleared. This is termed as nonlinear pharmacokinetics or zero order kinetics and occurs with capacity limited elimination of drugs.

This is not a common phenomenon, however can occur in dangerously high concentrations with continued, unmonitored drug administration or prolonged infusion e.g. ethanol, salicylates, phenytoin. The elimination of drugs occurs at a constant rate irrespective of the drug concentration and when the concentration decrease to a certain level, i.e. the systems are not saturated anymore then the kinetics changes from zero order to first order.

Clearance (CL): It is defined as volume of blood or plasma from which the drug is completely cleared in unit time (mL/min). It is also defined as the rate of drug elimination (mg/min) per unit of blood or plasma concentration (mg/mL). A drug can undergo clearance by the kidneys, liver or other tissues. Total clearance is the sum of different ways of drug elimination through various organs in the body.

$CL = CL_R + CL_H + CL_\chi$ where CL_R is renal clearance, CL_H is hepatic clearance and CL_χ is clearance by other routes.

The clearance of a drug through an organ (renal or hepatic) depends on:

- Extraction ratio (ER)
- Organ plasma flow (Q)

$$CL = ER \times Q$$

Renal clearance can be directly measured or calculated by - CLR = dose of drug in the urine/AUC where AUC is the

Area **U**nder the plasma concentration-time **C**urve during the elimination phase. If the ratio of renal clearance to the total clearance of the drug is over 70% then the drug is predominantly eliminated by kidneys from the body. It implies that the drug will cumulate in renal failure, whereas if the CLR is less than 30% renal impairment has little or no impact on elimination.

Hepatic clearance—the extraction ratio is an overall ability of the liver to remove drug from the hepatic capillaries and reflects drug metabolism and biliary secretion.

$$ER = \frac{Ca - Cv}{Ca} = 1 - \frac{Cv}{Ca}$$

Where Ca is the concentration of drug in the mixed portal venous and hepatic arterial blood, Cv is the drug concentration in the hepatic venous blood. Elimination of the drugs by liver is dependent on:

1. Hepatic blood flow
2. Proportion of unbound drug in the plasma
3. Activity of the hepatic enzymes

$$CL_H = ER \times Q$$

It is also expressed in terms of blood flow and intrinsic clearance. Intrinsic clearance is the maximal ability of liver to irreversibly eliminate drugs by metabolism or biliary excretion and is independent of blood flow

$$CL_H = Q \times \frac{(CLi \times f)}{Q + (CLi \times f)} \qquad \text{Equation 1}$$

Where CLi is the intrinsic clearance, which represents the rate at which liver water is cleared of the drug (ml/min), *f* is fraction of unbound drug in plasma.

When the intrinsic clearance is relatively low compared to the blood flow then:

$Q + (CLi \times f) = Q$ (Subtituting in Equation 1)

$$CL_H = Q \times \frac{(CLi \times f)}{Q} = CLi \times f$$

In these circumstances, hepatic clearance is dependent on the intrinsic clearance and the unbound fraction of the drug in plasma. This is termed as capacity limited elimination e.g. phenytoin, warfarin, barbiturates and benzodiazepines. These drugs have limited first pass effect and after oral administration and low hepatic extraction ratio. Their clearance is relatively low and will be unaffected by changes in hepatic blood flow, however will be profoundly influenced by the activity of the hepatic enzymes. The unbound fraction of drug can have an effect as well, particularly for drugs with high degree of protein binding.

When the intrinsic clearance is relatively large compared to the blood flow then:

$Q + (CLi \times f) = (CLi \times f)$ (Subtituting in Equation 1)

$$CL_H = Q \times \frac{(CLi \times f)}{(CLi \times f)} = Q$$

In these conditions hepatic clearance is primarily dependent on hepatic blood flow, this is termed as flow limited elimination e.g. opioids analgesics, lidocaine, tricyclic antidepressants. The half life can be modified by alterations in hepatic blood flow, but is not affected by the hepatic enzymatic activity or protein binding. The hepatic extraction is high and there is substantial first pass effect for orally administered drugs.

Practical Applications

Establishing Steady State

- If a drug is given as a constant infusion, it will eventually reach a steady state (i.e. with the whole Vdss containing the drug at a stable concentration, and elimination occurring at the same rate as administration). It takes four to five elimination half-lives to achieve this steady state.
- The target concentration can be attained far more rapidly using an initial loading dose followed by further additional drug in a declining exponential fashion as redistribution to other tissues occurs.

Predictability

- Established pharmacokinetic and pharmacodynamic data are derived from averaged population studies. One must accept individual variation both in pharmacokinetics and pharmacodynamics even with most sophisticated dosage schemes for IV drugs
- In contrast, for inhalational anesthetic agents, although pharmacodynamic variability will still occur, pharmacokinetic behavior is far more predictable, because of the physics of gas/vapor solution in a liquid. Indeed, at equilibrium the partial pressure of an inhalational agent in blood (and other tissues) will precisely equal that in the inhaled gas mixture. Furthermore, the end-tidal partial pressure of an inhalational agent can be measured in real-time, providing a value very close to that in arterial blood.

Suggested Reading

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Section Two

Anaesthesia Equipment and Table Viva

- Arterial Blood Gas Analysis
- Mechanical Ventilation
- Pulmonary Function Tests
- Anesthesia Machine
- Vaporizers
- Anesthesia Breathing Systems
- Endotracheal Tubes, Double Lumen Tubes and Combitube
- Cardiopulmonary Resuscitation
- Airways, Connectors Laryngoscopes and Non-rebreathing Valves
- Electrocardiography
- Interpretation of X-rays
- Supraglottic Airway Devices
- Regional Anesthesia Instruments
- Nerve Locator and Peripheral Nerve Stimulator
- Oxygen Therapy Devices and Manual Resuscitator
- Videolaryngoscopes

40 Arterial Blood Gas Analysis

V Patil, J Doctor

Explain the basic physiological processes reflected in ABG

The ABG provides us with rapid information on three physiologic processes:

1. *Alveolar ventilation:* The maintenance of CO_2 level reflected by arterial CO_2 tension ($PaCO_2$) at any given moment depends on the quantity of CO_2 produced in body and its excretion through alveolar ventilation (VA) and can be expressed by the equation, $PaCO_2 \sim CO_2/VA$. The alveolar ventilation is that portion of total ventilation that participates in gas exchange with pulmonary blood. Thus $PaCO_2$ is the best index for assessment of alveolar ventilation. High $PaCO_2$ (> 45 mm Hg) indicates alveolar hypoventilation and low $PaCO_2$ (< 35 mm Hg) implies alveolar hyperventilation.
2. *Oxygenation:* The ultimate aim of oxygenation is to provide adequate delivery of oxygen to tissues. This is a function of cardiopulmonary system and various factors like arterial oxygen tension (PaO_2), hemoglobin content and saturation with oxygen and cardiac output. The PaO_2 and SaO_2 are primarily used for assessment of oxygenation status. Approximately 98% of oxygen is carried in blood in the combined state with hemoglobin. Hypoxemia is defined as PaO_2 of less than 80 mm Hg at sea level in an adult patient breathing room air PaO_2 must always be interpreted in relation to concentration of inspired oxygen FiO_2 and age. Relation between PaO_2 and FiO_2: PaO_2 alone provides little information regarding efficiency of oxygen loading into the pulmonary capillary blood. In other words, it does not quantitate the physiological shunt, which helps in assessment of the severity of underlying disease in lungs and in guiding oxygen therapy. Since the normal PaO_2 in an adult breathing room air with FiO_2 of 0.2 is 80 to 100 mm Hg, the normal values for PaO_2/FiO_2 ratio or oxygenation ratio are 400–500 mm Hg or 4.0 to 5.0 respectively. PaO_2/FiO_2 ratio of less than 200 most often indicates a shunt greater than 20%.
3. *Acid-base balance.*

How does age affect oxygenation?

$PaO_2 = 104–(0.27 \times age)$ more crudely, normal oxygenation for age is roughly 1/3 of the patient's age subtracted from 100. Using this estimation, a 60-year-old patient should have a PaO_2 of 80 mm Hg.

Explain terms: Shunt and dead space

Shunt: That part of the cardiac output that returns to the left heart without being exposed to ventilated alveoli.

Dead space: That part of inspired air that does not take part in gas exchange. It is normal for a small percentage of air in the lungs not to reach blood. Air in the nasopharynx, trachea and bronchi does not reach the alveoli before exhalation.

Classic shunt equation

$Qs/Qt = (CaO_2–CvO_2) / (CcO_2–CvO_2)$
CcO_2 = End capillary O_2 content
CaO_2 = Arterial O_2 content
CvO_2 = Mixed venous.

VQ ratio

Normal V (ventilation) is 4 L of air per minute.
Normal Q (perfusion) is 5 L of blood per minute.
So, normal V/Q ratio is 4/5 or 0.8.

When the V/Q is higher than 0.8, it means ventilation exceeds perfusion.

When the V/Q is < 0.8, there is a VQ mismatch caused by poor ventilation or excess perfusion as in one lung anesthesia.

When V=0 (collapsed non-aerated alveoli), a shunt is present.

When Q =0 (non-perfused alveolus), dead space is present.

What is alveolar–arterial gradient (A-a gradient)?

It predicts the degree of shunt by comparing the partial pressure of O_2 in the alveoli (A) to that in the artery (a).

The difference between them gives us an idea how well the oxygen is moving from the alveoli to the arterial blood. (PAO_2–PaO_2) The PaO_2 is obtained from the ABG. The PAO_2 (partial pressure of oxygen in the alveolus) is obtained from the Alveolar Gas equation:

$$PAO_2 = (760–47) \times FiO_2–PaCO_2 /R,$$

Where R is the Respiratory Quotient. R is usually 0.8, but may be taken as 1 for ease of calculation.

OR

$$PAO_2 = 150–1.25\ (PaCO_2)$$

A-a gradient = $[150–1.25\ (PaCO_2)]–PaO_2$

A normal A-a gradient is 10–20 mm Hg,

A-a gradient when the patient is not breathing room air is calculated as:

A-a gradient = $[(FIO_2)(760–47)–(1.25)(PaCO_2)]–PaO_2$

The A-a gradient is always widened in parenchymal lung disease. Thus a low PaO_2 with wide A-a gradient suggests lung disease as a cause of hypoxia,while a low PaO_2 with a normal A-a gradient suggests hypoventilation as a cause of hypoxia.

What is the difference between actual and standard bicarbonate?

Actual bicarbonate—The actual bicarbonate is the value calculated from the blood gas sample.

The standard/corrected bicarbonate is the value of the bicarbonate had the sample been corrected to 40 mm Hg and at room temperature. The standard bicarbonate gives an estimate of the metabolic component causing an acid base imbalance.

Base deficit or excess is the amount of alkali or acid that must be added to a solution to restore its pH to 7.4 after it has been equilibrated to a PCO_2 of 40 mm Hg.

The base deficit/excess is the amount of deviation of the standard bicarbonate from the normal.

What are indications for ABG analysis?

In some cases, noninvasive monitoring such as pulse oximetry, transcutaneous gas monitoring, or capnography may be sufficient. In other cases, the procedure may be reconsidered if it is not clear that the results of the test will alter the course of treatment.

Indications

- The need to assess ventilatory status, acid-base balance, and oxygenation and the oxygen-carrying capacity of the blood
- To assess a patient's response to therapeutic intervention like ventilator management, circulatory intervention or the progression of a disease process
- For surgical evaluation (pulmonary resections).

Explain role of kidneys in maintaining acid-base homeostasis.

Organic and mineral acids are products of normal metabolic processes. These are immediately buffered so pH remains stable. Buffers are bicarbonate, proteins and bone.

Kidneys excrete mineral ions (associated with producing these mineral acids) and regenerate the 60–70 meq of bicarbonate lost daily whilst buffering these acids. Kidneys do this by reabsorbing bicarbonate and producing ammonium.

Is it mandatory to have arterial line for ABG sampling?

An arterial line should be placed when multiple blood gas studies (more than 4 samples of arterial blood in 24 hours) are anticipated. For less than 4 samples/24 hours, collecting sample through arterial puncture should be performed but care should be taken to change the puncture site every time as repeated puncture of a single site increases the likelihood of hematoma, scarring, or laceration of the artery.

What are the contraindications for arterial puncture?

- Cellulitis or other infections over the puncture site of artery
- Absence of palpable arterial pulse
- Negative results of an Allen test/ modified Allen test (collateral circulation test)
- Coagulopathies or medium-to-high-dose anticoagulation therapy (e.g. heparin or coumadin, streptokinase, and tissue plasminogen activator but not necessarily aspirin) may be a relative contraindication for arterial puncture
- History of arterial spasms following previous punctures
- Severe peripheral vascular disease
- Arterial grafts.

What is the most preferred artery for puncture?

The radial artery on non-dominant hand is the ideal site for an arterial puncture for the following reasons:

- It is small, but superficial and easily accessible, and stabilized.

- It is easily compressible with better control of bleeding
- There is no nerve nearby to worry about.
- The collateral arch with the ulnar artery minimizes the risk of ischemia to the hand in case of occlusion of the radial artery. This must be tested using the Allen's test.

How will you check for adequacy of collateral circulation? What is Allen's test ?

- Patient elevates hand and makes a fist for 20 seconds.
- Firm pressure held against radial and ulnar arteries.
- Patient opens the hand which should be blanched white
- Examiner releases only ulnar compression.
- Normal result hand color flushes—color returns within 5 to 7 seconds.
- Abnormal result: Delayed or absent hand flushing indicating inadequate collateral circulation.

Alternative means for assessing collateral circulation

- Doppler ultrasound
- Finger plethysmography using a pulse oximeter.

What is modified Allen's test?

This is done before puncturing dorsalis pedis artery. Elevate patient's feet. Occlude dorsalis pedis artery; then blanch the great toe by compressing the toenail for several seconds. Release pressure on the nail and observe for flushing (rapid return of color indicates adequate collateral flow). Compress the posterior tibial artery if it is being used as the puncture site (pediatric population).

Describe procedure for arterial puncture

A comfortable setting is essential to proper technique. The patient should be lying down or sitting with the arm well-supported and the clinician should also be seated if possible. Thoroughly clean the skin around the site using your institutional protocol. A rolled towel positioned beneath the wrist helps hyperextend the site while the pulse is carefully palpated. Note that sometimes palpating too firmly can occlude the artery enough to prevent blood from flowing into the syringe, even though the artery has been penetrated. If the syringe needs to be repositioned, the tip should be withdrawn to the subcutaneous tissue to prevent damaging the artery or tendons with the needle.

Do's

- Communicate with the patient about the purpose of the procedure
- Always perform Allen test prior to drawing blood from a radial artery
- Apply pressure to the site for at least 5 minutes (normal clotting time) or more if patient has prolonged clotting time
- Keep ABG sample in an ice bag sample unless it is going to be analyzed within 10–15 minutes.

Don'ts

- Palpate too firmly, inhibiting blood flow
- Reposition a needle without first withdrawing the tip to subcutaneous tissue
- Ever leave bubbles in an ABG syringe or draw air in before de-airing
- Fail to adequately heparinize a sample to prevent clotting (we get preheparinized syringes for ABG collection. Preferably use these syringes. If unavailable, take a 2 cc syringe flush it with heparin and then use for ABG collection).

List complications of arterial puncture

- Pain
- Bruising and hematoma
- Nerve damage
- Aneurysm
- Spasms
- AV Fistula
- Infection
- Vasovagal response
- Air or thromboembolism
- Anaphylaxis from local anesthestic.

How would you time ABG sample?

To get the correct information about underlying pathophysiology blood sampling must be done during steady state i.e. whenever there is initiation or change in oxygen therapy; or changes in ventilatory parameters with patients on mechanical ventilation. In the patients without overt pulmonary disease a steady state is reached between 3–10 minutes and in patients with chronic airways obstruction it takes about 20–30 minutes after changes have been made to ventilatory therapy.

How does excess heparin affect your ABG report? How will you heparinize the syringe?

Heparin must be added to the syringe as an anticoagulant. Because the pH of heparin is near 7.0, and the PO_2 and PCO_2 of the heparin solution are near room air values, excess heparin can alter all three ABG measurements. Very little heparin is actually needed in the sample to prevent clotting; 0.05 to 0.10 mL of a dilute solution (1000 units/mL) will anticoagulate 1 mL of blood without affecting its pH PCO_2, or PO_2. After

flushing the syringe with heparin, a sufficient amount usually remains in the dead space of the syringe and needle for anticoagulation without distortion of the ABG determination.

Why proper handling of sample is necessary?

Leukocytes and platelets continue to consume oxygen in the sample after it is drawn into a syringe and carbon dioxide continues to be produced. This can cause a significant fall in PaO_2 and rise in PCO_2 over time at room temperature, especially in the setting of leukocytosis or thrombocytosis. It is essential that the ABG sample be analyzed within 10–15 minutes at room temperature immediately be put on ice. An ABG sample can remain stable on ice for at least 1 hour. One may argue that an iced sample can remain stable for up to several hours, but at that point it is no longer representative of the patient's current status and its value as a clinical tool is severely diminished. Failure to cool the sample properly is a common source of preanalytic error.

If air bubbles are not removed immediately, oxygen can diffuse into the sample and compromise the results. Air bubbles that mix with a blood sample will result in gas equilibration between the air and the blood. Room air has a PO_2, of approximately 150 mm Hg (sea level) and a PCO_2 of essentially zero. Thus, air bubbles that mix and equilibrate with arterial blood will shift the PaO_2 toward 150 mm Hg and significantly lower the PCO_2 of the blood sample with subsequent increase in pH.

Correcting patient temperature, once commonly applied to ABG samples, especially in patients on cardiopulmonary bypass is no longer the standard as studies have failed to show much clinical relevance of temperature-corrected PO_2 values.

What are the most common sources of error in ABG?

Accurate results for ABGs depend on collecting, handling, and analyzing the specimen properly. Clinically important errors may occur at any of these steps, but ABG measurements are particularly vulnerable to preanalytic errors (collection, storage and transport of sample). The most common problems include non-arterial samples, air bubbles in the sample, either inadequate or excessive anticoagulant in the sample, and delayed analysis of an un-cooled sample.

How will you choose the site?

Arterial blood samples are normally obtained from adults at the radial, brachial, femoral, or dorsalis pedis arteries. Because radial artery puncture is relatively safe and the site easily accessible as well as convenient for checking collateral circulation, this site is preferable. Before doing arterial puncture one should test for collateral circulation. If collateral circulation is absent, the radial artery should never be used.

The brachial artery is the second choice, as it is relatively large and easy to palpate, and has good collateral circulation; however, it lies deeper and its proximity to the basilic vein and median nerve makes it easy to hit them by mistake. In addition, the lack of underlying ligaments or bone support increases the risk of hematoma following the procedure.

The femoral artery is the third choice because it is relatively easy to palpate and it is sometimes the only site where sampling will be possible; however, it lies close to the femoral vein, poses increased risk of infection, and requires prolonged monitoring after puncture. It should be selected as a last resort and only within a hospital setting.

Dorsalis pedis artery is also a useful site for ABG punctures when a radial artery sample is not obtainable.

How do calculate oxygen content of blood?

Oxygen content can be measured directly or calculated by the oxygen content equation:

$CaO_2 = (Hb \times 1.34 \times SaO_2) + (.003 \times PaO_2)$

What are the normal values in ABG?

Table 40.1 Normal ABG values

Sample	PaO_2 (mmHg)	$PaCO_2$ (mmHg)	pH	HCO_3 mmol/L	Base Excess	SaO_2
Arterial	80–100	35–45	7.36–7.44	22–26	+/–2	94–100
Venous	37–42	42–50	7.34–7.42			

	Normal range
Anion gap	12–16 mmoL/lt
Osmolar gap	10 mmoL/lt
PaO_2/FiO_2 ratio	> 300

What are the different methods of analyzing acid base disorders?

There are 2 methods of evaluation of acid base disorders:

1. Classic or traditional approach.
2. Stewart approach.

1. The classic or traditional bicarbonate based approach essentially revolves around the Henderson Hesselbach formula $pH = pK + [\log (HCO_3)/PaCO_2]$
   Respiratory disorders are due to a change in PCO_2 whereas metabolic disorders are due to alterations in bicarbonate. Based on this approach acid base disorders are classified into 6 primary disorders
   - Metabolic acidosis (High anion gap/ Normal anion gap)
   - Metabolic alkalosis

- Acute and chronic respiratory acidosis
- Acute and chronic respiratory alkalosis.

2. The Stewart approach on the other hand is independent of the bicarbonate.
   - Its variables are pCO_2, strong ion difference (SID) and Atot (Total weak acids)
   - Respiratory disorders are due to alterations in pCO_2 similar to the traditional approach
   - Metabolic disorders are due to primary alterations in strong ion difference (SID) and Strong ion gap (SIG) or Atot
   - The cations are Na^+, K^+, Ca^{++} and Mg^{++}
   - The anions are Cl^- , Lactates and other strong ions
   - SID apparent SIDa= (Na+k+Ca+Mg)–(Cl) –Lactate-other strong ions
   - (Na + k + Ca + Mg) – (Cl) Normal strong ion difference is about 40 mEq/L
   - SID effective SIDe = (HCO_3) + (A^-) where A^- is the total concentration of dissociated weak noncarbonic acids, mainly albumin and PO_4^-
   - Strong ion gap (SIG) or Atot = SIDa- SIDe. Normal Strong ion gap is zero.

   Atot represents all non bicarbonate buffer pairs primarily albumin, inorganic phosphate, hemoglobin.
   Atot = [HA] + $[A^-]$

Table 40.2 Stewart approach

Parameter		Acidosis	Alkalosis
Respiratory		↑PCo2	↓PCo2
Non respiratory (Metabolic)			
SID	Abnormal SID	↓SID	↑SID
	Anion excess	↓SID ↑ Lactates	
Atot (non- volatile weak acids)	Albumin	↑	↓
	Inorganic phosphate	↑	↓

What is compensation? State the rules of compensation and expected compensation in various disorders.

Maintenance of acid-base balance is normally accomplished by buffer systems which react to changes in the hydrogen ion concentration of body fluids. When there is an imbalance in one parameter (primary disturbance), the other moves in the opposite direction to minimize the impact of the primary disturbance on the pH.

Rules of compensation:

1. The compensatory response depends upon the proper functioning of organ system involved in the response (lungs or kidneys) and on the severity of acid-base disturbance.
2. Acute compensation in case of kidneys occurs within 6–24 hours and chronic within 1–4 days. Respiratory compensation occurs faster than metabolic compensation.
3. In clinical practice, it is uncommon to see complete compensation. The maximum compensatory response in most of the cases is associated with only 50–75% return of pH to normal.
4. Overcompensation never occurs.

Expected compensation for simple acid-base disorders

1. Metabolic acidosis: Expected $PaCO_2$ decreases by 1–1.3 mm Hg for ever 1 meq/L decrease in HCO_3 or expected $PaCO_2$ = last two digits of pH.
2. Metabolic alkalosis: Expected $PaCO_2$ increases by 0.5–0.7 mm Hg for every 1 meq/L increase in HCO_3 or expected $PaCO_2$ = last two digit of pH.
3. Respiratory acidosis: In acute acidosis, HCO_3 increases by 1 meq/L for every 10 mm Hg increase in $PaCO_2$. In chronic acidosis, the acidosis, the HCO_3 increases by 3.5 meq/L for each 10 mm Hg increase in $PaCO_2$.
4. Respiratory alkalosis: In acute alkalosis HCO_3 decreases by 2 meq/Lfor every 10 mm Hg decrease in $PaCO_2$. In chronic alkalosis HCO_3 decreases by 5 meq/L for every 10 mm Hg decrease in $PaCO_2$.

How do you determine if compensation is present, and to what extent?

When compensation is present, we will see two imbalances. The question then becomes, which is the primary problem, and which imbalance is due to compensation. The clue is the pH. If the pH is leaning toward acidosis or alkalosis, then the parameter with the matching imbalance is the primary problem, and the other is due to compensation. Also knowing patients proper history will help us identify the primary pathology.

Single acid-base disorders do not lead to normal blood pH. Although pH can end up in the normal range (7.35–7.45) with a single mild acid-base disorder, a truly normal pH with distinctly abnormal HCO_3- and $PaCO_2$ invariably suggests two or more primary disorders.

What are the types of respiratory failure and causes?

Type I respiratory failure; is also known as hypoxemic failures and type II is called hypercapneic failure.

Table 40.3 Types of respiratory failure

Type I	Type II
Atelectasis	CNS depression (drugs/sleep/head injury)
Pulmonary edema (cardiogenic/non cardiogenic)	High spinal cord lesion
Pneumonia	Phrenic nerve lesions
Pleural effusion	Neuromuscular disorders
Hemopneumothorax	Severe kyphoscoliasis
	COPD
	Type I causes in advanced states

How is metabolic acidosis produced?

Metabolic acidosis is produced when:

1. Acid production is in excess of kidney ability to excrete acid and regenerate bicarbonate.
2. Increased loss of bicarbonate from extracellular fluid (e.g. GIT loss or renal).
3. Renal pathology causing reduction in ability to excrete acid and regenerate bicarbonate.

What do you understand by anion gap?

Anion gap is difference between measured cations and measured anion. There is no difference in actual cations and actual anions. Body fluids including blood may contain a variable number of ions, but the total number of anions (negative ions) and cations (positive ions) are roughly the same. The ions that are usually measured in blood are cations like sodium and potassium and anions including chloride and bicarbonate. There are unmeasured ions in both groups (cations and anions), which also contribute to the ionic constitution of blood. The measured cations are usually greater than the measured anions by about 8–12mmol/L. This is because the unmeasured anions constitute a significant proportion of the total number of anions in blood. Proteins make this up predominantly, but also included are sulfates, phosphates, lactate and ketones.

Anion gap = $(Na + K)–(Cl + HCO_3)$ Normal AG is 14-16 if we include potassium.

Also, anion gap = $(Na)–(Cl + HCO_3)$. Normal AG is 8–12 if we do not include potassium.

What are the causes of metabolic acidosis?

High anion gap: Use mnemonics—KULT or MUDPILES

1. Ketoacidosis—diabetic
   a. Starvation
   b. Alcoholic (ethanol)
2. Lactic acidosis
3. Uremia
4. Toxins
   a. Methanol
   b. Ethylene glycol
   c. Propylene glycol
   d. Salicylates
   e. Paraldehyde.

Normal anion gap acidosis

1. Hypokalemic
   a. GI losses of HCO_3
      i. Ureteral diversion
      ii. Diarrhea
      iii. Ileostomy
   b. renal losses of HCO_3
      i. proximal RTA
      ii. carbonic Anhydrase inhibitors
2. Normokalemic or hyperkalemic
   a. Renal tubular disease
      i. Acute tubular necrosis
      ii. Chronic tubulointerstitial disease
      iii. Distal RTA (type I and IV)
      iv. Hypoaldesteronism
   b. Pharmacological
      i. Ammonium chloride
      ii. Hyperalimentation
      iii. Dilutional acidosis.

Decreased anion gap acidosis

1. Hypoalbuminemia
2. Paraproteinemia (multiple myeloma)
3. Spurious hypercholeremia
4. Bromide intoxication
5. Spurious hyponatremia
6. Hypermagnesemia.

What is corrected anion gap?

Critically ill patients generally are hypoalbuminemic and hypophosphatemic. Albumin is a major contributor for anion gap. In the presence of low serum albumin, anion gap is reduced by approximately 2.5 mEq/L for every 1g/dL fall in albumin. Alternatively a corrected value for a normal anion gap (assuming K is included in calculation of anion gap) can be obtained from:

Corrected normal anion gap = 0.2 (albumin) × 1.5 (phosphate) where albumin is in g/dL and phosphate in mmol/L.

Also, corrected anion gap = Observed anion gap + 2.5 x (Normal albumin–observed albumin)

Normal albumin is taken as 4.4 g/dL

How do you treat metabolic acidosis?

One should always be cautious when interpreting any result. The aim should not be to correct a number but the underlying abnormality that is represented by abnormal number. Metabolic acidosis points towards a specific underlying pathology which needs to be adressed. For example raised lactic acidosis mostly suggests a hypoperfusive state. So treatment should be directed towards improving perfusion by optimizing fluid status and using inotropes rather than giving bicarbonate.

Indiations for bicarbonate therapy in acidosis.

1. pH < 7.2 and bicarb < 5 mEq/L.
2. Patients with bicarbonate loss(normal anion gap acidosis).
3. In raised AG acidosis, treat the underlying pathology.

Complications of bicarbonate therapy

- Volume overload
- Hypernatremia
- Hyperosmolarity
- Hypokalemia
- Intracellular acidosis
- Overshoot alkalosis
- Shift of oxyhemoglobin dissociation curve to left.

What is urine anion gap?

The urine anion gap helps in defining the cause in case of normal anion gap acidosis.

It is defined as Na^+ K^- Cl^- in urine.

The UAG is normally negative as a result of excretion of ammonium into urine. If UAG is positive it reflects an impairment of ammonium excretion as seen in patients with renal tubular acidosis (RTA). Urinary pH then further helps in knowing type of RTA. If pH is > 6 a distal RTA is present and if pH is low and remains low inspite of bicarbonate infusion proximal RTA is the underlying pathology.

What are the causes of metabolic alkalosis? How do you treat it?

Causes of metabolic alkalosis:

1. Loss of H^+ ions (e.g. vomiting, diuretics)
2. Increased reabsorption of bicarbonate
   - Low intravascular volume
   - Hypokalemia
   - High pCO_2
   - Increased mineralocorticoids (aldosterone).
3. Administration of alkali (in setting of renal impairment) e.g. Ringer's lactate where lactate gets metabolised to bicarbonates in liver adding to alkali pool.

Treatment

- Correct dehydration (intravascular volume) with chloride rich fluid and potassium
- If the patient has cardiac failure (or primary excess mineralocorticoids) use aldosterone antagonist (spironalactone)
- Use of carbonic anhydrase inhibitors like acetazolamide to enhance loss of bicarbonates from kidneys

What are the causes of respiratory acidosis?

Any condition that results in hypoventilation can cause respiratory acidosis. These conditions include:

1. Airway/pulmonary parenchymal disease
   a. Upper airway obstruction
   b. Lower airway obstruction
   c. Pulmonary alveolar process:
      i. Cardiogenic pulmonary edema
      ii. Pneumonia
      iii. ARDS
      iv. Pulmonary perfusion defect—PE—air/fat/tumor
2. Normal airway/pulmonary parenchymal
   a. CNS depression
   b. Central nervous system depression related to head injury
   c. Central nervous system depression related to medications such as narcotics, sedatives, or anesthesia
3. Neuromuscular disease and impairment—Impaired respiratory muscle function related to spinal cord injury, neuromuscular diseases,or neuromuscular blocking drugs
4. Ventilatory restriction—due to pain, chest wall injury/deformity, or abdominal distension.

What are the causes of respiratory alkalosis?

Any condition that causes hyperventilation can result in respiratory alkalosis. These conditions include:

1. CNS stimulation: Fever, pain, thyrotoxicosis, cerebrovascular accidents.
2. Hypoxemia: pneumonia, pulmonary edema, severe anemia.
3. Drugs/hormones: Medroxyprogesterone, catecholamines, salicylates.
4. Miscellaneous: Sepsis, pregnancy
5. Psychological responses, such as anxiety or fear.

What happens to serum sodium level in hyperglycemia? How do you calculate correct sodium in presence of hyperglycemia?

Since normal blood glucose is relatively low its contribution of osmotic pressure is relatively insignificant. However,

in presence of severe hyperglycemia as may be seen in uncontrolled diabetics, blood glucose exerts significant osmotic pressure resulting in raised serum osmolality that leads to shift of water from intracellular to extracellular compartment. This will dilute electrolytes in blood and as a result serum sodium concentration appears to be low. This is called as fictitious hyponatremia. In presence of significant hyperglycemia one can calculate corrected serum sodium as follows:

Corrected sodium= Na^+ **(**(BS in mg%–100)/100**)** × 1.4

What is osmolar gap? What are the causes of osmolar gap?

The plasma osmolality can be calculated by the formula

Plasma Osmolality = 2 x sodium (mEq/L) + glucose (mg/dl)/18 + urea (mg/dl)/2.

If the calculated osmolality differ from the measured osmolality by 15 mosm/kg H_2O, this is called as osmolar gap and further investigations need to be done. Causes are:

1. Ethanol
2. Isopropyl alcohol
3. Methanol
4. Glycine
5. Mannitol
6. Ethylene glycol
7. Glycerol
8. Chronic renal failure.

A 55-year-old man presents with shortness of breath. He is hypertensive on chlorthiazide and aspirin. He is chronic smoker with 60 pack year's history.

On room air his ABG shows pH 7.53, PaO_2 62 mm Hg, $PaCO_2$ 37 mm Hg, HCO_3- 30 mEq/L Hb 14 gm% and SaO_2 87%. How would you characterize his state of oxygenation, ventilation, and acid-base balance?

Oxygenation: The PaO_2 and SaO_2 are both reduced on room air. Since $P(A\text{-}a)O_2$ is elevated (approximately 43 mm Hg), the low PaO_2 can be attributed to V-Q mismatch, i.e. a pulmonary problem. SaO_2 is reduced, in part from the low PaO_2 and from elevated carboxyhemoglobin as he is a smoker. The arterial oxygen content is adequate.(CaO_2 16.5 mL O_2/dL)

Ventilation: Adequate for the patient's level of CO_2 production; the patient is neither hyper- nor hypoventilating.

Acid-Base: Elevated pH and HCO_3—suggest a state of uncompensated metabolic alkalosis, most likely related to the patient's diuretic; his serum K^+ should be checked for hypokalemia.

A 46-year-old man has been in the hospital two days with pneumonia. He was recovering but has just become diaphoretic, dyspneic, and hypotensive. He is breathing oxygen through a nasal cannula at 3 l/min.

pH 7.40 $PaCO_2$ 20 mm Hg PaO_2 80 mm Hg SaO_2 95% Hb 13.3 gm% HCO_3 12 mEq/L. How would you characterize his state of oxygenation, ventilation, and acid-base balance?

Oxygenation: The PaO_2 is lower than expected for someone of this age and receiving supplemental oxygen, and points to significant V-Q imbalance. The oxygen content is adequate. (CaO_2 17.2 mL O_2/dL)

Ventilation: $PaCO_2$ is half normal and indicates marked hyperventilation.

Acid- Base: Normal pH with very low bicarbonate and $PaCO_2$ indicates combined respiratory alkalosis and metabolic acidosis.

Which is primary pathology?

Patient has deteriorated acutely. Only compensation that works faster is respiratory. Metabolic full compeansation takes 2–3 days. Since compensation is full, compensating process is respiratory and primary pathology is metabolic. Patient is hypotensive hence sepsis should be strongly considered, especially in someone with a documented infection.

What are mixed acid base disorders and how do you diagnose them?

The simple, or primary, acid-base disorders (respiratory and metabolic acidosis and alkalosis) evoke a compensatory response that produces a secondary acid-base disturbance and reversion of the blood pH towards (rarely to) normal; e.g. a simple metabolic acidosis will result in a secondary respiratory alkalosis, both of which will ordinarily be reflected in the patients' acid-base-related analysis in blood. When two primary acid-base disturbances arise simultaneously in the same patient, the complex is called a mixed acid-base disorder. If three primary disturbances occur together, the patient is described as having "triple acid-base disorder."

For example, the development of a primary metabolic alkalosis in a patient with chronic obstructive airway disease who is being treated with diuretics should alert the clinician to the possibility of potassium depletion. To diagnose them always follow following rules:

1. Do not interpret any blood gas data for acid-base diagnosis without also examining the corresponding serum electrolytes.

2. Single acid-base disorders do not lead to normal blood pH. Although pH can end up in the normal range (7.35–7.45)
3. Simplified rules predict the pH and HCO_3—for a given change in $PaCO_2$. If the pH or HCO_3 is higher or lower than expected for the change in $PaCO_2$, the patient probably has a metabolic acid-base disorder as well.
4. In maximally-compensated metabolic acidosis (which takes about 12– 24 hours), the following formula applies: Expected $PaCO_2$ = last two digits of pH ± 2.
   In contrast, the compensation for metabolic alkalosis (by increasing $PaCO_2$) is highly variable, and in some cases there may be no or minimal compensation.

Enumerate the steps for analyzing the ABG

Step 0: Before starting make sure the report is compatible. To do this, use the following formula:

$$H \times HCO3/PaCO_2 = 24 (+/- 2 \text{ nmol/L})$$

To calculate H^+ in nEq/L from pH, use the following table.

pH	6.80	6.90	7.00	7.10	7.20	7.30	7.40	7.50	7.60	7.70	7.80
(–) from			100	90	80				85	90	95
(H^+)	160	130	100	80	60	50	40	30	25	20	15

Once you are sure the report is compatible, use following steps to analyse the ABG:

Step 1: Look at the pH.

Is the patient academic (H < 7.35) or alkalemic (pH > 7.45)

Step 2: Is it a primary metabolic or respiratory disturbance?

Acidemia: With HCO_3 < 20 mmol/L = metabolic. With PCO_2 > 45 mm Hg = respiratory

Alkalemia: With HCO3 >28 mmol/L =metabolic. With PCO_2 < 35 mm Hg = respiratory

Go to step 3 and 4 if Respiratory and step 5 to 7 if metabolic.

Step 3: If there is a primary respiratory disturbance, it is acute? Expect Δ pH = 0.08 x Δ PCO_2 / 10

(For respiratory acid (PCO_2–40) = Δ PCO_2)

(For respiratory alkalemic (40–PCO_2) = Δ PCO_2)

If not acute go to step 4, if yes –also consider steps 5–7.

Step 4: For a respiratory disorder is renal compensation OK?

Respiratory acidosis: < 24 hrs: Δ (HCO_3) = 1/10 × Δ PCO_2

> 24 hrs: Δ (HCO_3) = 4/10 × Δ PCO_2

Respiratory acidosis: 1–2 hrs: Δ (HCO_3) = 2/10 × Δ PCO_2

> 2days: Δ (HCO_3) = 5/10 × Δ PCO_2

Step 5: If the disturbance is metabolic is the respiratory. Compensation appropriate ?

For metabolic acidosis: Expect PCO_2 = [1.5 × (HCO_3)] + 8 ±2

For metabolic alkalosis: Expect PCO_2 = [0.7 × (HCO_3)] +21 ± 1.5

If not actual PCO_2 > expected: Hidden respiratory acidosis, actual PCO_2 < expected: Hidden respiratory alkalosis

Step 6: If there is metabolic acidosis, is there an anion gap?

Na – (Cl + HCO_3) = Anion gap usually < 12. If gap is more than 12, patient has an anion gap acidosis.

The usual causes are remembered by MUDPIES

- Methanol
- Uremia
- Diabetic Ketoacidosis
- Paraldehyde
- Infection (lactic acid)
- Ethylene Glycol
- Salicylate

Step 7: Does the anion gap explain the change in bicarbonate? [(HCO_3)+(gap- 12)] =24

If parameter is greater than 24; consider additional hidden metabolic alkalosis.

If parameter is less than 24; consider a hidden non anion gap metabolic acidosis.

This is valid only in patients who do not have a chronic respiratory acid base disorder.

A 45-year old male is admitted to the ICU with pancreatitis. His ABG is 15, pH 7.33, $PaCO_2$ 30 mm Hg, HCO_3, lactates = 6, albumin = 2 gm/dl, Na=136, K=4.5, Cl =108, Ca=3.3, Mg= 1.6, (mEq/L) PO4 = 2 mmol/L. Interpret it using traditional and the Stewart approach.

Using the classic approach:

PH =7.33 is suggestive of acidosis, HCO_3 of 15 is suggests primary metabolic acidosis with a compensatory fall in CO_2 because of hyperventilation.

Anion gap = (Na) – (Cl + HCO3) = 136–123 =13

Corrected anion gap for albumin= observed AG + 2.5 (Normal albumin- Observed albumin)

=13+ 2.5(4.4–2)=13+6 =19

Hence, this is a high anion gap metabolic acidosis due to elevated lactates.

Using the stewart approach:

pH =7.33 indicates acidosis

SID = (Na + K +Ca + Mg) – (Cl)

= (136+4.5+3.3+1.6) – (108)

=145.4–108 = 37.4

Since SID is less than 40 this is suggestive of metabolic acidosis with anion excess-lactates.

Suggested Readings

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41 Mechanical Ventilation

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How are ventilators classified?

There are numerous ways to classify ventilators.

Single Circuit Versus Double Circuit Ventilators

Ventilators that mix and deliver compressed gas directly to the patient are single circuit ventilators. They expel the expired gases to the atmosphere during expiration. Ventilators used in critical care are of this type. A double circuit ventilator is analogous to a reservoir bag powered by a single circuit ventilator. These ventilators deliver a driving gas, usually compressed oxygen or air, to a bellows assembly. The driving gas pressurizes the rigid space outside the bellows, causing the bellows to squeeze its contents of patient gas into the breathing circuit and the patient's lungs. For example, North American Drager and Ohmeda anesthesia ventilators.

Classification According to their Power

a. *Low powered ventilators:* These generate only modest gas pressures required to deliver reasonable tidal volumes to the lungs (in a normal or near normal lung with respect to resistances and compliances). Their use is limited as in a damaged lung the tidal volume delivered will be insufficient. Thus, when these ventilators are used monitoring of the ventilation becomes mandatory.
b. *High powered ventilators:* A ventilator needs to be powerful to maintain its performance in deteriorating lung conditions like changes in lung-thoracic compliance and airway resistances. Up to 80 cms of water is sufficient without alteration in gas flow (Flow generators). These require a safety valve (pressure relief valve) which can be preset or adjustable. They may also require a flow restrictor to avoid build up of high pressure in the lungs.

Classification According to Cycling

A ventilator is said to cycle in 2 phases-inspiratory and expiratory.

A. Inspiratory cycling

a. *Volume cycling:* These ventilators recognize the point at which the predetermined volume of the gas has left the ventilator and switches its internal mechanism to allow exhalation to occur.
b. *Time cycling:* These use mechanical, pneumatic or electronic timers to operate the cycling mechanism, which work independently of the delivered tidal volume. It may allow the tidal volume to be delivered early in the inspiratory cycle, followed by a pause to allow better distribution of the gases prior to the start of the expiratory phase.
c. *Pressure cycling:* Here the inspiratory phase is terminated when the predetermined pressure is reached.
d. *Flow cycling:* Flow pattern changes are used to cycle the ventilator. Now rarely used.

B. Expiratory cycling

a. *Volume cycling:* Here the expiratory phase is terminated when the bellows have filled to the desired tidal volume required for the next inspiration.
b. *Time cycling:* It is the most popular and the most versatile method of controlling the expiratory phase. It uses electronic or pneumatic timers.
c. *Pressure cycling:* It identifies a selected airway pressure at end of exhalation that would trigger the next inspiration.
d. *Flow cycling:* It senses the end of exhalation when the desired flow rate is reached and then switches to inspiration.

Classification According to Application

a. *Mechanical thumbs:* It is a simple T-piece system, the open end of which is occluded with the anesthetist's thumb thus inflating the patient's lungs. On release of the thumb pressure expiration occurs passively. Thus, rhythmically intermittent positive pressure can be achieved. In

ventilators like Sheffield and Amsterdam, the thumb is replaced by an electrical solenoid valve, which is cycled by an electronic circuit.

b. *Minute volume dividers:* These derive their power from the pressure of gases from the outlet of anesthetic machine. The whole of the driving gas is delivered to the patient and there is no rebreathing. Principle—A reservoir R which is continually pressurized by a spring or a weight or its own elastic recoil is continuously being filled by the fresh gas flow. 2 valves, V1 and V2 are linked together and operated by a bistable mechanism. When V1 is open and V2 closes, the reservoir discharges gas to the patient (inspiration). When V1 is closed and V2 opens expiration occurs. For example, East freeman automatic vent, Manley MP2, MN2 and MP3.
c. *Bag squeezers:* This is a bag in bottle type of ventilator (double circuit type of ventilators). The bag or bellows can be squeezed mechanically (gears or lever or motors) or pneumatically. For example, Cape ventilator, Manley Sevovent, Oxford, Penlon.
d. *Intermittent blowers:* These are driven by a continuos flow of gases and air pressure of 45 – 60 psi is required. For example, Bird Mark 7, Penlon-AP.

Discuss different types of bellows in anesthesia ventilators with their advantages and disadvantages.

Automatic ventilators include bellows that expand (fill with gas) during expiration and are compressed (to deliver gas) during inspiration in response to cycling ventilator drive pressure. Some units have ascending (rising or upright) bellows, which are attached to the base of the bellows housing and expand upward. Other units have descending (hanging) bellows, which are suspended from the top of the bellows housing and expand downward.

For an ascending bellows to expand, the expiratory pressure provided by the patient's passive lung and chest wall recoil must be sufficient to overcome the weight of the bellows. Conversely, the weight of a descending bellows assists expansion, allowing the bellows to expand—and actually draw in gas—even when no expiratory pressure is produced by the patient. Thus, when a leak or disconnection is present in the breathing circuit, a descending bellows will still expand (drawing air from the atmosphere through the leak and into the bellows) and be compressed as the ventilator cycles.

The disadvantages of the descending bellows are unrecognized disconnection (due to their design, they may fill even when disconnected from the patient), and also collection of exhaled humidity in bellows; risking infection and lessening delivered tidal volume. The modern type is ascending. Only one current machine, the Dräger Julian, uses a hanging bellows, but incorporates capnography and sensors to detect failure of the bellows to fill, both of which may lessen unrecognized disconnects.

To remember the classification: "Ascend" and "descend" have "e" in them - so look at them during expiration. Ascending bellows ("standing") ascend during expiration (modern type—preferred by many) and descending bellows ("hanging") descend during expiration. Ventilator relief valve gives 2 – 3 cm water pressure positive end-expiratory pressure (PEEP). This is true for almost all mechanical ventilators-exceptions are the new Dräger Divan ventilator, which has a horizontal piston and the Julian hanging bellows. The ventilator relief valve (spill valve) only allows scavenging during expiratory phase.

The hanging design was chosen for the Julian for compactness and ease of sterilization of the entire breathing circuit. The Julian hanging bellows housing, unlike older designs, lacks an internal weight, and senses when the bellows do not return to the full "down" position. These factors, plus integration of disconnect alarms based on chemical (capnograph), and mechanical (pressure, volume, and flow sensors) detection, make piston or hanging bellows designs safe. The placement of the hanging bellows below the writing surface makes visual detection of disconnects difficult; also it is less easy to determine if the patient is breathing spontaneously in addition to the rate set on the mechanical ventilator. The user must rely more on the pressure and capnography waveforms as opposed to the bellows. Water may gather in the bellows (lessening tidal volume and creating an infection risk), but this tendency should be opposed by the heated absorber head.

How does a positive pressure ventilator work?

A positive pressure ventilator is essentially just an air compressor and two valves. It pushes air in during inspiration and expiration occurs passively. That means during inspiration the inspiratory valve is open and the expiratory valve is closed. While the reverse happens during expiration; the expiratory valve opens, at the level of applied PEEP with a PEEP valve and the inspiratory valve is closed. If you have applied an inspiratory plateau period, then both valves are closed and the lung is held distended at the pressure which is the true Plateau pressure. This reflects the state of alveoli.

What is a mode of ventilation?

A mode is a particular pattern of spontaneous and mandatory breaths delivered by the ventilator. Common modes include pressure support, volume control, and pressure control

ventilation. There are multiple different 'modes' of ventilation, all with different names and initials.

What are the types of breaths?

Controlled breath: Breath initiated and delivered by the ventilator.

Assisted breath: Breath initiated by the patient and assisted to the set target (volume / pressure) parameter by the ventilator.

Spontaneous breath: Fully spontaneous breath, no ventilator assistance.

What are the phases of respiration during mechanical ventilation?

Phases of ventilation: A breath during mechanical ventilation can be divided into 4 phases. All modes can be described in terms of these phases. Once we understand these phases it is easy to understand even newer and more complicated modes.

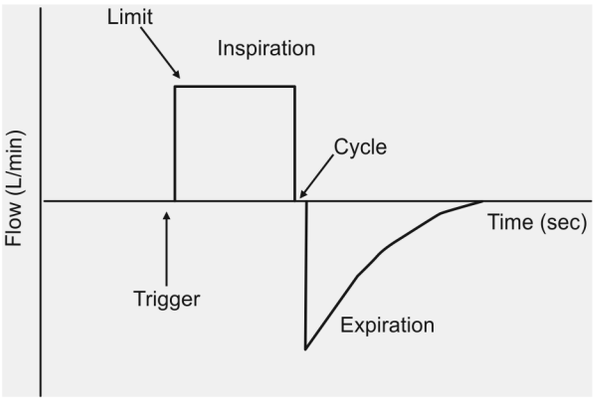

Fig. 41.1: Phases of respiration during mechanical ventilation

Expiratory phase: During this phase the inspiratory valve is closed, and expiratory valve is open and lungs empty passively. This can be further divided into time when there is no flow (expiratory pause) and when expiratory flow takes place. Positive end expiratory pressure (PEEP) is applied throughout the ventilatory cycle, including during expiration. Continuous positive airway pressure (CPAP) is nothing but PEEP during spontaneous breathing.

Changeover from expiration to inspiration: This tells the ventilator that expiration is over and inspiration should begin. The process is called triggering. Inspiration may be triggered or initiated by the patient during an assisted or spontaneous breath, or by the ventilator (controlled breath). During assisted or spontaneous breathing "triggering" allows the ventilator to sense the patient's inspiratory effort. The two common ways by which ventilators do this are pressure and flow triggering. Pressure triggering: Patient needs to generate a small negative inspiratory pressure (generally negative 1–3 cm H_2O), which is sensed by the ventilator, and it changes over to inspiration and delivers the next breath. Flow triggering: Here a constant flow is provided in the patient circuit during expiration, flow rate being measured upstream and downstream of the patient. When the patient begins a breath the downstream flow decreases relative to the upstream flow. The ventilator senses this difference in flow and an inspiration is triggered. Flow triggering is often set in the range of 1.5–3 L/min. Flow triggering may require less work for the patient when compared to pressure triggering. During controlled ventilation the inspiration is time triggered i.e. it begins after a set time. Other ways of triggering a breath are volume, impedance or motion triggering. Trigger sensitivity denotes how easily a breath may be triggered by the patient. Too low trigger sensitivity may lead to autocycling or autotriggering, which is breath delivery without effort from the patient. If the trigger is too high the patient may waste effort without getting a breath, leading to respiratory muscle fatigue. Wrong trigger setting can lead to patient—ventilator asynchrony.

Inspiratory phase: This phase begins with the initiation of the breath, and ends when the ventilator cycles into expiration. The PIFR and inspiratory time settings determine time available for expiration.

Limit: During the inspiratory phase, the inspiratory valve opens, and pressure, volume, and flow all rise above their end-expiratory phase. If one (or more) variables rises no higher than a preset value, it is called a limit variable. Limit variable is the one which limits the way gas keeps flowing in the lungs during inspiration. However, the limit variable does not terminate the inspiration (that is known as cycling—Figure 41.1); it allows inspiration to continue till the cycling criterion is reached, but at the set limit. This phase may also contain the inspiratory pause, during which the inspiratory valve and expiratory valve are both closed. During the inspiratory pause, there is no gas flow in or out of lungs and the pressure during this phase denotes the alveolar distending pressure. This pressure is also called as Plateau pressure.

Changeover from inspiration to expiration: This occurs when the ventilator recognizes that the inspiratory phase is over, the process is called cycling. Different modes have different ways of cycling. These are: volume cycling (volume controlled ventilation VCV), time cycling (pressure control ventilation PCV) and flow cycling (pressure support ventilation).

What are the common or old modes of mechanical ventilation? What are the differences between IMV, SIMV and assist control (AC)?

The standard or established modes are volume controlled mode, pressure controlled mode and pressure support mode. The controlled modes can be further of following types: Intermittent Mandatory Ventilation (IMV), Synchronized Intermittent Mandatory Ventilation (SIMV) and Assist Control (AC). Sometimes two modes may be combined during ventilation; for example; SIMV may be combined with pressure support. There are a wide variety of different modes available, many of which are proprietary and ventilator specific.

In IMV, the ventilator delivers the set number of breaths at set time, regardless of which phase of respiration the patient is in. In SIMV, the inspiration is synchronized with patient effort, but the ventilator will deliver only fixed number of breaths; whereas in Assist Control, the ventilator will deliver a set number of breaths synchronized with patient effort and will also assist any extra breaths (to the level of set tidal volume) that the patient may initiate.

Discuss volume controlled ventilation (VCV). What are the advantages and disadvantages?

VCV is a controlled mode where the tidal volume is controlled and it also allows the patient breath additional breaths. This can also be used in SIMV or Assist Control Mode.

1. *Changeover from expiration to inspiration:* This can be pressure or flow triggered, and each breath will have the same set tidal volume.
2. *Inspiratory phase:* During VCV, the settings are tidal volume to be delivered each breath, Peak Inspiratory Flow Rate (PIFR) usually 1–1.5 L/min, and inspiratory time. The combination of PIFR and the inspiratory time will determine how fast the breath will be delivered by the ventilator, how much inspiratory pause will be available and the time available for expiration. A very high inspiratory flow rate leads to high airway pressures, and a low inspiratory flow rate may lead to ventilator dysynchrony and air hunger. The length of inspiration depends on the tidal volume and inspiratory flow rate that have been set. The flow during this mode is constant flow (square wave pattern).
   Limit: The flow rate rises to the set limit (set flow rate), or the delivered tidal volume; provided the limit is not exceeded. In VCV, the limiting variables is pressure—so the modern VCV mode is Volume Controlled Pressure Limited mode. The limit prevents delivery of excessive pressures to the lungs which means that the preset tidal volume will not be delivered as well. So the limit setting is very important to prevent barotrauma.
3. *Changeover from inspiration to expiration or cycling:* This mode is volume cycled, which means that the ventilator cycles to exhalation when the set tidal volume has been delivered.
4. *Expiratory phase:* Positive end expiratory pressure may be added.
   Advantages: VCV ensures delivery of set minimum tidal and minute ventilation regardless of patient effort and allows the patient to rest by reducing work of breathing. Patient can however increase the minute ventilation by breathing spontaneously, however the spontaneous breaths will not be supported.
   Disadvantages: Airway pressure varies as per compliance of patient lungs. High inspiratory flow demands from patient may not be met as the PIFR is fixed. During this mode adverse hemodynamic effects may occur due to positive intrathoracic pressure. Inappropriate hyperventilation may occur if the patient takes additional breaths above the set rate.

Discuss pressure control ventilation with advantages and disadvantages.

In this mode, inspiratory airway pressure and inspiratory time are fixed by the clinician.

1. *Changeover from expiration to inspiration:* Inspiration is time triggered with PCV, and the respiratory rate is set. When used as P-SIMV or PCV–AC patient may initiate additional breaths.
2. *Inspiratory phase:* The peak pressure is set. The ventilator quickly delivers inspiratory flow to reach the set pressure and maintains the flow to keep the pressure constant. Therefore, the flow type is called decelerating flow (decreasing flow). Thus, peak inspiratory pressure is generated quickly and maintained constant throughout inspiration. With a prolonged inspiratory time, the inspiratory flow rate may fall to zero before the end of inspiration. The tidal volume delivered depends on the set pressure and inspiratory time, and on patient factors such as respiratory mechanics i.e. airway resistance and respiratory compliance. Tidal volume may vary but the peak airway pressure is constant.
   Limit: Gas flow occurs till the preset airway pressure is reached. This pressure is maintained constant throughout the set inspiratory time.
3. *Changeover from inspiration to expiration:* PCV is time cycled, which means that the ventilator cycles into expiration when the set inspiratory time is over. A long inspiratory time will increase the tidal volume delivered.

4. *Expiratory phase:* Expiration occurs passively and PEEP can be added to pressure control ventilation.
   Advantages: PCV ensures a control over peak airway pressure and allows the patient to rest by reducing work of breathing. Minimum minute ventilation is not ensured. This mode may be of some use in patients with small leaks (such as bronchoplueral fistula) to tide over the initial crisis, till definite treatment can be undertaken. The decelerating flow may compensate for small leaks.
   Disadvantages: Tidal volume (and minute ventilation) varies as per compliance of patient lungs and minimum minute ventilation is not assured. Inappropriate hyperventilation may occur if the patient takes additional breaths above the set rate.

Discuss pressure support ventilation (PSV) with advantages and disadvantages.

Pressure support is a spontaneous mode of ventilation. The patient initiates each breath, the ventilatory flow takes the inspiratory pressure to the set level and the patient terminates the inspiration. Apnea back up ventilation must be set while using this mode.

1. *Changeover from expiration to inspiration:* The patient triggers (pressure or flow) each breathe in PSV.
2. *Inspiratory phase:* The clinician sets the inspiratory pressure. The ventilator delivers a high initial flow rate, to achieve set pressure, and then the flow declines to maintain the set airway pressure constant throughout inspiration. The idea is to set the support pressure so that the patient generates adequate tidal volumes with a comfortable respiratory rate.
   Limit: Gas flow occurs till the set airway pressure is reached. This pressure is maintained constant throughout the inspiratory time.
3. *Changeover from inspiration to expiration:* Pressure support is flow cycled. As the patient's inspiratory flow rate falls to 25% of the peak inspiratory flow, the ventilator cycles into the expiratory phase.
4. *Expiratory phase:* PEEP can be combined with pressure support.
   Advantages: PSV is supposed to ensure patient-ventilator synchrony, reduce work of breathing and thus maintain patient comfort.
   Disadvantages: If the patient stops breathing apnea back up is the only ventilation. The tidal volume and minute ventilation will vary as per patient's lung compliance. Presence of leaks in the ventilator circuit will interfere with cycling.

Summarize the differences between VCV and PCV.

The table below summarizes the differences between VCV and PCV.

Table 41.1 Differences between VCV and PCV

Variable	Volume/Flow control	Pressure control
Tidal Volume	Set, constant	Variable
Peak inspiratory pressure	Variable	Set, constant
Inspiratory time	Set	Set
Inspiratory flow	Set	Variable
Inspiratory flow waveform	Set, constant	Decelerating type, variable

What is positive end expiratory pressure (PEEP)?

Positive End Expiratory Pressure (PEEP) refers to a constant baseline pressure that is present throughout the ventilatory cycle (not just on expiration). Continuous Positive Airway Pressure (CPAP) is PEEP during spontaneous breathing.

How does PEEP improve oxygenation?

PEEP improves oxygenation by preventing end-expiratory collapse of alveoli and by recruiting non-ventilated (shunt), or poorly ventilated (low V/Q) alveoli. Cyclic opening and closing of alveoli and distal bronchioles with each breath during ventilation, depletes surfactant and contributes to Ventilator Induced Lung Injury (VILI). PEEP keeps the alveoli inflated and prevents cyclic opening and closing of alveoli. PEEP improves oxygenation in patients with pulmonary edema by creating hydrostatic forces which move fluid from the alveoli and airways into the interstitium and by reducing left ventricular afterload.

What are the adverse effects of PEEP?

Inappropriate PEEP may create overinflation of alveoli leading to lung injury. In patients with hypovolemia and high dead space (COPD patients) PEEP may worsen of ventilation due to an increase in dead space. PEEP may worsen right heart failure by increasing right ventricular afterload. PEEP may precipitate hypotension due to raised intrathoracic pressure.

What is intrinsic PEEP or auto PEEP? How do you detect it?

Intrinsic PEEP is PEEP generated within patient's own lungs and is distinct from externally applied PEEP. It develops when expiratory time is short and expiration remains incomplete. Thus when the next breath comes in, some amount of previous tidal volume is still present in the alveoli,

thus the next ventilator breath 'stacks' on the previous breath. This leads to hyperinflation of the alveolus and generation of positive pressure, this intrinsic increase in end-expiratory pressure known as 'intrinsic or auto-PEEP'. This process is seen commonly in COPD patients is called dynamic hyperinflation.

Ways to detect auto-PEEP: Many ventilators will permit direct measurement of auto-PEEP by doing an expiratory hold maneuver.

Clinical detection: Development of auto-PEEP should be anticipated in patients with a high minute ventilation, high respiratory rate, short expiratory times, and airway obstruction. Since auto-PEEP can cause severe hyperinflation, patient has to spend more effort; this is seen clinically patients failing to trigger ventilator or patient-ventilator dysynchrony. If expiratory sounds are heard throughout the expiratory phase on auscultation right up to the inspiratory phase of the next breath then auto-PEEP should be suspected.

From flow waveform: Look at the expiratory flow versus time curve on the ventilator. If expiratory flow does not return to baseline (i.e. zero) before the next inspiration, then auto-PEEP is present.

What are the clinical implications of auto-PEEP?

Auto-PEEP causes an increase in intrathoracic pressure and therefore can cause adverse hemodynamic effects. Auto-PEEP can increase the work of breathing required to trigger the ventilator. This additional imposed work of breathing can be very uncomfortable and will eventually lead to patient fatigue.

How do you manage auto-PEEP?

Auto-PEEP is due to short expiratory time and is managed by increasing the expiratory time on the ventilator. This is achieved by reducing respiratory rate, reducing tidal volume or increasing the peak inspiratory flow rate. In patients with bronchial asthma, apart from these changes to ventilatory setting, bronchodilator therapy will reduce auto-PEEP.

What are the contraindications to PEEP?

Relative contraindications to the use of PEEP include:

- Hypotension due to volume depletion and dehydration
- Right ventricular failure since PEEP increases right ventricular afterload
- Right to left intracardiac shunt
- Increased intracranial pressure particularly if CVP is also high. Since hypoxemia worsens neuronal damage, risk of increased ICP with PEEP must be balanced with likelihood of an improvement in oxygenation.
- Patients with hyperinflation such as emphysema and COPD. However, external PEEP can be used to balance auto-PEEP in spontaneously breathing patients as it makes triggering easier for these patients.
- Bronchopleural fistula. High levels of PEEP may increase air leak with loss of ventilation. PEEP may also prevent healing.

Discuss initial set up of the ventilator for elective ventilation of a postoperative patient.

First we have to select a mode. There are studies to suggest a difference in outcomes if either VCV or PCV are used. Select a mode that you are most familiar with. Since the patient is going to start breathing soon, an AC mode is useful as it will also assist all spontaneous breaths. The initial settings thus are as follows:

FiO_2: 1.0 always start with 100% O_2. This should be reduced to 0.5 as soon as possible, preferably within next 24 hours.

Tidal volume: 8–10 ml/Kg body weight

Respiratory rate: 12–14 breaths/minute

PEEP: Start with 5 cm H_2O, may be increased in increments of 2–3 cm H_2O for achieving adequate oxygenation.

Trigger sensitivity: 1–3 L/min or -1 to -2 cm H_2O

Inspiratory flow rate: 1–1.5 L/kg, higher rates may be needed for asthma patients.

The settings may be adjusted after assessing patient response and an arterial blood gas drawn 30 minutes later.

What alarms are set during ventilation? Explain the function of each alarm.

The most common set alarms are:

1. High pressure alarm: Sets off when the compliance is bad or patient coughs or when there are with mechanical problem with circuit.
2. Low pressure alarm: Sets off with leaks in the circuit, accidental disconnection, or accidental extubation.
3. Low minute ventilation: Sets off with leaks in the circuit or inappropriate settings.
4. High minute ventilation: When the patient is breathing too fast, due to need for high MV or if patient not sedated well enough.
5. High or low respiratory rate.
6. High and low FiO_2.
7. Apnea alarm: circuit disconnection, apnea (during spontaneous modes). When using spontaneous modes, backup ventilation must be set up.

What are the complications of mechanical ventilation?

Related to Intubation

- Endobronchial intubation and massive atelectasis
- Esophageal intubation
- Failed intubation
- Trauma to upper airway and tracheobronchial tree
- Blocked tracheal tube
- Accidental extubation
- Sinusitis
- Cuff leak
- Tracheal stenosis
- Tracheoesophageal fistula
- Tracheoinnominate fistula

Due to Positive Intrathoracic Pressure

- Hypotension
- Reduced organ perfusion
- Raised intracranial pressure
- Ventilator malfunction
- Meteorism (Gaseous distension of GI tract)
- Ventilator induced lung injury (VILI) or ventilator associated Lung Injury (VALI).

Suction Related Complications

- Hypoxia
- Arrhythmias

Related to ventilator setup

- Unintended hyperventilation
- Unintended hypoventilation (acute respiratory acidosis)
- Ventilator initiated lung injury
- Inadvertent air trapping and auto-PEEP
- Respiratory distress (due to patient ventilator asynchrony-'fighting the ventilator')
- Excessive patient work of breathing (due to incorrect setting)
- Inappropriate use of paralytic agents
- Oxygen toxicity
- Ventilator associated pneumonia.

Technology Gap in Patient Management

- Bedside communication failure
- Muscle weakness
- Barotrauma
- Subcutaneous emphysema
- Pneumomediastinum
- Pneumothorax.

What is barotrauma?

Pneumothorax, pneumomediastinum and subcutaneous emphysema are manifestations of extra-alveolar air, and are collectively termed "barotrauma" in mechanically ventilated patients, although in most instances alveolar overdistension (high peak inflation volume) is probably responsible for their occurrence rather than high airway pressure. The occurrence of alveolar rupture during mechanical ventilation is influenced markedly by the presence and nature of underlying lung pathology. Barotrauma is less common in patients with normal lungs, and more common in patients with severe obstructive lung disease as well as in ARDS.

Manifestations of Barotrauma

- Pulmonary interstitial emphysema
- Pneumomediastinum
- Subcutaneous emphysema
- Pneumopericadium
- Systemic air embolism
- Pneumothorax: May be asymptomatic, or present with worsening oxygenation, an increase in peak inspiratory airway pressure, or a fall in lung-chest wall compliance. Tension pneumothorax is defined by its adverse effects on cardiovascular function than by its radiographic characteristics. The latter include mediastinal displacement away from the pneumothorax, reversal of the diaphragmatic curve, and overall enlargement of the affected hemithorax. Most commonly the patient with tension pneumothorax presents with agitation and respiratory distress, hypotension and other signs of cardiovascular collapse.

What is ventilator induced lung injury (VILI)? What are the mechanisms which lead to (VILI)?

Injury to the lung that occurs in the course of and due to mechanical ventilation is termed ventilator-induced lung injury. The following mechanisms of lung injury have been described:

1. Atelectrauma: Cyclical collapse of alveoli i.e. repeated opening and closing of alveoli and surfactant depletion. Normal or stable alveoli do not collapse completely at the end of expiration. During ARDS, some alveoli may become unstable and undergo cyclical collapse. Ventilation with high FiO_2 aggravates alveolar collapse due to absorption atelectasis.
2. Oxygen toxicity: While this is well-known, it is not clear what concentration of oxygen is toxic over what period of time. It is generally assumed that $FiO_2 < 0.6$ is not toxic, however an attempt must be made to maintain the FiO_2 as low as possible.
3. Volutrauma: Ventilation at high volumes and pressures can lead to alveolar over distension, causing increased

permeability in the uninjured lung with pulmonary edema and enhanced edema in the injured lung.

4. Cyclical shear stress injury: Cyclic opening and closing of atelectatic alveoli during mechanical ventilation create tremendous shear stress at their junctions with open alveoli. This results in damage to the capillary endothelium and the alveolar membrane.
5. Biotrauma: Alveolar overdistension along with the repeated collapse and reopening of the alveoli can result in a whole cascade of proinflammatory cytokines which induce a pulmonary and systemic cytokine response, aggravating lung injury and causing systemic multiorgan dysfunction.
6. Barotrauma: Pneumothorax, pneumomediastinum, interstitial emphysema.

How can we monitor the mechanics of the respiratory system during mechanical ventilation? Explain the terms peak pressure and plateau pressure.

The respiratory mechanics can be monitored by looking at the pressure-time graph present on all modern ventilators. The highest pressure during inspiration in a volume controlled mode is Peak Pressure or Ppeak. When you apply an inspiratory pause, the alveoli are held distended with no gases flowing in or out of the lungs. This pressure is called Alveolar Distending Pressure of Plateau Pressure (Pplat). In patients with alveolar pathologies, this pressure will go up.

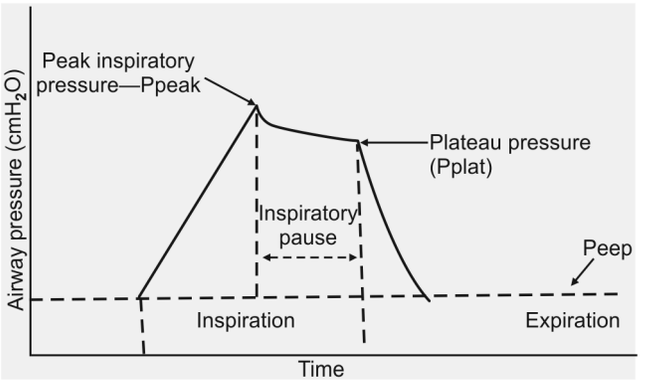

Fig. 41.2: Pressure-time graphic used to monitor respiratory mechanics

What is the compliance of the respiratory system? How do you measure static and dynamic compliance?

Compliance relates to the change in volume of the system on application of pressure. Static compliance is measured when there is no movement of gases occurring in the system (Fig. 41.2) i.e. alveoli are held open at the end of inspiration. So plateau pressure is a surrogate of static compliance. Static compliance is given by the following formula: Cstat = Tidal volume/Pplat–PEEP. Alveolar pathologies such as pneumonia or ARDS will cause a decrease in static compliance and an increase in Pplat. Dynamic compliance is measured when gas is flowing into the lung. This means the flow has to overcome the resistance of the breathing system starting with inspiratory limb of the circuit, endotracheal tube and the tracheobronchial tree. Thus at the level of peak pressure, if there is anything wrong with the inspiratory arc of the circuit—such as a linked ET tube, bronchospasm, it will lead to requirement of increased inspiratory pressures to overcome the resistance. Dynamic compliance is given by the following formula:

Cdyn = Tidal volume/Ppeak–PEEP. Ppeak is a surrogate of dynamic compliance. The effect of therapy in a patient with bronchospasm can be monitored by tracking a decrease in Ppeak or Cdyn.

How is resistance of the respiratory system calculated?

Resistance of any system is given by the formula resistance = Pressure across the tubing / flow in the system. For respiratory system this will be given by resistance = Ppeak–Pplat/ Inspiratory flow. (Figure 41.2). At the bedside the difference between Ppeak and Pplat can be easily monitored as a surrogate of the calculated resistance. If Ppeak is high but Pplat is normal, the patient has a resistive problem. Once you start treating the bronchospasm, this difference will reduce. In a patient with alveolar or lung parenchymal pathology, both Ppeak and Pplat will be high and close together.

What is bilevel inspiratory positive pressure ventilation (BIPAP)?

This is probably the most confusing term in the area of ventilation. Theoretically all pressure limited modes can be called BIPAP as they work between two pressures: the inspiratory pressure and the expiratory pressure. The various types of BIPAP are described below.

BIPAP type 1. (BIPAP, Respironics): This is completely spontaneous mode. The clinician sets the Inspiratory Positive airway Pressure (IPAP) and an Expiratory Positive airway Pressure (EPAP). The patient triggers the breath, ventilator supplies a decelerating flow, the breath is limited by the IPAP. Cycling is flow sensed and the expiration then ends at the EPAP pressure level. Thus, it is similar to a CPAP + PS mode where IPAP is the PS level and CPAP is EPAP.

BIPAP type 2 (BIPAP, Drager): This is a controlled mode of ventilation. The clinician sets the Inspiratory Positive airway Pressure (IPAP) and an Expiratory Positive airway Pressure (EPAP), inspiratory time and expiratory time. The ventilator triggers the breath which is pressure controlled, time cycled and ends at the set EPAP level.

BIPAP type 3: (Drager Evita): This mode is similar to BIPAP 2 and the settings are similar except that the time settings at IPAP and EPAP are set longer (5 to 10 seconds each) and the patient can breathe spontaneously. Thus this can be considered to be a double CPAP mode, where patient breaths alternately between two levels of pressure (IPAP and EPAP).

BIPAP type 4: This is a variation of type 3 breath. The settings are similar to the type 3 mode above, except that the IPAP time is too short. The patient can breathe spontaneously at the lower pressure level i.e. CPAP but not at the IPAP level. Thus the inspiration is a pressure limited, time cycled breath. This is similar to a pressure limited and time cycled form of SIMV. Additional breaths are spontaneous unsupported breaths or they may be pressure supported breaths.

BIPAP type 5: This is another variation on type 3 BIPAP. Here the patient breaths spontaneously only at the IPAP level. The expiratory time is very short and does not allow spontaneous breathing.

What is pressure control inverse ratio ventilation (PC-IRV)?

PC-IRV is a type of pressure control CMV where all breaths are pressure limited and time cycled and ventilator triggered. The inspiration is longer than the expiration. It is also simply called Inverse Ratio Ventilation. In patients with ARDS, the alveoli in various lung regions may have different time constants i.e. some alveoli are "slow" while some are "fast"; this basically means that these alveoli are slow (or fast) to fill or empty. When we use inverse ratio ventilation, the slower alveoli get time to be filled.

Describe airway pressure release ventilation.

It is actually a variation of BIPAP one. Actually, this is pressure controlled mode with inverse ratio ventilation. It is often described as two levels of positive pressure applied for preset time between these pressure levels breaths spontaneously. Because the patient breaths between two positive pressures this has also been called Bilevel Airway Pressure (BiPAP). Other synonyms are Variable Positive Airway Pressure (VPAP), Intermittent CPAP and CPAP with release. Here the mandatory breaths are pressure controlled, time triggered, pressure limited and time cycled. APRV allows the patient to breath spontaneously during any phase of ventilators' mechanical cycle. The clinician sets the pressure High (generally the plateau pressure) and the pressure low or release pressure (generally set as 0) and the time spent at each pressure. Thus both time high (generally 5–6 secs) or inspiratory time and time low or time for expiration (generally 0.5–1 secs) are also set. The expiratory time is set such that the expiration is incomplete. Thus, oxygenation improves because of prolonged inspiratory time (helps recruit and ventilate the slow alveoli) and development of Auto-PEEP due to incomplete expiration.

Describe high frequency ventilation

This is an infrequently used mode of ventilation. The ventilators used are totally different and the settings also are different from the conventional ventilators. Here one uses very small tidal volumes and very high frequencies (180-300 Hz). The tidal volumes are generally less that the anatomical dead space. All breaths are ventilator triggered and time cycled. A recent review described mechanism of gas exchange that can be briefly summarized as convection and convection–diffusion. The diffusion occurs due to turbulent movement of gases and direct ventilation of close alveoli. Due to different patterns of inspiratory and expiratory velocity profiles of the tidal ventilation, axial movement of gas flow occurs leading to gas exchange with the expired gas. Other mechanisms include pendelluft, cardiogenic mixing, laminar flow with Taylor dispersion, collateral ventilation between neighboring alveoli, and molecular diffusion takes place. This mode has mostly been shown to be effective in management of ARDS in neonates. It can also be used during surgery of the airway. Disappointingly, the studies of HFO in adult patients in ARDS have failed to show benefit.

What are dual modes of ventilation? How do these modes work?

Dual control modes are capable of controlling either pressure or volume based on a measured input variable. These modes switch between pressure control and volume control ventilation. This switch can occur either within single breath: e.g. Volume Assured Pressure Support (VAPS) ventilation and Pressure Augmentation (PA) or the switch can occur between two breaths: Volume Support (VS) or Pressure-Regulated Volume Control (PRVC). These modes are also called closed loop modes of ventilation i.e. as per the sensed change in requirements of patient; the ventilator changes the delivery of breath. In theory, closed-loop ventilation should lead to increased safety to the patient, greater patient comfort and by virtue of their very definition show a rapid response to change in patient condition. Unfortunately, the introduction of these modes has not been shown to contribute to reduced length of mechanical ventilation or improved ICU outcomes.

Dual control breath to breath: These modes are essentially pressure limited modes that aim to maintain minimum peak pressure to achieve the set tidal volume. The tidal volume is maintained consistently in face of varying compliance or

resistance, thus we get an automated reduction of pressure and flow while constant minute volume is maintained. The examples include PRVC and volume support.

Dual control within a breath: In this type of ventilation, the modes aim to maintain minimum peak pressure required to achieve the tidal volume set by the clinician. (A more subtle volume control mode.) The tidal volume is maintained in face varying compliance or resistance by automatic reduction or increase of pressure and flow and thus eventually maintaining the desired minute volume.

What is neurally adjusted ventilatory assist (NAVA)? How does it work?

The neural signal of respiration originates in respiratory center which is then transmitted through phrenic nerve to excite the diaphragm. In the novel mode called Neurally Adjusted Ventilatory Assist (NAVA), a nasogastric tube containing 7 electrodes is placed in the stomach so that the electrodes are at the level of diaphragm. The signals coming to diaphragm from the respiratory centre are sensed by these electrodes and the information is sent to the ventilator. The ventilator then provides assistance to the patient commensurate with the degree of patient effort. Thus with this mode patient's own respiratory centre is in direct control of the mechanical support required on a breath-by-breath basis, and any variation in neural respiratory demand is responded to by the appropriate corresponding change in ventilatory assistance. So in theory NAVA should be very useful in reducing patient ventilator asynchrony. This mode is relatively new and clinical studies are ongoing regarding utility of this mode.

What is automatic tube compensation (ATC)? How does it work?

Automatic tube compensation (ATC) is a new option which can be used with other modes of ventilation. During inspiration and expiration, it can compensate for the non-linear decrease in pressure through the endotracheal or tracheostomy tube. ATC compensates for the tube-related additional work of breathing. This is also sometimes termed the "electronic extubation". If the clinician thinks patient is ready for extubation, he can put the ATC on. If the patient appears to breath he may be extubated. However, patient can still fail extubation due to upper airway edema.

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42 Pulmonary Function Tests

V Patil, M Desai

What are pulmonary function tests (PFTs)?

Pulmonary function tests (PFTs) is a term used to indicate a group of studies or maneuvers that may be performed using standardized equipment to measure lung function. PFTs can include simple screening spirometry, formal lung volume measurement, diffusing capacity for carbon monoxide, and arterial blood gases. These studies may collectively be referred to as a complete pulmonary function survey. It includes:

1. Spirometry
2. Lung volumes by helium dilution or body plethysmography
3. Diffusing capacity for carbon monoxide
4. Bronchial challenge testing
5. Blood gases: This is discussed in a separate chapter
6. Exercise tests
7. Pulse oximetry:

PFTs identify and quantify many functional and structural abnormalities of respiratory system. PFTs give information regarding the volume and velocity of the movement of air moving in and out of lungs, compliance of the lungs and chest wall, diffusion characteristics of membrane, and quantify the response to a procedure (lobectomy/pneumonectomy) and therapy. They only support or exclude a diagnosis. A combination of a thorough history and physical examination, as well as supporting laboratory data and imaging will help establish a confirmed diagnosis.

What are the limitations of PFTs?

1. There is some variability in normal predictive value as with many other tests
2. The accuracy of the test depends on well-trained technician as well as patient's cooperation unlike other laboratory tests. Patient should use maximum efforts and technician should be able to recognize submaximal efforts.
3. PFTs must be interpreted in the context of a proper history, physical examination, and ancillary diagnostic tests. Used alone they generally cannot distinguish among the potential causes of the abnormalities.

What are the requirements for good PFT's?

The American Thoracic Society (ATS) has published guidelines for the standardization of spirometry equipment and performance which include criteria for acceptability and reproducibility. Because spirometry is such an effort dependent test, each spirogram should be examined using performance criteria set forth by the ATS.

1. Lack of artifact induced by coughing, glottic closure, or equipment problems (primarily leak).
2. Satisfactory start to the test without hesitation.
3. Satisfactory exhalation with six seconds of smooth continuous exhalation and/or a plateau in the volume time curve of at least one second or a reasonable duration of exhalation with a plateau.

Criteria for reproducibility after obtaining three acceptable spirograms include:

1. Largest FVC within 0.2 L of next largest FVC.
2. Largest FEV_1 within 0.2 L of next largest FEV_1.

If the two above criteria have not been met, additional spirograms should be obtained.

Up to eight efforts should be allowed in order to meet acceptability and reproducibility criteria. Beyond eight efforts, fatigue may play a role in the results and the interpretation of the test may not be reliable. If all the criteria are not met, abnormal results should be interpreted with caution.

What are the indications for spirometry?

Diagnostic

- To evaluate symptoms, signs or abnormal laboratory tests
  - Symptoms: Dyspnea, wheezing, orthopnea, cough, phlegm production, chest pain

  - Signs: Diminished breath sounds, overinflation, expiratory slowing, cyanosis, chest deformity, unexplained crackles
  - Abnormal laboratory tests: Hypoxemia, hypercapnia, polycythemia, abnormal chest radiographs
- To measure the effect of disease on pulmonary function –Obstructive or restrictive
- To screen individuals at risk of having pulmonary diseases
  - Smokers
  - Individuals in occupations with exposures to injurious substances affecting lungs e.g. asbestosis
- To assess preoperative risk—this is especially true of patients who are:
  - Known to have pulmonary disease
  - Obese
  - Undergoing an upper abdominal or a thoracic operation
  - Have a history of smoking, cough or wheezing
- Part of routine physical examinations
- To assess prognosis (Idiopathic pulmonary fibrosis, lung transplant, etc.)
- To assess health status before enrollment in strenuous physical activity programs.

Monitoring

- To assess therapeutic interventions
  - Bronchodilator therapy
  - Steroid treatment for asthma, interstitial lung disease, etc.
  - Management of congestive heart failure
  - Other (antibiotics in cystic fibrosis, etc.)
- To describe the course of diseases affecting lung function
  - Pulmonary disease
  - Obstructive airway disease
  - Interstitial lung disease
  - Cardiac disease, congestive heart failure
  - Neuromuscular disease, Guillain-Barre syndrome
  - To monitor persons in occupations with exposure to injurious agents
  - To monitor for adverse reactions to drugs with known pulmonary toxicity (Bleomycin, Amiodarone, etc.)

Public Health

- Epidemiologic surveys
- Comparison of health status of populations living in different environments
- Validation of subjective complaints in occupational/ environmental settings
- Derivation of reference equations for interpretation of PFTs for age, sex, race.

Are there any contraindications for spirometry?

There are no absolute contraindications for performing PFTs. Relative contraindications for spirometry include:

- Hemoptysis of unknown origin
- Pneumothorax
- Recent myocardial infarction
- Unstable angina pectoris
- Thoracic, abdominal and cerebral aneurysms
- Recent abdominal or thoracic surgical procedures
- Patients with a history of syncope associated with forced exhalation
- Recent eye surgery (raised intraocular pressure during forced expiration).

What is spirometry?

Spirometry is one of the most commonly performed pulmonary function test which measures the rate at which the lungs change volume during quiet and forced breathing maneuvers. It can only measure lung volume compartments that exchange gas with the atmosphere.

There are two methods of recording FVC:

1. Volume exhaled is plotted as a function of time, where we commonly measure FEV_1 and the average forced expiratory flow rate over the middle 50% of FVC i.e. FEF 25-75% which is effort independent.
2. Volume exhaled is plotted against flow (flow volume curve). Flow volume loop is a plot of inspiratory and expiratory flow on the Y-axis against volume on the X-axis during the performance of maximally forced inspiratory and expiratory maneuvers.

Both the curves reflect same data and most of the machines that are used presently are computerized and give both curves.

Enumerate physiological factors that affect normal values of PFTs.

Spirometry is typically reported in both absolute values and as a predicted percentage of normal. Standards for normality are based on age, sex, height, weight and race with an abnormal value occurring if there is a difference of $\pm$20% from the predicted mean value.

- *Age:* Natural elasticity of the lungs decreases with aging which results in gradual decrease of lung volumes and capacities.
- *Gender:* Lung volumes and capacities of males are more than those of female individuals. Even when males and females are matched for height and weight, males have larger lungs than females. Because of this gender-dependent lung size difference, different normal tables must be used for males and females.

- *Body height and size:* An individual with small stature has smaller PFT result than a man of the same age who is much larger. Normal tables account for this variable by giving predicted PFT data for males or females of a certain age and height. In obese patients, body fat to lean body mass ratio increases, the abdominal mass prevents downward excursion of the diaphragm and the PFT results will demonstrate a smaller measured PFT outcome than expected. This leads to smaller measured values than the predicted values (predicted values from the normal tables).
- *Race:* Caucasians PFTs are different than those of Blacks, Hispanics, and Native Americans.

How will you decide normal range of PFTs for a patient with kyphoscoliosis?

Body height should not be used in these patients as the measured height is less than actual skeletal height due to deformed spine. Measurement of arm span is better marker of actual height in these patients.

Give definitions of various volumes and capacities that are mentioned in PFT reports.

TV (Tidal volume) is the volume of air that is inhaled or exhaled with each breath when a person is breathing at rest.

FEV_1 (Forced expiratory volume in 1 second in liters) is the volume of air forcibly expired from a maximum inspiratory effort in the first second.

FRC (Functional residual capacity in liters) is the volume of air in the lungs following a tidal volume exhalation = ERV + RV.

FVC (Forced vital capacity in liters) is the total volume that can be forcefully expired from a maximum inspiratory effort.

ERV (Expiratory reserve volume in liters) is the maximum volume of air that can be exhaled from the tidal end-expiratory position.

IC (Inspiratory capacity in liters) is the maximum volume of air that can be inhaled from tidal volume end-expiratory level; the sum of IRV and VT.

IRV (Inspiratory reserve volume) is the maximum volume of air that can be inhaled from the end-inspiratory tidal position.

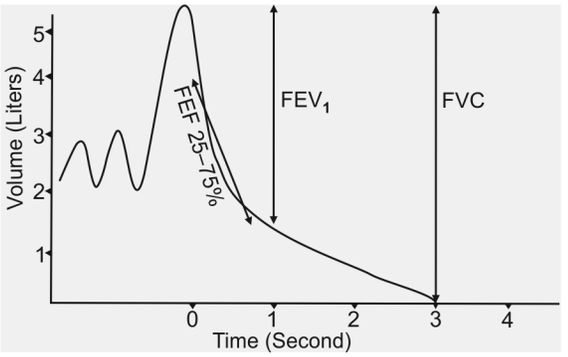
PEF (Peak expiratory flow) is the highest forced expiratory flow (L/second).

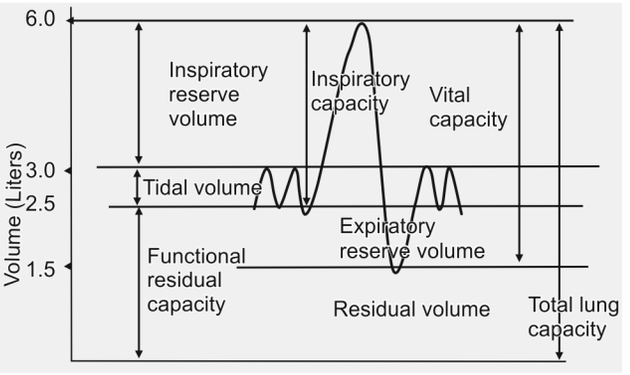
RV (Residual volume) is the volume of air that remains in the lungs after maximal exhalation.

TLC (Total lung capacity) is the total volume of air in the lungs at full inhalation; the sum of all volume compartments (IC + FRC or IRV + VT + ERV + RV).

VC (Vital capacity) is the maximum volume of air that can be exhaled starting from maximum inspiration.

TLC (Total lung capacity) can be measured either as slow vital capacity (SVC) or forced vital capacity (FVC).





What do you understand by static and dynamic lung volumes?

Static lung volumes reflect the elastic properties of the lungs and chest wall. These tests include all the capacity measurements: Vital capacity, forced vital capacity, total lung capacity and functional residual capacity. Dynamic lung volumes reflect the caliber and integrity of the airways. Spirometry records lung volume against time during an FVC maneuver.

What is a restrictive lung disease?

The test is most reliably interpreted as showing restrictive abnormality on the basis of total lung capacity. If the total lung capacity not available, one may interpret a reduction in the VC without a reduction of the FEV1/VC ratio as a restriction of the volume excursion of the lung. The severity of the abnormality might be graded as follows:

- **Based on the TLC**
  - Mild: Predicted TLC is less than lower limit of normal but ≥70%
  - Moderate: Predicted TLC < 70% and ≥ 60%
  - Moderately severe: Predicted TLC < 60%

- **Based on spirometry**
  - Mild: Predicted VC is less than lower limit of normal but ≥ 70%
  - Moderate: Predicted VC < 70% and ≥ 60%
  - Moderately severe: Predicted VC < 60% and ≥ 50%
  - Severe: Predicted VC < 50% and ≥ 34%
  - Very severe: Predicted VC < 34%

What are the causes of restrictive PFTs?

Restrictive lung volumes can be produced due to various factors that include lung pathology, pleural pathology, chest wall pathology or muscular problems.

- Lung parenchyma pathology
  - Resection of lung parenchyma e.g. prior pneumonectomy/lobectomy
  - Atelectasis
  - Fibrosis as in old healed Koch's/ interstitial fibrosis
  - Pulmonary edema as in congestive cardiac failure
  - Thickened pleura
  - Large tumor—intraparenchymal/pleural/mediastinal
  - Emphysema
- Interpleural pathology
  - Effusion
  - Cardiomegaly
  - Tumor
  - Displacement of chest wall/diaphragm
  - Ascitis
  - Pregnancy
  - Severe pain leading to splinting
  - Obesity
  - Kyphoscoliosis
  - Severe burns with contracture to scleroderma
- Neuromuscular problems
  - Phrenic nerve palsy—unilateral/bilateral
  - Any neuromuscular disorder involving thoracic muscles: Polio, Myasthenia gravis, Gullain Barre syndrome, etc.

What are the causes of obstructive PFTs?

Flow through the tubular passage of airways of the lung can be reduced for various reasons:

- Narrowing of the airways due to bronchial smooth muscle contraction: Asthma
- Narrowing of the airways due to inflammation and swelling of bronchial mucosa and the hypertrophy and hyperplasia of bronchial glands : Bronchitis
- Material inside the bronchial passage physically obstructing the flow of air: Mucus plugs, foreign objects or invasive tumors
- Destruction of lung tissue with the loss of elasticity and hence the loss of the external support of the airways : Emphysema
- External compression of the airways by tumors and trauma.

How do you grade severity of obstructive lung disease from spirometry values?

The American Thoracic Society has recommended gradation of severity as follows:

- FEV_1/FVC < predicted and FEV_1 > 100% of predicted—normal
- FEV_1/FVC < predicted and FEV_1 < 100% > 70% of predicted - mild obstruction
- FEV_1/FVC < predicted and FEV_1 < 70% > 60% of predicted - moderate obstruction
- FEV_1/FVC < predicted and FEV_1 < 60% > 50% of predicted -moderately severe obstruction
- FEV_1/FVC < predicted and FEV_1 < 50% > 34% of predicted—severe obstruction
- FEV_1/FVC < predicted and FEV_1 < 34% of predicted—very severe obstruction.

What are the criteria for reversibility of small airway obstruction on PFTs?

Patients showing small airway obstruction on PFT should be tested twice—once before bronchodilators, and then after administration of bronchodilators to evaluate the responsiveness to a bronchodilator medication. The drug almost always used is a β-2 selective sympathomimetic because it causes bronchodilation but which does not stimulate the heart to any great degree. If two out of three measurements (FVC, FEV_1 and FEF25%–75%) improve, then it can be said that the patient has a reversible airway obstruction that is responsive to medication.

1. FVC: An increase of 10% or more
2. FEV1: An increase of 200 mL or 15% of the baseline FEV_1
3. FEF25%-75%: an increase of 20% or more.

In patients with airway obstruction, absence of a response to a single exposure to a bronchodilator, however, does not preclude a beneficial response to maintenance therapy and many clinicians prefer to use bronchodilators even in the absence of above mentioned criteria.

What is MVV and its significance?

The subject is asked to breathe as hard and as fast as possible for 10–15 seconds and the value obtained is then converted to 60 seconds and reported as liters per minute. For example, if the patients MVV is 30 L in 15 seconds, it will be reported as 30 x 4 =120 L/m. This test reflects the status of the respiratory muscles, compliance of the chest wall and lung, and airway resistance. Since it parallels the

FEV_1, it can be used to test internal consistency and estimate patient cooperation. It is a quick and easy way to assess the strength of the patient's pulmonary musculature prior to surgery, a poor result on this test suggests that the patient may have pulmonary problems postoperatively due to muscle weakness. But this test is effort dependant, if patient does not perform the test properly; it cannot be then used to assess the true pulmonary strength and compliance. When the MVV is disproportionately low in a patient who seems to be cooperating, neuromuscular weakness should be suspected. Except in advanced neuromuscular disease, most patients can generate fairly good single-breath efforts (e.g. FVC). But since MVV is much more demanding, it can reveal diminished reserves of weak respiratory muscles. A low MVV can occur in any type of pulmonary disorder like obstructive, restrictive, or in patients with heart disease and very frail patients.

What are different methods of measuring residual lung volume?

1. Gas dilution techniques use either closed-circuit helium dilution or open-circuit nitrogen washout.
   - Nitrogen washout technique—In this technique, at the end of a normal expiration, the patient breathes 100% oxygen and all the nitrogen in the lung is "washed out." The exhaled volume and the nitrogen concentration in that volume are measured. The difference in nitrogen volume at the initial concentration and at the final exhaled concentration allows a calculation of intrathoracic volume, usually FRC.
   - Helium dilution technique—It is based on the inhalation of a known concentration and volume of an inert tracer gas, such as helium, followed by equilibration of 7 to 10 minutes in the closed-circuit. The final exhaled helium concentration is diluted in proportion to the unknown volume of air in the patient's chest (RV). Usually, the patient is connected at the end-tidal position of the spirometer; therefore, the lung volume measured is FRC.
2. Body plethysmography—It is based on the principle of Boyle's law, which states that the volume of gas at a constant temperature varies inversely with the pressure applied to it. In this technique, the patient sits in a closed "body box" with a known volume and the lung volumes are calculated based on the amount of air displaced from the box during ventilation.

