Chest X-Ray Interpretation

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Introduction

In general, but not always, PA radiographs are taken with the patient upright (standing or at least sitting erect with legs dangling). The X-ray tube is positioned 6ft behind the patient. (The farther the tube is from the object, the more the source resembles a point source and that reduces penumbra (fuzziness of edges); the standard (in the US) is 72"). The heart, anterior in the chest, is closer to the detector and so has less geometric magnification.

AP radiographs are taken with the tube in front of the patient with the detector behind the patient. These are usually done with portable equipment and the patient is typically in bed. Portable equipment typically has less power than fixed equipment and so the photons may be less energetic. The patient is typically semi-erect so some geometric distortion may be introduced.

How do you differentiate between PA and AP views?

PA view is the most commonly taken view of chest….and AP view is taken only in certain conditions where patient is completely bed ridden and is not able to stand or sit in an erect posture….further to differentiate PA from AP you can look into quite a few structures …

Scapular shadow will be outside lung filed in PA view

Clavicles - more angulated in PA view, some portion of apical lung field will be seen above the clavicle

Direction of the ribs - Anterior ribs are more prominent in PA view; Posterior ribs in AP view. (To look for rib fractures, AP view may be more appropriate.)

Heart shadow is broader in AP view.

The differences mentioned so far apply to the best-case PA view versus best-case AP view. For example, scapular shadow may overlap lung field in PA view due to inappropriate positioning of upper limbs or due to abnormalities like scoliosis. Similarly, labeling as apparent cardiomegaly may not always hold true, if you do not have a benchmark.

Be it PA or AP view, you can comment as "**Chest X Ray frontal projection**", if there is no label printed in the X ray indicating PA or AP view.

Chest X-Ray Interpretation - a structured approach

YouTube

Confirm details

Always begin by checking the following:

Patient details (name / DOB)

Date and time the film was taken

Any previous imaging (useful for comparison)

Assess image quality

A mnemonic you may find useful is '**RIPE**':

Rotation

The medial aspect of each clavicle should be equidistant from the spinous processes.
The spinous processes should also be in vertically orientated against the vertebral bodies.
Inspiration.
5-6 anterior ribs, the lung apices, both costophrenic angles and lateral rib edges should be visible

Projection

AP vs PA film
Tip- if there is no label, then assume it's a PA. Also, if the scapulae are not projected within the chest, it's PA.

Exposure

Left hemidiaphragm visible to the spine and vertebrae visible behind heart

ABCDE approach to reading CXR

Airway

Trachea:

Is the trachea significantly deviated?
The trachea is normally located centrally or just slightly off to the right
If the trachea is deviated, look for anything that could be pushing or pulling at the trachea.
Also inspect for any paratracheal masses/lymphadenopathy
Pushing of trachea – e.g. large pleural effusion / tension pneumothorax
Pulling of trachea – e.g. consolidation with lobar collapse
Rotation of the patient can give the appearance of a deviated trachea, so as mentioned above, check the clavicles to rule out rotation as the cause.

Carina and Bronchi:

The carina is located at the point at which the trachea divides into the left and right main bronchus.
On a good quality CXR this division should be visible and is an important landmark when assessing nasogastric tube placement, as the NG tube should bisect the carina if it is correctly placed (i.e. not in the airway).
The right main bronchus is generally wider, shorter and more vertical than the left main bronchus. As a result it is more common for inhaled foreign objects to become lodged here (as the route is more direct).
Depending on the quality of the CXR you may be able to see the main bronchi branching into further subdivisions of bronchi which supply each of lobe.

Hilar structures

The hilar consist of the main pulmonary vasculature and the major bronchi.
Each hilar also has a collection of lymph nodes which aren't usually visible in healthy individuals.
The left hilum is often positioned slightly higher than the right, but there is a wide degree of variability between individuals.
The hilar are usually the same size, so asymmetry should raise suspicion of pathology

The hilar point is also a very important landmark; anatomically it is where the descending pulmonary artery intersects the superior pulmonary vein. When this is lost, think of a lesion here (e.g. lung tumour or enlarged lymph nodes).

Breathing

Lungs

Inspect the lungs:

When looking at a CXR we divide each of the lungs into 3 zones, each occupying 1/3 of the height of the lung.

These zones do not equate to lung lobes (e.g. the left lung has 3 zones but only 2 lobes).

Inspect each of the zones of the lung first ensuring that lung markings occupy the entire zone.

Compare each zone between lungs, paying close attention for any asymmetry (some asymmetry is normal and caused by the presence of various anatomical structures e.g. the heart).

Some lung pathology causes symmetrical changes in the lung fields, which can make it more difficult to recognise, so it's important to keep this in mind (e.g. pulmonary oedema).