What is the significance of vital capacity?

Vital capacity reflects the patient's ability to take a deep breath, to cough, and to clear the airways of excess secretions, therefore, is an important preoperative assessment tool. It is normally 70 mL/kg ideal body weight.

- Significant reductions in vital capacity < 20 cc/Kg of ideal body weight indicates that the patient is at a higher risk for postoperative respiratory complications.
- Evaluating the patient's condition for weaning from a ventilator. If the patient on a ventilator can demonstrate a vital capacity (VC) of 10–15 mL/Kg of body weight, it is generally considered that there is enough ventilatory reserve to try weaning and extubation.

What is the significance of RV?

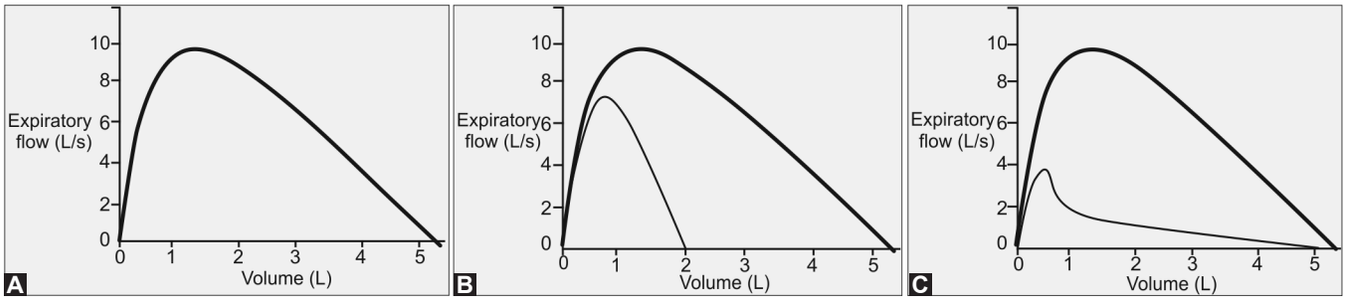
RV is the air that is remaining in lung at the end of forced expiration. It depends on two factors—limits of chest wall excursion and smaller airway collapse. In restrictive lung pathologies, RV is determined by chest wall compression by muscles. In obstructive diseases, the collapse of terminal airways prevents distal air from escaping the lungs and thus influences RV. RV and thus TLC are commonly increased in COPD patients but are normal in patients with asthma.

What is the significance of FRC?

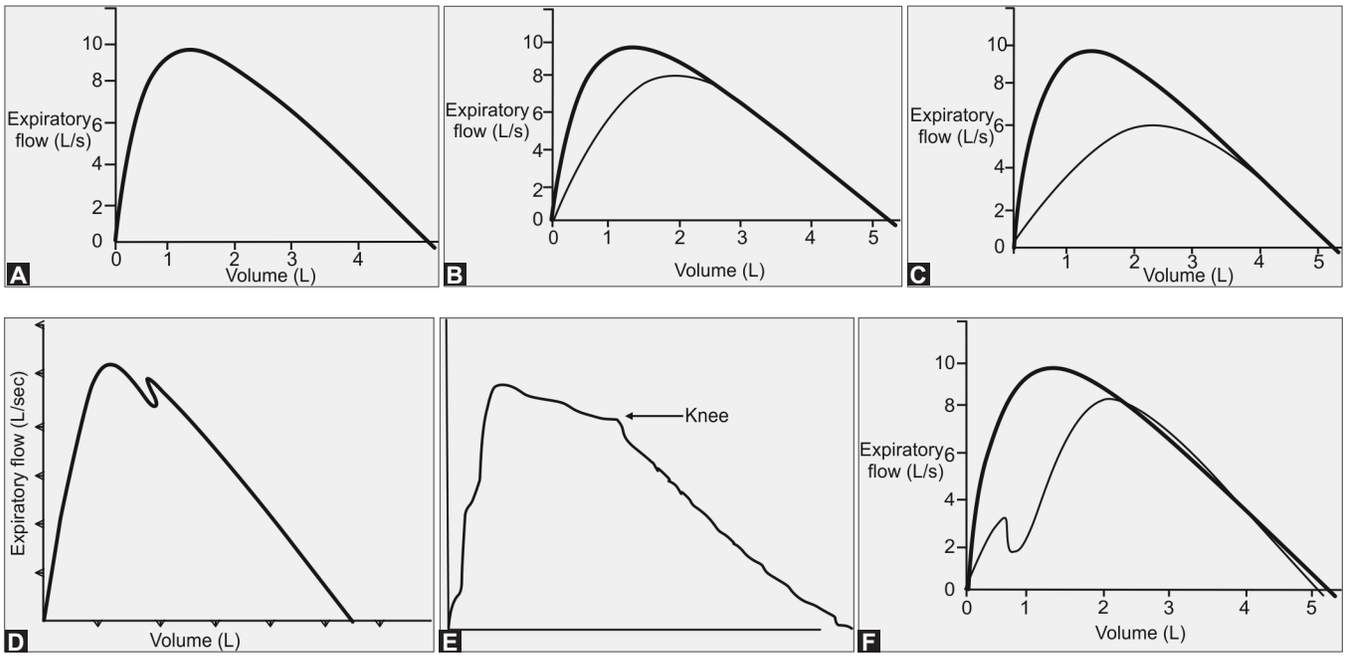
FRC is the lung volume at which the inward elastic recoil of lungs is balanced by outward elastic force of relaxed chest wall. It is usually 40–50% of TLC (30 mL/kg ideal body weight). FRC increases with reduced lung elasticity and decreases with increased lung recoil. It is also affected by posture. FRC is more in standing than supine position and this is one of the reasons why during postoperative recovery patients should be kept in head elevated position. Almost all anesthetics reduce muscle tone, which in turn lowers FRC to close to the residual volume in awake state. This is the cause of faster desaturation of obese, pregnant patients or patients with large intra-abdominal mass under anesthesia. FRC increases with age because of loss of elastic lung tissue. The preoxygenation prior to induction of anesthesia is to replace the FRC with 100% O_2 which helps in delaying the desaturation of apneic patient during intubation.

What is Gestalt approach of spirometry interpretation?

Here the patients flow volume curve is superimposed with the predicted normal flow volume curve for that patient so that instead of looking at numbers one can visually compare both curves. The difference in area provides a visual index of ventilatory limitation.



Looking at PFTs can you make out which are good and which are unaccepted maneuvers?



In the above figures figure **A** shows excellent efforts with rapid climb to peak flow followed by continuous decline of flows, figure **B** shows failure to exert maximum efforts initially but can be considered valid, figure **C** shows failure to exert maximally and test needs to be repeated, figure **D** shows coughing during the first second of expiration and hence test should be repeated, figure **E** shows knee and is a normal variant seen especially in young women, figure **F** shows hesitation in the start and test should be repeated.

What is slow vital capacity (SVC) test?

This test is performed by asking the patient to slowly and completely blow out all the air from his lungs. The SVC test eliminates the strong bronchoconstriction that usually accompanies a strong forced exhalation effort. Hence, the vital capacity of the patient may well be much larger after a SVC test because there is little or no airway collapse during a controlled and slow exhalation effort. If the vital capacity improves after a SVC test, then it can be assumed that the original small FVC was caused by airway collapse and does

not indicate the presence of restrictive disease. If the vital capacity does not improve either with the inhalation of a bronchodilator or does not improve with the administration of a SVC test, then restrictive pathologies must be considered as a possible cause for the small vital capacity results.

What is the difference between FEV1 and FEF 25%–75%?

FEV_1 represents volume expired in 1 second, reflecting average flow over first second of expiration. FEF 25%–75% is the average flow in the middle half portion of forceful expiration, starting when 25% of FVC is exhaled and ending when 75% FVC is completed. Though both measurements are reduced in obstructive airway pathology, FEF 25%–75% is supposed to reflect caliber of airways less than 2 mm in diameter (small airway), as FEF 25%–75% is measured at lower lung volumes. At lung volumes less than approximately 70% of FVC, flows become effort independent.

This effort independence is attributed to equal pressure point concept. During forced expiration, airway pressure gradually falls as air moves from distal most airways to proximal airway as airway caliber increases and corresponding resistance drops. At some point in the distal airway intraluminal pressure falls below external pressure compressing airway (pleural pressure + lung recoil pressure) and the airway collapses trapping air. The greater the expiratory effort (pleural pressure), the more proximal is equal pressure point. However the occlusion of airway interrupts airflow, intraluminal pressure rises due to trapped gas and reopens the airway. Thus at equal pressure point airflow is proportional to lung elastic recoil and inversely proportional to resistance of airway between alveoli and equal pressure point and not on pleural pressure.

What are the advantages of flow volume loop?

1. Flows at a particular point can easily be read from curve.
2. Overall shape of curve can tell about adequacy of patients effort and thus validity of report as mentioned prior.
3. Small expiratory efforts with long concave shape indicates small airway obstruction.
4. Large airway obstruction typically affect peak flow rates resulting in flat flow volume loop and depending on site of obstruction flattening occurs either in both inspiratory and expiratory limb of loop or either limb.

What is diffusion lung carbon monoxide (DLCO)?

DLCO is a measurement of the ease of transfer for CO molecules from alveolar gas to the hemoglobin of the red blood cells in the pulmonary circulation. In Europe, it is often called the transfer factor of carbon monoxide (TLCO mmol/min/kilopascal), which describes the process more accurately. DLCO is a measure of the interaction of alveolar surface area, alveolar capillary perfusion, the physical properties of the alveolar capillary interface, capillary volume, hemoglobin concentration, and the reaction rate of carbon monoxide and hemoglobin. In principle, the total diffusing capacity of the whole lung is the sum of the diffusing capacity of the pulmonary membrane component and the capacity of the pulmonary capillary blood volume.

- DLCO—The capacity of the lungs to transfer carbon monoxide (mL/min/mm Hg)
- DLCOc—DLCO corrected for hemoglobin (mL/min/mm Hg)
- DLVA—DLCO corrected for volume (mL/min/mm Hg/L)
- DLVC—DLCO corrected for both volume and hemoglobin (mL/min/mm Hg/L).

DLCO may be decreased in anemic patient as hemoglobin concentration is a very important measurement in interpreting reductions in DLCO. Because the level of hemoglobin present in the blood and diffusing capacity are directly related, a correction for anemic patients (DLCOc) is used to further delineate whether a DLCO is decreased due to anemia or due to parenchymal or interface limitation.

- Hb adjusted DLCO (adolescent males and men): Hb adjusted DLCO (DLCOc) = measured DLCO [(10.22 + Hb g/dL)/(1.7 Hb)]
- Hb adjusted DLCO (children < 15 years and women): Hb adjusted DLCO (DLCOc) = measured DLCO [(9.38 + Hb g/dL)]/(1.7 Hb)]

Reduction in the diffusing capacity is classified as:

- Mild: Less than the lower limit of normal (LLN) but greater than 60% of predicted
- Moderate: Between 40 and 60% of predicted
- Severe: Less than 40%.

For risk stratification of postoperative pulmonary complications based on DLCO refer chapter on pneumonectomy.

How is DLCO performed?

The most commonly used and standardized technique is the single-breath technique. In this technique, a subject inhales a known volume of test gas that contains 10% helium, 0.3% carbon monoxide, 21% oxygen, and the remainder nitrogen. The patient inhales the test gas and holds breath for 10 seconds. The patient exhales to wash out a conservative overestimate of mechanical and anatomic dead space. Subsequently, an alveolar sample is collected. DLCO is calculated from the total volume of the lung, breath-holding time and the initial and final alveolar concentrations of carbon monoxide. The exhaled helium concentration is used to calculate a single-breath estimate of total lung capacity and the initial alveolar concentration of carbon monoxide.

Patient should avoid smoking for several hours before the test. Alcohol vapors can affect the accuracy of some fuel cell types of CO analyzers, therefore alcoholic beverages should be withheld for 8 hours prior to test.

Are there any contraindications to perform DLCO?

Inability to follow instructions is a contraindication to a DLCO test. Patients should be alert, oriented, able to exhale completely and inhale to total lung capacity, able to maintain an airtight seal on a mouthpiece and able to hold a large breath for 10 seconds.

What are the conditions in which DLCO is affected?

Decreases in DLCO

- Obstructive lung diseases—Emphysema, cystic fibrosis
- Parenchymal lung diseases—Interstitial lung disease caused by fibrogenic dusts like asbestosis, biologic dusts like allergic alveolitis, drug reactions like amiodarone, bleomycin, Idiopathic, sarcoidosis
- Pulmonary involvement in systemic diseases—Systemic lupus erythematosus, progressive systemic sclerosis, mixed connective tissue disease, rheumatoid arthritis, Dermatomyositis-polymyositis, wegener's granulomatosis
- Cardiovascular diseases—Acute myocardial infarction, mitral stenosis, primary pulmonary hypertension, Pulmonary edema, acute and recurrent pulmonary thromboembolism, fat embolization
- Other—Diseases associated with anemia, chronic renal failure, chronic hemodialysis, marijuana smoking, cigarette smoking, bronchitis obliterans with organizing pneumonia (BOOP).

Increases in DLCO

- Diseases associated with polycythemia, pulmonary hemorrhage, diseases associated with increased pulmonary blood flow such as left-to-right intracardiac shunts and exercise.

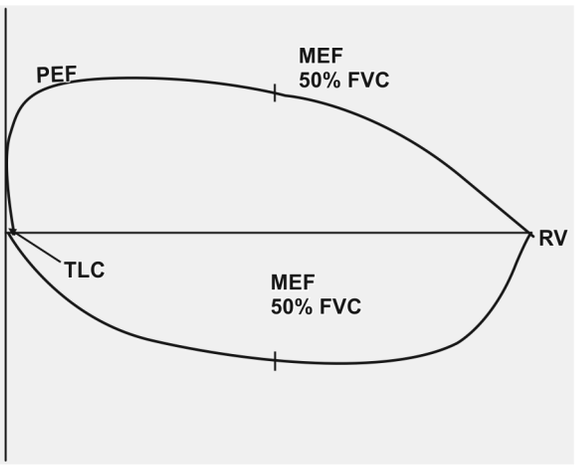
What is an exercise induced bronchoconstriction test?

Incidence of exercise induced bronchoconstriction is observed in 70–80% of patients with asthma. Before performing this test bronchodilators are withheld. Spirometry is then performed before and after 5,15 and 30 minutes postexercise. A positive test is diagnosed when FEV_1 and FVC is reduced by 15% after exercise as compared to before exercise. Exercise protocol is like stress test on treadmill where 80% of maximum heart rate is targeted to terminate exercise.

How would you diagnose site of obstruction from flow volume loops?

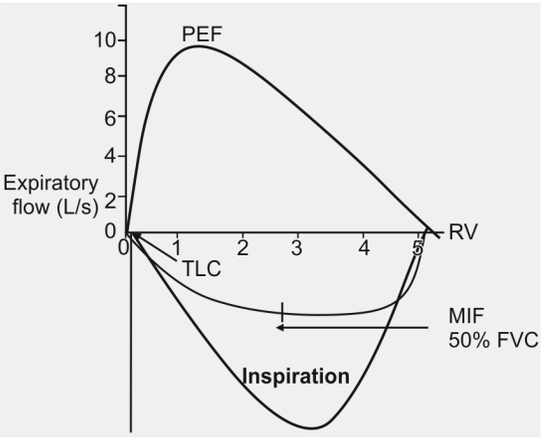
Fixed obstruction of upper airway (e.g. tracheal stenosis, bilateral vocal cord paralysis, goiter)

In fixed obstruction of the upper airway, flow is limited by the caliber of the narrowed segment rather than by dynamic compression, resulting in equal reduction of inspiratory and expiratory flow rates. The top and bottom of loop are flattened so that the configuration approaches that of a rectangle. The fixed obstruction limits flow equally during inspiration and expiration and MEF = MIF.



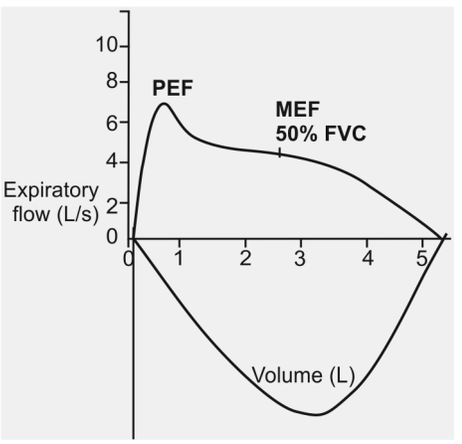
Variable extrathoracic obstruction (e.g. single vocal cord paralysis, tracheomalacia or neoplasm invading trachea)

When a single vocal cord is paralyzed, it moves passively in accordance with pressure gradients across the glottis. During a forced inspiration, it is drawn inward, resulting in decreased inspiratory flow. During a forced expiration, it is passively blown aside and expiratory flow is unimpaired, i.e. MIF 50% FVC is < MEF 50% FVC. During normal inspiration, airways within the thorax tend to dilate as the lung inflates while airways outside the thorax tend to collapse due to negative intraluminal pressure. During expiration, the reverse is true as airways within the thorax collapse but airways outside the thorax are held open by expiratory flow. As a result, a variable extrathoracic obstruction leads to flattened inspiratory limb with normal expiratory portion.



Variable intrathoracic obstruction (e.g. distal tracheomalacia, bronchomalacia):

During a forced inspiration, negative pleural pressure holds the "floppy" trachea open. With forced expiration, the loss of structural support results in narrowing of the trachea and a plateau of diminished flow (a brief period of maintained flow is seen before airway compression occurs). Thus a variable intrathoracic obstruction mainly affects the expiratory limb, again giving a flattened appearance to that aspect of the loop



Case-1

67-year old male, non-smoker, presents with carcinoma stomach for total gastrectomy.

He has a past history of pulmonary tuberculosis for which he has taken complete treatment. His effort tolerance is 1 flight of stairs.

On clinical examination—he is pale, PR- 88/min, regular BP: 140/86 mm Hg. His spirometry reveals:

Spirometry	Predicted	Actual	% Predicted
FVC	3.65	2.43	67
FEV1	2.83	2.08	73
FEV1/FVC	75	86	114

How would you interpret these values?

FVC, FEV1 both are reduced, but FEV1/FVC is normal/ increased. There is no obstructive element. This is suggestive of mild restrictive disorder.

How will you optimize this patient? What care is needed postoperatively?

This is a mild disorder; probably due to his old healed Koch's which leads to fibrosis and typical restrictive disorder.

- Check room air saturation, ABG if required (very unlikely to be abnormal)
- Bronchodilators are not of much help in restrictive pattern but if the patient has simultaneous obstructive pattern then he should be started with it.
- Chest PT/deep breathing exercises-This patient will have upper abdominal laparotomy where there is higher incidence of postoperative pulmonary complications. It is important that patient has good cough in postoperative period. Cough is related to inspiratory capacity/vital capacity, for which deep breathing would be of help. Epidural analgesia should be considered
- Counsel the patient regarding the postoperative outcome possibility of postoperative ventilation (though remote as his PFTs are just mildly deranged).

Postoperative care

- Maintain SpO_2 > 92%. Give supplemental O_2 with saline nebulization
- Early mobilization
- Chest PT/ DBE

Case-2

60-year old male with history of tobacco chewing and beedi smoking for 40 years. He is diagnosed with carcinoma of gallbladder and for radical cholecystectomy.

Spirometry	Predicted	Pre-bronchodilator actual	Pre-bronchodilator % Predicted	Post-bronchodilator actual	Post-bronchodilator % Predicted	Post-bronchodilator % change
FVC	3.37	2.5	74	2.77	82	11
FEV1	2.69	1.33	49	1.89	70	42
FEV1/FVC	76	53	70	68	90	28
FEF 25-75%	3.24	0.58	18	1.47	45	153
MVV	118	42	35	57	48	37

How would you interpret these values?

FVC, FEV_1, FEV_1/FVC and FEF 25%–75% are all reduced. MVV is decreased as well. This is suggestive of an obstructive pattern. Also a significant reversibility is noted post-bronchodilator administration.

How will you optimize this patient?

- Start/continue bronchodilators and optimize the patient on bronchodialators
- Inhaled steroids
- Chest PT/ deep breathing exercises.

How will you manage this patient in the perioperative period?

- Smooth induction and maintain deep planes on anesthesia.
- Monitor peak airway pressures (if patient develops bronchoconstriction in intraoperative period, peak airway pressures will rise).
- Since this patient is a chronic smoker, he will have some element of COPD also. Hence while ventilating this patient, keep lower respiratory rates and longer expiratory time to avoid auto-PEEP.
- Intraoperative ABG would be of help to know gas exchange status.
- If patient develops bronchospasm during intraoperative period, one can use Metered Dose Inhalers (MDIs). The circuit should not be disconnected and puffs given as the drug will not reach the lungs. For this purpose, a separate MDI adaptor should be attached to inspiratory limb of circuit before the 'Y' piece. Puffs should be given through this adaptor synchronized with inspiration. The catheter mount acts as a spacer.
- One can use IV aminophylline keeping in mind its narrow safety margin and side effects like tachycardia, tachyarrythmias.

Postoperative care

- Continue inhaled/ parental bronchodilators as per pre-operative dose schedule
- Supplemental O_2 to keep SpO_2 > 92%.
- Continue chest PT/ deep breathing exercises/incentive spirometry
- Venous thromboembolism prophylaxis.

Case-3

49-year old male patient a known case of sarcoidosis suffering from carcinoma tongue is planned for hemiglossectomy.

Spirometry	Predicted	Actual	% Predicted
FVC	2.70	1.88	70
FEV1	2.30	1.72	75
FEV1/FVC	80	91	114
MVV	92	110	119
DLCOunc	20.25	11.05	55
DLCOcor	20.25	10.95	55
DL/VA	6.10	3.46	57
VA	4.61	3.17	69

How would you interpret these values?

FEV_1 and FVC are both reduced but the FEV_1/FVC ratio is increased suggestive of mild restrictive pattern.

The diffusing capacity is also lost suggestive of loss of functional alveolar capillary membrane.

It should be remembered that in patients with interstitial lung disease spirometry values can be misleading.

The diffusing capacity decreases much before the changes in the spirometry and thus this study should be carried on regular basis for patients suffering from interstitial lung diseases to know progress of disease.

How would you optimize this patient?

- Baseline check SpO_2, ABG.
- Treatment for interstitial lung disease includes: Corticosteroid drugs, cytotoxic drugs like azathioprine, antifibrotic drugs like penicillamine. As disease advances they require oxygen therapy. Patients with very severe disease require lung transplant.

- Continue all drugs in perioperative period
- This patient will require higher FiO_2 in preoperative period and should be titrated to maintain SpO_2 >92%
- Careful premedication only in monitored environment with O_2 supplementation.

Intraoperative care

- Maintain SpO_2 and $ETCO_2$ to the baseline level. Higher FiO_2 may be required.
- Maintain a high index of suspicion for concomitant pulmonary hypertension. Echocardiography would help diagnose right heart function.
- Strict attention to fluid balance. Avoid fluid overload.

Postoperative care

- Maintain SpO_2 > 92%. Give supplemental O_2 if required.
- Progressive ventilatory compromise in these patients can lead to progressive hypoxia and hypercapnea which can be magnified by surgery and postoperative analgesia especially narcotics. Preoperative ABG should be done to know the baseline values and thus perioperative ventilatory strategies.
- Continue medications as per schedule.
- Chest PT/ deep breathing exercises / incentive spirometry
- Early mobilization.
- HDU/ ICU care.

Suggested Reading

1. Ferguson MK, Dignam JJ, Siddique J, Vigneswaran WT, Celauro AD. Diffusing capacity predicts long-term survival after lung resection for cancer. Eur J Cardiothorac Surg. 2012.
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3. http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/pulmonary/pulmonary-function-testing.
4. Kaminsky DA, Whitman T, Callas PW. DLCO versus DLCO/VA as predictors of pulmonary gas exchange. Respir Med. 2007;101: 989-94.
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43 Anesthesia Machine

N Amin, V Agarwal

Describe the parts of anesthesia machine

Anesthesia machine controls the release of pressurized gases at preset rates, adds vapour to the gases as required and delivers the mixture to a common gas outlet. Pressurized gases are supplied by cylinders or pipelines to the anesthetic machine. Continuous flow anesthetic machines date back to the first availability of compressed gases, and despite numerous modifications the modern apparatus retains many of the features of the original Boyle's machine, a British Oxygen Company trade name in honor of the British anesthetist H E G Boyle (1875-1941).

The basic structure of anesthesia machine has a steel framework which is mounted on 4 wheels. The machine can be divided into 3 parts:

- High pressure system consists of all parts of the machine which receive gases at cylinder pressure. It includes:
  - The hanger yoke which connects the cylinder to the machine, yoke block which is used to connect cylinders larger than size E or pipeline hoses to the machine through the yoke
  - The cylinder pressure gauge
  - The pressure regulator which converts the high, variable gas pressure into a lower more constant pressure, suitable for use in the machine.
- The intermediate pressure system includes components of the machine which receive gases at reduced pressures. It begins at the pipeline inlet pressures of 60 psig (50–55 psig in the US) and regulated cylinder pressures of 45 psig to the flow control valves where the pressures would be 14–26 psig. The parts of the intermediate pressure system are as follows:
  - Pipeline inlet connections which connect the machine to the hospital pipeline system.
  - Pipeline pressure gauges.
  - Ventilator power outlets which supplies driving gas to the ventilator.
  - Oxygen pressure failure devices which either interrupt the flow of anesthetic gases or provide an alarm when oxygen supply fails.
  - Second stage pressure regulators.
  - Oxygen flush which allows delivery of high flows of oxygen from the machine.
  - Flow control valves.
  - Auxiliary oxygen flow meters.
- Low pressure system takes the gases from the flow meters to the machine outlet. It is the part of the machine downstream of the flow meters in which the pressure is slightly above atmospheric. The components of the low pressure system are as follows:
  - Flow meters
  - Anti hypoxia devices
  - Vaporizers
  - Vaporizer circuit control valves
  - Back pressure safety devices
  - Outlet check valve
  - Common gas outlet

What are the various units for expressing pressure? What are their conversion factors?

The various units of pressure and their conversion factors are as follows:

100 κPa = 1000 mbar = 760 mm Hg = 1030 cm H_2O = 14.7 psi = 1 atmosphere

Therefore

1 kPa = 10 mbar = 7.6 mm Hg = 10.3 cm H_2O = 0.147 psi

How do you define compressed gas?

A compressed gas is defined as any mixture having in a container an absolute pressure exceeding 40 psi at 70^0F, or

regardless of the pressure at 70°F, having an absolute pressure exceeding 104 psi at 130°F, or any liquid having a vapour pressure exceeding 40 psia at 100°F.

Which are the compressed gases used in anesthesia?

Both liquefied and non liquefied compressed gases are used in anesthesia. The non liquefied compressed gases are oxygen, nitrogen, air and helium. These gases do not liquefy at ordinary ambient temperatures no matter how much pressure is applied. Liquefied compressed gases are gases which become liquid at ambient temperatures and at pressures ranging from 25–2500 psig. Examples are nitrous oxide, carbon dioxide.

What do Psi, Psig, Psia stand for?

Psi stands for pounds per square inch.

Psig stands for pounds per square inch gauge. It is the difference between the measured pressure and surrounding atmospheric pressure.

Psia stands for pounds per square inch absolute. Zero pressure for a perfect vacuum is taken as a reference point. Therefore Psia = psig + local atmospheric pressure.

What is the difference between gas and vapour?

A gas is a substance which is in the gaseous state at room temperature and atmospheric pressure as its critical temperature is below the room temperature.

A vapour is a gaseous substance which is below its critical temperature under ambient conditions. It exists as a liquid at room temperature and atmospheric pressure as its critical temperature is above the room temperature.

What do you mean by critical temperature? What is the critical temperature of oxygen and nitrous oxide?

Critical temperature is the temperature above which any gas cannot be liquefied no matter how much pressure is applied. The critical temperature of oxygen is -119°C due to which it exists as a gas in a cylinder at room temperature. The critical temperature of nitrous oxide is 36.5°C due to which it exists as a mixture of liquid and gas in a cylinder at room temperature. If the ambient temperature exceeds 36.5°C then nitrous oxide can only exist in the gaseous state.

What is critical pressure?

Critical pressure is the minimum pressure that is required to liquefy a gas at its critical temperature.

How and why is bulk oxygen storage done?

If oxygen consumption is very high then it is more economical to store oxygen in liquid form as a liquid would occupy only 1/860 of the space the gas would occupy. At a temperature of 15°C and atmospheric pressure, largest size J cylinder will give approximately 130 times its capacity of oxygen while one volume of liquid oxygen will give approximately 840 times its capacity of oxygen. The liquid oxygen is stored in special designed vessel called vacuum insulated evaporator (VIE). It has an outer shell made of carbon steel and an inner shell made of stainless steel. The two shells are separated by vacuum so that there is minimal temperature fluctuation in the inner shell. Oxygen is in a liquid form because the temperature inside the chamber is -153°C which is lower than its critical temperature. If there is an increase in temperature it will result in evaporation of the liquid oxygen. If oxygen is not being used it can result in pressure build up in the chamber, which is prevented by venting the excess gas through a safety valve. The pressure in an oxygen cylinder is 1900 psi whereas the pressure in a liquid oxygen tank is only 20–90 psi. There is gaseous oxygen above the liquid oxygen. When oxygen is being used it is first heated and the pressure is regulated to the pipeline pressure before delivering it to the hospital pipeline system.

What are cylinders made of?

Older cylinders were made of carbon steel and were heavier. Modern cylinders are made from alloy containing molybdenum and steel. As molybdenum steel is stronger the cylinders have thinner walls and therefore are lighter. MRI compatible cylinders are made of aluminium. The wall thickness is 5/64 to ¼ of an inch. In India the cylinders are made of manganese steel.

What are the different sizes of cylinders and what are their capacities?

Cylinders are available in various sizes. They are designated according to alphabets (size A to J). Size A is the smallest size. Size E is most commonly used in the anesthesia machine. The pressures and capacities of size E medical gas cylinders are mentioned in Table 43.1.

What do you mean by tare weight? What is the tare weight of size E oxygen and nitrous oxide cylinder?

Tare weight is weight of an empty cylinder. Tare weight of nitrous oxide cylinder is 7 kg.

What is the internal volume of cylinders of various sizes?

Sizes- D- 3 L, Size E- 5 L, Size F- 10 L, Size H- 46-50 L.

How can you measure the contents of a cylinder?

In a cylinder containing non liquefied gas such as oxygen, the pressure in the cylinder decreases as the contents are

Table 43.1 Sizes of cylinders and their content

		Oxygen	Nitrous oxide	Air	Nitrogen	Carbon dioxide
Size D	Content in L	400	940	375	370	940
	Pressure in psig (kPa)	1900 (13700)	745 (4400)	1900 (13700)	1900 (13700)	838 (5000)
Size E	Content in L	660	1590	625	610	1590
	Pressure in psig (kPa)	1900 (13700)	745 (4400)	1900 (13700)	1900 (13700)	838 (5000)
Size H	Content in L	6900	15800	6550	6400	
	Pressure in psig (kPa)	2200	745 (4400)	2200	2200	

used. Therefore the pressure on the pressure gauge can be used to measure the cylinder contents. The volume of the contents available at atmospheric pressure can be estimated using Boyle's law which states that at constant temperature pressure is inversely proportional to volume. Therefore if the temperature is constant then according to Boyle's law $P_1 \times V_1 = P_2 \times V_2$.

Example – We know that the volume of size E cylinder is 5 L (V_1). If the pressure gauge reads 1000 psi (P_1) then to know how much of oxygen (V_2) will evolve at atmospheric pressure of 14.7 psia (P_2) we have to apply Boyle's law.

$1000 \times 5 = 14.7 \times V_2$ Therefore $V_2 = 1000 \times 5 \div 14.7 = 340$ L.

If pressure gauge reads 600 psi then $V_2 = 600 \times 5 \div 14.7 = 204$ L.

Another method

A full cylinder at 1900 psi will evolve 660 L of oxygen, then at a pressure of 1000 psi how much oxygen will evolve? 1000 ÷ 1900 = 52% full hence it will evolve 660 x 52% = 340 L.

If pressure gauge reads 600 psi then it is 31% full and it will evolve 660 x 31% = 204 L.

If the cylinder contains a liquefied gas such as nitrous oxide then the pressure in the cylinder would not be indicative of the contents of the cylinder. This is because the pressure in the cylinder depends on the vapour pressure of the liquid gas. As a result the pressure would remain constant until all the liquid has evaporated, after which the pressure decreases as the cylinder empties. Hence for liquefied gases the weight of the contents of the cylinder can also be estimated by weighing the cylinder and subtracting the weight of the empty cylinder or the tare weight.

By Avogadro's Law, 1 gram molecular weight (i.e. the molecular weight in grams) of any gas or vapour occupies 22.4 L at standard temperature and pressure [STP: 0°C (which is 273 degrees Absolute or 273 Kelvin) and 760 mm Hg pressure (or one atmosphere)]. According to Charles' Law, at a constant pressure the volume of a fixed mass of gas is directly proportional to Absolute temperature. Room temperature is usually 20°C or 293 Kelvin (i.e, 273 + 20). Therefore, 32 grams of oxygen or 44 grams of nitrous oxide will occupy 22.4 x 293 ÷ 273 or 24 L at 20°C.

Example: How much nitrous oxide will evolve from a cylinder weighing 8 Kg?

Assume tare weight of size E nitrous oxide cylinder is 7 kg. The pressure gauge reads 745 psi and the weight of the cylinder is 8 kg then how much nitrous oxide will evolve at 20°C at atmospheric pressure. The mass of nitrous oxide is 1000 gms (8kg – 7kg). We already know that 44 gm of nitrous oxide will occupy 24 L.

The amount of gas that will evolve from 1000 gms will be 1000 x 24 ÷ 44 = 545 L.

If the weight of the cylinder is 7.5 kg then the mass of nitrous oxide is 500 gms and the amount of gas evolved will be 500 x 24 ÷ 44 = 272 L.

If all the liquid nitrous oxide has been used up and the cylinder contains only gaseous nitrous oxide then at this point we can apply Boyle's law to know how much nitrous oxide will evolve at atmospheric pressure. If cylinder contains only nitrous oxide gas and has a pressure of 745 psi (P_1).

Then: $745 \times 5 = 14.7 \times V_2$, therefore $V_2 = 745 \times 5 \div 14.7 = 253$ L. When the cylinder contains only nitrous oxide gas, it is 16% full (253/1590 L).

If the pressure gauge reads 300 psi then it will evolve 300 x 253 ÷ 745 = 100 L.

Is the pressure gauge a reliable indicator of the contents of a cylinder?

The pressure gauge would be a reliable indicator of the contents of a cylinder containing non liquefied compressed gases. However it is not a reliable indicator of the contents of cylinders containing liquefied gases like nitrous oxide. This is because; while using a nitrous oxide cylinder the pressure can remain constant if the temperature is constant. A constant temperature would ensure the vapour pressure

remains constant and the pressure would not change as the cylinder is being used. However in practice the temperature does not remain constant. Heat energy is required for the evaporation of the liquid in the cylinder. As the temperature falls the vapour pressure also falls. There is a progressive fall in the cylinder pressure as the nitrous oxide is used. The rate at which the pressure falls is related to the flow rate.

Why do you get frost on the outer surface of nitrous oxide cylinders?

When a liquefied gas like nitrous oxide is being used, the latent heat of vaporization causes cooling of the cylinder. The fall in the temperature of the cylinder is much more if the high flow rates of nitrous oxide are used. As the cylinder cools, water vapour from the surrounding can condense on the external surface of the cylinder. If the temperature is very low the water vapour may even freeze resulting in frost formation on the outer surface of the cylinder. Cooling of nitrous oxide cylinder during use indicates that there is residual liquid nitrous oxide in the cylinder.

How are the cylinders tested?

Periodic visual inspection of the cylinder to check for any distortion, cracks, dents, leaks, etc.

Tensile test on 1 out of every 100 cylinders manufactured. Strips of the cylinder are cut and stretched.

Flattening test, Bend test and impact test are carried out on at least one in every 100 cylinders

Hydraulic test—the cylinder is tested to 1.66 times it service pressure. This helps detect any leaks and to determine the retention of structural strength. A cylinder must be subjected to internal hydrostatic pressure testing at least every 5 years. With a special permit it can be extended up to every 10 years.

What precautions should be taken while transporting a cylinder?

- Upright position
- Wear protective footwear/safety gloves
- Do not lift by protective cap/guard as you may damage valve
- Ensure all cylinders are properly labeled as to the contents
- Do not subject cylinders to temperature extremes
- Do not drag slide or roll- use cylinder cart
- Cylinders should be secured at all times.

What should you check while receiving a gas cylinder?

When a gas cylinder is received, it shall be checked for the following:

- A stamped hydraulic test date within the last five years
- A stenciled or labeled identification of its contents
- Cylinder is in an acceptable condition, and
- Presence of a valve protection cap.

Cylinders should be rejected if the test date, identification, markings or cap are not in order or if the cap is rusted or inoperable.

What precautions should be taken while storing compressed gas cylinders?

- Cylinders should not be stored in the operation theatres. There should be a designated area for storing cylinders.
- Storage area should be well ventilated, cool and clean and made of fire resistant materials.
- Larger cylinders should be stored in upright position and secured with chains. Smaller cylinders should be stored horizontally in racks.
- Cap cylinders when not in use.
- Ensure all cylinders are properly labeled as to the contents.
- Always secure cylinders with chain or cart.
- Do not secure more than four cylinders in any one row.
- Keep cylinders away from radiators and other sources of heat.
- Do not subject cylinders to temperature extremes.
- Separate storage of flammable and oxidizing cylinders by at least 20 feet or a fire resistant barrier of at least five feet high and having a fire rating of at least one hour.
- Mark used cylinders "Empty". They should never be stored with other full cylinders.
- Do not store cylinders where water is freestanding or may collect.
- Do not store cylinders where they can become part of an electrical circuit.
- Do not store cylinders in hallways, closets or vestibules.
- Do not store cylinder in front of eyewash/emergency shower stations, spill kits or fire extinguishers.
- Cylinders should not be exposed to continuous dampness, corrosive chemicals or fumes.
- Cylinders should not be covered during storage. Wrappings should be removed from the cylinders during storage.

What is the service pressure of a cylinder?

The service pressure is the maximum pressure to which the cylinder may be filled at 70°F.

What are the filling limits for any cylinder? Why do you need filling limits?

When a gas is kept in a closed container, a rise in temperature will cause a rise in pressure. If a cylinder which has been

filled to a safe pressure at normal temperature is subjected to very high temperatures, the pressure can increase to very dangerous levels. This can even cause the cylinder to explode. In order to prevent this from happening the Department of Transportation has established regulations limiting the amount of gas a cylinder may contain.

Filling limits for non liquefied gases:

- The pressure in the filled cylinder at 70°F should not exceed the service pressure marked on the cylinder. However some non-liquefied gases such as oxygen, helium, carbon dioxide-oxygen mixtures, helium-oxygen mixtures which may be allowed an additional 10%.
- At 130^{0}F, the pressure in cylinders containing non liquefied gases may not exceed 1¼ times the maximum permitted filling pressures at 70°F.

Filling limits for liquefied gases:

In cylinders containing liquefied gases the pressure will remain nearly constant as long as there is liquid in the cylinder. To prevent the cylinder containing liquefied gas from being overfilled, the maximum amount of gas that can be contained is defined by a filling ratio for each gas.
Nitrous oxide- 68%
Carbon dioxide- 68%
Cyclopropane- 55%

Define filling density (Filling ratio)

Filling ratio is the percent ratio of the weight of the gas in the cylinder to the weight of the water a cylinder can hold at 60°F.

Is the filling ratio for nitrous oxide same in all the countries?

In tropical countries the filling ratio is 0.68 and in temperate climates it is 0.75.

How would you identify the contents of a cylinder?

The cylinder contents can be identified by the color of the cylinder and the label on the cylinder. Each cylinder must have a conspicous label which contains all the important information. It mentions the name, chemical symbol and physical state of the contents of the cylinder. It also mentions the maximum cylinder pressure, the cylinder contents in L, cylinder size code and product licence number. The label not only helps to identify the contents of the cylinder, it also gives a warning of the principal hazards associated with the cylinder or its contents. As there are many medical gases available the cylinders are color coded for a specific gas as an added safety feature. The chemical symbol of the gas contained in the cylinder is marked on the cylinder valve.

Table 43.2 Identification of cylinders through color coding

Gas	International	United states
Oxygen	Black body and white shoulders	Green
Nitrous oxide	Blue	Blue
Carbon dioxide	Grey	Grey
Air	Black body and shoulders with black and white quarters	Yellow
Helium	Brown	Brown
Entonox	Blue body and shoulders with blue and white quarters	
Cyclopropane	Orange	Orange

Just like United States there are many other countries which do not follow the international codes, hence colour coding is only an aid to identifying the cylinder contents and cannot be completely relied upon. The label is the primary means of identification. In India we follow international color code.

Can you describe the cylinder valve?

Cylinders are filled and discharged through the cylinder valves. The valves are attached to the neck of the cylinder by means of tapered threads. The valves are made of brass with chromium plating. Small cylinder valves are used for cylinder sizes A to E and large cylinder valves are used on cylinder sizes F to H. Each valve has the following parts:

- The body which forms the basic structure.
- The port is the point of exit for the cylinder contents. It fits over the nipple of the yoke (on small cylinders) or the regulator (on large cylinders). Any damage to the port may prevent a tight seal and result in a leak. Hence the port should be covered during transit. While installing the cylinder if the port is mistaken for the conical depression on the opposite side of the valve then, inserting the retaining screw into the port may damage the port and the index pins.
- The stem or shaft, when rotated will either open or close the valve. The stem has to be rotated in the anticlockwise direction to open the valve and in the clockwise direction to close it.
- There is a handle or hand-wheel for turning the valve stem.
- Safety relief device which allows discharge of cylinder contents to the atmosphere under certain conditions of exposure.
- There is a conical depression on the small cylinder valves just above the safety relief device. It receives the retaining screw of the yoke. If the retaining screw is tightened into the safety relief device then the device may be damaged and the cylinder contents may escape.
- The pin index is a non-interchangeable safety system which prevents the attachment of the incorrect cylinder to the yoke.

What are the differences between direct and indirect acting valves?

Table 43.3 Differences between direct and indirect acting valves

Direct acting	Indirect acting
Packed valves	Diaphragm valves
Turning the stem causes the seat to turn	Turning the stem moves a metal diaphragm against the opposing force of spring acting on the seat assembly
Stem is sealed by resilient packing like teflon	No packing
Less expensive.	More expensive
Good performance with gases under high pressure.	It is used for gases like nitrous oxide which liquefy under pressure. These valves operate better at low temperatures which are produced when these liquids vaporize. Less wear and tear on the seat These valves are tighter at the stem and the seat does not turn, therefore they are less susceptible to leaks
These valves open fully in 2-3 full turns.	These valves open fully in ½ to ¾ turns

Most cylinder valves are of the packed type.

What safety feature in a cylinder valve protects against build up of high pressure in the cylinder? Can you describe any one of them?

All cylinder valves have a safety relief device which is designed to prevent rupture of the cylinder under certain conditions of exposure. The safety relief devices are of 3 types namely frangible disc assembly, fusible plug and safety relief valve.

Frangible disc assembly: It consists of a frangible disc and a safety cap. The ports of this assembly may be filled with a fusible alloy. The disc is made of copper. This disc closes the discharge channel and prevents the release of the contained gas. The bursting pressure is the minimum pressure for which the disc is designed to burst so that the gas can escape through the discharge channel. The burst pressure is determined by the material, thickness and shape of the disc and the diameter of the discharge channel. It is a non reclosing device and is used in oxygen, air, nitrous oxide, carbon dioxide, nitrogen, helium and carbon dioxide-oxygen and helium-oxygen cylinders. It protects against high pressures resulting from high temperature or overfilling.

Fusible plug: It is usually made of a metal alloy with low melting point. This alloy occludes the discharge channel and melts at a predetermined temperature. Woods metal alloy is most commonly used. The yield temperature is the temperature at which the alloy will melt. It protects against excessive pressure resulting from high temperature but not from overfilling.

A combination of frangible disc and a low melting point fusible plug is available. This prevents bursting at a predetermined pressure unless the melting temperature has also been attained. This device does not protect against high pressure due to overfilling.

Safety relief valve: It consists of a valve seat which occludes the discharge channel. It is held in place by a spring. When the set pressure is exceeded the valve opens and the gas escapes through the vents in the safety valve. Once the pressure is reduced the valve closes again. This type of pressure relief device is more susceptible to leaks.

What is woods metal?

Woods metal is a fusible alloy which contains bismuth, lead, tin and cadmium. Depending on the composition it melts at two different temperatures either 70°C–75°C or 95°C–105°C. It is named after American metallurgist B. Wood.

Are there any other safety features on the cylinder valve?

The pin index safety system on the cylinder valve prevents an incorrect cylinder from being attached to the anesthesia machine. It was first used in 1952. This is an additional safety feature as the color coding of cylinders is not a foolproof method of protecting against human error.

What is the pin index safety system?

The pin index safety system is used in small cylinder valves (size E or less) with yoke type connections. It consists of 2 pins projecting from the inner surface of the yoke. They are positioned in such a way that they fit into two corresponding holes drilled into the cylinder valve. The holes are positioned in an arc below the outlet port on the cylinder valve. The arc has a radius of 9/16 inch. The six holes are placed at an angle of 12^0 from the port, while the seventh hole is at the middle of these holes. The port will not seat against the washer of the yoke unless the pins and holes are aligned. The pins are 4 mm in diameter and 6 mm long. Pin 7 is slightly thicker.

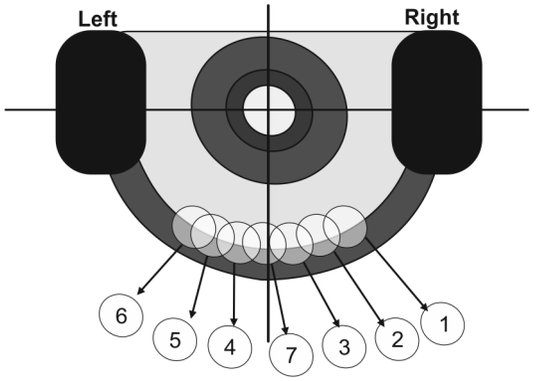


Fig. 43.1 Pin index safety system

The pin index for different gases is as follows:

Table 43.4 Pin index for different gases

Gas	Pin index
Oxygen	2,5
Nitrous oxide	3,5
Carbon dioxide	3,6
Air	1,5
Cyclopropane	3,6
O_2-CO_2 (CO_2 < 7.5%)	2,6
O_2-CO_2 (CO_2 > 7.5%)	1,6
O_2-He (He > 80.5%)	4,6
O_2-He (He < 80.5%)	2,4
Entonox	7

The pin index system is also used when the cylinders are being refilled. This prevents a wrong gas from being filled into the cylinder.

What are the fallacies of the pin index safety system?

In spite of having a pin index system, one can still attach a wrong cylinder if the pins on the yoke are broken. If someone places 2 washers on the port then the pin index would be nullified and a wrong cylinder can be placed on the yoke.

What is the safety feature for valve outlet connections for large cylinders?

As the large cylinders cannot directly connect to the yoke, the pin index system cannot be used. The valves of large cylinders have threaded outlet which mesh with those of the nut. When the nut is turned clockwise the nipple seats against the valve outlet and the gas channel of the valve is aligned with the channel of the nipple. Different gas cylinders will have different threaded valves. The valve threads may be external, internal, right hand, left hand. Also the diameters of the threads vary. Further separation is made by varying the size and shape of seats and nipples for any given thread size.

Why do you need to open the cylinder valve slowly?

When the cylinder valve is opened very fast, the gas quickly passes into the space between the cylinder valve and the yoke or regulator. The sudden recompression of the gas generates large amounts of heat (Joule-Thompson effect). Since there is very little time for the dissipation of heat, it results in an adiabatic process (heat is neither gained nor lost from the environment). If there is any dust or grease in this space, it can be ignited by the heat causing flash fire or explosion. When the valve is opened slowly, the recompression of the gas is prolonged. This allows some time for the dissipation of heat thereby reducing the risk of fires and explosions. Also when the valve is opened rapidly the sudden release of gases may damage the pressure gauge and regulator attached to the machine. Before the cylinder is fitted to the machine the valve should be opened slowly till the pressure on the gauge stabilizes. The gas which escapes helps to blow off the dirt present on the valve outlet. This dirt can damage the pressure regulator or even cause an explosion.

The cylinder valve if it is too tight, so the OT attendant gives a suggestion of applying grease on the valve cylinder. What will you do?

You should not apply grease on the valve cylinder. The reasons have already been mentioned above.

What do you mean by cracking the valve?

Before connecting the cylinder to the anesthesia machine the protective cap should be removed and the valve should be opened slowly and briefly. While opening the valve the port should point away from the user or other personnel. This is known as cracking the valve. It helps to clear dust and other foreign materials from the valve outlet before it is connected to the machine. It reduces the possibility of fires and explosions when the valve is opened after connecting it to the machine. It also prevents the dust from being blown into the machine and clogging the filters.

Is it a safe practice to keep cylinder valve open at all times?

No, the cylinder valve should not be kept open at all times as it can result in slow depletion of gases from the cylinder. Usually the pipeline pressures are higher than the regulated cylinder pressures. If the cylinders are kept open, then whenever the pipeline pressure drops below the cylinder pressure (during the use of ventilators or oxygen flush) gases will preferentially be used from the cylinder. This will result in depletion of gases from the cylinder and no reserve will be available if the pipeline supply fails. Hence the cylinder should only be opened while checking the cylinder or when the pipeline is not available.

Can you summarise the safety features of cylinders?

- Label on the cylinder
- Pin index safety system
- Safety relief valve on the cylinder
- Symbol of the gas on the cylinder valve
- Color coding.

What is entonox?

Entonox is the trade name for a compressed gas mixture containing 50% oxygen and 50% nitrous oxide by volume.

Entonox is stored in cylinders at a pressure of 13700 kPa (1900 psig). It is mainly used for labor analgesia.

What is the Poynting effect?

Poynting effect or overpressure effect is the change in the critical temperature and pressure of one gas when it is mixed with another gas. Poynting effect is seen in Entonox cylinders which contains a mixture of 50:50 of nitrous oxide and oxygen. Normally oxygen is stored in the cylinder in a gaseous state as its critical temperature is very low (-119°C) whereas nitrous oxide is stored as a liquid. When gaseous oxygen is bubbled through liquid nitrous oxide there is vaporization of the liquid resulting in the formation of mixture of oxygen and nitrous oxide in a gaseous state. Thus due to the Poynting effect nitrous oxide which is normally present in cylinders as liquid, is present as a gas in the mixture with oxygen. The Poynting effect reduces the critical temperature of N_2O so Entonox has a pseudocritical temperature of -6°C. The physical property of entonox gas is different from the individual gasses.

The cylinders should be stored in horizontal position at a temperature between 10°C – 38°C. It is important to store Entonox cylinders above its pseudocritical temperature of -6°C. If the temperature drops below -6^{0}C there is liquefaction and separation of its two components. This is known as lamination. There is a liquid mixture which mostly contains nitrous oxide with about 20% oxygen dissolved in it. Above this liquid is a gas mixture containing a high concentration of oxygen. As the cylinder is being used initially a high concentration of oxygen will be supplied. As the liquid evaporates the concentration of oxygen will go on decreasing. When the cylinder is nearly empty the patient will get hypoxic mixtures containing less than 20% oxygen. This process can be reversed by rewarming the cylinder and mixing its contents. If a cylinder has been exposed to cold below -6°C it should be warmed for 5 minutes in a 37°C water bath or for 2 hours in a room at 15^{0}C. It should then be inverted three times before use.

How can liquefaction and separation of Entonox be prevented?

To prevent liquefaction and separation of Entonox the cylinders should be stored in horizontal position at a temperature of 5°C for at least 24 hours before use. The horizontal position increases the area of diffusion. The cylinder should also be inverted several times before use. In large cylinders there is a dip tube with its tip ending in the liquid phase. This ensures that the liquid is used first, thus preventing the delivery of oxygen at concentration less than 20%.

Can you describe the hanger yoke assembly?

The hanger yoke orients and supports the cylinder, provides a gas tight seal and ensures unidirectional flow of gases to the machine. It consists of following parts:

The body is the main framework and supporting structure. It has a toggle handle or swivel gate which cannot be closed if the cylinder valve is not properly placed.

The retaining screw tightens the cylinder into the yoke and helps to establish a seal. The tip of the retaining screw fits into the conical depression on the cylinder valve.

The nipple is the part of the yoke through which the gas enters the machine. It fits into the port on the cylinder valve.

The index pins prevent the attachment of an incorrect cylinder to the yoke.

The washer helps to form a seal between the cylinder and the yoke.

Filter is present between the cylinder and the regulator to prevent particulate matter from entering the machine and damaging the components of the machine.

Check valve assembly which ensures a unidirectional flow of gases through the yoke. It prevents the flow of gases from the machine or from another cylinder to atmosphere when the yoke is empty. When double yokes are present the check valves prevents transfer of gas from a cylinder at high pressure to a cylinder at low pressure. It also allows an empty cylinder to be exchanged with minimal loss of gas, even as the gas flow continues from the other cylinder.

What are the fallacies of the check valve assembly?

The check valves do not act as permanent seals for empty yokes. Small amounts of gas can escape especially if the cylinder pressure is low. In order to minimize the losses, the yokes should not be left vacant for extended periods. An empty cylinder should be replaced as soon as possible. The valve of the empty cylinder should be closed if it cannot be replaced. A yoke plug can also be used to prevent gas leak. Also if the check valve is defective then trans-filling of the cylinders can occur. In order to avoid this only one cylinder should be opened at a time.

Can you enumerate the uses of yoke block?

Yoke blocks are a piece of metal shaped like a cylinder valve. They have a port and conical depression to fit the yoke. They are also pin indexed. They are used to:

- Connect cylinders larger than size E to the machine.
- To connect pipeline supply to the machines that does not have pipeline connections.
- They are used to block the yoke when there are no cylinders present. This helps prevent small gas leaks from the machine if the check valves are defective.

Why are yoke blocks no longer used nowadays?

The use of yoke blocks have been associated with several hazards and hence are no longer used nowadays. If the yoke block is used to connect a high pressure cylinder without a regulator, then the hose carrying the gas at high pressure can pose a hazard to personnel if it leaks or breaks. It is safer to use pipeline inlet connections than yoke blocks because yoke blocks can be responsible for crossover of gases in the anesthesia machine. Some yoke blocks do not have pin index system and can be inserted into any yoke. The pin indexed yoke can be inserted upside down if they have a short top. The pressure regulators in the machine which are designed to accept gases at cylinder pressure may not function properly when supplied with gases at reduced pressure. This can lead to fluctuations of gas pressure supplied to the flow control valve. Due to these reasons yoke blocks are no longer used and pipeline inlet connections are used whenever cylinders larger than size D or E are used.

Can you describe the cylinder pressure gauge?

The cylinder pressure gauge is bourdon tube type. It indicates the pressure in the cylinder when the cylinder valve is opened. It has a hollow metal tube which is bent into a curve. One end is sealed and the other end is open. The sealed end is connected to a clock-like mechanism. The open end is connected to a gas source and soldered into a socket. There is a constriction at the entry point of the gas. This helps to prevent any pressure surges that can damage the gauge. It also prevents rapid loss of the gas when the pressure gauge raptures. When the cylinder valve is opened the gas enters the tube. The high pressure in the tube causes it to straighten. The movement of the sealed end is transmitted to the indicator attached to the clockwork mechanism. The indicator moves on a scale calibrated in units of pressure. They have a heavy glass window and a safety flap on the back. If the bourdons tube raptures then the gas will be vented from the back of the case rather than blowing out the glass window in the front. The full scale pressure indication is at least 33% greater than the maximum cylinder pressure. The pressure gauges are color coded and also identified by the name or symbol of the gas being measured. They must be positioned in such a way that they are easily visible and not covered by other items.

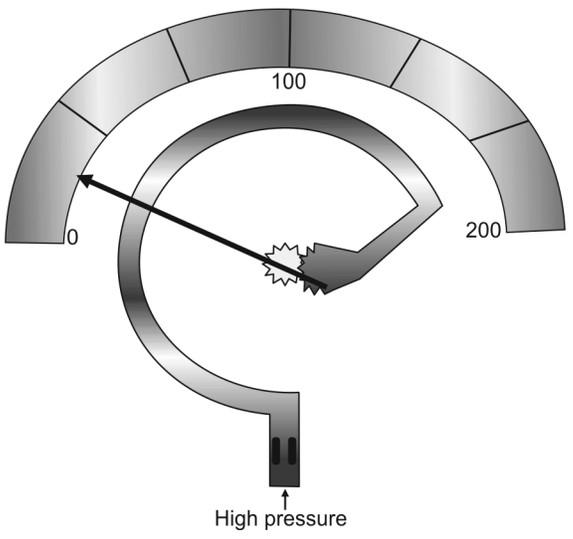


Fig. 43.2 Burden pressure gauge

Why do you need pressure regulators?

- The pressure reducing valves reduce the variable high pressure in the cylinder to a constant low pressure suitable for use in the anesthesia machine. The cylinder pressures are reduced to 375 kPa (45 psi).
  - When a cylinder is being used the pressure decreases due to which the flow also reduces. As a result the flow control valve has to be frequently adjusted to maintain a steady flow. Pressure regulators were used to ensure constant flow with changing supply pressure thus eliminating the need for frequent adjustments in the flowmeter.
- When there is high pressure it is difficult to make small adjustments in the flow meter. By lowering the pressures it allows fine adjustments to be made easily.
- Also lowering the pressure reduces the possibility of dangerous pressure build up which can damage the anesthesia machine.

What is the working principle of a pressure regulator?

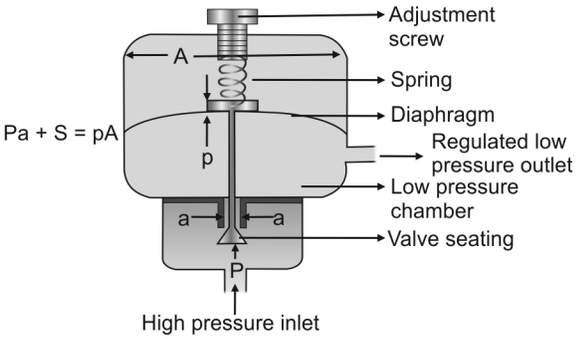


Fig. 43.3 Pressure regulator

Pressure is defined as force acting against an area. The force can be increased by increasing the pressure or by increasing the area over which the pressure acts. In a pressure regulator, large pressure (P) acting over a small area (a) is balanced by smaller pressure (p) acting over a large area (A).

The pressure regulator has a high pressure chamber with an inlet that has a filter. The high pressure chamber has a valve which leads to a low pressure chamber with an outlet. The low

pressure chamber has a diaphragm attached to a spring. The gases from the cylinders pass through the inlet into a high pressure chamber. The gases then pass through the valve into the low pressure chamber. The increased pressure in the low pressure chamber tends to distend the diaphragm. The distension of the diaphragm is opposed by the tension in the spring (S). When there is sufficient increase in pressure the diaphragm moves and the valve closes. The pressure at which the valve closes can be preset. This is done by adjusting the screw which alters the tension in the spring. When the gas is allowed to flow through the outlet, there is a drop in pressure in the low pressure chamber and the valve opens. If the outlet is closed the pressure again builds up and the valve closes. Thus the force exerted by the high pressure gases tries to close the valve while the opposing force of the diaphragm and spring tries to open the valve. The balance between the two opposing forces helps to maintain a constant gas flow.

What is Adams valve?

Adams valve is a pressure regulating valve which was used in the past. The working principle is the same as the newer pressure regulators, but the internal structure is different. Instead of the push rod there is lazy tongs toggle arrangement. It reverses the direction of thrust transmitted from the diaphragm.

Why do you have fins on the Adams regulator for nitrous oxide?

Earlier nitrous oxide contained significant amounts of water vapor as impurity. When nitrous oxide was being used, the cooling of the valve caused condensation of the water vapor on the valve seating. When there was excessive cooling the water vapor used to freeze and jam the valve. The fins on the nitrous oxide regulator helped in conducting heat from the surrounding. This prevented excessive cooling of the valve which in turn prevented the freezing of water vapor. Modern pressure regulators do not have fins as nitrous oxide does not have water vapor as impurity.

What are the safety features on pressure regulators?

Pressure regulators have safety relief valves downstream of the regulators. If due to any reason there is build up in the pressure regulator then the safety valve blow off at a set pressure of 525 kPa (70 psi). The spring loaded valves close when the pressure reduces. Some valves may rapture and remain open till they are repaired. The safety feature is not present in Boylis Machine but present in newer machines

The older pressure regulators were coded and labelled for specific gases.

What is the normal working pressure in the anesthesia gas machine and cylinders?

The hospital pipeline is the primary source of all gases and the pressure within the pipelines is 60 psig (50 psig in US), which is the normal working pressure of most machines. The oxygen cylinder pressure is 2000 psi which is regulated to approximately 45 psi after it enters the machine. A full nitrous oxide cylinder pressure is regulated from 745 psi to approximately 45 psig. Air cylinder pressures are similar to oxygen.

Why is the regulated cylinder pressure lower than the pipeline pressure?

The pipeline pressures are 55–60 psi and the regulated cylinder pressures are 45 psi. Regulating the cylinder pressure below the pipeline pressure reduces the risk of premature emptying of cylinders if the cylinders are accidentally left open. Even if the cylinder is left open the pipeline gas will be preferentially used.

What are the safety features in the pipeline system?

The terminal wall units for the pipelines have Schrader socket assembly which accepts the Schrader probe for a specific gas. The terminal wall units are colour coded and the identity of the gas is also mentioned on the socket. The socket assembly has a primary valve (automatic shutoff valve) which allows the gas to flow when the pipeline is inserted and which closes automatically when the pipeline is disconnected from the wall outlet. The socket assembly also has an isolating valve which prevents any gas leak when the primary valve is removed for cleaning or replacement.

The pipeline system consists of flexible non crushable hoses which connect the terminal wall outlets to the anesthesia machine. The pipeline has a Schrader probe which fits into the Schrader socket in the terminal wall outlet. Though the size of the Schrader probe is the same for all gases, the probes have a collar indexing system specific for a gas. The collar for each gas has a unique diameter which will fit the matching recess on the Schrader socket assembly for that gas. Nowadays instead of Schrader probes there are quick connectors which are gas specific and manufacturer specific. The quick connectors on the wall outlet will not accept pipeline schrader probe. The pipeline tubing's are also color coded.

The machine end of the pipelines has the diameter index safety system (DISS) or non interchangeable screw thread system (NIST) to minimise the risk of misconnections. The connections for oxygen, nitrous oxide and air have different diameters.

There are check valves at the pipeline inlets to the machine, which prevent the backflow of gas from the machine to the pipelines or to the atmosphere when pipelines are not attached to the machine.

How will you shut off the flow of gases if there is leakage from the wall outlet?

If there is any need for maintenance, repair, testing or expansion then the shutoff valves can be used to isolate specific areas of the pipeline system. These valves are of 2 types. The manual shutoff valves are easily visible and can be accessed at all times. The service shutoff valves are secured and locked to prevent accidental closing and can be accessed only by authorized personnel. For all the pipelines entering the operation theaters, there is a shutoff valve (bypass valve) just outside the operation theaters. If there is any gas leakage from the wall outlet or the pendants then this bypass valve can be used to shut off the gas supply to the operation theaters.

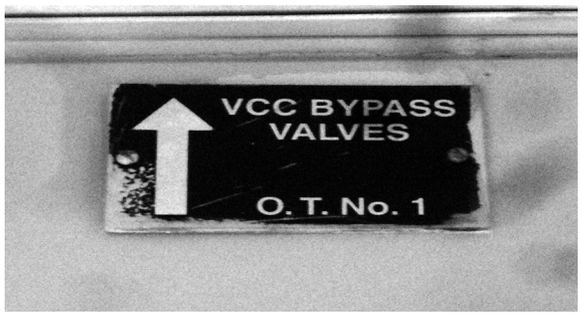


Fig. 43.4 Shutoff valve

What should be the ideal position of pipeline pressure gauge?

The pipeline pressure gauge should ideally be on the pipeline side of the check valve. If it is on the machine side of the check valve it will detect the machine pressure and not the pipeline pressures.

How would you know what is the position of the pipeline pressure gauge?

The pipelines should be disconnected and the cylinders should be opened. If the pipeline pressure gauge reads zero then the pressure gauge is on the pipeline side. If there is a reading then it is on the machine side.

Why do some machines have secondary pressure regulators?

Some machines have secondary pressure regulators in the intermediate pressure system just upstream of the flow meters. They receive the gas from the pipeline or cylinder regulator and reduce it to 14 psig for oxygen and 26 psig for nitrous oxide. The use of ventilators and oxygen flush can sometimes cause sudden pressure fluctuations which can affect the performance of the flow meters. A secondary pressure regulator helps to minimize these pressure fluctuations. The flowmeter settings remain constant as the pressure is reduced below the normal fluctuation range. Thus oxygen flow from the flowmeter will remain constant as long as the oxygen pressure is above 14 psig.

Also the mechanically linked anti-hypoxia devices at the flowmeter level need a constant oxygen supply pressure to maintain accurate gas flows. However these devices cannot detect altered gas flow rates caused by changing pressures. Whenever the oxygen supply pressure decreases below 20 psig the pressure sensor shutoff valve cuts off supply of all the other gases. Due to the secondary pressure regulators in Datex Ohmeda machines the pressure of oxygen is lower than that of nitrous oxide which ensures that oxygen would be the last gas flow to decrease.

What are the flows and pressure at which oxygen is delivered from the oxygen flush valve?

Oxygen flush valve receives oxygen from the pipeline inlet or oxygen cylinders at a pressure of 45–55 psi and delivers it to the common gas outlet at flow rates of 35–75 l/min. In the older machines the oxygen flush valve had a locking mechanism. This can cause barotrauma if the valve remains in the locked position. In the modern machines the oxygen flush button is in a recess to prevent accidental activation and there is no locking mechanism.
It bypasses the flowmeter and vaporizers.

What are the hazards of oxygen flush valve?

There are several hazards associated with the oxygen flush valve.

- Barotrauma can result from sticking of defective or damaged valve in the fully open position. In valves with locking mechanism, accidental locking of the valve can cause barotraumas. The oxygen flush valves on modern machines do not have locking mechanism.
- If the oxygen flush valve is incompetent then the oxygen flowing through the valve can dilute the inhaled anesthetic resulting in awareness in the patient. Repeated use of oxygen flush intraoperatively can also dilute the inhaled anesthetic.
- If the anesthesia machine does not use fresh gas decoupling or inspiratory pressure limiter then the use of oxygen flush during the inspiratory phase of positive

pressure ventilation can cause barotrauma in patients. In the traditional machines the ventilators relief valve is closed and APL valve is out of circuit during controlled ventilation, due to which the excess gas cannot be vented out during the inspiratory phase of controlled ventilation.

- If the vaporizers are placed downstream of the common gas outlet then the use of oxygen flush can deliver large quantities of inhaled anesthetic to the patient.
- Oxygen flush should not be used to detect leaks in the low pressure circuit in anesthesia machines with one way check valve at the common gas outlet. Major leaks in the low pressure system can go undetected because the backpressure from the breathing system closes the one way valve making it air tight.

What are the safety devices for oxygen supply pressure failure?

These devices prevent the delivery of hypoxic mixture resulting from a decrease in oxygen supply pressure. They are fail safe valve which are present in the gas line supplying all the flowmeters except that for oxygen. In the older machines whenever the oxygen supply pressure decreases below 50% of the normal supply pressure or below 30 psi the valve shuts off or proportionately decreases the supply pressure of all the other gases. This is also called as master and slave mechanism where oxygen is the master and nitrous oxide is the slave. Here the tension in spring of the nitrous oxide pressure reducing valve is replaced by the pressure of oxygen coming from the oxygen pressure regulating valve. If the oxygen pressures drop below 30 psi, the valve in the nitrous oxide pressure regulator closes and no further nitrous oxide can be delivered.

In Datex Ohmeda machine there is the pressure sensors shutoff valve with a threshold pressure of 20 psig. Whenever the oxygen supply pressure falls below 20 psig the valve closes and completely stops the supply of nitrous oxide to the flow control valve.

In Drager machines there is the oxygen failure protection device (OFPD). This device is based on the proportioning principle and not on the threshold principle. Whenever there is a decrease in the oxygen supply pressure there is a proportional decrease in pressure of all the gases controlled by the OFPD. When the oxygen supply pressure falls below 10 psig the supply of other gases to the respective flowmeters is completely cut off.

The Fabius GS Dräger machine, uses a Sensitive Oxygen Ratio Controller (S-ORC) shuts off nitrous oxide if the oxygen flow is less than 200 mL/min, or if the oxygen fresh gas valve is closed.

When is the oxygen pressure failure warning device activated?

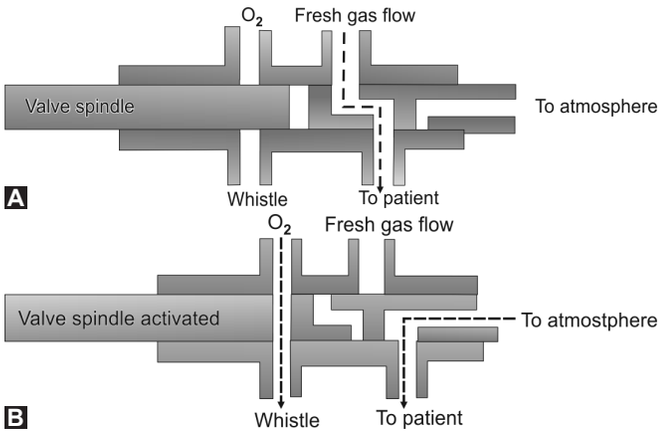


Fig. 43.5A and B (A) Oxygen failure warning device; (B) When Oxygen supply pressure falls below 200 kPa then whistle is activated

Ritchie whistle is an oxygen failure warning device. It was introduced in the mid 1960s. Anesthetic gases will flow into the machine as long as the pipeline pressure is 55–60 psig. When the pressure drops below 260 kPa (38 psig) the anesthetic gas cut off valve begins to close and there is an audible alarm. At a pressure of 200 kPa (30 psig) the anesthetic gases are cut off. The alarm continues till the oxygen pressure falls to 40.4 kPa (6 psig). However there is a mechanism which allows the patient to inspire room air.

In Fabius GS machine there is an audible and visual alarm when the oxygen pipeline pressure drops below 20 ± 4 psi.

What are the limitations of the oxygen pressure failure safety devices?

The oxygen pressure failure safety devices depend on pressure and not on flows. Therefore machines that do not have a flow proportioning system can still deliver hypoxic mixture. These devices only prevent hypoxia due to problems occurring upstream of them e.g. disconnected oxygen hoses, low oxygen pressures in the pipelines and depletion of oxygen cylinders. They do not protect against hypoxic mixtures from being delivered due problems downstream of these devices. Hypoxic mixture can result from problems like crossover in the pipelines, wrong contents in the gas cylinder, leaks in the equipment and operation errors like inadvertently closed oxygen flow control valve. In these situations the normal oxygen supply pressure keeps the other gas lines open.

What mechanisms are incorporated in an anesthetic machine to prevent delivery of a hypoxic mixture?

There are various devices that can help prevent the delivery of hypoxic mixture.

Minimum mandatory oxygen flow: In some anesthesia machines there should be a minimum oxygen flow of 50–250 ml/min. before other gases can flow. This is achieved by providing a mechanical stop on the oxygen flow control valve or a resistor which allows a small flow to bypass the oxygen flow control valve. However minimum oxygen flow by itself will not prevent the delivery of hypoxic mixtures.

The minimum oxygen ratio or the proportioning devices prevent the delivery of hypoxic mixture. Oxygen and nitrous oxide are linked either pneumatically or mechanically to ensure a minimum oxygen concentration of 23%–25% at the common gas outlet. Therefore the flow meters are adjacent to each other. The Datex Ohmeda has Link-25 Proportion Limiting Control System while Drager has Oxygen Ration Monitor Controller (ORMC). Newer Drager machines have sensitive oxygen ratio controller(S-ORC).

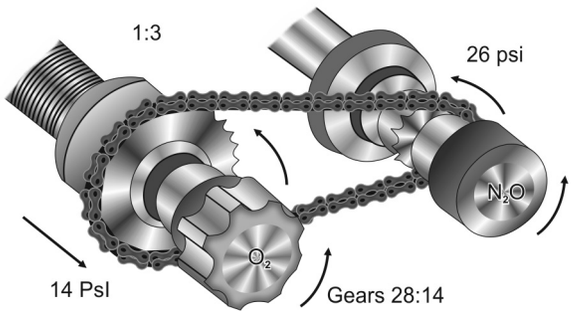


Fig. 43.6 Datex ohmeda link-25 system

Mechanical devices: In the Datex Ohmeda Link-25 system a 28-tooth sprocket is attached to the oxygen flow control valve and14 tooth sprocket is attached to the nitrous oxide flow control valve. The sprockets are linked by a chain with a gear ratio of 2:1 (nitrous oxide: oxygen). As a result of this when nitrous oxide flow control valve turns 2 revolutions; oxygen flow control valve turns 1 revolution. A second stage pressure regulator reduces the pressure at the nitrous oxide flow control valve to 26 psig and at the oxygen flow control valve to 14 psig. So the final flow ratio is 3:1. **The final oxygen concentration is a result of this combination of mechanical and pneumatic aspects of the system.** This system actually increases the flow of oxygen to prevent the delivery of a fresh gas mixture with an oxygen concentration of less than 25%. Whenever nitrous oxide is flowing the oxygen flow control knob can be opened independently, but it cannot be closed below a setting which will result in less than 25% oxygen in the mixture. Another safety feature of this system is that a mechanical stop on the oxygen flow control valve ensures a minimum flow of 175–250 ml of oxygen even when the valve is closed.

Pneumatic devices: In the Drager machines the ORMC and S-ORC are pneumatic oxygen-nitrous oxide interlock system which ensure a fresh gas oxygen concentration of at least 25% $\pm$ 3%. They actually limit the flow of nitrous oxide to prevent the delivery of hypoxic mixture. This system has an oxygen chamber, a nitrous oxide chamber and nitrous oxide slave control valve which are all interconnected by a horizontal shaft. The slave control valve consists of a spring loaded ball which is linked to O_2 diaphragm. It cuts off flow across the N_2O needle on the base of its flow meter, if O_2 supply fails or if O_2 flow meter is turned off. The oxygen and nitrous oxide flow control valves have resistors downstream. Back pressure generated due to flow of gases is transferred to the oxygen and nitrous oxide chambers. There is a diaphragm interposed between the two chambers. The relative values of the resistors determine the value of the controlled fresh gas oxygen concentration. The value of the oxygen flow tube resistor is 3–4 times that of the nitrous oxide flow tube resistor. Backpressure in the oxygen and nitrous oxide chambers cause movement of a horizontal shaft attached to the rubber diaphragm. The movement of the shaft controls the nitrous oxide slave control valve which feeds the nitrous oxide flow control valve.

- O_2 flow meter turned on: it pushes the shaft to the left thus pushing the ball away from the N_2O needle. N_2O is thus ready to be turned on.
- O_2 flow turned off: the shaft moves to the right to return to its neutral position, this allows the spring on the ball to recoil and close the N_2O needle. N_2O cannot be turned on.
- If one attempts to increase the N_2O flow: pressure in the N_2O chamber increases and moves the shaft to the right, thus preventing its increase. As the shaft moves to the left, the spring relaxes and the ball partly occludes the flow from the N_2O needle.
- If one attempts to reduce O_2 flow: pressure in the O_2 chamber decreases and the shaft moves to the right, which reduces N_2O flow from the needle.

In this system, if one tries to increase N_2O flow, they will not be able to do so as the flow of N_2O depends on O_2 flow.

Electronic devices: In Penlon machines there is a paramagnetic oxygen analyzer. Whenever the oxygen concentration is less than 25% there is an audible alarm and nitrous oxide supply is cut off.

Can these systems prevent delivery of hypoxic mixture to the patient?

No. These systems work only for two gases namely O_2 and N_2O. Third or fourth gases such as air or helium are not linked. In addition these systems prevent hypoxic mixture

only until the common gas outlet. If a circle system is used, inappropriately low flow settings can lead to hypoxic mixture within the circle system.

What are the circumstances in which a hypoxic mixture can be delivered even when the hypoxic guard system is used?

1. Wrong supply gas in oxygen pipeline or cylinder.
2. Defective pneumatics or mechanics (the hypoxic guard system is broken).
3. Leaks down stream of flowmeter control valves.
4. As the proportioning system links only oxygen and nitrous oxide the administration of a third inert gas like helium, nitrogen or carbon dioxide can cause a hypoxic mixture.
5. The hypoxic guard system only connects oxygen and nitrous oxide. The volatile inhaled anesthetics are added to the gas mixture downstream from the flow meters and proportioning system. Hence when using high concentration of low potency inhaled anesthetics (maximum dial setting for desflurane is 18%) it is possible to create a hypoxic mixture when you give desflurane in air.

Therefore it is essential to monitor the inspired concentration of oxygen being delivered from the machine outlet.

How would you describe a flow meter assembly?

The flowmeter assembly has a needle valve, valve seat and tapered glass tube which is calibrated for the particular gas. The flowmeter is made up of glass and is made gas tight at both the ends by 'O' rings and washer. Each flowmeter is calibrated for the specific gas at 20°C at ambient pressure of 760 mmHg.

The flow meters are designed to deliver a range of flows from a single tube. The bore of the tube increases from the bottom to the top. This is achieved by either single tapered or double tapered tubes. Some anesthetic machines have a pair of tubes for each gas: one to be used for low flows and the other for higher flows. As the pair is arranged in series, the second tube indicates total flow. It usually contains a bobbin or ball which is free to rotate and to move up or down depending upon the flow rate.

How do you read flows from flow meters with bobbins?

The flows are read off from the top end of the bobbin. A freely rotating bobbin confirms that the flowmeter is delivering the gases at the flow rate indicated. They have grooves cut at an angle which force it to rotate as the gas flows from the lower end of the tube. A mark on the bobbin aids in visual confirmation that it is rotating.

Some flow meters such as those attached to cylinders in the ward or wall mounted flow meters have a sphere as the bobbin within them. The flow rate reading is taken from the equator of the sphere.

How does gas flow around the bobbin?

At low flows, the gap between the flange of the bobbin and the flowmeter wall behaves like a tube (distance between the flange and the wall is small compared to the length of the flange). Therefore gas flow depends on viscosity. At high flows, the bobbin is pushed up the flowmeter tube. Here the gap between the flange and the wall of the flowmeter will behaves like an orifice (distance between the flange and the wall is great when compared to the length of the flange). Therefore gas flow depends on density.

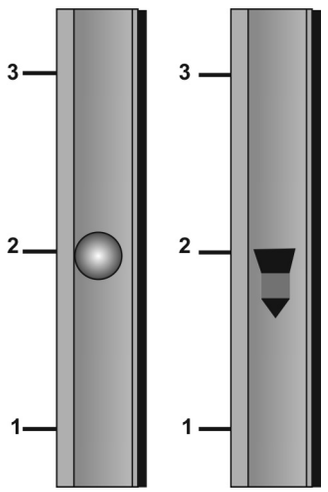


Fig. 43.7 Funetioning of flow meter

What are the principles behind the flow meters?

It is a variable orifice (area)/constant pressure flowmeter. The variable area is accounted by the tapered flow meter tubes. As the bobbin moves upwards the area or space around it increases therefore variable area (orifice). If the pressure across (above and below) the bobbin is measured, it is always constant; and hence the constant pressure flowmeter.

The downward force on the bobbin is due to the force of gravity acting on its mass. This can be assumed to be constant for the entire height of the flow meter (as it is very short). Therefore the force needed to balance (by gases flowing from the bottom of the flowmeter) the bobbin against its weight will also be constant.

What factors may contribute to erroneous reading of a flow meter?

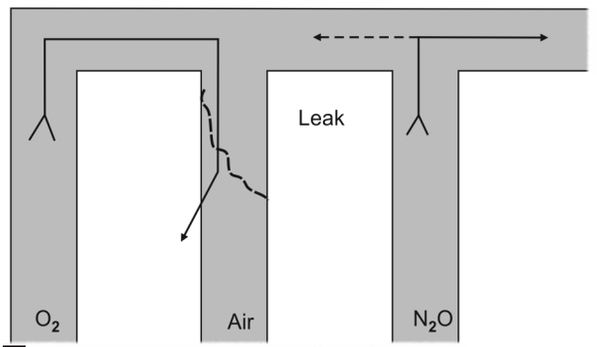
Spinning of the bobbin is essential for accurate reading. The bobbin may stick to the flow meter tube under the following circumstances:

- Dust in the flowmeter—filter is incorporated at the needle valve at the base of the flowmeter to trap any dust.
- Static electricity—the glass is coated with a conductive material (gold or tin oxide) to prevent building up of static electricity.
- Tilting of the flowmeter at its mounting—not significant in a modern anesthetic machine.
- Backpressure from a ventilator—the bobbin under-reads, but the actual flow is not significantly altered.
- Translocation of flow meters after servicing—flow meters are colour coded; the symbol of the gas is etched on it and indexed at the needle valve thus reducing risk of translocation.

How are the flow meters arranged in an anesthetic machine?

Conventionally, from left to right it is O_2, N_2O and air. O_2 is carried by a separate line on top of the flow meter block and it is the last gas to join the back bar. For example, let us consider the following scenarios -

Scenario 1—the anesthetist is using O_2 and N_2O a crack develops in the air flow meter which is switched off, O_2 will flow out through this crack preferentially and a hypoxic mixture will be delivered at the common gas outlet.



Figs 43.8: Arrangement of flow meter in anesthetic machine

Scenario 2—rearrangement of the flowmeter block - the anesthetist is using O_2 and N_2O, a crack develops in the air flow meter which is switched off, N_2O will be vented off through the crack and as oxygen is the last gas to enter the gas mixture, thus preventing delivery of hypoxic gas.

What safety measures are built into the flow meter block?

- All the needle valve control knobs are color coded and clearly marked.
- O_2 control knob- is larger, octagonal profile and protudes out more than nitrous oxide knob when completely closed.

Fig. 43.9: Color coded knobs in flow meter

- The torque required to turn the knobs is high enough to prevent adjustments secondary to casual contact.
- Some manufacturers use a plastic shield to cover the knobs to prevent alterations in flow settings on casual contact.
- Illuminated back helps visualization of the flowmeter block in dim light.
- Sequence of various gases to minimize delivery of hypoxic mixture.

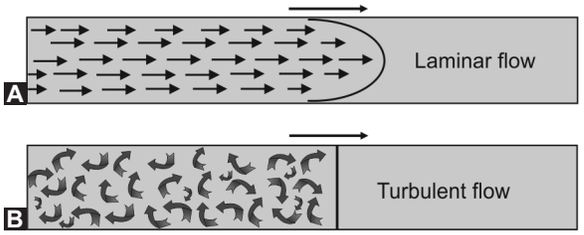
How do electronic flow meters work?

In S5 Datex machines: a chamber collects gases that pass through the needle valve. As the volume of the chamber is fixed, the pressure within it increases. The chamber is controlled by a solenoid valve that opens and lets the gas out when a specific pressure limit is reached. The number of times the solenoid valves opens per minute is related to flow per minute. The display is either numerical (Drager) or as a bar (Datex S5). Electronic flow meters allow data to be stored and printed off as a part of electronic anesthetic chart.

Define flow. What do you understand by laminar and turbulent flow?

Flow is defined as the quantity of fluid (gas or liquid) passing a point in unit time.

Laminar flow—the fluid moves in a steady manner and there is no turbulence or eddy current. The flow is greatest in the central column (twice the mean flow) laterally the flow is slower compared to the center until approaches zero at the sides. It is present in smooth tubes at lower flow rates. See diagram below.



Figs 43.10A and B: (A) Laminar flow; (B) Turbulent flow

Laminar flow is directly proportional to pressure gradient across the tube and the radius of the tube and indirectly proportional to the viscosity and the length of the tube. So decreasing the radius by half decrease the flow by a factor of 16 and halving the length doubles the flow. This is explained by the Hagen-Poiseuelle equation:

$$Q \alpha \frac{\Delta P r^4}{8 \eta l}$$

where Q is the flow, ΔP is the pressure gradient across the tube, r is the radius of the tube, η is the viscosity and l is the length of the tube.

Anesthetic implication – during fluid resuscitation a short length wide bore cannula is preferred for e.g. 14 G as the flow rate is much superior to a 20 G cannula or a central line.

Similarly, intubating patients with a very small tube increases the resistance to flow and thus the pressure increases to deliver the same amount of flow through the endotracheal tube.

Turbulent flow: Turbulent flow is when there is eddies in the fluid. It is seen in the following conditions:

- Rough or irregular inner surface of the tube
- Acute bends or acute narrowing in the tube
- Flow velocity exceeds a critical level.

What is Reynolds number?

Reynold's number is

$$Re = v\rho D / \eta$$

v – Velocity of the fluid, ρ – density of the fluid, η – viscosity, D – diameter of the tube.

A Reynold's number in excess of 2000 favours turbulent flow. If it is less than 2000 then laminar flow exists. The density of the fluid is in the numerator, hence a fall in the density would reduce Reynolds number and so encourage laminar flow.

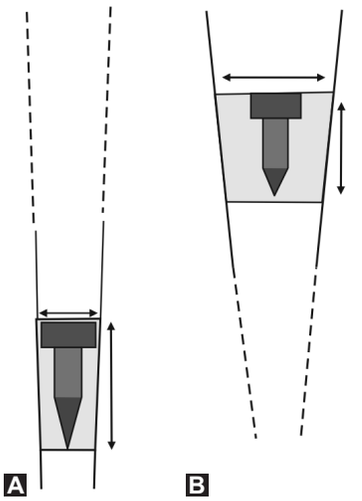
How is this clinically important?

In cases of upper airway obstruction, there is turbulent flow which increases the work of breathing. Administering helium, which has very low density decreases the Reynolds number and thus changes the characteristics of flow from turbulent to laminar and decreases the respiratory work. This is the reason behind using heliox to reduce respiratory distress due to upper airway obstruction.

Can the flowmeter for different gases be used interchangeably?

No. A flow meter is tapered. At low flows, the gap between the walls of the flowmeter and the bobbin behaves like a tube so viscosity is important. Viscosity is independent of barometric pressure so at low flows it reads accurately. At high flows the gap between the bobbin and the walls of the flowmeter behave like an orifice and the flow is dependent upon the density of the gases. As different gases have different densities the flow meters are calibrated for a specific gas and cannot be used interchangeably.

See diagram below.



Figs 43.11A and B: Uses of flow meter for different gases. (A) Low flow rate; (B) High flow rate

Can the same flow meters be used at different altitudes?

No. Barometric pressures affect the density but do not affect viscosity of the gas. At high flow rates the flow is dependent upon the density of the gases. At high altitude, density decreases so the flowmeter under reads at high flows. In a hyperbaric chamber it over reads at high flows.

Circle system

What are the components in a circle system?

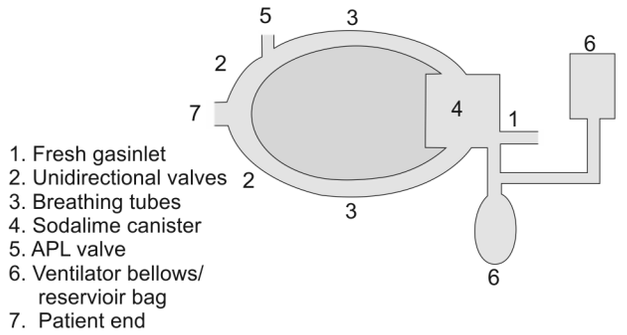


Fig. 43.12: Circle system

Although there are many possible arrangements of the components, in practice the arrangement is dictated by certain rules and practicalities.

Fresh gas flow (FGF): ideally located downstream to the soda lime canister and upstream to the inspiratory one way valve. During expiratory pause continuous FGF pushes the CO_2 enriched exhaled gases retrograde past the soda lime canister to be vented via the pressure limiting valve (APL). This prolongs the life of soda-lime.

If FGF is high, it flushes out gases that have been cleaned up by soda lime, and if high enough, it might be lost via the APL valve. In addition, changes in FGF composition is rapidly reflected in the inspired gases.

FGF joining upstream of the soda lime has the following disadvantages:

- Its composition may not immediately reflect the gas content of inspired gases.
- Activation of flush may carry dust.
- Inhaled anesthetic agent is absorbed by soda lime.
- High flows may dry up soda lime.

If FGF enters between the patient and the expiratory unidirectional valve, there is a risk of barotrauma on activation of the O_2 flush at a critical time of respiratory cycle.

Unidirectional valves

These are light disc sitting on a knife edge seat. The disc is held in place by a cage. The valve is designed to prevent the disc from sticking to valve seat. Gases normally flow from under the disc, lifting the disc off the seat and flowing out under the dome. Pressure from under the dome firmly seats the disc and prevents retrograde flow. Some unidirectional valves are positional and depend on gravity for seating the disc. Historically, valves were placed on a block near the patient end.

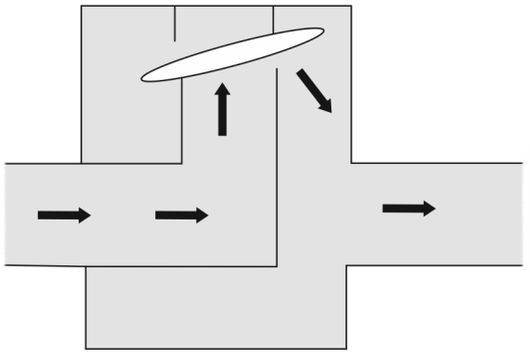


Fig. 43.13: Unidirectional valves

Reservoir Bag: Commonly between the expiratory valve and the canister. This reduces the work of expiration which is the only work the respiratory muscles do under IPPV. If it is located between absorber and inspiratory valve, it reduces work of inspiration in spontaneous ventilation. Any positions between the patient and the uni-directional valves are not permitted as it would lead to rebreathing.

APL valve: usually located between the expiratory unidirectional valve and the canister. This allows exhaled gases to escape before passing through the soda lime canister. Downstream the canister will lead to loss of gases that are conditioned (fresh gas flow and gas from the canister). Downstream to inspiratory unidirectional valve leads to rebreathing, in addition to loss of conditioned gases.

Other components in the circle housing:

A selector switch toggles between ventilator and the reservoir bag. In newer machines the APL valve is automatically isolated (along with the bag) when switched to ventilator mode.

PEEP valve may be added by replacing the dome of the expiratory port. In newer machines it is incorporated in the dome of the expiratory unidirectional valve.

Gases aspirated for analysis are returned to the circle system by a port that is functionally upstream of the canister. The APL valve is connected to a scavenging system.

A disposable circle system is manufactured by intersurgical that is useful in infective cases and patients who are susceptible to malignant hyperpyrexia.

What are the advantages and disadvantages of a circle system?

Advantages

- Economical—
  - Gases and Inhalational anesthetic agents.
  - Scavenging volume / load decreases.

- Better preservation of heat and humidity within the circuit.
- Arterial CO_2 tension depends on minute ventilation and not on FGF.
- Low dead space if no faults in the system. The dead space in the circle system consists of the Y-piece which connects the inspiratory and the expiratory limb.
- Reduced atmospheric pollution.

Disadvantages

- Many components and therefore there is a risk of disconnection and misconnection.
- Slow change in the inspired gas composition particularly with vaporizer-out-of circuit and low flow. Change in the composition of inspired gases requires the fresh gas flow rate to be increased.
- Dry soda lime or bara lyme adsorbs anesthetic agents.
- Accumulation of trace gases – Carbon monoxide (CO), H_2, acetone, methane, and ethanol. CO comes from smokers and from breakdown of Hb.
- Acrylic monomer is exhaled after cementing. A brief period of higher FGF would vent this out.
- Greater resistance to breathing:
  - Position of the bag influences the resistance to that part of respiratory cycle. For e.g. in a patient breathing spontaneously, if the bag is in the inspiratory limb, it reduces inspiratory work of breathing.
  - A high fresh gas flow reduces inspiratory resistance but increases expiratory resistance.

How is CO_2 absorbed in the circle system?

The principle of CO_2 absorption is base neutralizing an acid. Carbon dioxide reacts with water to form carbonic acid which is neutralized by hydroxides of alkali. This reaction takes place between ions; hence water has to be present so that the acid and base can exist in the ionic form. CO_2 combines with water to form H_2CO_3 which is neutralized by Na or Ca OH. The final reaction generates water and a carbonate.

What is the composition of CO_2 absorbents?

There are 2 commercial preparations namely soda lime and Barium lime. Soda lime is made up of approximately 80% calcium hydroxide, 4% sodium hydroxide and 15–20% water. Sodium hydroxide has hygroscopic properties and improves the reactivity of the mixture. Absorbents with low water content would exhaust rapidly, while those with high water content would get sticky and provide increased resistance for gas flow. Small amounts of silica and kieselguhr are added as hardners. They help to form the required granule size. They are no longer used in the modern manufacturing process. KOH is also no longer added. Indicators are also present.

Barium lime is made of 80% calcium hydroxide and 20% barium hydroxide. Barium hydroxide has its own naturally occurring water of crystallization. It does not require any hardeners as water of crystallization prevents dust formation. Indicator is present.

Why are hardners added to soda lime?

Soda lime granules powder very easily to produce fine dust. If there is excessive powder it can cause channelling, caking and increased resistance to flow. If the dust is blown into the patient it can irritate the respiratory mucosa. Small amount of silica is added to soda lime to increase its hardness. Kieselguhr is added to silica as it tends to clog the pores and reduce the efficiency of soda lime.

What should be the hardness number and how is it tested?

Hardness is tested by agitating a weighted amount of absorbent with a steel ball and agitating it. The soda lime is then sifted through 8 mesh screen; the amount of soda lime in percentage left on the mesh screen is the hardness number. Normally it should be greater than 75.

Which is the more active component in soda lime?

Sodium hydroxide is the more active component in soda lime.

Then why do you have only 4% sodium hydroxide and 80% calcium hydroxide in soda lime?

Sodium hydroxide is more hygroscopic. If you increase the concentration of sodium hydroxide then it will absorb water whenever there is high humidity. This will result in clogging of the pores of the granules and caking during use. Also lot of heat is produced during this reaction.

What is the chemical reaction taking place during CO_2 absorption by soda lime?

Carbon dioxide first reacts with the water on the surface of the soda lime granules to form carbonic acid.

$$CO_2 + H_2O \longleftrightarrow H_2CO_3$$

As it is a weak acid it incompletely dissociates into its ions

$$H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$$

$$\downarrow$$

$$H^+ + CO_3^-$$

Sodium and calcium hydroxide also dissociate into their ions

$$NaOH \longleftrightarrow OH^- + Na^+$$

$$Ca(OH)_2 \longleftrightarrow 2OH^- + Ca^{2+}$$

The sodium and calcium ions combine with carbonate ions to form sodium and calcium carbonate. Hydrogen and hydroxyl ions combine to form water.

$$2NaOH + 2\,H_2CO_3 + Ca(OH)_2 \longleftrightarrow CaCO_3 + Na_2CO_3 + 4H_2O$$

This is an exothermic reaction which produces a lot of heat.

Is the size and shape of soda lime granules important?

The size and shape of granules are important. Irregular surface of the granules provide maximum surface area for absorption. Also if the granules are smaller in size they provide greater surface area for absorption and decrease channelling. But there is increased resistance and caking with smaller granules. If the granules are large they provide lesser resistance to air flow but have lesser surface area for absorption. The optimum size of the granules is 1.5–5 mm or 4–8 mesh size (USA) and 3–10(UK). A size 4 mesh size means the sieve has squares of 1/4 inch and size 8 means there are squares if 1/8 inch. Thus 4-mesh size is bigger than 8-mesh.

How do you know that soda lime is exhausted?

Indicators are added to the absorbent to indicate that they are exhausted. These indicators are acids or bases and their color depends on hydrogen ion concentration. Various indicators are available, these are summarized in Table 43.5:

Table 43.5 Exhausted of soda lime through various indicators

Indicator	Color when fresh	Color when exhausted
Phenolphthalein	White	Pink
Ethyl violet	White	Purple
Clayton yellow	Deep pink	Off white
Ethyl orange	Orange	Yellow
Mimosa Z	Red	White

What are the absorptive capacities of CO_2 absorbents?

The absorptive capacity of soda lime is 25 L of CO_2 per 100 gm, and of barium lime is 27 L of CO_2 per 100 gm of absorbent. However during continous use the outer surface of the granule is exhausted before the whole granule is used up. Therefore it appears that the soda lime is exhausted even before its absorptive capacity is reached. Hence during use the 500 gm soda lime canister appear exhausted at a CO_2 load of 10–12 L per 100 gm and the 2 kg jumbo canister appear exhausted at a CO_2 load of 17 L per 100 gm.

Why does soda lime which is exhausted show reversal of colour when allowed to rest?

The reversal of colour is due to regeneration of soda lime. The length of the rest period will determine the amount of regeneration. Sodium hydroxide is more active than calcium hydroxide. So it preferentially binds to carbon dioxide to form sodium carbonate. As sodium carbonate is more soluble it dissolves in the moisture on the granules and penetrates into the granules. Within the granules it reacts with calcium hydroxide to form calcium carbonate (which is insoluble) and sodium hydroxide. This regenerated sodium hydroxide imparts renewed activity to the absorbent.

Can you predict the exhaustion time of soda lime canister?

No we cannot predict the exhaustion time of soda lime canister. This is because the exhaustion time will depend on various factors.

- Channelling: The exhaustion time is reduced due to channelling which can result from improper packing or canister design.
- Size of absorber: Larger the absorber longer the exhaustion time.
- Rate of CO_2 production: higher CO_2 production, shorter the exhaustion time.
- Fresh gas flow rate: high fresh gas flow rate longer the exhaustion time.
- The acceptable inspired CO_2 level vary between 0.1–1%.
- The position of the absorber in relation to the various components of the circle system will also influence the exhaustion time.

What do you mean by channelling?

Whenever a gas takes the path of least resistance it is called channelling. If the soda lime canister design is faulty or if the granules are loosely packed then the gas bypasses bulk of the absorbent and follow the path of least resistance. Channelling usually occurs at the inlet and the inner tube and along the walls. Soda lime near the channels gets exhausted quickly as compared to the other parts. This reduces the efficiency of CO_2 absorption.

However even after exhaustion there is a blind spot in the distal third of the canister where the granules are still capable of absorbing carbon dioxide.

Why is barium lime not used nowadays?

Anesthetic agents containing CHF2 moiety (desflurane, enflurane and isoflurane) react with warm and dry soda lime and barium lime to produce varying amounts of carbon monoxide. As compared to soda lime barium lime has a greater tendency to produce carbon monoxide especially if its water content is less than 2%. The interaction between barium lime and sevoflurane produces very high

temperatures. This can cause melting or burning of the components of the absorber. The increase in temperature is much less with soda lime. Due to these problems barium hydroxide is no longer used.

What are the differences between the traditional soda lime and the newer soda lime?

Traditional soda lime was a high alkali absorbent. It contained high concentrations of potassium and /or sodium hydroxide. The desiccated soda lime would react with volatile anesthetics to produce carbon monoxide. Also sevoflurane is degraded to produce compound A which is nephrotoxic. They do not change color if they are dry. Decrease in moisture content reduces the capacity to absorb CO_2.

The newer absorbents are alkali free absorbents. They mainly contain calcium hydroxide. Even if they are desiccated there is no carbon monoxide formation with volatile anesthetics. Sevoflurane can be used in a closed circuit with desiccated absorbent as there is little or no Compound A formation. They change colour even if they are dry and the color does not revert after they are exhausted. Their CO_2 absorption capacity is not affected when they are dry.

Amsorb is a newer soda lime which contains CaOH, $CaCl_2$, setting agents ($CaSO_4$ and polyvinylpyrrolidine). The advantages are lack of sodium and potassium hydroxide eliminates the production of CO and compound A. Absorbs 10.2 L CO_2/100 gm of the absorbent.

Lithium hydroxide is also used as a CO_2 absorbent. It reacts with carbon dioxide to form lithium carbonate. It does not react with volatile anesthetics even when it is dry. The major drawback is that it is expensive and can cause burns to the eyes, skin and respiratory tract if not handled properly.

What are the problems with the use of inhalational anesthetics with CO_2 absorbents?

At high temperatures in the soda lime canister, sevoflurane is degraded to fluorinated metabolite called sevo-olefin or compound A. Increasing levels of compound A are seen with increased concentration of agent, prolonged anesthesia, low FGF, increased temperature within the absorber and desiccated absorbents. Compound A is nephrotoxic in rats however human studies show conflicting results. During closed circuit anesthesia Halothane is degraded to form a halokene which is nephrotoxic in rats but not in humans. Anesthetic agents containing CHF_2 moiety (desflurane, enflurane and isoflurane) react with warm and dry soda lime and barium lime to produce varying amounts of carbon monoxide. The production of carbon monoxide increases in the presence of desiccated absorbent, high temperatures in the absorber, presence of strong alkalis in the absorbent, high fresh gas flows. Carbon monoxide production is greatest with (desflurane > = enflurane > isoflurane >> halothane = sevoflurane).

What are the advantages of using CO_2 absorber?

The exhaled gases can be reutilized after CO_2 absorption. It has the following advantages:

- It is economical as low fresh gas flows can be used
- There is less theatre pollution
- The hazards of explosion are reduced as the in flammable mixtures are enclosed
- There is conservation of heat and humidity.

If you have a choice between a jumbo CO_2 canister and smaller CO_2 canister; which one would you prefer and why?

The efficiency of CO_2 absorption depends on

- How fresh is the soda lime

Table 43.6 Differences between traditional soda lime and newer soda lime

Component	Soda lime	Baralyme	Medisorb	Dragersorb 800+	Amsorb
$Ca(OH)_2$ %	84	80	70–80	80	83
NaOH %	2–4		1–2	2	–
KOH %	1–3	May contain some	0.003	2	–
$CaCl_2$ % (humectant)					1
$CaSO_4$ (hardener)					1
Polyvinylpyrrolidine % (hardener)					1
Water Content %	14–19	As water of crystallization	16–20	14	14.5
$Ba(OH)_2$–$8H_2O$%	–	20	–	–	–
Size (mesh)	4–8	4–8	4–8	4–8	4–8
Indicator	Yes	Yes	Yes	Yes	Yes

- How much surface area of the granules is available for absorption
- The length of time the gas is in contact with the granules.

The jumbo canisters containing 2 kg of soda lime have a large volume. Due to this more surface area of the granule is available for absorption and the expired gases are in contact with the granules for a longer time. Hence the jumbo canisters are more efficient in CO_2 absorption. They need to be changed less frequently because they last 5 times longer than the 500 gm canister.

The smaller canisters are easier to maintain, to clean and have fewer leaks. They are also supplied as disposable cartridges. However they need frequent changes. Smaller canisters are preferred due to the concerns of carbon monoxide production with dry stale soda lime. Frequent change of the soda lime canister will reduce the risk of carbon monoxide production.

When will you change the sodalime canister?

The time to change the canister will be indicated by the inspired concentration of CO_2 when monitoring end tidal capnometry, change in the colour of the CO_2 absorber.

Can one deliver hypoxic mixture via a circle system?

When using low fresh gas flows, the exhaled gases exert an increasing influence on the composition of the gases within the circle system. If the inspired concentration of O_2 is gradually falling, it might be due to either of the following reasons:

- The O_2 supply: consumption imbalance. Either the supply is low (reduced O_2 from the flowmeter or reduced alveolar ventilation) or demand is high (hypermetabolic states or high sympathetic activity).
- Denitrogenation process was not complete during the high fresh gas flow period (if one is using O_2 and N_2O).
- If nitrous oxide is used, particularly after a long anesthetic, the body gets saturated (equilibrated) with nitrous oxide and its uptake decreases progressively with time i.e. it accumulates in the circuit. If nitrous oxide is administered at the same flow rate, the fractional concentration of O_2 decreases secondary to reduced uptake of nitrous oxide resulting in a hypoxic mixture. It is mandatory to use inspired oxygen concentration monitor whenever nitrous oxide is used in a closed system.
- Addition of other gases such as helium or desflurane may also affect FiO_2.

Therefore it is mandatory to measure O_2 concentration within the circle system, preferably at the patient end.

What are the objectives in a circle system?

- Maximum re-use of dead space and fresh gases.
- Maximum venting of alveolar gases (not if the system is completely closed).
- FGF should join the inspiratory limb. Some circuits have the FGF conducted via separate tubing (within the inspiratory limb) close to the Y piece. This allows changes in the anesthetic machine to be rapidly reflected in inspired gases. This is important if one uses low flow.
- For pediatric use, low diameter tubes should be used.

What is the ideal arrangement of components in a circle system?

- Unidirectional valves should be located between the patient and the reservoir bag one in each limb
- No gases should flow towards the patient via the expiratory limb during inspiration.
  - FGF cannot enter between the patient and expiratory unidirectional valve.
  - Reservoir bag should not be located between the patient and the expiratory valve.

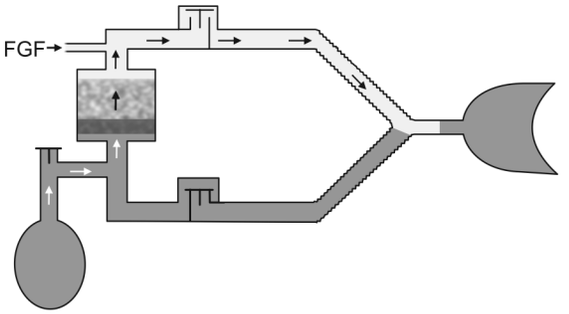


Fig. 43.14: Finctional analysis of circle system

- No gases should flow from the patient into the inspiratory limb during expiration.
  - APL valve cannot be located between patient and inspiratory valve
  - Reservoir bag should not be located between the patient and the inspiratory valve.

Certain important considerations

- The capacity of the reservoir bag should be greater than the patient's inspiratory capacity (approximately 30 ml/kg body weight).
- The soda lime contains 50% – 70% air around the granules, so for optimal efficiency, the volume of the canister should be at least twice the tidal volume of the patient.

What is low flow anesthesia?

It is an anesthetic technique via a circle (rebreathing) system in which fraction of recycled (CO_2 free) gases account for at least 50% of the total flow.

Low flow anesthesia implies the total gas flow rate is less than 1 litre/min.

Minimal flow anesthesia implies the total gas flow rate is less than 0.5 litre/min.

Metabolic flow implies the total gas flow rate is approximately 0.25 litre/min.

What are the advantages and disadvantages of low flow anesthesia?

The advantages are:

- Economical—decrease in consumption of gases and inhalational anesthetic agents.
- Effectively warms and humidifies inspired gases.
- Minimises atmospheric pollution—Inhalational agents along with nitrous oxide destroy the ozone layer and thus contribute to the green house effect. Though the contribution to this effect by anesthetist is minimal (every little helps).

The disadvantages are:

- Initial need for capital investment for anesthesia machine and gas monitoring equipment.
- Increase consumption of soda lime.
- Accumulation of unwanted substances within the circle such as alcohol, carbon monoxide, methane. Low flow anesthesia is contraindicated in intoxicated patients, and carbon monoxide poisoning.
- Accumulation of products of reaction with soda lime – compound A is formed when sevoflurane is used in the circle system at low flows and high temperature. It has been shown to be nephrotoxic in rats (at a concentration of 250 ppm), however it is considered safe in humans as the concentrations in the breathing circuit during a typical anesthetic is maximum of 15 ppm. Because of this concern the US FDA set a 2 litre/min as the lower limit for carrier gas flow during sevoflurane anesthesia. This was later revised in December 1997, to 1 litre/min.
- Technically more difficult compared to conventional anesthetic.

What is the first device that will inform you of a crossover (non-oxygen gas in the oxygen pipeline)? Is it the fail-safe valve or the hypoxic guard?

It is important to understand that the fail-safe guards against decreased oxygen pressure and not against crossovers or mislabelled contents. As long as there is any pressure in the oxygen line, nitrous oxide (and any other gases) will continue flowing. If oxygen pressure is lost, the fail-safe shuts off the flow of all other gases. The hypoxic guard system works on oxygen pressure as well. It controls the ratio of oxygen and nitrous oxide so that there is minimum 25% oxygen. It does not analyze what is in the oxygen pipeline for the presence of oxygen. **Hence the oxygen analyzer is the first and only device that will detect a crossover**. If the oxygen analyzer is ignored then hypoxia will set in. This will be detected by a pulse oximeter on the patient. However the cause of hypoxia cannot be determined using the pulse oximeter.

For suspected crossover, what two actions must be taken?

1. Turn on backup oxygen cylinder.
2. Then disconnect oxygen supply source at the wall.

The pipelines are at a slightly higher pressure (60 psi) than the cylinder regulator (45 psi). If the pipeline supply hose at the wall is not disconnected, the pipeline pressure exerted on the oxygen cylinder regulator diaphragm (downstream side) will prevent the cylinder gas from flowing.

What should you do if you lose oxygen pipeline pressure?

Just like a crossover,

1. Open the emergency oxygen cylinder fully (not just the three or four quick turns used for checking)
2. Disconnect the pipeline connection at the wall. And always use oxygen analyzer! Allernative ventilation apparatus in the form of AMBU bag with reservoir with flow meter assembly and tubing should be present in every operation theatre to overcome such situations at all times

The pipeline supply of oxygen has failed. How can you make your emergency E cylinder oxygen supply last as long as possible?

Oxygen is used as a driving gas for ventilators on the anesthesia machine. If the pipeline supply fails then using the cylinder gas to drive the ventilator will cause rapid depletion of the cylinder. In general the amount of oxygen needed to drive the ventilator is approximately equivalent to the minute ventilation set to be delivered. (e.g. TV = 500 ml, RR = 10/min then MV = 5 L). So manually ventilate the patient, assist spontaneous ventilation if possible, use air or nitrous oxide with oxygen if possible, and use low flows.

Your pipeline supply fails, and your cylinder gauge shows 800 psi. How long will your emergency oxygen supply last?

As mentioned earlier the contents of a cylinder containing non-liquefied compressed gases can be estimated from the pressure. According to Boyle's law at constant temperature, the volume of a mass of a gas is inversely proportional to pressure.

Contents in L / Gauge Pressure = Capacity in L / Service Pressure

In the example, X L/800 psi = 660 L/1900 psi

Therefore X = 277 L.

If oxygen is used at a rate of 2 L/min oxygen, the cylinder will last 138.5 minutes.

What is the safety feature on the back bar?

The back bar is the horizontal part of the anesthesia machine between the rotameter block and common gas outlet. Vaporizers are mounted on the back bar. They add volatile agents to the fresh gas which is delivered to the common gas outlet. The pressure in the back bar ranges from 7–10 kpa (70–100cm H_2O) at the rotameter to 1 kpa (10 cm H_2O) at the common gas outlet. The back bar has the selectatec system for mounting the vaporizers. The vaporizers can easily be removed and there is a greater flexibility in the choice and use of vaporizers. The interlocking selectatec system prevents more than one vaporizer being used at the same time. If the locking lever is not engaged the control dial on the vaporizer cannot be moved. Also the vaporizers can be changed rapidly without interrupting the flow of carrier gas to the patient. There is a pressure relief valve which has a threshold pressure of 30–40 kpa. It protects the rotameter block and vaporizers from overpressure damage. There is also a spring loaded non return valve on the back-bar which prevents the back pressure surges from reaching the vaporizers (Datex)

Why do older anesthesia machines have trilene lock on the back bar?

The trilene vaporizers were always placed downstream of the trilene lock. Whenever trilene was being used this switch diverted all the fresh gas flows directly to the common gas outlet and not to the circle system. This is because CO_2 absorption is an exothermic reaction which produces a lot of heat. At high temperatures trilene gets decomposed to dichloroacetylene and phosgene which are neurotoxic.

What is the preferred bellows design, ascending or descending?

Bellows are said to be ascending or descending depending on what they do during expiration. An ascending bellows stands, while a descending bellows hangs down during expiration. Hence it is difficult to detect any disconnections with descending bellows. Since it hangs during expiration it will fill even if it is disconnected from the patient. Also there is a risk of infection due to the collection of exhaled humidity in bellows. As a result of this most modern machines have ascending bellows.

If there is an emergency life threatening situation where there is no time to check the anesthesia machine, what is the minimal safety check that must always be done?

1. Do a high-pressure test of the breathing circuit which ensures that there is no leak distal to common gas outlet.
2. During pre oxygenation observe the movements of the reservoir bag which ensures that there is good mask fit and adequate gas flow.
3. Check that the suction is working.

Artificial manual resuscitator should be present in all operation theatres.

Do you know of any intermittent flow anesthesia machines? Where were they used?

McKeesons Simplor, Juno Mk II, Walton 5, Cyprane A E, Nargraff: these are few intermittent flow anesthesia machines. They were used for dental anesthesia. They were also called demand flow machines.

How do you calibrate an oxygen analyzer?

There are two types of oxygen analyzers: a galvanic type sensor (an older "plug in" type), and the paramagnetic. For the galvanic oxygen sensor, first calibrate it to room air. It should take 15–20 seconds to read 21%. If it takes longer than 40–60 seconds, then change the sensor. Then expose to 100% oxygen and ensure it reads close.

Newer paramagnetic sensors use internal calibration routines. So they only need periodic (every 3-6 months) exposure to calibration gas. These sensors are longer lasting.

During a general anesthetic the oxygen analyzer is reading an FIO2 of 0.2 (and declining) what will you do?

1. Always trust the monitors until they are proven wrong.
2. Call for help
3. Turn on emergency oxygen cylinder and disconnect pipeline from wall
4. If inspired oxygen concentration doesn't increase in spite of adequate fresh gas flow, manually ventilate the lungs with an Ambu bag and room air. Use a portable oxygen cylinder if available.
5. Start CPR early.

How would you check for leaks in the low pressure system of the machine?

Leaks in the low pressure system could be either due to cracks in the rotameter which can result in hypoxic gas mixture or due to leaks in the vaporizers which can lead to patient awareness. Leaks could also result from failure of tubing connection.

A positive pressure leak test is carried out in machines which do not have outlet check valve (Drager). After shutting off all the gas flows and the vaporizers a known amount of positive pressure (usually about 50 cm H_2O) is applied to the common gas outlet. The manufacturer of each machine would specify how much of leak is acceptable over a specified period of time. This test is again repeated with the vaporizer turned on.

A negative pressure leak test is carried out in machines which have outlet check valves (Datex Ohmeda). All the gas flows and vaporizers are shut off. Then a negative pressure leak test device is connected to the common gas outlet via a 15 mm diameter connector. This device has a squeeze bulb which when evacuated applies a negative pressure of -65 cm H_2O to the low pressure system. The bulb reinflates if there is any leak in the system. This test is repeated with the vaporizer turned on. If it takes more than 30 seconds for the bulb to reinflate then it is usually acceptable. As the bulb has a volume of 15 ml, reinflation in 30 seconds would indicate a leak of 30ml/min. The negative pressure leak test is the universal test for all machines.

Describe the procedure for checking a Boyle's anesthetic machine; which uses cylinders as the source of compressed gases.

If an oxygen analyser is available, use it. It is the only way to verify the contents of an oxygen cylinder.

- Check that cylinders are securely attached and turned off.
- Open all flow meter control valves and check there is no flow.
- Turn on oxygen cylinder and check its contents on pressure gauge. Set the rotameter to read 4 L/minute. If a second oxygen cylinder is present, turn off the first and check the contents of second. Check there is no flow at the nitrous oxide rotameter. Turn off the second cylinder open the flow meter and bleed the system
- Turn on the nitrous oxide cylinder and check the contents on the pressure gauge. Try to start nitrous oxide flow. There should not be any flow of nitrous oxide if the master slave mechanism is functional. If a second nitrous oxide cylinder is present turn it on to check its contents then turn it off again.
- Turn off the oxygen cylinder and empty system via oxygen flush. The oxygen warning device should sound (if fitted), and should vent all gases from the machine. There should be no flow at the common gas outlet.
- Turn on the oxygen cylinder again.
- Check that the vaporizers are properly fitted to the back bar, with no leaks. They should contain an adequate amount of volatile anesthetic agent and the controls operate throughout their full range without sticking.
- If the anesthetic machine is fitted with a pressure relief valve it should be tested by occluding the common gas outlet with a thumb whilst gas is flowing. The pressure relief valve (PRV) should open with an audible release of gas. Do not do this test if a PRV is not fitted as it may damage the vaporizers.
- Check your breathing circuit to ensure that it has been assembled correctly, close the valve, fill with gas and squeeze the reservoir bag to ensure there are no leaks. Open the valves following this check and ensure circuit empties.
- Check the function of other equipment such as ambu bag, suction apparatus and laryngoscopes and ensure that all the drugs, endotracheal tubes, facemasks and airways you may require are present.

1993 FDA Anesthesia Apparatus Checkout Recommendations are applicable to the majority of anesthesia machines in use worldwide. Since all workstations are not the same the machine check has to be modified accordingly. 2007 guidelines elaborate only specific systems and subsystems that must be evaluated. Ultimately the user, in conjunction with workstation manufacturers, and local policy should develop institution specific guidelines for machine check. It is very important to refer to manufacturer's operator's manual for special procedures or precautions related to particular workstations.

Refer to 2007 guidelines for pre anesthesia checkout procedures.

Refer to www.ASAHQ.org/clinical/FDA.htm for some institution specific checkout procedures.

Summarise the safety features on anesthesia machine.

- Antistatic low friction black rubber wheels to the machine trolley
- Pipelines :
  - The wall outlets for pipelines are labelled and colour coded for the specific gas.
  - Primary valve or automatic shutoff valve at the wall outlet.
  - Secondary valve or Isolation valve at the wall outlets.
  - The Schraeder probes or quick connectors for the pipeline terminal outlets are gas specific.
  - Also the DISS on the machine end of the pipeline prevents interchangeability of the gas connections.
  - The pipeline hoses are color coded.
- Cylinders:
  - Color coding
  - Pin index safety system

  - Safety relief valve on the cylinder
  - Symbol of the gas on the cylinder valve.
  - Label on the cylinder
  - Filling on cylinder within the service pressure.
- Check valves at the cylinder inlet and pipeline inlet on the machine.
- Bourdons pressure gauge.
- Pressure regulators.
- Pressure relief valves downstream of the pressure regulators.
- Oxygen flush valve for emergency oxygen.
- Oxygen failure safety alarm.
- Oxygen supply pressure failure safety device.
- Hypoxia guard at the flow meter.
- Flow meters:
  - Colour, touch and placement coding
  - Rotating bobbin
  - Antistatic spray in the flow meters
  - Downstream placement of Oxygen flow meter.
  - Radio-florescent plastic sheet behind flow meters
- The interlocking selectatec system prevents more than one vaporizer being used at the same time. If the locking lever is not engaged the control dial on the vaporizer cannot be moved. The Selectatec block on the back bar of the anesthesia machine allows the vaporizers to be changed rapidly without interrupting the flow of carrier gas to the patient.
- In the older machines:
  - Placement of the vaporizer
  - Copper plunger in Boyle's bottle
  - Trilene interlock with closed circuit (in older machines)
  - Black tubings of breathing systems.
- Non return valve downstream of the vaporizer prevents backpressure which might cause output of high concentration of vapour. (Datex)
- Pressure relief valve situated downstream of the vaporizer prevents damage to the flowmeters or vaporizers if the common gas outlet is obstructed.
- Soda lime and it's indicator in closed circuit.

The Gas Laws

Boyle's Law

For a constant amount of gas at a constant temperature, the product of the pressure and volume of the gas is a constant.

$$P_1V_1 = P_2V_2$$

Charles's Law

For a constant amount of gas at a constant pressure, the volume of the gas is directly proportional to the absolute temperature.

$$V_1/T_1 = V_2/T_2$$

Gay-Lussac's Law

At a given temperature and pressure, equal volumes of gas contain equal numbers of moles. This is sometimes also known as Avogadro's law.

$$P_1/T_1 = P_2/T_2$$

Avogadro's hypothesis

Equal volumes of gases at the same temperature and pressure contain equal numbers of molecules.

Thus, 1 mole of any gas (i.e. 6.02 x 1023 gas molecules) at 1 atmosphere pressure and 0°C occupies a volume of approximately 22.4 L.

Each of these laws is a special case of a more general law. That general law is called the Ideal Gas Law.

$$P V = n R T$$

At standard temperature and pressure (commonly abbreviated as STP), the value of the temperature is 0°C (273 K) and the pressure is at 1 atmosphere (760 mmHg or 760 torrs or 1.01 x 105 Nm-2 [pascals]).

This implies that at STP, 32.0 g of oxygen gas will occupy a volume of 22.41 L. In addition, 44.0 g of nitrous dioxide will also occupy a volume of 22.41 L at STP.

Dalton's Law

Dalton's Law of Partial Pressure states that the pressure of a gas mixture is the sum of the partial pressures of the individual components of the gas mixture.

Graham's Law of diffusion

The rate at which gases diffuse is inversely proportional to the square root of their densities.

Henry's Law

Henry's Law states that at a given temperature, the amount of gas dissolved in a solute is directly proportional to the pressure of the gas above the substance.

Suggested Reading

1. Circle system. http://www.frca.co.uk/article.aspx?articleid=100143#
2. Dorsch, A Dorsch. E Understanding Anesthesia Equipment. 5th Edition
3. Davey J, Diba Ali. Ward's Anesthetic equipment. 5th Edition
4. Miller D. Miller's Anesthesia 6th Edition.
5. Parbrook GD, Davis PD, Parbrook EO. Basic physics and measurement for anesthetists. 4th Edition.

44 Vaporizers

P Ranganathan, M Shetmahajan

Define the following and explain the importance to anesthesia

- *Critical temperature:* **The temperature above which a substance in the gaseous phase cannot be condensed into a liquid, no matter how much pressure is applied.**
- *Vapour and gas:* A **vapour** is a substance that is in the gaseous phase below its critical temperature. This means that the vapour can be condensed to a liquid or to a solid by increasing its pressure without reducing the temperature, e.g. water vapour is the vapour form of water. The term **"gas**" is used to describe a substance that is in the gaseous phase above its critical temperature, e.g. nitrogen is a gas at room temperature and atmospheric pressure. It is important to know the difference between gases and vapour because gas laws are applicable to gases and may not always be applicable to vapours at normal room temperature, oxygen and nitrogen are gases whereas carbon dioxide, halothane and ether are vapours. The critical temperature of nitrous oxide is 36.5 °C. So at room temperature of 20 °C, it is a vapour. However in the tropics where the atmospheric temperature is more than 36. 5 degree celcius, it is a gas.
- *Vaporizers:* A vapourizer is an instrument designed to facilitate the change of a liquid anesthetic into its vapour and add a controlled amount of this vapour to the flow of gases going to the patient.
- *Vapour pressure:* It is the pressure exerted by the molecules escaping from the surface of a liquid to the gaseous phase in a closed container.
- *Saturated vapour pressure:* When the liquid and its vapour are in equilibrium, the vapour pressure is now called the saturated vapour pressure (SVP). SVP is dependent on only the temperature and nature of liquid.
- Vapour pressure of an agent determines how much of vapour will be formed from 1 mL of the liquid. Since different anesthetic agents have different vapour pressures, they need separate vaporizers, and the vapourizer cannot be interchanged between agents.
- *Boiling point:* The temperature at which SVP becomes equal to atmospheric pressure and at which all the liquid agent changes to the vapour phase is the liquid's boiling point.
- *Vaporizers output:* Refers to the concentration of vapour at the outlet of a vapourizer.
- *Vaporizers concentration:* Denotes the concentration delivered by a vapourizer when fresh gas containing no vapour passes through it. Therefore, in an out-of-system vapourizer, vapourizer output equals vapourizer concentration. In an in-system vapourizer, expired gases contain some volatile anesthetic, and therefore vapourizer output can exceed the vapourizer concentration.
- *Vaporizers capability:* Refers to the maximum concentration that can be delivered by a vapourizer at the highest setting of the concentration dial. Vapourizer capability depends on the MAC of the agent. For example, Sevoflurane has a higher MAC than Isoflurane and therefore needs a vapourizer with a higher capability (maximum 8% in case of Sevoflurane as compared to 5% with Isoflurane)
- *Vaporizers efficiency:* Means the ability of a vapourizer to saturate the carrier gas passing into the vapourizing chamber at the temperature of the liquid. Vapourizer efficiency is increased by using wicks, baffles and longer vapourizing chambers to increase the surface area available for vaporization.

How are gas concentrations expressed?

Two methods are commonly used to express the concentration of gas or vapour; partial pressure and volumes percent.

- *Partial pressure:* The part of the total pressure due to any one gas in the mixture is called the partial pressure of that gas.
- *Volumes percent:* It is defined as the number of units of volume of a gas in relationship to a total of 100 units of volume for the total gas mixture.

$$\frac{\text{Partial pressure}}{\text{Total pressure}} = \frac{\text{Volumes percent}}{100}$$

Why is ethyl chloride kept under pressure?

Ethyl chloride has a boiling point of 12.5 °C and is therefore a gas at room temperature. In order to liquefy it, it needs to be pressurized.

Calculate the amount of vapour produced by 1 gram of its liquid agent. How much liquid agent does a vapourizer use per hour?

1 mole of a gas weighs 1 gram molecular weight.
The molecular weight of Isoflurane is 185
Therefore, 1 mole of isoflurane vapour weighs 185 g
As per Avogadro's hypothesis, 1 mole of a substance = 22.4 liters of gas
22.4 liters of isoflurane vapour weighs 185 g
Therefore, density of isoflurane vapour is 185/22.4 = 8.25 g/L
Therefore, 1 gram = 1000/8.25 mL of vapour
Density of Isoflurane liquid is 1.5 g/mL
Therefore, 1 gram = 1/1.5 mL of liquid
1/1.5 mL of liquid = 1000/8.25 mL of vapour
1mL of liquid = 1000/8.25 × 1.5 = 180 mL vapour
For most modern agents, 1 mL of liquid volatile agent yields about 200 mL vapour.
The amount of vapour used per minute = fresh gas flow rate × time × concentration setting
For example, to deliver 1% Isoflurane at FGF of 2 liters per minute for 60 minutes, one would need
1/100 × 2000 mL × 60 min = 1200 mL of Isoflurane vapour
This would correspond to 1200/180 = 6.7 mL of liquid isoflurane
Another formula which can be used with most agents is
3 × Fresh gas flow (FGF) (L/min) × volume % = mL liquid used per hour

Classify vaporizers with examples

The most commonly used classification is the Dorsch and Dorsch classification.

1. **Method of regulating output**
   a. Variable bypass: Ether bottle, TEC vaporizers
   b. Measured flow: Copper kettle, side-arm vernitrol
2. **Method of vaporization**
   a. Flow over:
      - with wick—TEC vaporizers
      - without wick—Goldman bottle
   b. Bubble through: Copper kettle
   c. Flow-over or bubble-through: Ether bottle, depending on the position of the plunger
   d. Injection: Desflurane, Kion though Desflurane vapourizer has been described as an "injector", it is more ideally classified as a gas-vapour blender.
3. **Temperature compensation**
   a. Thermocompensated:
      - By altered flow—TEC vaporizers
      - By supplied heat—copper kettle
      - By both supplied heat and altered flow: EMO
   b. Non compensated: Ether bottle.
4. **Specificity**
   a. Agent specific: TEC vaporizers
   b. Multi agent: Goldman bottle.
5. **Resistance**
   a. Plenum (high resistance): TEC vaporizers
   b. Draw over (low resistance): Goldman bottle, EMO.
6. **Location**
   a. In circuit: VIC – EMO, Goldman bottle
   b. Out of circuit: VOC – TEC vaporizers.

Vaporizers may or may not be concentration calibrated. For example, the Goldman vapourizer is a variable-bypass vapourizer. However, the calibrations are arbitrary and do not indicate actual concentrations delivered. The Desflurane vapourizer has concentration calibrations. However, it is neither measured-flow nor variable-bypass.

Describe the functioning of variable-bypass vaporizers with an example.

Variable bypass vaporizers accept the total gas flow from the anesthesia machine and deliver the gas flow with a predictable concentration of vapour to the common gas outlet.

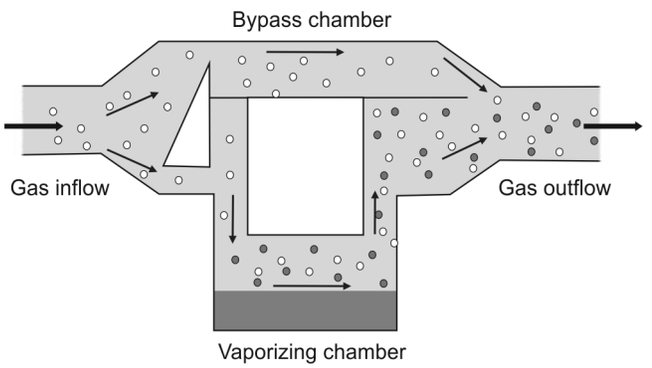


Fig. 44.1 Variable bypass vaporizer

The carrier gas flow entering the vapourizer is divided into 2 parts—one part going through the bypass chamber, and the other part going through the vapourizing chamber where it is saturated with the vapour of the liquid anesthetic agent.

Define splitting ratio

The ratio of the carrier gas flows through bypass and vapourizing chambers is called the splitting ratio.

- SVP of halothane at 20 °C is 243 mm Hg.
- At 20 °C and 760 mm Hg, halothane in the vapourizing chamber will contribute to 243/760 = 32% of volume (as per Dalton's law of partial pressures).
- Remaining 68% is made up of carrier gas.

Therefore, if 100 mL of carrier gas enters, it picks up 100/68 × 32 = 47 mL of halothane.

- Therefore, 147 mL of carrier gas + vapour exits the vapourizer.
- We want 1% halothane at total gas flow of 5 L/min to patient.
- We want 50 mL of halothane.
- Remaining 4950 is carrier gas.
- We know that 100 mL gas picks up 47 mL halothane vapour.
- To pick up 50 mL halothane, we need 106 mL gas.
- So 106 mL gas enters the vapourizing chamber and rest 4844 mL gas (4950 – 106) goes through the bypass.
- Therefore, for 1% halothane the splitting ratio is 106:4844 or 1:46.

The splitting ratio is affected by the concentration setting on the dial, and by the temperature-compensating mechanism in the vapourizer.

Splitting ratios for different agents—can vaporizers be interchanged?

Different anesthetic agents have different saturated vapour pressures at a given temperature. Therefore, the partial pressure exerted by the vapour in a mixture of gases will be different. Hence, splitting ratios are agent-specific and vaporizers cannot be interchanged unless 2 vapours have the same vapour pressure.

Describe the functioning of a measured-flow vapourizer.

This type of vapourizer utilizes a measured flow of carrier gas, usually oxygen, to pick up anesthetic vapour. The vapourizer system consists of 3 parts:

1. A vapourizer fitted with a thermometer
2. A flow-meter assembly
3. A vapourizer circuit control valve.

A measured amount of carrier gas flows through a flow meter and onto the vapourizer circuit control valve, which directs it to the vapourizer. The gas saturated with vapour is returned to the vapourizer circuit control valve, where it is diluted by the remaining fresh gas flow and goes to the patient.

To calculate the vapourizer output, one must know the vapour pressure of the agent, the atmospheric pressure, the total flow of gases and the flow to the vapourizer.

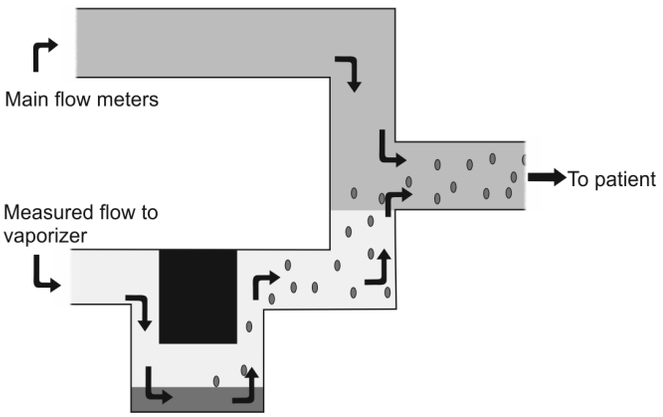


Fig. 44.2 Measured-flow vaporizer

% concentration of agent = 100 × vapourizer output of anesthetic/total flow

It is important to remember that:

1. The oxygen flowing through the vapourizer should be counted as additional to that going directly to the patient.
2. The vapourizer should not be turned on until the flows on the other flow meters have been set—a lethal concentration of agent may be delivered to the patient.

What does method of vaporization mean?

The method of vaporization refers to the pathway followed by the carrier gas as it travels through the vapourizing chamber.

What are the factors affecting efficiency of vaporization in a flow-over vapourizer?

1. *Area of the gas-liquid interface:* This can be increased by using wicks or baffles.
2. *Velocity of carrier gas flow:* Time is required to establish equilibrium between the fluid and its vapour, and this may not be possible at very high gas flows.
3. *Height of the gas flow above the liquid:* Gases passing closer to the surface of the liquid agent pick up more vapour than gases flowing at a height over the liquid. For example, in the ether bottle, vaporization can be increased by depressing the plunger so that gases pass under the hood and over the surface of the liquid

What are the factors affecting efficiency of vaporization in a bubble-through vapourizer?

1. *Size of the bubbles:* The smaller the bubbles, the more are the surface area available for equilibration. For example, in the copper kettle, a sintered bronze plate is used to break the gas flow into small bubbles to increase efficiency of vaporization.
2. *Depth of the liquid:* The deeper the liquid, the more is the time required for bubbles to reach the surface, allowing more time for equilibration. For example, in the ether bottle, depressing the plunger below the level of the liquid increases vaporization.
3. *Velocity of carrier gas flow:* The faster the flow of bubbles, the less is the time available for equilibration.

Define heat of vaporization, specific heat, thermal capacity, and thermal conductivity.

1. *Heat of vaporization:* It is the amount of heat required to vapourize one gram of a liquid at its boiling point with no change in temperature. Heat required to vapourize anesthetic agents is drawn from the remaining liquid and the surroundings. As vapour is generated, the temperatures of the vapourizer and remaining liquid fall. This causes the vapour pressure to decrease and would result in decreased vapourizer output if no compensatory mechanism were provided.
2. *Specific heat:* The specific heat of a substance is defined as the quantity of heat required to raise the temperature of 1 g of the substance through 1°C. Specific heat is important when considering the amount of heat that must be supplied to an anesthetic agent to maintain a stable temperature when heat is lost through vaporization. Specific heat is also important in the choice of material from which a vapourizer is constructed. A substance with a high specific heat will change temperature more slowly, and will therefore provide a more stable temperature for vaporization.
3. *Thermal capacity:* Product of specific heat and mass (amount of heat stored in the vapourizer body).
4. *Thermal conductivity:* It is a measure of speed with which heat flows through a substance. Substances with a high thermal conductivity can supply more heat to the liquid anesthetic in them. For example, copper has a moderately high specific heat (0.1 cal/°C/g) and a very high thermal conductivity. Hence, it is used in the construction of a number of vaporizers like the copper kettle. The copper kettle is placed on a copper surface to further enhance conductivity.

What are the different techniques of temperature compensation in vaporizers?

Temperature compensation in vaporizers can be by:

1. *Supplied heat:* Use of copper—copper kettle, which is placed on a copper surface, Water jacket—EMO, Electrically heated—TEC 6 vapourizer for Desflurane.
2. *Altered flow:* At lower temperatures, a higher percentage of carrier gas flows through the vapourizing chamber. Bimetallic strip—TEC vaporizers. Bellows valve—EMO. Annular valve of dissimilar metals.

What are the differences between in-system and out-of-system vaporizers?

Vaporizers are classified as being in or out-of-system depending on their location in relation to the breathing circuit.

Out-of-system vaporizers

All measured flow vaporizers are located out of the breathing system and have a dedicated flow meter. Plenum vaporizers (high-resistance) are also used out of system. These may be attached:

- Between the flow meters and the common gas outlet:
  - Advantage is that the vapourizer is securely mounted on the machine.
- Between the common gas outlet and the breathing system.
  - Advantage is that it is more convenient to move the vapourizer from one location to another
  - Disadvantages are:
    - Chances of tipping
    - Can have incorrect connections (reversal of flow through vapourizer)
    - Backpressure changes especially if the oxygen flush is used.

In-system vaporizers

In-system vaporizers are those in which the patient's inspiratory and expiratory gases go through the vapourizer. In-system vaporizers can be used as follows:

- In the circle system: Either in the inspiratory or the expiratory limb.
- As inhalers: Air is drawn through the vapourizer by the patient's respiration. facilities for supplementing oxygen and assisting ventilation may be provided.

Plenum vaporizers cannot be used as in-system vaporizers because:

- They offer resistance to breathing so they cannot be used as inhalers
- If used in the circle-system:

- In the expiratory limb, they offer resistance to inhalation
- In the inspiratory limb:
  - Vapourizer output is very high because the expired gas passing through the bypass chamber also contains vapour.
  - Flow rates during ventilation are very high and the vapourizer may not be calibrated for these flow rates.

Table 44.1 Differences between in and out-of-system vaporizers

In-system vaporizers	Out-of-system vaporizers
Low resistance is required as patient breathes through vaporizer. Draw over vaporizers and vaporizers-in circuit are in-system vaporizers e.g. Goldman, EMO	Low and high resistance vaporizers can be used as resistance is overcome by pressure of carrier gas. e.g. TEC 3 onwards
Expired gas passes through vaporizer increasing vaporizer output. Inspired anesthetic concentration (IAC) is always higher than the dialed concentration. Increasing gas flows decreases IAC to approximate dialed concentration	Vaporizer output and vaporizer concentration are equal. Inspired anesthetic concentration is lower than the dialed concentration
Vaporizer with low capability desirable	Vaporizers must have high capability
Vaporizer output is dependent on minute ventilation	Vaporizer output is not dependent on minute ventilation
Must possess easy access for washing and drying because it is exposed to expired gases from the patient	It does not require cleaning and washing

Describe the back-pressure changes that occur in vaporizers with the use of positive-pressure ventilation.

The use of controlled or assisted ventilation leads to transmission of positive pressure along the breathing system to the vapourizer. This can also occur with the use of the oxygen flush device. The intermittent back-pressure can lead to:

- Pumping effect: Increase in vapourizer output
- Pressurizing effect: Decrease in vapourizer output.

Mechanism of pumping effect

In variable bypass vaporizers, an increase in positive pressure at the outlet causes compression of gas in the vapourizing chamber and bypass. When the back pressure decreases suddenly, the pressure in the bypass chamber falls more quickly because it is a larger chamber, with less resistance to flow. The gas in the vapourizing chamber which is saturated with vapour exits from both the outlet and the inlet of the vapourizing chamber, and thus vapour-rich gas enters the bypass. Therefore, the overall vapourizer output is increased.

In measured-flow vaporizers, when back pressure is applied, gas is forced back into the vapourizing chamber where it picks up additional vapour, thus increasing vapourizer output.

The pumping effect is more with low flows, low dial settings and when the level of liquid agent in the vapourizer is low:

Modifications to minimize pumping effect.
Modifications in variable-bypass vaporizers.

- Changing the size of the bypass and the vapourizing chamber:
  - By making the vapourizing chamber smaller and the bypass larger—TEC 4 vapourizer has a much smaller vapourizing chamber than the TEC 2.
- Preventing extra gas flow to the vapourizing chamber from picking up anesthetic vapour:
  - By making the inlet of the vapourizing chamber longer
  - By excluding wicks at the inlet of the vapourizing chamber
  - By incorporating a baffle system or a positive pressure assembly in the vapourizing chamber.
- Back-pressure valves:
  - A one-way valve can be inserted at the vapourizer outlet.

Modifications in measured-flow vaporizers:

- Smaller vapourizing chamber
- Longer outlet tube
- Relief valve or check valve at the outlet

Modifications in the machine:

- Pressure relief valve or check valve to limit transmission of pressure to the vapourizer
- As per ASTM machine standard, the pressure transmitted to the vapourizer during use of oxygen flush should not be more than 100 cm H_2O above the normal working pressure, and the vapourizer output should not vary by more than 20% when the oxygen flush is used.

Mechanism of pressurizing effect

Pressurizing effect occurs when carrier gas is compressed in the vapourizing chamber. However, since the number of molecules of vapour in the vapourizing chamber does not increase, the carrier gas cannot pick up additional molecules of vapour. This results in a dilution of anesthetic in the vapourizing chamber and a decrease in vapourizer output.

The pressurizing effect is more at high gas flows. There is usually an interplay between the pressurizing and pumping

effects. Changes in vapourizer output are more with the pumping effect and are of greater clinical significance.

What are the hazards of vaporizers?

- *Incorrect concentration delivered*
  - High concentration:
    - Liquid anesthetic in delivery tube
    - Pumping effect
    - Reversed flow
    - Agitation
  - Low concentration:
    - Cooling of liquid
    - Pressurizing effect
    - Wicks covered with liquid
    - Very low/high flows.
- *Incorrect agent:* Use of the wrong agent in the vapourizer can lead to delivery of dangerously high concentrations. This is minimized by:
  - Agent specific filling devices
  - Color coding
  - Use of agent monitors.
- *Tipping:* This can lead to entry of liquid agent into the bypass chamber. Dangerously high concentrations of vapour will be delivered when the vapourizer is turned on. In free-standing vaporizers, liquid agent can directly enter the breathing circuit and the patient's lungs. This can be prevented by:
  - Mounting vaporizers on the manifold
  - Draining the vapourizer before being moved
  - Newer vaporizers like the TEC 4 are designed to avoid spillage (baffle system) even when turned 180 degrees
  - The TEC 6 vapourizer for Desflurane goes into standby mode if it is tilted more than 10 degrees. If tipping is suspected, the vapourizer should be flushed with high gas flows for 15 to 20 minutes before being used.
- *Overfilling:* The problems with overfilling are:
  - Liquid agent can enter the fresh gas line leading to high concentrations
  - If the wicks are completely submerged, the surface area for vaporization is decreased, leading to a fall in vapourizer output. This is prevented by:
    - Low level filling port and liquid level indicator glass
    - Use of agent specific filling devices
    - Filling only up to level indicated.
- *Reversal of flow:* With reversal of flow through the vapourizer, output is increased. This can be prevented by.
  - Mounting the vapourizer on the manifold
  - In free-standing vaporizers, use of the arrows to indicate the direction of gas flow.
- *Leaks:* Can lead to:
  - Wastage of agent
  - Delivery of wrong concentrations
  - OT pollution
  - Prevented by
    - Machine check including negative pressure check
    - Vigilance for vaporizers needing excessive filling or for odor of agent
    - Agent monitoring.

Enumerate the safety features of vaporizers.

- Agent specific filling systems
  - Prevent accidental filling with wrong agent.
  - Reduce air pollution during filling.
  - Prevent water and contaminants from entering the vapourizing chamber.

  Consist of a color-coded bottle collar and a matching color-coded adaptor, which fits into the vapourizer filling receptacle
- *Low filling port:* This minimizes overfilling because the filler port is located at the maximum safe liquid level.
- *Secured vaporizers* (less ability to move them about minimizes tipping).
- *Interlocks:* Prevent administration of more than one inhaled anesthetic at a time.
- Concentration dial increases output in all when rotated counterclockwise (as seen from above).

Classify and give a brief description of Flagg's can

Flagg's can consists of a square tin can, later replaced by plastic. The lid had a hole through which a rubber tube was used to connect the can to the endotracheal tube. Side holes in the lid allowed entry of air and could be partially closed to adjust the concentration of ether. The area of the holes was equal to the area of the trachea, to minimize resistance to breathing. Oxygen could be supplemented by inserting a catheter connected to oxygen source via one of the holes. The tip of this catheter should be kept above the level of the liquid ether to prevent bubbling of ether leading to dangerously high concentrations.

Hazards of Flagg's can

Tipping, leading to direct entry of ether into the tube and respiratory tract.If side holes were completely obstructed, hypoxic mixture will be delivered to the patient.

Describe devices used for ether anesthesia.

Schimmelbusch mask, Yankauer's mask

Draw over, non-temperature compensated, multi-agent. Used for open-drop method of anesthesia. Could be used with ether, chloroform, ethyl chloride, etc.

Metal frame over which gauze or lint is spread. In case of ether, the liquid is dropped evenly over the whole surface, whereas with chloroform, it is restricted to one-half of it to ensure that air is freely drawn and the concentration of the vapour is not too high.

For ether, 16 layers of gauze are used whereas for chloroform or ethyl chloride, 12 layers of gauze or (295.7 mL) 1 layer of lint are applied.

When ether is being used, the high rate of vaporization requires so much latent heat that the mask becomes cool and water vapour from the exhaled air may condense on it and freeze. This causes resistance to flow of air through the gauze and also reduces the rate of vaporization. Therefore, a second mask or a fresh supply of gauze is needed during long administration.

When using the Schimmelbusch mask, the patient's face is covered with a piece of gamgee with a central hole to expose the patient's nose and mouth. This prevents vapour or liquid from entering the patient's eyes and also reduces the amount of air drawn in by the patient, between the mask and his face. A piece of gamgee may also be used to cover the mask

Ogston's inhaler

Semi-open drop anestheisa. Consists of a mask surmounted by a wire frame around which a napkin or some gauze could be erected to keep the vapour trapped.

Classify and give a brief description of Boyle's bottle

Boyle's bottle (vaporizer)

Classification:

- Variable bypass, flow over without wick, out of system bubble through.
- No temperature compensation, not calibrated.
- Agents used are:
  - Ether
  - Methoxyflurane
  - Trilene
  - Chloroform
  - Halothane.

Function

- With the lever in 'off' position—gas flows through bypass entirely.
- As the lever is turned towards the 'on' position, an increasing proportion of gases pass through the bottle.
- In full on—all gases pass through bottle.
- When the cowl is up, gases flow directly from the U-tube to the outlet of the vapourizer.
- Gas may be diverted by lowering the cowl over the end of U-tube and gases are forced to impinge on surface of ether.
- If cowl is fully down, then gas bubbles up through liquid to produce maximum vaporization.
- The position of the cowl is adjusted by a plunger, which passes through a gland to maintain a gas-tight seal.
- The control drum rotates inside the body of the vapourizer and is positioned laterally by adjusting and locking rings at each end.
- Special grease is used to maintain a seal and yet allow rotation. There is spare grease in the grease injector cap (at the back of the vapourizer), and by turning this, it can be fed into the seating of the drum.
- As gases and vapours pass through the vapourizer, the grease tends to be slowly washed away and this results in stiffness if extra grease is not added.
- The plunger may become loose and tend to fall down on it. This can be corrected by tightening the gland nut.
- The packing in the gland may be of cotton, neoprene and nylon, and needs replacement when it wears out.
- There is a filling orifice that is blocked by a cork stopper, which is retained by a small chain. The cork should have a good fit to prevent leak of gases.
- In some vaporizers, the metal anchor for the retaining chain passes through the cork. If the chain is broken, the metal core of the cork can act as a spark plug is someone who is charged with static electricity touches it. Hence, it is important that the chain is intact. Sometimes, the top of the cork is insulated to prevent this hazard.
- The bottle sealing washer is made of cork and may have a canvas or metal insert. If this is damaged, there may be a leak of anesthetic gases and vapour.
- Copper in ether bottle acts as an anti-catalyst preventing decomposition of ether.
- Some bottle are colored (brown/green) to prevent decomposition by light.
- Two bottles—broad one for ether, narrow one for other agent.

Evaluation and use

- Graduation of control lever is arbitrary and as rule it starts functioning at mark II.
- Ether vapourizer—knob is turned on slowly, usually one-quarter of a division after 4 consecutive regular breaths.
- Ether bottle—270 mL of ether used.
- Trilene and chloroform—½ inch in depth of anesthetic used.
- Temperature is maintained by wiping bottle from outside or by wrapping the bottle with warm gamgee.
- Volatile agents used in the vapourizer must be removed from the bottle after use and be discarded in order to prevent a risk of being used in other bottle.

- The concentration of vapour depends on the temperature, the level of liquid anaesthetic agent, and the rate of gas flow.
- Minimum ether concentration with 10 oz ether filled, 8 liter gas flow is 4.5%.
- Maximum concentration with cowl at highest position 14.5%.
- Maximum concentration with cowl fully depressed for maximal bubbling 40%; but this falls rapidly as the ether cools and temperature falls.
- In case of chloroform and trichloroethylene, a sufficient concentration is produced by turning the control knob fully on, and depressing the plunger may lead to dangerously high concentrations.
- This bottle may not produce adequately high concentrations for induction with methoxyflurane.

Cleaning and care

- Liquid is removed after use
- Clean and dry vapourizer.

Hazards

- Lever gets stuck due to thymol, which is used as a preservative
- Plunger may get stuck.
- Ether is very volatile and there may be a build-up of vapour in the bottle when it is turned off, especially in warm environments. This can lead to a surge of high-concentration ether vapour when the vapourizer is turned on. This can be prevented by running a small quantity of gases through the vapourizer prior to commencement of anesthesia, or by removing the cork from the filler orifice for a brief period
- Since agents are not color coded, the residual agent in the Boyle's bottle may be poured back into the wrong bottle. It is preferable to discard residual agent, to avoid this complication.

What are the differences between the Boyle's bottles for ether and trilene?

The ether bottle has a broad base to allow greater surface area for evapouration. It has markings up to 300 cc. The trilene bottle is narrow to prevent excessively high concentrations of vapour. It has markings up to 100 cc.

What is the trilene interlock?

Trilene combines with soda lime to form phosgene which is a toxic gas. The trilene interlock is present on the back bar of the machine. This ensures that gases cannot flow to the soda lime canister when trilene is being used.

Classify and describe EMO

1. Variable bypass
2. Concentration calibrated
3. Draw over or flow over
4. Agent specific
5. Temperature compensated by supplied heat and by altered flow
6. Vapourizer in and out of system.

The EMO system consists of vaporizers alone or in series, valves, bellows or self-inflating bags and accessories.

The EMO inhaler (Epstein and Macintosh, 1956)

Construction

It is 23 cm in diameter, 24 cm in height and weighs 6.5 kg with the water compartment full. It has an annular ether vapourizing chamber (V), lined with wicks. Surrounding this is a water compartment (W) and an automatic thermocompensating valve (T). The air passages are relatively large, offering a low resistance to breathing very low resistance to flow (less than 1.25 cm H_2O at 40 L/min).

The air inlet communicates with a large chamber with two outlets. One gives access to the main ether chamber via the closing mechanism (L) which seals off the entry to the ether chamber when the control indicator is in the 'transit' position. Also incorporated in the closing mechanism is an inlet relief valve, which allows air to enter the inhaler if the main air inlet becomes blocked. The other outlet port leads into the small chamber in the center of the control rotor which is fully open in the 'transit', position but as the concentration indicator (C) is moved around towards the 20% position, it closes progressively, so directing a greater proportion of the air into the ether chamber.

The central chamber has a second inlet port leading from the ether vapourizing chamber via the thermocompensating valve (T). This port is fully closed in the 'transit' position but opens progressively as the indicator is moved towards the 20% position, permitting extra air to leave the vapourizing chamber. The control mechanism thus directs progressively larger proportion of the air through the ether chamber without altering the total flow through the inhaler. It also seals the ether chamber when the inhaler is not in use. The temperature compensating thermostat allows more air to pass through the vapourizing chamber when the ether gets colder, maintaining the ether output concentration constant between 15° and 30°C adjusting automatically for changes in barometric pressure. The active element is a sealed capsule containing liquid ether. It should normally last for 10 years.

The indicator on the compensating unit, which shows red if the temperature is two high (over 32°C). At normal working

temperatures (20–25°C), the metal top and black band will show. Mark III EMO's from 1964 do not have this feature. If the ether in the chamber becomes too hot (after storage in a hot environment), some ether must be vapourized by opening the filler and sucking air through with the bellows which has the effect of cooling the inhaler and will prevent a dangerously high concentration being delivered in the first few breaths. As ether vapourizes, heat is consumed at the rate of 89 calories per gram of liquid.

When supplied, the water jacket is dry and must be filled with approximately 1200 mL of cold distilled water via the filler (F). Mark I EMO's have aluminum water jackets. Water level must be checked every 3 months because chemical action may lead to generation of gas which may damage other parts of the inhaler. Mark II, Mark III, Mark IV EMOs have stainless steel jackets and need only yearly water level checks. In freezing conditions, automobile anti-freeze solution containing 25% glycol should be used.

When filling the control must be turned to the zero position rather than to the 'transit' position so that air can escape from the vapourizing chamber. As ether is poured in, the filler must be held down. Mark I inhaler have fillers, which must be lifted and turned. If left open by mistake, extra air enters the chamber and vapourizes too much ether. The inhaler must not be overfilled otherwise the wicks will be covered and vaporization of the ether will be decreased. 150 mL is required to fill it to the 'Empty' mark (this being the amount soaked up by the wicks), further 300 mL to raise the level to 'Full'.

An EMO requires no maintenance for 5 years. The main fault is that the control pointer may stick—this is most likely to occur if an inhaler is left for long periods full of ether without being used. It may also follow the use of ether dispensed from lacquered cans. Use of trichloroethylene or halothane, apart from corroding the metal of the interior, will also lead to the deposition of gummy material on the rotor, causing it to stick. If the unit is going to be stored for any length of time, drain the ether chamber and open the control fully to allow all the vapour to escape, otherwise oxidation of the ether will occur.

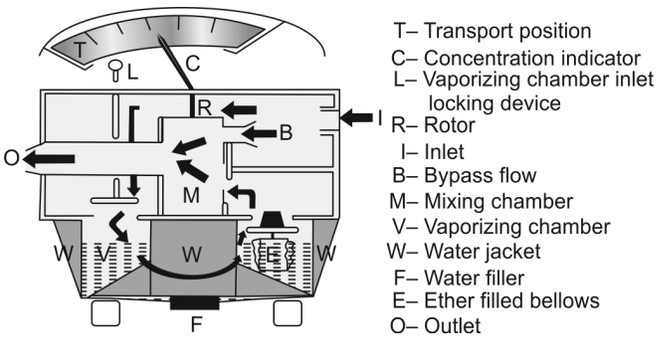


Fig. 44.3 Epstein-Macintosh-Oxford inhaler

What are the techniques of temperature compensation in EMO?

Temperature compensation in EMO occurs by:

1. Supplied heat by the water jacket
2. Flow alteration at the outlet of the vapourizing chamber via the ether bellows.

How is the EMO checked?

The level indicator is checked by inverting the inhaler (with concentration control at 'Transit'), when it should fall freely to the 'Full' position. Connect the bellows to the inlet and block the outlet. While the control knob is turned to the 'Transit' position, the filler held open, the bellows are compressed and no air should escape. The filler is released, the control pointer turned to 10%, and this is repeated to check that the filler is not leaking. Bellows is then attached to the outlet, inlet is blocked, and the control knob is set at '2' to suck air through the safety release valve. If the valve is working correctly, air can be heard hissing through.

Describe the Oxford Miniature Vapourizer (OMV)

This is designed for less volatile agents than ether, such as halothane, methoxyflurane and trichloroethylene. In particular it is meant as an induction unit for use with the EMO, when it should be plugged into the outlet of the latter. It should never be fitted to the inlet of the EMO, otherwise halothane will be drawn in, causing corrosion. It can be used with a continuous gas flow for supplementation of relaxant anesthesia. When combined with an inflating device it can be used as a draw-over anesthetic apparatus on its own to provide anesthesia or analgesia.

It is 13.5 cm high and weighs 1060 g (with the water jacket full). There is a scale, which gives volumetric percentage concentration at 25°C with small deviations from the indicated concentrations with time and temperature. The output is never greater than indicated and is unaffected by positive pressure, so it is safe to place it on the patient's side of the bellows. Alternative scale are for halothane (HALO – 4%), methoxyflurane (MOF – 0.6%) and trichloroethylene (TCE – 1.5%). The base contains a water jacket which acts as a heat source. When used with the EMO, the flow of gas through the vapourizer should be from the right to left (when looking at the front). Another version designed for gas machines in which the gas flows from left to right. The direction of flow is marked by an arrow.

The OMV has special filler designed to limit the volume of anesthetic used. It is a useful economy device operated by a lever which must be pressed fully down to open the filling port. The initial movement of the lever opens

an air relief valve; heavier pressure must be applied to open the actual filler. The built-in funnel surrounding the filler contains about 10 mL which is convenient for most induction purposes. It is enough to cover about one-eight of the level indicator. A second 10 mL dose may be added initially if desired, but no more. The indicator is designed so that some liquid remains in the chamber even when it can no longer be seen in the window. The wicks are made of stainless steel gauze, which will not be corroded by any anesthetic in current use.

What are the accessories for use with the EMO and OMV?

Oxford Inflating Bellows (OIB)

It is 3.5 kg and is 25 cm long by 23 cm high and 18 cm wide spring loaded concertina bellows with 2 non-return flap valves. It was originally intended for use with a simple spring-loaded expiratory valve. This arrangement works well with spontaneous respiration, for artificial respiration is less satisfactory. For IPPV, a non-rebreathing or inflating valve is usually preferred. Compression of the bellows during artificial respiration will produce full deflection of the valve flaps or bobbin during inspiration. In order to draw the bobbin or flaps back and hence to permit the patient to expire to atmosphere, a very small amount of air must pass back up the corrugated tube towards the bellows. If the distal bellows valve is not immobilized, air will be unable to pass back and the non-rebreathing valve will stick in the position of inspiration. A magnet is therefore provided for immobilizing the distal flap. The magnet must be fitted whenever a non-rebreathing valve is used. The bellows have internal springs; press-studs inside the concertinas keep them closed during storage and transport. The bellows are capable of delivering a volume of about 1300 mL; a 10 cm stroke will deliver about 800 mL.

The Penlon Bellows Unit (PBU)

This has a single flap valve designed specifically for use with a non-rebreathing valve. It must never be used with a simple spring loaded expiratory valve. It weighs only 1 kg.

A special Pediatric bellows of smaller diameter has a full stroke capacity of about 400 mL the EMO is also supplied as an alternative with the Ambu double-ended self-inflating bag. The bag is placed between the vaporizers and the non-rebreathing valve.

How is the EMO used with the anesthesia machine?

The flow meter is plugged directly into the air inlet of the EMO This arrangement can allow the EMO to operate as a plenum (constant gas flow) vapourizer, allowing a lower than indicated concentration of ether to be delivered if the fresh gas flow is less than 10 liters per minute. If the fresh gas flow is less than the patient's minute volume, air will still be drawn through the inhaler. Danger is that the bellows overfilling and a non-rebreathing valve sticking if the fresh gas flow is greater than the patient's minute volume.

What are the means of enriching the inspired mixture in an EMO with oxygen?

A tap for addition of oxygen is provided on the bellows, which is intended for use only in resuscitation and should always be kept closed during anesthesia. Oxygen is added to the inhaled air through a narrow delivery tube passed into the air inlet of the EMO However, the best way of introducing oxygen is with the T-piece, gas reservoir adapter and the reservoir tube, which avoids the dilution of anesthetic vapour, which occurs when oxygen is led directly into the OIB or PBU Concentration of oxygen achieved will depend both on the volume added and the patient's minute volume.

How is the EMO used for artificial respiration?

By virtue of its bellows, the EMO apparatus can be used for artificial respiration in any situation. This applies equally to the OIB (Oxford Inflating Bellows) and the Penlon Bellows Unit (PBU). In either case, an automatic non-rebreathing valve is essential. Alternatively, a self-inflating bag may be used. A magnet should always be used with the NRV on the expiratory side of the OIB, to prevent sticking of the valve. When using a non-rebreathing valve it should be remembered that the movements of the bellows or bag effectively operate the valve. With most types of automatic non-rebreathing valve, it is possible by gentle pressure to blow air across the valve and out through the expiratory port instead of into the patient's lungs. In the case of Ruben valve, which has a hard plastic bobbin, the bobbin can be made to vibrate rapidly to and fro, making a shuddering noise. In either of these cases, ventilation won't be effective. The inspiratory movement should therefore begin smartly and continue throughout inspiration as a firm and definite compression of the bag or bellows. Similarly, at the beginning of expiration, it is possible to lift the bellows so slowly that the valve remains in the inspiratory position and air is allowed to pass back up the tubing towards the vapourizer, instead of out to the atmosphere. For this reason, inspiration should end, not with the bellows held down, but with a sudden upward movement, which is continued until the bellows has been refilled with air for the next breath. Similarly, the bag should be allowed to re-expand immediately. A pause for expiration must then be allowed.

The magnet on the OIB should never be used to immobilize the distal flap valve when a spring loaded expiratory valve is used. Spring-loaded valves should never be used with the PBU In either case, rebreathing into the bellows would occur.

What are the principles for the arrangement of the EMO apparatus?

1. If vaporizers are to be placed in series, the vapourizer for the more volatile agent should be placed upstream (i.e. further from the patient) from the vapourizer for the less volatile. If this point is neglected, condensation of the less volatile agent (which will have a higher boiling point) will occur in a cooler downstream vapourizer. EMO should therefore always be upstream of the OMV When using serial OMVs for halothane and trichloroethylene, the halothane OMV should be upstream.
2. The bellows or inflating bag should be placed between the vapourizer and the patient. If air is blown through the vapourizer, the concentration delivered may be greater than indicated. This particularly affects the EMO because of its large internal volume, but OMV will not be affected, but it is nevertheless preferable to keep the vapourizer upstream. The exception is that when the PBU is used with the EMO and OMV, the bellows should be placed between the two vaporizers to avoid overbalancing them.
3. Oxygen and nitrous oxide (when used) should always be introduced on the upstream side of the vaporizers. This avoids the dilution of the vapour, which will occur if the gas is introduced downstream. The oxygen inlets on the OIB and PBU are only intended for use in resuscitation.
4. When using the OIB the magnet must be used to immobilize the downstream flap valve whenever an automatic non-rebreathing valve is used. This valve must not be immobilized if a simple spring-loaded expiratory valve is used.
5. Before using the apparatus to anesthetize the patient, the bag or bellows must be operated in order to ensure that the flow of air is in the correct direction (i.e. towards the patient).

How is the EMO used in pediatric patients?

In pediatric patients, the EMO cannot be used as a draw-over vapourizer rather,

The dead space of the non-rebreathing valves and connections used for elder children and adults are too great for infants and children below the age of 3 or 4. Moreover, the inspiratory flow rates generated by such children are too small for satisfactory vaporization of ether in the EMO For these reasons, the techniques described for adults are not suitable and the EMO must be used as a constant-flow over vapourizer. The pediatric entrainer was designed to provide a flow of oxygen enriched air through the EMO in order to vapourize the ether. It consists of fine jet, through which oxygen passes into a Venturi-shaped tube. Air is drawn in and mixes with the oxygen. The entrainer is plugged into the air inlet of the EMO

Alternative means of providing a flow may be used. Nitrous oxide and oxygen may be used (5.0 L/min of oxygen and 5.0 L/min of nitrous oxide) delivered from a gas anesthetic machine, via the EMO flowmeter attachment. Whichever source of gas flow is used, the EMO is used as a constant-flow or plenum vapouriser. In order to deliver the correct concentration, a total gas flow of about 10 L/min is needed.

The pediatric entrainer is driven from an oxygen cylinder, preferably via a regulator (reducing valve). The flow of gas from the cylinder is increased until the manometer reads 100 mm Hg, when the entrainer will deliver the correct flow of 10 liters per minute. By using oxygen as the driving gas, an inspired oxygen content of about 35% is achieved. Although this is clinically advantageous, overcoming most of the effects of ventilation-perfusion inequalities renders an ether mixture explosive. When compressed air is used as the driving gas, the ether remains flammable but is no longer detonable. For the infants, it is nevertheless safer to employ an oxygen-rich mixture to minimize the risk of hypoxemia developing as a result of the patient coughing or holding his breath. If the use of diathermy or open flames is essential, a non-explosive agent must be used instead of ether. The consumption of driving gas is 2 liters per minute. The entrainment ratio is 1.5:1. If the outflow from the entrainer is occluded the high pressure gas escapes through the air inlet holes and the maximum internal pressure achieved is 15 cm H_2O, which is unlikely to harm the patient.

The entrainer is inserted into the air inlet of the EMO the bellows and OMV or BSIU is left in the circuit but the bellows is pushed down into the 'closed' position. The anesthetic mixture is led to the patient via the delivery tube to T-piece, one end of which leads to the patient via a facemask or endotracheal tube. The other end has a reservoir tube, which should have a volume greater than the patient's tidal volume. Too short a tube will result in air-dilution. The delivery tube of the Ayre's circuit is attached to the outlet of the OIB in place of the long corrugated tubing. When the Penlon bellows is used, the OMV or BSIU remains on the downstream side of the bellows. The delivery tube is then attached to the outlet of the accessory vapourizer.

The minute volumes of anesthetized children are found to be 1.5 L in a 1 year old child, 2 L in a 3 year old and 3 L

in a 5-year old. A fresh gas flow of 2.5–3 times the minute volume should be used with Ayre's T-piece to prevent rebreathing of the expired gas. The flow delivered by the entrainer is therefore adequate for the largest child likely to be anesthetized by this method. The possible disadvantage of using a flow greater than three times the minute volume is that the resistance to expiration will be increased. A flow of 10 L/min may be unnecessarily high for neonates, the advantage of knowing the concentration of ether outweighs this disadvantage. The peak expiratory flow in a child under one year will be less than 6.6 liters a minute. The maximum flow in the expiratory limb of the T-piece will therefore be 16.6 L/min. Therefore, with a T-piece of 10 mm diameter as recommended by Ayre, the resistance is likely to be negligible.

Classify and describe Goldman's vapourizer.

Classification

1. Variable by pass, draw over or flow over, can be used with or without wick, in or out of system
2. No temperature compensation
3. Multiple agent.

Model

1. Mark I
   a. Self-locking in off position
   b. Three divisions between off and on position.
2. Mark II
   a. Click stops at each settings
   b. Three divisions between off and on position.
   c. Size and shape of ports differs from Mark I.
3. Mark III
   a. Two divisions between off and on position
   b. Click at each setting.

Construction

It consists of:

1. Small glass bowl which will hold up 20 cc E liquid
2. Bowl is attached to head which receives gas and divides gas between vapourizing chamber and by pass
3. A control lever on the top is used to alter the vapourizer output and is turned counterclockwise to increase the concentration.

Evaluation and use

1. Mainly it is designed for attachment to the outlet of an intermittent flow machine.
2. Low resistance vapourizer.
3. Easy to operate and does not deliver high-concentration.
4. When vapourizing chamber is full, output concentration is 2%.
5. Used inside a breathing circle, it maintains fairly constant halothane concentration.

Care and cleaning

1. Drained periodically
2. Vapourizer chamber should be cleaned out and allowed to dry.

Hazards : Agitation or tilting will increase the vapour output.

Table 44.2 Halothane percentages in Goldman's vaporizer

Drum position	Gas flow rate		
	2 liters/min	8 liters/min	30 liters/min
1	0.03	0.03	0.03
2	0.41	0.74	0.92
3	0.73	2.21	1.31
on	0.74	2.08	1.21

How can the output of a Goldman's vapourizer be increased?

1. Agitation or splashing from filling markedly increases halothane concentration to over 5%. However this can lead to liquid agent entering the breathing circuit, and is hence not recommended.
2. By adding wicks—concentration is increased between 3–4% at III setting.
3. Two Goldman vapourizer in series, turning one to the 3rd position and altering the setting of other to increase vapour output.

Classify and describe copper kettle

Classification

1. Measured flow
2. Bubble through
3. Out of system
4. Temperature compensated by supplied heat and manual flow alteration
5. Multiple agent.

Construction: Each copper kettle is supplied with a vapourizer circuit control valve and a flow meter assembly. Copper is used in construction because of its high heat capacity and thermal conductivity.

Gas flow: Gas enters the vapourizer from the inlet and passes upward through the central tube into the surge

chamber or loving cup. This chamber prevents the sudden surge of gas into the vapourizer. The gas then passes downward around the center tube and enters the diffuser at the base of the vapourizer. There is a sintered bronze disk at the top of the diffuser. The disk conducts heat to the gas-liquid interface to prevent cooling. As the carrier gas permeates through the disk, it is broken into small bubbles, which rise through the anesthetic liquid. This increases the area for saturation with vapour. Gas saturated with vapour rises to the top of the kettle and passes out through the discharge tube. A thermometer is present to indicate the vapourizer temperature. A level indicator window is present. The filling port was on top of the vapourizer in older models, and is at the back in newer ones. Newer models also have a back pressure check valve at the outlet. The copper kettle is available in 2 sizes with capacities of 160 mL and 400 mL. A chart is available to indicate the flow needed through the vapourizer as per agent, temperature and required output.

Hazards: Calculations are needed to determine the flow of gas needed through the vapourizer for a particular vapour output. Erroneous calculations can lead to lethal doses being delivered. Failure to turn on the main gas flow can result in undiluted vapour delivery to the patient. When filled from the top, older models could be overfilled leading to spillage into the delivery tube. This is eliminated in newer models by changing the filling port to the side.

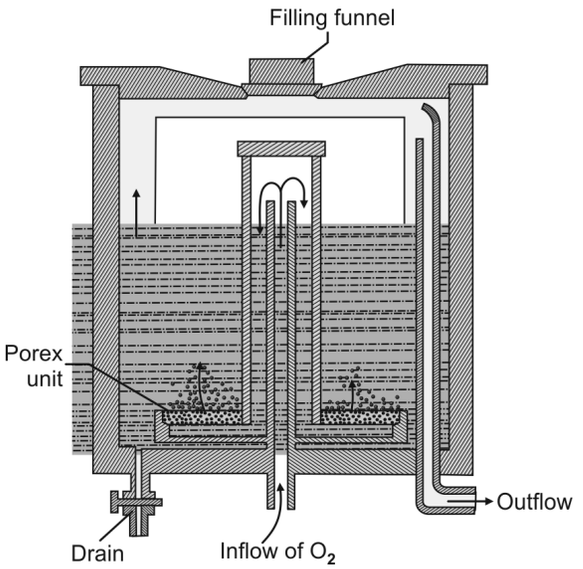


Fig. 44.4 Copper kettle

Classify and describe TEC vapourizer (TEC I to V) temperature compensated vaporizers – TEC

Classification

1. Variable bypass
2. Flow over with wick
3. Out of system
4. Temperature compensated by automatic flow alteration
5. Concentration calibrated
6. Agent specific.

Functioning of TEC 2, 3, 4, 5 and 7 vaporizers

The gases pass through the vapourizer by 2 channels, one leading through a bypass and the other through the vaporization chamber. The proportion of gases passing through the bypass is determined by a calibrated control knob that is attached to a spindle. In the off position, the vapourizing chamber is isolated from the bypass and all gases flow through the bypass to the fresh gas outlet. When the spindle is pulled towards the operator and rotated counterclockwise, the inlet and outlet ports of the vapourizing chamber are opened. The fraction of gas passing through the vapourizing chamber is also varied by a bimetallic thermostatic valve. This is located inside the vaporization chamber in TEC 2 vaporizers, but is in the bypass chamber in subsequent models. As the temperature in the vaporization chamber falls, the thermostatically operated valve opens wider and a greater proportion of the gas flow passes through the chamber. By this means, the vapour output is kept constant. The vaporization chamber contains a series of wicks, with a large surface area. This ensures that gases exiting the vaporization chamber are saturated with vapour.

TEC I

Introduced by Cyprane Company in 1956 as Fluotec I for halothane. All Mark I vaporizers were recalled, modified, and re-released as Mark II variants in order to correct a critical design flaw, which could cause the proportioning valve to stick thus posing a risk of overdosage to the patient.

TEC II

1. Halothane – FluoTec – Mark II
2. Methoxyflurane – Pentec - Mark I

Construction: parts

1. Vapour chamber is round and wicks are concentric and in series
2. Filling tap at side
3. Draining at bottom

4. Level indicator on side
5. External calibrated control knob is attached to a spindle that controls concentration—moves to right
6. Temperature compensation—bimetallic strip located at the outlet of vapourizing chamber and regulates the amount of gas that passes through the chamber.

Evaluation and use

1. Not accurate below 4 liters /min
2. With flows of 2 liters/min, at dial setting less than 2%, output is less than setting; with dial setting more than 2%, output is more than the setting
3. With nitrous oxide it gives greater output at lower setting and lower output at higher setting
4. With dial setting between off and 0%, some output can occur, varying with gas flows
5. Prone to back pressure changes—pumping effect at low flows and pressurizing effect at high flows
6. There can be a small leak in the off position.

Care and cleaning

1. Manufacturers recommend yearly return to company for servicing
2. Halothane: Preservative is thymol which accumulates in the vapourizer
3. Vapourizer is drained every 2 weeks
4. Drained agent: Discarded

Hazards

1. Thymol caused the operating spindle to stick
2. Back pressure forcing saturated vapour back into the bypass chamber
3. High concentration at low flows.

TEC III

Models

- Fluotec Mark III
- Pentec Mark II
- Enfluratec
- Fortec.

Construction

It consists of two sections:

1. Lower vapourizing chamber
2. Upper duct and valve system.

Single control dial: Controls the concentration delivered by this vapourizer. Movement of this dial opens and closes appropriate parts and thus regulates the proportion of gas passing through vapourizing chamber.

Parts

1. This model has larger bypass
2. The tube leading to the vapourizing chamber is longer and has an expansion area
3. Wicks are excluded from inlet area of vapourizing chamber – helps to decrease effects of back-pressure on vapourizer output
4. Concentration dial is on top and to the left of it is the locking lever which is depressed to turn on the vapourizer
5. Sight window for liquid level on left
6. Filling and draining at the bottom.

Evaluation and use

1. All are accurate with lower dial setting.
2. At higher dial setting, higher than expected concentration at low flow rates and lower than expected concentration at high flow rates.
3. Sudden increase or decrease in carrier gas flow, intermittent back pressure and upstream O_2 flush has negligible effect on vapour output.
4. Nitrous oxide has got little effect on output
5. Between OFF and 0.5% dial setting, output was less affected by fresh gas flows, as compared to TEC II
6. Small amounts of leaks in bypass in off position
7. Tipping up to 90 degrees does not affect output. Tipping more than 90 degrees causes an increase in subsequent output,

Care and cleaning

1. Manufactures annual preventative maintenance
2. Halothane to be drained— every 2 weeks.

Hazards

Besides common hazards, problems related to dial rotation have been reported:

1. Rotated beyond off position
2. Rotated by 180^0
3. Leak from dial setting became of damage or compression of gasket.

TEC IV

Models

- Fluotec – 4
- Enfluratec 4
- Fortec 4.

Construction

This is a modified version of TEC-III.These vaporizers are designed to be attached to the back bar of anesthesia

machine by means of Select-a-Tec manifold which allows easy removal and exchange of vapourizer.

Parts

1. On top is a control dial that is turned counterclockwise to increase the concentration. A release button located to the left of the concentration dial must be depressed before the vapourizer can be turned on.
2. There is a locking lever connected with the control dial, so that the vapourizer cannot be turned on unless it is locked to the manifold.
3. When the vapourizer is turned on, 2 plungers within the vapourizer operate to open the valve into the fresh gas stream. Also, when the vapourizer is turned on, 2 extension rods prevent the use of any adjacent vapourizer.
4. There are 2 filling mechanisms—a screw cap or a keyed filling system. A special adapter fitted the bottles of the specified agent only, whilst the other end was keyed for specific same agent vaporizers.

Gas flow

1. Off position—incoming gas flows from inlet through bypass and on to outlet
2. On position
   - Incoming gas is split into two streams by a rotary valve attached to the concentration dial
   - One stream is directed to the vapourizing chamber. That gas first enters one of two chambers, which surrounds the bypass chamber. After passing from this chamber it is directed over two concentric wicks, which enclose a copper helix, which converts this space into long spiral outlet channel. Wicks assure maximum contact between gas and agent. The vapour laden gas leaves via the second chamber surrounding bypass chamber to outlet
   - The remaining fresh gas flows to bypass chamber, which contains the temperature sensitive element
   - No spillage after tilting or inversion.

Evaluation and use

1. Fluctuating back pressures can increase the concentration by altering the flow distribution within the vapourizer
2. Greatest effects are observed at:
   a. Low flow rates
   b. Low dial setting
   c. Large and frequent pressure fluctuation
3. Output decreases slightly when nitrous oxide is used as a carrier gas
4. Vapourizer is filled and used in upright position—deviations do not affect the output or safety, but can give a misleading impression of the amount of agent in the vapourizer.

Care and cleaning

1. Annual servicing
2. When liquid level is low, drain it and discard it
3. Every two weeks, vapourizer is drained.

Problems with TEC IV

1. Difficulty in operation one-handed
2. Yearly service interval.

TEC V

Models

- Isotec – 5
- Fluotec –5
- Enfluratec – 5
- Sevotec – 5.

Construction

1. At the top is the control dial
2. Release button at rear of dial is pushed in before vapourizer is turned on
3. Rear end is locking lever connected to the dial so that vapourizer cannot be turned on until it is locked
4. Filling devices—Keyed filling system and screw cap.

Differences

1. Helical intermittent positive pressure assembly to minimize effects of positive pressure ventilation
2. Volatile agent capacity increased from 125 mL to 300 mL.
3. One-handed dial control and more obvious 'off' position.

Evaluation

1. Accuracy is maximum at flow of 5 L/min with dial setting < 3%
2. Decreased output with higher flows with higher setting
3. TEC V is more prone for increasing vapour output due to pumping effect, than TEC IV
4. Carrier gas composition affects the output. If air or nitrous oxide is used, the vapourizer output is lower than the setting at low flows, and higher than the setting at high flows
5. Reverse flow through the vapourizer increases output.

Maintenance

1. Every 2 weeks or when there is low level of liquid, the vapourizer needs to be drained
2. Every 3 years, servicing by company.

Enumerate differences and improved safety features between TEC II to V

Table 44.3 Differences and improved safety features between TEC II to V

	TEC II	TEC III	TEC IV	TEC V
Flow (L/min)		0.25–15	0.25–15	0.25–15
Temperature		18–35	18–35	18–35
Capacity (mL)				
Dry wicks	150	270	135	300
Wet wicks	50	35	100	225
Concentration dial	At side	On top	On top	On top
Range (%)	Off–4%	0.5–5.0	0.5–5.0	0.5–5.0
Weight (kg)		6.3	7.2	7.0
Temperature compensation	Yes, at outlet of vaporizing chamber		Yes, in bypass chamber	
Non-spillage	No	No	Yes	Yes
Tilt	No	90°	180°	180°
Interlock	No	No	Yes	yes

Why does Desflurane need a separate kind of vapourizer?

Desflurane has certain unique properties, which preclude its use in regular vaporizers.

1. Its vapour pressure is higher than other volatile agents. At 20 degrees Celsius, the vapour pressure of desflurane is 669 mm Hg. Therefore, larger quantities of desflurane are present in the vapour form in the vapourizing chamber. Therefore, much larger quantities of fresh gas flow through the bypass chamber would be needed to dilute this vapour to clinically meaningful concentrations using a traditional mechanical bypass. For example, at 1 atm and 20 °C, 100 mL/min of gas through the vapourizing chamber picks up 735 mL of desflurane as compared to 47 mL of Halothane. To dilute this to a 1% concentration, 73 L per minute of bypass flow would be needed for desflurane as compared to about 5 L per minute for halothane. Modern Aladin vapourizer is electronically controlled and therefore a separate vapourizer is not needed for desflurane.
2. Desflurane boils at 22.8 °C. Therefore, at room temperatures more than 22.8°C, the amount of vapour formed would be limited only by the heat available from the vapourizer.
3. The MAC of desflurane is higher than that of other modern inhaled anesthetics (6 to 7%). Therefore, the amount of desflurane needed over a period of time is larger than other anesthetics. This would cause cooling of the vapourizer, and a reduction in output, which cannot be compensated by the temperature compensating mechanisms available in contemporary vaporizers. Therefore, an external heating source is needed.

Classify and describe TEC VI vaporizer

The TEC VI vapourizer for desflurane is classified as:

- Concentration calibrated
- Thermocompensated (electrically supplied heat)
- Agent-specific
- Out-of circuit
- Plenum
- Gas-vapour blender.

The vapourizer has 2 independent circuits arranged in parallel. Fresh gas flows through one circuit, through a fixed resistor and emerges at the fresh gas outlet. Desflurane vapour flows through the other circuit through a pressure regulating valve. There is a pressure transducer between these 2 circuits which transmits the pressure difference to a control electronic system. This regulates the pressure regulating valve so that the pressure in the 2 circuits, is equalled (known as the working pressure) even if the fresh gas flow is changed. A distal variable resistor in the vapour circuit allows concentration of gas to be adjusted by the operator. The vapour mixes with the fresh gas at the outlet of the vapourizer.

The TEC VI vapourizer has a concentration dial at the top, which is calibrated from 1–18% (1% graduation till 10% and 2% till 18%). The dial release is at back and is depressed to start the vapourizer, and when concentrations higher than 12% are needed. There are separate filling and draining mechanisms. There is a front display panel for visual indicators for vapourizer function. This consists of a light-emitting diode. The various color-coded alarms are:

Amber—without audible alarm—warmup, along with audible alarm—low agent level below 50 mL

Green—operational status

Red—no output—due to:

- Low agent level less than 20 mL
- Power failure
- Malfunction
- Tilted vapourizer beyond 10 degrees
- A liquid crystal display (LCD) indicates the amount of liquid in the vapourizer
  - Once the vapourizer is connected to an electrical source, the vapourizer sump gets heated to 39°C. During this warm-up phase, the concentration dial is

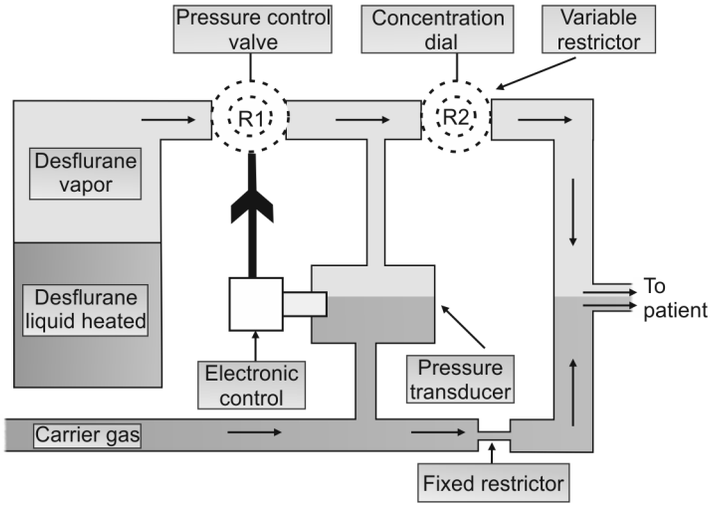


Fig. 44.5 Working of desflurane vaporizer

in standby mode and the vapourizer cannot be used. Once the proper temperature is achieved, the green light comes on indicating readiness for operation

- The TEC VI vapourizer cannot be classified as a measured flow or a variable-bypass vapourizer since it does not follow traditional methods of vaporization. It is best classified as a gas-vapour blender. Some authors have described it as an injector, though this term is ideally used for the technique of injection of liquid agent into the fresh gas flow.

Maintenance: Servicing every year.

Describe some of the newer vaporizers

Aladin cassette

- Available for all 5 agents
- Two parts
  - Cassette: Magnetic code which is agent specific
  - Anesthesia delivery unit: Part of the workstation – contains the software to recognize the agent
- Gas flow split into 2 parts
- Concentration of agent regulated by adjusting gas flow through cassette.

Concentration calibrated injector—Siemens "Kion"

- A calibrated throttle valve is opened or closed by the user.
- The more it is closed, the greater the pressure exerted by the fresh gas flow on the surface of the liquid anesthetic.
- This pressure tends to force liquid to atomize at the injector nozzle.
- The liquid droplets vapourize in the flowing fresh gas stream.
- No thermal compensation is required.

TEC VII

- Similar to TEC V
- "Easy-Fil" filler mechanism
- New ergonomics and design
- Planned factory service free
- Simplified filler mechanism
- Soldered sump assembly eliminating seals
- Improved sight glass design
- Clear agent color identification.

Describe the use of vaporizers in hyperbaric and hypobaric conditions.

Vaporizers are calibrated at normal atmospheric pressures (760 mm Hg). In the case of variable-bypass vaporizers, consider the example used in.

At an atmospheric pressure of 500 mm Hg, saturated vapour pressure of halothane would be 243 mm Hg. Therefore, halothane vapour in the vapourizing chamber would contribute to 243/500 = 48% volume and the remaining 52% volume will be constituted by carrier gas. From the previous example, we have seen that the splitting ratio for 1% halothane at a fresh gas flow rate of 5 L is 106:4844. Therefore, 106 mL of carrier gas would enter the vapourizing chamber. This will account for 52% volume and will pick up 106/52 × 48 = 98 mL of halothane vapour

Therefore, the gas exiting the vapourizing chamber would be 106 mL of carrier gas + 98 mL of halothane vapour. This would combine with 4844 mL of bypass flow. The final concentration of halothane would be 98 ml in a total volume of 98 + 106 + 4844 = 98/5048 = 1.9%

This is almost twice the 1% concentration achieved at atmospheric pressure

The partial pressure of halothane will be 1.9/100 × 500 = 9.5 mm Hg. This is only slightly higher than the partial pressure at atmospheric pressure.

Therefore, at hypobaric conditions, variable bypass vaporizers deliver a higher concentration in volumes percent, but the partial pressure increases only slightly. Since the anesthetic properties of a gas are determined by its partial pressure, there is no significant clinical implication.

Similarly, in hyperbaric conditions, delivered concentration decreases significantly but partial pressure reduces only slightly.

The new concentration (c′) achieved at altered ambient pressure (p′) can be given by the formula

$$c' = c \times p/p'$$

where c is the concentration set on the dial and p is the sea-level pressure

The TEC VI vapourizer works on the principle of differential pressure transduction. Therefore, when ambient pressures decrease or increase, the volume percent delivered will remain constant; however, this volume percent now represents a lower or higher partial pressure respectively, because of the change in ambient pressure. Hence, there will be a change in clinical effect.

What is the effect of change in carrier gas on vapourizer output?

Vaporizers are calibrated with 100% oxygen as the carrier gas.

When the carrier gas is switched from 100% oxygen to 100% nitrous oxide, there is a transient fall in vapourizer output, followed by an increase to a new steady-state level. This transient decrease is due to increased solubility of nitrous oxide in the volatile agent, leading to a reduction in the amount of gas leaving the vapourizing chamber.

What should the arrangement of vaporizers on the manifold be?

Old anesthesia machines had up to three variable-bypass vaporizers arranged in series such that fresh gas from the flow meters passed through each vapourizer to reach the common gas outlet of the anesthesia machine.

Without the use of an interlock, it was possible to turn on all vaporizers simultaneously. This could lead to

- Overdose of anaesthetic
- Vapour from one vapourizer could condense in the downstream vapourizer leading to contamination.

To prevent this, various techniques of arranging vaporizers have been described:

- Vaporizers of agents with lower boiling points should be upstream and those with higher boiling points should be downstream to prevent condensation.
- Vaporizers with less potent agents upstream and more potent agents downstream, so that even if there is contamination, it does not have clinical implications
- Difficult-to-clean vaporizers should be placed upstream.
- Agents with toxic by-products (like trilene) should be placed downstream.