Pleura

Inspect the pleura:

The pleura are not normally visible in healthy individuals, unless there is an abnormality such as pleural thickening.

Inspect the borders of each of the lungs to ensure lung markings extend all the way to the edges of the lung fields (if there appears to be an area lacking lung markings with decreased density this may suggest the presence of a pneumothorax).

Fluid (hydrothorax) or blood (haemothorax) can also accumulate in the pleural space, causing an area of increased opacity or a combination of both a pneumothorax and fluid (hydropneumothorax).

Cardiac

Assess heart size

In a healthy individual the heart should occupy no more than 50% of the thoracic width (e.g. a cardiothoracic ratio of <0.5).

This rule only applies to PA chest x-rays (as AP films exaggerate heart size), so you should not draw any conclusions about heart size from an AP film.

If the heart occupies more than 50% of the thoracic width (on a PA CXR) then this suggests abnormal enlargement (cardiomegaly). Cardiomegaly can occur for a wide variety of reasons including valvular disease, cardiomyopathy, pulmonary hypertension and pericardial effusion.

Assess heart borders

Inspect the borders of the heart which should be well defined in healthy individuals:

The right atrium makes up most of the right heart border.

The left ventricle makes up most of the left heart border.

The heart borders may become difficult to distinguish from the lung fields as a result of various pathological processes (e.g. consolidation) which cause increased opacity of the lung tissue.

Loss of definition of the right heart border is associated with right middle lobe consolidation

Loss of definition of the left heart border is associated with lingular consolidation

From the frontal projection, the cardiac silhouette can be divided into right and left borders:

Right heart border is formed by the right atrium

the superior vena cava entering superiorly and the inferior vena cava often seen at its lower margin

Left heart border is formed by the left ventricle and left atrial appendage the pulmonary artery, aortopulmonary window and aortic notch extend superiorly

On the lateral projection the cardiac silhouette is formed by:

Anterior border by right ventricle

Posterior border by left atrium (superiorly) and left ventricle (inferiorly) and the inferior vena cava.

Diaphragm

The right hemi-diaphragm is in most cases higher than the left in healthy individuals (as a result of the underlying liver). The stomach underlies the left hemi-diaphragm and is best identified by the gastric bubble located within it.

The diaphragm should be indistinguishable from the underlying liver in healthy individuals on an erect CXR, however if free gas is present (often as a result of bowel perforation), air accumulates under the diaphragm causing it to lift and become visibly separate from the liver. If you see free gas under the diaphragm you should seek urgent senior review, as further imaging (e.g. CT abdomen) will likely be required to identify the source of free gas.

There are some conditions which give the appearance of free gas under the diaphragm (pseudo-pneumoperitoneum), such as Chilaiditi syndrome which involves the colon becoming positioned between the liver and the diaphragm resulting in the appearance of free gas under the diaphragm (because the bowel wall and diaphragm become indistinguishable due to their proximity). As a result the imaging needs to be considered in the context of the patient's history and your findings on clinical examination. As a junior doctor however, you should always discuss a scan that appears to show free gas with a senior colleague immediately.

<u>Costophrenic angles</u>

The costophrenic angles are formed from the dome of each hemi-diaphragm and the lateral chest wall.

In a healthy individual the costo-phrenic angles should be clearly visible on a normal CXR as a well defined acute angle.

Loss of this acute angle (sometimes referred to as costophrenic blunting) can suggest the presence of fluid or consolidation in the area. Costophrenic blunting can also occur secondary to lung hyperinflation (seen in diseases such as COPD) as a result of diaphragmatic flattening and subsequent loss of the acute angle.

Everything else

<u>Mediastinal contours</u>

The mediastinum contains the heart, great vessels, lymphoid tissue and a number of potential spaces where pathology can occur. The exact boundaries of the

mediastinum aren't particularly visible on a CXR, however there are some important structures that you should assess.

Aortic knuckle:

Left lateral edge of the aorta as it arches back over the left main bronchus.

Loss of definition of the aortic knuckles contours can be caused by an aneurysm.

Aorto-pulmonary window:

The aorto-pulmonary window is a space located between the arch of the aorta and the pulmonary arteries.

This space can be lost as a result of mediastinal lymphadenopathy (e.g. malignancy).

Bones

Inspect the visible skeletal structures looking for any abnormalities (e.g. fractures / lytic lesions).

Soft tissues

Inspect the soft tissues for any obvious abnormalities (e.g. large haematoma).

Tubes / Valves / Pacemakers

Tubes – nasogastric tubes are something you'll often be asked to assess on a chest x-ray to confirm it is safe for feeding

Lines (e.g. central line / ECG cables).