Modern vaporizers are equipped with interlocking devices, which allow flow of gas through only one vapourizer at a time, and prevent simultaneous operation of more than one vapourizer.

Suggested Reading

1. Davis PD, Parbrook GD, Kenny GNC. Basic physics and measurement in anaesthesia. 4th ed. Publishers Butterworth-Heinemann. 1995.
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45 Anesthesia Breathing Systems

S Myatra, P Jain

How do you define breathing system?

A breathing system is an assembly of components which connects the patient's airway to the anesthetic machine creating an artificial atmosphere, into and from which the patient breathes.

What are the requirements of a breathing system?

Essential

1. Deliver the gases from the machine to the alveoli in the same concentration as set.
2. Delivery of gases in the shortest possible time.
3. Effective elimination of carbon dioxide.
4. Minimal apparatus dead space.
5. Low resistance.

Desirable

1. Efficient for spontaneous as well as controlled ventilation.
2. Adaptability for use in adults and children.
3. Economy of fresh gas.
4. Conservation of heat.
5. Adequate humidification of inspired gas.
6. Light weight and convenient to use.
7. Provision to reduce theater pollution.

What are the common components of a breathing system?

1. A delivery tube through which the fresh gases are delivered from the machine to the system and a fresh gas entry port.
2. Breathing tubes.
3. Reservoir bag.
4. Pressure relief valve.
5. Sleeves, connectors, adaptors.
6. CO_2 absorber (only if rebreathing is to be allowed).
7. Vigilance aids (e.g. FiO_2 sensor), humidification system may be present.

What is the function of the reservoir bag? How does it function as a reservoir?

1. Serves as a reservoir of gases during exhalation, which is available for next inspiration.
2. Reservoir of gases to provide Peak Inspiratory Flow during spontaneous breathing.
3. Permits rebreathing and allows greater economy of anesthetic gases and prevents air dilution.
4. To give a manual assisted or controlled breath.
5. Visual and tactile assessment rate and depth of respiration during spontaneous breathing.
6. Tactile assessment of resistance and compliance during manual assisted breathing.
7. Buffer for an increase in airway pressure in the system.
8. To provide a manual "sigh".

What are adjustable pressure limiting (APL) valves? Describe the Heidbrink valve?

- These are expiratory valves, which provide control of pressure in the breathing system.
- The purpose of this valve is to allow the escape of exhaled air and surplus gases from the breathing circuit, but without permitting entry of air from outside during a negative pressure in the system.
- Usually it is desirable that the pressure required to open the valve must be as low as possible in order to minimize resistance to expiration. It must however present sufficient resistance to prevent the reservoir bag from emptying spontaneously.

Heidbrink Valve: This is a commonly used expiratory valve. The components are shown in figure 45.1:

1. Female taper
2. Retaining screw
3. Disk

4. Spring
5. Valve top

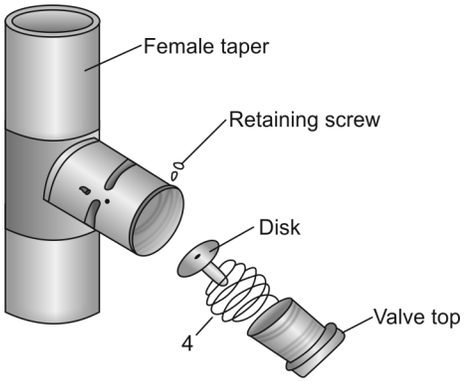


Fig. 45.1 Heidbrink valve

The body of the Heidbrink valve has a female taper with a retaining screw. The valve disk is very light and rests on a knife edge seating, which presents a small area of contact. This reduces the adhesion between the disk and the seating due to the surface tension of the condensed water from expired air. The disk has a stem which is located in a guide in order to ensure correct positioning on the seating and a coiled spring of light weight which promotes closure of the valve. The spring is a delicate coil and is of such dimensions that when the valve top is fully screwed "open" there is minimal pressure on the valve when seated. However, during the "blow off" phase the disk rises and shortens the spring so that the pressure it exerts on the disk is greater and will close it at the appropriate time. Screwing down the valve top produces progressively increasing tension in the spring. When the top is fully screwed down, the valve is completely closed. This metal valve is constructed so that it is easy to dismantle; to be cleaned and sterilized.

What are the ideal characteristics of tubing used in a breathing circuit? Describe and compare the corrugated rubber tube with the plastic disposable tubing.

Ideal characteristics of tubing used in a breathing circuit:

1. The tubing in a breathing system must be of such diameter as to present a low resistance to gas flow.
2. Its cross section must be smooth and uniform in order to promote laminar flow where possible.
3. It should be flexible and should not kink.
4. It should drape easily so that a loop may hang between the patient and the apparatus, which can trap moisture and prevent it from going back to the apparatus.
5. Transparency is desirable.

The corrugated rubber tubing is 22 mm in diameter and 110 cm in length. When compared with the plastic corrugated tubing, the advantage of the corrugated rubber tubing is that its ends are more easily stretched so it makes a better union with the other components of different diameters. It is also reusable. The disadvantage is that it is not transparent, and it is heavier than the plastic tubing. Its irregular walls cause much turbulence and it may harbor dirt and infection. Recently available silicon tubes have combined advantage of plastic tubes (transparent with smooth inner walls) and corrugated rubber tubes (non kinkable with easily stretchable ends) and are available in various sizes of 110, 130, 150 cms.

What is the significance of setting the fresh gas flow (FGF) and how is it set for the effective elimination of CO_2 in the breathing system with bi-directional flow?

Significance of setting the fresh gas flow (FGF)

- This is a very essential component of the breathing system with bi-directional flow. If there is no FGF the patient will suffocate.
- It is also required for the effective elimination of CO_2 and to adjust the amount of rebreathing.
- If the FGF is excessive; there will be wastage of gases.
- If a system has to deliver a set concentration of gases to the patient in the shortest time the FGF should be as near the patient as possible.

Elimination of carbon dioxide: An example to understand CO_2 elimination by the bi-directional flow systems is as follows. Normal production of carbon dioxide in a 70 kg adult is 200 mL per minute and it is eliminated through the lungs. Normal end-tidal concentration of carbon dioxide is 5%. Hence to eliminate 200 mL of carbon dioxide as a 5% gas mixture, the alveolar ventilation has to be:

$$\frac{200 \times 100}{5} = 4{,}000 \text{ mL} = 4\text{L}$$

This 4 L is the normal alveolar ventilation. Any breathing system should provide a minimum of 4 L/min of carbon dioxide free gas to the alveoli for eliminating carbon dioxide. If the alveolar ventilation becomes less than 4 L/min, it would lead to hypercarbia. If the alveoli are ventilated with 5 L/min of a gas containing 1% carbon dioxide, or 8 L/min of a gas containing 2.5% carbon dioxide, it could still eliminate 200 mL of carbon dioxide per minute from the alveoli.

What is rebreathing? What are the factor affecting rebreathing and what are the effects of rebreathing?

Rebreathing is said to occur when the expired alveolar gas containing CO_2 (and less oxygen than normal) is inspired as part of the next tidal volume. Circuits should have minimal rebreathing.

Rebreathing depends on

1. Fresh gas flow rate (when the inspired volume is more than the FGF).
2. Design of the breathing system.
3. Mode of ventilation (spontaneous or controlled).
4. Patients' respiratory pattern (tachypnea will increase rebreathing).
5. Mechanical dead space.

Effects of Rebreathing

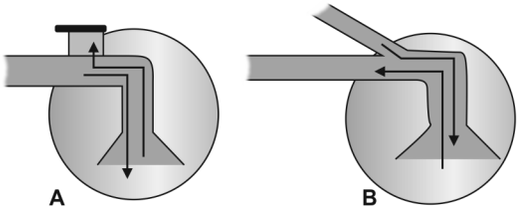
1. Retention of heat and water.
2. Alteration of inspired gas concentration.
3. Elevation of blood CO_2 levels.

What is patient and apparatus dead space?

Patient Dead Space: Patient dead space is that part of the respiratory system that is occupied by air **that** does not take part in gas exchange. In an adult, the volume of this space is 150 mL, but varies with the movement of the lungs during respiration. In a normal patient, this will mean only the anatomical dead space under ideal conditions.

- **Anatomical dead space** consists of the air passages from the lips or the nares up to, but not including the alveoli.
- **Physiological dead space** includes in addition to the anatomical dead space air that does not take part in the respiratory exchange, i.e. air in the non-perfused alveoli (dead space ventilation).

Apparatus Dead Space: This is the volume in the anesthetic equipment that is filled with exhaled gases at the end of expiration. In the non-rebreathing system this volume will be re-inhaled in the next breath and in the closed system this volume would be re-inhaled without passing through the CO_2 absorber. In other words, it is the volume of the breathing system from the patient-end to the point up to which, to and fro movement of expired gas takes place. Simplest definition of apparatus dead space is from the patient end to the point where inspired and expired gases meet and are separated. For example, apparatus dead space extends from the patient end of the circuit up to the following points (when the fresh gas flows are adequate) in the examples given in figure 45.1:



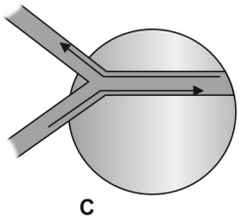


Fig. 45.2 Apparatus dead space. A. In an Afferent system- up to the expiratory valve; B. In an Efferent system - up to the Fresh gas flow; C. In a system with separate inspiratory and expiratory limbs (e.g. circle absorber) up to the point of bifurcation

Rebreathing of apparatus dead space does not contribute either to heating, humidification, but can reduce alveolar ventilation.

What are the causes of a discrepancy between the inspired and delivered concentration of gases?

- Rebreathing
- Leaks in the system leading to air dilution
- Dilution of gases because of repeated activation of flush valve
- Uptake of anesthetic agent by the system
- Release of anesthetic agent by the ststem.

What are the different ways of classifying circuits? How do you classify circuits by function?

Circuits can be classified in many ways

Dripps et al have classified them as **Open, Semi open, Semi closed and Closed,** taking into account the presence or absence of reservoir, rebreathing, CO_2 absorption and directional valves.

Table 45.1 Classification of circuits by function

Mode	Reservoir bag	Re-breathing	Example
Open	No	No	Open drop
Semi-open	Yes	No	Non-rebreathing circuit, or Circle at high FGF (> minute ventilation)
Semi-closed	Yes	Yes, partial	Circle at low FGF (< minute ventilation)
Closed	Yes	Yes, complete	Circle (with pop-off (APL) valve closed)

Table 45.2 Re-breathing and non re-breathing circuits

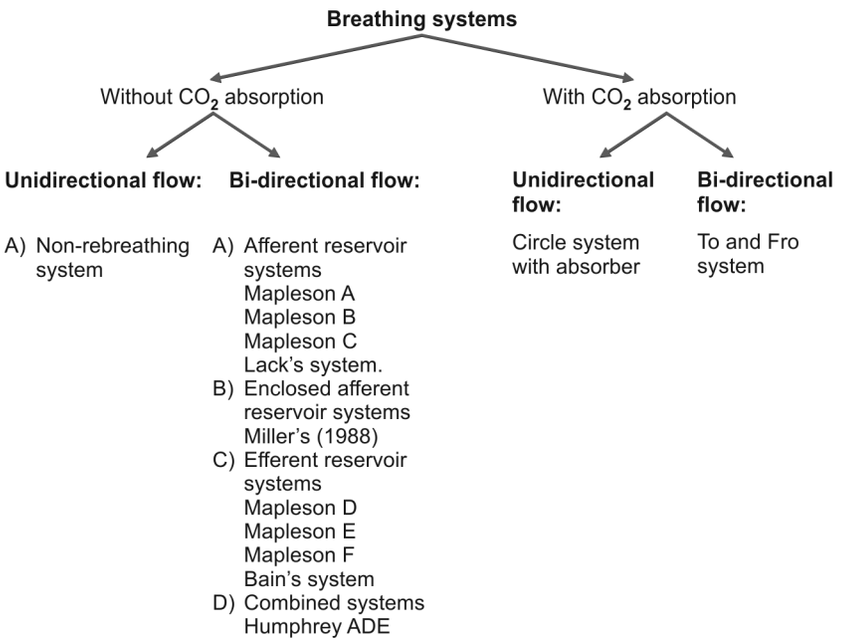
Parameters	Rebreathing	Non-rebreathing
Valve	Spill (APL)	Unidirectional
Access of expired gases to reservoir bag	Yes	No
Closed reservoir bag	+	±
Volume emerging from valve	equivalant to FGF	equivalant to MV

Breathing Systems with and without CO_2 Absorption

The ambiguity of the terminology used as open, semi-open, semi-closed and closed allowed inclusion of apparatus that are not breathing systems at all into the classification.

To overcome this problem Conway suggested that a **functional classification** be used and classified according to the method used for CO_2 elimination. Miller DM in 1988 widened the scope of this classification to include the enclosed afferent reservoir system.

Flow chart 45.1 Breathing system with and without CO_2 absorption



Draw diagrams of Mapleson's classification of semi-closed breathing systems. Which are the afferent and efferent reservoir systems? Rank the Maplelson breathing systems according to their superiority both for controlled and spontaneous ventilation.

Mapleson in 1954 classified the semi-closed anesthetic system according to the position of the expiratory valve, the fresh gas flow inlet and the position/presence of the reservoir bag. These are Mapleson's systems: A, B, C, D, E (No provision for CO_2 absorption). The Jackson Rees modification of The Ayre's T-piece is classified as a Mapleson F system although it was not included in the original description by Professor Mapleson.

Afferent reservoir systems: They have the reservoir in the afferent limb, and do not have an efferent limb: Mapleson A, Mapleson B and Mapleson C.

The afferent systems work efficiently during spontaneous breathing provided the expiratory valve and the reservoir bag and fresh gas flow are separated by one patient tidal volume; and the apparatus dead space is minimal. They are not very efficient for controlled ventilation. In Mapleson B and C, the

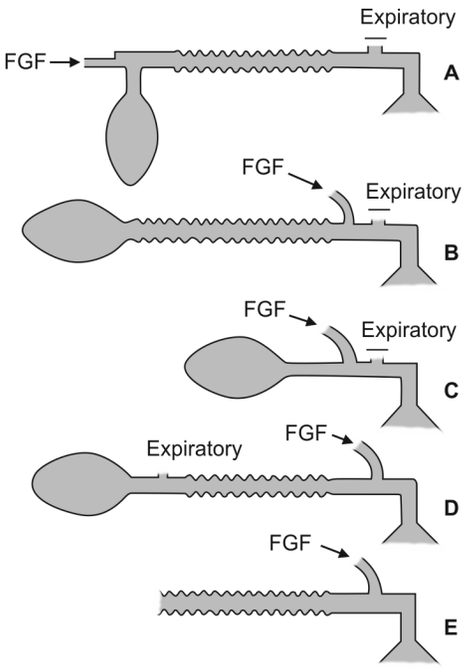


Fig. 45.3 Maplelson breathing systems

fresh gas flow is close to the expiratory valve, hence the system is inefficient both during spontaneous and controlled ventilation.

Efferent reservoir systems: They have valves or breathing bags at the expiratory end of the circuit: Mapleson D, Mapleson E and Mapleson F.

They act as T-pieces with the FGF delivered to the patient end of the circuit. These systems are all inefficient for spontaneous respiration.

At present only systems A, D, E and F and some of their modifications are used.

Ranking of Mapleson's systems based on superiority is as follows:

1. Spontaneous ventilation: A > D, F, E > C, B
2. Controlled ventilation: D, F, E > B, C > A.

What are the advantages and disadvantages of the Mapleson's breathing systems?

Advantages

1. Simple
2. Light weight
3. Not bulky, light weight and convenient to use
4. Inexpensive parts
5. Low resistance
6. Low dead space
7. All parts can be easily cleaned and sterilized.

Disadvantages

1. High fresh gas flows, hence wasteful and costly
2. Elimination of CO_2 depends on FGF so there can be re-breathing if FGF inadequate
3. Loss of heat and humidity (Except in co-axial circuits).

What are the assumptions made by Mapleson when he analyzed bi-directional flow systems?

1. Gases move en bloc. They maintain their identity as fresh gas, dead space gas and alveolar gas. There is no mixing of these gases.
2. The reservoir bag continues to fill up, without offering any resistance till it is full.
3. The expiratory valve opens as soon as the reservoir bag is full and the pressure inside the system goes above atmospheric pressure.
4. The valve remains open throughout the expiratory phase without offering any resistance to gas flow and closes at the start of the next inspiration.

Describe Mapleson A breathing system and give the functional analysis during spontaneous breathing and controlled ventilation. What are the advantages and disadvantages of this system?

The Mapleson A (Magill) system was designed by Sir Ivan Magill in the 1930's and remains an excellent system for spontaneous ventilation. Fresh gas enters the system at the fresh gas outlet of the anesthesia machine. It consists of a reservoir bag, a corrugated hose (42", volume that of one tidal volume, about 550 mL) and an expiratory valve (Heidbrink valve) which is very close to the patient to reduce the dead space.

Spontaneous breathing

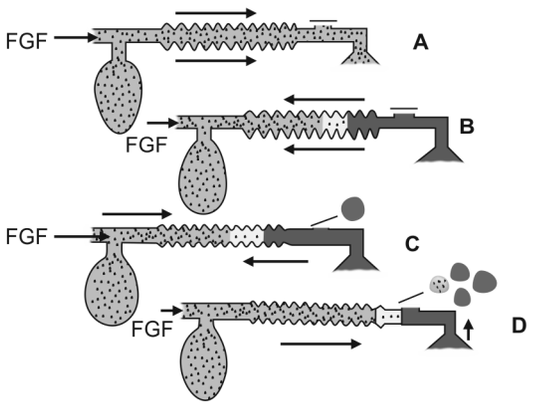


Fig. 45.4 Functional analysis during spontaneous breathing in Mapleson a Circuit

During inspiration, gas is inhaled from the 2 L reservoir (breathing) bag which partially collapses giving a visual confirmation that breathing is occurring (A). During expiration the bag and tubing are initially refilled with a combination of exhaled dead space gas (containing no carbon dioxide) and fresh gas flowing from the anesthetic machine (B). Once the bag is full the pressure within the breathing system rises and the expiratory valve near the patient opens allowing the alveolar gas (containing carbon dioxide) to be vented from the system (C). During the expiratory pause, more fresh gas enters the system driving any remaining alveolar gas back along the corrugated tubing and out through the valve (D).

If the fresh gas flow is sufficiently high all the alveolar gas is vented from the circuit before the next inspiration and no rebreathing will take place. With careful adjustment the FGF can be reduced until there is only fresh gas and dead space gas in the breathing system at the start of inspiration. When the system is functioning correctly, without any leaks, a FGF equal to the patient's minute ventilation is sufficient to prevent rebreathing. Thus, a FGF closer to the patient's

total minute ventilation (including dead space) is usually selected to provide a margin of safety. An adult's minute volume is approximately 80 mL/kg /min and thus for a 75 kg man a FGF of 6 L/min will prevent rebreathing. This is an efficient system for spontaneously breathing patients if carbon dioxide absorption is not available.

The opening pressure of the Heidbrink valve must be low to prevent expiratory resistance (0.5 cm H_2O), however, it must be higher than the collapsing pressure of the reservoir bag (0.25 cm H_2O) otherwise the system will not work during spontaneous breathing.

Controlled ventilation

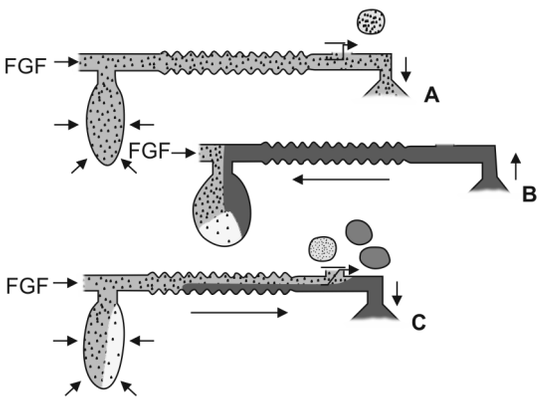


Fig. 45.5 Functional analysis during controlled ventilation in Mapleson A circuit

During controlled ventilation, the Magill circuit works in a different way and becomes wasteful and inefficient, requiring high FGF to prevent rebreathing. The inspiratory force is provided by the anesthetist squeezing the reservoir bag after partly closing the expiratory valve next to the patient. During lung inflation some of the gas is vented from the circuit and at the end of inspiration the reservoir bag is less than half full (A). During expiration, dead space and alveolar gas pass down the corrugated tubing and may reach the bag which will then contain some carbon dioxide (B). During the next inspiration when the bag is compressed, alveolar gas re-enters the patient's lungs followed by a mixture of fresh, dead space and alveolar gas (C).

A FGF of two and a half times the patient's minute volume is required to vent enough alveolar gas to minimize rebreathing (FGF of about 12–15 L/min) which is obviously very inefficient. In practice, the Magill circuit should not be used for positive pressure ventilation except for short periods of a few minutes at a time. Prolonged use is dangerous.

Advantages

- Best among all Mapleson's systems for spontaneous breathing
- Minimal wastage of gases during spontaneous breathing. No rebreathing if FGF is about one minute volume.

Disadvantages

- Not efficient for controlled ventilation—Considerable rebreathing requires high fresh gas flow.
- Expiratory valve required-produces slight resistance during expiration.
- Expiratory valve is heavy (Heidbrink valve).
- Expiratory valve near patient end inconvenient to use especially when head and neck is draped and difficult for scavenging.
- Not suitable for paediatric use.

Enumerate the modifications of the Magill's system and describe the Lack circuit.

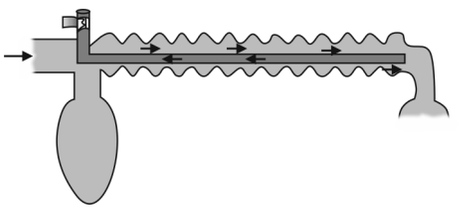


Fig. 45.6 Lack's co-axial system

Lack's Circuit

This is a simple modification of the Mapleson A circuit which made it more efficient for controlled ventilation. This is achieved by substituting a non-rebreathing valve (such as the Laerdel's valve) for the Heidbrink valve at the patient end of the circuit. Not only does this arrangement prevent rebreathing, but during manual ventilation the delivered minute volume will be the same as the desired FGF. It is, however, a dangerous arrangement for spontaneous respiration because the valve may jam if the fresh gas flow is greater that the patient's minute volume.

Lack's coaxial system: Another modification of the Magill's system originally described in 1976. A disadvantage of the Magill system is that the expiratory valve is attached close to the patient making it awkward to use. The Lack's system is a coaxial version of Mapleson A system with the expiratory valve located near the machine end towards which the exhaled gases are carried by efferent tubing placed coaxially. This also facilitates easy scavenging of expired gas. This system functions like a Mapleson A system both during spontaneous and controlled ventilation. The fresh gas flows required for both spontaneous and controlled ventilation are as described for the standard Mapleson A system. The dead space of the apparatus is from the patient end up to

the expiratory valve. The **advantages** of this modification are the easy accessibility of the expiratory valve (it is near the machine end unlike the Magill's circuit), easy to scavenge and better separation of gases before the APL valve is reached.

Testing the Lack's coaxial circuit:

1. Attach the endotracheal tube to the patient end of the circuit and blow through it after closing the valve fully. If there is a leak in the circuit, the bag will move.
2. Close both the limbs at the patient end. Open the expiratory valve and squeeze the bag. If there is a leak the bag will collapse and the gas will escape through the valve.

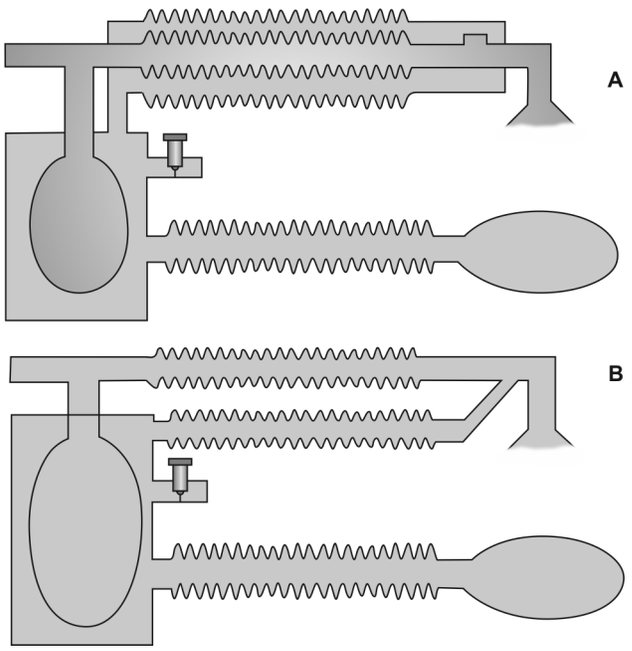


Fig. 45.7 Enclosed afferent reservoir

Enclosed Afferent Reservoir (EAR) system: This system consists of a Mapleson A system; enclosed within a non-distensible structure, and it was described by Miller and Miller in 1998. It may also be constructed by enclosing the reservoir bag alone in a bottle and connecting the expiratory port to the bottle with a corrugated tube and a one way valve (B). To the bottle is also attached a reservoir bag and a variable orifice for providing positive pressure ventilation.

This system allows for the selective elimination of alveolar gases in both spontaneous and controlled ventilation. A comparison with the Bain system in controlled ventilation demonstrated greater efficiency in eliminating carbon dioxide. A fresh gas flow of 70 mL/kg/min using an EAR system gave mild hypocarbia which equated to a FGF of 100 mL/kg/min using the Bain system. Smaller minute volumes of ventilation are required for optimal performance than with the Bain system. It is thus more efficient than the Bain system for controlled ventilation.

Describe Mapleson B and C system.

These systems are all functionally similar and also similar in construction, with the fresh gas flow entry and the expiratory valves located at the patient end of the circuit. The C system differs from B in that it does not have the corrugated tube. In order to reduce the rebreathing of alveolar gas and to improve the utilization of FG during controlled ventilation, the FG entry was shifted near the patient. However, since this allows a complete mixing of FG and expired gas these systems are not good for spontaneous or controlled ventilation. Fresh gas flows should be at least 2–3 times the minute volume to minimize rebreathing. Hence, they are not commonly used in anesthetic practice, although the C system is used on intensive care units. High flows of gases are needed to prevent rebreathing of CO_2. This system was at one time combined with a canister of sodalime to absorb CO_2 (Waters' "To and Fro" Circuit).

Describe and draw Ayre's T –piece? What is the Mapleson E system?

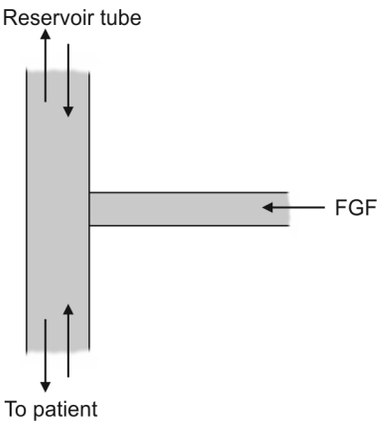


Fig. 45.8 Ayer's T-piece

This was described by Phillip Ayre in 1937. This consists of a light metal tube 1 cm in diameter, 5 cm in length with a side arm. It functions as a non-rebreathing system.

The Mapleson E is a modification of the Ayre's T piece. Fresh gas enters the system through the side arm and the expired gas is vented into the atmosphere through the expiratory limb, which also serves as a reservoir (there is no bag in this system). An expiratory limb volume greater than the patient's

tidal volume prevents entrainment of room air and thereby prevents dilution of anesthetic gases. A FGF greater than three times the minute ventilation prevents rebreathing. The dead space is minimal as it is only up to the point of FGF entry. Controlled ventilation is given by intermittently occluding the outlet with the thumb. The advantage of this system is that it has a low dead space, no valves and a low resistance, which makes it very suitable for use in children. The disadvantage of this system is that the pressure buffering effect of the bag is absent and we cannot make a visual and tactile observation of respiration. This system requires 2–3 times the minute ventilation to prevent rebreathing.

All the efferent systems are modifications of the Ayre's T–piece.

Describe the efferent reservoir systems (ER)?

The Mapleson D, E and F systems are efferent reservoir systems; they are all modifications of the Ayre's T-piece. They have a 6 mm diameter tube as the afferent limb that supplies the FGF from the machine. The efferent limb is a wide-bore corrugated tube to which the reservoir bag is attached and the expiratory valve is near the bag. In Mapleson E system, the corrugated tube acts as the reservoir. In Bain system, the afferent and efferent limbs are coaxially placed.

In an attempt to reduce FGF requirements, ER systems are constructed with reservoirs in the efferent limb. The functioning of all these systems is similar. These systems work efficiently and economically for controlled ventilation as long as the FGF entry and the expiratory valve are separated by a volume equivalent to at least one tidal volume of the patient. They are not economical during spontaneous breathing.

What is the Mapleson D system?

The Mapleson D can be described as a T piece with an expiratory limb. The fresh gas inlet is located near the patient end and the expiratory valve is present, which is close to the reservoir bag. The functional analysis is described with the Bain's circuit which is a modification of Mapleson D system.

Draw and describe the Bain's circuit. Give the functional analysis of this system during spontaneous breathing and controlled ventilation.

The Bain's circuit is the most commonly used modification of the Mapleson D system. It is a co-axial circuit, which was introduced in 1972 by Bain and Spoerel. This is often called a universal circuit as it can be used for adult and pediatric patients as well as for spontaneous and controlled ventilation. It is also the best among the Mapleson circuits for controlled ventilation. This is made of plastic tubings, which are arranged coaxially which reduces the bulk of the system. The inner

Functional Analysis

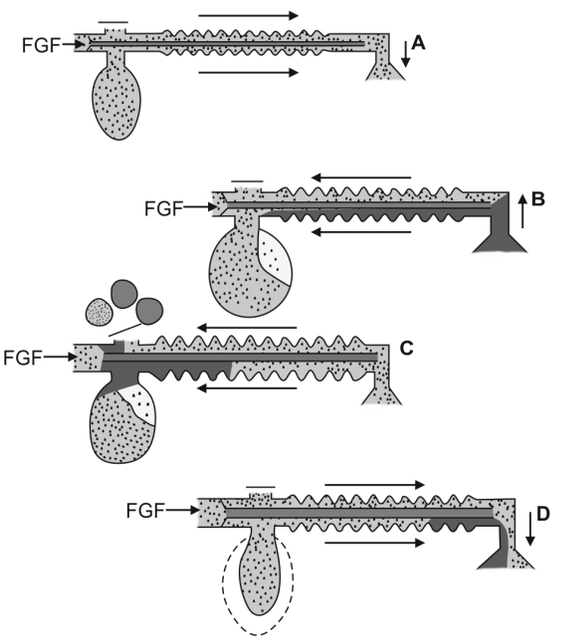


Fig. 45.9 Functional analysis of Bain's circuit during spontaneous breathing

tubing is green in color with an internal diameter of 7 mm through which the fresh gas flows to the patient and exhaled gases travel in the outer corrugated tubing with an internal diameter of 22 mm. The reservoir bag may be removed and replaced by a ventilator. It also has a pressure manometer and PEEP valve. The dead space of the circuit is the volume from the patient end up to the point of separation of the gases. If there is a leak in either tubing the entire volume of the tubing becomes the dead space.

Spontaneous respiration: Before connecting to the patient breathing system should be filled with FG. When the patient inspires, the FG from the machine, the reservoir bag and the corrugated tube flow to the patient (A). During expiration, there is a continuous FGF into the system at the patient end. The expired gas gets mixed with the FG as it flows back into the corrugated tube and the reservoir bag (B). Once the system is full, the excess gas is vented through the valve situated at the end of the corrugated tube near the reservoir bag. During the expiratory pause the FG continues to flow and fill the proximal portion of the corrugated tube while the mixed gas is vented through the valve (C). During the next inspiration, the patient breaths FG as well as the mixed gas from the corrugated tube (D). The composition of the inspired mixture is determined by several factors. They are FGF, respiratory rate, tidal volume, end expiratory pause and CO_2 production in the body. Factors other than FGF cannot be manipulated in a spontaneously breathing patient. It has

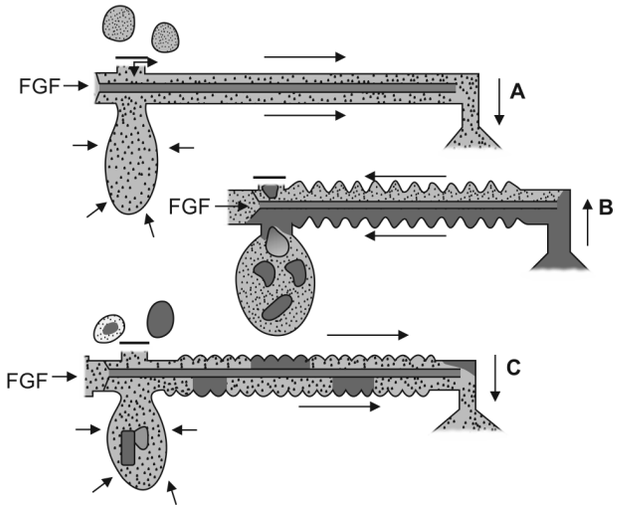


Fig. 45.10 Functional analysis of Bain's circuit during controlled ventilation

been shown that the FGF should be at least 1.5 to 2 times the patient's minute ventilation in order to minimize rebreathing.

Controlled ventilation: For positive pressure ventilation, the expiratory valve has to be partly closed so that it will open only after sufficient pressure has developed in the system. When the system is filled with fresh gas, the patient gets ventilated with the FGF from the machine, the corrugated tube and the reservoir bag (A). During expiration, the expired gas gets mixed with the fresh gas that is flowing into the system at the patient end. During the expiratory pause the FG will continue to enter the system and pushes the mixed gas towards the reservoir (B). When the next inspiration is initiated, the patient gets ventilated with the gas in the corrugated tube, i.e. a mixture of FG, alveolar gas and dead space gas (C). When the pressure in the system increases, the expiratory valve opens and the contents of the reservoir bag are released. This system functions more efficiently when used for controlled ventilation. The degree of rebreathing that occurs depends on the FGF. This system causes less rebreathing that Mapleson B and C.

Flow settings

- Controlled ventilation: 2 L/min < 10 kg 3.5 L/min = 10–50 kg 70 mL/kg > 60 kg
- Spontaneous ventilation: 200–300 mL/kg

What are the advantages and disadvantages of Bain's circuit? What are the tests that can be performed to check the Bain's circuit?

Advantages

- Universal circuit: Can be used for adult and pediatric patient. Spontaneous and controlled ventilation
- Best Mapleson system for controlled ventilation
- Light weight and coaxial arrangement makes it convenient to use
- Long length of the circuit makes it easy to be away from the patient, useful during MRI
- Disposable circuit, however can be easily sterilized and reused
- Warmth added to the inhaled gases by exhaled gas passing through the outer tubing.

Disadvantages: Due to the coaxial nature of the circuit there may be disconnection or kinking of inner tubing or a leak may get missed. If this occurs, the entire corrugated tubing becomes dead space. This can result in hypercarbia from inadequate gas flow or increased respiratory resistance

Tests: A careful examination of the Bain's circuit is essential. Special tests to check this circuit are given below:

1. The Pethick test**:** Flush high flow oxygen into the circuit and occlude the patient's end of the circuit until the reservoir bag is filled. The patient end is then opened and the circuit flushed with oxygen. If the inner tube is intact, the venturi effect occurs at the patient end, causing decrease in pressure within the circuit and the bag will deflate. If there is a leak in the inner tube FG will escape into the expiratory limb and the bag will inflate.
2. Block the inner tube at the patient end and flush the circuit. The flow meter bobbins will dip due to the backpressure if the inner tube is patent and there is no leak in the inner tube.

What are coaxial circuits? Describe the Penlon co-axial circuit?

Coaxial circuits have two tubings one inner and one outer tubing (arranged coaxially). This makes the system less bulky and convenient to use and also helps to warm the inspired gases**.**

Examples of coaxial circuits are**:**

- Lack's circuit (described earlier)
- Bain's circuit (described earlier)
- Penlon circuit

Penlon Circuit:

It is a modification of the Bain's circuit. Here the inner tube is made of antistatic rubber and the outer tube is transparent. The circuit is directly connected with the outlet of the anesthetic machine by means of a coaxial circuit valve. Fresh gas inflow occurs through this valve unit into a narrow metal tube the end of which connects the inner tube of the circuit. This valve unit thus directs the FGF, forms an expiratory valve

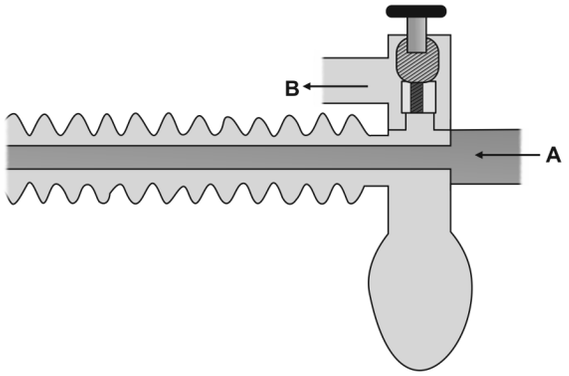


Fig. 45.11 Penlon circuit

for exhaled gases and an attachment for the reservoir bag. The resistance in this system (at a flow rate of 30 L/min) is less than 0.5 cm H_20.

Draw and describe the Jackson–Rees circuit in detail? What are its advantages and disadvantages?

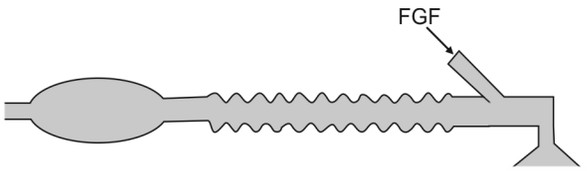


Fig. 45.12 Jackson-Rees circuit

Jackson-Rees modification of the Ayre's T-piece: This is classified as a Mapleson F system although it was not included in the original description by Professor Mapleson. An open ended reservoir bag is fitted to the expiratory limb of the Ayre's T-piece. This reservoir bag incorporates a relief mechanism for venting exhaled gases. The relief mechanism is either an adjustable valve at the distal end of the bag or a fenestration at the side of the bag. The internal volume of the tube between the patient and the bag should exceed the patient's tidal volume. Movement of the bag can be seen during spontaneous breathing, and the bag can be compressed to provide manual ventilation. It is light in weight; has low resistance and no valve which makes it suitable for use in children. As in the Bain's circuit, the bag may be replaced by a mechanical ventilator designed for use with children. This system is suitable for children under 20 kg. During spontaneous ventilation, exhaled gases pass down the expiratory limb and mix with the fresh gas. The expiratory pause allows the fresh gas to push the exhaled gas down the expiratory limb, including the reservoir bag. Fresh gas flows of 2–3 times minute volume should be used to prevent rebreathing during spontaneous ventilation, with a minimum flow of 3 L/min, e.g. a 4-year-old child weighing 20 kg has a normal minute volume of 3 L/min and would require a FGF of 6–9 L/min.

During controlled ventilation in children, normocapnia can be maintained with a fresh gas flow of 1L + 100 mL/kg/min; e.g. a 4-year-old weighing 20 kg would need a total FGF of around 3 L/min. It can be used in adult patients with controlled ventilation using FGF ranging from 70–100 mL/min per kg body weight.

Advantages

- Simple, easy to assemble
- Light weight
- Portable
- No valves
- Least resistance
- Suitable for pediatric anesthesia, especially head and neck surgery (due to the above factors)
- Easy to scavenge
- Inexpensive
- Equally effective for both controlled and spontaneous ventilation.

Disadvantages

- Wastage of gases—FGF 3 times minute volume required
- Lack's humidification (can be overcome by allowing FG to pass through a humidifier)
- Occlusion of the relief valve can increase airway pressure producing barotrauma.

Classify and Describe Water's to and fro canister? What are the advantages and disadvantages of this?

Fig. 45.13 Water's to and fro canister

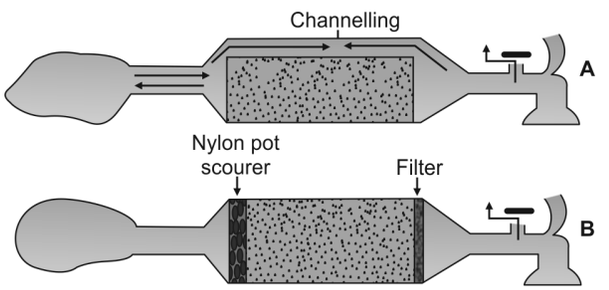


Fig. 45.14 Schematic diagram of Water's to and fro canister

This is a breathing system with CO_2 absorption with bidirectional flow. Here the patient breathes in and out of a closed bag, which is connected to the face mask or an endotracheal tube. The dead space is the part between the patient and the soda lime and hence this volume must be minimized, which means the soda lime canister must be close to the patients head and this lead to mechanical problems. A length of tubing can be placed between the canister and the bag without detriment. The fresh gases enter the system at the patient end and the expiatory valve is between this point and the canister.

The canister is usually placed horizontally for convenience and it is important that it is well-packed with soda lime, since if there is a void above the soda lime "channeling" (gas takes the path of least resistance) would occur. The soda lime may be conveniently compressed to prevent gaps by the insertion of a nylon pot scourer at one end. When the canister is closed the sealing washer should be checked to ensure that it is in the correct position. Any soda lime on the threads or the washer should be removed. The system should be checked for leaks. Cotton wool filters may be used at the patient end of the canister to prevent inhalation of soda lime.

Advantages

- Portable
- Easy to sterilize after an infected case (if made of metal –autoclave, if made of Perspex-chemical sterilization).

Disadvantages

- Cumbersome
- Risk of patient inhaling soda lime
- Danger of accidental extubation due to the weight of the canister and proximity to the patient
- Expiratory valve very close to patient end, difficult to use when head and neck are draped.

What are combined breathing systems? Describe in brief Humphrey's ADE system.

Combined breathing systems are those in which more than one Mapleson's systems are combined and used. This helps to overcome the difficulties of changing the breathing systems for different modes of ventilation.

Humphrey's ADE System

The Mapleson A circuit is inefficient for controlled ventilation as is the Mapleson D circuit for spontaneous ventilation. David Humphrey has designed a system called Humphrey ADE, with two reservoirs, one in the afferent limb and the other in the efferent limb. This consists of a single circuit that can be changed from a Mapleson A system to a Mapleson D by moving a lever on the metallic block, which connects the circuit to the fresh gas outlet on the anesthetic machine, while in use, only one reservoir will be in operation. The reservoir bag is situated at the fresh gas inlet end of the circuit, and gas is conducted to and from the patient down the inspiratory and expiratory limbs of the circuit. It can be used for adults as well as children. The functional analysis is the same as Mapleson A in afferent system mode and as Bain in efferent system mode. It is not yet widely used.

Depending on the position of the control lever at the Humphrey block, gases either pass through the expiratory valve or the ventilator port. When the lever is "up" the reservoir bag and the expiratory valve are used, creating a Mapleson A type circuit. When the lever is in the "down" position the bag and valve are bypassed and the ventilator port is opened creating a Mapleson D system for controlled ventilation. If no ventilator is attached and the port is left open the system will function like an Ayer's T-piece (Mapleson E).

It is essential that the anesthetist fully understands the function of a particular circuit. If the lever on the Humphrey block is moved from "up" to "down" while gases are flowing the breathing bag will remain full of gas but manual ventilation of the patient's lungs by compressing the bag will be impossible and may resemble complete obstruction of the breathing circuit. This has led to anesthetists occasionally concluding that their endotracheal tube required changing.

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46 Endotracheal Tubes, Double Lumen Tubes and Combitube

A Kulkarni, A Chatterjee

What is an endotracheal tube (ETT)?

An endotracheal tube is a device used to secure a patient's airway. It can be inserted via the oral or nasal route and is shaped accordingly.

What is the material of the ETT? How is it standardized?

The ideal material should have following characteristics:

1. Low cost.
2. Non-toxic and non-irritant to tissues.
3. Transparent so that exhaled air mist can be seen through it.
4. In case of non-disposable tubes should be easily sterilized and repeated sterilizations should not affect their durability.
5. Non-flammable.
6. Inside and outside surface of the tube should be smooth and non-wettable to prevent secretion build up, allow easy passage of suction catheter or bronchoscope and prevent trauma to oral/nasal structures during passage.
7. Thin walled with sufficient strength to prevent occlusion of lumen from extrinsic compression and kinking.
8. Thermoplasticity to conform to patients anatomy when in place.
9. Non reactivity with lubricants and anesthetic agents.
10. Latex free.

Endotracheal tubes are made of red rubber or polyvinyl-chloride (PVC). Other materials such as silicon rubber (polymethylsiloxane) or teflon has also been used.

Red rubber endotracheal tube

Advantages

- Easily cleaned, sterilized and reused.

Disadvantages

1. Becomes floppy and sticky with repeated use.
2. They are not transparent, hence secretions, blood cannot be seen.
3. Can easily kink and get clogged by secretions.
4. They do not soften at body temperature.
5. Risk of latex allergy (Red rubber tubes are coated with latex).
6. Thicker wall (in comparison to a PVC tube of the same size).

Polyvinylchloride (PVC): The routinely used endotracheal tubes.

Advantages

1. Disposable, inexpensive and have a lower tendency to kink as compared with red rubber tubes.
2. Nonirritant, compatible with tissues and cause less tissue reaction particularly with long-term intubation.
3. Stiffer, therefore facilitates intubation but soften at body temperature and conforms to patient's upper airway, reducing pressure at the point of contact.
4. Smooth non-wettable surface facilitates easy passage of a suction catheter or a bronchoscope.
5. Transparent.
6. Thin wall: Therefore a wider lumen and thus a reduction in airway resistance for the same size tube as compared to a red rubber endotracheal tube.

Disadvantages

Cannot be used in laser surgeries, as PVC tubes can ignite and cause airway fires.

Tissue reactivity of ETT

The material used for construction of the endotracheal tubes is tested to ensure that it is non-irritant. It must pass the American Society for Testing and Materials Standards

(ASTM) or American National Standards Institute (ANSI) testing. Tissue implantation test is carried out. The material is implanted in the paravertebral muscle of anesthetized rabbits under sterile conditions and the sites observed for any sign of inflammation. Tubes which pass the test have F-29, Z-79 or ASTM written on them. A CE mark on the tube also indicates compliance with the requirements of the medical devices directive.

Describe the design of the standard PVC endotracheal tube. What are the markings on the ETT?

Design of a PVC ETT

It is a transparent tube with a preformed curve and made of polyvinylchloride. It has 2 ends; a patient end (enters patients trachea) and a machine end, (which gets attached to the breathing circuit.). The tube may be shortened at this end if required. A typical ETT is shaped like an arc of a circle with a radius of curvature of 140 ± 20 mm.

The patient end is cut obliquely and is called the bevel. The angle that the bevel makes with the longitudinal axis of the tube is called the angle of the bevel and it is about 38 ± 10°. The bevel faces to the left and facilitates the visualization of the cords as the tube is being inserted from the right side of the mouth. On the opposite side of the bevel is a hole in the wall of the tube, called a Murphy eye. It provides an alternate pathway for gas flow should the bevel get occluded. The tracheal tube standard specifies that the area of a Murphy eye must not be less than 80% of the cross sectional area of the tube lumen.

Tracheal tubes which lack the Murphy eye are known as Magill tipped endotracheal tubes (PVC tubes). The tip of the tube is smooth and atraumatic. As there is no Murphy eye, the cuff is placed closer to the tip of the tube, decreasing the risk of inadvertent endobronchial intubation.

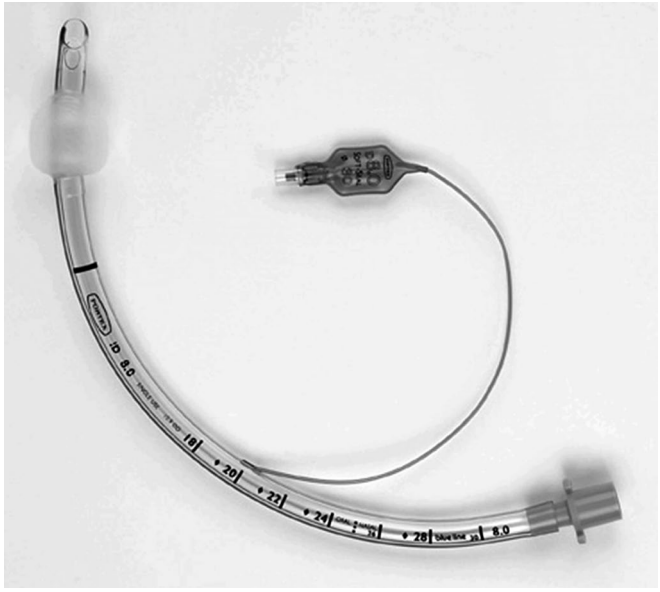

Fig. 46.1 PVC ETT

There is a radiopaque line along the entire length of the tube which helps to determine the correct position of the tube on an X-ray if required. The length of the tube has transverse black markings at each centimeter along the outer curvature which indicates the distance from the tip of the tube. A black transverse depth marker, which assists proper placement of the tube is present on certain tubes approximately 3 cm proximal to the cuff in size 6 and above. (note that this distance will vary according to manufacture) For pediatric cuffed tubes, the depth marking from tip of tube varies according to size as given in table.

Table 46.1 ETT marking in pediatric ETTs

Internal diameter (mm)	Distance of depth marking from tube tip (mm)	Cuff free subglottic tube length (mm)
3	24	9
3.5	27	10
4	30	12
4.5	34	12
5	39	16
5.5	45	16

The tubes are also marked with their internal diameter and outer diameter in mm, whether for oral or nasal use (red rubber tube) and the implantation test number. Note such as 'for single use only' also may be written on the tube. Endotracheal tubes have a uniform wall thickness. The machine end is circular and is fitted with a standard 15 mm universal connector which is compatible with standard circuits.

Describe the cuff system of the ETT and type of the cuff.

Cuff system of an ETT consists of:

1. *Cuff*
   The cuff is an inflatable sleeve near the patient end of the tube. ASTM/ISO standards—specify the maximum distance of the cuff from the tip of tube (varies with tube size as shown in table 46.1). Bonded edge of the cuff does not encroach on the Murphy's eye.
2. *Inflating system*
   Includes an internal (built in the wall of the tube) inflation tube, an external inflation tube, a pilot balloon (located near the midpoint of the inflation tube or adjacent to the inflation valve) and a one way valve. (Cuff on red rubber tubes has a cap and not a one way valve) The ASTM/ISOC standard for external inflation tube requires that its external diameter does not exceed 2.5 mm and it to be

attached to the tube at a small angle. The cuff material should be, thin, soft but strong and resistant to tearing.

Advantages of a cuffed tube

1. It prevents aspiration by sealing off the trachea from the oral cavity and gastric contents.
2. It avoids gastric distension during IPPV.
3. Allows efficient ventilation.

Overinflation of the cuff leads to a high pressure within the cuff which gets transmitted to the tracheal mucosa. The pressure on the lateral tracheal wall measured at end expiration should be approximately 20–30 cm of H_2O (15–22 mm Hg) in a normotensive adult. This could worsen during the periods of systemic hypotension thus, overfilling of the cuff is not recommended.

The increase in pressure depends on the cuff compliance. To avoid this problem:

- The cuff can be filled with either the gas mixture identical to that used for the anesthesia or with sterile saline
- The cuff should be inflated very gradually till the leak just disappears
- The intracuff pressure should be monitored at frequent intervals during anesthesia.

After prolonged intubation as in an intensive care setting, there can be mucosal sloughing and ulceration at the point of contact of the cuff with the tracheal mucosa and intracuff pressures should therefore be monitored regularly.

Nitrousoxide can diffuse into the cuff filled with air during an anesthetic and with time increase the volume of the cuff and the pressure exerted on the tracheal mucosa. The rate of diffusion depends on:

- The permeability of the cuff material
- The surface area of cuff exposed to N_2O
- The partial pressure of N_2O.

Types of Cuffs

1. *High volume low pressure:* As in PVC tubes. This cuff has a large resting volume and area of contact with the tracheal wall hence, the pressure is distributed over a large area rather than at a specific point.

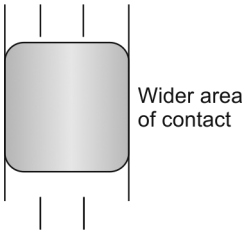

Fig. 46.2: Volume low pressure cuff

Advantages

- Exert minimal pressure on the tracheal wall.

Disadvantages

- Cuffs are made of a thin material and could be torn/damaged during intubation especially during a nasal intubation or if held with a Magill's forceps.
- The cuff may not be fully unfolded when seal is achieved; leaving some folds that may allow aspiration.

2. Low volume high pressure: As in red rubber or flexometallic/reinforced tubes.

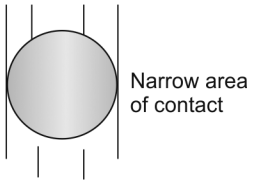

Fig. 46.3: Low volume high pressure cuff

Advantage: It gives better protection against aspiration of secretions into the lungs.

Disadvantage: Exert a high pressure on the tracheal wall and if left in-situ for a prolonged period may lead to necrosis of the tracheal mucosa.

3. *Foam filled cuffs:* They are soft, low pressure cuff. Self inflating polyurethane foam material replaces the air that is used to inflate the cuff. The cuff is actively deflated so as to collapse the foam prior to insertion. After intubation the pilot tube is opened to atmosphere which allows the foam to expand and fill the cuff. Since the pilot tube is left open, diffusion of N_2O does not further increase the cuff pressure.

Pilot balloon is situated near the inflating valve and gives an idea of the state of inflation and deflation of the cuff. (i.e. act as a pressure volume indicator)

Inflation valve is designed to fit a nozzle of the standard syringe. It is a one way valve which prevents the escape of air once inflated.

Enumerate the indications and contraindications of tracheal intubation?

Indications

1. *For surgery:* Trachea is intubated to administer general anesthesia.
   - To secure and maintain a free airway
   - To protect against aspiration of gastric/oral contents.
   - To provide positive pressure ventilation

2. *Nonsurgical conditions:* As a life-saving measure.
   - CPR
   - In an unconscious or semiconscious patient who is unable to protect his/her own airway. for example drug overdose, poisoning, CNS disease, head injury, polytrauma and patient with impaired laryngeal reflexes
   - When patient's own respiratory attempts/ drive is inadequate and need mechanical ventilation
   - Respiratory obstruction
   - Tracheobronchial toileting in severe sputum retention.

Contraindications: These are no contraindications to intubation. Intubation in certain scenarios can be tricky. Appropriate precautions should be taken when dealing with such patients. The difficult scenarios include:

- Severe airway trauma with distortion of upper airway, edema and bleeding makes endotracheal intubation difficult or impossible. Alternate mode of securing the airway is needed.
- *Laryngeal edema and epiglotitis:* intubation with a smaller tube can be life-saving.
- *Aneurysm of the arch of Aorta:* Longstanding aneurysm can erode into the tracheal wall and during intubation it can burst causing exsanguination.
- *Cervical spine injury:* There is a possibility of converting an incomplete cervical spine fracture into a complete one with resultant spinal cord damage. Intubation can be achieved with manual in-line stabilization on a spine board with absolutely no neck movement
- Traumatic laryngeal fracture.

Describe the procedure of endotracheal intubation

- Confirm starvation in elective circumstances. (In case of an emergency, i.e. a patient with a full stomach, a rapid sequence induction with Sellick's maneuver is carried out)
- Assess the airway to identify the ease of intubation
- Start essential monitoring like ECG, NIBP and pulse oxymetry
- Secure an intravenous access
- Things kept ready prior to induction: SALT (Suction, Airways, Laryngoscope, ETT)
  - A functioning suction apparatus with an attached catheter, oropharyngeal and nasopharyngeal airways to overcome upper airway obstruction if required, at least two working laryngoscopes with proper sized blades, proper sized endotracheal tubes and a syringe for cuff inflation or cuff inflation device.
- Position of the patient. The aim is to position the oral, pharyngeal and laryngeal axes in one line, i.e. in the line of vision of the anesthetist to optimize vocal cord visualization. This can be achieved by: (1) Flexion at the neck by keeping a head ring or thin pillow below the head of the patient to raise it by approximately 10 cm. (2) Extension at the atlanto-occipital joint. This position is called as sniffing the morning air position or the Chevalier Jackson position.
- Intravenous or inhalational induction of anesthesia till patient loses consciousness
- Administer muscle relaxant to facilitate tracheal intubation after checking adequate of mesk ventilation.
- Open the mouth with the right hand and introduce the laryngoscope into the right side of the mouth, using the left hand moving the tongue to the left. The blade (curved Macintosh) is passed over the surface of the tongue till it reaches the space between the base of the tongue and the epiglottis. Lift the laryngoscope in the direction of the handle without applying any pressure on the upper incisors. The glottis will be visible under the epiglottis as the epiglottis is lifted forward. If a straight blade (e.g. Magill's) is used, the tip is placed over the epiglottis and lifted as above. Could result in bradycardia as the dorsal surface of the epiglottis which is innervated by the superior laryngeal branch of the vagus is touched.
- After laryngoscopy, the proper size tube is passed from the right corner of the mouth through the vocal cords till the proximal edge of the cuff is beyond the cords or the transverse black line (if present on the tube) is at the cords. Confirm the intratracheal placement of ETT by auscultation and capnometry.
- The cuff is inflated till any leak of air disappears.

How would you select the appropriate size of the tube for your patient?

A size 7.5 or 8 mm internal diameter (ID) is chosen for an adult male and a size 7 or 7.5 mm ID is chosen for an adult female patient. However there is evidence to suggest that, the incidence of hoarseness and sore throat is increased with the use of tubes with ID greater than 7 mm. Therefore, currently there is a trend towards using smaller tubes even in adult patients. For nasal intubation, size of tube will be decided by size of external nares.

Pediatric endotracheal tube sizes are calculated based on the weight and age of the patient. The narrowest portion of a child's airway is the cricoid cartilage and circular, a correct size uncuffed tube will achieve a good fit with minimal air leak.

Uncuffed tubes had been in use in pediatric patients until 8–10 years of age till recently however with development of thin polyurethane cuffs, cuffed endotracheal tubes are available in all paediatric sizes. A commonly used formula for uncuffed tube selection in patients over 1 year of age is:

Age in years/4 + 4.5 mm,

The modified Cole formula [4 + (age/4)] for children aged 2 and older is also used.

The formula for calculating a cuffed tube size is:

Age/4 + 3 mm (Khine formula)

Microcuff endotracheal tubes developed for use in children consist of a short, ultra-thin polyurethane cuff located away from the subglottic area (the narrowest part of the glottis in children and hence vulnerable to cuff induced damage). The cuff effectively seals the tracheal wall at pressures as low as 10 cm H_2O and fills the gap between the tube and the tracheal wall without folds. The Murphy eye is absent.

Table 46.2: Selection of size of tube

Age	Tube size ID in mm
10 – 14 years	7.0 – 7.5
7 – 10 years	6.5 – 7.0
4 – 6 years	5.5 – 6.5
1 – 4 years	4.5 – 5.5
6 – 12 months	4.5 – 5.0
1 – 6 months	4.0 – 4.5
Neonate	2.5 – 3.5
Premature	– 3

A rough guide to estimate the size of the tube in case the age of the child is not known is to use a tube whose external diameter is the same as the distal phalanx of the little finger.

- In paediatric age group: Length of the tube (cm) = (Age/2) + 12 cm

These are rough guidelines. One has to remember that PVC tubes being thin walled usually one size larger tube will pass through the cords than the same external diameter red rubber tube. Since the narrowest portion in children is at the cricoid cartilage, the tube that is able to pass through the cords may not always pass through the subglottic region and should never be forced through it as it may cause severe trauma to larynx. Oral intubation is preferred in children (below 11 years), as there is a risk of injury to the hypertrophied adenoids and profuse bleeding during a nasal intubation.

How would you confirm the intratracheal placement of an endotracheal tube?

Following confirmatory criteria are used for intratracheal placement of ETT:

- Visualize the tube passing through the vocal cords
- A Capnograph /$EtCO_2$ (end tidal carbon dioxide) curve
- Feel and compliance of the reservoir bag
- Chest wall rise with inspiration
- Auscultation of chest wall
- Movement of the respiratory mist in the ETT during ventilation
- Absence of epigastric/gastric distention
- X-ray chest
- Fiberoptic confirmation
- Esophageal detector device
- Sonomatic confirmation of the tracheal intubation. (SCOTI): A lightweight battery-powered, sonomatic device which emits sound waves into the tube and analyzes the reflection
- Good patient color and pulse- oximeter value.

What are the other types of ETT which you know? Describe each in brief.

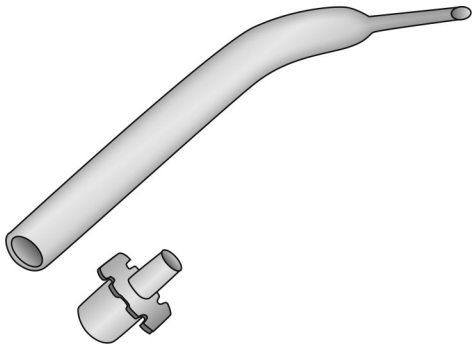

Fig. 46.4: Cole tube

It is used in neonates, designed for use in emergency neonatal resuscitation. Cole tube is an uncuffed tube with a tapering patient end. It has a shoulder at the junction of the tracheal end and the broad proximal end. The shoulder prevents endobronchial intubation as well impacts in the larynx and provide an airtight seal. It was designed to minimize the resistance to flow, however the resistance is paradoxically increased due to turbulence at the junction with the narrow portion of the tube. Cole tube is sized according to the internal diameter of the tracheal end. Can cause trauma to the larynx and trachea if, the shoulder is forced into the larynx. These tubes cannot be used for nasal intubation.

Preformed Tubes (RAE or Ring- Adair- Elwin Tubes)

These tubes have a preformed bend in the tube, so as to facilitate surgery on the head and face. They are made of PVC material. The external portion of the oral version (south polar) is bent at an acute angle so that it rests on patients chin with

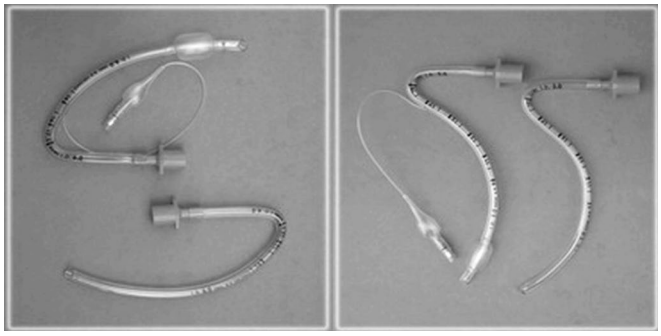

Fig. 46.5 RAE tube

the connector on chest away from operating field. It is used in cleft lip/palate/nasal surgeries. The nasal version (north polar) has a cephalic curve so that the connector rests on forehead. It is used for surgeries on the lower face, mandible and floor of mouth. The nasal and oral versions are available in various sizes both cuffed and uncuffed. There is a black mark at the bend, in most of the cases when it is positioned at the level of teeth or external nares in case of nasal tube, the tube is properly placed in the trachea. Selection of a tube of proper size is mandatory as the length of the tube beyond the bend is fixed. A smaller size tube may not comfortably reach below the glottis. While selecting the tube patients height and weight may be more useful than age.

Disadvantages: Tracheal suction is difficult due to bend in the tube however in emergency tube can be cut at the bend for proper suctioning. Selection of proper sized tube is essential for accurate positioning. The tube offers more resistance than a conventional tube.

RAE – flex tubes are also available which have conventional distal segment and spiral embedded proximal segment.

Oxford Tube

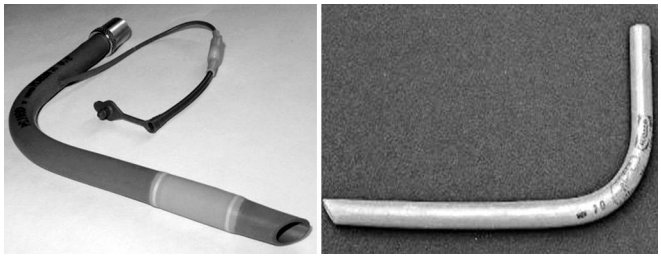

Fig. 46.6 Oxford tube

It is an L shaped tracheal tube made up of red rubber. May be cuffed or uncuffed. Used for upper face surgeries as it is non kinkable. The internal diameter is uniform throughout the tube but the thickness of tube wall varies. The proximal portion is thicker than the tracheal segment thus allowing bigger tube to be passed through the trachea and the thicker wall at the lips prevents compression by the mouth gag. The bevel is situated posteriorly and may abut against tracheal wall if the head is flexed. The distance from the bevel to the curve is fixed hence a proper sized tube must be used to avoid endobronchial intubation. The tube offers greater resistance, difficult to suction through and is not transparent.

Flexo-Metallic/Spiral Embedded/Reinforced/Armored ETTs

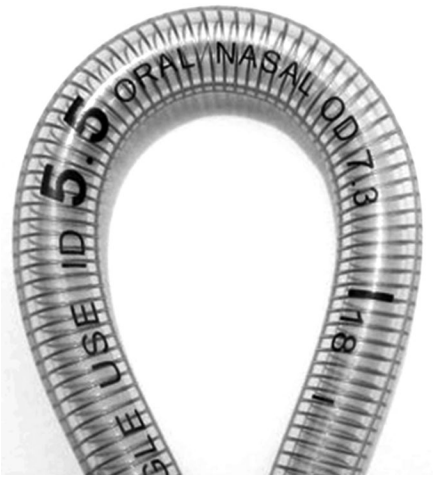

Fig. 46.7 Armored ETT

These are made of silicon rubber or PVC. Have a metal or nylon spiral within the wall of the tube which makes them flexible and non-kinking. The spiral does not extend into the proximal and distal ends. It is easy to pass this tube over the fibre optic bronchoscope.

Disadvantages

- *Thicker wall:* Hence a smaller ID for a given outer diameter.
- *Difficult to insert:* A forceps' or a stylet is required for intubation; tube may rotate on the stylet during insertion.
- Due to the spirals, it is not possible to cut the tube to the desired length. Risk of endobronchial intubation if not well-secured.
- The elastic recoil force may increase the incidence of accidental extubation hence the tube needs to be secured properly in place and a throat pack packed around the tube.
- On repeated use the tubes become sticky and soft especially at the junction of the start of the spiral and the connector and liable to kinking at this spot.
- *The tube does not have a Murphy eye:* may result in obstruction if the bevel abuts against the tracheal wall.

Specific indications for these tubes are head, face and neck surgeries where the head and/or the neck is rotated or flexed. For example, Neurosurgery, thyroidectomy, airway surgery like tracheal resection where the armored tube is passed through the tracheostomy stoma, spine surgery (prone position), etc.

Microlaryngeal tracheal surgery tubes (MLT)

It has a smaller external diameter and an ID of approximately 5 mm with an adult high-volume low pressure cuff (cuff diameter is the same as on a standard 8 mm ID tube). This large cuff helps the tube to be in the center of the trachea. Some MLT may have a yellow colored cuff for better visibility. It is designed for microlaryngeal surgeries allowing better visibility and access to the surgical field. The tube offers a high resistance to gas flows and requires ventilation to be controlled with a long expiratory phase to prevent incomplete exhalation. The tubes are not laser resistant.

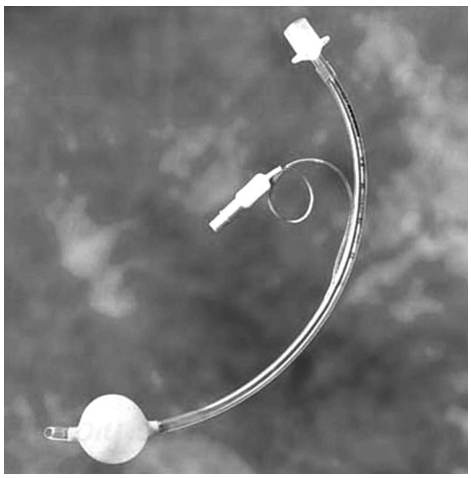

Fig. 46.8 MLT

What are the complications of endotracheal intubation?

Complications of endotracheal intubation include:

- At intubation
  - Trauma to lips, teeth, tonsillar pillar, tongue and nose, neck and jaw
  - Nasal, retropharyngeal, pharyngeal, uvular, laryngeal, tracheal, esophageal and bronchial trauma
  - Spinal cord and vertebral column injury
  - Vocal cord avulsions, fractures and dislocation of arytenoids
  - Hypertension, tachycardia, bradycardia and arrhythmias
  - Raised intracranial and intraocular tension
  - Laryngospasm, Bronchospasm, Laryngeal trauma
  - Airway perforation
  - Aspiration of gastric contents
  - Failed intubation
  - Esophageal intubation
  - Bacteremia especially with nasal intubation
- While the ETT is in place
  - Disconnection and dislodgement
  - Endobronchial intubation
  - Airway obstruction: By blood, secretions, foreign body, compression by a mouth gag, kinking
  - Tension pneumothorax: Due to overinflation of the lungs in a patient with an emphysematous bulla
  - Tracheal tube fire as in laser surgeries
  - Unsatisfactory cuff seal
- At extubation
  - Cuff related problems
  - ETT sutured to trachea or bronchus leading to difficult extubation.
  - Laryngeal edema
  - Aspiration of oral or gastric contents
- After extubation
  - Sore throat, hoarseness
  - Laryngeal edema
  - Nerve injury
  - Superficial laryngeal ulcers
  - Laryngeal granuloma
  - Glottic and subglottic granulation tissue
  - Laryngeal synechiae
  - Vocal cord paralysis and aspiration
  - Tracheal stenosis
  - Sinusitis in prolonged nasal intubation.

What is a double lumen tube (DLT) and why it is used?

A double lumen tube (DLT) is essentially two single lumen tubes of different lengths bound together and termed right or left sided depending on which main stem bronchus the tube is designed to fit. The shorter tube terminates above the carina and the longer tube (bronchial stem) extends into the main bronchus which it is designed to fit. The tube has 2 curves, an anterior curve to fit the oropharyngeal-laryngotracheal curvature and a second curve (bronchial curve) to the right or left depending on the bronchus that it fits into. The tube has two cuffs, one which seals the trachea and the other on the bronchial stem which seals the main bronchus. Both cuffs have separate inflation tubes, pilot balloons and connectors which are color coded for identification. The connectors are attached to the Y shaped catheter mount and then attached to the breathing system. DLT's are available in right and left-sided versions. The right sided version has an eye in the bronchial cuff to allow ventilation of the right upper lobe. There is no eye in the left sided DLT. The right sided double lumen tube is intended for the surgery on the left lung where the left lung needs to be collapsed with continued ventilation of the right lung. The left sided DLT is intended for the use during the surgery on either right or left lung. Left sided tubes are preferred even for left sided surgery because of

risk of occlusion of the right upper lobe bronchus with the bronchial cuff of a right-sided DLT.

Conditions where a right sided DLT is absolutely indicated are:

- Left pneumonectomy
- Tumor in the left main stem bronchus
- Left lung transplantation
- Left bronchial trauma or distorted anatomy
- Left bronchial stent in place (which could be displaced if a left DLT is inserted).

Size: Conventional PVC DLTs, were previously available in only adult sizes (35, 37, 39, and 41 Fr), but now smaller sizes are available. The smallest cuffed DLT is 26 Fr (Rusch) may be used in children as young as 8 years of age. Double-lumen tubes are also available in sizes 28 and 32 Fr (Mallinckrodt Medical), suitable for children10 years of age and older.

Marraro has described a bilumen tube for infants. This tube consists of two separate uncuffed tracheal tubes of different lengths attached longitudinally.

DLT's are used for separating the two lungs during lung surgery or other thoracic surgeries, allowing the anesthetist to selectively deflate a lung and ventilate the non deflated one.

What are the types of DLT?

Type of DLTs:

- *Carlen's:* Left-sided version with a carinal hook. One lumen anterior to the other. The carinal hook can traumatize the larynx or carina and make it difficult to insert. This tube may be especially useful with massive hemoptysis when verification of the tube placement is difficult. Lumen is small, hence greater resistance and difficult to suction down the tubes.
- *White:* A right sided version similar to Carlen's tube. Has a carinal hook. The cuff for the right main stem bronchus is circumferential superior to the opening to the upper lobe bronchus and continues distally behind the opening. Both Carlen's and White's tubes are not in clinical use due to their shortcomings and availability of better tubes in market
- *Robertshaw:* Made of red rubber. Both left and right versions available. Lumens are side by side and wider than older tubes. Used for multiple use.
- *PVC tubes:* Available from many companies.

PVC DLT

It is made up from tissue implantable plastic. The two D shaped lumen lie side by side with a bronchial lumen at an angle of 20° in right side DLT's and 40° in left side DLT's. The lumens are wider and hence offer less resistance to ventilation and allow suctioning. It has 3 ends and 2 curves, the tracheal end which lies in trachea and ventilates the lung being operated, the bronchial end lies in the main stem bronchus and ventilates the nonoperative lung and the machine end which is bifurcated to connect to the double catheter mount. The 2 curves are placed right angles to each other, the proximal corresponding to the oropharyngeal curve and the distal corresponding to the bronchial curve as the main stem bronchus is given off from the carina. There are 2 cuffs, bronchial (blue colored for easy identification through FOB) and tracheal (white) cuff, seal the respiratory tract from the GIT and atmosphere. In right sided DLT's, the bronchial cuff has a slot in its lateral aspect for upper bronchus ventilation. The carinal hook is absent. Malleable intubation stylet and suction catheters are packaged with the tube.

They are available in five sizes—41,39,37,35 and 28 corresponding to internal diameters of 6.5, 6.0, 5.5, 5.0 and 4.5 mm respectively. They are larger in size as compared to any other double lumen tube of same size and for single use only.

How is the right main stem bronchus different to the left and how does this affect right-sided DLT design?

The adult trachea begins at the lower end of the cricoid cartilage (C6 vertebral level) and bifurcates behind the manubriosternal joint (T4). It is 15 cm long. The main differences between the right and left main bronchi include the following:

- Right bronchus is shorter wider (supplies larger lung) and diverges away from the trachea at a 25° angle, while the left bronchus diverges at a 45° angle.
- Left bronchus divides into only upper and lower lobe branches while the right bronchus has upper, middle, and lower lobe branches.
- It is difficult to correctly position a right-sided DLT because of anatomic variations between individuals in the distance between the right upper lobe orifice and the carina often resulting in obstruction of right upper lobe bronchus and difficulty in ventilating the right upper lobe. The orifice of the right upper lobe bronchus from the carina is 15 mm in women and 20 mm in men, while that of the left upper lobe is 50 mm from the carina. Right-sided DLTs therefore have a slit in the bronchial cuff for ventilating the right upper lobe. Correct placement requires confirmation with a fiberoptic bronchoscope.

Choosing the appropriate size double-lumen endotracheal tube and insertion technique

See chapter on "Pneumonectomy".

What are the complications of DLTs?

Complications include:

1. Upper airway trauma during insertion.
2. Malpositioning resulting in hypoxia, inability to ventilate good lung, inability to deflate operative lung, etc.
3. Disruption of tracheobronchial tree.
4. Suturing the endobronchial cuff or tube to the bronchial stump resulting in difficult extubation.
5. Traumatic laryngitis.

What are the indications of one lung anesthesia?

Indications:

- Absolute
  - Isolation of one lung from the other to avoid spillage or contamination
    - Infection
    - Massive hemorrhage
  - Control of the distribution of ventilation
    - Bronchopleural fistula
    - Bronchopleural cutaneous fistula
    - Surgical opening of a major conducting airway
    - Tracheobronchial tree disruption
    - Life-threatening hypoxemia due to unilateral lung disease
  - Unilateral bronchopulmonary lavage
  - Video assisted thoracoscopic procedures
- Relative
  - Surgical exposure (high priority)
    - Thoracic aortic aneurysm
    - Pneumonectomy
    - Upper lobectomy
    - Mediastinal exposure
    - Thoracoscopy
  - Surgical exposure (low priority)
    - Middle and lower lobectomies and subsegmental resections
    - Esophageal surgery
    - Thoracic spine procedure
    - Minimally invasive cardiac surgery (MID-CABG)
  - Severe hypoxemia due to unilateral lung disease

Enumerate methods of isolation of lung other than DLT with brief description of each isolation of lungs without DLT

It may not be possible to pass a DLT in all cases. For example, In children, distorted airway anatomy, non-availability of the tube or appropriate size. In such cases, the isolation can be achieved by:

- Introducing a bronchial blocker into the operative side to prevent the blood and secretions trickling into the normal side and soiling it
- Introducing a Fogarty catheter or a Foley's catheter into the operated bronchus serving as the bronchial blocker
- Univent tube
- Passing a single lumen tracheal tube into the bronchus of the nonoperated lung (not a very good option).

What is combitube?

Combitube is a double lumen tube that allows blind placement in either the trachea or the esophagus. It is a single use device. Available in two adult sizes 37 Fr and 41 Fr only.

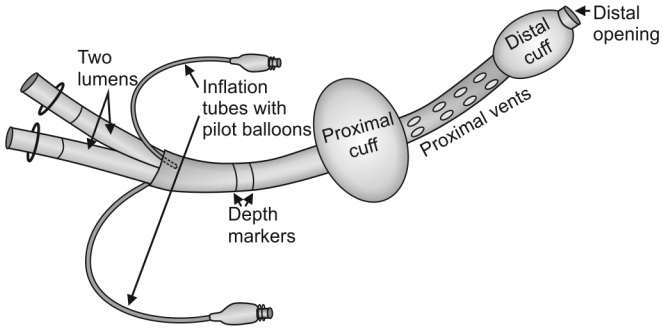

Fig. 46.9 Combitube

Consists of:

- A large proximal (oropharyngeal) cuff, inflated with 100 mL of air. Seals the pharynx by filling the space between the base of tongue and soft palate so that no gas escapes through the mouth
- A distal (esophageal/tracheal) cuff inflated with 15 mL of air
- Two lumens, fused longitudinally with a universal connector attached to each end. One lumen opens beyond the distal cuff, while the other lumen (usually colored blue) ends between the two cuffs (blind end) and has 8 ventilating ports located in between the two cuffs.
- Two inflation tubes with a pilot balloon and a self-sealing valve each. Pilot balloon to proximal cuff is also blue colored.
- A distal opening beyond the distal cuff opens in the trachea or the esophagus depending on the tube's position.
- There are two depth marks on the airway tube.

This tube is used for emergency airway management and not for anesthesia. It has been included as the alternative airway device in the guidelines for ACLS of the AHA, practice guidelines by Difficult Airway of USA, Canadian Airway Focus Group and also the European Resuscitation Group.

When inserted blindly, tube enters the esophagus in 95% cases. Ventilation is possible regardless of whether the tip is in the trachea or the esophagus and IPPV may be performed through either tube depending on the device's position.

The tube after blind insertion is advanced till the 2 black depth markers are at the level of the incisors. The proximal cuff is inflated first.

When the tip is in the esophagus, the distal cuff seals the esophagus and protects against aspiration. A gastric tube can then be placed through that lumen. The proximal cuff seals the pharynx by filling the space between the base of tongue and soft palate so that no gas escapes through the mouth. Always ventilate through the blue lumen first. The air escapes through the 8 ventilating ports between the two cuffs. It can not enter esophagus because of the seal formed by the inflated distal cuff. If ventilation through the blue lumen is not possible, then the distal tip is possibly in the trachea, ventilate through the other lumen.

If ventilation is not possible through either lumen, it may be because of obstruction of the larynx by the proximal balloon. The device should hence be withdrawn by 1–2 cm and ventilation attempted again.

The device has been in use in prehospital settings by paramedical personnel for over 20 years and has been used successfully in patients with difficult airway following severe facial burns, trauma, and upper airway bleeding and vomiting where it was not possible to visualize the vocal cords. However its use is contraindicated in the following scenarios

- Upper airway obstruction due to foreign body or tumor
- In case a patient has ingested some caustic agent.

Disadvantages

- Increased incidence of a sore throat, dysphagia and upper airway hematoma
- Risk of esophageal rupture
- Not possible to suck out the secretions and/ or blood from the pharynx.

How would you sterilize the endotracheal tubes?

Disposable tubes are available at such a low cost that it is usually not cost effective to use reusable tubes.

If the tube is reusable, secretions should be removed by rinsing and then soaking in a detergent solution. Remove the connector and rinse the tube thoroughly with soap water, making certain to flush the lumen and then dry the tube. This tube then can be sterilized by

1. *Steam sterilization*: (Autoclaving) steam sterilization under pressure kills all bacteria, spores and viruses. Repeated autoclaving makes them soft, more liable to kink and decreases the elasticity of the cuff.
2. *ETO* (*ethylene oxide*)*:* ETO is a popular method for sterilizing tracheal tubes as it is method of choice for heat sensitive items. However with repeated gas sterilization, rubber tracheal tubes soften and kink more easily.
3. Liquid chemical disinfection has been used for tracheal tubes. The inflation tube should be closed (capped) during immersion to prevent the solution from entering the cuff. The tube frequently floats and need to be held submerged with an object. Thorough rinsing is necessary after use of all chemical agents except alcohol. Pseudomembranous laryngitis has been reported following disinfection of tracheal tubes with glutaraldehyde.

Suggested Reading

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47 Cardiopulmonary Resuscitation

S Myatra, B Trivedi, A Kothekar

The 2010 American Heart Association (AHA) Guidelines for CPR contain the recommendations designed to improve survival from Sudden Cardiac Arrest (SCA).These guidelines are based on the most extensive review of literature of CPR and specially designed to reduce the information the rescuer needs to remember and also simplify the important skills he needs to perform.

These are published in: Circulation on October 18, 2010. The following article is based on these recommendations.

What is Cardiac Arrest and how will you clinically diagnose it? What is Cardiopulmonary Resuscitation?

Cardiac arrest is the cessation of all cardiac mechanical activity. Its clinical diagnosis is confirmed by:

- Unresponsiveness
- Absence of detectable pulse
- Apnea or agonal respiration (gasping type of breathing).

Cardiopulmonary Resuscitation is an attempt to restore spontaneous circulation through any of a broad range of maneuvers and techniques. If cardiopulmonary resuscitation is delayed or delivered in appropriately, the survivor may have various degrees of neurological dysfunction, which is undesirable. Thus all attempts at CPR should be performed with the aim of ROSC (return of spontaneous circulation) with intact cerebral function. Thus CPR has been rightly termed as CPCR (Cardiopulmonary Cerebral Resuscitation).

List the cardiac arrest rhythms, which of these are amenable to defibrillation?

The four cardiac arrest rhythms are:

- Asystole
- PEA (Pulseless Electrical Activity)
- Ventricular Fibrillation
- Pulse less Ventricular Tachycardia

Ventricular fibrillation and pulseless ventricular tachycardia are amenable to defibrillation.

What is the management of a patient who has collapsed in your presence?

Enumerate the sequence of BLS and ACLS.

Step 1

Early recognition of Sudden Cardiac Arrest (SCA) - Check Responsiveness and Breathing

- Check Response—gently tap the patient on her shoulders and check for a response.
- Check Breathing—no breathing or no normal breathing (i.e. only gasping). Remember short period of seizure-like activity or agonal gasps may occur in victims of cardiac arrest and often confuse the rescuer.
  Suspect cardiac arrest if there is no response and absent or agonal respiration.

Step 2

Activate the Emergency System: Activate the emergency system if present in your hospital or just shout for help. Ask for help with the defibrillator or if there is extra person send him to get the defibrillator.

Step 3

Check pulse: There is a de-emphasis on pulse check. Pulse check if done using a central pulse (carotid or femoral) for no more than 10 seconds. If pulse is not felt in 10 seconds or there is any doubt start chest compression.

Step 4

Start CPR: Initiate chest compressions before giving rescue breaths (A–B – C is now C – A – B)

Positioning

- Victim should lie supine on a hard surface.
- Rescuer kneels beside the victim's thorax (either side).

- Keep arms straight, elbows locked and shoulder directly above the hand.
- Hand placement-place the heel of the hand on the lower half of the victim's sternum in the center (middle) of the chest, between the nipples, then place the heel of the second hand on top of the first so that the hands are overlapped and parallel. Interlock fingers to avoid compression on the ribs.

Technique: During CPR remember to "push hard and push fast".

- Compression rate—at least 100 per minute (but not very high either).
- Depth of sternal compression – at least 2 inches, i.e. 5 cms (1/3 AP diameter in children and infants).
- Compression-Ventilation Ratio: Adult patient 30:2 (with 1 or more rescuers). Child or infant 30:2 (with 1 rescuer) 15:2 (with more than 1 rescuer).
- Compression-Relaxation Ratio: 1:1 (allow complete recoil of the chest).
- Perform 5 cycles (approximately 2 minutes) of compression and ventilation (ratio 30:2).
- Switch the compressor every 2 minutes (i.e. after every 5 cycles).
- Give 2 minutes of uninterrupted CPR (limit interruptions to < 10 seconds, interrupt only during intubation and just when you are ready to deliver a shock).

To easily achieve the above, one could use simple counting at the speed of approximately 100 per minute …"one and two and three and …." Every time you say a number – compress and when you say "and" you relax.

Airway and Ventilation

- Open the airway using a "head tilt – chin lift" maneuver (avoided when head and neck trauma is present or suspected) or "jaw thrust" maneuver.
- Give 2 slow rescue breaths using face mask and AMBU bag (using reservoir bag) to deliver 100% oxygen after every 30 compressions.
- Give one breath over one second (rapid ventilation which could cause gastric insufflations and increase the risk of aspiration).
- Give sufficient tidal volume to ensure visible chest rise.
- Reposition mask if there is a leak and insert an oral or nasal airway if there is airway obstruction due to tongue fall.
- Use of cricoid pressure during ventilation is generally not recommended.

Step 5

Attach defibrillator (AED or manual defibrillator) and shock if indicated

- As soon as a AED/defibrillator is available, attach it and shock if indicated, i.e in ventricular fibrillation (VF) and pulse less ventricular tachycardia (VT).
- Prefer a biphasic defibrillator, if unavailable use monophasic defibrillator.
- Electrode placement—anterior-lateral pad position (default). Alternative positions are anterior-posterior, anterior– left infrascapular and anterior–right infrascapular.
- Shock Energy—Biphasic: Use manufacturer recommendation (120–200 J), if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered if available. Monophasic: 360 J (In children and infants use 2–4 J/kg first and 4 J/kg for subsequent shocks, higher energy may be considered but not to exceed 10J/kg).
- Ensure that no one is touching the patient before you shock. Charge to the appropriate energy level, use the warning chant - "all clear" – then SHOCK.
- No pulse check is recommended after defibrillation, resume CPR immediately.
- If non-shockable rhythm (Asystole or PEA) continue chest compression.
- Reattach the defibrillator after every 2 minutes of CPR.
- Reduce time between the last compression and shock delivery and the time between shock delivery and resumption of compressions. CPR should be performed while the defibrillator is readied.
- There is no upper limit to the number of shocks you can give. Remember the shockable rhythms are the ones with the better prognosis so never give up. As long as VF/VT persists attempts at defibrillation must continue (as long as the myocardium has energy to produce VF, it has energy to produce a perfusing rhythm.
- AEDs can now be used even in infants with a pediatric dose attenuator, if manual defibrillator is not available. If neither is available AED without pediatric dose attenuator can be used (All AEDs are biphasic).
- The precordial thump may be considered in witnessed monitored unstable ventricular tachyarrhythmias only when a defibrillator is not available.
- Electric pacing is not recommended for routine use in cardiac arrest.

Step 6

Drug Therapy

- Use Intravenous (IV) or intraosseous (IO) route for bolus delivery of drugs. For IV use give bolus drug followed by a

20 mL saline push and raise the extremity. (Use special IO needles to deliver the drug; if using the IO route).

- If both IV and IO are unavailable then tracheal route, i.e through endotracheal tube (ETT) may be used. Epinephrine, Atropine, Lidocaine and Naloxone may be administered through this route. Optimal ETT doses of medications are unknown, in general experts recommend use of 2–2½ times the dose diluted 5–10 mL of distilled water or saline.
- Give a vasopressor soon after giving shock. Epinephrine IV/IO Dose: 1 mg every 3–5 minutes (Vasopressin IV/IO 40 units can replace first or second dose of epinephrine and repeated again after 20 minutes and once Vasopressin is given no need to give epinephrine for 20 mins). Pediatric dose of epinephrine 0.01 mg/kg (0.1 mL/kg 1:10,000) IV/IO and 0.1 mg/kg (0.1 mL/kg 1:1000) through ETT. Maximum dose is 1 mg IV/IO and 2.5 mg through ETT.
- Amiodarone should be given when VF/VT is unresponsive to CPR, defibrillation, and vasopressor therapy. IV/IO Dose: first dose: 300 mg bolus and second dose: 150 mg (after 3–5 minutes if VF/VT recurs or persists). This may be followed by a 24 hour infusion (1 mg/minute for 6 hours and then 0.5 mg/minute for the remaining18 hours. Use Lidocaine only if Amiodarone is unavailable.
- Atropine should not be used during PEA or asystole as it is unlikely to have a therapeutic benefit.
- Other drugs not routinely used and should be considered only in specific situations are:
  - Magnesiun Suphate (1–2 g) for hypomagnesemia and Torsades de pointes associated with a long QT interval (25–50 mg/kg IV/IO over 10–20 minutes in children)
  - Sodium bicarbonate (initial dose is 1 mEq/kg) repeat half this dose every 10 minutes thereafter. Should be used only if there is hyperkalemia (Class I), bicarbonate responsive acidosis or TCA overdose (Class II a). After prolonged resuscitation (IIb). It is harmful in hypercarbic acidosis (Class III)
  - Calcium gluconate/chloride should not be routinely used. Recommended only in the presence hyperkalemia, hypocalcemia or calcium blocker overdose.

Step 7

Advanced Airway

- Weigh the need for minimally interrupted compressions against the need for insertion of an advanced airway, i.e endotracheal tube or supraglottic airway (Laryngeal mask airway, esophageal tracheal tube or laryngeal tube).
- Continue bag mask ventilation if advanced airway is not placed.
- Confirm placement of advanced airway by clinical method (chest expansion and breath sound) and in addition use capnography to confirm and monitor correct placement. Record depth and secure the tube.
- Once advanced airway is in place give 8–10 breaths per minute and continue chest compressions at atleast 100 per minute independent of each other.

Step 8

Treat Reversible Causes

During each 2-minute period of CPR review the most frequent causes - 5 H's and 5 T's to identify factors that may have caused the arrest or may be complicating the resuscitation

Hypovolemia	Tension pneumothorax	Hypoxia
Tamponade, cardiac	Hydrogen ion (acidosis)	Toxins
Hypo/hyperkalemia	Thrombosis, pulmonary	Hypothermia
Thrombosis, coronary		

Step 9

Monitor CPR Quality throughout resuscitation

- Give emphasis on delivering high quality CPR. This means giving compressions of adequate rate and depth, allowing complete chest recoil between compressions, minimizing interruptions in compressions, avoiding excessive ventilation and rotating the compressor every 2 minutes.
- Use quantitative waveform capnography to monitor end-tidal CO_2 (expressed as a partial pressure in mm Hg - $PetCO_2$) in intubated patients. If $PetCO_2$ <10 mm Hg, attempt to improve CPR quality.
- If intra-arterial pressure monitoring is present then try to maintain the diastolic pressure >20 mm Hg during chest compression.
- ROSC can be confirmed by return of pulse or blood pressure or abrupt sustained increase in $PetCO_2$ (typically ≥ 40 mm Hg) or spontaneous arterial pressure waves with intra-arterial monitoring.

Step 10

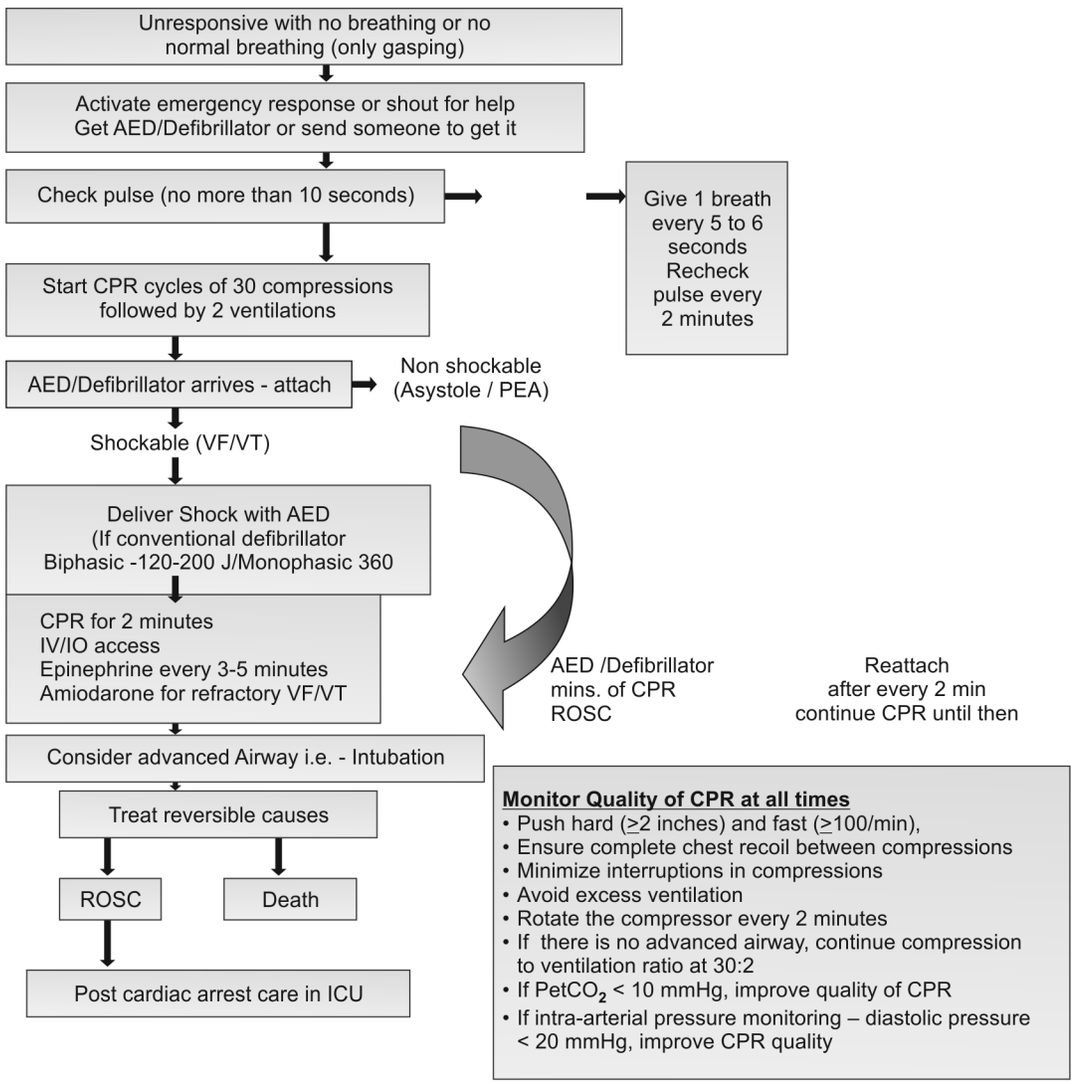
Post Cardiac Arrest Care after ROSC

- Goal—Optimize cardiopulmonary function and vital organ perfusion
- Transfer patient to an appropriate hospital or ICU with facility to deliver postcardiac arrest care.
- Optimize ventilation to minimize lung injury. Do Chest X-ray to confirm secure airway and diagnose pneumonitis, pneumonia, pulmonary edema. Use lung protective ventilation if there is pulmonary dysfunction; adjust settings using blood gas values. Avoiding excessive ventilation and hyperoxia. Once ROSC is achieved, the fraction of inspired oxygen (FiO_2) should be adjusted to

the minimum concentration needed to achieve arterial oxyhemoglobin saturation ≥ 94%, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery.

- Treat hypotension (SBP < 90) with fluid and vasopressor and treat the other reversible causes.
- Consider Induced hypothermia. Adult patients with persistent coma after ROSC should be cooled to 32°C to 34°C for 12 to 24 hours provided SBP > 90 mm Hg or MAP > 70 with or without vasopressors. Patients comatose before cardiac arrest or another reason to be comatose (e.g. drug overdose, status epilepticus) should be excluded. Cooling can be done using cold IV fluid bolus 30 mL/kg or using special endovascular catheters with feedback control to give cold fluids. This can be combined with surface cooling using ice packs or mattresses. Sedation/muscle relaxants may be used to control shivering, agitation, or ventilator dysynchrony as needed. After 24 hours start slow rewarming at 0.25°C/hr. Prevent hyperpyrexia > 37.7°C.
- Glucose control - moderate glycemic control (140 to 180 mg/dL). Use anticonvulsants if seizures present (use EEG monitoring if available).
- Identify and treat acute coronary syndrome (ACS). Patients with suspected ACS should be sent to a facility with coronary angiography and interventional reperfusion facility (primary PCI).
- Reduce the risk of multiorgan injury and support organ function, if required.

Flow chart 47.1 Managing cardiac arrest



What are the current guidelines for prognostication of a victim after cardiac arrest?

In patients treated with therapeutic hypothermia:

- Clinical neurologic signs, electrophysiologic studies, biomarkers, and imaging should be performed when available 3 days after cardiac arrest.
- Presently, there is limited evidence to guide decisions regarding limitation/withdrawal of life support in these patients as favorable outcomes have been seen in those in whom studies predicted poor outcome. Use your best clinical judgment based on this testing to make a decision.

In patients who have not undergone therapeutic hypothermia:

- Absence of pupillary response to light on the Day 3.
- Absence of motor response to pain by the Day 3.
- Absence of bilateral cortical response to median nerve somatosensory-evoked potentials in those comatose for at least 72 hours after a hypoxic-ischemic insult.

Limitation/Withdrawal of life support in this situation may be considered.

What is the Adult Chain of Survival? How is it different from the pediatric chain of survival?

The AHA uses 5 links in a chain to illustrate the important time-sensitive actions in victims of SCA. Survival from cardiac arrest depends on a series of critical interventions, the term "Chain of Survival" summarizes the present understanding of the best approach to treat patients with sudden cardiac arrest to optimize survival.

The five links of the adult chain of survival are:

- Immediate recognition of cardiac arrest and activation of the emergency response system
- Early CPR with an emphasis on chest compressions
- Rapid defibrillation
- Effective advanced life support
- Integrated postcardiac arrest care.

The five links of the pediatric chain of survival are:

- Prevention
- Early CPR
- Prompt access to emergency response system
- Rapid pediatric advanced life support (PALS)
- Integrated postcardiac arrest care.

The major cause of death in infants and children are respiratory failure, sudden infant death syndrome (SIDS), sepsis, neurologic disease and injuries. Since most of these causes can be prevented, 'prevention' of cardiac arrest is the first link in the pediatric chain of survival. Since the most common cause is respiratory arrest it is acceptable for the health care provider to give 2 minutes of CPR and then call for help.

How does defibrillation work? What is Automated External Defibrillator (AED)?

Defibrillation involves delivery of current through the chest and to the heart to depolarize myocardial cells simultaneously and eliminate VF. As soon as an AED/Defibrillator is available attach and defibrillate if indicated. Most adults with sudden witnessed non-traumatic cardiac arrest are found to be in ventricular fibrillation, for these victims the time from collapse to defibrillation is the single greatest determinant of survival. Survival from VF arrest decrease by approximately 7–10% for each minute without defibrillation.

AED (Automated external defibrillator) is a computerized completely automatic defibrillator which uses voice and visual prompts to guide lay rescuers and healthcare providers to safely defibrillate. They can automatically analyze cardiac rhythm, charge to appropriate energy level and shock if advised. This is the single greatest advance in CPR which has significantly reduced in and out of hospital mortality after SCA. Due to the increasing evidence for improved survival with early defibrillation and the introduction of AEDs, defibrillation which was earlier part of ACLS, has been now included as part of BLS. All AEDs are biphasic and are not recommended for use under the age of one year.

What is the difference between Defibrillation and Cardioversion?

Cardioversion is the electrical maneuver that is used for organized rhythms, e.g unstable tachycardia (SVT, rapid AF etc.). Here the timing of the shock with respect to cardiac cycle is important to avoid R on T phenomenon. The defibrillator/cardioverter have the ability to track the QRS, and to stick a visible marker on each one. This synchronization avoids shock delivery during the relative refractory period of the cardiac cycle when a shock could produce VF. The same machine is used for both cardioversion and defibrillation, however, you have to select the 'sync' button when you want to do cardioversion otherwise it will function as a defibrillator. Sedation will be required if the patient is awake as it is a painful and uncomfortable procedure. The recommended energy for cardioversion in atrial flutter is 50-100 J (biphasic), for atrial fibrillation is 120–200 J (biphasic) and for monomorphic VT it is 100 J (biphasic). In pediatric age group energy recommendations for cardioversion are: 0.5–1 J/kg and can go upto 2 J/kg.

Defibrillation on the other hand is used only for the shockable cardiac arrest rhythms, i.e. VF and pulseless VT. Here there is no synchronization of the shock. Sedation is not required as all cardiac arrest victims are unconscious and the energy used is also much higher than during cardioversion

What is the difference between Biphasic and Monophasic Defibrillation?

Modern defibrillators are classified according to 2 types of waveforms: monophasic and biphasic. Monophasic waveform defibrillators were introduced first, now biphasic waveforms are used in all AEDs and manual defibrillators sold today. Energy levels vary by type of device.

Monophasic waveforms deliver current of one polarity (i.e. direction of current flow). Monophasic waveforms can be further categorized by the rate at which the current pulse decreases to zero. The monophasic damped sinusoidal waveform (MDS) returns to zero gradually, whereas the monophasic truncated exponential waveform (MTE) current is abruptly returned to baseline (truncated) to zero current flow. Few monophasic waveform defibrillators are being manufactured but many are still in use.

Biphasic Waveform delivers bi-directional current. Current research indicates, however, that when doses equivalent to or lower than monophasic doses (360 J) are used, biphasic waveform shocks are safe and effective for termination of VF. Research confirms that it is reasonable to use selected energies of 120 J to 200 J with a biphasic truncated exponential waveform. Outcome with the use of biphasic defibrillation is superior to monophasic defibrillation.

Why A-B-C has been changed to C-A-B in latest guidelines?

During the first minutes of VF SCA, rescue breaths are probably not as important as chest compressions because the oxygen level in the blood remains high for the first several minutes after cardiac arrest. In early cardiac arrest, myocardial and cerebral oxygen delivery is limited more by the diminished blood flow (cardiac output) than a lack of oxygen in the blood. Doing chest compression before ventilation saves 30 seconds. Both ventilations and compressions are important for victims of prolonged VF SCA, when oxygen in the blood is utilized.

During CPR blood flow to the lungs is substantially reduced as the cardiac output is 25 to 33% of normal, so oxygen uptake from the lungs and CO_2 delivery to the lungs are also reduced. As a result, low minute ventilation (lower than normal tidal volume and respiratory rate) can maintain effective oxygenation and ventilation during CPR.

Excessive ventilation is unnecessary and is harmful because it increases intrathoracic pressure, decreases venous return to the heart, and diminishes cardiac output and survival. Avoid delivering breaths that are too large or too forceful. Such breaths are not needed and may cause gastric inflation and its resultant complications.

Describe the special considerations during CPR in some special situations.

A. Drowning
- Drowning is a preventable cause of death. The duration and severity of hypoxia sustained as a result of drowning is the single most important determinant of outcome.
- Rescuers should remove drowning victims from the water by the fastest means available and should begin resuscitation as quickly as possible.
- Provide CPR; particularly rescue breathing, as soon as the unresponsive submersion victim is removed from the water.
- When rescuing a drowning victim of any age, the lone healthcare provider should give 5 cycles (about 2 minutes) of CPR before leaving the victim to activate the EMS system.
- Mouth-to-mouth ventilation in the water may be helpful when administered by a trained rescuer.
- Chest compressions are difficult to perform in water, may not be effective, and could potentially cause harm to both the rescuer and the victim.
- Only victims with obvious clinical signs of injury or alcohol intoxication or a history of diving, waterslide use, or trauma should be treated as a "potential spinal cord injury," with stabilization and possible immobilization of the cervical and thoracic spine.
- If defibrillation is required dry the chest area well before applying the paddles.

B. Hypothermia
- In an unresponsive victim with hypothermia, assessments of breathing and pulse are particularly difficult because heart rate and breathing may be very slow, depending on the degree of hypothermia. Thus, life-saving procedures should be initiated unless the victim is obviously dead (e.g. rigor mortis, decomposition, hemisection, decapitation).
- Victim should be transported to a center where aggressive re-warming during resuscitation is possible, as soon as possible.
- If the adult victim is unresponsive with no breathing or no normal breathing (i.e. only gasping), healthcare providers can check for a pulse, but should start CPR if a pulse is not definitely felt within 10 seconds. Do not wait to check the victim's temperature and do not wait until the victim is rewarmed to start CPR.
- To prevent further heat loss, remove wet clothes from the victim; insulate or shield the victim from wind, heat,

or cold; and if possible ventilate the victim with warm, humidified oxygen.
  - If VF is detected, emergency personnel should deliver shocks using the same protocols used for the normothermic cardiac arrest victim.
  - After ROSC, patients should continue to be warmed to a goal temperature of approximately 32° to 34°C; this can be maintained according to standard post-arrest guidelines for mild to moderate hypothermia in patients for whom induced hypothermia is appropriate.

C. Pregnancy
  - During CPR; to improve the quality left uterine displacement is advocated to remove the aorto-caval compression. This is done manually by 1-hand or 2-hand technique or by using a wedge of predetermined angle.
  - Anticipate difficult airway (due to modified anatomy and the tilt).
  - Chest compressions should be given slightly higher than normal to adjust for the elevated diaphragm.
  - Defibrillation to be used as in non-pregnant adults.
  - IV should be secured above the level of diaphragm.
  - If there is no ROSC within 4 minutes; prepare for emergency Cesarean section (hysterotomy). Aim for delivery within 5 minutes of initiating resuscitative efforts.
  - Therapeutic hypothermia to be considered on individual basis (with continuous fetal monitoring).
  - To simultaneously search and treat for the possible causes of cardiac arrest.

How will you recognize a foreign body airway obstruction (FBAO) in an adult and how will you proceed with resuscitating such a victim?

- Any FBAO should be recognized and relieved quickly.
- If mild obstruction is present and the victim is coughing forcefully, do not interfere with the patient's spontaneous coughing and breathing efforts. Attempt to relieve the obstruction only if signs of severe obstruction develop: the cough becomes silent, respiratory difficulty increases and is accompanied by stridor or the victim becomes unresponsive. Activate the EMS system quickly if the patient is having difficulty breathing.
- For responsive adults and children >1 year of age with severe FBAO, case reports show the feasibility and effectiveness of back blows or slaps, abdominal thrusts and chest thrusts. The abdominal thrust is applied in rapid sequence until the obstruction is relieved. If abdominal thrusts are not effective, the rescuer may consider chest thrusts.
- Abdominal thrusts are not recommended for infants <1 year of age because thrusts may cause injuries.
- Chest thrusts should be used for obese patients if the rescuer is unable to encircle the victim's abdomen and if the choking victim is in the late stages of pregnancy.
- If the adult victim with FBAO becomes unresponsive, the rescuer should carefully support the patient to the ground, immediately activate EMS and then begin CPR. Each time the airway is opened during CPR, the rescuer should look for an object in the victim's mouth and remove it. Simply looking into the mouth should not increase the time it takes to attempt the ventilations and proceed to the 30 chest compressions.
- A finger sweep should be used only when the provider can see solid material obstructing the airway of an unresponsive patient as blind sweep can push the foreign body further in.

What are the current recommendations regarding the route of drug administration during CPR?

Peripheral venous access is the procedure of choice during CPR; cannulate the large easily accessible peripheral veins like the cephalic or external jugular vein. During cardiac arrest follow all administered drugs with a bolus of 20 mL IV saline while using a peripheral line and a limb raise. Use a central line if already in place, do not attempt central venous cannulation if peripheral access is available as this requires asepsis, skills and takes more time than taking a peripheral line.

If IV access cannot be established, intraosseous (IO) delivery of resuscitation drugs will achieve adequate plasma concentrations (you will need special intraosseous needles for this). Resuscitation drugs can also be given via the tracheal tube, but the plasma concentrations achieved are variable and substantially lower than those achieved when the same drug is given by the IV or IO route. If IV access is delayed or cannot be achieved, IO access should be considered.

What are the current recommendations for the use of vasopressors during cardiac arrest?

Both epinephrine and vasopressin may be used as vasopressors during cardiac arrest.

1. *Epinephrine:* Indicated for all cardiac arrest rhythms, i.e. VF, pulseless VT, asystole and PEA. IV/IO dose is 1 mg administered every 3–5 minutes followed by 20 mL saline flush. In large randomized clinical trials, doses of epinephrine greater than 1 mg were not beneficial and did not improve survival. There is no upper limit on how much adrenaline can be given.
2. *Vasopressin:* It is a naturally occurring antidiuretic hormone, becomes a powerful vasoconstrictor when

used at higher does than normally observed in the body. It produces the positive effects of epinephrine in terms of vasoconstriction and increasing blood flow to the heart and brain during CPR without its adverse effects on the heart, such as increased ischemia and irritability and, paradoxically the propensity for VF.

It can be used for all the 4 cardiac arrest rhythms using a single one time bolus dose of 40 U IV/IO the effect of which lasts for 20 minutes. After a single dose of vasopressin if there is no clinical response within 20 minutes, it is acceptable to return to adrenaline 1 mg every 3–5 minutes. There is insufficient evidence to support or refute the use of vasopressin as an alternative to or in combination with, epinephrine in any cardiac arrest rhythm. Both these drugs can be given through tracheal route if IV/IO is not available at 2–2½ times the dose diluted with 5–10 mL of distilled water or saline.

Which are the investigations you will perform in a cardiac arrest victim during resuscitation?

Immediate investigations

- ABG (to look for acidosis)
- Blood glucose
- Serum electrolytes (to look for hyperkalemia).

What is Impedance Threshold Device?

The impedance threshold device (ITD) is a valve that limits air entry into the lungs during chest recoil between chest compressions. It is designed to reduce intrathoracic pressure and enhance venous return to the heart. The addition of the ITD improved the hemodynamics during standard CPR in 5 laboratory studies and 1 clinical study. It is not in regular use. Only centers with expertise may use it as an adjunct.

How does chest compression help during CPR? What alternate techniques and devices other than standard chest compression that have been described to assist circulation and what is their current status?

Chest compressions create blood flow by increasing intrathoracic pressure or by directly compressing the heart. When rescue breathing is provided using oxygen and blood is circulated to the lungs by chest compression the victim will receive oxygenation to the brain and the other vital organs. 'Effective' chest compressions are essential for providing blood flow during CPR (Class I). To give 'effective' chest compressions, "push hard and push fast."

Open Chest CPR

Open-chest CPR is not recommended for routine use. It should be considered only for special situations, such as cardiac arrest in cardiothoracic surgery, patients on table or in the early postoperative phase after or when the chest or abdomen is already open as during surgery or in case of penetrating chest trauma.

Prone CPR

When the patient cannot be placed in the supine position, rescuers may consider providing CPR with the patient in the prone position, particularly in patients with an advanced airway in place, like in the operating room. The chest compressions are given with the hands of the rescuer in the midline between the two scapulae (i.e. at T7–10 position) with rest of the technique same as for supine CPR. Several cases series have documented survival to discharge when this technique was used.

Compression-Only CPR

Recent studies suggest improved outcome toward higher compression rates and more time spent for compression with less emphasis on ventilation.

The outcome of chest compressions without ventilations is significantly better than the outcome of no CPR for adult cardiac arrest. Laypersons should be encouraged to do compression-only CPR if they are unable or unwilling to provide rescue breaths (Class IIa), although the best method of CPR is compressions coordinated with ventilations.

Others Techniques and Devices:

- Interposed Abdominal Compression IAC-CPR.
- Active Compression-Decompression ACD-CPR.
- Load Distributing Band CPR—The load distributing band (LDB) is a circumferential chest compression device composed of a pneumatically actuated constricting band and backboard.
- Mechanical (Piston) CPR.
- Lund University Cardiac Arrest System CPR—The Lund University Cardiac Arrest System (LUCAS) is a gas-driven sternal compression device that incorporates a suction cup for active decompression.
- Phased Thoracic-Abdominal Compression-Decompression CPR—Phased thoracic-abdominal compression-decompression (PTACD) CPR combines the concepts of IAC-CPR and ACD-CPR.
- Minimally Invasive Direct Cardiac Massage—Minimally invasive direct cardiac massage (MIDCM) involves insertion of a plunger-like device through a small incision in the chest wall to enable direct compression of the heart.

Many of these devices and techniques have shown promising results both in laboratory and human studies. However there is insufficient data to support it's use for routine resuscitation.

Describe the rate and technique of chest compression for infants and children?

If there is no pulse or pulse is < 60 bpm despite oxygenation and ventilation begin chest compressions. While giving chest compression "Push Hard Push Fast" at a rate of at least 100 times per minute to depress the chest to 1/3 AP diameter or 2 inches (5 cms) in children and 1½ inch (4 cm in infants). Release completely to allow full recoil of the chest. Minimize interruptions to chest compression. Compression ventilation ratio 30:2 (one rescuer) and 15:2 (two rescuers).

In Infants

a. 2-finger chest compression technique (lone rescuer)– compress the sternum with 2 fingers placed below the intermammary line.

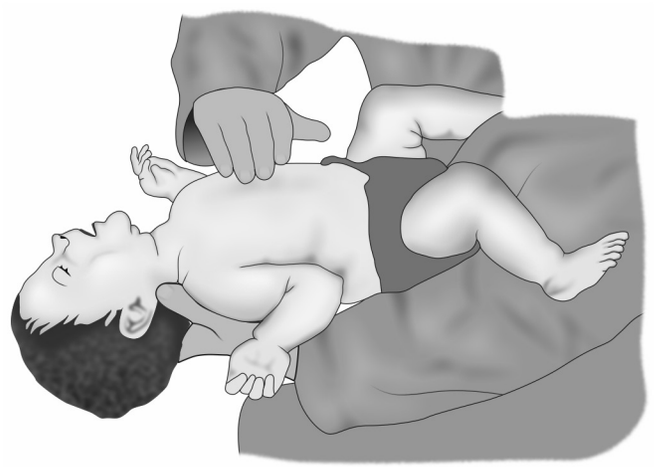


Fig. 47.1 Two-finger chest compression technique in infant (1 rescuer)

b. 2-thumb encircling technique (2 rescuers) – encircle the infant's chest with both hands, spread your fingers and place your thumbs together over the lower-half of the sternum and forcefully compress the sternum with your thumb.

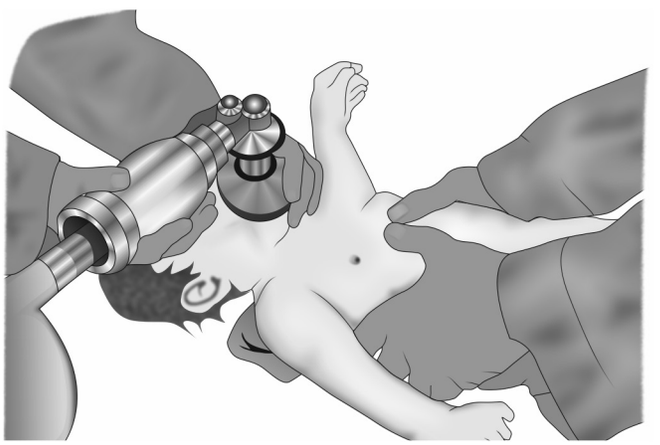


Fig. 47.2 Two thumb-encircling hands chest compression in infant (2 rescuers)

The 2-thumb encircling technique is recommended when 2 rescuers are present. It is also preferred because it produces better coronary perfusion and may generate higher systolic and diastolic pressures.

In child

Compress the lower end of the sternum with the heel of one hand or two hands but should not press on the xiphoid or the ribs.

Enumerate the key differences in between adult, paediatric and infant BLS

Table 47.1 Differences between adult, pediatric and infant BLS

Recommendations			
Component	Adult	Child (1year to puberty)	Infant (<1 year)
Recognition	Unresponsive		
	No breathing or no normal breathing (i.e. only gasping)	No breathing or only gasping	
	No pulse palpated within 10 seconds		
CPR Sequence	C-A-B		
Compression technique	Heel of both hand	Heel of one hand	2 finger chest compression technique/2 thumb encircling technique
Compression rate	At least 100/min		
Compression depth	At least 2 inches (5 cm)	At least 1⁄3 AP diameter About 2 inches (5 cm)	At least 1⁄3 AP diameter About 1 1/2 inches (4 cm)
Chest wall recoil	Allow complete recoil between compressions rotate compressors every 2 minutes		
Compression interruption	Minimize interruptions in chest compressions limit interruptions to <10 seconds		
Airway	Head tilt–chin lift (suspected trauma: jaw thrust)		
Compression-to-ventilation ratio (until advanced airway placed)	30:2 1 or more rescuers	30:2 Single rescuer 15:2 2 HCP rescuers	
Ventilations with advanced airway	1 breath every 6-8 seconds (8-10 breaths/min) Asynchronous with chest compressions About 1 second per breath Visible chest rise		
Defibrillation	Attach and use AED as soon as available. Minimize interruptions in chest compressions before and after shock; Resume CPR beginning with compressions immediately after each shock.		
	Biphasic -120-200 J Monophasic: 360 J	2-4 J/kg. first and 4 J/kg for subsequent shocks, higher energy may be considered but not to exceed 10J/kg	

Which are the common causes of Cardiac arrest under anesthesia?

Cardiac arrest can occur during induction, maintenance, intra-operatively or in postoperative period. It is more common during induction and reversal.

Cardiac arrest can occur anytime during the course of the anesthetic. The causes are related to:

1. Airway:
   a. "Cannot Ventilate Cannot Intubate" is a dreaded complication. Rescue airway devices should be immediately used.
   b. Many airway-related problems may occur during the course of anesthetic like blocked tube, disconnections, displacement of tube. Continuous $EtCO_2$ is helpful in recognizing these problems early.
   c. Premature extubation if unrecognized.
   d. Foreign body: Any unrecognized foreign body in the oropharynx, e.g. throat pack, dislodged tooth.
2. Breathing:
   a. Laryngospasm at induction or post-extubation.
   b. Intra-operative/postoperative bronchospasm when severe can cause hypoxia and bradycardia leading to cardiac arrest.
   c. *Tension pneumothorax:* Patients with pre-existing bullae or emphysema are at risk.
   d. *Aspiration:* Can occur at induction or post-extubation.
   e. *Residual anesthetic effect:* Residual narcotic/inhalation effect or residual paralysis can lead to hypoventilation and hypoxemia.
   f. *Pulmonary edema:* Due to fluid overload/aspiration.
3. Circulation:
   a. *Embolism:* Pulmonary thromboembolism, e.g. Air (laparoscopic, neurosurgery), Fat (rimming of long bones), Amniotic fluid (at placental separation), tumor (cancer surgeries). Continuous $EtCO_2$ and BP monitoring is helpful in recognizing the problem.
   b. Perioperative Myocardial Infarction.
   c. Massive uncontrollable perioperative blood loss.
   d. *Vasovagal:* Generally occurs in young individuals with high vagal tone.
   e. Patients for emergency surgery with severe cardiac depression in SIRS/sepsis.
4. Drug induced:
   a. Succinylcholine induced hyperkalemia.
   b. *Anaphylaxis:* To drugs/fluids.
   c. Accidental intravascular or intrathecal injection of local anesthetic.

What is the mechanism of cardiac arrest under Spinal Anesthesia?

Decrease in preload due to vasodilatation causes reflex severe bradycardia. In patient with high vagal tone this severe bradycardia leads to cardiac arrest.

What is the treatment of postspinal bradycardia?

Atropine 0.4–0.6 mg is first drug of choice. Glycopyrrolate is ineffective, hence not recommended.

If bradycardia is not reverted with atropine; ephedrine (25–50 mg), or epinephrine (0.2–0.3 mg) may be considered.

If bradycardia leads to cardiac arrest, follow CPR flow chart.

What are the causes of cardiac arrest in pediatric patient?

Cardiac causes predominantly hypovolemia from blood loss is one of the causes of cardiac arrest in children especially in patients of spinal fusion or craniotomy. Hyperkalemia of transfusion is another cause. Halothane is sometimes implicated.

Severe Laryngo-spasm can sometimes lead to cardiac arrest in children.

Suggested Reading

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48 Airways, Connectors, Laryngoscopes and Non-Rebreathing Valves

N Amin

Airways

Define an airway.

An airway is a device which when inserted either into the oropharynx or nasopharynx, helps to maintain the patency of the air passage for unobstructed breathing.

There are various maneuvers like chin lift, jaw thrust and tracheal intubation which help to maintain a patent airway. However, unlike these maneuvers insertion of an airway does not affect the stability of the cervical spine.

How are airways classified?

Airways are classified as:

1. Oropharyngeal airways
2. Nasopharyngeal airways
3. Modified airways
   a. Laryngeal mask airway
   b. Cuffed oropharygeal airway.

What are the uses of airways?

1. Prevent obstruction of the upper air passage; by lifting the tongue and epiglottis away from the posterior pharyngeal wall.
2. Prevent biting of and occlusion of the orotracheal tube.
3. Protect the tongue during biting and seizure activity.
4. Facilitate oropharyngeal suctioning.
5. Provide a better mask fit for ventilation.
6. Help insertion of tubular devices (suction catheters, ryles tube) into pharynx or esophagus.

Give a brief description of an oropharyngeal airway.

It is a suitably curved apparatus made of metal, plastic or hard rubber. There is a flange at the proximal end which can be used to fix the airway. The flange also prevents the airway from moving deeper into the mouth. The bite portion is straight and firm so that the patient cannot close the air channel by biting hard. When an appropriate sized airway is in place; the bite portion lies between the lips and teeth and the flange lies outside the lips. The pharyngeal end is the curved portion of the airway that extends upwards and backwards to correspond to the shape of the tongue and palate. It separates the tongue from posterior pharyngeal wall and it also pulls the epiglottis slightly forwards by exerting pressure along the base of the tongue. When inserted the distal end should lie just above the epiglottis so as to not irritate the laryngeal inlet. The cross-section of an oropharyngeal airway is either oval or circular in shape. These airways are available in various lengths and diameter so that they can be used in a neonates as well as adults.

How do you estimate the appropriate size of an oropharyngeal airway? Describe the insertion technique.

An estimate of the appropriate sized airway is obtained by selecting an airway with the length corresponding to the vertical distance between the patient's incisor and the angle of the jaw. If the airway is too small it may cause kinking of the tongue and obstruct the movement of gas. If an airway is too large it may cause trauma to the larynx or it may displace the epiglottis and cause airway obstruction.

Insertion technique

Open the patient's mouth and insert a well-lubricated airway into the oral cavity with it curvature facing the upper lips. Once it is halfway down it is rotated to its normal position and advanced further until the distal end lies in the oropharynx. This rotation technique minimizes the chance of pushing the tongue backwards and downward. Jaw thrust can be given to overcome any resistance during insertion. Another method of insertion is to depress the tongue with a tongue

depressor and insert the airway with it's concavity facing the tongue. The flange is pushed to slide the airway behind the tongue. If the patients jaw is not supported it may cause the airway to be pushed out. However, if the airway repeatedly comes out it should be replaced with a smaller-sized airway. Correct placement is shown by improvement in airway patency and by seating of the flattened reinforced section between the patient's teeth or alveolar margins if the patient is edentulous.

Give a brief description of the various types of oropharyngeal airways.

Guedel airway (Sizes – 0 to 6)
Description: It is made up of rubber. It is a hollow oropharyngeal airway with a large flange and a reinforced bite portion. It has a gentle curve that follows the contour of the tongue. It has a tubular channel for air exchange and suction. It is most frequently used.

Sterilization: Cleaning with soap and water. However, repeated sterilization may cause softening.

Problems: Latex allergy. Softening may allow the lumen to be occluded and not prevent the tube from biting.

Waters airway: It is named after Ralph Waters. (Sizes – 00, 0,1 to 7).

Description: It is a metallic oropharyngeal airway with two holes at the pharyngeal end. It is hollow and has a right or left nipple on the flange for attachment of oxygen line and suction.

Sterilization: Cleaning with soap and water.

Advantages: It is cheap, reusable and easy to store.

Connell airways (Sizes – 00, 0, 1 to 7).

Description: It is a metallic hollow oropharyngeal airway. It is similar to waters airway but without the insufflation nipple.

Berman airway (Sizes – infant, small child, child, medium adult, large adult).

Description: It has a flange at the buccal end. It has no enclosed air channel. The sides are cut-open and there is support through the center (H-shaped in cross-section) through which a tracheal tube can be passed. The sides allow passage of suction catheters and provide air channels. The center may have openings in it to permit suction if the airway becomes lodged sideways.

Advantage: It is easier to clean and is less likely to become obstructed with foreign body or mucus.

Which airways can be used for resuscitation if you do not have manual resuscitator? Can you describe any one of them?

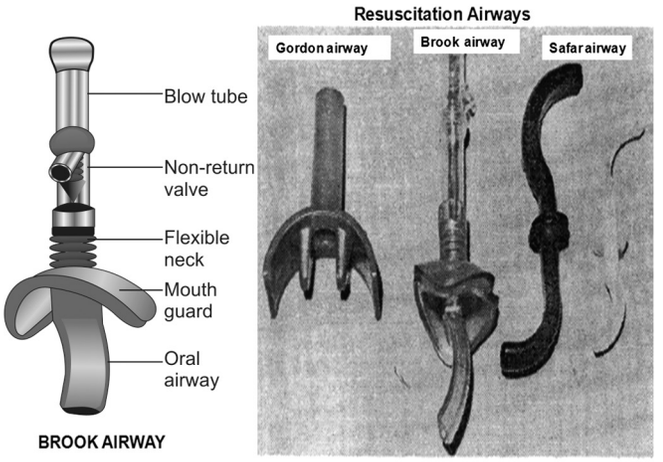


Fig. 48.1 Various types of airway

Safar airway (Sizes – Adult and pediatric)
Description: In 1958, Safar and McMahon described an S-shaped oropharangeal airway which consisted of two Guedel-type airways soldered together. It is a new and aesthetic modification for mouth-to-mouth artificial respiration. A smaller version is available for use in infants and children. It is made of non-traumatic soft rubber.
Use: Mainly for artificial resuscitation.

Brook Airway

This airway was devised by Morris Brook in 1957. Like Safar he started with a Guedel airway to which he attached a mouth guard flap which fit snugly over the patient's lips. A flexible rubber neck with a straight plastic tube was attached to the mouth guard. The straight tube had a built in spring one way valve which allowed air to be blown into the tube but at the same time the expired air from the victim would escape via the valve through a side exit. The advantage of Brook's airway was the flexible neck which allowed the tube to be angled if necessary. A suction channel with an attached suction-bulb has been added in the later models.

Which airways are used as an aid for fibreoptic intubation?

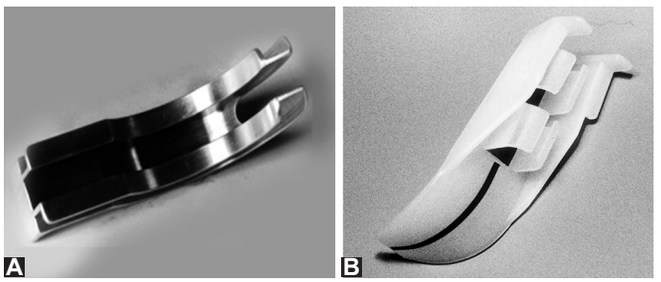
Ovassapian Fiberoptic Intubating Airway: It is designed for use during fibreoptic intubation.
Description: The proximal end is flat and narrow and functions as a bite block. The distal end gradually widens. It has 2 vertical side walls at the buccal end. Between the side walls

there are 2 pairs of curved guide walls which allow the passage of a tracheal tube up to 9 mmID. The guide walls are flexible so that the airway can be removed from around the tracheal tube after intubation has been completed. The proximal end is tubular so that it can function as a bite block. Since there is no posterior wall in the distal half of the airway, it is easy to maneuver the fibreoptic scope in the oropharynx. Also during fibreoptic intubation it is not necessary to remove the tracheal tube connector when using this airway.

Patil-Syracuse oral airway or Patil endoscopic airway

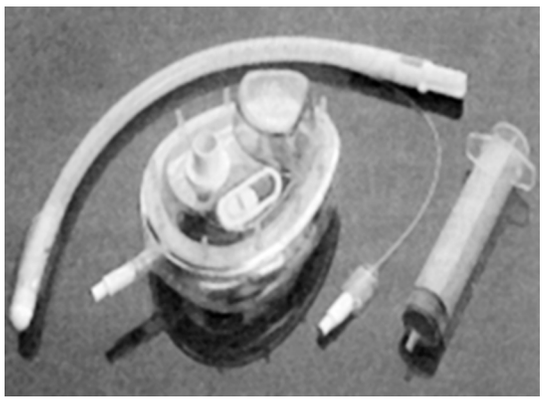
It was designed as an aid to fiberoptic intubation.

Description: It has suction channels at the sides. On the lingual surface there is a grove at the center which allows the passage of the fiberscope and guides it in the midline. The distal end has a slit which allows the fiberscope to be manipulated in the anteroposterior direction.



Figs 48.2A and B (A) Patil-Syracuse oral airway (B) Ovassapian airway

Esophageal obturator airway



Figs 48.3 Egophageal obturator airway

This airway has a face mask at the proximal end, which fits over the mouth and nose of the victim. There is a blowing device in the center of the facemask through which air or oxygen can be blown during artificial respiration. This blowing tube continues below as a curved and elongated hollow tube with a blind end. There is an esophageal balloon just before the blind end. The balloon needs to be inflated with 10–20 mL of air to obliterate the esophagus and prevent regurgitation and aspiration. There are multiple small holes along the walls of the tube. The air that is blown through the tube passes through these holes to enter the patients lungs.

Use: This is used for the artificial resuscitation.

Which oropharyngeal airways can be used for IPPV?

Cuffed oropharyngeal airway and laryngeal mask airway can be used for IPPV.

What are the advantages of a nasopharyngeal airway over an oropharyngeal airway?

Nasal airway is better tolerated in a semi-awake patient than an oral airway and is less likely to be accidentally displaced or removed.

They offer an alternative to the oral airway when the patient has limited mouth opening, awkward or fragile dentition, trauma or pathology of oral cavity, or where oral airway is frequently displaced by marked overlapping bite.

They have also been used to aid in pharyngeal surgery, to reduce trauma while passing fiberoptic bronchoscope, to apply CPAP, to facilitate suctioning and to help in airway management of a patient with Pierre-Robin syndrome.

Give a brief description of a nasopharyngeal airway.

Nasopharyngeal airways are made of soft plastic, polyurethane or latex rubber. At its proximal end there is either a fixed or adjustable flange which limits insertion of an excessive length so that the device lies just above the epiglottis. The tube is curved to fit the curvature of the nasopharynx. A beveled distal-end makes its passage through the nose less traumatic. Sometimes there may be a hole cut into the wall opposite the bevel which ensures airway patency even if the bevel becomes blocked with mucus during insertion. It is available in a range of lengths and internal diameters.

How would you choose an appropriate size nasopharyngeal airway? Describe the insertion technique.

The length of the airway needed for the patient can be estimated 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.

Insertion Technique

Before insertion it should be lubricated along its entire length. Use of vasoconstrictors before insertion of the airway may not always be beneficial. Check the patency of the right nostril. The airway is held in the hand on the same side as it is to be inserted. It is inserted through the nares, bevel end first. It is

passed vertically along the floor of the nose with slight twisting action. The curve of the airway should be directed towards the patients feet. If resistance is encountered the airway should be redirected. If excessive resistance is encountered the other nostril or smaller airway should be used. When properly placed it extends from the nose to the pharynx with the pharyngeal end above the epiglottis and below the base of the tongue and with the flange just outside the nostrils.

What are the various types of nasopharyngeal airways that you know of?

Bardex airway: It is made of rubber. It has a large flange at the nasal end and a bevel at the pharyngeal end.

Rusch airway: It is made of red rubber. It has a short bevel at the pharyngeal end and an adjustable flange at the nasal end.

Saklad's airway: It is almost like Bardex airway, except that it does not have a circular flange in the proximal end, making it vulnerable to slip into the oropharynx.

Linder nasopharyngeal airway: It is a clear plastic airway with a large flange. The distal end is flat rather than beveled. The airway is supplied with an introducer which has a balloon. A luer lock syringe attached to a one way valve at the other end of the introduce can be used to inflate or deflate the balloon.

Binasal airway: This airway consists of 2 nasal airways joined together by a connection that has a15 mm adaptor for attachment to the breathing system. The tube is positioned so that the distal tips are near the larynx just below the tip of epiglottis. After the airway is inserted the soft tissues usually seal the hypopharynx permitting assisted or controlled respiration.

What are the precautions to be taken during the insertion of an airway?

- Pharyngeal and laryngeal reflex should be depressed to prevent coughing and laryngospasm
- There should be selection of correct size
- The airways should be well lubricated before insertion.

What are the problems that can arise during the use of an airway?

- Airway obstruction – too large an airway, foreign body may be lodged in the air channel
- Airway can be compromised due to epistaxis resulting from trauma to the septal mucosa, nasal polyps or adenoidal tissue during insertion of nasopharyngeal airway
- Trauma to the tongue, nose, uvula, pharynx and teeth
- Long oropharyngeal or nasopharyngeal airway can cause laryngospasm
- Insertion of oropharyngeal airway in light planes of anesthesia can result in gagging or retching
- Latex allergy.

How are the airways sterilized?

Airways are cleaned with soap and water.

Connectors

What is the difference between a connector and an adaptor?

A connector is a fitting intended to join together two or more components. An adaptor is a specialized connector that establishes functional continuity between otherwise disparate or incompatible components. An adaptor or a connector may be distinguished by:

- Shape (straight, right angle or elbow, T or Y)
- Components to which it is semi-permanently attached
- Added feature (with nipple or pop-off)
- Size and type of fitting at either end (15 mm male, 22 mm female).

All anaesthesia systems terminate at the patient end by connection to a mask or endotracheal tube adaptor. All face masks, both adult and paediatric, have a 22 mm female and a 15 mm male fitting.

Hence connectors should have:

- 15 mm male fitting at the machine end and either
- 22 mm male/15 mm female fitting for connection to either tracheal tube adaptor or a mask at the patient end
- 15 mm female fitting for connection to a tracheal tube adaptor.

Often this component is a right-angle connector and may be known as elbow adaptor, elbow joint, elbow connector, face mask angle piece, mask adaptor or mask elbow.

What are their uses?

- To extend the distance between the patient and the breathing system (head and neck surgery).
- To change the angle of connection between the tracheal tube and the breathing system.
- To allow a more flexible and/or less kinkable connection between the tracheal tube and the breathing system.

What are the principles to be kept in mind while selecting a connector?

While selecting a connector, several principles should be kept in mind. These are:

- Connectors increase dead space (significant in infants).
- Addition of a connector increases the number of possible locations at which at which disconnections can take place.

- Connectors with sharp curves and rough side walls (Cobb's suction union and Rowbotham adaptors) can markedly increase the resistance.
- Straight connectors offer less resistance than corrugated connectors; hence to minimize the resistance endotracheal tube adaptors should preferably be straight or gently curved.

Describe a tracheal tube connector?

It is a 15 mm male to female ISO tapered connection used to connect the endotracheal tube to the components of the breathing system. The male connector has a tapered cone which is pushed into the lumen of the endotracheal tube. While fitting the connector the tube material has to be stretched as it is slightly bigger than the tube. This helps to produce a more secure connection.

What is the difference between adult and pediatric connectors?

Prior to any standardization, the sizes of the connector depended on the manufacturer (Magill connectors,13 mm; Worcester, 12 mm; Metal Cardiff, 12 mm; Knight, 9 mm; Bennett,14 mm). However, subsequently a 15 mm ISO connection was standardized for use in adults and large children. Recently, 8.5 mm connectors are available for endotracheal tube sizes 2–6 mm ID as the standard 15 mm ISO connector may not be suitable for use in infants and neonates. However, these (8.5 mm) connectors will not fit our standard 15 mm ISO connector.

What are the ISO recommendations for tapered connections or adaptors?

Tapered connections or adaptors are used to join rigid tubes or other components in such a manner that the joint will not leak. This joint has a male-half and a female-half which is pushed together with a slight twist to form a gas tight fit. These connectors and adaptors should be easy to dismantle and reassemble.

Current ISO recommendations are:

- 30 mm tapered connections for attachment of scavenging hose to breathing systems.
- 22 mm tapers for connection within the breathing system.
- 15 mm connection for attachment of breathing system to an endotracheal tube.

How would you clean and sterilize the connectors?

Cleaning: After use connectors should be rinsed under a running tap, then placed in a solution of detergent and water and soaked.

Disinfection and/sterilization: Rubber and plastic connectors may be sterilized with ethylene oxide or in a liquid such as gluteraldehyde. Metal connectors may be boiled, autoclaved or pasteurized.

Laryngoscopes

How would you define a laryngoscope?

Laryngoscopes are devices that have been designed to visualize the interior of the larynx including the vocal cords so as to aid endotracheal intubation.

History: 1895 Alfred Kirstein developed direct vision laryngoscope. It was popularized by Sir Robert Mackintosh and Sir Ivan Magill in the early 1940.

What are the uses of a laryngoscope?

- For Intubation
- Foreign body removal
- Upper airway lesion biopsy
- Visualization of vocal cord and larynx
- For insertion of nasogastric tube.

Can you briefly describe the parts of a laryngoscope?

Its parts consist of the following:

Handle: Its function is to apply suitable leverage to the blade. It has a rough surface for traction and usually houses the batteries for the light source. Handles designed to accept blades that have a light bulb with a metallic contact. When the handle and blade are engaged in the operating position it completes an electrical circuit. Handles are available in several sizes. A narrow handle is useful while intubating a small patient. Short handles are useful in patients in whom the chest and/or breasts contact the handle during use, or if cricoids pressure is being given.

Fitting: The connection point between the handle and the blade is called the fitting. The hook on fitting is most commonly used. Another type of fitting employs a pin which fits through holes in the blade and handle. When ready to use most blades form a right angle with the handle, but they may also form acute (Guedel, Bennett) or obtuse (Polio, Bowen-Jackson) angles. An adaptor may be fitted between the handle and blade to allow the angle to be altered. The Patil-Syracuse handle can be positioned and locked in 4 different positions.

Blade: It is a rigid component that is inserted into the mouth. It helps to elevate the lower jaw and tongue. The blade further consists of:

- *Base:* It is the part that attaches to the handle. It has a slot for engaging the hinge pin of the handle.
- *Heel:* The proximal end of the base is called the heel.
- *Spatula (tongue):* It is the main shaft. It serves to compress and manipulate the soft tissues and the lower jaw so that a direct line of vision to the larynx is achieved. The long axis of the tongue may be straight or curved in part or all of its length.

- *Flange:* It is parallel to the tongue and is connected to it by the web. It serves to guide instrumentation and deflect interfering tissues. The flange determines the cross-sectional shape of the blade.
- *Tip (beak):* It contacts either the epiglottis or vallecula and directly or indirectly elevates the epiglottis. It is usually blunt and thickened to decrease trauma.
- The light source is a bulb screwed on to the blade. The electrical conduit runs at the back of the blade. An electrical connection is made when the blade is opened and the light comes on and the laryngoscope is ready for use. Most of the time the light source is near the tip of the blade, but in some blades it is in the base of the blade.

What have been the recent advances in the laryngoscope design?

Some designs place the bulb in the handle and the light is transmitted to the blade by means of fibreoptics. The fibreoptic bundle may be an integral part of the blade or it may be detachable. The light intensity is greater than that provided by the regular light bulb. As there is no bulb or electrical contact in the blade cleaning and sterilization are easier and the laryngoscopes are more reliable. Even if the light is left on for extended period of time, the blade stays cool. Also there is no danger of the bulb getting detached and acting as a foreign body. Rechargeable nickel cadmium batteries are becoming popular as power source for laryngoscopes. Xenon gas filled bulbs have brighter light than conventional tungsten type bulbs; however, they require power from alkaline batteries to work efficiently.

Which is the most commonly used laryngoscope? Is there a standard laryngoscope for all patients?

The Macintosh laryngoscope is most commonly used. The spatula has a smooth gentle curve that extends from the base to the tip. There is a flange at the left to push the tongue out of the way. In cross-section the spatula and flange form a reverse 'Z'. Its tip is blunt and hence causes lesser trauma to the tissues. It has 4 sizes, i.e. infant, child, medium adult, and large adult. Its blades are interchangeable.

There is no standard laryngoscope for all patients. Depending on the indication; various laryngoscope handles and blades can be used. There are different laryngoscopes for adults and pediatric patients. Also the laryngoscope blades come in different sizes.

What are the unique features of laryngoscopes used for difficult intubations?

McCoy Laryngoscope

It is based on the standard Macintosh blade with the addition of an adjustable tip that is operated by a lever on the handle. It is inserted in the normal way and if the larynx is not clearly visualized then the tip can be flexed so that it elevates the epiglottis. Less force is required to bring the larynx into view. The point on the blade which acts as a fulcrum; is moved further into the pharynx; thereby eliminating any inadvertent contact with the upper teeth. It is suitable both for routine use and in cases of difficult intubation. This laryngoscope may use either a traditional bulb in the blade or a fiberoptic light source.

Bullard Laryngoscope

The blades are designed so that varying degrees of jaw protrusion and upper neck extension will bring either a part or the entire larynx into direct view. It has a curved blade which is designed to pass behind the tongue and lift it away from the posterior pharyngeal wall without any neck extension. The fiber optic viewing channel and light source incorporated in the blade helps in the visualization of the larynx by indirect laryngoscopy.

The blade may be passed anterior to the epiglottis as with a Macintosh blade or posterior to it as with a straight blade. A suitable endotracheal tube is preloaded on a wire fitted to the side of the device and runs parallel to the blade. The fibreoptics allow the visualization of the tip of the guide as it has a slight bend. It also allows the visualization of the tube as it enters the larynx. Intubation is easier with a reinforced tube. This laryngoscope is useful in patients with limited neck movement and/or restricted mouth opening. Both adult and pediatric sizes are available.

Which laryngoscope would be useful in obese patients?

Polio Blade

It is also a modification of the Macintosh blade. The blade is offset from the handle at an obtuse angle to allow intubation of patients in iron lung respirators or body jackets, after anesthesia screen is in place, obese patients, patients with large breasts, kyphosis with severe barrel chest deformity, short neck or restricted neck mobility.

Disadvantage: Little force can be applied and control is minimal.

What is an Oxiport Macintosh?

It is a conventional Macintosh blade with a tube added to deliver oxygen.

What are the uses of a left handed makintosh laryngoscope blade?

The left handed Macintosh laryngoscope blade has a flange on the opposite side from the usual Macintosh blade. This blade is useful for abnormalities of the right side of the

face (e.g. cleft palate) or oropharynx, left-handed persons, intubating in the right lateral position and positioning a tracheal tube directly on the left side of the mouth.

Which laryngoscope is useful in patients with limited mouth opening?

Bizarri-Guiffrida Blade

It is a modified Macintosh. The flange is removed except for a small part that encases the light bulb so that space required to insert the blade in the mouth is minimum. This was done in an attempt to limit damage to the upper teeth. It was designed particularly for patients with limited mouth opening, prominent incisors, receding mandible, short thick neck or anterior larynx.

Can you describe the laryngoscopy technique?

Laryngoscopy consists of several steps: positioning the head, opening the mouth, inserting the blade, identifying the epiglottis and finally viewing the larynx.

Position for Laryngoscopy (2 positions)

Optimal position for most patients is flexion of lower cervical spine and extension of the head at atlanto-occipital joint (sniffing position). The lower part of the cervical spine can be maintained in a position of flexion by means of a small pillow under the head. Extension at the atlanto-occipital joint is achieved by pressure on top of the head and/or upward traction on upper teeth or gums by the laryngoscopist's hand.

Head and neck in full extension (Chevalier Jackson position): In children, it may be unnecessary to flex the lower cervical vertebrae, and in neonates it may be necessary to elevate the shoulders.

The laryngoscope is held in the left hand. The fingers of the right hand are used to open the mouth and spread the lips apart. The blade is inserted at the right side of the mouth. This reduces the likelihood of damage to the incisors and helps push the tongue to the left. The blade is advanced on the side of the tongue towards right tonsillar fossa, so that the tongue lies on the left side of the blade. The right hand keeps the lips from getting caught between the teeth or gums and the blade. When the right tonsillar fossa is visualized, the tip of the blade is moved towards the midline. The blade is then advanced behind the base of the tongue, elevating it, until the epiglottis comes into view. The jaw and the tongue are lifted away from the posterior pharyngeal wall (taking care not to lever on the front teeth) to expose the larynx and epiglottis. This maneuver creates a space that is larger on the right hand side for insertion of the endotracheal tube. There are 2 methods for elevating the epiglottis depending whether a straight or curved blade is being used.

Straight Blade

After exposure of the epiglottis, the blade is withdrawn slightly to scoop under the epiglottis and lift it forwards. If the larynx is not visualized downward external pressure on the larynx may be useful. If the laryngoscope is advanced too far, it will override the slanting glottis and enter the esophagus. If it is then withdrawn too far, the tip of the epiglottis will be releazed and will flop down over the glottis. Straight blades are useful in infants and children. It is also useful in adults with long floppy epiglottis.

Curved Blade

When the epiglottis is seen, the blade is advanced till the tip fits into the vallecula. Traction is then applied along the handle, at right angles to the blade to carry the base of the tongue and epiglottis forward. If the glottis is not seen then external pressure on the larynx may be helpful. The handle should not be pulled backwards as it will cause the tip to push the larynx upwards and out of sight and could cause damage to the teeth and gums.

Can you enumerate the complications of laryngoscopy?

- Damage to the teeth, gums and dental prosthesis.
- Bruising and laceration of lips and tongue, abrasion and laceration of hard and soft palate, the posterior wall of pharynx, epiglottis and valecullae, and blunt trauma or lacerations of larynx and/or esophagus. The risk of trauma and bruising is higher with the straight blade. Lingual nerve may be injured.
- Injury to cervical spinal cord.
- Changes in the cardiac rhythm and blood pressure are common during laryngoscopy.
- Swallowing or aspiration of a foreign body (e.g. bulb).
- *Burns:* Malpositioning of blade on handle can produce a short circuit, which leads to rapid heating of handle.
- *Laryngoscope malfunction:* Most common is failure of the light to illuminate. This may be a result of defective power source, defective lamp, faulty socket or poor contact between blade and handle.
- Laryngeal spasm.
- Vagal stimulation.

How are laryngoscopes sterilized?

Blades are cleaned with soap and water followed by spirit or chlorhexidine swabbing to disinfect it. Blades can be autoclaved without the light carrier.

Non-rebreathing Valves

Define non-rebreathing valves?

Non-rebreathing valves ensure unidirectional flow of gases. The various components are arranged to ensure that during the inspiratory phase gas flows only into the patient port and during the expiratory phase the exhaled gas escapes through the expiratory port without mixing with the fresh gas stored in the bag.

What are the properties of an ideal NRV?

Ideal NRV should have:

- Minimal dead space
- Minimal resistance
- No forward/backward leak
- Light weight
- Transparent
- Easy to clean and sterilize
- Non-noisy
- Non-sticky
- Compact and inexpensive
- Minimal opening pressure.

What are the advantages of NRV?

The advantages of NRVs are:

- Prevents rebreathing if valve is functioning properly.
- Inspired concentration of gases and vapours can be controlled as there is no mixing.
- Helps dissipation of heat and water vapour.
- Due to low resistance it can be used for spontaneous and controlled respiration.
- Can be used to measure the minute volume. The sum of the fresh gas flows on the flowmeter will be equal to the minute volume if the fresh gas flows are adjusted such that the reservoir bag is 3/4 inflated at the end of expiration.
- Eliminates hazards of carbon dioxide absorption (heat retention, inhalation of irritant dust, exhausted soda lime).
- Compact, light-weight and mobile.
- Inexpensive.
- Dead space is minimal in a properly functioning valve.
- While using NRVs the feel of the bag is a good indicator of compliance as the only distensible parts of the system are the patient's lungs and the reservoir bag.

What are the disadvantages of NRVs?

The disadvantages of NRVs are:

- Wastage of expensive anesthetic gases as relatively large flows is needed for adults. This increases the cost, causes theatre pollution, there is a hazard of explosions when flammable agents are used.
- There is considerable loss of heat and humidity from the patient.
- It is bulky and hence may cause the tube to kink or be displaced downwards.
- Causes theater pollution as scavenging is difficult (except in Laerdel valve).
- Some of the valves are noisy and sticky when wet.
- If the expiratory valve becomes stuck then high pressures may be built up leading to barotrauma.
- Cleaning and sterilization may be difficult.
- During spontaneous respiration frequent adjustments in the fresh gas flows may be required to match the patient's minute volume.
- When used in adult patients, the higher gas flows required may cause increased resistance.
- In some valves the dead space can be relatively high for pediatric patients.
- There is no standardization of the valves.
- If the expiratory valve sticks during spontaneous respiration then it can cause air dilution.
- In some valves both hands are required for assisted/controlled ventilation.
- In some valves if the bag release is delayed then it can cause rebreathing due to backward leak.
- During controlled ventilation in some valves the gas can escape pest the expiratory valve during the initial part of inspiration. This is known as forward leak which can reduce the tidal volume delivered to the patient.
- The feel of the bag is different as compared to other systems.

How would you classify NRVs?

NRVs can be classified depending on the type of respiration and depending on the position of the valve.

Sykes Classification Depending on Type of Respiration

From functional point of view, the main difference between spontaneous and controlled ventilation is the pressure inside the valve during inspiration. During controlled ventilation there is positive pressure inside the valve and during spontaneous ventilation, the patient exerts a negative pressure which is transmitted to the inside of the valve.

Valves designed for spontaneous respiration, e.g.—Stephen Slater valve

During inspiration, the negative pressure exerted by the patient closes the exhalation port. During exhalation the pressure in the valve increases and gas escapes through the exhalation port. If this valve is used for controlled or assisted ventilation, it is necessary to close the exhalation port with a finger during inspiration.

Valves designed for controlled respiration, e.g.—ambu resusitation valve

In this type of valve a pressure increase opens the inlet and closes the exhalation port. If the patient is allowed to breathe spontaneously, he will inspire room air through the exhalation port. It cannot be useful for general anaesthesia.

Valves designed for both spontaneous and controlled ventilation, e.g. Ruben valve, Ambu E, Ambu E2, Ambu Hesse, Laerdal valve and Lewis Leigh valve.

In this type of valve during inspiration the exhalation port is closed and the inlet opens with either controlled or spontaneous respirations. During expiration the exhalation port opens to allow free exhalation and inspiratory port closes to prevent rebreathing. Only dual purpose valves are used in anesthesia.

Depending on the Position

Positional valve works on gravity and hence must be horizontal.

Non-positional valves are closed by elastic tension of rubber or by a spring and may be used in any position, e.g.—flap valve, fish mouth valve, disk type.

How would you describe a non-rebreathing valve?

While describing a NRV first classify it. Then give a brief description of the valve and explain how it functions. What is the dead space and resistance of the valve? Then talk about its advantages and disadvantages. Finally describe how the valve is sterilized.

Give a brief description of the Ruben valve. How does it function?

Classification (a) Ruben resuscitation valve—it is used only for controlled respiration.
(b) Ruben mark 2 dual purpose valve.

Construction: It has a clear plastic body with metal fittings. It is a multi colored valve in which blue color is inlet, gold color is outlet and red color end is patients end. Ruben valve has a spring loaded bobbin within the valve housing. The exhalation port is perforated and has a disk-shaped valve.

Working: In resting position, the spring holds the bobbin away from the expiratory port and against the inspiratory port allowing unhindered exhalation to occur via patient port. When the bag is squeezed, the bobbin is forced across the valve housing to occlude the expiratory port. The inspiratory port is now connected to the patient port and the inspiratory gases enter the patients lungs without leaking out through the expiratory port. In spontaneous respiration, the patient draws air from the inspiratory limb thus forcing the bobbin to move and occlude the expiratory port. When the pressure at the inlet drops, the force of the spring pushes the piston against the inlet. During exhalation the pressure of the exhaled gases causes the disk valve to open. The gas then passes to the atmosphere.

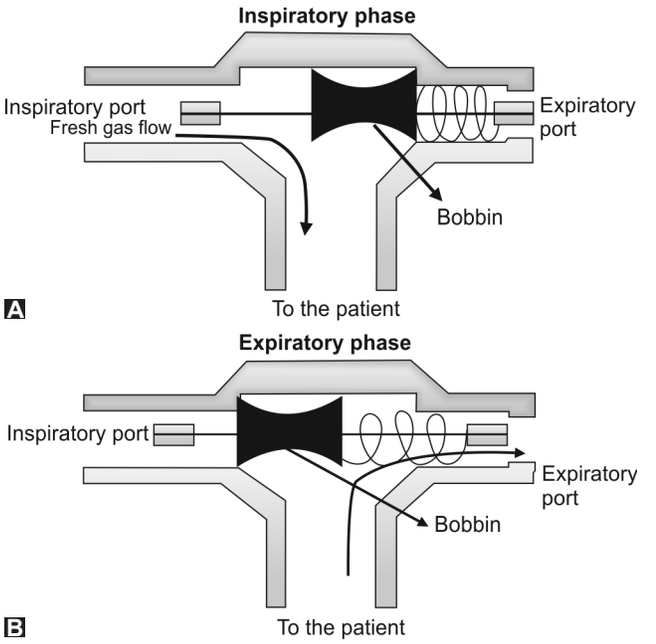
When the valve is to be used with resuscitation equipment the exhalation valve is not present. This allows the patient to inspire from the atmosphere.

Dead space: 9 mL
Resistance: Inspiratory—0.8 cm H_2O at 25 liters/min
Expiratory—1 cm H_2O at 25 liters/min

Disadvantages

- Noisy.
- Cleaning is difficult.
- Presence of backward leak (some exhaled gases may go back to reservoir bag). With fast bag release the backward leak is 10–12% of tidal volume, with slow bag release it is 32–44% and the backward leak could be as high as 76% of tidal volume in patients with increased resistance and decreased compliance.
- If valve sticks in inspiration, high pressure may be formed leading to barotrauma.
- If the valve is not correctly assembled then it can lead to increased resistance and rebreathing.
- If exposed to heat it may deteriorate.

Ruben valve



Figs 48.4A and B Ruben valve. (A) Inspiratory phase; (B) Expiratory phase

Care and Cleaning: Valves can be cleaned by flushing the channels with soap and water. It can also be gas sterilized.

What are the types of Ambu valves available? What are the differences between them?

Ambu Valves

Types (A) For controlled ventilation, e.g. ambu valve.
(B) For dual purpose, e.g. ambu E, ambu hesse, ambu E2.

Ambu valve: This is commonly used with ambu resuscitation bag.

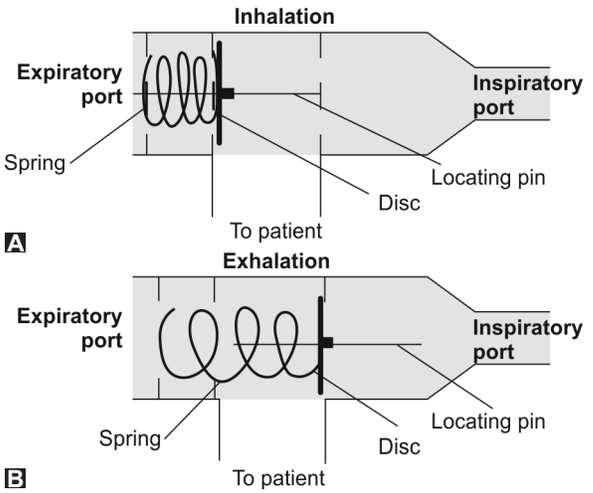
Classification: Controlled respiration.

Construction: Valve is made of metal or plastic material. It has a yellow plastic disc which is the movable part. It is held in place by a spring. A locating pin centers the disk.

Function: During controlled respiration, when the bag is squeezed the force of the gas pushes the disk and closes the exhalation port, thus allowing the patients lung to be inflated. During exhalation the pressure on the inlet side of the disk falls and the spring pushes the disc towards the inlet. The exhaled gases pass from the patient out through the exhalation port. During spontaneous ventilation, the interior of disc does not move and the patient inhales atmospheric air.

Care and Cleaning: It can be disassembled for cleaning. When reassembling it is essential to insert the guide pin through the appropriate channel. The valve should be checked for competence after reassembling.

Ambu valve



Figs 48.5A and B Ambu valve. (A) Inhalation; (B) Exhalation

Ambu E: In this the unidirectional flow of gases is controlled by two labial flap valves.

Classification: Spontaneous or controlled respiration.

Construction: It is made up of transparent plastic. The inlet connection is blue colored. It has 2 labial flaps one for inhalation and one for exhalation.

Working: During spontaneous respiration the negative pressure created by the patient during inspiration closes the valve at the expiratory port, while the fresh gases flow in through the inspiratory port. During expiration, the valve at the inspiratory port closes and the valve at the expiratory port opens so as to allow the expired gases to be voided to the atmosphere. The valve at the expiratory port prevents inhalation of the downstream gases during spontaneous ventilation. This downstream gas may be air when the valve is used as a resuscitator, but it could be expired gases if used in a circle breathing system. During expiratory pause, excess gases may pass outside straight:away as both valves open partially.

When Ambu E is used for controlled ventilation, initially very high pressures are required to produce sufficient movement of the valve at the inspiratory port in order to completely seal the expiratory port. At lower inspiratory flow rates this valve is inefficient because there is incomplete sealing of the expiratory port which allows some of the inspiratory gas to pass straight across the valve resulting in lower than expected tidal volumes.

Dead space: 10 mL

Resistance: Inspiratory resistance is 0.6 to 2.1 cm H_2O at 5 to 40 liters/min, expiratory resistance is 0.6 to 2.5 cm H_2O at 5 to 40 liters/min.

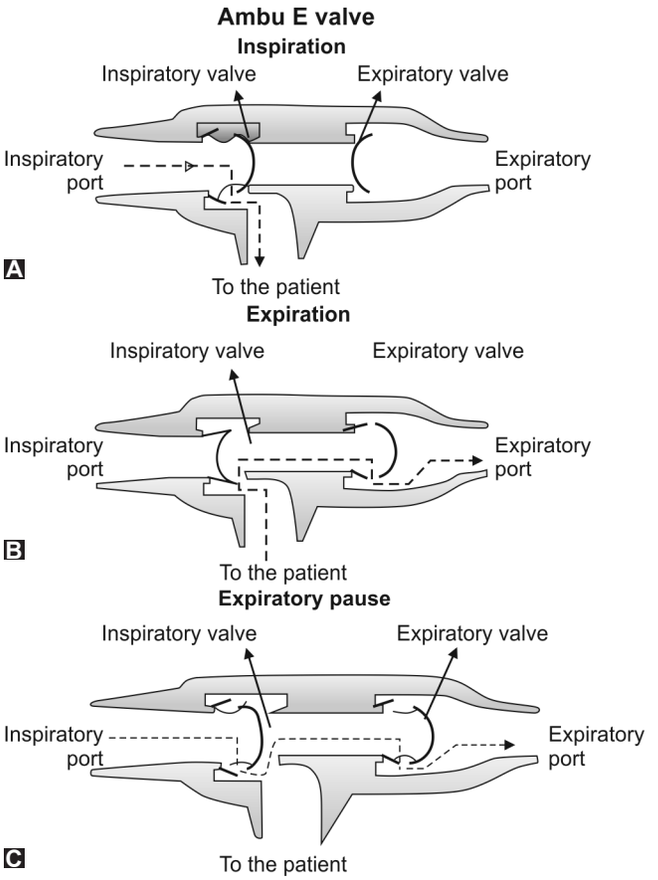
Advantage: Prevents excessive build-up of pressure.

Disadvantage: Cannot be used with resuscitators that do not produce initial high surge of gas required to produce an effective seal. The backward leak is 9% of the tidal volume with slow bag release and 1.8% with fast bag release.

Ambu E2: Contains single unidirectional valve. Has no valve at the expiratory end.

Classification: Spontaneous and controlled respiration.

Working: The negative pressure created by the patient during spontaneous inspiration opens the inspiratory port. It also draws air from the expiratory port. The result is that a mixture of fresh gas and air is inhaled. During controlled or assisted respiration the positive pressure from bag squeezing causes the inspiratory vave to occlude the expiratory port,



Figs 48.6A to C Ambu E valve. (A) Inspiration; (B) Expiration; (C) Expiratory pause

so that fresh gases pass to the patient. During expiration the inspiratory port closes and exhaled gases pass through the expiratory port to the atmosphere.

Dead space: 10 mL

Backward leak is 9% of tidal volume with slow bag releaze and 1.8% with rapid releaze.

Care and Cleaning: Parts can be cleaned with soap and water. The parts may also be boiled, autoclaved or gas sterilized.

Ambu HESSE: It is similar to Ambu E valve, but is slightly larger.

Classification: Spontaneous and controlled respiration.

Construction: This valve overcomes the potential problem of leakage across the Ambu E valve at low flow rates. The valve mechanism has three components ie an inspiratory leaf, an expiratory leaf and a mushroom valve.

Working: During controlled ventilation, manual compression of the self-inflating bag causes a small increase in pressure within the bag. This causes the elastic mushroom valve to expands and totally occlude the expiratory port providing a complete seal. If the bag is compressed further compression it opens the inspiratory leaf valve, forcing gas from the bag into the inspiratory port thus providing inspiratory flow. At the beginning of exhalation phase the self inflating bag starts to re-expand. The reduced pressure within causes the mushroom valve to collapse and the inspiratory leaf valve to close, sealing the bag off from the main valve. The exhaled patient gases then leave the system through the expiratory port via the expiratory leaf valve.

If the valve is used for spontaneous breathing, the negative pressure produced by the patient during inspiration causes the expiratory leaf valve to close, sealing the expiratory port. This ensures that the inspired gas is drawn from the bag and not from the ambient air through the expiratory port. During expiration the valve functions the same as during controlled ventilation.

Dead space: 14 mL

Resistance: Inspiration resistance is 0.2–0.7 cm H_2O at flows from 5–40 L/min, Expiration resistance is 0.2–0.9 cm H_2O at flows from 5–40 L/min.

Backward leak is 7.3% of the tidal volume with slow bag release and 0.9% with rapid releaze.

Care and Cleaning: The valves can be disassembled for cleaning. The parts may be boiled, autoclaved or gas sterilized.

Which NRV is most commonly used and why?

The Laerdal valve is most commonly used. It is light weight, has a transparent body, smallest dead space, minimal resistance to breathing. It can be used for both spontaneous and controlled ventilation. It can be easily dismantled and sterilized.

How does a Laerdel valve work?

Classification: Spontaneous or controlled ventilation.

Construction: It has a clear plastic body and contains a rubber inspiratory fishmouth valve with a circular flange. There is a circular exhalation flap valve which occludes the exhalation port. It is available in three sizes, i.e. adult, child and infant. All sizes have 23 mm external diameter expiratory port and 22 mm external diameter/15 mm internal diameter inspiratory port to minimize misconnections.

Working: In spontaneous respiration the fishmouth valve opens and allows the gas to pass to the patient during inspiration. Simultaneously the expiratory port is sealed as the outer disk portion of the valve is pushed against the apertures in the valve body. The exhalation flap prevents the room air from being inhaled through the exhalation ports. In the expiratory phase,

positive pressure from the elastic recoil of the patient's lungs causes the fishmouth valve to close preventing re-breathing into the bag. It also lifts the outer disk shaped portion of the expiratory apertures thus allowing exhaled gases to escape.

During controlled ventilation the pressure at the inlet opens the fishmouth valve and pushes the disk portion of the flap valve against the exhalation ports during inspiration. This allows the fresh gases to pass to the patient and prevents its loss through the exhalation ports. During exhalation the valve functions the same as with spontaneous respirations.

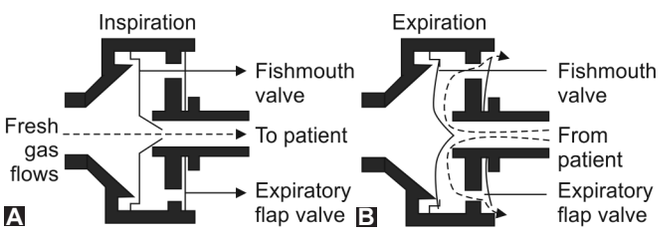
The child and infant models have overpressure safety valves built into the inspiratory port and are set to blow off at a pressure of 45 cm H_2O. If these pressures need to be exceeded, the safety valve can be overridden by finger pressure or a lock clip over the valve.

Dead space: 7 mL

Resistance: Inspiration resistance is 0.32–1.82 cm H_2O for flows from 4.7–44.6 L/min Expiration resistance is 0.16 – 2.83 cm H_2O for flows from 5.0–54.4 L/min.

Care and Cleaning: The valve can be disassembled to clean or repair broken parts. It can be cleaned with chemical solutions, boiling or autoclaving.

Laerdal Valve



Figs 48.7A and B Laerdal valve: (A) Inspiration; (B) Expiration

Give a brief description and working of the Lewis-Leigh valve

Classification: Spontaneous or controlled ventilation.

Construction: It has a clear plastic body with an exhalation chimney which may be rotated 900. There is a disk valve at the top of the chimney and an exhalation valve seat at the bottom of the chimney. The chimney is rotated clockwise for controlled respiration and counter-clockwise for spontaneous respiration. Rotation of the chimney causes changes in the position of the valves which in turn facilitates respiration. This valve is a modification of the DigbyLeigh valve which had a simple disk type exhalation valve instead of the chimney. The exhalation valve had to be closed with a finger during controlled or assisted ventilation.

Working – Controlled ventilation: During inspiration, the gas flows to the patient as the flap valve moves away from its seat on the body and closes over the base of the chimney. At the end of inspiration, when the pressure in the system returns to atmospheric, the flap valve returns to its resting position over the fresh gas inlet. As the patient exhales, the flap valve seats on the ridge and the exhaled gases pass up the chimney lifting the disk valve. Gas then flows to the patient. When the chimney is rotated 900 counter clockwise, the gas passes through the flap valve and travels to the patient during inspiration. The disk valve in the chimney prevents air from being drawn into the valve during spontaneous ventilation. During exhalation the exhaled gases pass into the chimney. Any excess gas can then leak through the valve to atmosphere thereby preventing buildup of pressure inside the valve.

Resistance: The resistance is 0.1 cm H_2O for flows from 3 – 5 L/min.

Backward leak: With fast bag release backward leak is 8–11% of tidal volume and with slow bag release the backward leak is 10–29% of tidal volume.

Care and Cleaning: The valve can be gas-sterilized.

Which valve has the smallest dead space and which valve has the largest dead space?

Laerdal valve has the smallest dead space of about 7 mL and Ambu Hesse valve has the largest dead space of about 14 mL.

Which are the valves with backward leak?

Ambu E, Ambu hesse, Ambu E2, Lewis Leigh and Ruben valves.

Suggested Reading

1. Andrew J Davey and Ali Diba. Ward's Anaesthetic equipment. 5th edition, 2005.
2. Jerry A Dorsch, Susan E Dorsch. Understanding Anesthesia Equipment. 5th edition, 2008.
3. Understanding anesthesia equipment, Dorsch 1st edition.

49 Electrocardiography

R Ambulkar

Describe the various leads used to obtain ECG.

A 12-lead ECG provides a complete picture of the electrical activity of the heart by recording information from 12 different views of the heart.

Limb leads: There are 6 limb leads (Lead I, II and III, aVR, aVF, aVL). Lead I, II and III require a positive and negative electrode and are hence called bipolar leads. aVR, aVF, aVL are augmented unipolar leads as they require only positive electrode. In these leads the small waveforms are enhanced or augmented. The last letter refers to position of the lead.
Chest leads: V1, V2, V3, V4, V5 and V6 are unipolar leads as only positive electrode is required for generating a waveform, the negative electrode being the center of the heart.

Enumerate leads and corresponding regions of the heart.

- *Lead I and AVL:* Left side of the heart.
- *aVF, Lead II and Lead III:* Inferior wall of the heart.
- *V2, V3 and V4:* Anterior surface of the heart.
- *V1 and V2:* Septal area.
- *V5 and V6:* Apical surface.
- *Mirror image changes in leads V1 – V4:* Posterior surface.

Region of heart	Arterial blood supply
Anteroseptal	Left anterior descending artery
Anteroapical	Left anterior descending artery
Anterolateral	Circumflex artery
Inferior	Right coronary artery
Posterior	Right coronary artery

SA node and AV node is supplied by the Right coronary artery in more than 80% of individuals. Patients with inferior wall MI have an increased incidence of developing a complete heart block.

Describe the ECG paper.

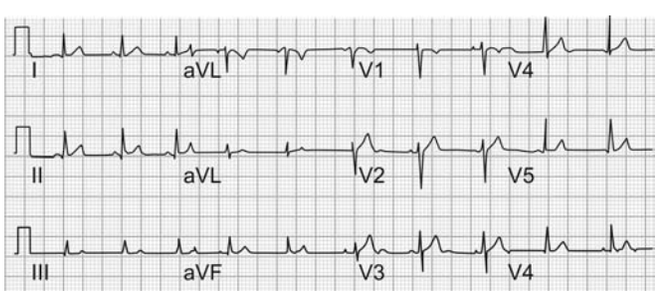
The paper consists of squares—large and small. 5 small squares make 1 large square. 300 large squares or 1500 small squares are covered in a minute of ECG recording (with paper speed of 25 mm/sec). This makes 1 small square = 0.04 sec, 1 large square = 0.2 sec and 5 large squares = 1 sec. Vertical axis represents the voltage and 1 small square in the vertical axis is equal to 0.1 mV at normal settings.

How would you describe a normal ECG by a systematic approach?

Standardized sequence of steps to analyze the ECG are:

- Rate
- Rhythm
- Axis
- P wave
- PR interval
- QRS complex
- ST segment
- T wave
- QT interval
- U wave
- Conclusion.

Normal ECG

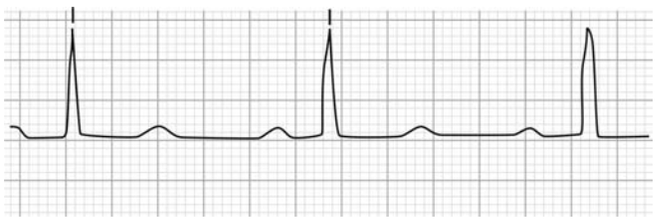


How do you calculate heart rate from ECG?

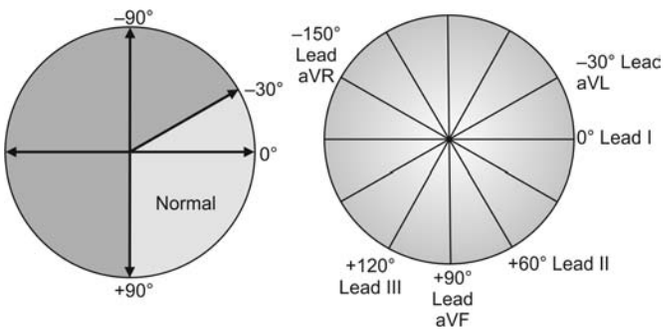
When rhythm is regular one can calculate rate by either:

- Dividing 1500 by the number of small squares between one R-R interval or
- Dividing 300 by number of large squares between one R-R interval.
- When rhythm is irregular one can calculate rate by:
  - Counting the number of R-R intervals in 3 sec (15 large squares) and multiplying by 20.

Example: Heart Rate = 300/4=75



How do you calculate the axis?



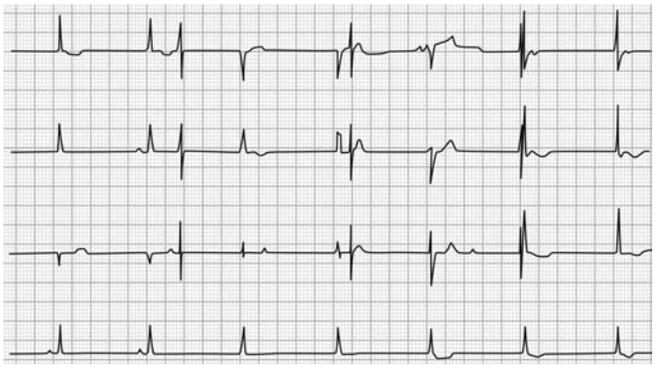
- Normal range – 30° to + 90°
- Left axis deviation – 30° to – 90°
- Right axis deviation + 90° to + 150°

Calculate the net QRS deflection for leads I and aVF (you can also take other two pairs, i.e. II and aVL or III and aVR but stick to these pairs only, do not mix them) by adding up the number of small squares that correspond to the height of the R wave (positive deflection), and subtracting the number of small squares that correspond to the depth of the Q and S waves (negative deflection). Now plot these points on the X and Y-axis, draw perpendiculars from these points and mark a point where these perpendiculars meet. Pass a line from center of circle to this point which will give you the axis.

Other way of calculating axis: examine six limb leads I, II, III, aVR, aVL, aVF

- Locate the lead (X) with equiphasic deflection (the lead with equal forces in the positive and negative direction)
- The axis would run perpendicular to that lead (X)
- Since there are two perpendiculars to each isoelectric lead, chose the perpendicular that best fits the direction of the other ECG leads.

ECG with Normal axis



Explain the Waves and Complexes seen in the ECG

Table 49.1 Waves and complexes seen in ECG

Waves/Complexes		Duration
P wave	It represents the spread of electrical activation (depolarisation) through the atrial myocardium	3 mm in height (0.3 mV) or 3 mm horizontally (0.12 sec)
PR interval	PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. Represents the time taken by the electrical impulse to travel from the sinus node through the AV node to bundle of his	0.12 to 0.2 s
QRS complex	QRS complex represents ventricular depolarization	0.08–0.12s. Voltage varies according to the leads, position of heart, and abnormality
T wave	T wave represents the repolarization of the ventricles	0.12 – 0.16s
U wave	U wave represent repolarization of the papillary muscles or purkinje fibers	
QT interval	QT interval is measured from the beginning of the QRS complex to the end of the T wave. The Q-T interval represents the time for both ventricular depolarization and repolarization (ventricular action potential). QTc- independent of heart rate	QT: 0.2 to 0.4 seconds QTc: <0.44sec
ST segment	It is the time at which the entire ventricle is depolarized and roughly corresponds to the plateau phase of the ventricular action potential	0.08 -0.12s
J Point	J point is the junction between the QRS complex and the ST segment. It is point from where the degree of ST elevation or depression is measured	

What does the p wave signify? What are the abnormalities seen with p wave?

It represents the spread of electrical activation (depolarisation) through the atrial myocardium. Normally does not exceed 3 mm in height (0.3 mV) or 3 mm horizontally (0.12 sec).

Abnormalities

- Absent
  - Atrial fibrillation
  - Sino-atrial block
  - Nodal rhythm
- Inverted
  - Dextrocardia
  - Incorrect electrode placement
- Wide and notched
  - P-mitrale—left atrial enlargement
- Tall and peaked
  - P-pulmonale—in right atrial enlargement

What abnormalities are seen in T wave?

T Wave Inversion

- Q wave and non-Q wave MI
- Myocardial ischemia
- Subacute MI or old pericarditis
- Myocarditis
- Myocardial contusion (from trauma)
- CNS disease causing long QT interval

Tall T wave

- Hyperkalemia
- Post-wall MI.

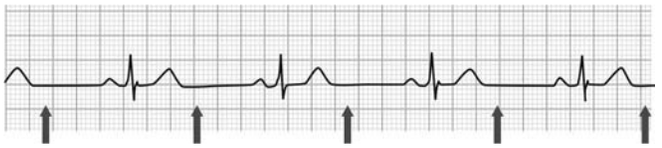
Flat T wave

- Emphysema
- Percardial effusion
- Myxoeedma
- Hypokalemia
- Hypocalcemia.

When do you see prominent U waves?

- Hypokalemia
- Hypercalcemia
- Thyrotoxicosis
- Drugs: Digitalis, epinephrine, and antiarrhythmics
- Intracranial hemorrhage.

An inverted U wave can be found in myocardial ischemia or left ventricular volume overload. See ECG below:



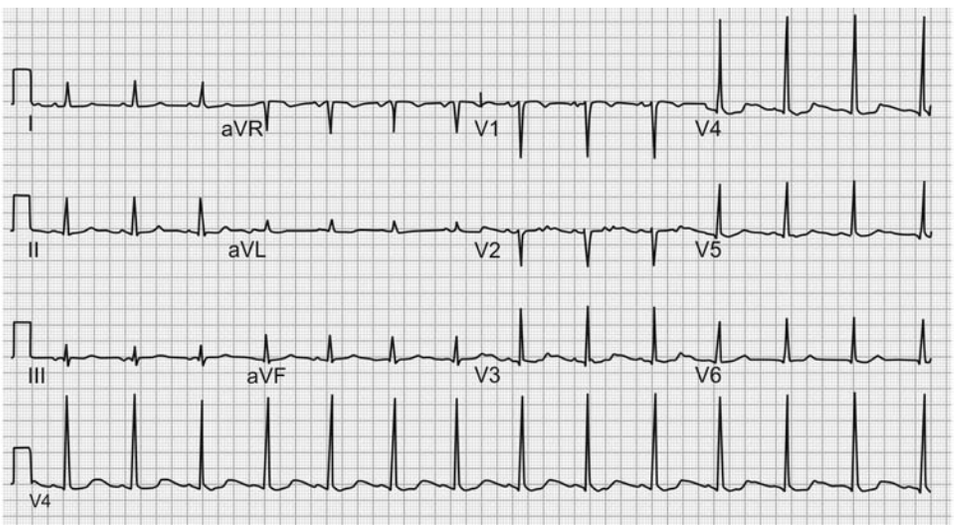
What is the significance of Long QT Syndrome and what are its causes?

- Long QT Syndrome (QTc > 0.47 sec for males and > 0.48 sec in females).
- Significance: Increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death.

Causes

- Drugs (many antiarrhythmics, tricyclics, phenothiazines)
- Electrolyte abnormalities (decreased K^+ or Ca^{++} or Mg^{++})
- CNS disease (especially subarrachnoid hemorrhage, stroke, trauma)
- Hereditary LQTS (e.g. Romano-Ward Syndrome)
- Coronary Heart Disease (post-MI patients).

Example: See the ECG below:



What is corrected QT interval?

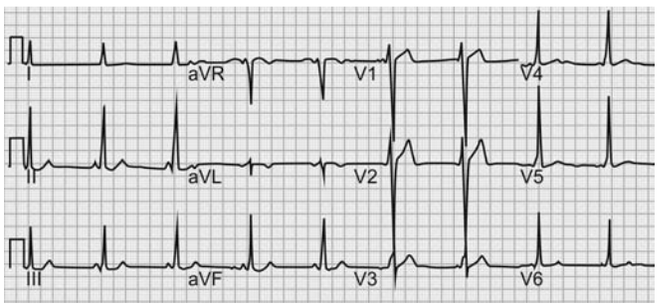
The QT interval is dependent on the heart rate (the faster the heart rate the shorter the QT interval) and needs to be adjusted to improve the detection of patients at increased risk of ventricular arrhythmia. This is calculated by using Bazett's formula: QTc = QT/ √ RR.

How can one differentiate between Wolff-Parkinson-White and Lown-Ganong-Levine Syndromes?

Wolff-Parkinson-White Syndrome:

An accessory pathway (called the "Kent" bundle) connects the right atrium to the right ventricle and the left atrium to the left ventricle, and this causes an early activation of the ventricles (delta wave) and a short PR interval. The features are:

- Short PR interval (<0.12 s)
- Initial slurring of QRS complex (delta wave) representing early ventricular activation through normal ventricular muscle in region of the accessory pathway
- Prolonged QRS duration (usually > 0.12 s)
- Secondary ST-T changes due to the altered ventricular activation sequence.
  See the ECG below:



LGL (Lown-Ganong-Levine) Syndrome

An AV nodal bypass track exists in the His bundle, and this permits early activation of the ventricles without a delta-wave because the ventricular activation sequence is normal.

List the differential Diagnosis of ST Segment Depression.

Normal Variants or Artifacts

- Pseudo-ST-depression (wandering baseline due to poor skin-electrode contact)
- Physiologic J-junctional depression with sinus tachycardia.

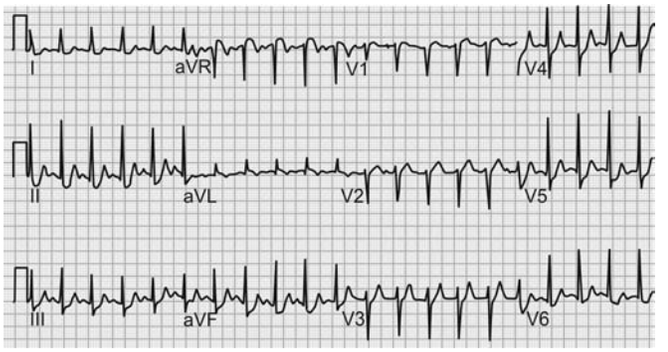
Ischemic Heart Disease

- Non Q-wave MI
- Reciprocal changes in acute Q-wave MI (e.g. ST depression in leads I and aVL with acute inferior MI).

Non-ischemic Causes of ST Depression

- RVH (right precordial leads) or LVH (left precordial leads, I, aVL) – called as strain pattern
- Digoxin effect on ECG
- Hypokalemia
- Mitral valve prolapse
- Subdural hematoma.

Example:



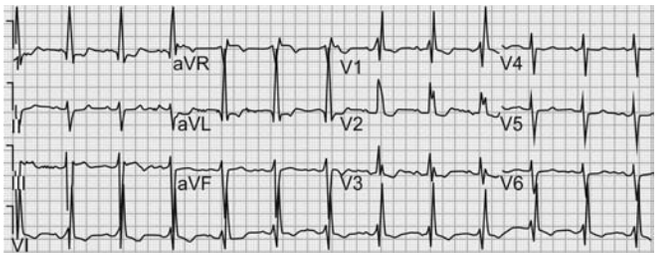
What are the causes of RBBB and criteria for diagnosis?

Causes

- Normal individuals
- Transient in PE
- Coronary artery disease
- ASD.

Criteria for Diagnosis

- Right axis deviation
- QRS duration >0.12 s
- Secondary R wave is seen in V1 and V2 resulting in M-shaped QRS complex
- Deep, slurred S waves in left precordial leads (typically V4,V5,V6).



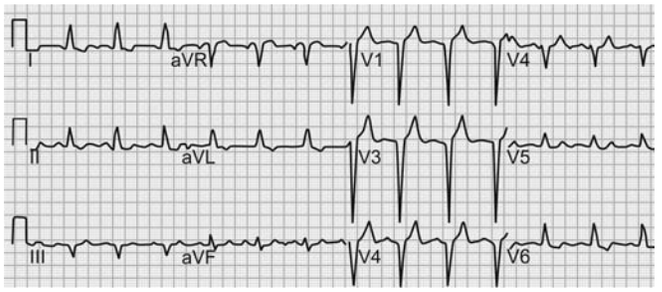
How do you diagnose LBBB on ECG?

- Left axis deviation
- QRS duration > 0.12 s
- M pattern QRS complexes in V4, V5, V6

State the Causes of LBBB

- IHD
- Myocarditis
- HT
- Cardiomyopathy
- Aortic valve disease.

Example:



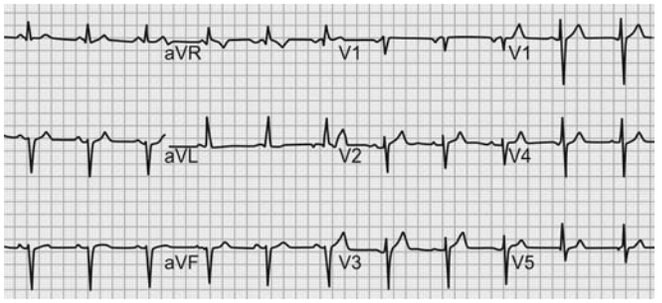
How would you diagnose LAHB and LPHB?

Hemiblock: Conduction is delayed in one of the divisions of Left bundle branch (LBB).

LAHB: Conduction is delayed in antero-superior division of LBB so activation is through postero-inferior division.

Criteria for LAHB: Left axis deviation with QRS axis more negative than -30°, deep S waves in inferior limb leads (leads II, III, aVF).

Example:



LPHB: Conduction is delayed postero-inferior division of LBB.

Criteria for LPHB: Right axis deviation, tall R wave in all inferior limb leads (leads II, III, aVF).

What are the diagnostic criteria for 1st degree heart block?

1st degree heart block is a delay in conduction of impulse passing through AV node.

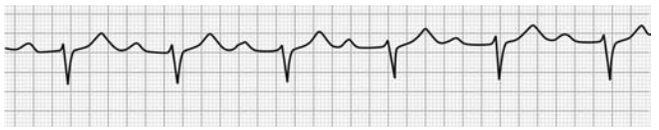
Criteria

- Prolongation of PR interval >0.2 sec
- Rhythm is regular
- All beats conducted from atria to ventricle.

Causes

- Idiopathic
- Coronary artery disease
- Digitalis toxicity
- Dugs β-blockers.

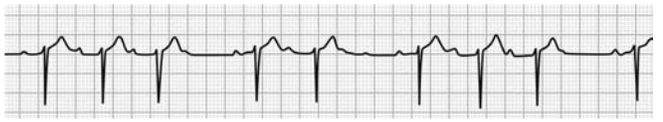
Example:



How do you differentiate between Wenckebach heart block and Mobitz type II heart block?

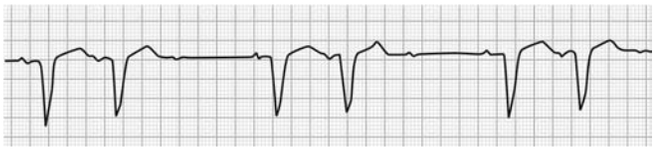
Mobitz Type I (Wenckebach Phenomenon)

- PR interval increases gradually until a P wave fails to conduct to the ventricles.



Mobitz Type II

- PR interval constant
- Intermittent block in conduction of the P wave to the ventricles
- Lesion usually in Bundle of His.



Causes of Mobitz Type I and II

- Acute rheumatic carditis
- Coronary heart disease
- Drugs: Digitalis and Quinidine toxicity.

How do you diagnose complete heart block on ECG?

- Characterised by complete interruption of AV conduction: A-V dissociation
- P wave bear no relationship to QRS complexes—P walking through QRS
- P waves coming at regular interval in different rate and QRS coming at regular interval at different rate
- A slow ventricular rate.

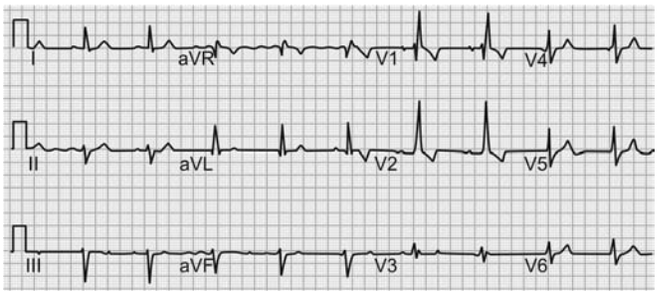
What are the indications for insertion of transvenous pacemaker?

Refer to the chapter on Anesthesia for a patient with pacemaker.

What are bifascicular and trifascicular blocks?

Bifascicular block: More commonly the combination of right bundle branch block (RBBB) with left anterior fascicular block (manifest as LAD), and less commonly RBBB with left posterior fascicular block (manifest as RAD).

Example: RBBB + LAHB



Trifascicular block: Combination of bifascicular block with 1st degree heart block.

Rhythm Abnormalities

What is sinus arrhythmia?

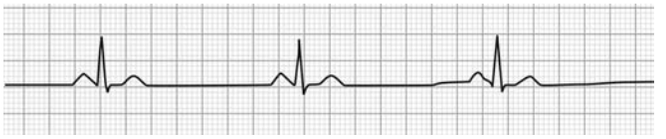
- It is a naturally occurring variation in heart rate that occurs during a breathing cycle, causing an increase in heart rate during inspiration and decrease during expiration.
- It is due to nucleus ambiguus (center in the medulla oblongata), which increases the parasympathetic nervous system input to the heart via the vagus nerve. Upon expiration the cells in the nucleus ambiguus are activated and heart rate is slowed down and during inspiration there are inhibitory signals to the nucleus ambiguus.
- It is more marked in younger age group.
- Normal PQRST complexes with alternating long and short PP interval.
- *Accentuated:* Carotid sinus compression.
- *Abolished:* Exercise, atropine.

Define sinus bradycardia and sinus tachycardia?

Sinus bradycardia: When SA node discharges at a rate slower than 60/min.

Sinus tachycardia: When SA node discharges at a rate faster than 100/min.

Both are associated with normal PQRST complexes.



What are causes of sinus bradycardia?

Physiological

- Sleep
- Athletes
- Starvation.

Pathological

- Raised ICP
- Hypothyroidism
- Obstructive jaundice (action of bile salts on SA node)
- Glaucoma
- *Drugs:* Digitalis effect, Quinidine, B blockers, calcium channel blockers.

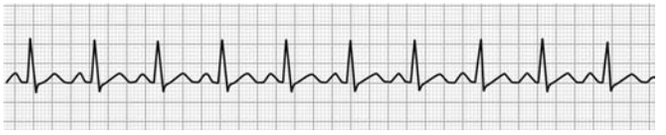
What are causes of sinus tachycardia?

Physiological

- Exercise
- Pregnancy
- High altitude

Pathological

- Pain
- Hypermetabolic states like Thyrotoxicosis, fever
- Anxiety
- Excessive caffeine
- Hemorrhage
- PE
- *Drugs:* Atropine, adrenaline

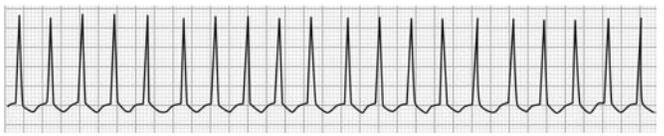


What do you understand by paroxysmal supraventricular tachycardia? Enumerate the causes and the treatment?

- Circus movement or reciprocating tachycardias utilizing the mechanism of re-entry.
- The onset is sudden, and also stops abruptly—paroxysmal.
- Narrow-QRS tachycardias (unless there is preexisting bundle branch block or rate-related aberrant ventricular conduction).
- May be benign.
- Persistence would precipitate heart failure.

Causes

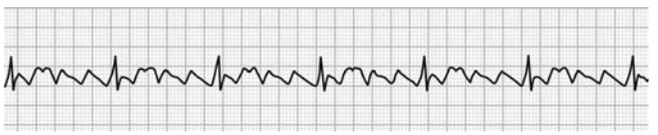
- Idiopathic
- Thyrotoxicosis
- Excessive caffeine intake
- Drugs: Adrenaline, Amphetamines
- Sepsis.



Differentiate between atrial flutter and fibrillation.

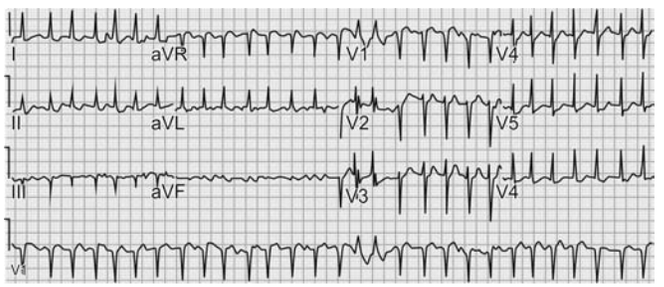
Atrial Flutter

- Rapid and regular excitation contraction of heart
- P wave—saw-tooth appearance
- Can be due to circus movement or focal discharge from an ectopic atrial focus
- Ventricular rate depends on efficacy of AV conduction.



Atrial Fibrillation

- Excitation and recovery of atria are disorganized and chaotic
- Causes small twitches of the atrial myocardium
- Irregularly irregular ventricular rhythm
- The atria are functionally fractionated into a chaotic state of islets at various stages of excitation and recovery.



State the causes of atrial fibrillation.

Causes

- Rheumatic fever
- CHD
- Thyrotoxicosis
- Excessive caffeine
- *Drugs:* Adrenaline, Amphetamines.

What is ventricular tachycardia?

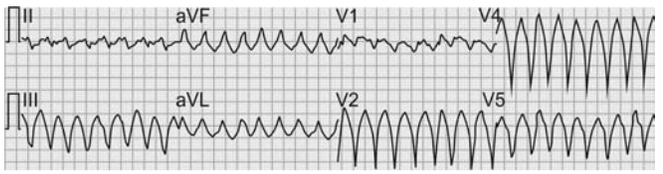
Ventricular tachycardia (VT) refers to rhythm faster than 100/min where pacemaker is distal to the bundle of His.

Types: Monomorphic (uniform morphology) versus polymorphic versus Torsade-de-pointes.

Monomorphic VT: A single focus giving rise to the ventricular activation hence the electrocardiographic pattern (morphology) remains the same, and the rhythm is called monomorphic VT.

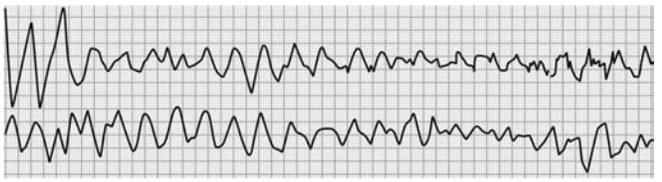
PolymorphicVT: When the ventricular activation sequence varies due to multiple foci, the electrocardiographic pattern differs, and the rhythm is called polymorphic VT.

Torsade-de-pointes: A polymorphic ventricular tachycardia associated with the long-QT syndromes characterized by phasic variations in the polarity of the QRS complexes around the baseline. Ventricular rate is often >200 bpm and ventricular fibrillation often result.



What is ventricular fibrillation?

It is a disorganized and chaotic activity of the heart resulting in irregular chaotic deflections of various height, shape and width.



What are the diagnostic criteria for right atrial enlargement, left atrial enlargement and biatrial enlargement?

Right atrial enlargement (RAE): Tall and peaked (p-pulmonale) P wave amplitude > 2.5 mm in II and/or >1.5 mm in V1 (criteria are not specific or sensitive).

Left atrial enlargement (LAE): Wide and notched (p-mitrale association with mitral valve disease)—P wave duration > 0.12s in frontal plane (usually lead II).

Biatrial enlargement: Features of both RAE and LAE in same ECG. P wave in lead II > 2.5 mm tall and > 0.12s in duration.

What are the diagnostic criteria and causes for right and left ventricular hypertrophy?

Causes of Right Ventricular Hypertrophy

- Pulmonary hypertension
- Fallot tetralogy
- Pulmonary valve stenosis
- VSD
- COPD.

Criteria

- Right axis deviation
- Tall R wave in right ventricular leads V1-V4
- Right ventricular strain pattern (depressed convex – upward ST segment with an inverted T wave)
- Tall R in aVL.

Causes of Left Ventricular Hypertrophy

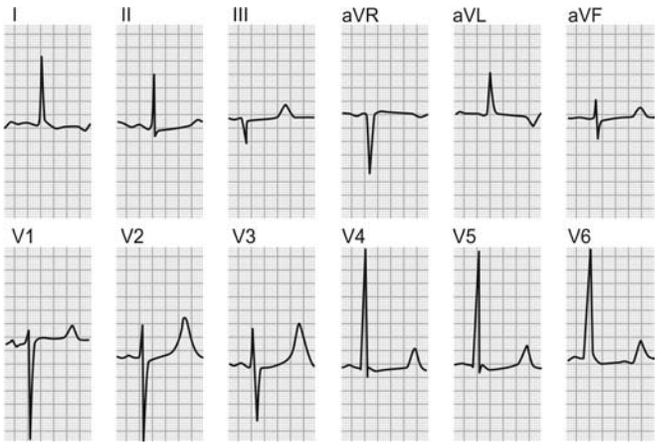
- Aortic stenosis
- Aortic regurgitation
- Hypertension
- Cardiomyopathies.

The Sokolow-Lyon Index

- S in V1 or V2 (Leads oriented to RV) + R in V5 or V6 ≥ 35 mm (Leads oriented to LV)
- R in aVL ≥ 11 mm (Lead oriented to LV).

The Cornell voltage criteria for diagnosis of LVH involves:

- S in V3 + R in aVL > 24 mm (men)
- S in V_3 + R in aVL > 20 mm (women)



Left ventricular strain: A depressed convex upwards ST segment and an inverted T wave in left ventricular leads V4 – V6. Strain can be due to increased tension within the myocardium or a mismatch between oxygen supply and demand.

What are characteristics features of myocardial infarction?

Hyper acute MI: This phase is characterized by ST segment elevation oriented towards the injured site, ST segment depression of the leads oriented towards the uninjured surface. Tall and widened T wave can exceed the R wave.

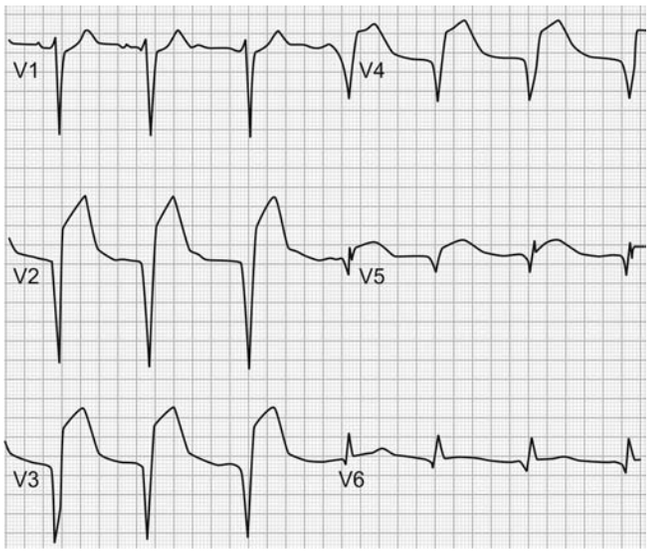
This phase is quite important as VF occurs most commonly during this phase.

Fully Evolved AMI: Necrosis is reflected by Q waves which are oriented towards the necrotic tissue. Myocardial injury is reflected by ST segment deviation towards the injured tissue. Myocardia ischemia is reflected by inversion of T waves oriented towards the ischemic area.

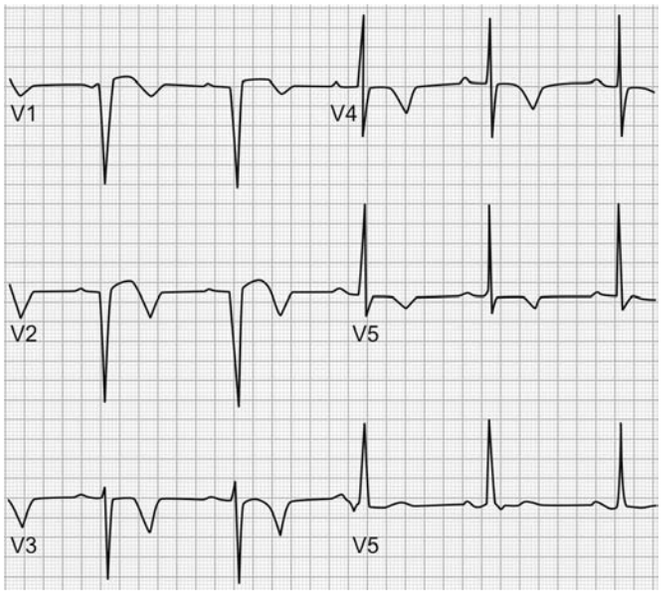
Resolution phase: Gradual return of ST segment to baseline. Tall T waves can appear in leads oriented to uninjured surface. Abnormal Q wave may persists in leads oriented to injured site.

Ant wall infarction: Extensive anterior infarction: Lead I , aVL, all precordial leads. Anteroseptal infarction: Leads V1-V4. Anterolateral infarction: Lead I, aVL, Lead V4-V6.

Example: Anterior MI

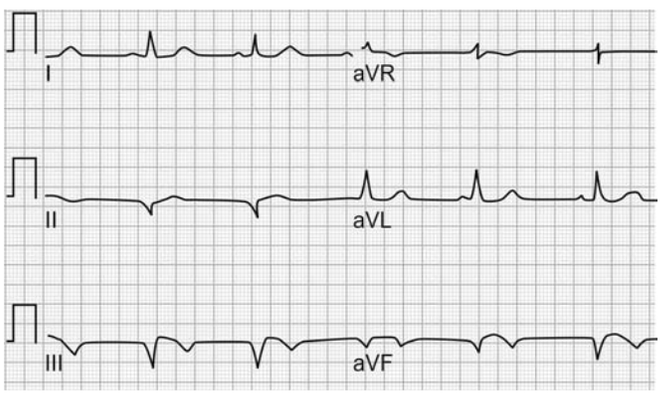


Example: Anteroseptal MI



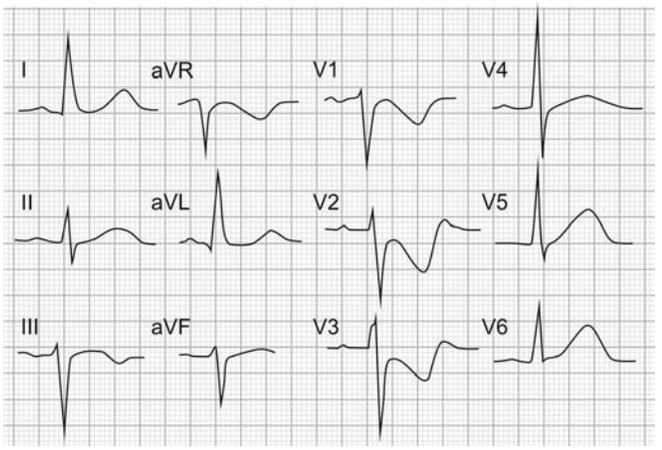
Inferior Wall M Infarction

Lead II, III, aVF



True Posterior Infarction

Classical Q wave, ST segment elevation, inverted T wave do not occur (none of the leads are oriented towards the posterior surface). Diagnosis is made by the reciprocal changes in the leads oriented towards the uninjured surface (anterior surface—V1 and V2). Tall and wide R waves, tall T waves, ST segment depression: V1-V2.



What are ECG findings in massive Pulmonary Embolism?

- S1Q3T3 pattern: large S waves in Lead I, large Q waves in V3 and T wave inversion in lead III.
- Sinus tachycardia is the commonest
- Right axis deviation
- T wave inversion in the right precordial leads
- Non-specific T wave changes and non-specific ST segment elevation or depression.

Can ECG help in diagnosing Pericarditis?

Acute Pericarditis

- Normal voltage
- Raised, concave upward ST segments in leads oriented to heart surface.

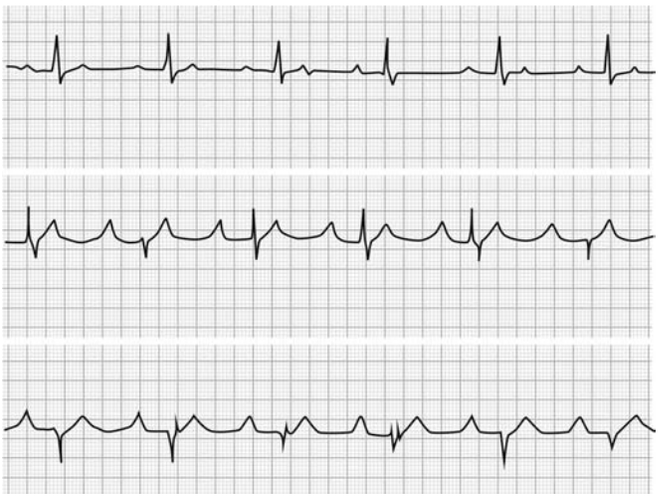
Chronic Pericarditis

- Generalized low voltage
- Low or inverted T wave in leads oriented to heart surface.

What are ECG changes associated with digitalis toxicity and quinidine toxicity?

- Digitalis toxicity
- Sinus bradycardia
- Premature beats
- Arrythmias: supraventricular and ventricular
- Blocks: 1st, 2nd and 3rd degree blocks and bundle branch blocks.

Example: Atrial tachycardia with 2:1 block



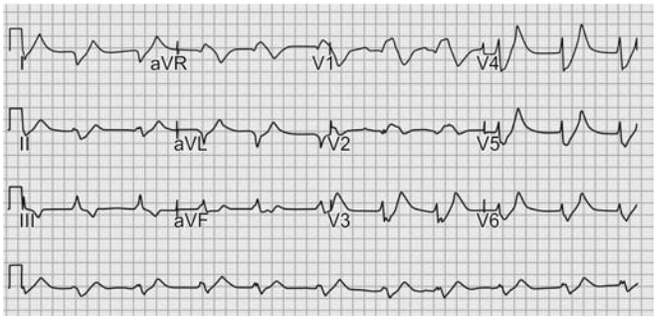
Quinidine Toxicity

- *Blocks:* 1st, 2nd and 3rd degree blocks
- Bundle branch blocks
- AV junctional rhythm
- Ventricular premature beats, VT, VF.

What are ECG changes associated with hyperkalemia and hypokalemia?

Progressive Changes of Hyperklemia

- Appearance of tall, pointed, narrow T waves
- Amplitude of P wave decreases
- Widened QRS complexes
- ST segment changes (elevation/depression)
- Advanced intraventricular block (very wide QRS with RBBB, LBBB, bi- or tri-fascicular blocks) and ventricular ectopics.
- Absent P waves, very broad and bizarre QRS complexes, AV block, VT, VF or ventricular asystole.



ECG Changes of Hypokalemia

- ST segment depression, decreased T wave amplitude, T wave inversion, increased U wave height
- Cardiac arrhythmias: atrial and ventricular ectopics, atrial tachycardia, heart blocks
- Increased PR interval.

Hypercalcemia

- Decreased Q-T interval and ST segment duration.

Hypocalcemia

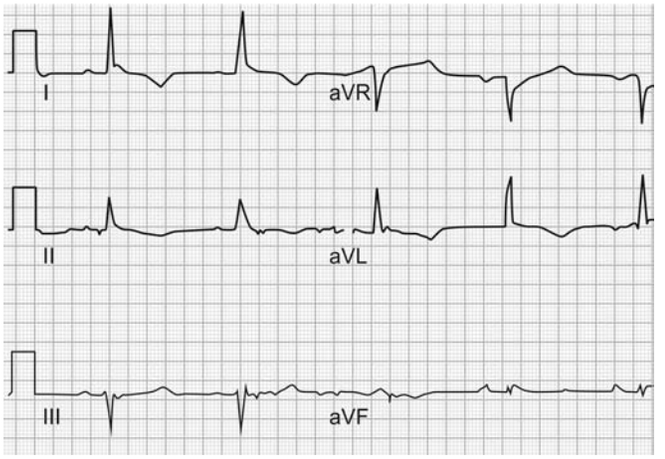
- Increased QT interval and ST segment duration.

Magnesium and ECG

- Hypomagnesemia resemble that of hypokalemia
- Hypermagnesemia resemble that of hyperkalemia.

Do Changes in Temperature Affect ECG?

- ECG changes associated with Hypothermia are:
- Sinus bradycardia
- Shivering artifact
- Prolonged QT interval
- Prolonged PR interval
- J wave (Osborne wave): This occurs when the body temperature falls below 25°C. It appears as an extra deflection at the end of the QRS complex just overlapping the beginning of the ST segment.



What are ECG changes in hypothyroidism?

The ECG changes here are due to interstitial myocardial edema and pericardial effusion.

- Sinus bradycardia
- Low voltage complexes (P, QRS and T waves)
- ST segment depression
- Prolonged P-R interval
- A-V block
- Prominent U waves
- Corrected QT interval may be prolonged.

Hyperthyroidism

- Sinus tachycardia
- Arrhythmias (atrial fibrillation, atrial flutter or atrial tachycardia).

Suggested Reading

1. An Introduction to Electrocardiography. Leo Schamroth. Publisher - Blackwell Scientific.
2. Sheppard LP, Channer KS. Acute coronary syndromes CEACP. 2004;4:175–80.

50 Interpretation of X-rays

A Kulkarni, R Sareen, A Chatterjee

Reading a X-rays is an essential skill for the anesthetist. In the examination you are likely to be questioned on chest X-rays, X-rays of the cervical spine and abdomen. Sometimes X-rays of trauma victims are kept and your viva may go in the direction trauma diagnosis and management. It is beyond the scope of this write-up to discuss all the X-rays in full detail. Instead, we will discuss principles of reading the X-rays, and with each X-ray is a list of pertinent questions you may be asked.

Reading a chest X-ray

X-rays should be read in a systematic manner. Check the following before you read the chest X-ray. The same principles apply to other X-rays.

Label: This is to ensure that you are actually seeing the X-ray of a patient you are interested in. This will generally be in the form of a case record number. What we want is correct patient, correct date and time and correct (radiology) examination.

Projection: Most chest X-rays done in the OPD will be PA views (X-ray source behind the patient and the plate placed in front of the chest) taken with the patient standing. Most of those done in the intensive care unit (i.e. portable) are AP views (X-rays coming from the front and the plate under the patient) with the patient lying down. In AP view, because the mediastinum is close to the X-ray source, it will appear enlarged as the X-rays tend to fan out while reaching the plate. It is therefore difficult to comment on cardiomegaly, unless gross, in an AP view. In the erect view, air-fluid level may be seen in chest (if there is a lesion), or in the gastric fundus. Sometimes the AP film may be marked as supine. There are several differences supine AP view is compared to PA erect view, these being smaller lung fields, broader heart and wider mediastinum, clavicles projected higher up and horizontal appearing ribs.

Side: The radiographer will always put a side marker on the film. We must check this, if we do not want to miss a dextrocardia or situs invertus!

Then you check the quality of the film by looking at rotation, penetration, timing and adequacy of the film:

Rotation: To check whether the patient is rotated, see the sternal end (medial end) of the clavicles, they should be equidistant from the midline (spinous processes of the thoracic vertebrae). If the patient is rotated we can not comment on the mediastinal shift or compare the quality of lung fields.

Exposure or penetration: In the PA view, you should be able to see the spinous processes of the first four thoracic vertebrae and the intervertebral disk space should be just visible. If the film is overexposed, the lung fields will appear blacker than normal. If underexposed, the lung fields will look too white. Penetration/exposure is important as in an overexposed film you may miss a pneumothorax.

Timing (in relation to respiration): Most chest X-rays are end-inspiratory films, you should be able to see six anterior ribs or 10 posterior ribs above the diaphragm. End expiratory films are sometimes done when the radiographer is asked to detect a small pneumothorax. ICU films may be done at any time as the patient may have no control over respiration. In full inspiration you should be able to count 6 rib interspaces.

Adequacy of film: You should be able to see the full area of interest. In chest X-ray you should see both apices as well as both costo-phrenic angles.

When you describe a chest X-ray, you should say: A labeled chest X-ray with side marked, PA view, well-centralized (or not) adequate film with good exposure done in inspiration, before

you proceed to actually reading the chest X-ray. Then we can proceed to reading a chest X-ray. There are numerous ways of going about reading a chest X-rays. The most important thing is to be systematic so that no anatomical structure is missed. We can either proceed from center (mediastinum) to periphery or the other way round (Table 50.1). Both these ways are easy to follow.

Centre to periphery	A, B C, D
Mediastinum	Airway (Trachea and Bronchi)
Lung fields from apices to diaphragm	Lung fields and bones/soft tissues
Bones and soft tissues	Heart shadow
Abdominal shadows	D – Diaphragm
Foreign bodies	Others

Mediastinum: Check the size, shape, position of the mediastinum. Check whether trachea is central. The mediastinum is bound by right brachiocephalic vessels, the ascending aorta, superior vena cava and the right atrium and rarely IVC on the right and left brachiocephalic vessel, aortic arch, pulmonary trunk, left atrial appendage and the left ventricle on left. The left atrium and right ventricle are not seen under normal conditions. Calculate the cardiothoracic ratio: Draw a line in the center (of spine). Draw lines to the right and left heart borders where the distance is maximum from center (commonly designated a and b). Measure the widest thoracic diameter (called c) cardiothoracic ratio from the inner margins of the ribs. The cardiothoracic ratio is given by (a + b)/c, normally it is < 0.5.

Lung fields from apices to diaphragm: Compare size of lungs, check for proper inflation. Look at the pulmonary parenchyma, you should find equal radio-lucency at similar levels on both sides. Sometimes it helps if you cover mediastinum with a piece of paper so that it does not distract you from the lungs. The costo-phrenic and cardio-phrenic angles should be clear. For descriptive purposes the lung fields are non-anatomically divided into upper (above anterior end of the 2nd rib), middle (between anterior ends of 2nd and 4th ribs) and lower (below anterior end of the 4th rib). Look for abnormal radiodensities or radiolucencies, masses, presence of infiltrates and calcifications. Small lung lesions may be easily overlooked in some areas: the apices, pleural margins and behind the calcified anterior first rib cartilage, heart, and the diaphragm.

Check pulmonary vasculature: The left pulmonary artery (left hilum) is higher (1–2 cm) than the right. The vessel size is more in lower than upper areas (effect of on low pressure pulmonary vasculature). If this is reversed, then redistribution of blood flow has occurred as in congestive cardiac failure (CCF).

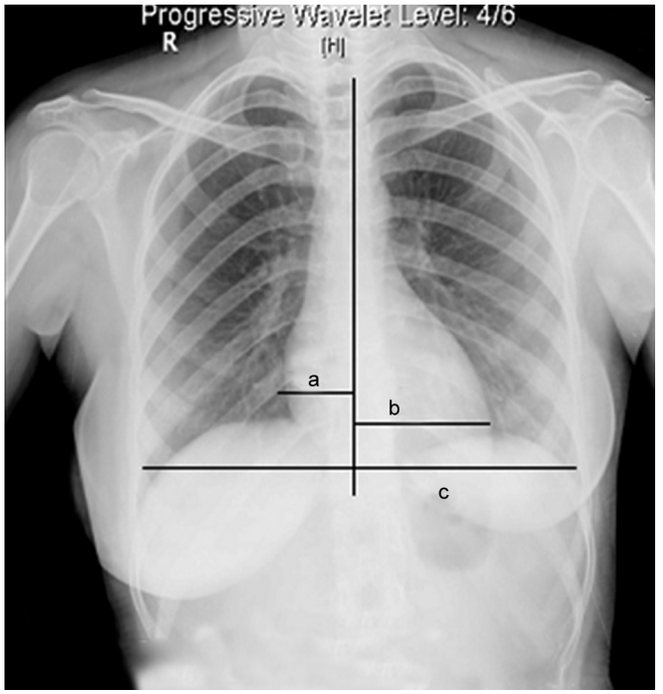


Fig. 50.1 Cardiothoracic ratio a + b/c

Look at the hemi-diaphragms, both should be smooth and curving upwards, right slightly higher (2.5 cm) than left. Follow pleura around the rib cage.

Bone and soft tissues: Check soft tissue shadows, note any obvious mass, calcification, and look for subcutaneous emphysema which may give a hint about underlying pneumothorax, rib fractures. Look at the bony skeleton: note bone densities, look for masses. Check breast shadows, a missing breast shadow in mastectomy patient may be the only abnormality (NORMAL X-ray for exam).

Abdominal shadows: Check below the diaphragm for bowel gas, organ size, abnormal calcifications, free air, etc.

Foreign bodies: Check for iatrogenic foreign bodies: endotracheal tube (should be above carina), nasogastric tube (below the diaphragm), tracheostomy tube, central venous catheter (CVC) (tip above the level of carina, same as pleuropericardial reflection. If the CVC punctures the SVC wall below this level cardiac tamponade will occur) and intercostal drains (all holes should be inside the chest, none in subcutaneous plane, no kinking or touching the heart), pacemaker box and lead (check for continuity, note any fractures) artificial cardiac valves. Then look for other foreign bodies.

Chest X-rays for Practice

You will find some X-rays below. We will talk about diagnosis/differentials and or etiologies and then questions pertinent to that diagnosis that you may be asked in the examination. You should know something of medical theory about each condition and of course all relevant anesthetic/clinical management for that condition. Sometimes in the bracket are the answers.

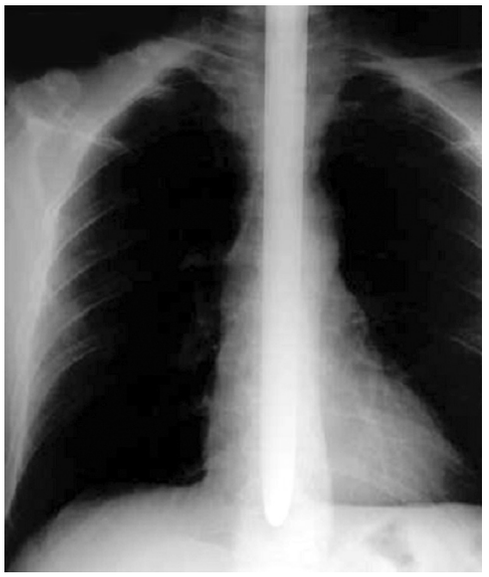


Fig. 50.2 CXR 1. Sword swallower

CXR 1. Foreign body

This is a very dramatic X-ray with a very obvious diagnosis. You might get a chest X-ray with a needle in one of the bronchi. A systematic review of X-ray will help you to pick it up. The usual questions asked with this X-ray are:

- Where is the foreign body–trachea or esophagus?
- How will you confirm the location? (A lateral chest X-ray)
- How will you anesthetize the patient for removal of a) tracheal b) esophageal FB? How will you induce anesthesia? (Crash induction)
- Will you use muscle relaxant? (Preserve spontaneous breathing as far as possible) Differences between organic versus non-organic foreign bodies, recent versus old (impacted), central versus peripheral?

CXR 2. Pneumothorax

Supine chest X-ray in a trauma victim showing rib fractures, tension pneumothorax and subcutaneous emphysema.

The usual questions are:

- What is the etiology of pneumothorax?
- How do you treat a pneumothorax?
- What is tension pneumothorax?
- How do you diagnose it?
- Will you wait for confirmatory X-ray?

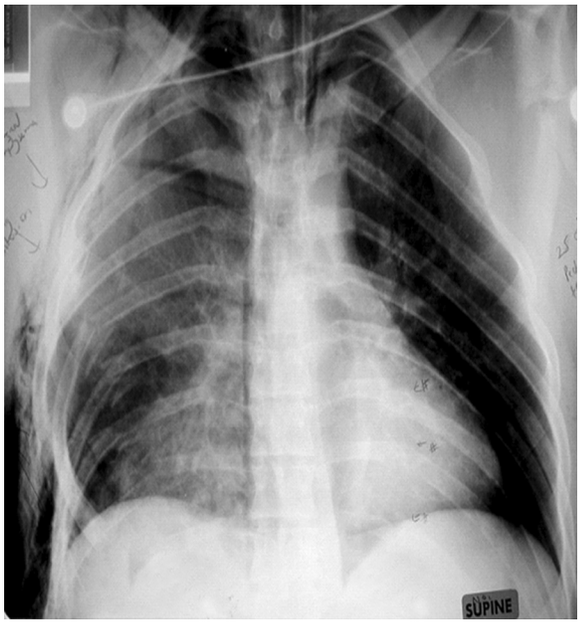


Fig. 50.3 CXR 2. Tension pneumothorax

- How do you treat a tension pneumothorax?
- How do you differentiate between a tension pneumothorax and cardiac tamponade (clinically)?
- How do diagnose cardiac tamponade?
- How do you treat cardiac tamponade?
- Describe the technique for insertion of intercostal drain? Describe care of intercostal drain?
- When do you use one bottle, two bottle, etc. of intercostal drain? Describe care during transport of a patient with an intercostal drain.

CXR 3. Pleural effusion.

Erect PA view showing left-sided pleural effusion.

- What is the etiology of pleural fluid?
- Why is the top of a pleural effusion C-shaped?
- How much fluid should be present in the thoracic cavity for it to be detected on a chest X-ray?
- How do you estimate the amount of pleural effusion?
- Will you drain pleural effusion in all patients?
- Which patients need to have the effusion drained?
- What precautions will you take during induction of anesthesia in a patient with pleural effusion?
- Describe techniques available for drainage of a pleural effusion?
- What is a loculated pleural effusion?
- Why are they difficult to drain?
- How will you differentiate loculated from non-loculated effusion?
- What are the causes of bilateral pleural effusion?
- Do we need to drain bilateral pleural effusions?

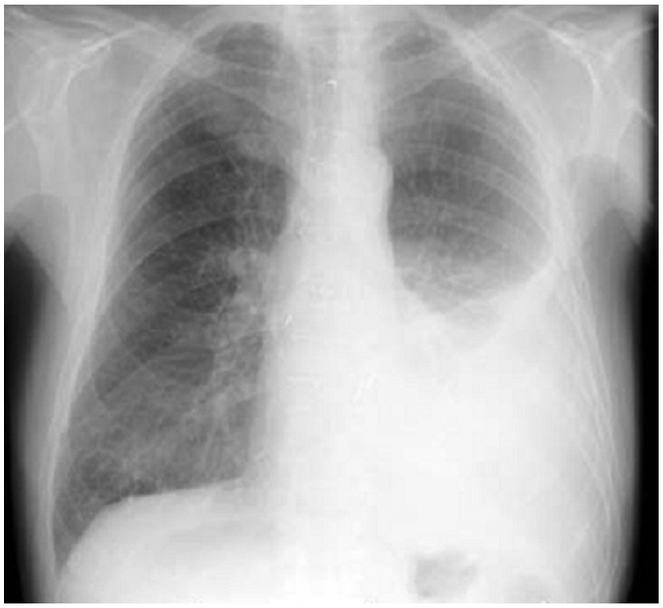


Fig. 50.4 CXR 3. Pleural effusion

CXR 4. Hydropneumothorax.

- When the top fluid level in pleural cavity is flat (instead of being C-shaped) it indicates air above the fluid.
- Trauma can frequently lead to hemopneumothorax, so when you see a CXR showing hydropneumothorax, always suspect a trauma case, and look for rib fractures, subcutaneous emphysema and other additional injuries if any.
- What are the causes of hydropneumothorax?

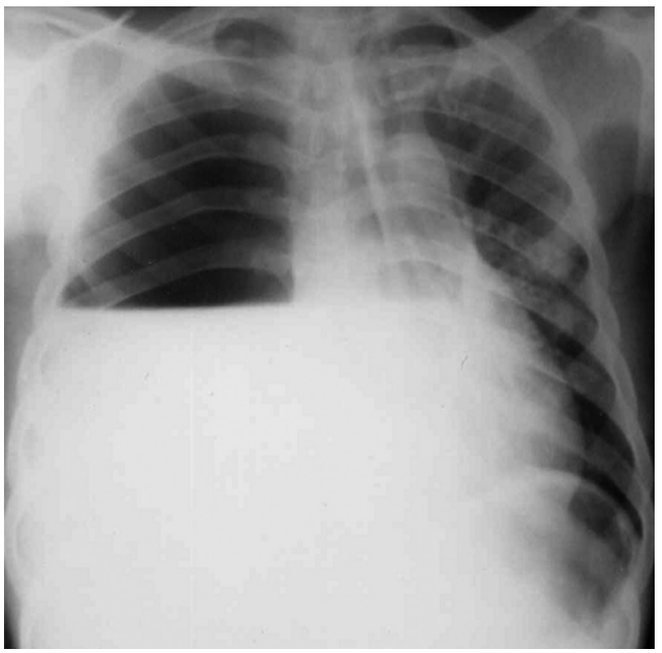


Fig. 50.5 CXR 4. Hydropneumothorax

- How do you treat hydropneumothorax?
- Does this patient need emergency intubation (if it were a trauma patient?
- Will you intubate first or put an intercostal drain first?
- Can this be a post-pneumonectomy X-ray?
- (No, normally the mediastinum shifts to the operated side due to compensatory hyper-expansion of the remaining lung) You may or may not see an intercostal drain.

CXR 5. Opaque hemithorax

- What are the differentials of opaque hemithorax?
- Lung collapse: volume loss, mediastinal shift towards affected side. Pleural fluid: transudate/exudates, empyema, haemothorax, chylothorax. Pneumonectomy: look for surgical clips and thoracotomy. Tumor: fibrous tumor of pleura; pleural metastases; mesothelioma. Extensive consolidation: pneumonia; pulmonary edema; obstructing neoplasm. Lung agenesis.
- The viva will then proceed towards medical management of these conditions and relevant anesthetic management when patient presents with those conditions.

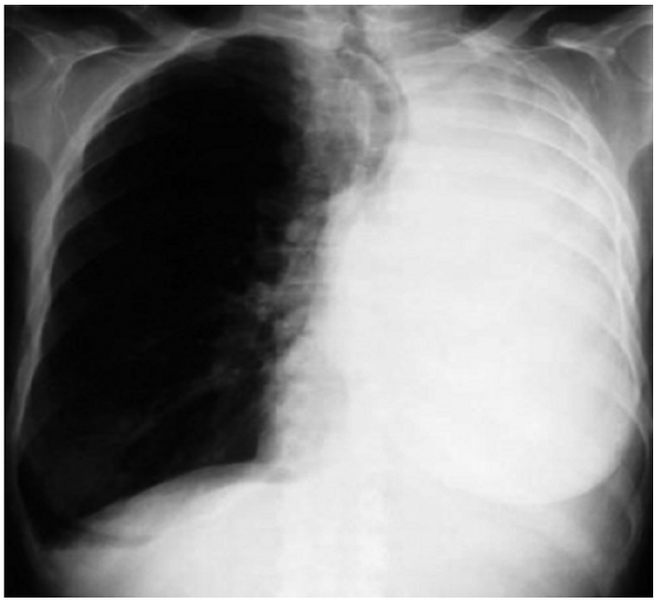


Fig. 50.6 CXR 5. Opaque hemithorax

CXR 6. Congenital diaphragmatic hernia (CDH)

- X-ray of a child with bowel loops in the hemithorax.
- If you see CXR of an adult with bowel loops in the hemithorax, consider diagnosis of trauma with diaphragmatic rupture hernia and look for other pointers to trauma in the CXR.
- Another differential for bowel loops in the chest is hiatus hernia.

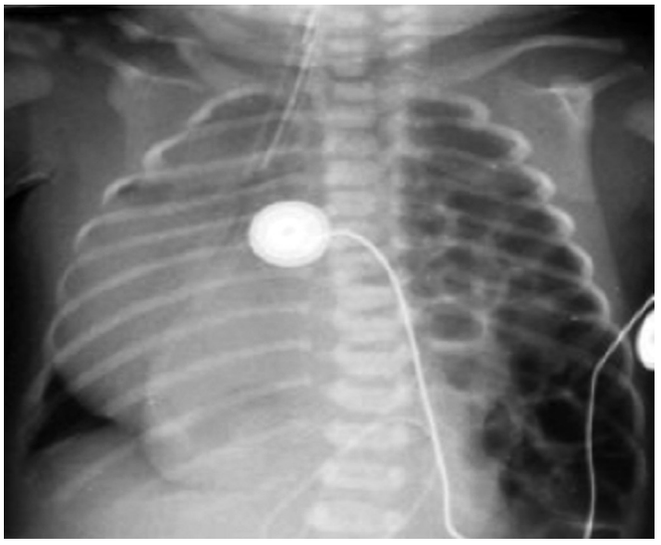


Fig. 50.7 CXR 6. Congential diaphragmatic hernia

- With CDH the viva will be for anesthetic management of CDH patients.
- What are the pulmonary problems in CDH patients?
- How do these problems affect anesthesia induction?
- What are the options for management of pulmonary hypertension?
- Do these patients need postoperative mechanical ventilation?
- How will you ventilate these patients?

CXR 7. Gas under diaphragm

Postoperative patient, so look for evidence of recent surgery (clips, drains, etc).

- Post-laparoscopy patients, perforated viscus
- Gas gangrene of the bowel, penetrating abdominal trauma (look for other injuries), peritoneal dialysis
- Fallopian tube insufflation, Chilaiditis syndrome (interposition of hepatic flexure of the colon, between the upper border of liver and the right dome of diaphragm may mimic gas under the diaphragm)
- What are the considerations in anesthetic management for emergency laparotomy? What problems do you anticipate? How will you resuscitate these patients? How will you induce anesthesia? Which muscle relaxant will you use?
- What are the likely electrolyte and metabolic disturbances?
- Will you ventilate this patient postoperatively?

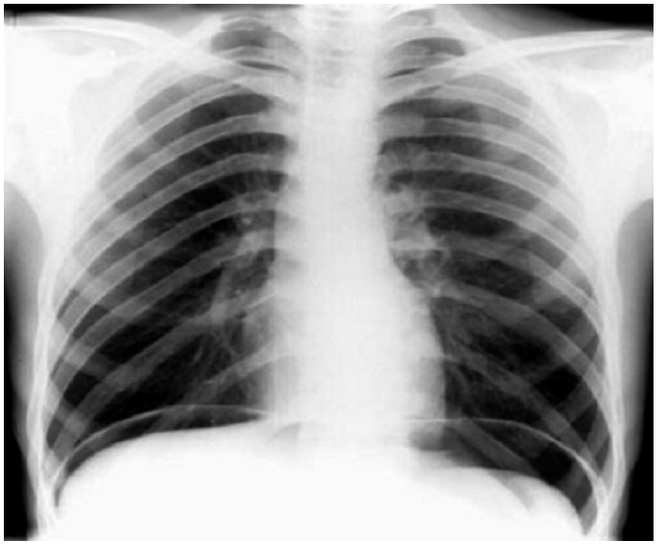


Fig. 50.8 CXR 7. Gas under diaphragm

CXR 8. Emphysema with right ventricular hypertrophy (RVH)

Emphysema is characterized by hyperinflation (>10 posterior ribs above the diaphragm), barrel chest, widened intercostal spaces, depressed or flattened diaphragm, tubular elongated heart or boot-shaped heart [in case of right ventricular hypertrophy (RVH)].

The usual questions are:

- What are the differences in pathophysiology of COPD, Asthma and Emphysema?
- How will you investigate this patient for preoperative evaluation?
- What are the spirometric features COPD, asthma and emphysema?
- What are the bedside pulmonary function tests?

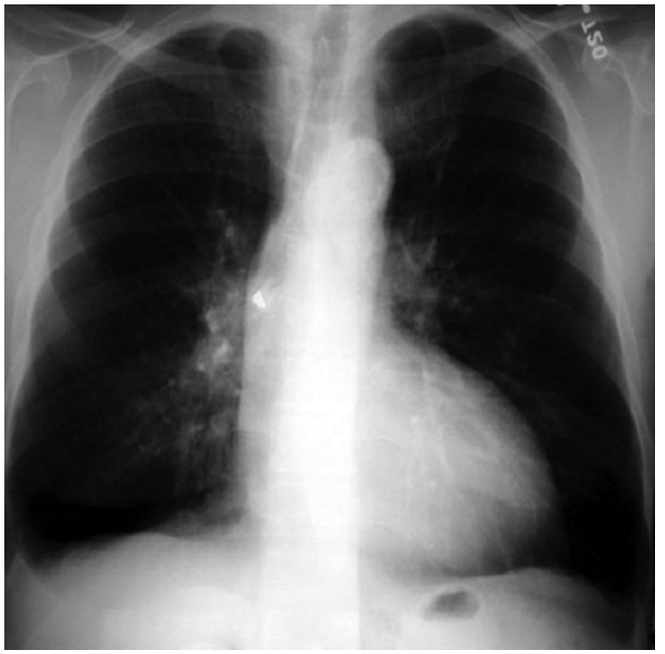


Fig. 50.9 CXR 8. Emphysema with RVH

- How do you optimize COPD patients for surgery?
- How will you predict
  - Likelihood of postoperative pulmonary complications?
  - Need for postoperative ventilation?

CXR 9. Bronchiectasis

Honeycombing pattern seen at both bases may occur unilaterally. You may get a bronchography X-ray as well showing bronchiectasis. Usual questions are:

- What is bronchiectasis?
- What is the etiology and pathophysiology of bronchiectasis?
- How will you optimize this patient for surgery?
- Discuss various position used for postural drainage in patients with bronchiectasis.
- How will you anesthetize this patient for bronchography?
- What radio contrast media are used for bronchography?
- Describe the bronchography technique.

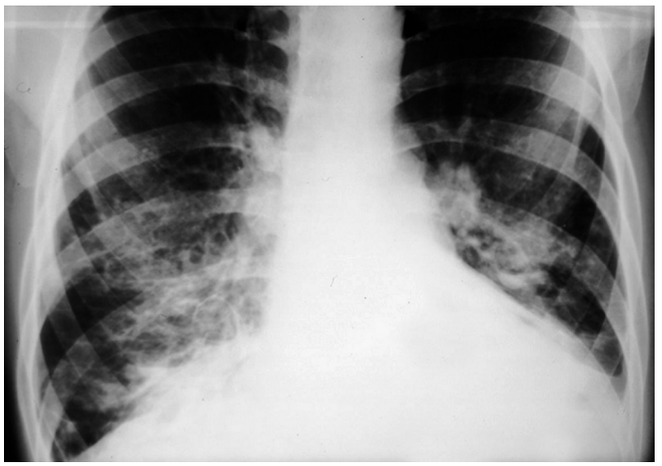


Fig. 50.10 CXR 9. Bronchiectasis

CXR 10. Lobar collapse

Features: mediastinal shift towards side of collapse, elevation of the hemidiaphragm, reduced vasculature on that side, hilar mass (if endobronchial tumor) or foreign body (if this has caused collapse), endobronchial intubation could be a cause. What are the causes of lobar collapse? Luminal mass (neoplasm, foreign body, mucus plug/inflammatory exudates, endoluminal metastasis, misplaced endotracheal tube) bronchial wall inflammation (TB, sarcoid), extrinsic compression (lymph nodes, aneurysm). What is treatment of postoperative lobar collapse? What is the silhouette sign? An intra thoracic radio-opacity, if it is in anatomic contact with a border of heart or aorta, will obscure that border. If the lesion is not anatomically contiguous with a border or a normal structure, it will not obliterate that border. Thus the anterior structure will be silhouetted against the posterior structure.

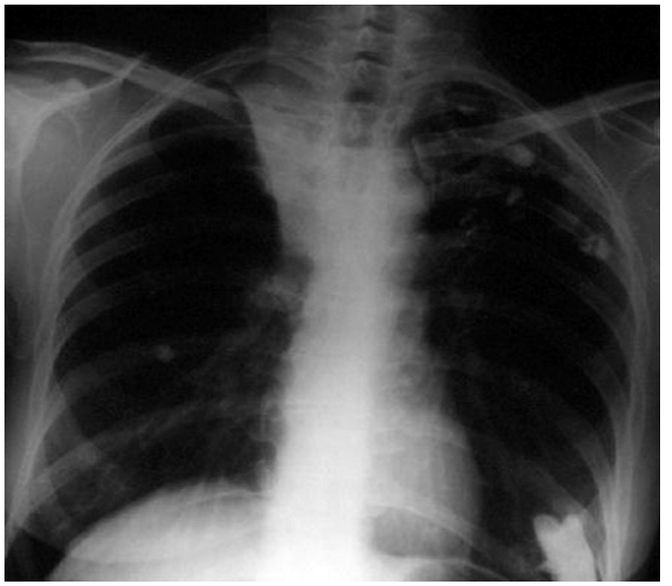


Fig. 50.11 CXR 10. Right upper lobe collapse

- What are the causes of postoperative lobar collapse?
- What are the common postoperative pulmonary complications?
- Which patients are susceptible to postoperative pulmonary complications?
- What preventive measures can be undertaken to prevent postoperative pulmonary complications?

CXR 11. Mediastinal Mass

This will be seen as superior mediastinal widening.

- What is the differential diagnosis superior mediastinal widening seen on chest X-ray?
- What clinical signs/symptoms will alert you to likely intraoperative problems?
- What findings are likely to find on spirometry?
- How will you investigate this patient for anesthesia?
- What are the features of fixed/variable and intrathoracic and extrathoracic obstruction on flow-volume loop?
- How does fibreoptic bronchoscopy help in evaluation of these patients?
- How will you induce anesthesia? Intravenous or inhalational?
- How will you intubate this patient?
- What muscle relaxant will you use during induction and maintenance of anesthesia?
- What problems do you expect immediately following induction of anesthesia? (Airway obstruction/failure to intubate/failure to ventilate)
- How do you surmount these problems?

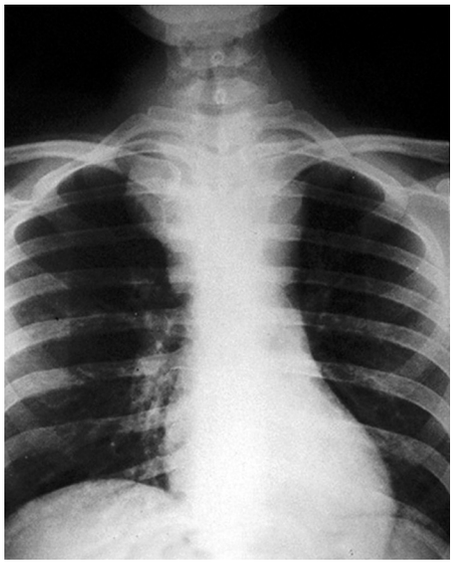


Fig. 50.12 CXR 11. Mediastinal mass

- What additional preparation will you keep ready to overcome these problems?
  - (rigid bronchoscope and a competent surgeon, enough people to change patient position to lateral/prone in case of failure to ventilate)

CXR 12. Mitral Stenosis (MS) and pulmonary hypertension (PH) or Cardiomegaly

- What are the radiological signs of MS with PH?
  - straightened left heart border, enlarged pulmonary conus, elevation of left main bronchus, "antler" or "mustache sign", double atrial shadow, indentation of esophagus on barium swallow (lateral X-ray), left lower lobe collapse due to left atrial enlargement, Kerley B lines.
- What are the types kerleys lines? (ABC)
- What does each one signify?
  - Kerley A at least 2 cm unbranching lines coming diagonally from the periphery toward the hila in the inner-half, caused by distension of anastomotic channels between peripheral and central lymphatics. Kerley B lines are short (< 1 cm) parallel lines at right angles to the pleura the lung periphery representing interlobular septa. Kerley C lines are short, fine lines throughout the lungs, with a reticular appearance.
  - Caused by thickening of anastomotic lymphatics or superimposition of many Kerley B lines.
- What are the differentials for cardiomegaly? Ischemic heart disease, Valvular heart disease Congenital heart disease, dilated cardiomyopathy (Viral, Alcohol, Metabolic), pericardial effusions.

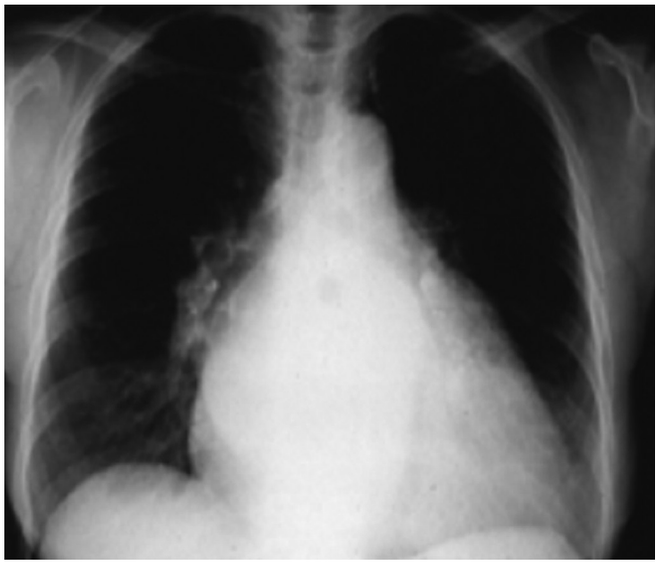


Fig. 50.13 CXR 12. MS with PH

CXR 13. Pacemaker

- This X-ray shows dual lead pacemakers (two leads). The right one is functioning, obviously placed to replace the fractured (marked with a circle) left-sided leads.
- It is most important to trace lead to make sure they are not fractured. Also look for a pneumothorax (if the X-ray done post-procedure). Commonest site of lead fracture is between first rib and clavicle.
- What are the indications for placement of permanent pacemaker?

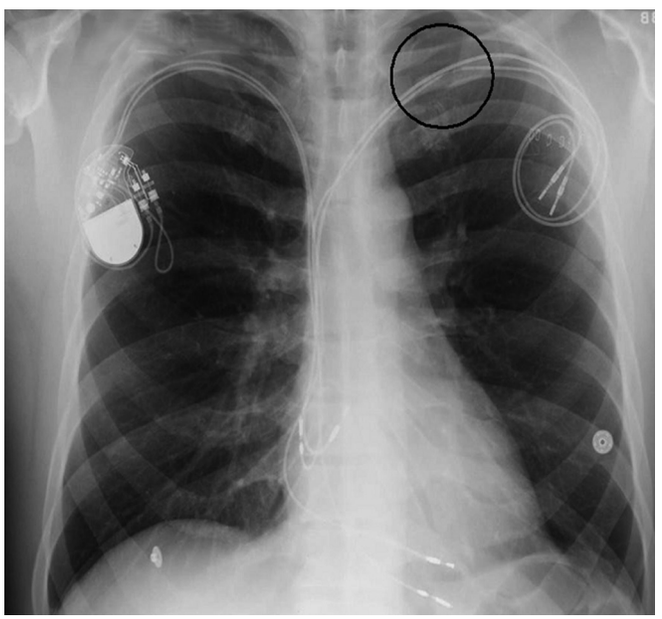


Fig. 50.14 CXR 13. Pacemaker

- How do you diagnose presence of IHD in a patient with a pacemaker?
- What types of pacemakers are available?
- How do you classify pacemakers?
- How do you test that the pacemaker is working properly preoperatively?
- What precautions do you take while anaesthetizing the patients with a pacemaker?
- What precautions do you take with the electrocautery? Do you alter pacemaker settings before anaesthesia induction? In what way?

CXRR 14. Artificial heart valve

Presence of sternal wires should alert you to intrathoracic surgery. It is sometimes difficult to distinguish mitral and aortic valve. A line drawn from right cardiophrenic angle to the pulmonary conus, help us do that. A valve below that line is mitral valve and above is the aortic valve.

- How do evaluate patients with prosthetic valves coming for non-cardiac surgery?
- How do you manage anticoagulation in these patients? When do you stop Warfarin?
- How to anticoagulate the patient once Warfarin is stopped? What are the differences between unfractionated and low molecular weight heparin?
- When do you stop heparin before surgery and when do you restart it after surgery?
- What other drugs (antibiotics) do you need to prescribe before surgery?
- How do you manage postoperative bleeding?

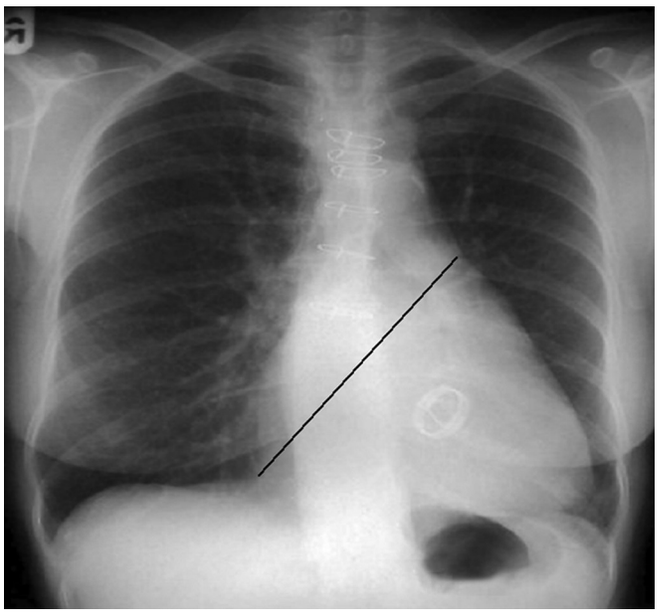


Fig. 50.15 CXR 14. Prosthetic valve (Mitral)

CXR 15. Cardiomegaly with pulmonary edema

- What are the common causes of cardiomegaly?
- What are the causes of pulmonary edema? (Cardiogenic vs. non-cardiogenic, left ventricular failure (ischemic, cardiomyopathy), valvular heart disease (Aortic/mitral), near drowning, aspiration, ARDS, altitude, raised intracranial pressure, renal failure and fluid overload)
- How do you treat cardiogenic pulmonary edema? How does PEEP reduce left ventricular afterload?
- What causes neurogenic pulmonary edema?
- What is negative pressure pulmonary edema? What can cause unilateral pulmonary edema?

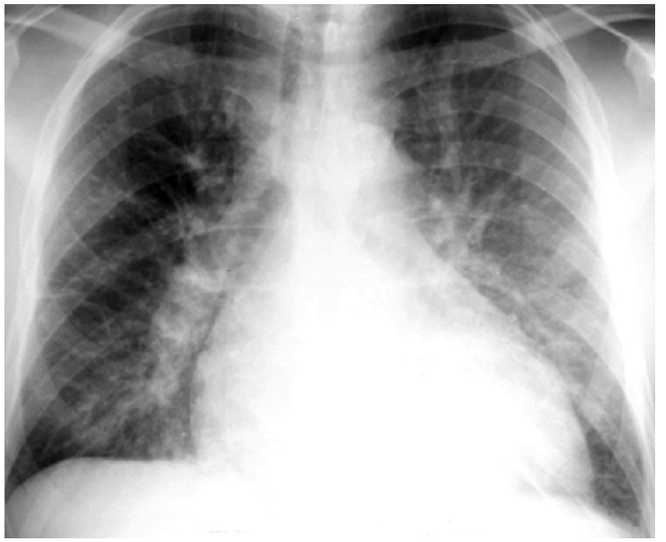


Fig. 50.16 CXR 15. Cardiomegaly with pulmonary edema

Reading cervical spine X-rays

Cervical spine clearance is important in trauma patients, particularly those with extensive TBI and when the spine cannot be cleared clinically. Lateral neck X-ray also gives an idea about the airway. Adequate film of c-spine must include skull base to upper body of first dorsal vertebra. The smoothness of five lines (marked in the figure below) indicates preserved integrity of cervical spine (at least non-displaced fractures). Also widening of the space between first and second line (retropharyngeal space) beyond 7 mm indicates a retropharyngeal collection, hematoma or abscess. In such circumstances patient might have difficulty in breathing and or difficult intubation. Please also check the height of cervical spines. A compressed fracture may not mar the smoothness of the lines but fracture may be present nonetheless.

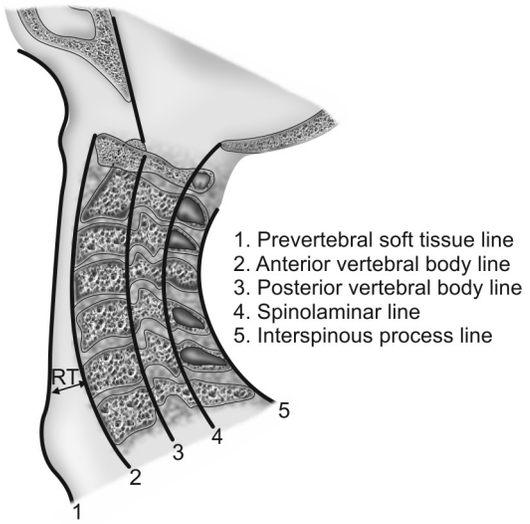


Fig. 50.17 Cervical spine

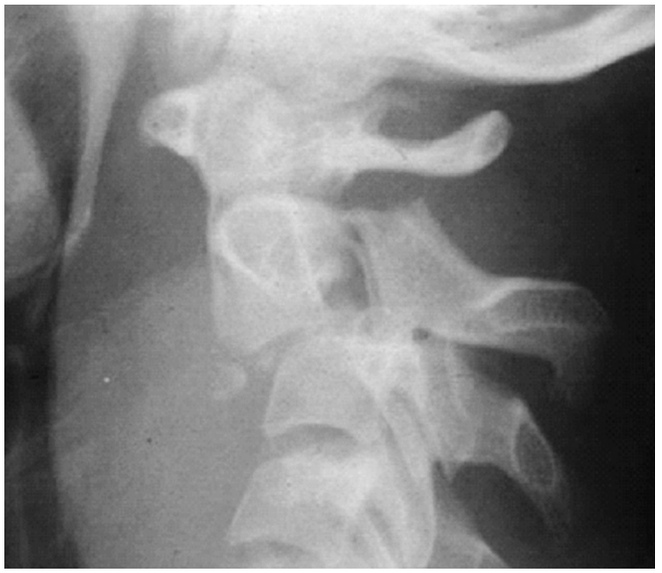


Fig. 50.18 CXR 16. Cervical spine fracture with widened retropharyngeal space

XR 16 shows cervical spine fracture with widened retropharyngeal space. The likely questions with such X-rays will be regarding management of the airway in a trauma patient, methods adopted for intubating these patients and other general management of trauma patients.

Suggested Reading

1. Hopkins R, Peden C, Gandhi S. Radiology for Anaesthesia and Intensive care. London: Greenwich Medical Media.
2. Corne J, Pointon K. Chest X-ray made easy. Churchill Livingstone Elsevier.

51 Supraglottic Airway Devices

V Agarwal, A Kulkarni

Who developed laryngeal mask airway (LMA)?

Dr Archie Brain developed LMA in 1982 at Royal London Hospital by as a modification of the Goldman Dental Mask.

Compare LMA with a face mask.

In comparison to a facemask the advantages of LMA are:

- It provides a more secure and reliable means of ventilation than the face mask.
- The mandible does not need to be supported; as a result hands of the anesthetist are not occupied and do not fatigue.
- Can be easily inserted by a clinical/non-clinical staff as it requires minimal training, particularly during cardiopulmonary resuscitation.
- Can be used in patients with abnormal facial contour provided the mouth opening is adequate for LMA insertion.
- Lower risk of aspiration with LMA (0.012%).

In comparison to a facemask the disadvantages of LMA:

- It does not ensure absolute protection against aspiration but is superior to a face mask.
- Postoperative sore throat (17% vs 3% for LMA and face mask respectively).

Compare LMA with an endotracheal tube.

In comparison to an endotracheal tube the advantages of LMA are:

- Reduced stress response as compared to laryngoscopy and intubation and during emergence.
- Minimal increase in intraocular pressure following insertion.
- Reduced requirement of anesthetic agents for airway tolerance.
- Neuromuscular blockers are not needed for insertion.
- Can be easily inserted by a clinical/non-clinical staff as it requires minimal training, particularly during cardiopulmonary resuscitation.
- Lower incidence of postoperative sore throat compared to endotracheal tube (45% vs 17% for endotracheal and LMA respectively).
- It can be inserted easily in patients with cervical spine injury with immobilization of the head and neck.
- Insertion of LMA does not require laryngoscopy and/or visualization of the vocal cords.
- It provides effective ventilation almost similar to endotracheal tube.

In comparison to an endotracheal tube the disadvantages of LMA are:

- It does not ensure absolute protection against aspiration but is superior to a face mask.
- It is contraindicated in patients at risk of acid aspiration (e.g. history of gastroesophageal reflux, hiatus hernia, gastroparesis as in diabetes mellitus, inadequate duration of fasting).
- Lower seal pressure, so if ventilated with high pressures can lead to gastric insufflation and predispose to regurgitation of gastric contents.

Can you describe a classic LMA?

It is supraglottic airway device used for anesthesia and maintaining patency of the upper airway. It forms a low-pressure seal around the laryngeal inlet and thus permits gentle positive pressure ventilation.

It consists of an elliptical silicone mask with an inflatable outer rim, a pilot tube and balloon, a latex free silicone tube, a standard 15 mm male adaptor attached to the inflatable outer rim. The elliptical cuff has bars on the laryngeal inlet surface.

- 40 uses
- Autoclaving–sterilization

Fig. 51.1 Classic LMA

How will you decide the size of the LMA for a patient and what are the cuff inflation volumes?

LMA is available in various sizes and depends on the body weight of the patient.

Body weight (kg)	Appropriate LMA size	Air volume (mL)
Upto 5	1	4
5–10	1.5	7
10–20	2	10
20–30	2.5	14
30–50	3	20
50–70	4	30
70–100	5	40
>100	6	50

What preparation is necessary prior to insertion of LMA?

- Examine the surface of the LMA for damage, including cuts, tears, or scratches.
- Examine the 15 mm connector to ensure it fits tightly into the airway tube. Do not twist the connector as this may break the seal.
- Carefully insert a syringe into the pilot balloon valve port and fully deflate the cuff so that the cuff walls are tightly flattened against each other. Examine the cuff walls to determine whether they remain tightly flattened.
- Over inflate the cuff with air from complete vacuum and look for any signs of leaks.
- Prior to insertion, deflate the cuff tightly so that it forms a spoon shape. This may be accomplished by pressing the aperture side down onto a flat surface with the non dominant hand. Alternatively the cuff deflator device can be used.
- Lubricate the posterior surface of the LMA with water-soluble lubricant just before insertion. Lignocaine jelly is not suitable for this purpose as it has been shown to increase the incidence of postoperative sore throat, Lignocaine application also anesthetises the airway predisposing the patient to the risk of acid aspiration postoperatively.

Describe the technique of LMA insertion.

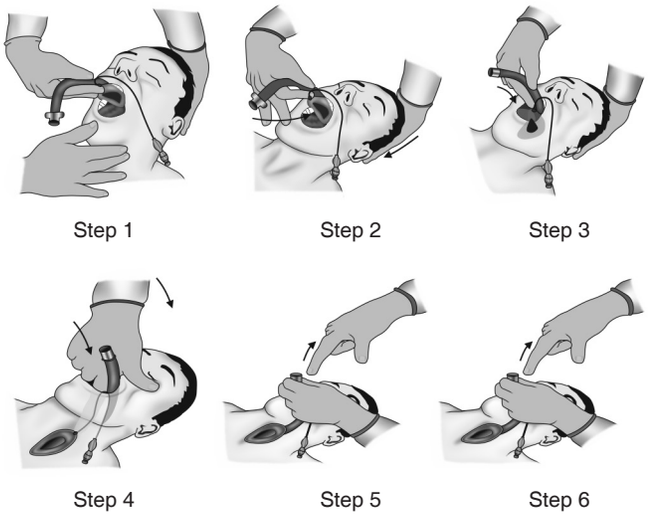


Fig. 51.2 Techniques of LMA insertion

- Position the patient with the neck flexed and head extended
- After the necessary preparation once the patient is anesthetized; grasp the LMA by the tube, holding it like a pen as near as possible to the mask end
- Place the tip of the LMA against the inner surface of the patient's upper teeth
- Insert the mask into the oral cavity with the tip directed upwards against the hard palate
- Using the index finger, glide the LMA along the hard palate into the pharynx to ensure the tip remains flattened and avoids the tongue
- Advance the mask into the pharynx with the dominant hand index finger till it cannot be advanced any further, then with the non-dominant hand hold the tube in place and withdraw the dominant hand
- Gently push the LMA into the pharyngeal cavity to ensure the mask is fully inserted
- Inflate the mask with the recommended volume of air
- The LMA rises up slightly out of the hypopharynx; as it is inflated to find its correct position (Fig. 51.2).

How is the LMA positioned at the laryngeal inlet?

When appropriately positioned, the distal tip of the silicone cuff rests against the upper esophageal sphincter, the sides

of the cuff in the pyriform fossa and the upper part of the cuff against the tongue base.

What are the indications and contraindications for LMA?

The indications for LMA use are:

- The LMA may be used as an alternative airway during general anesthesia in elective surgical cases.
- It is an essential part of the difficult airway trolley, as it can be used in both anticipated and unanticipated difficult airway to allow ventilation in case there is difficult mask ventilation.
- During cardiopulmonary resuscitation it can be used to secure an airway.
- Relative indication in professional singers; to avoid complications of endotracheal intubation.

The contraindications to LMA use are:

- Any patient at risk of aspiration e.g. patients undergoing emergency surgery.
- Patients with major local pathology in the pharynx and larynx such as tumor, abscess, edema, and/or hematoma.
- Patient with trismus, facial or upper airway trauma.
- Patients who are morbidly obese, more than 14 weeks pregnant, have received prior opioids medication or any condition associated with delayed gastric emptying.
- Patients with reduced lung compliance/increased resistive work of breathing such as pulmonary fibrosis, status asthmaticus. LMA forms a low pressure seal around the laryngeal inlet and ventilating patients with these conditions may require higher pressure and so the ventilation may be inadequate. Also gastric insufflation may occur as ventilation is delivered at higher pressure and gases may leak around the cuff as the sealing pressure is low (20 cmH_2O).

What is the seal pressure for LMA?

The seal pressures for classic LMA, unique LMA, flexible LMA is 20 cmH_2O and for ProSeal LMA it is 30 cmH_2O.

What are the common causes of LMA malposition?

The most common causes of LMA malposition are:

- Placement during inadequate depth of anesthesia and / or suppression of airway reflexes (pharyngeal muscle and/ or laryngeal spasm)
- Inappropriate size selection
- Correct placement may be more difficult in patients with a small mouth, a large tongue and/or tonsils. It larynx is posteriorly placed; it blocks the advancement of the tip of the LMA into the hypopharynx.

What are the complications or adverse effects of LMA?

The most common complication is postoperative sore throat (the incidence is as mentioned earlier).The other adverse events reported are arytenoid dislocation, trauma to the pharyngeal cavity, airway edema, tongue edema, rarely hypoglossal nerve injury, tongue numbness secondary to lingual nerve injury, tongue macroglossia, recurrent laryngeal nerve injury, inferior alveolar nerve injury and vocal cord paralysis. The nerve injuries are probably due to malposition of LMA or excessive intracuff pressure; which causes compression of nerves and/or blood vessels. Cuff malposition or excessive cuff pressure may be exacerbated by incorrect mask size, prolonged surgery, and use of nitrous oxide. Simply monitoring the cuff pressure intraoperatively (and evacuating air to maintain constant cuff pressure) was shown to reduce the incidence of postoperative sore throat. A study found that use of a simple device called continuous cuff pressure regulator allowed the cuff pressure to be maintained at a constant level. (max intracuff Pr < 60 cm OF H_2O)

Negative pressure pulmonary edema has been reported particularly during emergence from anesthesia. Large subatmospheric negative pressure may develop if the patient tries to breathe against either closed glottis (due to laryngospasm) or against closed tube (when patient bites the tube). Reports also suggest that proSeal use might be more likely to be associated with this rare but disastrous complication as compared to the other LMAs.

What is the intubating LMA (ILMA or LMA Fastrach)? Can you describe it?

As the name suggests, it was designed to act as a conduit for endotracheal intubation (Figs 51.3 and 51.4). It consists

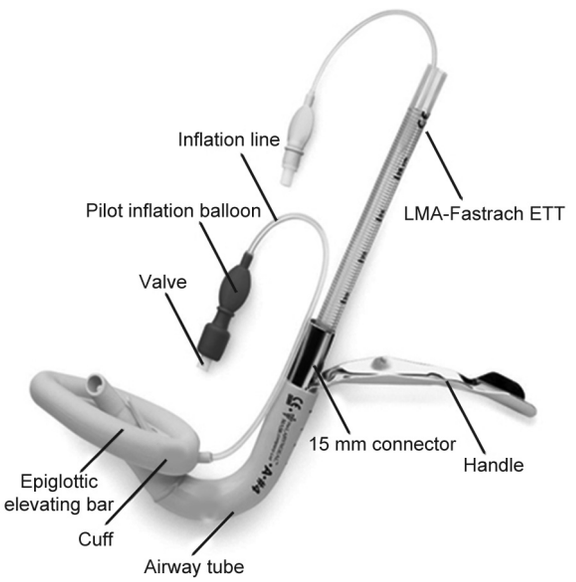


Fig. 51.3 LMA Fastrach

Fig. 51.4 Single use LMA Fastrach

of all the parts similar to the classic LMA however; there are certain differences. It has an elliptical silicone cuff with rigid anatomically curved steel airway tube cuff which is in turn attached to a rigid handle. The silicone cuff consists of an epiglottic elevating bar instead of the grid as in the classic LMA, which helps in the passage if the endotracheal tube. Preparation of the ILMA and patient is similar to the classic LMA.

What are the available sizes and inflation volume?

Body weight (Kg)	ILMA size	Air volume (mL)
30–50	3	20
50–70	4	30
70–100	5	40

Describe the insertion technique for ILMA.

After preparation and head in the neutral position, the ILMA is introduced in the oral cavity holding the rigid handle parallel to the patient's chest. Glide the mask along the palate till the straight part of the rigid tube is parallel to the chin, thereafter rotate the rigid handle directing towards the patient's nose till it cannot be advanced any further. Inflate the cuff with appropriate volumes taking care not to exceed the intracuff pressure > 60 cmH_2O. Secure the ILMA in place.

Describe the LMA Fastrach endotracheal tube and the technique of inserting this tube through ILMA

LMA Fastrach endotracheal tube (FETT) is a straight, reinforced (with embedded spiral wire in the wall) cuffed endotracheal tube with an internal diameter of 8.0 mm and can be passed easily through the ILMA. The ETT has a marker, a transverse line, at 15 cm; this coincides with the epiglottic elevating bar in the elliptical cuff. It is recommended to use this tube with the ILMA. Using the conventional endotracheal tube (ETT) through the ILMA can lead to laryngeal trauma.

Prior to insertion, fit the connector to the FETT. Lubricate it well. The LMA FETT has a longitudinal black line on the tube; introduce the tube with this line facing the rigid handle of the ILMA. The FETT should not be introduced beyond the 15 cms mark. Now grip the LMA handle firmly and lift (without levering) it forwards by a few millimeters, this increases the seal pressure and optimally aligns the tracheal and FETT axes (the Chandy maneuver). The mask may sometimes have a tendency to flex and the FETT can then pass in the esophagus rather than the trachea, this is avoided by the Chandy maneuver. Thereafter advance the FETT gently beyond the 15 cms mark; at this point it is lifting the epiglottic elevating bar. Advance the FETT further using clinical judgment. Inflate the FETT and attach it to the breathing circuit to check and confirm endotracheal position.

What is the Chandy's maneuver?

Chady's maneuver consists of two steps. The first step is rotating the LMA in coronal and sagittal plane in an attempt to find a position that offers least resistance to ventilation. The second step consists of grasping the handle firmly and use it to draw the LMA forward 2–5 mm in a lifting action without levering on the teeth. This maneuver increases seal pressure and aligns the axes of the trachea and FETT and facilitates blind intubation.

What are the causes of failed intubation through the ILMA?

The causes are:

- Folding of the epiglottis downwards
- Inappropriate size of the ILMA
- Inadequate depth of anesthesia/neuromuscular blockade.

How will you remove the ILMA prior to extubation?

It is recommended to remove the ILMA following insertion of FETT as it can lead to pharyngeal edema and increased mucosal pressure because of the rigid steel tube in the ILMA. If the ILMA has to be kept in situ, the cuff of the ILMA should be deflated to a pressure of 20–30 cmH_2O.

- With the stabilizer rod measure the distance between the proximal end of the FETT and the patient's incisors.
- Administer 100% O_2 for few minutes and then disconnect the FETT and the breathing circuit.
- Deflate the ILMA completely but keep the FETT cuff inflated.
- Gently displace the ILMA along the pharyngeal curvature till the FETT machine end tip is at the level of the universal

connector of the ILMA. Now insert the stabilizing rod in the FETT to keep it in place.
- Remove the ILMA gently over the stabilizing rod until it is clear of the oral cavity.
- Stabilize the FETT to prevent accidental extubation.
- Remove the ILMA and the stabilizing rod and reconnect the FETT to the breathing circuit and confirm position again.

Describe ProSeal LMA.

The ProSeal LMA (PLMA) (Fig. 51.5) was designed especially for use with positive pressure ventilation at high pressures (in contrast to Classic LMA which can be used only with low pressure ventilation). The uses, indications and contraindications and limitations of ProSeal LMA are similar to the Classic LMA. Only the different features of ProSeal LMA are mentioned below.

- The elliptical cuff of the ProSeal extends to the posterior or the pharyngeal side of the PLMA thus improving the seal pressure. (Dorsal Cuff)
- The mask has two tubes attached to it, a drain tube and a reinforced airway tube and a built in bite block. The double tube arrangement prevents device rotation.
- The mask has a drain tube incorporated within the cuff which communicates with the upper esophageal sphincter permitting drainage of the gastric contents and insertion of gastric tube. The position of the drain tube inside the cuff is designed in such a way to prevent the epiglottis from occluding the airway tube, thus eliminating the need for aperture bars.
- The reinforced airway tube prevents kinking.
- The built in bite block reduces the possibility of airway obstruction.
- The mask has a strap for PLMA introducer anteriorly between the tube and mask. This also allows insertion of finger or thumb for manual insertion as described for the Classic LMA

Fig. 51.5 ProSeal LMA

- PLMA can also be inserted by inserting a bougie in the drain tube and then passing the bougie in the esophagus after a gentle pharyngoscopy. The PLMA is then guided over the bougie in place. Since the bougie is already in esophagus, the PLMA automatically sits in front of the laryngeal inlet. The bougie may be left in place during surgery, if it is not in the way of the surgeons; this lends stability to the PLMA.

Discuss in brief about LMA supreme.

LMA Supreme™ (Fig. 51.6) is a latex free single use LMA similar to the ProSeal LMA. The tube part is rigid, preshaped to adopt to the contour of the anatomy. It also has a fixation tab for securing the LMA after insertion; and a built-in bite block and two ports: the airway port and the drain tube port. The cuff has molded fins which prevent epiglottis from obstructing the airway lumen. The tip of the cuff is reinforced to prevent it from folding during insertion. The patients can be ventilated with pressures up to 37 cmH_2O. Insertion is similar to that of the ProSeal LMA with a introducer, except no introducer is required. The tip has an opening for the gastric tube. Intubation is not possible with LMA Supreme. It is available in both adult and pediatric sizes.

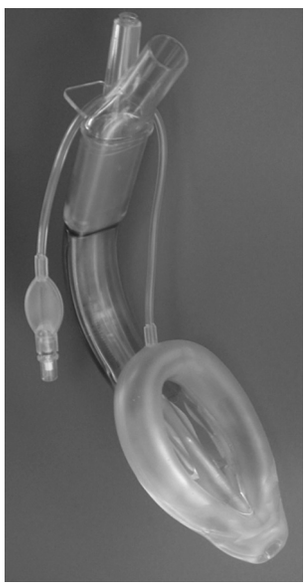


Fig. 51.6 LMA supreme

What other supraglottic airway devices are available? Describe these devices.

In the past, the choice of an airway device essentially was limited to the facemask or the endotracheal tube (ETT).

Tracheal intubation is the "gold standard" for securing the airway and providing adequate ventilation. However, training in tracheal intubation requires time, appropriate instruments, and adequate circumstances with respect to space and illumination. Furthermore, it requires continued practice and has its own set of complications.

The practice of airway management has become more advanced in recent years. This advancement is demonstrated by the introduction of new airway devices, several of which have been included in the American Society of Anesthesiologists (ASA) Difficult Airway Algorithm. The standard laryngeal mask airway was introduced into clinical practice in 1988 and rapidly transformed airway management. Since then, there has been an explosion of new supraglottic airway devices to compete with the Classic LMA, particularly single-use devices. Supraglottic airway (SGA) is a general term that includes airways with and without sealing characteristics. According to Miller, there are 3 main sealing mechanisms. The explanation for each provides reasons for the differences observed in the sealing pressures achieved with each of the different types of devices. The indications, contraindications, advantages and disadvantages of the supraglotttic airway devices are similar to the laryngeal mask airway. One study found that I-gel produced less increase in intraocular pressure (and LMA Supreme) and lower hemodynamic response than endotracheal intubation.

LMA Classic Excel

It differs from the Classic LMA (Fig. 51.7), such as it has an increased angle which aids intubation, armored airway connector with a removal 15 mm adaptor which helps during fiber optic intubation. Because of the reinforce airway tube it can be reused up to 60 times making it more cost-effective. The airway tube can accommodate and ETT size 7.5 mm ID. The silicone cuff has an epiglottic elevating bar to aid intubation.

Fig. 51.7 LMA Classic Excel

LMA Unique

It is single use version of Classic LMA (Fig. 51.8).

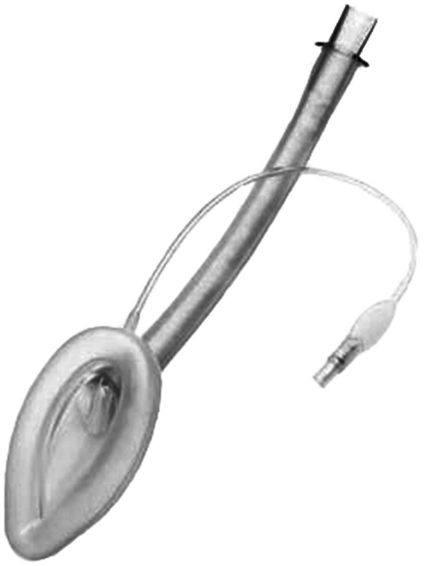


Fig. 51.8 LMA Unique (Disposable classic version)

Flexible LMA

As the name suggests it has a flexible wire reinforced airway tube which makes it suitable for head and neck surgeries. The length of the airway tube is longer and the diameter smaller compared to the other LMA devices. The length and the flexibility makes it is easier to move away from the surgical area. Because of the length it is not suitable for endotracheal intubation. It is available in both pediatric and adult sizes.

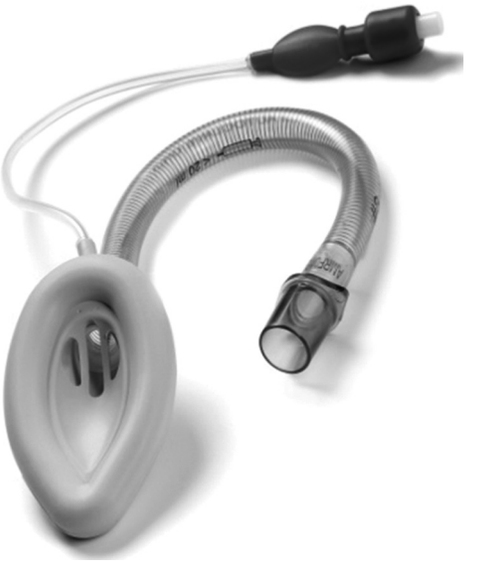


Fig. 51.9 Flexible LMA

Miller's supraglottic classifications

	Cuffed perilaryngeal sealers		Cuffed pharyngeal sealers		Cuffless preshaped sealers
	Without directional sealing	With directional sealing	Without esophageal sealing cuffs	With esophageal sealing cuffs	
Devices	LMA ILMA SSLM Ambu LM ILA	PLMA	CobraPLA	LT LTD ETC	SLIPA Baska MaskTM
Sealing mechanism	Cuff surrounds the laryngeal inlet. This limits the seal pressure due to direction of forces by the cuff	Forms a seal at the laryngeal inlet and the seal pressure is improve means of directional sealing cuff	Pharyngeal cuff seals at the base of the tongue		Seals the outlet from pharynx at the base of the tongue right up to the esophageal inlet because of the tough walls Baska Mask the preformed cuff expands with each PP breath

Abbreviations: LMA—Classic laryngeal mask airway; ILMA—Intubating LMA; SSLM—Softseal laryngeal mask; ILA—Intubating laryneal airway; PLMA—ProSeal LMA; Cobra PLA—Cobra perilaryngeal airway; LT—Laryngeal tube; LTD—Laryngeal tube with suction; ETC—Esophageal tracheal combitube; SLIPA—Streamlined liner of the pharynx airway. (Adapted from Miller: Anesth Analg. 2004;99:1553–9)

Fig. 51.10 Single use Flexible LMA

Classify SGA devices.

The SGA devices can be classified as:

Cuffed perilaryngeal sealers for example, Ambu Laryngeal mask, SSLM (Softseal laryngeal mask), ILA (Intubating laryngeal airway), LMA, ILMA

Cuffed pharyngeal sealers for example, CobraPLA (Cobra perilaryngeal airway).

Cuffless preshaped sealers: for example SLIPA.

Describe the insertion technique of the SGA devices.

The method of device insertion technique is similar for most of the supraglottic devices and therefore to avoid repetition, a generic method of device insertion is described below. Wherever the technique differs it is mentioned with device.

Insertion Technique

It is important that the individual who inserts and/or ventilates through SGA is familiar with the warnings, precautions, indications, and contraindications in the use of that particular device. Before insertion, the following points are of the highest importance:

- The size of the SGA must be appropriate for the patient. Use the SGA recommendations and clinical judgment to select the correct size.
- If the device is too small, clinicians tend to overinflate the cuff to achieve seal, this may lead to complications.
- Check the SGA cuff and lubricate with water soluble lubricant as directed.
- Do not use excess force for insertion.
- Preoxygenate and implement standard monitoring procedures.
- Ensure adequate depth of anesthesia before attempting insertion.Trainees or inexperienced users should insert SGA'a when the patient is at a deeper plane of anesthesia.
- If not contraindicated, use the "sniffing position" (neck flexed and head extended) for insertion.
- The device should be held between the fingers and thumb of the dominant hand, the line on the tube aligned midline and direct it against the hard palate. One may have to use jaw lift maneuver for insertion.The airway should be advanced into the oropharynx with gentle twisting motion to negotiate the tongue and tonsils.With the non-dominant hand hold the 15 mm connector and push the airway caudally until resistance is encountered.

- The cuff should be inflated with air until a "just-seal" pressure is obtained. To ensure an adequate seal and minimal trauma it is recommended to maintain intracuff pressure to a maximum of 60 cmH_2O. Pressure higher than this increase the incidence of sore throat and may cause nerve injuries as well.
- After the cuff inflation the airway or tube moves out slightly and fullness is noticed in the neck indicating correct placement.
- The airway or tube is connected to the breathing system and adequacy of ventilation assessed.
- Indicators of correct SGA placement are: Auscultation of bilateral lung sounds, bilateral chest excursion, absence of gastric insufflation, and capnography.
- A bite block may be inserted to prevent tube occlusion during emergence from anesthesia
- Tube should be taped on to the patient's face.

Describe the ambu laryngeal mask

It is a cuffed perilaryngeal sealer. The Ambu Laryngeal Mask (ALM) (Fig. 51.11) is a sterile, single-use product made of polyvinylchloride (PVC), moulded in one piece. It has a built- in curve replicating the anatomy of the oropharynx-hypopharynx. The curve ensures that the patient's head remains in a natural, supine position when the mask is in use. It has a reinforced tip; which along with the curve; facilitates insertion of the mask.The internal ribs in the curve make the airway tube flexible to adapt to individual anatomical variations and head positions. The cuff is elliptical and when inserted rests in the hypopharynx at the base of the tongue. The cuff does not have epiglottic bars and this allows easy access for fiberoptic bronchoscope. It has a single inflation line. It is latex-free and is available in both adult and pediatric sizes.The proper size is based according to the weight and size of the patient. Ambu® Aura40™ is a reusable Ambu LMA, where as Aura- i™ and AuraOnce™ are disposable products. Ambu AuraFlex™ has a flexible tube without a built in curve.

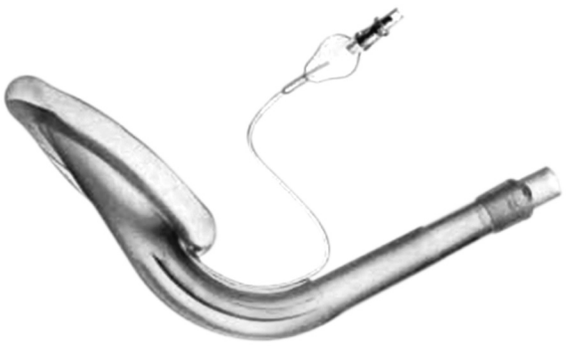


Fig. 51.11 Ambu laryngeal mask

Patient weight (kg)	Size	Maximum cuff volume (mL)
30–50	3	20
50–70	4	30
70	5	40

Describe the cuffed pharyngeal sealers—laryngeal tube and the laryngeal tube suction™ (LTS).

The LT consists of a silicon reusable airway tube provided with two cuffs (pharyngeal and esophageal) a single balloon for pressure control and a 15 mm standard adapter, which is designed to be reused up to 50 times.The airway tube is short and "J" shaped, has an average diameter of 11.5 mm

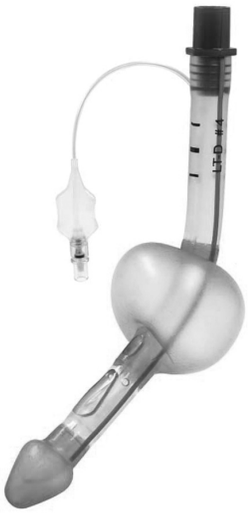


Fig. 51.12 Laryngeal tube

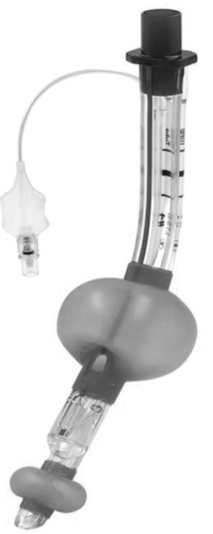


Fig. 51.13 Laryngeal tube suction

and a blind tip (Fig. 51.12). It is suitable for neonates to large adults. LT size selection is based on the patient's weight for infants and children and height for adults.

Size	Use	Size	Color
0	Infants < 5 kg	0	Transparent
2	Children 12–25 kg	2	Green
3	Adults with height <155 cm	3	Yellow
4	Adults with height 155–180 cm	4	Red
5	Adults with height >180 cm	5	Violet

After device insertion, the proximal cuff lies in the hypopharynx and the distal in the upper esophagus. Both cuffs are high volume and low pressure to avoid ischemic damages and permit a good seal. The proximal cuff is protected by a bend located in a "V" dent of the pharyngeal cuff. With cuff inflation, soft tissue is deflected from this opening, helping to maintain a patent airway. A wedge shaped block closes the tip of the tube, thus diverting the ventilation to the trachea. The cuffs should be inflated, with the aid of the laryngeal cuff pressure gauge, to 60 cmH_2O. Due to a single inflation line, both cuffs will inflate simultaneously.

Three side eyelets on each side allow improved collateral ventilation; should the epiglottis obstruct the main ventilation orifice. This version has also been manufactured 1.0 cm longer to facilitate deeper placement of the device, thus assuring proper positioning of the two main ventilation orifices and location of the proximal cuff beneath the tongue so that the epiglottis is more likely to be raised with cuff inflation in much the same manner as with a Macintosh laryngoscope blade.

The Laryngeal Tube Suction™ II (LTS-II) (Fig. 51.13) is double lumen silicon version of the LT. The additional lumen is situated behind the lumen for ventilation. This lumen is for insertion of gastric tube allowing suction of gastric contents.

Insertion technique and device removal

- The device has to be steam autoclaved, disposable versions are available.
- The LT should be held in the dominant hand and inserted blindly along the midline of the tongue sliding the tip along the hard palate until resistance is felt.
- The cuffs can then be inflated with the Cuff Pressure Gauge up to 60 cmH_2O. Alternately a syringe can be used, cuff inflation volumes vary with the size.
- Due to the single inflation line, the proximal cuff fills first stabilizing the tube according to the anatomy of the patient, the distal cuff then inflates automatically.

Mask Size	Maximum Cuff Volume (ml)
0	10
1	20
2	35
3	60
4	80
5	90

- The LT is well-tolerated until the return of the protective reflexes. Slight cuff deflation at this point allows better toleration of the oropharyngeal cuff.
- The device should be removed with the patient either deeply anesthetized or totally awake, otherwise laryngospasm, coughing or gagging may occur.
- Before removal of the device the cuffs should be completely deflated.Inadequate cuff deflation can make removal difficult, risking cuff damage and discomfort for the patient.

Indications and advantages

- The slim profile of the LT allows easy insertion with small mouth opening, thus it can be considered for airway management in patients with restricted mouth opening.
- In difficult airway situation can be used as an emergency device to secure airway.

LT as a guide to endotracheal intubation

Tracheal intubation can be done blindly through the LT using a small size endotracheal tube (ETT) (up to 6.5 mm). The ETT should be well-lubricated and the connector removed. If there is any resistance it is likely to be secondary to incorrect positioning of the LT, obstruction by the epiglottis. After correct placement of ETT, the LT cuffs should be deflated and the device can then be removed. The diameter of the ETT is limited by the LT size. Removing the LT over the ETT can be problematic, so in such a situation when a larger diameter ETT is desired, tube exchanger can be used. ET intubation can also be performed fiberoptically using airway exchange catheter.

Discuss the cuffed pharyngeal sealer—Cobra PLA™.

The CobraPLA™ (Pulmodyne®) (Fig. 51.14) consists of a breathing tube with a distal circumferential inflatable cuff proximal to the ventilation outlet. It is named so because of the widened distal part resembles the hood of the Cobra. This distal part prevents soft tissue collapse and allows positive pressure ventilation. When in properly inserted; the Cobra head sits over glottis and seals off the hypopharynx. Tip of the CobraPLA lies proximal to the esophageal inlet unlike other SGAs. A ramp present within this head, which directs the ventilation or a blindly passed ETT into the glottic aperture.

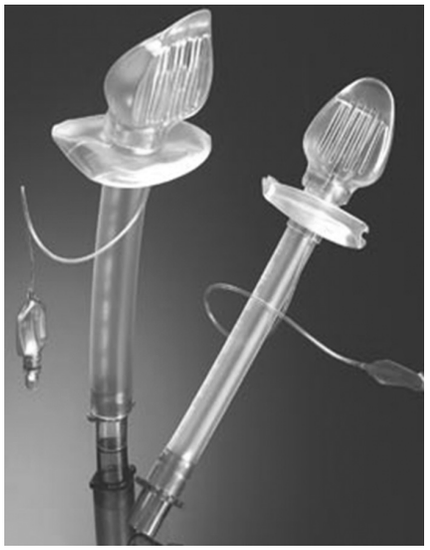


Fig. 51.14 Cobra PLA

A soft flexible grill placed anteriorly prevents epiglottis from downfolding. The grill allows passage of an ETT when required. It has a circumferential cuff which rests in the hypopharynx at the vallecula. When inflated, it lifts the base of the tongue exposing the glottic aperture, and also seals the airway. The CobraPLA is available in 8 sizes according to the weight and size of the patient including adult and pediatric.

CobraPLA sizes according to weight of the patient

Size	Weight (kg)
1/2	2.5 – 7.5
1	5 – 15
1 1/2	10 – 35
2	20 – 60
3	40 – 100
4	70 – 130
5	100 – 160
6	> 130

CobraPLA cuff inflation volumes suggested by manufacturer.

Size	Volume (ml)
3	< 65
4	< 70
5	< 85

Insertion

- Deflate the cuff completely and fold it back against the breathing tube.
- Generously lubricate front and the back of the Cobra head and the cuff, but take care not to obstruct the grill.
- Position the patient's head is positioned in the sniffing position and open the mouth with the left hand.
- Direct the distal end of the CobraPLA between the tongue and hard palate and not the hard palate. Once fully inserted in the mouth, perform a jaw lift and insert the CobraPLA.
- Pushing the jaw downwards makes insertion more difficult. Slight neck extension may aid passage of the device, as it turns towards the glottis at the back of the mouth.
- When positioned properly, the flexible tip is situated below the arytenoids, the cuff lies in the hypopharyx at the vallecula and the ramp and grill elevate the epiglottis.
- Once you are happy that CobraPLA is properly inserted, inflate the cuff. Do not use the maximum volume advocated by the manufacturers. Rather inflate slowly while ventilating so that you get no leak. This is called the minimal leakage technique.
- Confirm correct placement by gentle positive pressure ventilation (Paw not greater than 25 cmH_2O).
- Measure and document the pressure at which an audible leak is heard.

Removal

- Once the patient becomes conscious deflate the cuff partially and gently withdraw the device from the mouth. This helps in removing collected secretions from patients oral cavity.

Size of ETT that can pass through various sizes of CobraPLA

Cobra size	ET size (mm)
1/2	3.0
1	4.5
1 1/2	4.5
2	6.5
3	6.5
4	8.0
5	8.0
6	8.0

Discuss the cuffless preshaped sealer—streamlined liner of the pharynx airway™ (SLIPA)

The SLIPA™ is a latex free device and it is shaped like a slipper (Fig. 51.15). It is preformed airway that is a cuff-less and lines the pharynx when in place. It is made of medical grade thermoplastic material that is stiff enough to facilitate easy insertion. After insertion it warns up to body temperature and softens, improving the seal and comfort. It has a hollow, chamber similar to a boot in shape. There is a toe which sits at the upper end of esophageal opening, a bridge that comes to rest at the valeculla. Finally, the heel of SLIPA lends stability to the device by fitting in the nasopharynx. The bridge in the

Fig. 51.15 SLIPA

center of the chamber has two lateral bulges: these bulges occupy the pyriform fossa of the larynx. These prevent downfolding of the epiglottis in front of glottic opening.

The chamber has a variable capacity (maximal of 50 mL SLIPA of size 53). This can store secretions and gastric contents, preventing their aspiration in the airway. It is available in six adult sizes: 47, 49, 51, 53, 55, and 57. The numbers correspond to the maximal transverse diameter (in mm) of the bridge. The best way of selecting SLIPA of the correct size is to see the distance between two cornu of the thyroid cartilage and selecting a size that will match the width of the bridge.

Patient size	SLIPA size	Median height	Height range
Very small female	47	152 cm (5'0")	145–160cm (4'9"–5'3")
Small female	49	160 cm (5'3")	152–168 cm (5'0"–5'6")
Medium female	51	168 cm (5'6")	160–175 cm (5'3"–5'9")
Large female - Small male	53	173 cm (5'8")	163–182 cm (5'4"–6'0")
Medium male	55	182 cm (6'0")	173–193 cm (5'8"–6'4")
Large male	57	190 cm (6'3")	180–200 cm (5'11"–6'7")

Note: If unsure between two sizes, choose the larger size

Insertion technique and device removal

- Unlike LMA, we do not have to glide SLIPA along the hard palate. Its preformed curve allows the device to pass oropharyngeal curve easily.
- The head and neck are in sniffing position and after opening the mouth (and doing a forward jaw lift if required), the SLIPA is advanced towards the esophagus. The heel of the device will automatically fits into the nasopharynx.
- The shape of the toe reduces the chances of airway obstruction, due to any reasons i.e. either laryngospasm or downfolding of epiglottis.

Indications and advantages

- The SLIPA is a cheap uncomplicated disposable device. The risk of aspiration is minimal with SLIPA.
- The SLIPA design has a large empty compartment, which has a space for collection of almost 50 mL oropharyngeal content, therefore the patient may be protected against aspiration of gastric content.
- Low cost compared to other SGA's.

Disadvantage

- There are no studies evaluating use of SLIPA used as conduit to fiberoptic bronchoscopy and intubation.
- Selecting the correct size is difficult.
- First attempts at insertion are successful in 76–85% patient.

I-Gel Supraglottic Airway

I-gel is a soft, gel like, cuffless supraglottic airway made of thermoplastic elastomer (Fig. 51.16). It creates a noninflatable anatomical seal of the pharyngeal, laryngeal and perilaryngeal structures. Because of its noninflatable seal it avoids compression trauma that can occur with inflatable supraglottic airway devices.

Design of I-gel, makes it an anatomical device as the distal soft non-inflatable cuff achieves a mirrored impression of the pharyngeal, laryngeal and perilaryngeal structures and thus positions itself over laryngeal framework providing a reliable perilaryngeal seal. The tip of the distal non-inflatable cuff lies in the proximal opening of the esophagus, isolating the esophageal opening from the laryngeal inlet. The proximal end of the cuff has an epiglottic rest with a ridge. The rest prevents the epiglottis from down folding whereas the ridge is in contact with the base tongue preventing the upward and outward movement of the I-gel.

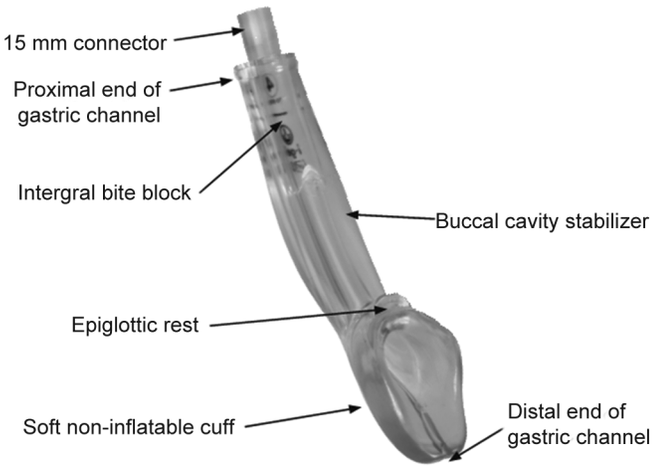


Fig. 51.16 I-Gel supraglottic airway

It consists of elliptical buccal cavity stabilizer which incorporates a circular airway lumen and a lumen for gastric tube insertion. The elliptical shape provides vertical stability and axial strength after insertion. Size 1 I-gel does not have a gastric lumen. It also has a built in bite block incorporated within the tube. There is a horizontal line in the integrated bite block (in Size 3, 4, and 5) which is guide for correct depth of insertion. If correctly inserted should coincide with the patient's teeth. The pediatric sizes do not have the horizontal line due to the greater variability in the length of the oro-pharyngeal-laryngeal arch in children.

Advantages

- Provides minimal risk of tissue compression because of the noninflatable cuff in comparison to other supraglottic airways.
- Latex free, sterile, single patient use device.

I-gel size	Patient size	Patient weight (kg)
1	Neonate	2–5
1.5	Infant	5–12
2	Small pediatric	10–25
2.5	Large pediatric	25–35
3	Small adult	30–60
4	Medium adult	50–90
5	Large adult	90 +

Insertion Technique

- Lubricate the posterior aspect of the distal non-inflatable cuff
- Extend the patient head and flex the neck gently (sniffing the morning air position) then introduce the I-gel in the oral cavity with the concavity facing downwards towards the chin.
- Glide the cuff along the palate until resistance is felt
- The horizontal line should coincide with the patients teeth, if it is not so then it is incompletely inserted. In such a case, removal the I-gel and reinsert it with jaw thrust. If this still does not solve the problem, then opt for a smaller size.
- Connect to the breathing apparatus.

Removal

- Once consciousness is regained and the protective reflexes are present, suction the oral cavity and pharynx.
- I-gel can be removed when the patient is awake and easily arousable with verbal commands. Alternately, in patients with hyper-reactive airway (i.e. smokers, asthmatics or patients with COPD) can be removed in deeper plane of anesthesia.

Describe the Elisha airway device (EAD).

The Elisha airway device is unique airway because of its ability to combine three functions: i.e. ventilation, intubation (blind and/or fiberoptic-aided) and gastric tube insertion. In comparison to the other supraglottic airways EAD allows for simultaneous ventilation and intubation of the patient. It has 3 channels – ventilation, intubation and gastric tube insertion (Fig. 51.17). The ventilation and the intubation ports are adjacent to each other whereas the gastric channel is on the posterior aspect of the device. The ventilation and intubation ports are separated at the proximal end of the device, however they communicate with each other at the distal end in to a laryngeal outlet. The ventilation channel has a 15 mm connector attached to the device. The intubation channel permits an 8.0 mm ID endotracheal tube for blind or fiberoptic-guided intubation.

The device consists of 2 high volume, low pressure cuffs with a single inflation port. The proximal cuff seals the nasopharynx and oropharynx, whereas the distal cuff seals the esophagus.

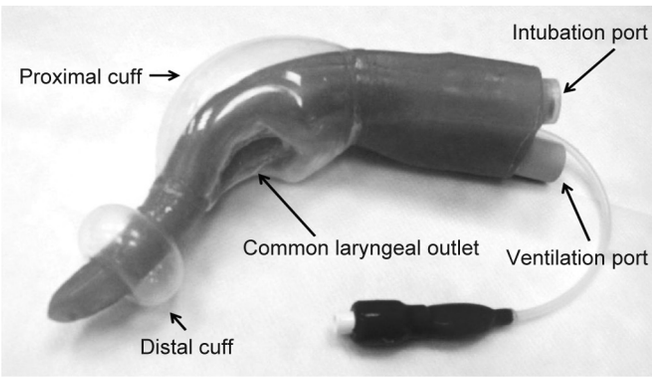


Fig. 51.17 Elisha airway device

Describe the Baska Mask®

The Baska mask® (Logikal Health Products Pvt Ltd., Morisset, NSW, Australia) is a novel supraglottic airway device similar to the LMA. It has a non-inflatable cuff, thus avoiding the risk of overinflation of cuff. The cuff is continuous with the airway channel and thus gets inflated with positive pressure ventilation, reducing chances of gastric insufflation and aspiration. The Baska Mask has an opening that sits in the upper esophagus, and the dorsal surface of the cuff is made in a way that it directs any oropharyngeal contents such as secretions towards the side channels to which suction can be attached. It comes with an elbow connector for suctioning. The tube also has an in-built bite-block, reducing the risk

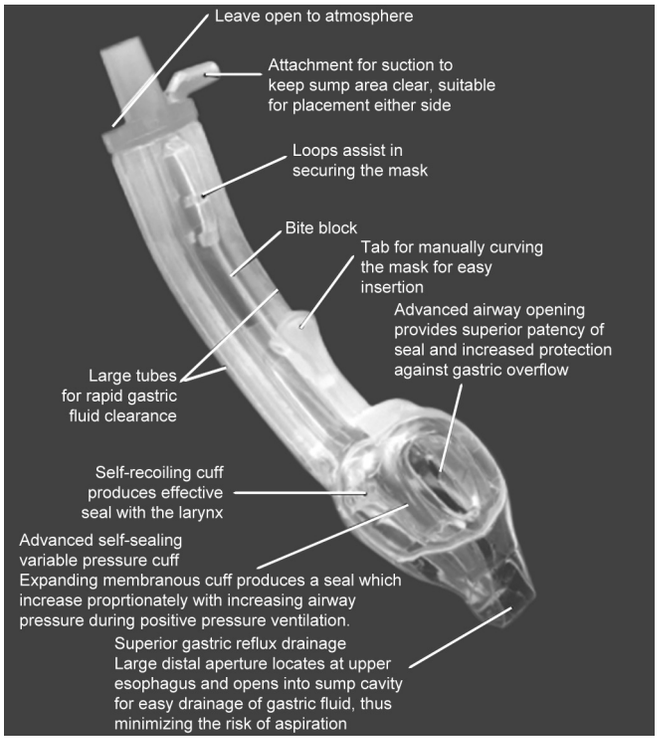


Fig. 51.18 The Baska Mask®

of patients' biting the airway. Anteriorly to the cuff, there is a hand-tab attached which allows manipulation during insertion; which is done in the neutral position. At the proximal end there are loops which help in fixation of the mask. This device was recently introduced and though it looks promising we need to see more studies on clinical utility. It comes in 4 sizes that have color coded 15 mm connecters.

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52 Regional Anesthesia Instruments

P Jain, A Kulkarni

What is the special advantage of using regional anesthesia?

Regional anesthesia techniques offer several advantages most of which relate to postoperative recovery.

- Minimal or no emergence time.
- Low or no incidence of postoperative nausea, vomiting, drowsiness and dizziness provided deep sedation and narcotic use is avoided.
- Minimal need for nursing care
- Eliminate the need for postoperative narcotics and its related side effects
- Facilitates rapid discharge from recovery and hospital

What is the Bromage scale of assessing the degree of motor block?

Bromage scale of assessing motor block is given in Table 52.1.

Table 52.1 Bromage scale

Scale	Criteria	Degree of block
0	Free movement of legs and feet able to raise extended leg	None
1	Inability to raise extended leg, knee flexion decreased, full flexion of feet and ankles	Partial (33%)
2	Inability to raise leg or flex knees; flexion of ankle and feet present	Partial (66%)
3	Inability to raise leg, flex knee or ankle, or move toes	Complete

What is the modified Bromage scale described by Breen?

Table 52.2 Modified bromage scale.

Score	Criteria
1	Complete block (unable to move feet or knees)
2	Almost complete block (able to move feet only)
3	Partial block (just able to move knees)
4	Detectable weakness of hip flexion while supine (full flexion of knees)
5	No detectable weakness of hip flexion while supine
6	Able to perform partial knee bend

What is the desired level of anesthesia required for various surgeries?

Types of spinal analgesia (Table 52.3):

1. Saddle block: Analgesia up to S1, usual puncture site between L4 and L5 or L5 and S1, cystoscopy, perineal or anal surgery can be done.
2. Low spinal: Analgesia up to L1 or T12, usual puncture site between L3 and L4, perineal or anal surgery, prostatectomy, etc.
3. Medium spinal: Analgesia upto T10–T8, usual puncture site between L2 and L3 or between L3 and L4. Lower abdominal surgery can be done.
4. High spinal: Analgesia up to T4, usual puncture site between L2 and L3, all abdominal surgeries can be done.
5. Unilateral: Patient should be kept in lateral position after injection of analgesic solution for some minutes to fix the drug. It helps to minimize the sympathetic block and hemodynamic.

Table 52.3 Level of anesthesia required for various surgeries.

Level	Surgical procedure
T4–5 (Nipple)	Upper abdominal surgery
T6–8 (Xiphoid)	Intestinal (appendicectomy, gynecologic, pelvic, ureter and renal pelvic surgery)
T10 (Umbilicus)	Transuretheral resection, obstetric, and hip surgery
L1 (Inguinal ligament)	Transuretheral resection, thigh, lower limb amputations
L2–3 (Knee and below)	Foot surgery
S2–5 (Perineal)	Perineal surgery, hemorrhoidectomy, anal dilation

What is the Stouts's principle of spread of local anesthetic in spinal anesthesia?

- Height of anesthesia varies directly with concentration of solution

- Extent of anesthesia is inversely proportional to rapidity of fixation
- Extent of anesthesia is directly proportional to the volume of drug used
- Extent of anesthesia is inversely proportional to spinal fluid pressure
- Extent of anesthesia is directly proportional to specific gravity for hyperbaric solution
- With isobaric or hypobaric solution extent depends on position of patient.

What is combined spinal-epidural anesthesia?

Combined spinal-epidural anesthesia is a technique in which the extradural space is located in the usual way as for extradural anesthesia. Then spinal anesthesia is performed either alongside or, more commonly by inserting a long spinal needle through the extradural needle to puncture the dura. The advantages of spinal anesthesia (speed of onset, density of block) are therefore combined with those of extradural anesthesia (flexibility, the ability to extend for intraoperative or postoperative analgesia). In addition the technique allows finer spinal needles to be used if a needle through needle method is employed, since the position of the needle tip can be estimated more easily as it passes through the extradural space into the dura. This technique has been widely used in obstetric analgesia and anesthesia and other types of surgery.

Techniques of needle insertion are:

- *Single pass:* Described by Soresi, where an epidural injection was performed and then the same needle was advanced into the subarachnoid space.
- *Needle through needle:* The epidural space is identified as if performing an epidural block. A spinal needle is then passed through the epidural needle and beyond its tip, until the dura is punctured. Subarachnoid drug is administered and, after removing the spinal needle an epidural catheter is placed. This technique requires that subarachnoid blockade is initiated before epidural catheter placement. Potential problems include failure of the spinal component, inadvertent insertion of the catheter into the subarachnoid space and damage to either of the needles by friction between them.
- *Separate needles:* Separate interspaces require the placement of the epidural catheter first followed by the subarachnoid block.
- *Combined needle:* It is made by joining a spinal needle along the length of a Tuohy needle, including bending around the tip. They were designed to avoid friction between spinal and epidural needle and to ensure that dural puncture was separated from epidural catheter placement.

Describe the various types of spinal/epidural needles?

Types of needles used for spinal and epidural blockade are described below:

The *standard spinal needle* consists of three parts: a hub that is fused to a cannula with a point, and a fitted removable stylet that occludes the distal lumen and point of the cannula.

Spinal needles are carefully manufactured without surface irregularities and with a tight fitting removable stylet that completely occludes the needle lumen. A wide variety of sizes with different applications are available from 16 gauge to 30 gauge.

The points of the cannulae usually are made of stainless steel and must meet standards of stiffness, flexibility, and resistance to breakage. Luminal sizes of the spinal needle cannulae vary from 3.5–4 inches in length; smaller cannulae

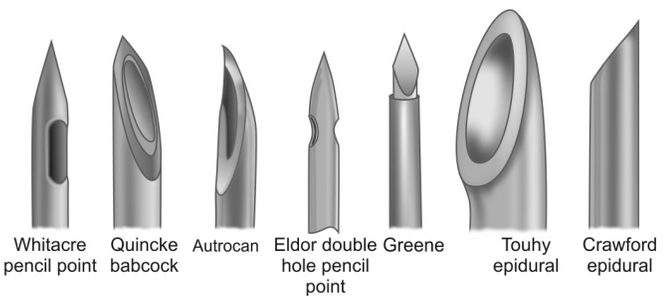

Fig. 52.1 Spinal and epidural needles

of 28 gauges and 30 gauges are available. However, the "feel" for tissue during puncture is lost with these, and they are used, if at all, in neonates and infants.

The needles are either sharp or blunt at the tip with either end-injection or side injection and either sharp or round (blunt) bevel edges. The *Quinke-Babcock* needle is considered the standard spinal needle. It has a small hub, medium bevel length with sharp edges, a sharp point, and end injection. There is a fitted stylet with a matching beveled tip to the cannula point. The hub is designed with a Luer lock connector.

The *Pitkin* needle has a short, and sharp bevel with a pointed end injection. The stylet also has a short bevel which fits the point of the cannula.

Two common alternatives are the Greene and Whitacre needles. The *Greene* needle has a small hub and a Luer lock connector. There are rounded, non-cutting edges to the bevel, and the bevel is of medium length. There is also a stylet with a rounded bevel to fill the opening point of the cannula. This needle functions as a type of pencil point and,

because of the non-cutting edges of the bevel, it separates rather than cut the dural fibers. Available sizes vary from 20–26 G and length ranges from 3–3.5 inches.

The *Whitacre* and other *Pencil-point* needles have a rounded bevel, no cutting edges, and the orifice of the needle is on one side of the cannula about 2.5 mm proximal to the tip of the cannula; a fitted stylet occludes the exit port on the side. The point cleanly separates all the fibers and the dural puncture site is small so that the incidence of PDPH is low because of less CSF leak. The factors affecting PDPH are:

- *Size of the needle:* A 25 gauge Quincke needle—less leakage than with 22 gauge needle
- *Type of needle design:* Whitacre pencil point or Greene conical produce less leakage than needles that have a bevel with a cutting edge
- *Orientation of the bevel:* A 22 gauge needle parallel to longitudinal dural fibers produces less leakage than when it is perpendicular to the dural axis
- *Angle of approach to dural puncture:* Less leakage at an angle of 30° than at an angle of 60° or 90°.

Epidural Needles

- The standard epidural needle is typically 16–18 gauges, 3 inches long, and has a blunt bevel with a gentle curve of 15–30° at the tip. This blunt bevel and curve allow the needle to pass through the ligamentum flavum and abut against dura, pushing it away rather than penetrating it. This creates the negative pressure that identifies the epidural space. The most common version of this needle is referred to as the Tuohy needle and the curved tip is the Huber tip.
- *Crawford point needle:* It is short beveled at 40 to 45° with smooth edges. The distal end is open but a well-fitted stylet closes the orifice for insertion. It is made of stainless steel with a 2% molybdenum content which improves resistance to corrosion in these reusable needles.
- *Hustead needle:* It is a modified Tuohy needle with a rounded tip and a bevel opening which is located 2.7 mm from the tip.

Describe the features of spinal/epidural catheters.

Teflon plastic tubing is the most widely used material. It is available in a radiopaque form and fitted with a fine wire stylet. Sizes vary from tubing with and outside diameter of 0.99 mm permitting passage through an 18 gauge, thin wall, directional Huber point needle to tubing with an outside diameter of 0.25 mm, which can pass through a 20 or 21 gauge needle. Some have single terminal opening, while others may have several lateral holes with or without a terminal opening. Preferred are the disposable, radiopaque Teflon catheters, 20 gauge by 90–100 cm, fitted with a fine wire stylet and a syringe adaptor marked at 5,10,15 and 20 cm from the distal end. The catheter will pass through an 18 guage thin wall needle.

Discuss factors affecting epidural and spinal drug spread

This can be discussed under the following heads:

1. *Patient factors:* The age, sex, weight and height will determine the spread of the drug. High intra abdominal pressure and change in the anatomy of the epidural space (pregnancy), anatomical deformities of spine, volume of CSF will also affect the drug spread.
2. *Technical factors:* Position of the patient, site of injection (level), type of needle used, placement of intrathecal catheters will also affect the spread of the drug. Barbotage previously thought to affect the spread has been shown not to affect the spread of the drug. Rapid injection may cause the drug to spread more when hypobaric solutions are used.
3. *Properties of the injected solution:* Baricity of the injectate: Solutions are made hyperbaric by addition of sugar. Higher volume will spread more. Larger dose or higher concentration will affect the height of block.
   Higher viscosity will increase the spread of the drug.

What are special precautions taken in patients who are on anticoagulation therapy?

With the twice-daily US dosing regimen of low molecular weight heparin, the first dose is usually given 12–24 h after surgery, and removal of the epidural catheter is recommended before initiation of thromboprophylaxis. If the epidural catheter is left in place during thromboprophylaxis with the twice-daily low molecular weight heparin dose, a 24 hour delay between the last heparin dose and removal of the epidural catheter is recommended. Combination with other drugs influencing the coagulation system including prophylactic heparin may be dangerous. Options for instituting central nerve block before elective surgery in patients receiving warfarin are:

- Stop drug at least 5 days prior to procedure
- Place patient on heparin infusion if continued anticoagulation required
- Check INR on day of procedure and ensure < 1.5 INR units
  If INR >1.5 (a) Delay surgery
  (b) Consider alternative anesthetic/analgesic technique if surgery urgent
  (c) Consider reversal with factor IX concentrate in an emergency.

Table 52.4 ASRA recommendations for use of neuraxial anesthesia with postoperative initiation of LMWH thromboprophylaxis

Regimen	Timing of initial dose(s)	Use of catheters
Twice-daily dosing (i.e. with enoxaparin)	Administer first dose no earlier than 24 h postoperatively, regardless of anesthetic technique, and only in presence of adequate hemostasis	Remove indwelling catheters before starting LMWH therapy With a continuous technique, epidural catheters may be left indwelling overnight and removed the following day; administer first LMWH dose 2 h after catheter removal
Single-daily dosing (i.e. with dalteparin)	Administer first dose 6–8 h postoperatively. Administer second dose no sooner than 24 h after first dose	Indwelling neuraxial catheters may be safely maintained but should be removed a minimum of 10–12 h after last LMWH dose; start subsequent LMWH dosing a minimum of 2 h after catheter removal

Table 52.5 ASRA recommendations for placement and removal of epidural catheter in patient anticoagulants and anti platelet drugs.

	Minimum delay between last dose and placement or removal of epidural catheter	Minimum delay after placement or removal of epidural catheter and subsequent dosing
Heparin		
SC Unfractionated heparin (mini-dose)	No contraindication The risk of neuraxial bleeding may be reduced by delay of the heparin injection until after the block, and may be increased in debilitated patients after prolonged therapy. Patients receiving heparin for greater than four days should have a platelet count assessed prior to neuraxial block and catheter removal.	
IV Unfractionated heparin	4 h (2–4 h)	1 h (0.5–1 h)
Full anticoagulation of cardiac surgery	Currently, insufficient data and experience. Postoperative monitoring of neurologic function and selection of neuraxial solutions that minimize sensory and motor block is recommended to facilitate detection of new/ progressive neurodeficits	
Low molecular weight heparin	12 h (10–12 h)*	4 h (2–12 h)
Traumatic needle or catheter placement may signify an increased risk of spinal hematoma, and it is recommended that this consideration be discussed with the surgeon. It does not necessitate postponement of surgery. However, initiation of LMWH therapy in this setting should be delayed for 24 hours postoperatively		
Patients receiving higher (treatment) doses of LMWH, will require delays of at least 24 hours to assure normal hemostasis at the time of needle insertion. Neuraxial techniques should be avoided in patients administered a dose of LMWH two hours preoperatively		
ADP receptor antagonists		
Clopidrogel	> 7 days (7 to > 7 days)	Immediately
Ticlopidin	> 10 days (10–14 days)	Immediately
COX inhibitors		
Non-selective, NSAIDs	0 day (0–2 days)**	Immediately
COX-2-selective (Rofecoxib,Celecoxib)	0 day (0 day)	Immediately
Aspirin (60–325 mg=day)	0 day (0–2 days)**	Immediately
GPIIb=IIIa antagonists		
Abciximab	2 days (24–48 h)	4 h (2–4 h)
Tirofiban	1 day (4–8 h)	4 h (2–4 h)
Eptifibatide	1 day (4–8 h)	4 h (2–4 h)
Fondaparinux	No epidural catheter recommended (not recommended to 24 h)	No epidural catheter recommended (not recommended to 6 h)
Melagatran, ximelagatran	12 h (8–10 h)	4 h (2–4 h)
Thrombin inhibitors	Due to the lack of information available, no statement regarding risk assessment and patient management can be made	

If epidural is to be removed, INR < 1.5, PTT must be corrected with FFP or Vitamin K.

Similar considerations apply to spinal or epidural catheter removal. Mechanical methods of DVT prophylaxis (e.g. intermittent calf compression boots or foot pumps) are effective alternatives in any situation where there is a wish to avoid pharmacological methods completely.

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53 Nerve Locator and Peripheral Nerve Stimulator

N Amin

What is the difference between a nerve locator and peripheral nerve stimulator?

Peripheral nerve stimulator (PNS) is used to assess the degree of neuromuscular block. Needle or surface electrodes are used to stimulate a suitable peripheral nerve and the evoked response of the muscles innervated by the nerve is either recorded or palpated. Electrical nerve locators are used as an aid to peripheral nerve blockade. Specialized insulated needles are used to elicit a response from a motor nerve. Both are battery operated equipments. However an electrical nerve locator can deliver currents in the range of 0.1–5 mA while a peripheral nerve locator can deliver currents in the range of 10–100 mA. When using needle electrodes lesser current is needed to stimulate a nerve due to close proximity to the nerve. While using surface electrodes higher currents are needed as skin resistance has to be overcome. Also in a peripheral nerve stimulator different patterns of stimulation (TOF, DBS, PTC) are available.

What are the advantages of a nerve locator?

A nerve locator is helpful because:

- It has higher success rate
- Easier to locate nerve—not totally dependent on anatomy
- Better localization of nerve trunks in difficult situations
- Less chances of nerve damage
- No discomfort or pain to the patient
- Patient cooperation is not required
- Can be used on anesthetized non-paralysed patients
- Toxic dose of LA avoided by giving titrated volume
- Valuable tool for training purpose
- Blocks can be performed in pain clinics and rehabilitation centers.

What are the limitations of a nerve locator?

A nerve locator has 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. Its use has been described for pure sensory nerves but it is not very common in clinical practice
- 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.

What do you understand by chronaxy and rheobase?

The amount of electrical energy required to propagate a nerve impulse is a product of the stimulus strength (mA) and duration of current (ms). Thus stimulation of a nerve depends on the relationship between the strength and duration of current flow. This relationship can be explained by the terms rheobase and chronaxy. Rheobase is the minimum current strength required, to generate an action potential. For any nerve type there is a minimum current strength below which, an impulse will not be generated; no matter for how long the current is applied. The chronaxy is the length of time the current has to be applied to the nerve to generate an action potential when the current strength is twice the rheobase.

Why is stimulation of a peripheral nerve not painful for the patient?

As chronaxy varies in different nerve fibers it is useful for comparing different nerves. Myelinated fibers have a shorter chronaxy than unmyelinated nerve fibers and require less

electrical energy for stimulation. Since the large motor fibers (Aα) have shorter chronaxy (0.05–0.1 ms) they can be stimulated with pulses of current with a shorter width. Thus when stimulating a motor nerve, the nerve fibers responsible for pain do not get stimulated as they have a longer chronaxy (Aδ fibers have a chronaxy of 0.150 ms and C fibers have a chronaxy of 0.4 ms). As a result of this the patient does not feel any pain during peripheral nerve blocks except when the needle is piercing the skin or is touching the nerve.
In awake, cooperative patients sensory nerves can be located by using longer width pulses of current.

What is the importance of threshold current and what should be the threshold current for stimulating a nerve?

If the needle tip is further away from the nerve, then more current strength is required at the tip of needle to stimulate the nerve. According to Coulombs law $E = K (Q/r^2)$, where E is the threshold current required at the nerve, K is a constant, Q is the minimal current from the needle tip and r is the distance from the nerve. Therefore, current required to stimulate a nerve is inversely proportional to the square of the distance. This principle is used to calculate the distance from the needle to the nerve using a constant current stimulus. For example, if the nerve is getting stimulated with the current threshold of 1mA and the distance of nerve from tip of needle is 1 cm, then if the distance increases to 2 cm the current required will be 4 mA and for a distance of 0.5 cm the current will be 0.25 mA. If needle is too far away then stimulation of the nerve with higher current strengths can be very painful. Also if the nerve can be stimulated with a lower threshold current then it indicates that the needle tip is very close to the nerve. If muscle twitch is elicited with a current of 0.3-0.4 mA, it indicates that the needle tip is very close to the nerve and injection of local anesthetic at this point will result in successful block. However, if muscle twitch is elicited with a current strength of less than 0.2 mA, there is very strong possibility that the needle has penetrated the epineurium. Injection of local anesthetic at this point increases the risk of intraneural injection which may cause temporary or permanent neural damage.

Will the polarity of the stimulating electrode make any difference during the use of a nerve locator?

If the stimulating needle is the cathode (negatively charged), then the flow of current alters the resting membrane potential of the surrounding cells. This produces an area of depolarization, making it easier to stimulate the nerve. Thus significantly less current is required to stimulate the nerve if the stimulating electrode is a cathode. If the stimulating electrode is the anode (positively charged), then the flow of current causes an area of hyper polarization adjacent to the tip of the needle and an area of depolarization just distal to the tip. This arrangement is not very efficient and very high current strength is required for stimulating the nerve. In most modern PNS the needle is negative by default and it cannot be changed by the operator.

What is the significance of the stimulus frequency during the use of a nerve locator?

The muscle response produced by a stimulating needle indicates that the needle is approaching the target nerve. If the needle is being advanced very fast and the stimulus frequency is very low then the needle may either miss the nerve or it may go through the nerve resulting in damage to the nerve. However, if the stimulus frequency is very high it can be very painful for the patient. In clinical practice a frequency of 2 Hz is adequate and does not cause patient discomfort. If you are getting a motor response at a current strength of less than 1 mA, it indicates that the needle is very close to the nerve and then it should be advanced slowly at a speed of 1mm/sec so that you do not miss the nerve.

Can you describe the parts of an electrical nerve locator? What is the importance of each part?

Electrical nerve locator has various parts that help to improve the efficiency and safety of the nerve locator.

Current generator produces the electric current required to stimulate the nerve.

Constant current output: The set current is delivered between the tip of the needle and the remote electrode. Modern electrical nerve locators compensate for the changes in resistance and ensure that a constant current is delivered. The resistance in the circuit will vary with position of the electrode from the tip of the needle and the tissue impedance.

Current meter: The intensity of current with which the nerve is stimulated gives an idea about the approximate distance from the tip of the needle to the nerve. Hence it is important to have an accurate idea of the intensity of current, which is used to stimulate the nerve. The current meter gives a digital display of the current delivered in the circuit. The maximum and minimum current delivered by most electrical nerve locators is 5 mA and 0.1 mA respectively. For patients with neuropathy and sensory nerve stimulation, higher current output is needed.

Current output control: The operator or assistant can precisely control the amount of current passing through the circuit with the help of the current output control. In some modern

nerve locators the control knob can be easily sterilized by autoclaving. Foot operated current intensity controllers can be used whenever there is no assistance available.

Frequency control: In most electrical nerve locators the impulses are delivered at a frequency of 1 or 2 Hz. Needle manipulation is better with higher frequencies as there is a frequent feedback to the operator as the needle is being advanced. The frequency may have to be reduced if the patient experiences pain during stimulation.

Connection/disconnection indicator: This is an important safety feature. Machines may either have flashing lights or pulsating beeps, which indicate that the connections are intact. Loss of these signals indicates that the connections are lost and the current will not be delivered. The most common cause of circuit disconnection is incomplete connection and/or poor electrode contact. Current may not be delivered if there is loss of circuitry, unit malfunction or battery failure.

Can a peripheral nerve stimulator used for neuromuscular monitoring be used for peripheral nerve blocks?

Peripheral nerve stimulators used in assessing recovery of muscle relaxation should not be used in locating nerves for blockade because of the possibility of high intensity currents (sometimes >100 mA), causing neural damage at close approximation to the nerve.

What is the significance of the pulse width?

As mentioned earlier motor fibers have a shorter chronaxy while the pain fibers have a longer chronaxy. A pulse width of 0.05– 0.1 ms will preferentially stimulate the motor fibers and not the pain fibers. A longer pulse width of > 0.15 ms can be used for locating pure sensory nerves like lateral femoral cutaneous nerve. Patients will complain of paresthesias along the distribution of the nerve.

What are the important features of a stimulating needle?

For precise nerve location, specially designed thin insulated needles have to be used with the nerve locator. Due to current dispersion, larger current is required to stimulate a nerve with a non-insulated needle. Specialized needles are completely insulated with the exception of the bevel or the tip as a result of which lower currents are required to stimulate the nerve. Use of these needles increases the accuracy, but it may be more difficult to locate the nerve as the current density is focused at the tip. Accidental damage to the nerves can be further minimized by the use of needles with non-cutting tip. Needles are available in varying lengths (commonly 50, 100 and 150 mm) and the needle selection would depend on the depth of tissue plane required for a particular block. If the nerve is superficial then a thin and short needle can be used and if the nerve is deep then a long needle with a larger diameter will be needed. It is easy to manipulate long needles with a larger diameter, but it also increases the chances of tissue damage. To prevent current leakage, the needle has an insulated cable, which is connected to the cathode of the nerve locator. The needle also has an extension tubing which minimizes the chances of needle movement while injecting the local anesthetic. The extension tubing should be flushed to remove any air as it can cause patchy block.

What are the local anesthetic doses for peripheral nerve block?

Large volumes of local anesthetic would be needed to perform a peripheral nerve block (PNB). Accidental intravascular injections of this large volume may cause CNS and cardiac complications. Therefore, the appropriate drug/dose selection, monitoring and availability of resuscitative drugs and equipment are essential while performing PNB. When a combination of local anesthetic is used the toxicity would be additive. The concentration and volume of local anesthetic should be reduced in elderly (patients > 65 years) and in ASA 3 patients with significant underlying cardiac/ hepatic/renal disease.

Therapeutic dose limits of commonly used local anesthetic for PNB are:

Bupivacaine, L-bupivacaine, ropivacaine- 3–4 mg/kg

Lidocaine with epinephrine- 5–7 mg/kg

Lidocaine without epinephrine- 3–5 mg/kg.

What is the appropriate evoked motor response for each peripheral nerve block technique?

The appropriate evoked motor response (EMR) for each peripheral nerve block technique is given below:

EMR for various blocks

Peripheral nerve block technique	Optimal EMR
Interscalene	Flexor—deltoid, biceps, pectoralis major
	Extensor—triceps, brachioradialis, wrist extensors
Deep cervical plexus	Rhomboids, shoulder girdle
Infraclavicular	Muscles of wrist and hand
	Radial—extension of wrist and fingers
	Median—flexion of wrist and fingers
	Ulnar—adduction of thumb/4th and 5th finger flexion
Femoral	Quadriceps—patellar snap
Sciatic	Inversion, plantar flexion

How can you improve the success rate of a block?

To minimize the latency (onset of action) and improve the success of the block:

- The local anesthetic must be deposited as close to the nerve as possible. Appropriate end points with a peripheral nerve stimulator can increase the success rate of the block. While performing an axillary block highest success rate is achieved with an evoked motor response of the radial nerve and with sciatic nerve block evoked motor response of inversion is associated with shortest latency and highest success rate.
- Success rate is highest when the desired evoked motor response is obtained with a stimulating current which is between 0.2–0.4 mA. If evoked motor response is obtained at currents higher than 0.5 mA it indicates that the needle tip is far from the nerve and it may result in failed block. If a stimulating current of less than 0.2 mA elicits an evoked motor response then there is an increased risk of nerve damage due to the possibility of intraneuronal injection.
- Needle should be immobile during the injection
- Judicious selection of local anesthetic drug and concentration.

Local anesthetics for blocks.

LA with epinephrine (1:300,000)	Latency (mins) surgical anesthesia	Duration (h) surgical anesthesia	Duration (h) postoperative analgesia
Lidocaine 1.5–2%	10–20	2–3.5	3–5
Bupivacaine 0.5–0.625%	15–30	5–6	12–24
L-bupivacaine 0.5–0.625%	15–30	5–6	12–24
Ropivacaine 0.5%	10–20	3–4	10–15

Latency is longer with axillary and sciatic nerve blocks than with interscalene and femoral nerve blocks.

Can you describe the technique of peripheral nerve block?

- Explain the procedure to the patient. Take a written informed consent.
- Check for history of bleeding disorder.
- Before performing a peripheral nerve block, take an IV access and ensure that the resuscitation drugs and airway equipments are readily available. The patient should be monitored during the block.
- Test the function of the nerve stimulator. Failure to check for circuit completion increases potential for neural damage (especially when performing nerve blocks in unconscious patients).
- Apply the electrode, ensuring good contact.
- Disinfect the skin, create a skin weal and, if necessary, infiltrate the puncture channel.
- Connect the earth lead to the surface electrode. Connect the syringe containing local anesthetic to the injection line and flush it.
- Switch on the stimulator and select the pulse duration (0.1 ms for mixed nerves), pulse frequency (2 Hz) and stimulation current (1 mA).
- After inserting the needle into the skin and subcutaneous tissue manipulate the needle to get the desired evoked motor response. Ask the assistant to reduce the stimulating current in small increments till the contractions are just barely visible at a threshold level of 0.2–0.3 mA. Evoked motor response at a current < 0.2 mA indicates that the needle may be intraneural.
- If there are visible contractions at a threshold current < 0.4 mA but not < 0.2 mA, then inject 1 mL of local anesthetic after negative aspiration test. At this point the twitching should disappear. If the patient experiences pain on injection or if the twitches do not disappear then the needle should be withdrawn slightly as the needle may be intraneural.
- After a negative aspiration test the rest of the local anesthetic is injected slowly and the needle is removed
- Separate kits are available for continuous infusion techniques (e.g. Contiplex).

Are the peripheral nerve stimulator settings the same for any block?

No, the peripheral nerve stimulator settings change depending on the patient and the type of nerve to be stimulated.

Mixed nerve (most PNB) blocks: Current -1 mA, current duration - 0.1 ms, frequency - 1–2 Hz.

Sensory nerve (e.g. saphenous nerve) block: Current—2–5 mA, Current duration—1 ms, Frequency–1 Hz.

Diabetic neuropathy: Current- 2 mA, current duration- 0.3 ms, frequency—1–2 Hz.

How does one avoid an intraneural injection?

While it is important to place local anesthetic accurately, it is vital to minimize the chance of causing nerve damage. The following list of seven points may help to avoid intraneural injection.

- Equipment checks—check the integrity of the circuit and whether the device is delivering the current. Complete the circuit by connecting the negative and positive electrode and switch on the nerve stimulator. There will be an audible beep if the current is being delivered. If there is no audible beep it indicates that current is not being delivered and the device should not be used.

- Appropriate anatomical knowledge
- Threshold—no muscle twitch at or below 0.2 mA
- Twitch should disappear immediately after injection starts
- Minimal resistance to injection
- Patient should be awake to see if there is pain on injection
- Avoid multiple attempts at nerve location.

Why does the twitch disappear immediately on injection of local anesthetic?

Immediate disappearance of the twitch on injection local anesthetic is due to the mechanical displacement of the nerve away from the needle tip.

What are the contraindications to peripheral nerve blocks?

Absolute contraindication- Patient refusal, infection at the site of block, allergy to local anesthetics, severe coagulopathy

Relative contraindication—Mild Coagulopathy, distorted local anatomy (can consider blocks under ultrasound guidance along with a nerve stimulator).

What are the motor supply areas of the upper and lower limbs?

Motor Supply Areas of Upper Limb

Peripheral nerve	Muscle	Function
Axillary nerve	Deltoid muscle	Abduction of the arm in the shoulder joint
Musculocutaneous nerve	Biceps brachii muscle Coraco-brachial muscle	Bends the elbow in supination
Median nerve	Flexor carpi radialis muscle	Flexes and abducts wrist radially
	Flexor pollicis brevis muscle	Pronates the forearm (flexes proximal phalanx of thumb)
	Flexor digitorum profundus	Flexes and adducts the thumb, flexes muscle (I–III) fingers I–III (Flexes distal interphalangeal joint of fingers)
Radial nerve	Triceps brachii muscle	Extends elbow
	Extensor carpi radialis (brevis) muscle	Extends and abducts wrist radially
	Extensor digitorum muscle	Extends and flexes the hand dorsally Extends and spreads the fingers
Ulnar nerve	Flexor carpi ulnaris muscle	Flexes and abducts wrist in an ulnar direction
	Flexor digitorum profundus	Flexes fingers (IV–V) muscle (IV–V)

Motor Supply Areas of Lower Limbs

Peripheral nerve	Muscle	Function
Femoral nerve	Quadriceps femoris muscle	Flexes the hip, extends the knee
Obturator nerve	Adductors of thigh at the hip joint	Adducts thigh
Tibial nerve	Biceps femoris muscle	Flexes knee and rotates leg laterally
	Semimembranosus muscle	Flexes knee
	Semitendinosus muscle	Extends thigh, flexes leg and rotates it medially
	Flexor hallucis longus muscle	Flexes foot
	Flexor digitorum longus muscle	Flexes toes
Common peroneal nerve	Tibialis anterior muscle	Dorsiflexion and inversion of foot
	Extensor digitorum muscles, Extensor hallucis muscles, Peroneal muscles	Extend, evert and pronate the outer foot

How can you further improve the safety and success of peripheral nerve blocks?

Performing the blocks under ultrasound guidance can further improve the safety and success rate of peripheral nerve blocks.

Describe various peripheral nerve blocks.

Interscalene block

Indications	Local anesthetic drug and doses	Duration of analgesia	Potential problems	contraindications
Shoulder arthroscopy, reconstructive shoulder surgery, Upper arm surgery	Bupivacaine 0.5% with epinephrine 1:200000 Volume 35–40 mL	12–18 hours	Common: Phrenic nerve block (inevitable),Horner syndrome, recurrent laryngeal nerve block (hoarseness) Rare: Intravascular injection (vertebral artery) pneumothorax, inadvertent spinal and epidural block	Patients with significant pre-existing pulmonary disease

Axillary block

Indications	Local anesthetic drug and doses	Duration of analgesia	Potential problems
Elbow, forearm and hand surgery	Bupivacaine 0.5% with epinephrine 1 : 200000, Volume 40–50 mL	12–24 hours	Hematoma, intravascular injection, peripheral nerve injury

Femoral nerve block (3 in 1 block)

Indications	Local anesthetic drug and doses	Duration of postoperative analgesia	Potential problems
Reconstructive knee procedures	Bupivacaine 0.5% with epinephrine 1:200,000. volume 20–30 mL	12–18 hours	Hematoma, intravascular injection, quadriceps femoris weakness. Patient should be prevented from bearing weight on the blocked extremity and instructed to crutch walking

Sciatic nerve block/ Popliteal nerve block

Indication	Local anesthetic drug and doses	Duration of postoperative analgesia	Potential problems
Surgical procedures below knee	Bupivacaine 0.5% with epinephrine 1:200,000 volume 25–30 mL	Bupivacaine 12–24 hours	Incomplete block, inability to bear weight. Patient should be instructed to crutch walking

Ankle block

Indications	Local anesthetic drug and doses	Duration of postoperative analgesia	Potential problems
Surgery on foot.	Bupivacaine 0.5%, volume 30–40 mL No epinephrine	Bupivacaine 12–24 hours	None

Peripheral Nerve Stimulator

Why do we need neuromuscular monitoring?

Traditionally clinical criteria were used to assess recovery from neuromuscular blockade. Numerous studies have shown that even if there is clinical recovery of neuromuscular function there is significant residual curarization in the post operative recovery room. The residual curarization can lead to increased postoperative complications. The clinical signs of recovery from neuromuscular blockade can be influenced by various other factors other than the degree of neuromuscular blockade. Response of the muscle to nerve stimulation would give precise information about the status of the neuromuscular function.

During neuromuscular monitoring a peripheral nerve stimulator is used to evaluate the muscular response to supramaximal stimulation of a peripheral motor nerve.

What are the various ways in which a nerve can be stimulated? Which method of stimulation is commonly used and why?

A nerve can be stimulated by an electrical or magnetic stimulus. Electrical nerve stimulation is commonly used in clinical practice. Unlike electrical nerve stimulation, magnetic nerve stimulation is less painful and does not require physical contact with the body. However, it is not used in clinical anesthesia because the equipment is bulky and heavy, and also it cannot be used to achieve supramaximal stimulus or train of four stimulation.

What are the principles of peripheral nerve stimulation?

Peripheral nerve stimulator is used for neuromuscular monitoring. A suitable peripheral nerve is stimulated by applying electric current via surface or needle electrodes. The evoked muscle response is either palpated or recorded. The current is usually applied transcutaneously, by using ECG electrodes.

A nerve will contain many motor nerve fibers. All of these fibers will need to be stimulated in order to produce a maximal muscle contraction. An electric current of sufficient magnitude would have to be applied for sufficient duration in order to generate an action potential in all of the nerve fibers in a motor nerve. Hence the nerve has to be stimulated with supramaximal stimulus. Maximal stimulus is the current which generates a response in all nerve fibers and results in maximal muscle contraction. Supramaximal stimulus is a current which is 25% above the maximal stimulus. However, a submaximal current should be used during recovery as supramaximal stimulus can be painful in an awake patient. After administering a neuromuscular blocking agent the response of the muscle will decrease in parallel to the number of fibers blocked. Therefore, the reduction in response during constant stimulation reflects the degree of neuromuscular blockade.

The shape of each stimulus should be monophasic and rectangular (also known as square wave stimulus). A biphasic stimulus will produce 2 responses to a single stimulus due to submaximal stimulation of the nerve by the second part of the biphasic stimulus pulse. The recorded response is a summated response not suitable for scientific analysis.

The duration of the square wave stimulus should be 0.2–0.3 ms so that it falls within the absolute refractory period of the motor units in the nerve. Pulse duration less than 0.1ms will require very high intensity of stimulation because the stimulus duration falls below the rheobase of the motor units. A pulse exceeding 0.5 ms may stimulate the muscle directly or cause repetitive firing.

What are the properties of an ideal nerve stimulator?

It should be able to generate square wave monophasic electrical stimuli of 0.2–0.3 ms duration of sufficient intensity in the required pattern. The nerve stimulator should be capable of stimulating the nerve at a constant current as compared to a constant voltage, as current is the determinant of nerve stimulation. It should be able to generate a current of at least 60–70 mA, which is a commonly used range. There should be a digital display of the delivered current. It should be battery operated and small in size. There should be audible signal on delivery of the stimulus. It should have an audible alarm for poor electrode contact. The polarity of the electrode leads should be indicated. Also it should be capable of delivering the following modes of stimulation: single twitch, Train of Four (TOF), Double Burst Stimulation(DBS), Posttetanic Count(PTC). It should have means of recording the evoked response.

What is the current required for supramaximal stimulation?

The current for supramaximal stimulation is 5–8 mA for needle electrodes and for skin electrodes it is 20–60 mA depending on the skin resistance (0–2.5 kΩ). A higher current is needed for surface electrodes because the skin resistance has to be overcome to ensure constant stimulating current.

What are the factors which will influence the delivery of constant current?

Any factor which affects the skin resistance will influence the delivery of constant current. The skin resistance varies with temperature, adequacy of electrode placement, disease states.

Diseased states like diabetics and CRF, and patients with thick and dry skin or obese patients require higher current intensity. Also cooling the skin would increase the skin resistance. This reduces the current intensity leading to a decrease in response to stimulation. It is recommended that the negative electrode should be the stimulating electrode. It should be placed directly over the most superficial part of the nerve. The positive electrode should be placed proximally along the course of the nerve, to avoid direct muscle stimulation. ECG electrodes of the silver/silver chloride variety ensure good electrical contact with the skin. The actual conducting area should be small, approx 7–8 mm in diameter. Otherwise the current produced in the underlying nerve may not be adequate. The skin should always be cleansed adequately (preferably with alcohol swabs) before applying the electrodes. This ensures that there is minimal resistance to the delivery of the current. If supramaximal stimulus cannot be obtained with surface electrodes then needle electrodes can be used.

What are the conditions where neuromuscular monitoring is essential?

- When either prolonged infusions of neuromuscular blocking drugs, or long-acting neuromuscular blockers are used as in ICU
- Prolonged duration of anesthesia using muscle relaxants
- In certain conditions like respiratory disease or morbid obesity where inadequate reversal may have devastating effects
- In conditions where administration of a reversal agent may cause harm, for example, tachyarrhythmias, cardiac failure and reversal agent are better avoided
- When pharmacokinetics of muscular relaxants may be altered like in liver or renal dysfunction
- If muscle relaxants are being used in patients with neuromuscular disorders like myasthenia gravis, Eaton–Lambert syndrome
- Surgeries requiring profound neuromuscular blockade e.g. neurosurgery, vascular surgery in vital areas like thoracic cavity.

What are the various methods available for quantifying the degree of neuromuscular blockade?

Visual and tactile assessment of the evoked muscle response cannot be relied upon to assess the degree of neuromuscular blockade. Objective methods of neuromuscular monitoring in which the evoked muscle response is measured and recorded is much more sensitive in excluding clinically significant residual neuromuscular blockade. Five methods are available namely

- Mechanomyography: Measurement of evoked mechanical response of muscle
- Electromyography: Measurement of evoked electrical response of muscle.
- Acceleromyography: Measurement of acceleration of muscle response
- Piezoelectric neuromuscular monitors: Measurement of evoked electrical response in a piezoelectric film sensor attached to the muscle
- Phonomyography.

What is the principle of mechanomyography (MMG)?

Mechanomyography: This measures evoked muscle tension. The adductor pollicis muscle in the thumb is most commonly used. A strain gauge is attached to the thumb or any other part of the body. The movement of the thumb is limited by a fixed amount of tension (preload). When the ulnar nerve is stimulated at the wrist, the adductor pollicis contracts and causes the thumb to move. However, as the thumb is stabilized the strain gauge transducer detects a change in tension in the adductor pollicis muscle. This change in tension is measured and converted to an electrical signal, which is then displayed and recorded. The arm and hand must be fixed and movement of the thumb must be along the length of the transducer to ensure that the readings are accurate. This technique is the gold standard as it can be used for assessment of any pattern of nerve stimulation. However as it is very cumbersome, it is not routinely used in clinical practice.

What is the principle of electromyography (EMG)?

Electromyography: It records the compound action potential that occurs during muscular contraction. Adductor pollicis and ulnar nerve are the most commonly used sites. Five electrodes are used to measure the electrical changes that occur in muscle during stimulation. The 2 stimulating electrodes are placed over the ulnar nerve. Of the remaining 3 electrodes one should be placed over the muscle belly and the reference electrode should be placed over the tendon insertion site. The third electrode is the ground electrode which should be placed between the stimulating and recording electrodes. EMG equipment is easy to assemble and not very bulky. The arm and hand need not be fixed during the recording. Muscles like diaphragm and larynx can also be monitored, which is not possible with MMG. Motor block during regional anesthesia can be assessed with EMG. Thus EMG has many advantages over the MMG. However, it also has some disadvantages. Direct muscle stimulation can also cause inaccurate reading and there can be interference from diathermy. Any change in the skin temperature and hand

movements can adversely affect the readings to a greater degree than with MMG.

What is the principle of Acceleromyography?

Acceleromyography: Measurement of acceleration of the contracting muscle. It is based on Newtons second law—Force = mass × acceleration. Acceleration would be directly proportional to the force if the mass is constant. A piezoelectric transducer is attached to the thumb. When the nerve is stimulated, the movement of the thumb will produce a voltage, which is proportional to its acceleration. This voltage is converted to an electrical signal and displayed. Unrestricted movement of the thumb is required for accuracy. Acceleromyography is mainly used to monitor the TOF ratio.

Are you aware of any other method of measuring the evoked responses?

Three new methods are available for measuring the evoked responses:

Pizoelectric neuromuscular monitors: They measure the evoked electrical response in a piezoelectric film sensor attached to the muscle. It is based on the principle that stretching or bending a flexible piezoelectric film, in response to nerve stimulation generates a voltage that is proportional to the amount of stretching and bending.

Phonomyography: It is a relatively new method. Special microphones are used to record the intrinsic low frequency sounds generated by contraction of the skeletal muscles. The advantage of this method is that it can be applied to any muscle (diaphragm, larynx, adductor pollicis and eye muscles) and is easy to apply.

Kinemyography: There is a piezoelectric sensor placed between the thumb and forefinger. Movement of the thumb following nerve stimulation changes the shape of the sensor material. This results in a flow of current, which is measured as a change in potential that is proportional to the amount of distortion. This technology is used to measure single twitch, TOF and double burst.

Which is the most common site for neuromuscular monitoring and why?

Ulnar nerve is the most common site for stimulation. Direct muscle stimulation is avoided as muscle and nerve lie on the opposite side of the arm. The electrodes are placed along the ulnar border of the forearm, with assessment of thumb adduction. The distal (stimulating) electrode should be placed 1 cm proximal to the point at which the proximal flexion crease of the wrist crosses the radial side of the tendon to flexor carpi ulnaris muscle. The proximal electrode should be placed 2–6 cm proximal to the distal electrode. Muscle contraction is easily seen, felt and quantified. Easily accessible at all times. The adductor pollicis muscle is sensitive to neuromuscular blocking drugs and closely reflects the muscles of the upper airway.

Which other sites can be used for neuromuscular monitoring?

Tibial nerve: Electrodes are placed behind the medial malleolus, with assessment of big toe plantar flexion.

Facial nerve: The stimulating (negative) electrode is placed anterior to the tragus of the ear, with assessment of facial muscle contraction. The positive electrode can be placed over the forehead. Underestimation of the degree of blockade is common, because of direct muscle stimulation and relative insensitivity of the facial muscles to neuromuscular blocking drugs.

Mandibular nerve: Stimulating electrode is placed anterior and inferior to zygomatic arch and the positive electrode is placed over the forehead. Stimulation of the nerve causes closure of the jaw due to contaction of the masseter.

Accessory nerve: One electrode is placed behind the mastoid process and the other at the posterior border of sternomastoid. Stimulation causes contraction of the sternomastoid and trapezius muscles and is easier to visualize than following stimulation of the facial nerve. There have been reports of asystole following tetanic stimulation when the upper electrode was placed anterior to the ear. It has been attributed to the stimulation of the vagus via the cranial root of the accessory nerve.

Common peroneal nerve: Electrodes are placed lateral to the neck of the fibula, with assessment of foot dorsiflexion.

What are the various patterns of stimulation? Can you describe any one pattern of stimulation?

- *Single twitch (0.1–1.0 Hz):* This pattern of stimulation is useful before induction to determine the level at which supramaximal stimulus is obtained. A single twitch at 1

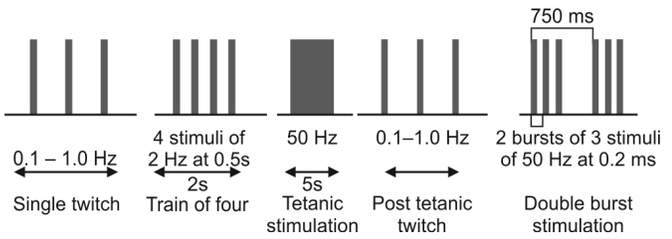

Fig. 53.1: Various patterns of stimultation

Hz (1 twitch every second) is used. The current at which there is no further increase in the evoked response will be the maximal stimulus. The supramaximal stimulus will be 25% above the maximal stimulus.

A single twitch at 0.1 Hz (1 twitch every 10s) is useful to determine the onset of neuromuscular block. There will be progressively diminished response if the stimulus frequency is greater than 1Hz. This can result in overestimation of the block. Prerelaxant control response is noted and then subsequent responses are expressed as % of control (T1% or T1/Tc). In both depolarizing and non-depolarizing blocks there is a progressive decrease in twitch height. The twitch response will only be depressed when a neuromuscular blocking agent occupies 75% of the postsynaptic nicotinic receptors. To assess deep neuromuscular block single twitches are also used in the post-tetanic count, but in this instance a control twitch height is not required. The major limitation to this technique is the need to measure a control twitch before administering the neuromuscular blocking agent. Also it cannot distinguish between depolarizing and non depolarizing block.

- *Tetanic stimulation* (50-100 Hz) for 3-5 seconds may be repeated every 5-10 minutes. It should not be repeated more frequently than every 2 mins. In normal muscle sustained contraction is seen while with a non-depolarizing block there is fade in the response. Muscular fatigue may develop at higher frequencies (100–200 Hz) of stimulation. A stimulation frequency of 50 Hz does not cause muscle fatigue and the degree of fade corresponds more closely to the degree of neuromuscular block. Tetanic stimulation does not cause a fade during partial depolarizing block. The amplitude of the evoked response will be lower but the tetanic contraction will be maintained. This pattern of stimulation is very sensitive and can elicit minor degrees of neuromuscular block, which is potentially useful in the postoperative recovery room. However, its use is limited by the fact that it is extremely painful. Also during the late phase of neuromuscular recovery, tetanic stimulations can cause antagonism of neuromuscular blockade in the stimulated muscle. As a result of this the response in the tested site may no longer be representative of other muscle groups.
- *Post-tetanic count:* Used to assess intense neuromuscular blockade. There may be no response to TOF or single twitch stimulation during profound non-depolarizing neuromuscular block. In such situations, a post-tetanic count (PTC) may be useful. A 5 second tetanic stimulus at 50 Hz is administered, followed 3 second later by further single twitches at 1 Hz. There would be no response to the single twitches during intense neuromuscular blockade. However, when the blockade dissipates there would be a first response to post tetanic twitch stimulation even before the first response to TOF reappears. For any neuromuscular blocking drug the number of post-tetanic twitch responses at a given time would be related to the time until return of first response of TOF stimulation. It should not be performed more than once in 6 minutes.
- *Train-of-four (TOF):* Four pulses at 2 Hz for 2 seconds, i.e. 4 stimuli at 0.5 second interval. In depolarizing block there is equal depression in the twitch height and in non-depolarizing block there is a fade in the twitch height. Fade in the TOF response after administration of succinylcholine indicates the development of phase II block. During non-depolarizing block the TOF count (number of twitches) correlates with the degree of blockade. There is successive decrease in the four responses, with eventual disappearance of the 4th, 3rd, 2nd and 1st twitches at 75%, 80%, 90% and 100% blockade respectively. During recovery the twitches reappear in the reverse order. The advantages of TOF over other patterns of stimulation are that no control twitch is needed; it is less painful than tetanic stimulation and it does not affect the degree of neuromuscular block.
- *Double-burst stimulation:* DBS was developed to enable the anesthetist to detect even small degrees of neuromuscular block clinically. It is used to assess recovery from non-depolarizing blockade. In DBS, two short bursts of tetanus at 50 Hz at a supramaximal current are applied to a nerve. Typically, each burst will have three impulses (DBS3,3) lasting 0.2 ms. Each impulse is delivered every 20 ms and the two bursts are separated by 750 ms. In unparalyzed muscle, two separate muscle contractions of equal intensity will occur. In muscle partially paralyzed with a non-depolarizing agent, the response to the second burst is reduced. This is the phenomenon of fade. The ratio of the magnitude of the second stimulus to the first is known as the DBS ratio. It is more sensitive at detecting fade than TOF. However, one has to remember that the absence of fade in the tactile response to DBS and TOF does not exclude residual neuromuscular blockade.

What do you mean by TOF count and TOF ratio?

TOF count is the number of palpable muscle twitches. Twitch suppression of 90% would equate to a TOF count of 1 or less. TOF ratio is the force of the first twitch divided by the force of the fourth. It may be repeated every 10-15 seconds.

Suggested suitable values during anesthesia;

- TOF count of 1 or PTC should be zero for tracheal intubation
- TOF count of 1-2 for maintenance; deeper levels may be required for complete diaphragmatic paralysis

- TOF count of 3-4 before attempting reversal of blockade, especially with long- acting drugs.

Relationship between TOF ratio and neuromuscular recovery:

- TOF ratio 0.4—patient is unable to lift the head or arm. Tidal volume may be normal but vital capacity and inspiratory force may be reduced.
- TOF ratio 0.6—most patients are able to lift the head for 3 seconds open their eyes widely and protrude their tongue, but inspiratory force and vital capacity is reduced.
- TOF ratio 0.7—patient can cough and lift their head for atleast 5 seconds but their grip strength is low
- TOF ratio > 0.8—vital capacity and inspiratory force are normal, but patient may have diplopia and facial weakness
- TOF ratio (at the thumb) of 0.9 for adequate maintenance of spontaneous ventilation. Studies have shown that if TOF ratio is < 0.9 there is functional impairment of muscles of pharynx and upper esophagus, which can predispose to regurgitation and aspiration.

Why do you get a fade on tetanic stimulation?

Fade in response to tetanic stimulation is considered a pre-synaptic event. Tetanic stimulation results in the release of large amounts of acetylcholine release from the immediately available stores in the nerve terminal. When these stores are depleted the rate of acetylcholine decreases until there is equilibrium between mobilization and synthesis of acetylcholine. In spite of the decrease in acetylcholine release the muscle response is maintained as the amount of acetylcholine released is much greater than that required to evoke a response. However after giving a non-depolarizing neuromuscular blocking drug the number of free cholinergic receptors on the postsynaptic membrane is reduced. The neuromuscular blocking drug may also impair the release of acetylcholine within the nerve terminal. As a result of this the decrease in release of acetylcholine during tetanic stimulation produces fade.

What is post tetanic facilitation?

During moderate non-depolarizing block if you give tetanic stimulation (50 Hz) for 5 seconds followed by post–tetanic twitch stimulation (1.0 Hz) there is an increase in the twitch height, which is due to post-tetanic facilitation of transmission. This occurs because tetanic stimulation causes mobilization and synthesis of acetylcholine which continues for some time even though the stimulation has stopped. The degree and duration of post-tetanic facilitation depends on the degree of neuromuscular blockade.

What are the clinical signs of postoperative neuromuscular recovery?

Unreliable – sustained eye opening, protrusion of tongue, arm lift to opposite shoulder, normal tidal volume, normal or near normal vital capacity, maximum inspiratory pressure < 40–50 cmH_2O

Reliable – sustained head lift for 5 seconds, sustained leg lift for 5 secs, sustained hand grip for 5 secs, sustained tongue protrusion, maximum inspiratory pressure > 40–50 cm of H_2O, effective cough or eye opening for 5 secs without any diplopia. The ability to resist the removal of a tongue depressor from between the clenched teeth would be a more sensitive test.

Is neuromuscular monitoring reliable in hypothermic patients?

No, neuromuscular monitoring is not reliable in hypothermic patients. Both central and peripheral cooling reduces the twitch tension and TOF ratio. Cooling affects nerve conduction, reduces the rate of acetylcholine release and muscle contraction. Cooling reduces the current delivery by increasing skin impedance. This results in a submaximal response. Also cooling causes vasoconstriction, which reduces the blood flow to the muscles which in turn decreases the rate of removal of muscle relaxant from the neuromuscular junction. As a result of all these factors the evoked muscle response will be different between a warm and cold extremity.

What is your choice of stimulation pattern and which muscle would you monitor during induction of anesthesia?

The central muscles like diaphragm and larynx have a very good blood supply, as a result of which the onset and offset of the block is faster. The peripheral muscles like adductor pollicis have a poor blood supply due to which the onset of block is delayed. However, they are very sensitive to neuromuscular blockers due to which the recovery is slow. The muscles of upper airway and pharynx behave like the central muscle at onset (faster onset) and like the peripheral muscles during recovery (delayed recovery). Also as compared to the peripheral muscles the diaphragm is the most resistant to both depolarizing and non-depolarizing agents. During induction of anesthesia and tracheal intubation the diaphragm and the muscles of the jaw and larynx must be completely paralyzed. If the adductor pollicis is used to monitor the neuromuscular function during induction then the absence of response to TOF would not ensure complete paralysis of the diaphragm. The orbicularis

oculi is probably the ideal muscle to monitor at the time of induction as it is more similar to a central muscle—onset of block will be similar to the laryngeal muscles and diaphragm. During induction single twitch stimulation should be used to determine the supramaximal stimulus. During intubation TOF count of 1 will correspond to optimal intubating conditions.

Which muscle should be monitored during maintenance of anesthesia and why?

The diaphragm is relatively resistant to neuromuscular block. If you monitor a more sensitive peripheral muscle, such as the adductor pollicis it may not adequately reflect the degree of block required during maintenance of anesthesia. A central muscle which is resistant to neuromuscular block, for example, orbicularis oculi, will reflect the diaphragm more closely and should be monitored during maintenance of anesthesia.

During maintenance of anesthesia the TOF count should be 1or 2. For profound neuromuscular block the PTC should be zero.

Which muscle should be monitored during reversal?

During reversal a peripheral muscle, such as adductor pollicis should be monitored. As the peripheral muscles have a slower recovery time, the recovery of the peripheral muscles would indicate the respiratory muscles have recovered to a greater degree. This provides a larger margin of safety.

Before administering a neuromuscular antagonist, the TOF count should be at least 3 or TOF ratio should be 0.7.

If you are using neuromuscular monitoring then when would you decide to extubate the patient?

While extubating any patient all 4 responses to TOF should be present and the strength of all 4 response should be the same (TOF ratio of 0.9 – 1). However, visual and tactile assessment of the strength of the stimulus is not very sensitive. The strength of DBS is better appreciated than the stimulus strength of TOF. Hence use DBS to assess the adequacy of reversal if you do not have a monitor which can measure the TOF ratio. However it is important to remember that tactile evaluation is not a very sensitive method and even though both responses to DBS appear same in magnitude it still does not exclude clinically significant residual blockade. Hence manual evaluation of responses to nerve stimulation should always be considered in relation to reliable clinical signs and symptoms of residual neuromuscular blockade. Objective assessment is ideal and to exclude clinically significant residual neuromuscular blockade the TOF ratio should be > 0.9.

Suggested Reading

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54 Oxygen Therapy Devices and Manual Resuscitator

N Amin, V Patil

Which patients need oxygen therapy?

American College of Chest Physicians and National Heart Lung and Blood Institute recommendations for instituting oxygen therapy are:

- Cardiac and respiratory arrest
- Hypoxemia (PaO_2 < 60 mm Hg, SaO_2 < 90%)
- Hypotension (systolic BP < 100 mm Hg)
- Low cardiac output and metabolic acidosis
- Respiratory distress (respiratory rate > 24/min).

While providing oxygen therapy, attempts must be made to find the underlying pathology and treat it.

What is the difference between hypoxia and hypoxemia?

Hypoxia is lack of oxygen at the tissue level whereas hypoxemia is low oxygen tension (< 60 mm Hg) in the arterial blood.

What are the possible causes of hypoxia?

Hypoxia could be due to arterial hypoxemia or failure of the oxygen hemoglobin transport system.

- Arterial hypoxemia
  - Low inspired oxygen partial pressure (high altitude)
  - Alveolar hypoventilation (sleep apnea, narcotic overdose, neuromuscular disorders, anesthetics and sedatives)
  - Ventilation-perfusion mismatch (atelectasis, pneumonia acute asthma)
  - Right to left shunts (pneumonia, pulmonary embolism, arteriovenous malformation)
- Failure of oxygen hemoglobin transport system
  - Inadequate tissue perfusion (cardiac failure, myocardial infarction, hypovolemic shock)
  - Low hemoglobin concentration (anemia, trauma)
  - Abnormal oxygen dissociation curve (hemoglobinopathies, high carboxyhemoglobin)
  - Increased oxygen requirements in hypermetabolic states
  - Histotoxic poisoning of intracellular enzymes (cyanide poisoning, septicemia).

Depending on the cause and severity, oxygen can be supplemented from a range of oxygen therapy devices. These devices deliver oxygen at or just above atmospheric pressure (normobaric oxygen therapy). There are some patients in whom, either the oxygen carrying capacity of hemoglobin is compromised (e.g. carbon monoxide poisoning) or extra tissue oxygen is required (in severe burns and tissue infections). In these patients, supplemental oxygen may be delivered by dissolving it under pressure, in plasma (hyperbaric oxygen therapy).

What are the indications for oxygen therapy in the postoperative period?

The indications for oxygen therapy would be:

- *Patient factors:* Cardiorespiratory disease, elderly, shivering, obesity
- *Surgical factors:* Thoracic surgery, upper abdominal surgery
- *Physiological factors:* Hypovolemia, hypotension, anemia
- *Postoperative analgesic technique:* Patient controlled analgesia, IV opioid infusion.

How would you classify oxygen therapy devices?

There are a number of methods of classifying oxygen therapy devices.

They can be classified as follows:

- Performance of the device: Fixed (venturi mask) or variable performance (nasal prong, face mask)

- *Flows delivered by the device:* Low flow (nasal prongs, face mask) or high flow (venturi mask)
- *Patient:* Dependent (face mask, nasal prongs, CPAP device) or patient-independent (Ventilators)
- *Degree of dependency:* Low dependency (face mask, nasal prongs), medium dependency (CPAP mask and equipment), high dependency (noninvasive or invasive positive pressure ventilation) .

What is the importance of peak inspiratory flow rates while choosing the oxygen therapy devices?

If a patient is breathing spontaneously from a T- piece with no reservoir and zero fresh gas flow (FGF), then all the inspired gas would be room air. If the FGF is increased to100 L/m, then all the inspired gas would be taken from the FGF and the FiO_2 will be the same as the fresh gas. If the FGF are decreased from 100 L/m, there will be a point at which the FGF will not meet the patient's requirements. Below this flow, the patient will entrain room air which will dilute the Flo_2 of the fresh gas. This occurs when the FGF is less than the peak inspiratory flow.

For example: If a patient is breathing at a rate of 10 breaths/m, tidal volume is 500 mL and inspiratory-to-expiratory ratio is 1:2 then each respiratory cycle will be of 6 seconds with an inspiratory time of 2 seconds. To inspire a tidal volume of 500 mL over 2 seconds, the mean inspiratory flow should be 0.25 L/s or 15 L/min. If the patient is breathing with a square wave pattern, the peak inspiratory flow would be the same as the mean inspiratory flow. However, if the breathing pattern is sine wave the peak flow must be higher than the mean flow. Normally, the peak inspiratory flow rates are 60–70 L/min and it can be higher in patients who are hyperventilating or exercising.

If a patient receives a FGF of 10 L/m then at the start of inspiration, all inspiratory gas will be taken from FGF. However, entrainment of room air will occur when the inspiratory flow exceeds 10 L/m. After this point, the Flo_2 will depend on the amount of room air that is entrained. Therefore, in order to deliver a known and fixed Flo_2 to the patient via a face mask, the FGF should exceed peak inspiratory flow.

What do you mean by fixed or variable performance devices? Give examples of each.

Supplemental oxygen can be supplied by a variety of devices. The effectiveness of each device is determined by the capacity of each device to deliver sufficient oxygen at high enough flow rate to match the patients spontaneous inspiratory flow rate. In a non-intubated patient breathing in an "open" system the capacity of the oxygen therapy device to meet the patient's inspiratory flow will determines how much room air will be entrained. The FiO_2 delivered from the oxygen source will be diluted by the entrained room air. Therefore, the oxygen delivery systems are categorized as either variable performance (no control on FiO_2) or high-flow fixed performance (controlled FiO_2) systems.

The variable performance systems are 'patient- dependent' because the FiO_2 that the patient receives will change with changes in the respiratory parameters. For example, nasal catheters, nasal cannulae and masks with or without a rebreathing bag.

The fixed performance systems are usually considered as 'patient-independent' because regardless of changes in respiratory parameters, the patient will receive a constant, predetermined inspired oxygen concentration (FiO_2). For example, Ventimask.

What are the factors which control the FiO_2 in variable performance devices?

In variable performance devices FiO_2 varies with the phase and pattern of respiration. Entrainment of room air occurs during the use of these devices, as the oxygen flow delivered by these devices is less than patient's peak inspiratory flow. During normal respiration the rate and tidal volume varies with each breath (Fig. 54.1). The exact proportion of air dilution cannot be known due to the irregular respiratory pattern. As a result of this, the FiO_2 not only varies with each breath but also varies from patient to patient.

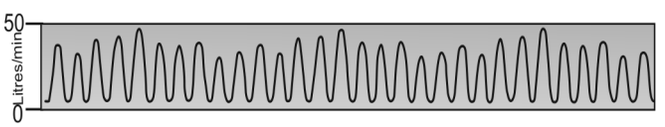


Fig. 54.1 Variation in tidal volume

The factors which control the FiO_2 are:

- Equipment factors—Oxygen flow rate, mask volume (100–200 mL) and volume of the reservoir bag (600–1000 mL), fit of the mask to the face, size and placement of the vents.
- Patient factors—respiratory rate, tidal volume and peak inspiratory flow rates, anatomical reservoir. The anatomical reservoir varies with the size of the subject. In infants the anatomical dead space is 3.3 mL/kg. It decreases to the adult value of approximately 2 mL/kg by the age of 6 years. During the period of development the intrathoracic anatomical dead space remains constant at 1 mL/kg while the volumes of the nose, mouth and pharynx change relative to the body weight. From early adulthood, the anatomic dead space increases by approximately 1mL/year.
- Additional factors—presence of other gases or vapors e.g. humidifier.

Can you explain how the inspired FiO_2 varies with a variable performance device?

We have already mentioned the various factors that affect the fraction of inspired oxygen (FiO_2).

When giving oxygen therapy there is pooling of oxygen in the anatomical reservoir of the upper airways, at the end of exhalation. The oxygen collected in the anatomical and the equipment reservoir is available for the next inspiration. The anatomical reservoir (nose and nasopharynx) is 50 mL, mask space varies from 100–200 mL and reservoir bag if present may have a volume of 600–1000 mL. Depending on the reservoir capacity it may or may not be able to meet the tidal volume requirements. If oxygen flow rate is constant then the FiO_2 will be low if the respiratory rate, tidal volume and peak inspiratory flow rates are high.

Consider the following example of a normal breathing pattern: Tidal volume (VT) 500 mL, respiratory rate 20/min, inspiratory time 1 s, expiratory time 2 s (including end-expiratory pause 0.5 s), anatomic reservoir 50 mL. If this patient is receiving oxygen at 2 L/min what would the FiO_2 be?

- 2 L/min = 33 mL/s (2000 mL/60 s)
- 17 mL of 100% oxygen from the anatomic reservoir (0.5 s end-expiratory pause x 33 mL/s)
- 33 mL of 100% oxygen during inspiration (inspiratory time- 1 s)
- 450 ml of 21% oxygen (room air) = 95 mL of 100% oxygen
- 17 + 33 + 95 = 145 mL of 100% oxygen
- FiO_2 = 145 mL of oxygen/500 mL = 0.29%
- 6 L/min = 100 mL/s (6000 mL/60 s)
- 50 mL of 100% oxygen from the anatomic reservoir (0.5 s end-expiratory pause × 100 mL/s)
- 00 mL of 100% oxygen during inspiration (inspiratory time- 1 s)
- 350 mL of 21% oxygen (room air) = 73 mL of 100% oxygen
- 50 + 100 + 73 = 223 mL of 100% oxygen
- FiO_2 = 223 mL of oxygen/ 500 mL = 0.44%
- If VT-500 mL and RR is 10 then inspiratory time is 2 s, expiratory time 4 s, end expiratory pause 0.5 s. Now doing the above calculations with 2 L/min = 33 mL/s (2000 mL/60 s)
- 17 mL of 100% oxygen from the anatomic reservoir (0.5 s end-expiratory pause x 33 mL/s)
- 66 mL of 100% oxygen during inspiration (inspiratory time 2 s × 33 mL)
- 417 mL of 21% oxygen (room air) = 87 mL of 100% oxygen
- 17 + 66 + 87 = 170 mL of 100% oxygen
- FiO_2 = 170 mL of oxygen/500 mL = 0.34%.

These are just approximate values. There are various other factors which can influence the FiO_2 like changes in the tidal volume or the end expiratory pause.

What do you mean by low-flow system? Give examples.

Oxygen therapy devices are also classified as high or low-flow systems, depending on the total gas flows that are delivered to the patient. Low-flow systems deliver oxygen at flows that are less than the patient's inspiratory flow rate leading to air entrainment. As a result of this inhaled oxygen concentration (FiO_2) may be low or high, depending on the specific device and the patient's inspiratory flow rate. As oxygen delivery of low-flow systems varies with the patient's inspiratory flow they are also classified as variable performance oxygen delivery systems. However, an anesthetic breathing system which has a tight fitting mask and a reservoir is a low flow system which can deliver a known FiO_2. Examples of low flow systems:

- No capacity system: Nasal cannula, nasopharyngeal catheters
- Low capacity system (capacity < 100 mL): simple and nebuliser face mask for children, tracheostomy mask
- Medium capacity system (capacity 100–250 mL): Simple and nebulizer face mask for adults
- High capacity system (capacity 250–1500 mL): Face mask with reservoir bag
- Very high capacity system (capacity >1500 mL): Incubators, oxygen hood, oxygen tents.

How would you describe any oxygen therapy device?

First classify the device according to the flows (low flow or high flow device), the performance of the device (variable or fixed performance) and the capacity of the device (no capacity, low capacity, medium capacity or large capacity). Then give a brief description of the device. What is the FiO_2 delivered, where it is used, what are the advantages, disadvantages and limitations of the device. How is the device sterilized.

Can you give a brief description of any low-flow oxygen delivery system?

No Capacity Systems

Nasal cannulae are low flow variable performance devices with no capacity. They consist of two soft prongs that arise from oxygen supply tubing. The prongs are inserted into the patient's anterior nares, and the tubing is secured to the patient's face by an adjustable strap. Oxygen is delivered through the cannula, but at low flow rates between 0.5–5.0 L/min. The patient's nasopharynx acts as an anatomic reservoir. When the patient inspires, the oxygen is diluted with room

air. The degree of dilution depends on the patients inspiratory flows. The resulting FiO_2 depends on the flow rate of oxygen, patient's minute ventilation and inspiratory flow and the volume of the nasopharynx. The FiO_2 cannot therefore be precisely controlled, but the maximal tracheal FiO_2 is not likely to exceed 0.4 (40%). Mouth breathing causes inspiratory airflow. This produces a Venturi effect in the posterior pharynx entraining oxygen from the nose.

Approximately nasal cannulas deliver 4% oxygen per liter flow

- Flow 1 L/m: 24%
- Flow 2 L/m: 28%
- Flow 3 L/m: 32%
- Flow 4 L/m: 36%
- Flow 5 L/m: 40%.

Higher flow rates should not be used as they have a drying and irritating effect on the nasal mucosa and do not result in much higher FiO_2. High flows can cause headaches and nose bleeds. Since it is a variable performance system a fixed FiO_2 cannot be assigned to patients on low-flow system. Patients are more compliant with the nasal cannula than the face mask as they are able to speak, eat and drink (Fig. 54.2).

Fig. 54.2 Nasal cannula

Advantages

- Comfortable for patient
- Ideal for claustrophobic patients
- Ideal for oxygen dependent patients requiring small amounts of oxygen especially at home
- Humidification not required up to 4 L/m.

Disadvantages

- Unpredictable FiO_2
- Maximum estimated FiO_2 0.4
- Not appropriate for patients in respiratory distress.

Limitations

- Any change in the patient's respiratory pattern can cause air entrainment leading to fluctuations in FiO_2
- Prongs are difficult to keep in position, especially in small infants
- Use may be limited by the presence of excessive mucus drainage, mucosal edema, or a deviated septum
- Maximum flow should not be more than 2 L/m in infants and newborns.

Equipment Related Potential Harm

- Skin irritation can result from material used to secure the cannula or from local allergic reaction to polyvinyl chloride
- Improper sizing can lead to nasal obstruction or irritation
- Displacement can lead to loss of oxygen delivery
- Inadvertent continuous positive airway pressure (CPAP) may be administered depending upon the size of the nasal cannula, the gas flow, and the infant's anatomy
- Irritation and drying of nasal mucosa can result if flows are excessive.

Nasopharyngeal catheters are variable performance low flow device with no capacity. They are soft tubes with several distal holes. Catheters are available from 8–14 FG size. The catheter should be well-lubricated prior to insertion. The depth of insertion should be equal to the distance from the ala nasi to the tragus. The tube is secured to the patient's face and connected to oxygen supply tubing. Oxygen flows from the catheter into the patient's oropharynx, which acts as an anatomic reservoir. The FiO_2 varies with the patient's inspiratory flow. The patient should be observed for evidence of catheter occlusion, and the catheter should be alternated between nares every 8 to 12 hours and changed daily.

Limitations

- Method is in less common use because of the complexity of care
- FiO_2 is difficult to control and measure
- Use may be limited by excessive mucus drainage, mucosal edema, or the presence of a deviated septum
- Catheter should be cleared frequently to prevent occlusion of the distal holes
- Catheter sizes less than 8 Fr are less effective in oxygen delivery
- Lower oxygen concentrations are delivered if the catheter is placed in the nose rather than in the pharynx.

Equipment Related Potential Harm

- Improper insertion can cause gagging and nasal or pharyngeal trauma
- Improper sizing can lead to nasal obstruction or irritation
- Excessive oxygen flow can produce pain in the frontal sinuses
- Excessive secretions and/or mucosal inflammation can result
- Skin irritation may result from material used to secure the cannula and/or from local allergic reaction to polyvinyl chloride
- Occlusion of distal openings may occur
- Excessive flow may cause gastric distention.

Small Capacity Systems

Simple oxygen masks are low flow variable performance device with small capacity. The pediatric face masks have low capacity (70–100 mL) and the adult facemask have medium capacity (100–250 mL). They are plastic reservoirs designed to fit over the patient's nose and mouth. An elastic strap is used to secure the mask around the patient's head. Higher FiO_2 can be delivered if the mask volume is increased. Some masks may have a deformable metal which allow the mask to be shaped around the nose to achieve a closer fit. Oxygen is delivered through a small-bore tube connected to the base of the mask. The oxygen that gets collected in the apparatus dead space at the end of expiration is inhaled at the beginning of the next breath. If there is no expiratory pause, alveolar gas may be rebreathed from the mask at the start of inspiration. Exhaled gases are vented out through the holes on each side of the mask, which also serve as room-air entrainment ports. As it is a low flow device there is air entrainment during inspiration and the Fio_2 varies with the patient's inspiratory flow, mask fit, and patient respiratory pattern. The Fio_2 can be reduced if the spontaneous inspiratory flows are high, mask is loose or oxygen flow is low.

Simple oxygen masks can provide 35–60% FiO_2, depending on fit, at flow rates from 5–10 L/m (Table 54.1). Caution should be taken when using a simple mask where accurate delivery of low concentrations of oxygen is required (Fig. 54.3).

Table 54.1 Flow rate and Fio_2 with simple mask

O_2 flow rate L/min	FiO_2
5–6	0.4
6–7	0.5
7–8	0.6

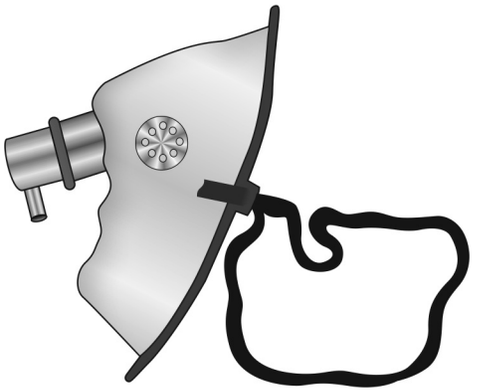


Fig. 54.3 Simple otygen mask

Advantages

- Quick and easy to setup and apply.

Disadvantages

- Unpredictable FiO_2
- Not more than 0.5 FiO_2 can be delivered.

Watch for

- CO_2 rebreathing can occur with flow rates less than 2 liters/min or if minute ventilation is very high.

Aerosol (Nebulizer) Face Mask

It is a variable-oxygen, low flow device with medium capacity. The mask with large side holes is attached by large-bore tubing to a nebulizer, which delivers water/drug in aerosol form. Humidified oxygen can be delivered with a nebulizer mask.

Advantages

- Humidified gases.

Disadvantages

- Nebulizer is noisy
- Unpredictable FiO_2
- Maximum FiO_2 estimated at 0.50 (same as plain mask).

Watch for

- Attempts to minimize noise by decreasing oxygen flow on flowmeter will interfere with effective nebulization and thus humidification.

Tracheostomy mask is low flow variable performance device with low capacity.

Advantages

- Designed for patients with a tracheostomy.

Disadvantages

- Can require frequent cleaning or replacement if patient is coughing up sputum.

High Capacity Systems

In all of the above systems, most of the oxygen stored in device during exhalation gets wasted as it is blown away by the patient's exhaled gases. Oxygen also gets wasted if the oxygen flow rates are more than the patient's inspiratory flow rates. The large capacity devices have been designed to store this wasted oxygen.

Reservoir Face Mask

It is a high-oxygen, low-flow, variable performance device. These masks provide a higher FiO_2 as there is a reservoir bag attached to the face mask. Oxygen flows directly into the reservoir bag. The flow rate of oxygen is adjusted so that the bag remains completely or partially distended throughout the respiratory cycle. The patient inhales oxygen preferentially from the reservoir. If the reservoir empties during inspiration, then ambient air is entrained from the vents in each side of the mask body if necessary. Oxygen delivery can be maximized by using a mask with a good fit. FiO_2 varies depending on mask fit, capacity of reservoir bag, oxygen flow rates and patients inspiratory flow.
The reservoir face mask can be a partial rebreathing mask or a non rebreathing mask (Fig. 54.4).

Partial rebreathing mask is a simple mask with a reservoir bag. Oxygen flow should always be supplied to maintain the reservoir bag at least one third to one half full on inspiration. The reservoir receives fresh gas plus exhaled gas approximately equal to the volume of the patient's anatomic dead space. The oxygen concentration of the exhaled gases combined with the supply of fresh oxygen, permits the use of flows lower than those necessary for other devices (e.g., non-rebreathing masks), and potentially conserves oxygen use. At a flow of 6–10 L/min the system can provide 40–70% oxygen.

Non-rebreathing masks are similar to partial rebreathing masks but do not permit the mixing of exhaled gases with the fresh gas supply. A series of one-way valves ensures a fresh oxygen supply with minimal dilution from the entrainment of room air. The one-way valve over the reservoir bag prevents entry of expired gas, and the one-way valve over one of the side ports limits entrainment of room air during inspiration. As the flap valves are on the outside they do not impede exhalation. This design provides a higher FiO_2 than the simple and partial-rebreathing masks and the nasal devices provided the mask fits correctly. There should be a minimum flow of 10 L/min. The delivered FiO_2 of this system is 75–90% at flow rates of 12–15 L/m. Some of these masks may have a device which gives a visual indication of the patient's respiratory efforts (respi-check face mask) (Table 54.2).

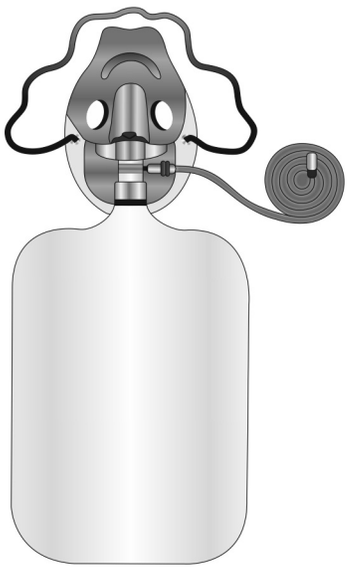


Fig. 54.4 Reservoir face mask

High inspired oxygen concentrations can be achieved with:

- High oxygen flow rates
- Large reservoir bags
- Inclusion of flap valves
- Slower respiratory rates (to allow reservoir bags to fill)
- Smaller tidal volumes.

Advantages

- Fast and easy to set up.

Disadvantages

FiO_2 is variable if there is air entrainment due to poor mask fit.

Watch for

- Reservoir bag must always remain inflated. Deflated bag indicates that the oxygen flow rates are low
- These masks cannot be used for long-term as there is no humidification of gases.

Limitations

- FiO_2 may be unpredictable depending on inspiratory flow and construction of the mask's reservoir.
- May not be well-tolerated by claustrophobic patients
- May interfere with feeding

- The maximum FiO_2 attainable with a simple, non-rebreathing or partial rebreathing mask in neonates, infants, and children has not been well-documented.
- Performance is altered if the entrainment ports are blocked.

Equipment related potential harm

- Aspiration of vomitus may be more likely when a mask is in place in patients with obtunded airway reflexes.
- Irritation may result from tight application.
- Rebreathing of carbon dioxide may occur if total oxygen flow is inadequate.
- Not appropriate for use in neonates.

Table 54.2 Flow rate, Fio_2 with reservoir face mask

O_2 flow rate L/min	No valves	One way valve
3	0.5–0.65	0.45–0.6
6	0.6–0.75	0.6–0.75
9	0.65–0.80	0.65–0.80
12	0.7–0.85	0.7–0.85
15	0.75–0.9	0.75–0.9

A 35 years male patient who is very tachypneic, is receiving oxygen therapy via a Hudson mask with oxygen flows of 6 L/min. He is maintaining a saturation of 90%. Reservoir mask is not available. The nurse thinks that she can increase the FiO_2 if she closes all the leaks around the mask including the holes in the side of the mask by applying sticking plaster. Is she doing the right thing?

No she is not doing the right thing. The Hudson mask is a low flow device. With 6 L/min of oxygen flows approximately 54 L of room air would have to be entrained to match the patient's peak inspiratory flow rates. The air entrainment occurs from the holes at the sides of the mask. If there is a tight seal, there would be no air entrainment and the patient would feel suffocated. Also the holes at the side are for the egress of exhaled gases. Closure of these holes would result in rebreathing.

What do you mean by a high-flow system? Give example.

High-flow systems are fixed-performance oxygen delivery systems as they can provide a specific FiO_2 at flows that meet or exceed the patient's inspiratory flow requirement. While there is no defined cut-off point between low and high flow, most authors place it at about 10–15 L/m. These devices create a constant proportion of air-oxygen mixture in excess of patient inspiratory flow rate and are independent of patient factors or fit to the face. There is no rebreathing with these devices as the CO_2 gets washed out by the gas flow which constantly exceeds the patients demand. However, when the flow is less than the peak inspiratory flow, even the high flow devices become patient-dependent.

To work efficiently and to prevent air dilution a fixed performance device should incorporate:

- A large volume face piece (not less than 280 mL).
- Gas mixture flowing directly towards the nose and mouth.
- Vents positioned well away from the patient airway. For example, ventimask.

What is Venturi effect?

The Venturi effect is based on the Bernoulli principle. The relationship between the velocity and pressure exerted by a moving liquid is described by the Bernoulli's principle: as the velocity of a fluid increases, the pressure exerted by that fluid decreases. This principle can be applied to both liquids and gases.

When a fluid flows through a constriction the fluid velocity must increase to satisfy the equation of continuity. Due to the principle of conservation of energy any gain in kinetic energy due to the increase in fluid velocity must be balanced by a drop in pressure distal to the constriction.

The venturi principle describes how a second fluid can be entrained into the stream of the first. It can either be done through a coaxial arrangement or through a side arm opening into an area of low pressure.

Which other devices are based on the Bernoulli principle?

The Venturi effect may be observed or used in the following:

- In Venturi masks used in medical oxygen therapy
- Sanders jet injector
- Inspirators that mix air and flammable gas in grills, gas stoves, Bunsen burners
- Steam siphons use the kinetic energy from the steam pressure to create a partial vacuum
- Water aspirators that produce a partial vacuum using the kinetic energy from the faucet water pressure
- Atomizers that are used to disperse perfume or spray paint.
- Foam fire fighting nozzles and extinguishers
- Carburetors use the Venturi effect to suck gasoline into an engine's intake air stream
- Industrial vacuum cleaners which operate on compressed air
- Venturi scrubbers used to clean fuel gas emissions.

- Injectors used to add chlorine gas to water treatment chlorination systems
- Steam injectors use the Venturi effect to deliver feed water to a steam locomotive boiler
- Sand blasters used to draw fine sand in and mix it with air.
- A scuba diving regulator to assist the flow of air once it starts flowing
- Modern vaporizers to optimize efficiency
- In recoilless rifles Venturi effect is used to decrease the recoil of firing
- Ventilators
- Automated pool cleaners.

Can you give a brief description of the Venturi mask?

It is a high-flow, fixed performance device. An air-entrainment mask contains a jet orifice and air entrainment ports and is designed to fit over the patient's nose and mouth. The oxygen is supplied to the mask through narrow bore oxygen tubing under pressure. When oxygen under pressure is forced through a small jet orifice entering the mask, a negative pressure is created, which causes room air to be entrained into the mask through the apertures in the Venturi barrel. The resultant air-oxygen mixture containing the prescribed O_2 concentration flows into the face piece for patient breathing. Due to the high-flow rates, the excess gas flushes out the expired CO_2 through the holes on the sides of the mask. As a result, of this there is no rebreathing or no increase in dead space. When oxygen flow is increased, more air is entrained, to maintain a constant oxygen concentration. The system is called HAFOE (high airflow with oxygen enrichment). Variations in the size of the entrainment ports determine the FiO_2 and variation in the oxygen flow rate determines the total flow delivered. The entrainment mechanism is based on the principles described by Bernoulli. The FiO_2 can be precisely controlled from 0.24–0.6 (24%–60%) at high-flow rates by simply exchanging the interchangeable jet nozzles and adjusting the oxygen flow rate (Table 54.3).

In the Ventimask, the Venturi is part of the mask so that air is entrained into the mask, close to the patient. The masks are color-coded and labeled with the inspired oxygen concentration that they will deliver and the flow of oxygen that is required to achieve this. A known FiO_2 can also be delivered to patients breathing spontaneously with an endotracheal tube by attaching the Venturi device to the T piece. Since the device is away from the patient, they receive the gasses at a lower pressure. The patient will continue to receive the set FiO_2 as long as the fresh gas flows exceed the peak inspiratory flow rates.

Table 54.3 Flow rate and Fio_2 with Venturi mask

FiO_2 Provided by the Venturi valve	Oxygen flow to the valve (L/min)	Amount of air entrained (L/min)	Total flow to patient (L/min)
0.24	2	51	53
0.28	4	41	45
0.31	6	41	47
0.35	8	37	45
0.40	10	32	42
0.60	15	15	30

How does one get a fixed FiO_2 with a Venturi mask?

The Venturi oxygen delivery device has two variables—oxygen flow and the entrainment ratio. The entrainment ratio is set by the manufacturer. Any back pressure or suction will change the entrainment ratio and more air will be entrained. If all of the oxygen that is delivered to the mask takes part in the total flow, it is possible to derive a mathematical equation which will calculate the amount of air entrained. The total gas flow will be the sum of the oxygen flow and the amount of air entrained.

Entrained air flow is given by the equation:

$$\text{Entrained air flow} = \frac{O_2 \text{ flow from wall} \times (1 - FiO_2)}{FiO_2 - 0.2}$$

For example, if the oxygen flow is 10 liter/min and FIO_2 0.6, the ratio will be 1:1; i.e. for each liter of oxygen, 1 L of air will be entrained. Tables 54.3 and 54.4 shows the effects of different FiO_2 and different flow rates of oxygen on the total gas flows delivered to the patient.

Table 54.3 Total gas flows with variable Fio_2

FiO_2 L/min	Oxygen flow (driving) L/min	Air flow (entrained) L/min	Total flow L/min
1.0	10	0	10
0.6	10	10	20
0.6	20	20	40
0.5	10	17	27
0.5	15	25	40
0.4	10	30	40
0.24	2	38	40

To get FiO_2 of 1.0 with oxygen flow rates of 10 L/m there should be no air entrainment and the total gas flows delivered to the patient is 10 L/m. If the patient's peak inspiratory flow rates exceed 10 L/m there will be air dilution and the delivered FiO_2 will be lower. From the Table 54.4, we can see that to deliver FiO_2 of 0.6 the oxygen flow rates and the amount of air entrained

are the same. If oxygen flow rate is increased to 15 L/m, then to get FiO_2 of 0.6 the air entrainment should be 15 L/m. But if the patient's peak inspiratory flow rates exceed 30 L/m there will be air dilution which reduces the FiO_2 actually received by the patient. Furthermore, the group of patients who need a high FiO_2 are likely to have peak inspiratory flows higher than normal. Generally, oxygen flow of 10 L/m are sufficient to deliver an FiO_2 of 0.4 as the peak inspiratory flow rate of 40 L/m usually matches with the total flows going to the patient. While designing the Ventimask it must be taken into account that any change in downstream resistance will reduce the air entrainment which in turn will reduce the total flows being delivered to the patient. Also there must be sufficient space available beyond the Venturi to allow mixing of driving and entrained gas. Therefore, a Venturi from one device may not function as expected if fitted to another manufacturer's product.

Can you describe any medium dependency system?

Medium dependency oxygen delivery devices are used when the patient needs some amount of respiratory assistance along with oxygen supplementation. It can only be used in spontaneously breathing patients. For example, CPAP mask. The CPAP device consists of a face mask with head strap, O_2-CPAP valve, flow generator and wide bore tubings. The masks are available in various sizes and cover the mouth and nose. The mask is secured with a harness. O_2-CPAP valves provide CPAP from 2.5–20 cm of water. The flow generator can be plugged into wall outlet for oxygen. It has an ON switch, as well as a switch for adjusting the oxygen flow and oxygen concentration. Wide bore tubing connects the flow generator to the mask. There are 2 one way valves present in the body of the face mask. One is the inlet valve and other is the outlet valve to which the CPAP valve is attached. The facemask, CPAP valve and the wide bore tubings are for single use only.

How do you give oxygen therapy to neonates and infants?

Enclosure systems are used to provide oxygen therapy to neonates and small infants (Figs 54.5A to F).

Oxygen hoods are transparent enclosures designed to surround the head of the neonate or small infant. It has an opening for the neck and comes in different sizes. Hoods are well-tolerated by infants and allow easy access to chest, trunk, and extremities. Due limitation of the size, these hoods can be used only in children below 1 year of age. A continuous flow of humidified oxygen is supplied to the hood. The hoods deliver 80–90% oxygen at a flow rate of 10–15 L/m.

Oxygen tents: Transparent enclosures in larger sizes (so-called tent houses or huts) are available for patients who are too big for the hoods. The subject lies in the tent and CO_2 is removed by soda lime and water vapor by calcium chloride. The temperature in the tent is regulated by flowing oxygen and air over ice chunks (to prevent accumulation of heat of exothermic reaction). The tents are designed for children. FiO_2 of 0.6–0.7 can be achieved with flow rates of 10–12 L/m. The air changes 20 times/h. High output nebulizers can circulate cooled mist in the tent.

Limitations

As the oxygen concentrations may vary within the hood it should be measured as near the nose and mouth as possible. There is a decrease in oxygen concentration whenever the enclosure is opened. Nasal oxygen may need to be supplied during feeding and nursing care of patients who are confined to hoods. Flows > 7 L/m are required to wash out carbon dioxide.

- Devices can be confining and isolating.
- FiO_2 can vary from 0.21 to 1.0.

Temperature of the gases in the hood should be maintained to provide a neutral thermal environment.

Closed incubators are transparent enclosures that provide a warm environment for small infants with temperature instability. Supplemental oxygen can be added to incubators but may result in an increased oxygen concentration. The primary purpose of an incubator is to provide a temperature-controlled environment. FiO_2 is maintained at 0.4. Humidified oxygen can be provided.

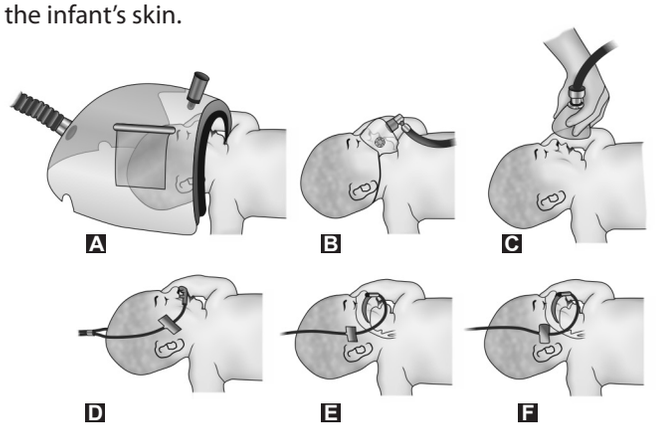
Equipment related potential harm

Prolonged exposure to humidified oxygen may increase risk for cutaneous fungal infection.

Hypoxia or hypercapnia can result from inadequate or loss of gas flow.

Temperature within enclosures should be closely monitored to reduce the potential for cold stress or apnea from overheating in neonates.

Use of an improperly sized hood can result in irritation of the infant's skin.



Figs 50.5A to F (A) Headbox oxygen; (B) Facemask; (C) Oxygen administration by holding an oxygen source near the infant's face; (D) Nasal cannulae; (E) Nasal catheter; (F) Nasopharyngeal catheter

How would you clean and sterilize the oxygen therapy devices?

Low-Flow Systems

Under normal circumstances, low-flow oxygen systems do not present clinically important risk of infection and do not require routine replacement on the same patient.

Nasopharyngeal catheters should be changed every 24 hours.

Transtracheal catheters should be changed every 3 months.

Reservoir systems: Under normal circumstances, reservoir systems do not present clinically important risk of infection and do not require routine replacement on the same patient.

High-Flow Systems

Large-volume nebulizers should be changed every 24 hours when applied to patients with an artificial airway.

Enclosure systems: There is no recommendation regarding the frequency of changing oxygen hoods and reservoirs while in use on the same patient.

What factors decide the choice of an oxygen therapy device?

Need is determined by measurement of inadequate oxygen tensions and saturations by invasive or noninvasive methods and or the presence of clinical indicators. Supplemental oxygen flow should be titrated to maintain adequate oxygen saturation as indicated by pulse oximetry SpO_2 or appropriate arterial or venous blood gas values.

Nasal cannulae and nasopharyngeal catheters are used to provide low level supplemental oxygen. They are also used to supplement oxygen in patients who are being fed so that there is no interruption in oxygen supplementation.

Simple oxygen masks are used to provide supplemental oxygen in the moderate range (0.35 to 0.50, depending on size and minute ventilation) for short periods of time (e.g., during procedures, for transport, in emergency situations).

Air-entrainment nebulizers, can be used when high levels of humidity or aerosol are desired.

Partial rebreathing masks are used to conserve the oxygen supply when higher concentrations (FiO_2 > 0.4, < 0.6) are warranted (e.g. during transport).

Non-rebreathing masks are used to deliver concentrations > 0.60.

Venturi masks are used when precise FiO_2 has to be delivered e.g. COPD.

Hoods are used to provide:

- Controlled FiO_2 in infants and small children
- Controlled FiO_2 and or increased heated humidity to patients who cannot tolerate other devices
- Controlled FiO_2 when the chest, abdomen, and extremities must be accessible to caregivers.

Manual Resuscitators

What are the uses of manual resuscitators?

These are portable manual ventilating devices used for ventilating the patient during:

- During resuscitation and other critical situations.
- Transport of the patient.
- As a standby measure for non-functioning of anesthesia machine.
- Administering anesthesia when anesthesia machine is not available e.g. In field situation.
- For manual ventilation during magnetic resonance imaging.

What are the various types of resuscitators that you know of?

- Laerdal resuscitator bag: Most commonly used
- The AMBU bag resuscitator, Denmark.
- Cardiff infant bag
- Samson Blease bag
- Sanjivani adult resuscitator
- The air-viva resuscitator.

What does AMBU stand for?

Artificial Mandatory Breathing Unit or Air Mask Bag Unit.

Is there a standard resuscitator for all patients?

- No. There is no standard resuscitator for all patients. They are supplied in 3 sizes
- Adult: It has a capacity of 1600 mL and is capable of delivering tidal volume of 600 mL. It can be used in adults weighing more than 30 kg
- Child: They have the capacity of 500 mL and can be used in patients from weight between 7 kg to 30 kg.
- Neonatal: capable of delivering the tidal volume up to 20 to 50 mL and can be used in infants up to 7 kg
- Maximum tidal volume that can be delivered: Adult-1000 mL, Child-350 mL, Infant-205 mL.
- Expiratory resistance—approximately 0.045 cmH_2O/L/min. The expiratory resistance is lower for flows less than 10 L/min.
- Inspiratory resistance—approximately 0.05 cmH_2O/L/min.

Describe the various components of a manual resuscitator.

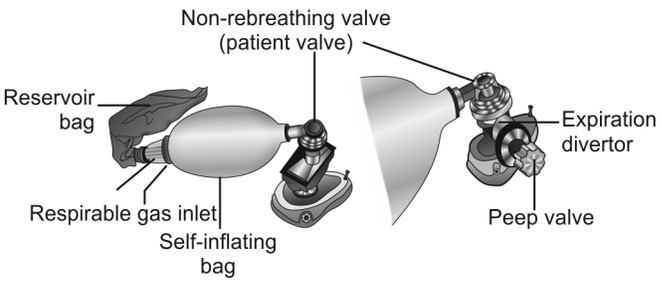


Fig. 54.6 Manual resuscitator

Design and Function

Manual resuscitators have a compressible self-expanding bag, a bag refill valve and a patient intake valve (non-rebreathing valve). Optional components include a pressure-limiting device, reservoir bag with demand valve, PEEP valve, and mechanism for scavenging expired gases (Fig. 54.6).

Self-Inflating Bag

This is oval, cylindrical or sometimes shaped like a base ball. It is made up of silicone and is cream—white in color. The bag is strengthened either by making its wall thicker, or incorporating circular 'ribs' of identical material during manufacture, or by lining it with thick foam so that in the resting state it is expanded. The respirable gas inlet mechanism is housed at one end and the non-rebreathing valve at the other end. Some bags collapse like an accordion during storage. The bag expands during expiration. If the volume of oxygen from the delivery source is inadequate to fill the bag, the difference is made up by intake of air. The rate at which the bag reinflates will determine the maximum respiratory rate. The size of the operator's hands determines the actual tidal volume going to the patient and if the hands are small then it can cause hypoventilation. With prolonged exposure to cold conditions, the cycling rate of the bag may be reduced and the unit may become incapable of delivering satisfactory ventilation.

The respirable gas inlet: It has several components:

Bag refill (intake) valve: It is a one way flap valve which is fitted to the inlet of the self-inflating bag. When the bag is squeezed, the gas pressure inside the bag causes the flap valve to close. This prevents the escape of gas back through the inlet. When the bag is released, its self-inflating characteristics cause entrainment of fresh gas from the respirable gas inlet. This may normally be either air, oxygen or a mixture of both.

A small bore nipple: This is mounted on the inlet, to allow oxygen supply to be attached.

A wide-bore gas inlet: This supplies the bulk of gas (either air or oxygen) entering the bag. The final concentration of oxygen delivered depends on the oxygen flow rates and air entrainment.

A reservoir system: The inlet may be fitted with a reservoir system which stores the oxygen supply. Use of a reservoir significantly increases the oxygen concentration in the inspiratory air. With each bag compression up to 100% oxygen can be delivered to the patient if the oxygen flow rates are greater than the volume given to the patient. The reservoir assembly for the resuscitators consists of a valve unit and either an inflatable bag or an elongated wide bore hose (popular in paediatric resuscitators). The size of the reservoir may limit the oxygen delivered. If the volume of the reservoir is less than that of the bag, the inflowing oxygen may not be sufficient to make up the difference and room air will be drawn in. A large reservoir makes the resuscitator more cumbersome. The reservoir assembly is attached to the wide-bore connector in the rear of the intake valve. The valve unit consists of an overflow valve to prevent overfilling from too high a flow of oxygen and an entrainment valve to entrain air if the bag collapses due to underfilling.

When the bag is not filling, i.e. during squeezing and in the fully expanded state, the oxygen inflow will initially partly fill the ventilation bag and partly flow out through the open end of the intake valve and fill the reservoir bag. During bag re-expansion there is negative pressure in the ventilation bag which causes the inlet valve to open and oxygen from the reservoir enters the bag.

If the ventilation bag is not squeezed, and the mask is not held tightly over a patients face, oxygen will flow continuously through the lip membrane of the patient valve. However, if the mask is held tightly in place, or when patient valve is connected to a tracheal tube, excess oxygen will escape through the outlet membrane of the reservoir valve until the ventilation bag is squeezed. During ventilation if the reservoir bag remains flat it indicates that there is no oxygen supplementation.

Demand Valve

A demand valve connecting a compressed gas source to the self-expanding bag will consistently provide a high inspired oxygen concentration. A negative pressure in the bag triggers a flow of oxygen, which stops at the preset pressure. A demand valve provides warning of supplemental oxygen. Should the demand valve become stuck or the oxygen supply depleted, the bag will not refill.

Patient Valve (Non-Rebreathing Valve)

It is also called as direction control valve. The valve should have a transparent housing which will enable the user to check

the valve function during use. The various components of the valve ensure that during the inspiratory phase the gas flows from the bag into the patient port only and during expiratory phase the exhaled gases escape from the expiratory port without mixing with the fresh gas stored in the bag.

The inspiratory port is the opening through which gas enters the valve from the bag. Expiratory port is the opening through which exhaled gases pass to the atmosphere. The expiratory port may have a tapered 30 mm connector for attachment of the transfer tube of a scavenging system. The ASTM standard requires that such a connector have ridges in its internal lumen so that it cannot accept a 22-mm male connector. The patient valve has a standard15 (ID)/22(OD) mm patient end which connects to all standard masks or tube adaptors. The adult masks and the pediatric circular mask size 2, fit outside the patient valve connector. All other (pediatric) sizes fit inside, to reduce the dead space.

During inspiration, as the self-inflating bag is pressed, the valve directs the gas from the bag to the patient. At the same time, expiration port is blocked. When the squeezing of the bag is stopped and the bag is released, the expiration port opens and allows exhalation of the gases to the atmosphere. The valve may have means to prevent air intake so that a spontaneously breathing patient will inhale only from the bag. A patient who breathes spontaneously can inhale oxygen through the resuscitator with minimal resistance. The capacity of the valve is 7 mL.

Inspiration pressure regulator: The infant and child resuscitators have a patient valve with a special pressure limiting device mounted on the upper valve housing. It is also called as pressure relief valve or pop-off valve. This device is usually in the form of spring-loaded disk with the tension on the spring adjusted so that it opens at a desired pressure. It opens at an opening pressure of 45 cm of water for infant and pediatric bags. It prevents lung injury because of barotrauma and also prevents gastric inflation. In adults the opening pressure is kept at 60 cm of water. One can override this mechanism by either placing a finger over it or fixing it with adhesive tape.

Expiration Diverter

An expiration diverter can be snapped onto the patient valve. The diverter provides an airtight seal to the valve housing but does not prevent the swivel function (possibility of horizontally rotating the bag without interfering with the position of mask or tube) of the valve connecter.

A PEEP valve or equipment for measuring, scavenging or monitoring expired gases can be attached to the standard (30 mm OD) outlet port of the diverter.

What is the FiO_2 delivered to a patient using a laerdal resuscitator?

When used with oxygen the manual resuscitator is a high flow high oxygen device. They can be used with or without supplemental oxygen. Concentrations delivered to the patient depend on oxygen flow rate, use of a reservoir and ventilation technique, e.g. tidal volume, ventilation frequency, time relations during compression—release cycles. The higher minute volume and greater the I:E ratio, the lower the delivered oxygen concentration. The amount of oxygen concentration can be increased by increasing the oxygen flow rate. However the maximum oxygen that can be delivered by a flow meter is 15 L/min. Oxygen concentration can also be increased by attaching reservoir tubing to the fresh gas inlet where the excess oxygen is stored.

Wastage of oxygen can be avoided by adjusting the oxygen flows so that the reservoir bag does not fill completely during ventilation cycles. If 100% oxygen is desirable, best oxygen economy is obtained when flow is regulated so that reservoir neither fills fully nor empties completely during ventilation cycles.

The Tables 54.5 to 54.8 show typical oxygen concentrations that are delivered under a variety of conditions with two popular makes of resuscitators. The differences reflect the size of the relevant reservoirs used.

For delivered oxygen concentrations in laerdal resuscitator see below:

Adult: ventilation bag volume—1600 mL
Reservoir bag volume- 2600 mL

Table 54.5 Delivered Fio_2 with laerdal bag (Adult)

O_2 flow L/min	Tidal vol (mL) × bag cycling rate per min. O_2 concentration (%) using reservoir (without reservoir in brackets)					
	500 × 12	500 × 24	750 × 12	750 × 24	1000 × 12	1000 × 24
3	56 (37)	39 (32)	47 (33)	34 (29)	41 (32)	30 (28)
5	81 (52)	52 (38)	62 (41)	42 (33)	52 (39)	38 (31)
10	100 (73)	84 (48)	100 (56)	65 (42)	84 (55)	53 (39)
12	100 (84)	97 (53)	100 (61)	74 (45)	94 (60)	59 (42)
15	100 (89)	100 (59)	100 (69)	86 (48)	100 (69)	66 (44)

Child: Ventilation bag volume–500 mL
Reservoir bag volume–2600 mL

Table 54.6 Delivered Fio_2 with laerdal bag (Padiatric)

O_2 flow L/min	Tidal volume (mL) × bag cycling rate per min			
	250 × 20		100 × 30	
	Oxygen concentrations (%)			
	With reservoir	Without reservoir	With reservoir	Without reservoir
10	100	75	100	90

Infant: ventilation bag volume—500 mL
Reservoir bag volume—2600 mL

Table 54.7 Delivered Fio_2 with laerdal bag (Infunts)

O_2 flow L/min	Tidal volume (ml) x bag cycling rate per min			
	40 × 30		20 × 40	
	Oxygen concentrations (%)			
	With reservoir	Without reservoir	With reservoir	Without reservoir
4	98	89	98	98

Oxygen concentrations (%) in the Ambu system with reservoir

Table 54.8 Delivered Fio_2 with Ambu bag

O_2 flow L/min	Ventilation volume (mL) x frequency			
	250 × 12	600 × 12	750 × 12	1000 × 12
2	74	43	38	34
5	100	76	65	54
10	100	100	100	87
15	100	100	100	100

Can you give PEEP with a laerdel resuscitator?

Yes. A PEEP valve can be attached to the outlet port of the expiration diverter. The expiration diverter can be attached to the patient valve.

What is the functional analysis of the manual resuscitator?

The laerdal valve is the commonly used valve. It contains two unidirectional valves, a fish mouth valve which ensures unidirectional flow from the bag to the patient, and a disk membrane which ensures flow from patient to atmosphere.

Inspiration: Bag compression causes positive pressure in the ventilation bag. This opens the lip-shaped membrane in the patient valve, and also closes the intake valve.

Spontaneous inhalation: This causes negative pressure in the patient valve. This closes the external disk membrane, to prevent inappropriate intake of ambient air.

Exhalation: Whether spontaneous or passive, exhalation causes positive pressure in the front part of the patient valve. This closes the lip membrane, preventing expired air from entering the bag. At the same time the external disk membrane is lifted away from its seat, allowing expired air to escape to atmosphere.

Bag refill (intake) valve is a one way flap valve which is fitted to the inlet of the self-inflating bag.

Open: Bag re-expansion or inhalation efforts of a spontaneously breathing patient cause's negative pressure in the ventilation bag. This lifts the disk membrane from its seat, leaving the intake ports open so that ambient or oxygen enriched air will be drawn into the bag.

Closed: The intake valve stays closed with neutral or positive pressure in the ventilation bag. This occurs when the bag has reexpanded fully, is being compressed, or when no inhalation effort is made by the patient.

How do you sterilize a manual resuscitator?

Decontamination

Washing and rinsing is the first step in the decontamination process. Disassemble all the parts and rinse in lukewarm water. Then wash the parts in warm water using a detergent that is compatible with the resuscitator material. Rinse all the parts thoroughly in clean water until all traces of detergent is removed. Allow to dry.

Disinfection/Sterilization

The sterilization methods apply to all parts except reservoir bags, head straps, storage pouch and containers. The high level disinfection methods apply to all parts (Table 54.9).

Table 54.9 Disinfenction/Sterilization

	Method	Process parameters		Post-treatment
		Parameters/ concentrations	Exposure time	
Sterilization	Steam autoclaving	Auotoclave at 132°C–135°C	15 mins	Allow parts to cool
	Ethylene oxide	Temperator 55°C, 30%-80% R. h Ethylene oxide Concentrution 630 mg/L	180 mins	Aeration time: 24 h Ambient temperature
High level disinfection	Glutaral-dehyde, (Cidex)	Concentration 2% Ambient temperature	60 mins	Rinse all parts in sterile water and dry
	0.5% sodium hypochlorite	Concentration 0.5% Ambient temperature	20 mins	Rinse all parts in sterile water and dry

How would you check the valve functions to ensure proper operation of the resuscitator after each disassembly-reassembly?

Test valve functions to ensure proper operation of the resuscitator after each disassembly-reassembly. A test lung is required to complete the test procedures.

Intake Valve (Bag Refill Valve)

Compress the ventilation bag with one hand and close its neck opening with your other hand. Release the grip on the bag. Rapid bag re-expansion confirms efficient air intake.

Close the neck opening and try to compress the bag. If the bag cannot be compressed with reasonable force, or if bag

compression forces the air out between your hand and neck of the bag, the valve efficiently prevents backward leakage of air.

Patient Valve (Non-Rebreathing Valve)

Ensure that the patient valve is properly assembled. Attach the patient valve to the bag. Hold a reservoir bag over the patient port. Ensure a tight seal between the patient port and reservoir bag and that the external disk membrane is not blocked. Compress the bag with your other hand several times. Inspect that the lip valve opens during compression. Filling of the reservoir bag in this set-up confirms that the patient valve efficiently directs air to the patient

With the filled reservoir bag held firmly to the valve connector, compress the reservoir bag while watching the external disk membrane mounted on the outside of the patient valve housing. Lifting of the disk membrane from its seat confirms that air is correctly directed to atmosphere instead of being returned to the ventilation bag.

Patient Valve with Pressure Relief Valve

Close patient port connector with your thumb while compressing the bag several times. Visual and audible opening of the relief valve confirms its operation.

Oxygen Nipple in the Respirable Gas Inlet

After compressing the ventilation bag, close the reservoir valve connector on the intake valve and then release the bag compression. There will be slow re-expansion of the bag which confirms that the oxygen nipple is open.
Reservoir flap valves (located in the reservoir valve assembly) Attach a reservoir bag to the reservoir valve assembly. Close port of the reservoir valve and press on the reservoir bag. Compression of the reservoir bag and visual rise of the outlet flap valve confirms that the reservoir valve efficiently vents excessive gas to atmosphere.

With the patient valve in place and the reservoir attached to the reservoir valve assembly, perform several compression-release cycles on the ventilation bag until the reservoir bag is flat and empty. Rapid re-expansion of the ventilation bag after flattening of the reservoir bag confirms that the reservoir valve efficiently lets in ambient air to compensate for lack of gas in the reservoir or insufficient gas flow through the oxygen tubing and nipple.

What are the possible hazards during the use of a manual resuscitator?

- *High airway pressure:* If excessive force is used to compress the bag in intubated patients.
- Barotrauma due to use of high tidal volume.
- Hypocapnea due to hyperventilation.
- Rebreathing can occur if the non-rebreathing valve in stuck in the inspiratory position or is improperly assembled.
- Failure of the pressure limiting device in pediatric resuscitator.
- Hypoventilation if sufficient force is not applied to the manual pressing.
- Low delivered oxygen concentration may result due to insufficient oxygen flow, detached or defective oxygen tubing or problems with the oxygen enrichment device.
- Risk of infection due to improper sterilization.

What are the differences between the laerdel, AMBU and air viva resuscitator?

Table 54.10 Camparision of laerdel AMBU and air viva resuscitator

Laerdel resuscitator	AMBU	Air viva
It is made up of silicone and is cream—white in color and oval in shape. The bag is strengthened either by making its wall thicker, or incorporating circular 'ribs'	It is a black colored self-inflating double ended bag, with foam rubber lining	It is green colored, self-inflating single ended bag
Available in 3 sizes-adult, child and neonate	Available in 2 sizes-adult and baby	
Laerdel non-rebreathing valve is attached to the front of the bag	Ambu E2 non rebreathing valve is attached to the front of the bag	It has an expiratory valve with a common port for inspired and expired gases. Bag refilling occurs during the expiratory phase as a result of which the expired gases get drawn in resulting in partial rebreathing. Oxygen nipple in the front
Distal end has air inlet valve and oxygen inlet connection with or without attached oxygen reservoir	Distal end has air inlet valve and oxygen inlet connection with or without attached oxygen reservoir	It is closed distally
Can be used for anesthesia purpose		Cannot be used for anesthesia purpose. Not advisable if prolonged resuscitation is required

Suggested Reading

1. Dorsch J A, Dorsch SE. Understanding Anesthesia Equipment. 5th Edition.
2. Davey AJ, Diba A. Ward's Anaesthetic equipment. 5th Edition.
3. Ely J, Clapham M. Delivering oxygen to patients. BJA. CEPD Reviews. 2003;3:43–5.

55 Videolaryngoscopes

A Kulkarni, M Desai, S Bhosale

Introduction

Fiberoptic intubation is the gold standard in the management of an anticipated difficult airway. According to the ASA, difficult intubation is defined as need for more than 3 attempts to intubate in 10 minutes. As per the Canadian Society of Difficult Airway, intubation is considered difficult when it is not possible to visualize any portion of vocal cords with conventional laryngoscopy or when the intubation requires more than one attempt, a change in blade, an adjunct to direct laryngoscopy or use of an alternative device. Cormack and Lehane grade III and IV with conventional direct laryngoscopy have been demonstrated to be associated with difficult intubation. In the literature, 5–6% of the intubations in operating room and 16% intubations outside operating room, are reported as unanticipated difficult intubations.

Video-assisted airway management using various forms of video laryngoscope design has been introduced in the last decade to bridge the gap between direct laryngoscopy and fiberoptic bronchoscopy for an anticipated or unanticipated difficult airway. In 2001, Canadian surgeon John A. Pacey was the first to embed a miniature video chip (complementary metal oxide semiconductor) into a modified Macintosh laryngoscope: the GlideScope videolaryngoscope.

What is a videolaryngoscope?

Videolaryngoscopes are indirect laryngoscopes and consist of a handle, laryngoscope blade and a micro video-chip camera embedded into the end of the blade. The image of the laryngoscopic view is transmitted to an external monitor; liquid crystal display (LCD) through a video system. Thus, videolaryngoscopes provide a wider viewing angle, making alignment of the oral, pharyngeal, and tracheal axes less mandatory. It allows the operator to perform tracheal intubation while watching the video screen instead of looking through the small opening of the mouth.

The indirect-optical laryngoscopes are often called videolaryngoscopes; because these laryngoscopes generally use video-technology, so the image of the glottis is transmitted to a remote video screen.

What is the working principle of the video-laryngoscopes?

Direct laryngoscopy is the direct visualization of vocal folds and the glottis. The conventional direct laryngoscope uses a line of sight provided by a rigid viewing instrument with a light on the blade or intraoral portion which requires a direct view of the larynx (Fig. 55.1).

Indirect laryngoscopes allow the operator to visualize the virtual image of patient's vocal cords by a means other than obtaining a direct line of sight. For the purpose of intubation, this is facilitated by fiberoptic bronchoscopes,

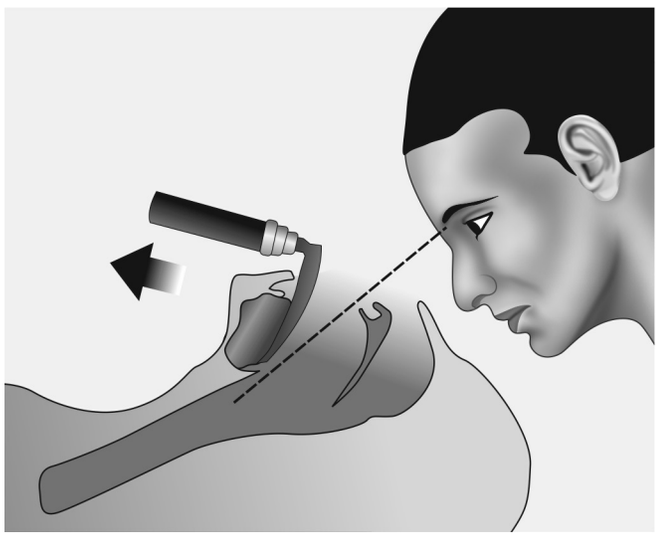


Fig. 55.1 Direct laryngoscopy-line of sight

videolaryngoscopes, fiberoptic stylets and mirror or prism optically-enhanced laryngoscopes.

How are the videolaryngoscopes classified?

Videolaryngoscopes are classified in various categories depending on several features:

1. Method of tube advancement
   a. Videolaryngoscopes with integrated tube channel e.g. Airtraq, Pentax airwayscope
   b. Videolaryngoscopes without integrated tube channel e.g. Glidescope, McGrath, C-Mac.
2. Depending upon the curvature of the blade
   a. Videolaryngoscopes with standard Macintosh Blades, e.g. V-Mac, C-Mac
   b. Videolaryngoscopes with angulated blades, e.g. McGrath series 5 video-laryngoscope, Glidescope video-laryngoscopes (Glidescope original, Ranger and Cobalt), C-Mac with D blade
3. Optical laryngoscopes: Optical laryngoscopes can be equipped with a video-camera and thus function as videolaryngoscopes with a remote screen, e.g. Truview, Airtraq optical laryngoscope, Bullard laryngoscope.

What are the advantages of videolaryngoscopy?

- Display systems of the videolaryngoscope give large, bright and higher resolution image as compared with the small monocular image obtained from fiberscopes.
- Because of the design of many videolaryngoscopes, the anterior angulation of the camera enables the operator to "look around the corner" which is not possible with conventional direct laryngoscopy.
- Videolaryngoscopy allows other healthcare providers to visualize the anatomy that the operator is viewing. Thus, the instructor can guide the trainee operator during procedure, making these devices extremely useful in teaching and they may replace 'over the shoulder' laryngoscopy teaching in the future.
- The assistant can help facilitate intubation by providing external laryngeal manipulation to provide optimal visualization of cords as he is himself able to see the field of vision on the monitor.
- It is possible to record the procedure for records and teaching.
- Movement of the cervical spine is less during videolaryngoscopy as compared to Macintosh laryngoscope, as there is no need to align the oral, pharyngeal, and tracheal axes. For the same reason, significantly less force is exerted on the maxillary incisors during videolaryngoscopy, minimizing potential for dental damage.

What are the limitations of videolaryngoscopes?

- Requires additional training to develop the psychomotor skills for hand-eye coordination necessary for the correct handling of the endotracheal tube while looking at the screen.
- Videolaryngoscopy often allows a good view of the glottis, even in patients who have CL III and IV grade views with conventional laryngoscopes. However, despite good view of the laryngeal inlet with videolaryngoscope, endotracheal intubation may prove to be difficult. This is because though view is obtained "around the corner" intubation still needs some alignment of axes, as the endotracheal tube keeps going posteriorly unless pre-shaped stylets are used.

What is the advantage of videolaryngoscopes over fiberoptic bronchoscope?

In fiberoptic intubation, when the tip of the bronchoscope is in the trachea, railroading of the endotracheal tube over the fiberscope remains a "blind technique" and may result in injury to laryngeal structures where tube impingement commonly occurs. With videolaryngoscopes the larynx remains in view throughout the intubation process.

What are the limitations of videolaryngoscopes when compared with fiberoptic bronchoscope?

- Videolaryngoscopes cannot be introduced through nose or tracheostomy
- Some space is required for introducing videolaryngoscopes, so these devices cannot be used in patients with complete trismus or wired jaw.
- Videolaryngoscopes are not helpful in confirming the placement of a double lumen tube, bronchial blocker or performing pulmonary toilet.

What are the complications of videolaryngoscopy?

- "Time to intubation" is significantly longer with videolaryngoscopes. Prolonged apnea time, rather than laryngeal manipulation with the laryngoscope, increases the blood pressure and heart rate and may be more stressful to the patient.
- In addition, prolonged apnea with a videolaryngoscope may cause hypoxia in patients with reduced oxygen store, such as obese patients, obstetric women, etc. These patients have minimal tolerance to apnea as they desaturate faster due to reduced functional residual capacity.
- The difficult part is to direct the tube toward the glottis and between the vocal cords, which are not in the line of sight. Difficulty during insertion of the tracheal tube may

result in injuries to the soft palate, oropharynx, and tonsils; even penetration of the palatal arch has been reported.
- In inexperienced hands, in particular, there is possibility of endotracheal tube impingement at the vocal cords or luminal surface of the anterior tracheal wall. Persistent threading of the tube results in the tube bending further instead of passing down the trachea. This can often be overcome by rotating the tube at 180° after withdrawing the stylet.

What is the role of videolaryngoscopes in emergency settings?

A recent meta-analysis found that, compared with the direct laryngoscopy, videolaryngoscopy failed to achieve better glottic views, higher rates of successful intubation, or shorter time to intubate. The possible explanations could be:

- In an emergency situation, patients may not be well prepared for intubation as in case of an elective intubation in operating room. The patient may not be positioned properly and there may be bleeding or copious secretions particularly; because of previous attempts of laryngoscopy which may obscure the lens of the videolaryngoscope and obstruct the glottic view.
- Insufficient training with the use of videolaryngoscope.

Do videolaryngoscopes have any role in pediatric difficult airway?

An unanticipated difficult airway is extremely rare in children. Most of the times, it is associated with congenital syndromes like Pierre Robin, Treacher Collins, Arnold Chiari, microcephaly, etc. The history and appearance of the child almost always alerts the anesthetist to anticipate a difficult airway. Most of the videolaryngoscopes are also useful in older children, only four are available in sizes that may be used in children younger than 2 years of age.

- Airtraq disposable optical laryngoscope
- GlideScope videolaryngoscope
- Storz DCI videolaryngoscope
- Truview PCD infant.

The Airtraq is available in two pediatric sizes. 'Infant' (size 0) accommodates tube size 2.5–3.5 and 'Pediatric' (size 1) accommodates tube size 3.5–5.5. Both laryngoscopes require a mouth opening of 12–13 mm. Combining the Airtraq with a stylet is not recommended as it increases the risk of airway injuries in children.

The pediatric GlideScope Cobalt has a reusable video baton (handle) and single-use laryngoscopy blades in two sizes. Owing to the 70° angulation of the blade, it can be used only for indirect laryngoscopy.

The Storz DCI Videolaryngoscope (SVL) is available with two slim Miller-like straight laryngoscope blades, size 0 and 1. It can be used in small infants with very limited mouth openings because of the small height of the blade (only 5 mm) and the distally located lens. In a series of 42 neonates weighing as little as 500 g, all were successfully intubated using Storz DCI videolaryngoscope.

The Truview PCD infant is small handy laryngoscope; with an eyepiece and optics; which provides a wide and magnified laryngeal view at a 46° anterior refracted angle. The height of the laryngoscope blade is only 8 mm and therefore can be used even in neonates. The side port allows insufflation of oxygen which significantly prolongs apnea time before desaturation occurs in neonates.

Describe various types of GlideScope.

In 2001, Canadian surgeon John A Pacey introduced the GlideScope videolaryngoscope.

Three types of GlideScope videolaryngoscopes are available: GlideScope original, GlideScope Cobalt and GlideScope Ranger.

The original GlideScope is reusable. It has non-detachable curved blade, a handle and 7" LCD monitor and a camera. (Fig. 55.2A) The laryngoscope blade is made up of high resistance medical grade plastic and the distal third of the blade angulates upwards, at approximately 60°. The maximum width of the blade at any place does not exceed 18 mm.

The Glidescope is introduced from the middle rather than the angle of the mouth, in the oropharynx till epiglottis is visualized. The use of a preshaped stylet; with curvature matching the shape of the blade; is recommended to facilitate intubation. Table 55.1 displays comparative features of the three types of GlideScopes.

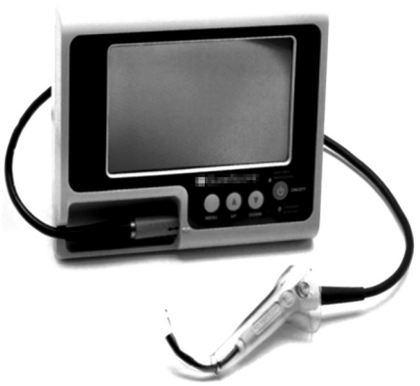


Fig. 55.2A GlideScope

Table 55.1 The features of three types of GlideScope

Glide-Scope	Blade shape, size	Monitor	Portability	Disposability	Antifog mechanism
Glide-scope original	Angulated, sizes 2 to 5	Separate, 7" LCD monitor	-	Reusable	Yes
Glide-Scope Cobalt (GVL Stat)	Angulated, sizes 1 to 4	Separate, 7 " LCD monitor	-	Yes (Has a plastic blade which slides over the flexible video baton)	Yes
Glide-Scope ranger	Angulated reusable: Sizes 3 and 4 Single-use sizes: 1 to 4	Separate, 3.5 LCD monitor	Yes, used in military, pre-hospital settings	Reusable or disposable	Yes

Disinfection and sterilization

Autoclaving, ultrasonic cleaning or pasteurization is not recommended. The GlideScope GVL is a non-sterile, reusable device. The GlideScope cobalt video baton is a non-sterile, reusable device. When used as intended, it is protected from direct contact with the patient by the sterile, single-use GVL Stat (Fig. 55.2B). Low-level disinfection (70% Isopropyl alcohol with minimum 1 minute exposure or Sodium Hypochlorite 500 ppm with minimum 1 minute exposure) is recommended for the video baton after every patient use. Clean the exterior of the monitor and the video cables, wipe with 70% Isopropyl alcohol or bleach, i.e. sodium hypochlorite (100 ppm) or a mild detergent and water.

Describe McGrath laryngoscope.

The McGrath series 5 video laryngoscope is a anatomically shaped, disposable videolaryngoscope. It has a video camera embedded into a "camera stick" completely, in such a way that no part of the handle or the camera comes in contact with the patient's mouth.

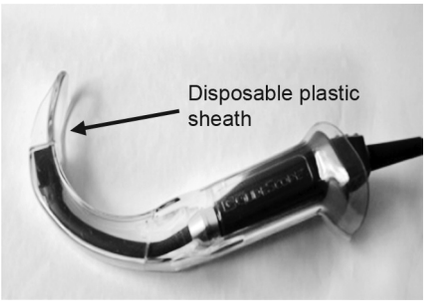


Fig. 55.2B GlideScope STAT

The single use transparent plastic blade (available in three adult lengths) is 13 mm wide and covers the steel bodied camera stick .The length of the camera stick can be easily increased to a maximum of 34 mm; using a simple ratchet mechanism for different-size patients (Fig. 55.3). The resulting video image is displayed on a small colored Liquid crystal display (1.7") mounted on top of the laryngoscope. The handle with the camera stick can be disassembled for sterilization. The device is powered with a single 1.5 v AA battery (life of 60 min of non-continuous use) and features a single electronic control: an on/off switch which is located at the top of the unit.

Method of insertion: Introduce the laryngoscope in the midline or slightly to the left (not angle of the mouth). As with other videolaryngoscopes like the GlideScope, a stylet must be used with the endotracheal tube, to allow it to be manipulated through the vocal cords.

Advantage: The device is light weight (350 g), therefore it is easily portable.

Disadvantage: McGrath video laryngoscope has no video output jack so the image cannot be seen on a remote screen. Also it does not have integrated antifogging mechanism.

Describe Storz C-Mac videolaryngoscope in brief.

In 2003, Kaplan and Berci introduced the Storz C-Mac videolaryngoscope. The C-Mac laryngoscope has a standard Macintosh reusable blade (Sizes 2–4) (Fig. 55.4A) with an integrated video camera. In addition, angulated blade is available only in size 4 (Fig. 55.4B) which is called C-Mac "D" Blade. The camera is housed within the laryngoscope handle and the magnified image is displayed on a screen. In the C-Mac laryngoscope, a fiberoptic bundle is coupled to the internal video camera, directed at approximately 25° from the line-of-sight. The newer C-Mac DCI laryngoscope is a part of the direct coupler interface (DCI) video-intubation system, in

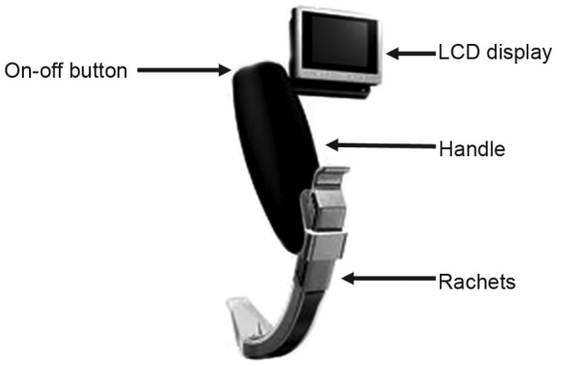


Fig. 55.3 McGrath videolaryngoscope

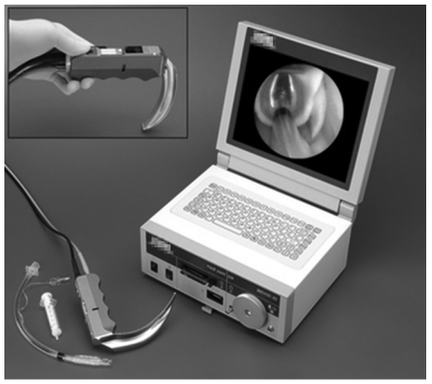


Fig. 55.4A C-Mac videolaryngoscope Macintosh blade

Fig. 55.4B C-Mac videolaryngoscope D blade

which the blades are interchangeable with flexible fiberoptic and straight Miller blades. C-Mac consists of only two parts, a laryngoscope and a monitor, connected via a single cable. Therefore, it is portable, more robust and less expensive as compared with the older V-Mac.

Disinfection and sterilization

Encrustations are removed using an active cleaner and a disinfected using a soft brush. The C-MAC blades and electronic modules are validated for low-temperature preparation methods of sterilization up to a maximum temperature of 60°C. It can be cleaned manually or using a machine and sterilized with Sterrad (low-temperature, hydrogen peroxide gas plasma technology to sterilize instruments and medical devices).At the end of the process, the optical surfaces of the image chip and LED illuminator are rewashed with a cotton tip applicator soaked in 70% isopropyl alcohol. Ultrasound bath and steam sterilization is not recommended.

Describe Bullard rigid fiberoptic laryngoscope.

The Bullard laryngoscope is an S shaped rigid fiberoptic laryngoscope with anatomically curved blade. A sheath is provided for introduction of fiberoptic bundles for illumination and visualization on the posterior aspect of the handle. The handle may contain batteries and a halogen bulb and/or have an adaptor for a flexible cable that is connected to a high-intensity halogen or xenon light source. Light from the source is transmitted to the distal blade through the fiberoptic bundle. It is, therefore, a rigid and fiberoptic laryngoscope together in the same apparatus. A viewing arm with an eyepiece is located at an angle of 45 degrees from the handle (Fig. 55.5).

A working channel for suction and /or insufflation of oxygen extends from the body of the scope to the tip where the light bundles end. The proximal end of the handle has two openings. The first opening is of Luer-taper type of connection. This can be used to connect a Luer-lock connection for equipment such as syringes for medication, local anesthetic or saline. It can also be used for suction or insufflation of oxygen during the procedure. The second opening has an attachment for the introducing stylet. The introducing or intubating stylet or multifunctional stylet attaches near the base of the viewing arm with a spring-loaded, locking mechanism. It is thin, with a curve to the left at 20° near the tip. This brings the end of the stylet into the field of vision and facilitates passage of the endotracheal tube into the laryngeal inlet.

The multifunctional stylet is a long, hollow tube fixed to and parallel to the laryngoscope body. Flexible fiberscope, tracheal tube exchanger, or a small catheter can be introduced through it. It is more difficult to maneuver than

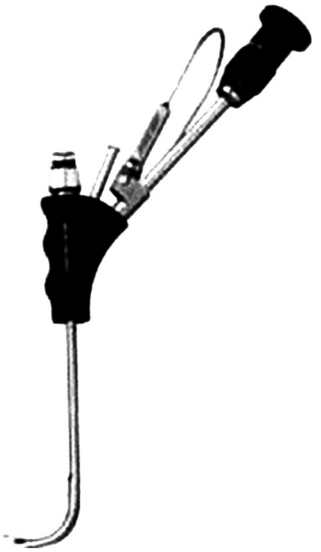


Fig. 55.5 Bullard laryngoscope

the introducing stylet. The adult tracheal tube is threaded over the lubricated stylet so that the tip of the stylet protrudes through the Murphy eye.

The Bullard laryngoscope is available in three sizes: pediatric, pediatric long, and adult. The adult has a deeper curve than pediatric blades. Also, a plastic tip extender is available to lengthen the tip of the adult blade. This is useful for intubating male patients but is usually not required for female patients. For infants and children and patients with height less than 5′ a pediatric, pediatric long size laryngoscope blade is used respectively. In children more than 8–10 years of age and patients more than 5′ but less than 6′ the adult size blade is used. In patients more than 6′, adult blade with tip extender is used (Usually required in male patients).

Advantages: It can be used in patients with a mouth opening as small as 6 mm. Jet ventilation or oxygen insufflation can be given through the working channel. The channel can also be used for suction. Administration of oxygen helps to clear secretions, to prevent fogging and desaturation. It can be converted into a videolaryngoscope.

Method of insertion

For oral intubation, endotracheal tube is mounted on the stylet. The blade is inserted in the midline of the oral cavity, with the handle horizontal. As the blade is advanced, the handle is rotated to the vertical position so that the blade slides over the tongue. The blade is then elevated against the dorsal surface of the tongue with minimal upward movement exerted along the axis of the laryngoscope. This upward movement will result in the blade of the Bullard laryngoscope lifting the epiglottis, providing complete visualization of the glottis opening. The endotracheal tube is advanced over the stylet, and under direct vision it is pushed into the trachea. The stylet is detached and the scope is gently removed after ascertaining that the endotracheal tube is in place. In some cases, the tip of the tracheal tube will impact on the right arytenoid, in such situations the tube may be rotated 180° on the stylet.

Discuss the Airtraq optical laryngoscope.

Airtraq was developed and patented by Dr Acha. It is a disposable, optical laryngoscope with two parallel conduits, one optical and the other a tube guiding channel. A low temperature battery operated light is present at the tip of the blade which acts as an antifogging. The battery life is approximately one hour. It is available in adult and pediatric sizes, and a size to accommodate DLT (Fig. 55.6).

To use the Airtraq, the endotracheal tube is preloaded in a channel next to the optical pathway, and the device is inserted in oropharynx in midline. When the glottis is visualized, the ETT is advanced into the trachea. The Airtraq is then removed, disengaging the ETT with a lateral movement. If the vocal cords are not visible, it usually means that either the laryngoscope is not in the midline or placed too posteriorly in the vallecula. In case laryngeal structures are not recognized, withdraw the Airtraq slightly.

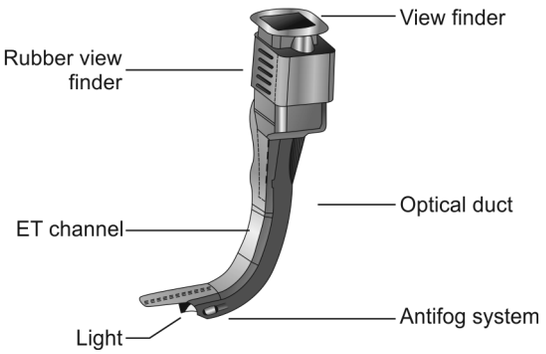


Fig. 55.6 Airtraq optical laryngoscope

Advantages: It has an anti-fog system built into the optical channel which requires 30–45 s warm-up time prior to use. It can be converted into videolaryngoscope by attaching optional wireless or wired video system; which should be mounted on top of the Airtraq; after removing the original rubber viewfinder.

Disadvantage: The view through the Airtraq is less panoramic when compared with Macintosh and other videolaryngoscopes. The oral injuries may be unnoticed if they are outside the optical field of Airtraq. Table 55.2 describes various sizes and types of blades available with Airtraq.

Table 55.2 Sizes and types of blades of Airtraq

Description	ET tube sizes	Mouth opening required for insertion	Use
Regular (Size 3-blue)	7.0–8.5	18 mm	Oral intubation
Small (Size 2-green)	6.0–7.5	16 mm	Oral intubation
Pediatric (Size 1-purple)	3.5–5.5 (Cuffed or Uncuffed)	12.5 mm	Oral intubation
Infant (Size 0-gray)	2.5–3.5	12.5 mm	Oral intubation
Infant nasal (white)	Not applicable	12.5 mm	Nasal intubation
Adult nasal tracheal intubations (orange)	Not applicable	18 mm	Nasal intubation
Double Lumen Endobronchial tubes	35–41 Fr. Standard	19 mm	Double lumen endobronchial tubes

Describe PENTAX airwayscope in brief.

The PENTAX airwayscope has anatomically shaped polycarbonate blade (PBlade) with a guide channel and 2.4 "LCD monitor built into the top of the handle. The handle houses a built-in charge-coupled device camera and a light-emitting diode attached to the tip of the scope and a disposable blade powered by two AA batteries lasting approximately an hour. The single use PBlade completely encloses and protects the image tube and camera from oral contamination. The PBlade has two side grooves where the tracheal tube (external diameter between 8.5 and 11.0 mm) and suction catheter (4.0 mm diameter or less) are preloaded (Fig. 55.7).

The endotracheal tube is attached to the blade and the tip of the tube is captured on the display screen even before insertion of the device. Therefore, the location of the tube tip can continuously be monitored during intubation. If the advancement of the endotracheal tube is difficult, a tube exchanger or bougie can be passed through the loaded endotracheal tube into the trachea and then the tracheal tube from the PBlade can be detached and ETT is railroaded over the bougie.

Advantages

PENTAX airwayscope is portable and water resistant. The display screen can be fully rotated to facilitating intubation from a position where the operator is facing the patient. A target mark on the monitor is designed to give direction for advancing the endotracheal tube and is intended to facilitate intubation. The PBlade has a separate channel for suction catheter.

Disadvantage: Bulky and the length may make its use difficult in patients with large breasts or barrel chest.

Describe in brief the Truview laryngoscope.

The Truview is a reusable video and optical laryngoscope with a straight blade. It incorporates a prism and lens assembly. It consists of a combination of optical system with specially profiled 12.8 mm slim steel blade, optical apparatus which provides 42° angled deflection view through 15 mm eye piece (Fig. 55.8).

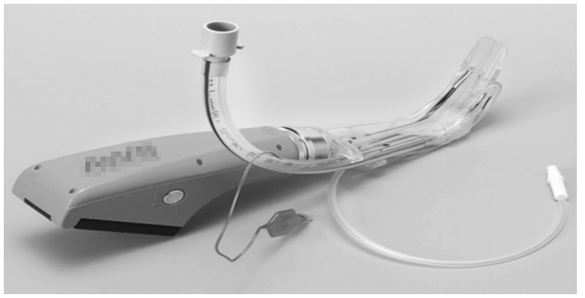


Fig. 55.7 PENTAX airwayscope

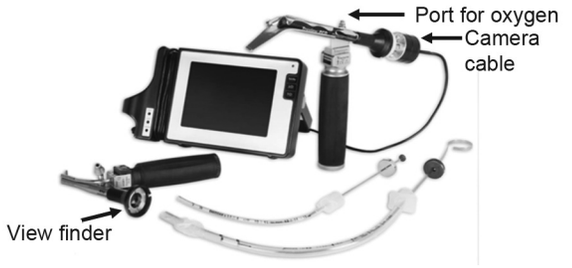


Fig. 55.8 Truview

Method of insertion: The endotracheal tube is loaded on the preshaped stylet supplied with the laryngoscope. The laryngoscope is inserted through middle of the mouth and advanced partly into the oropharynx. Then start looking through the viewing lens (or the monitor) and advance while lifting the mandible gently until the glottis is visualized. The trachea is then intubated with the endotracheal tube over the stylet and the stylet is withdrawn.

Advantages: The Truview has an oxygen port on the blade for oxygen insufflation and thus it slows the desaturation of anesthetized patient, also prevents the accumulation of mist and secretions on the lenses and serves as antifogging mechanism. Recommended oxygen flows to prevent fogging are up to 5 L/min for blade sizes 0 and 1 and up to 10 L/min for blade sizes 2, 3 and 4. It is lightweight and portable device (Table 55.3).

The Truview laryngoscope blades are available in a 5 sizes. (Neonate to adult)

It can be converted to a videolaryngoscope by connecting a magnetic camera to the blade and the image is displayed on 5" LCD monitor.

Table 55.3 Patients' age and weight and selection of Truview laryngoscope blade

Blade name	Blade mark	Patient's age (years)	Weight range (Kg)
Neonatal	0	< 1	0.8–4
Infant	1	1–3	4–8
Child	2	3–16	8–60
Adult	3	> 16	50–120
Large adult	4	> 16	> 120

Cleaning, sterilization and disinfection

In Trueview size 2, 3 and 4 some components may be disassembled. Truview blade can be cleaned in its assembled form. The removable components of the blade (the replaceable FiberClip—fiberoptic light guide and the optical view tube) may be disassembled for easier handling during cleaning.

Immediately after use, the Truview laryngoscope blade should be immersed in water. External surfaces should be gently scrubbed in soapy water with soft brush to remove encrusted deposits. Rinse the blades in demineralized water and dry thoroughly before disinfection and/or storage. Oxygen port can be cleared and/or cleaned using an air or gas jet.

High level disinfectants such as, Cidex Plus (2% Glutaraldehyde for minimum 20 minutes exposure or) and Cidex OPA (0.55% w/w orthophthaline aldehyde 12 minute exposure) may be used. Chemical detergents containing caustic or alkaline ingredients such as povidone-iodine solutions, peroxide solutions, sodium hypochlorite are not recommended.

Do not steam autoclave the Truview blade 0 and 1. If Truview PCD™ blade sizes 2, 3 or 4 are to be sterilized in a steam autoclave, remove the optical view tube and set aside to be sterilized separately as described above. The remaining parts may be placed in a steam autoclave. Ultrasonic cleaning and "flash" autoclaving are not recommended.

Describe in brief the bonfils retromolar intubating fiberscope.

The bonfils fiberscope was developed 20 years ago for use by ENT surgeons. The device was introduced in clinical management of patients with difficult airway in mid 1990s. The Bonfils (Fig. 55.9A) is a 40 cm long, semi-rigid optical stylet with an external diameter of 5.0 mm and a fixed anterior tip curvature of 40°. Its fiberoptic bundle is encased in a stainless steel tube that provides 15,000 pixel resolution and is available with and without a 1.2 mm working channel. The adult stylet can accommodate 6.5 mm or larger endotracheal tubes and the pediatric stylet can accommodate 2.5–6.0 mm endotracheal tubes. Image of the glottis can be seen through the eyepiece, allowing direct visualization during intubation. The other version has a "Direct Coupled Interface" that displays the image directly on a video monitor. The eyepiece can also be converted to project the image onto a remote monitor to provide better visualization and enhance teaching. There is an adaptor "slide cone" for fixation of the endotracheal tube. This adaptor has a side port that allows oxygen insufflation.

Method of insertion: The endotracheal tube is loaded over the Bonfils optical stylet and fixed in such a way (keeping the tip of the stylet at the endotracheal tube bevel) that a crescent of illumination is seen at the right side of the screen (Fig. 55.9B). Bonfils fiberscope is held like a pen and introduced through right angle of the mouth. The Bonfils fiberscope is advanced through the retromolar space and then turned cephalad to visualize the glottis. Preloaded ET tube is then advanced into the trachea and the Bonfils fiberscope is withdrawn by rotating it toward foot-end of the patient.

Advantages

It is especially useful when patient has expensive/delicate dental work, inconveniently placed solitary tooth, loose teeth, large gaps between upper teeth which can trap laryngoscope blade and impede its movement. The external diameter of the adult Bonfils scope allows the use of endotracheal tubes as small as the 5 mm microlaryngeal tube, double-lumen tubes.

Disadvantages

Learning curve for its use is very high. It cannot be used for nasal intubations. The rigidity of the scope may increase the risk of airway trauma in inexperienced hands. The non-malleable shaft may limit the ability to angle the scope in cases where the larynx is extremely anterior.

Fig. 55.9A Bonfils retromolar intubating fibrescope

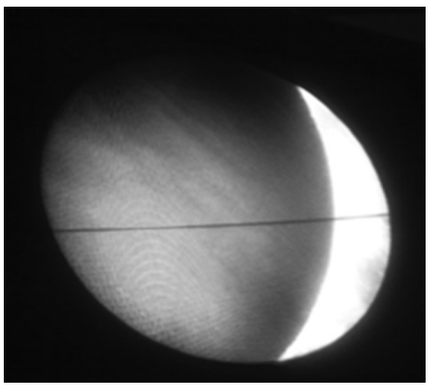


Fig. 55.9B Crescent

Suggested Reading

1. Ahmed-Nusrath A. Videolaryngoscopy. Curr Anae Critical Care. 2010;21:199–205.
2. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Anesthesiology. 2003;98: 1269–77.
3. Asai T. Videolaryngoscopes: Do they truly have roles in difficult airways? Anesthesiology. 2012;116:515–7.
4. Holm-Knudsen R. The difficult pediatric airway–a review of new devices for indirect laryngoscopy in children younger than two years of age Paediatr Anaesth. 2011;21:98–103.
5. Lee YK, Chen CC, Wang TL, Lin KJ, Su YC. Comparison of video and direct laryngoscope for tracheal intubation in emergency settings: A meta-analysis. Acute Med. 2012;2:43–9.
6. Niforopoulou P, Pantazopoulos I, Demestiha T, Koudouna E, Xanthos T. Video-laryngoscopes in the adult airway management: a topical review of the literature. Acta Anaesthesiol Scand. 2010;54:1050–61.
7. The bonfils retromolar intubation fibrescope: Advantages and practical aspects of its use. AJA- Online.com 2011;12:40–8.

Plate i

Fig. 1 Red rubber endotracheal tube

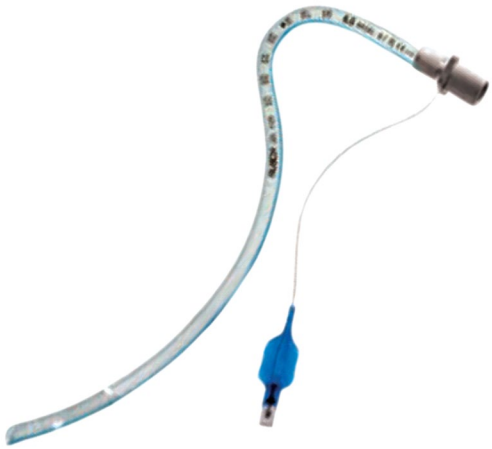


Fig. 2 North pole tube

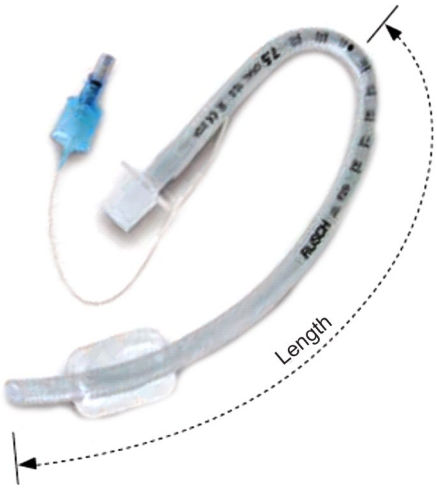


Fig. 3 South pole tube

Fig. 4 Armored endotracheal tube

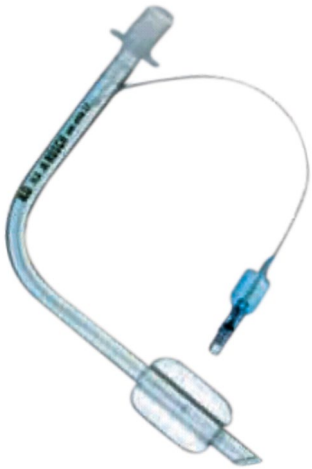


Fig. 5 Oxford tube

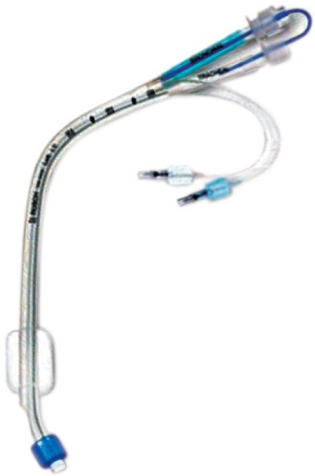


Fig. 6 Double lumen tube

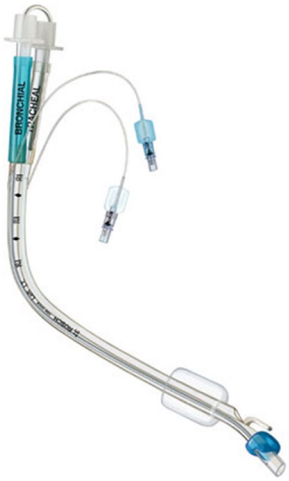


Fig. 7 White double lumen tube

Fig. 8 Carlens tube

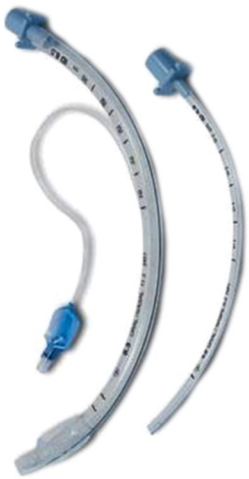


Fig. 9 Portex adult and pediatric endotracheal tube

Fig. 10 Cole tube

Plate iii

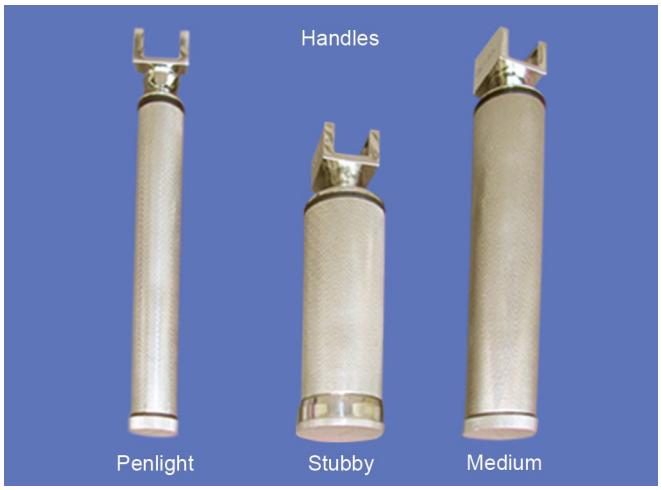


Fig. 11 Handles

Fig. 12 Macintosh blades

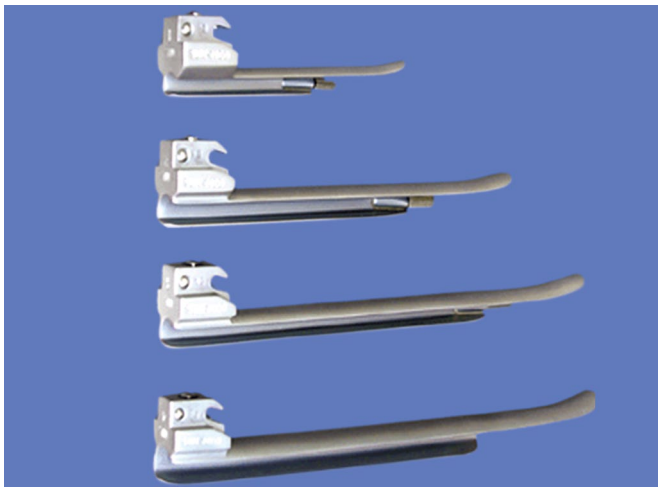


Fig. 13 Miller blades

Fig. 14 Polio blade

Fig. 15 McCoy laryngoscope

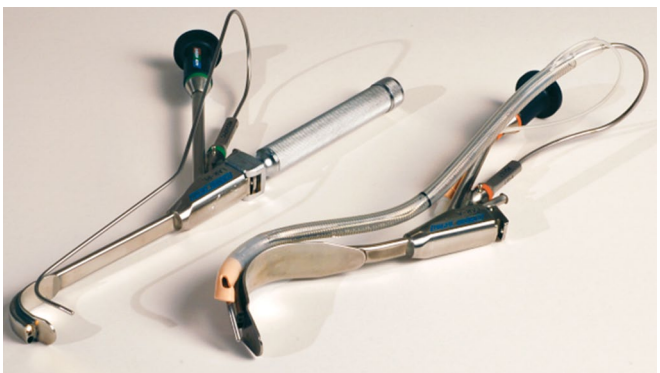


Fig. 16 Bullard laryngoscope

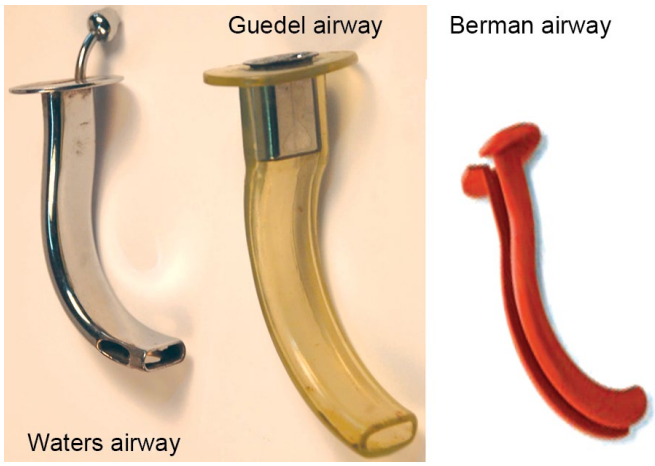


Fig. 17 Waters airway, Guedal airway and Berman airway

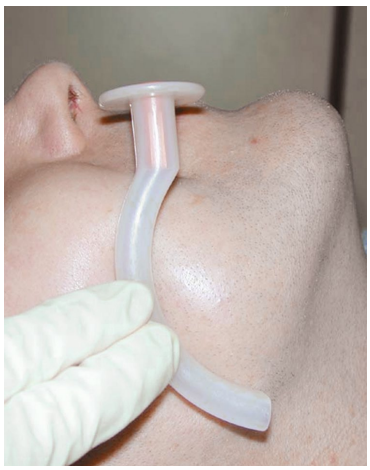


Fig. 18 Selection of appropriate oropharyngeal airway

Fig. 19 Binasal airway

Fig. 20A Portex nasopharyngeal airway

Fig. 20B Red rubber nasopharyngeal tube

Plate v

Fig. 21 Classic LMA

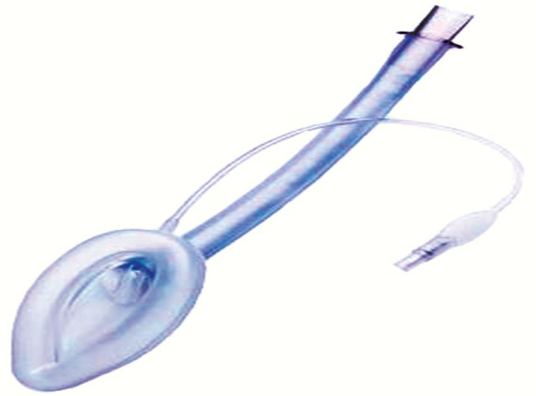


Fig. 22 LMA unique (Disposable classic version)

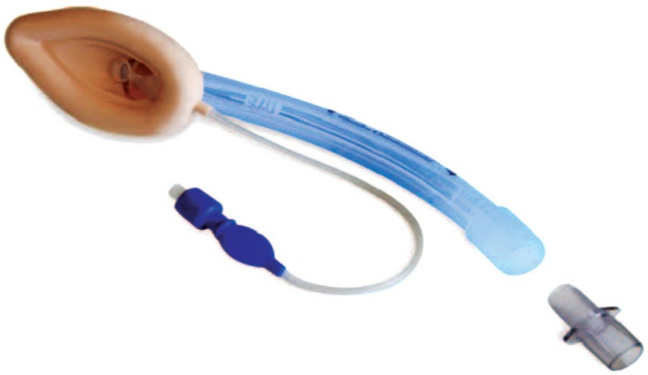


Fig. 23 LMA classic excel

Fig. 24 Flexible LMA

Fig. 25 Single use flexible LMA

Fig. 26 ProSeal LMA

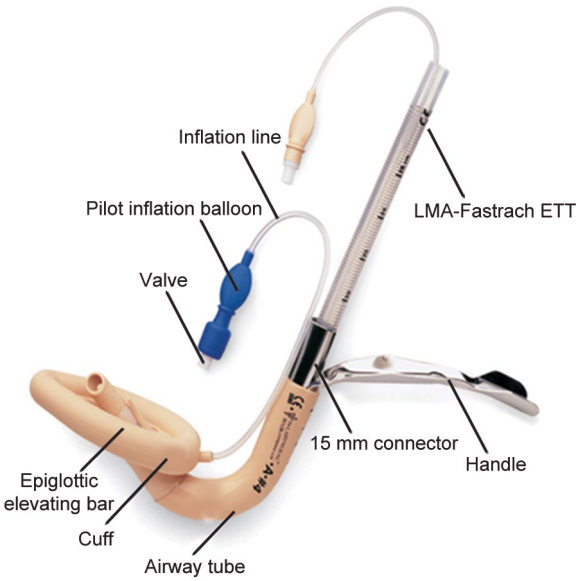


Fig. 27 LMA Fastrach

Fig. 28 Single use LMA fastrach

Fig. 29 LMA supreme

Fig. 30 Ambu LMA

Fig. 31 Laryngeal tube

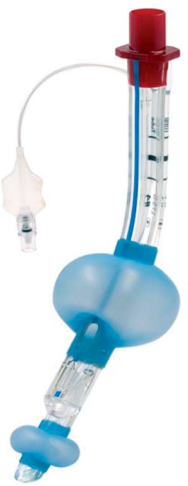


Fig. 32 Laryngeal tube S

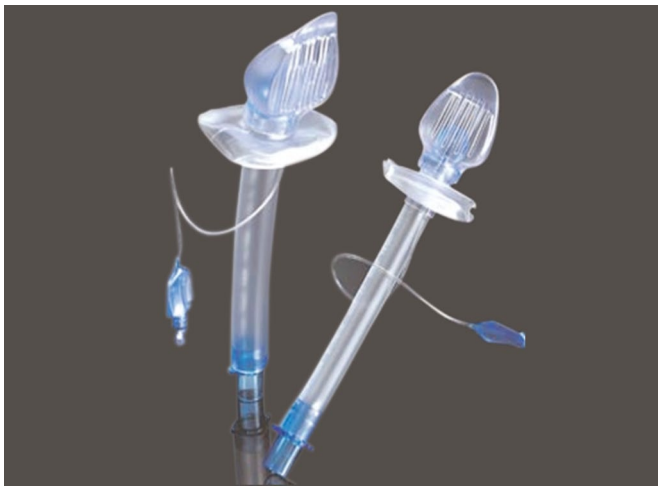


Fig. 33 Cobra PLA

Fig. 34 SLIPA

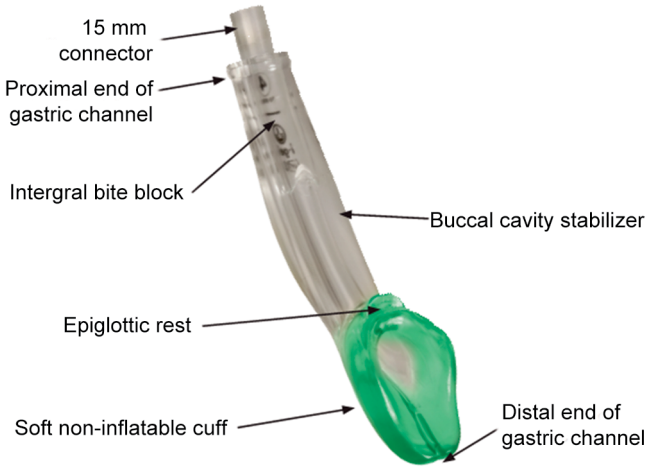


Fig. 35 I-gel

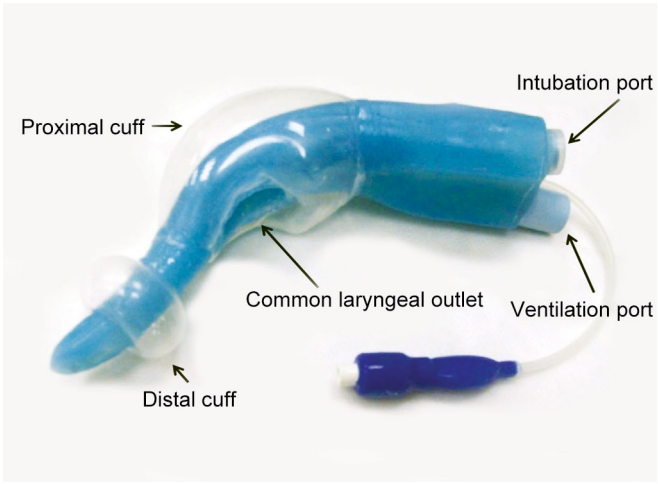


Fig. 36 Elisha airway device

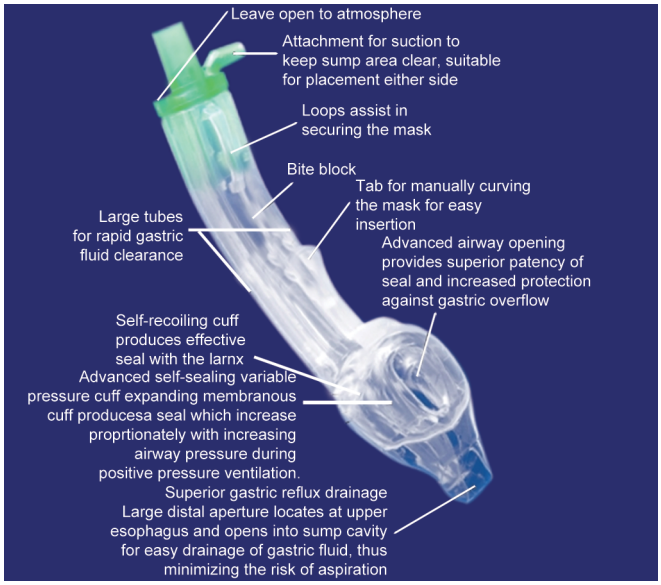


Fig. 37 The Baska Mask®

Fig. 38 Flagg's can

Plate viii

Fig. 39 Schimmelbusch mask

Fig. 40 Yankauer's mask

Fig. 41 Ogston's inhaler

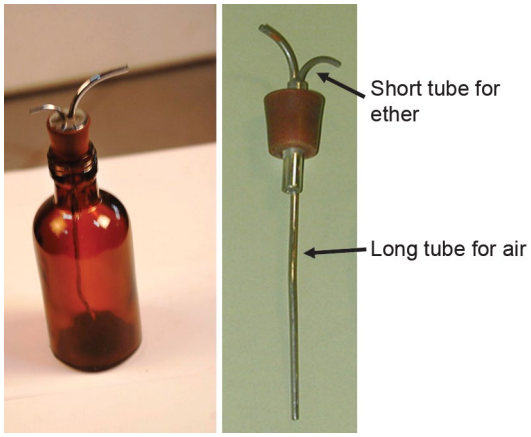


Fig. 42 Bellamy Gardner ether dropper

Fig. 43 Boyle's bottles for ether and trilene

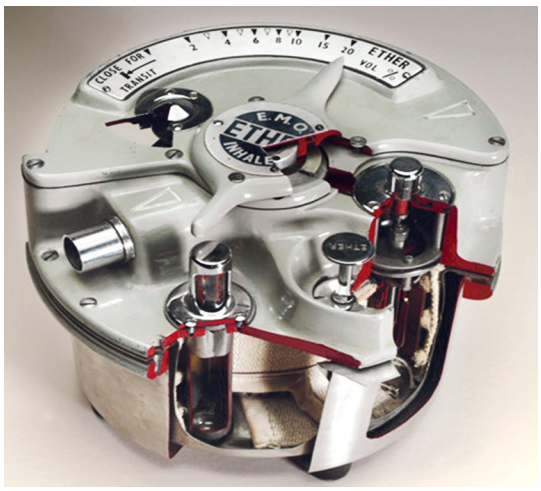


Fig. 44 EMO

Fig. 45 Oxford miniature vaporizer

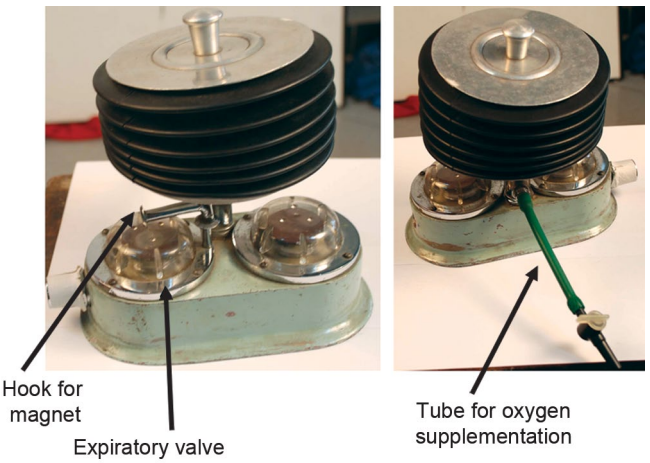


Fig. 46 Oxford Inflating Bellows

Fig. 47 Goldman's vaporizer

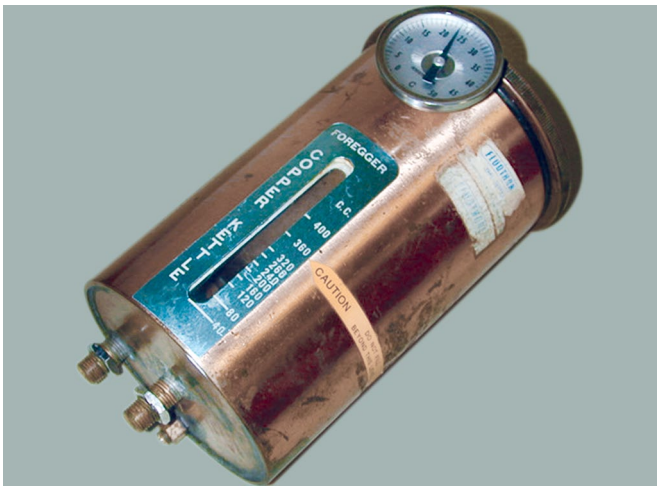


Fig. 48 Copper Kettle

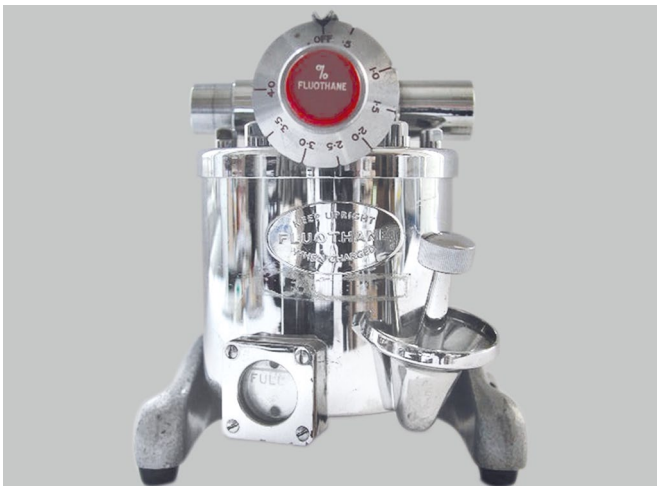


Fig. 49 TEC 2 vaporizer

Fig. 50 TEC 3 vaporizer

Fig. 51 TEC 6 vaporizer

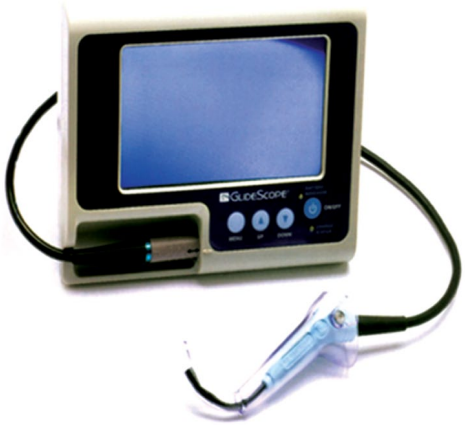


Fig. 52A GlideScope

Fig. 52B GlideScope STAT

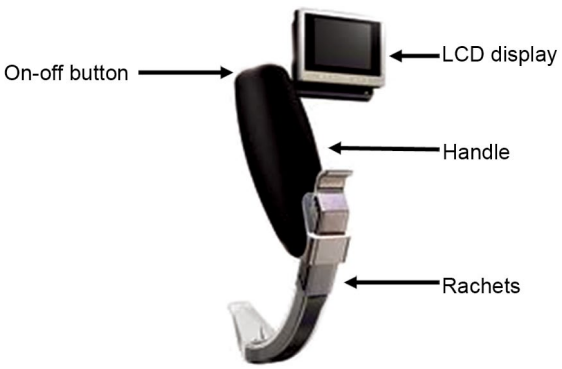


Fig. 53 McGrath videolaryngoscope

Fig. 54A C-Mac videolaryngoscope Macintosh blade

Fig. 54B C-Mac videolaryngoscope D blade

Fig. 55 Bullard laryngoscope

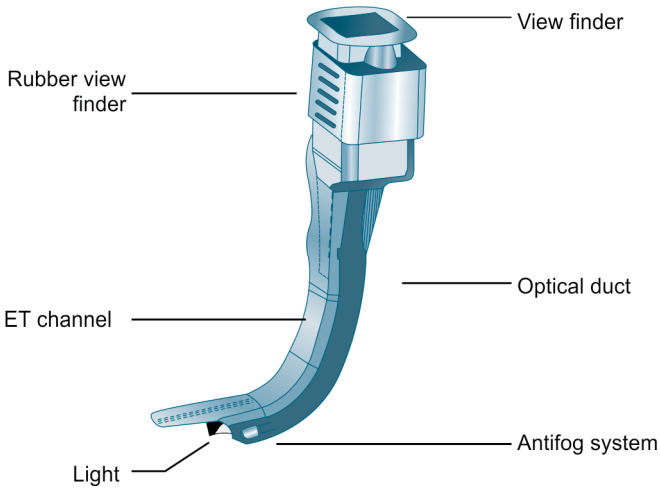


Fig. 56 Airtraq optical laryngoscope

Fig. 57 Pentax airwayscope

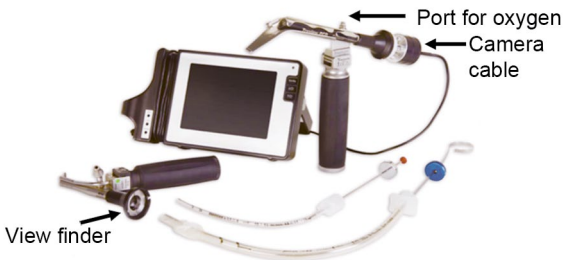


Fig. 58 Trueview

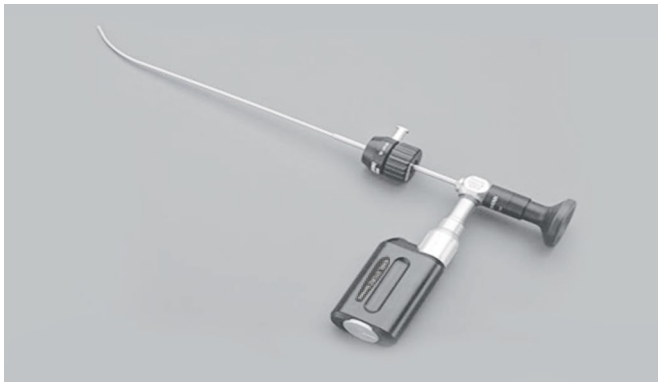


Fig. 59A Bonfils retromolar intubating fiberscope

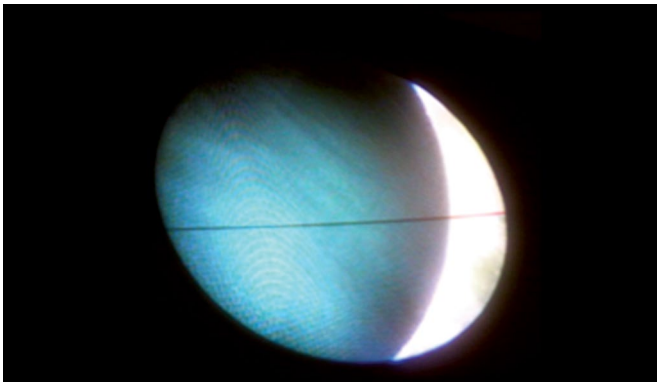
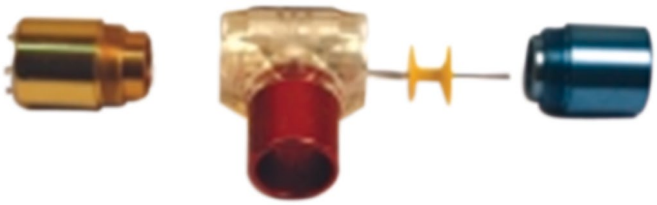


Fig. 59B Crescent

Ruben valve Ambu valve

Fig. 60 Nonrebreathing valves



Ruben valve

Fig. 61 Nonrebreathing valves

Laerdal's valve

Fig. 62 Nonrebreathing valves

Fig. 63 Pressure reducing valves

Plate xiii

Fig. 64 Pressure reducing valves

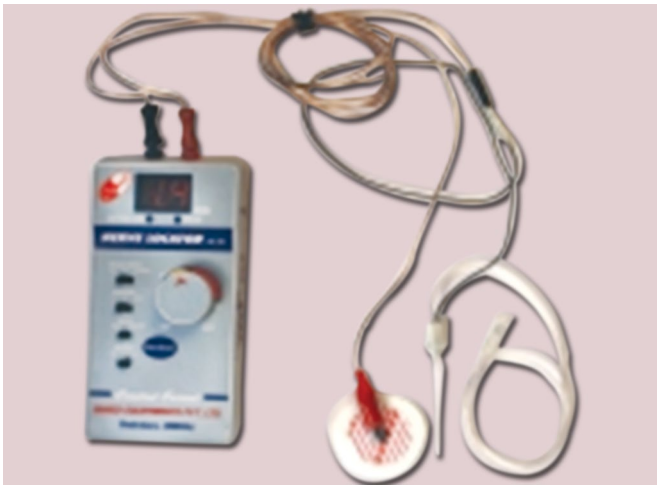


Fig. 65 Nerve locator

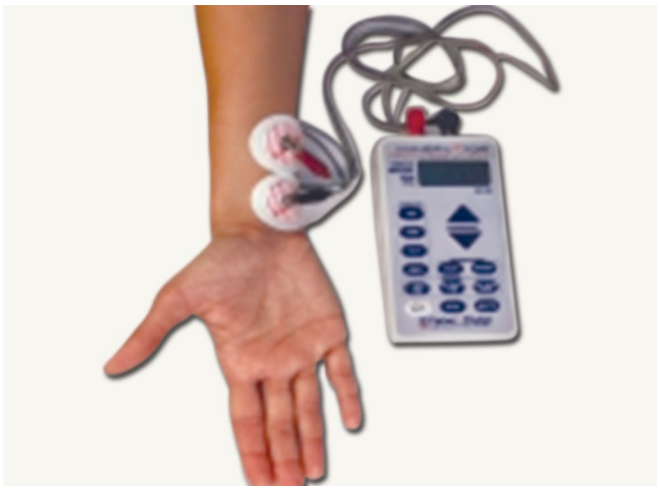


Fig. 66 Peripheral nerve stimulator

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