Artificial valves (e.g. aortic valve replacement).

Pacemaker (often located below the left clavicle).

Review areas

Lastly, before completing your assessment, always ensure you've looked at the 'Review areas' which are:

Lung apices

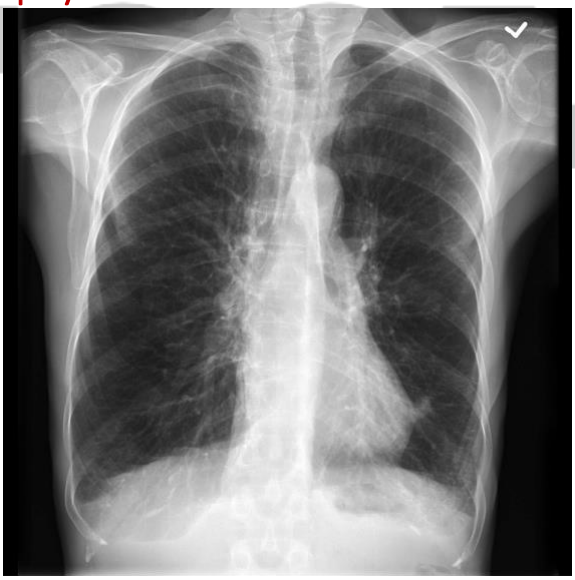
Retrocardiac

Behind the diaphragm

Peripheral lungs

Hilar

Case 1: Chronic emphysema



Chest x-ray demonstrates very marked hyperinflation of both lungs. Over 11 posterior ribs are seen, the diaphragms are flattened and there is enlargement of the retrosternal airspace. Pulmonary vasculature not terribly distorted, although there is some prominence of the pulmonary arteries.

Floating heart sign

The lungs may be so hyper-expanded that the inferior border of the heart becomes visible – the heart appears to float above the diaphragm

Watch out for bullous emphysema. Occasionally bullae are seen as discretely outlined holes in the lungs which resemble bubbles

Look for features of infective exacerbation. Check for large areas of black, due to bullae. You may also find a large area of consolidation in the left lung is due to pneumonia.

Emphysema is diagnosed by alveolar septal destruction and airspace enlargement, which may occur in a variety of distributions. Formation of giant bullae may lead to compression of mediastinal structures, while rupture of pleural blebs may produce spontaneous pneumothorax/pneumomediastinum.

Findings of chronic bronchitis on chest radiography are non-specific and include increased bronchovascular markings and cardiomegaly. In chronic bronchitis, bronchial wall thickening may be seen in addition to enlarged vessels. Repeated inflammation can lead to scarring with bronchovascular irregularity and fibrosis.

Emphysema manifests as lung hyperinflation with flattened hemidiaphragms, a small heart, and possible bullous changes. On the, lateral radiograph, a "barrel chest" with widened anterior-posterior diameter may be visualized. The "saber-sheath trachea" sign refers to marked coronal narrowing of the intrathoracic trachea (frontal view) with concomitant sagittal widening (lateral view).

Q & A

https://www.onlineanesthesiatools.com/copd

1. How do you define COPD?

   COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease. Spirometry is required to establish the diagnosis in this clinical context; the presence of a post-bronchodilator FEV1/FVC < 0.70 confirms the presence of persistent airflow limitation and thus of COPD in patients with appropriate symptoms and significant exposures to noxious stimuli.

   The key indicators are:

   Dyspnoea that is:
   - Progressive over time;
   - Characteristically worse with exercise;
   - Persistent.

   Chronic cough:
   - May be intermittent and may be unproductive,
   - Recurrent wheeze.

   Chronic sputum production:
   - Any pattern of sputum production may indicate COPD.

   Recurrent lower respiratory tract infections

   History of risk factors:
   - Host factors (such as genetic factors, congenital/developmental abnormalities etc),
   - Tobacco smoke (including popular local preparations).
   - Smoke from home cooking and heating fuels.
   - Occupational dusts, fumes, gases and other chemicals.

   Family history of COPD and/or Childhood factors:
   - For example low birth weight, childhood respiratory infections etc.

2. What is the pathophysiology?

   Expiration is normally a passive phenomenon, driven by the elastic recoil of the lungs. Airways closure, limiting expiration, is determined by the transmural pressure gradient between the pressure inside the conducting airways (generated by the elastic recoil) and the external pleural pressure.

   The causes of expiratory airflow limitation in these patients are multifactorial and the following features occur in varying proportions (Fig. 2.1):

   Chronic mucosal inflammation caused by repeated exposure to foreign material (e.g. smoking)

   Active bronchospasm and excessive bronchial secretions with mucosal plugging

   Destruction of normal alveolar architecture

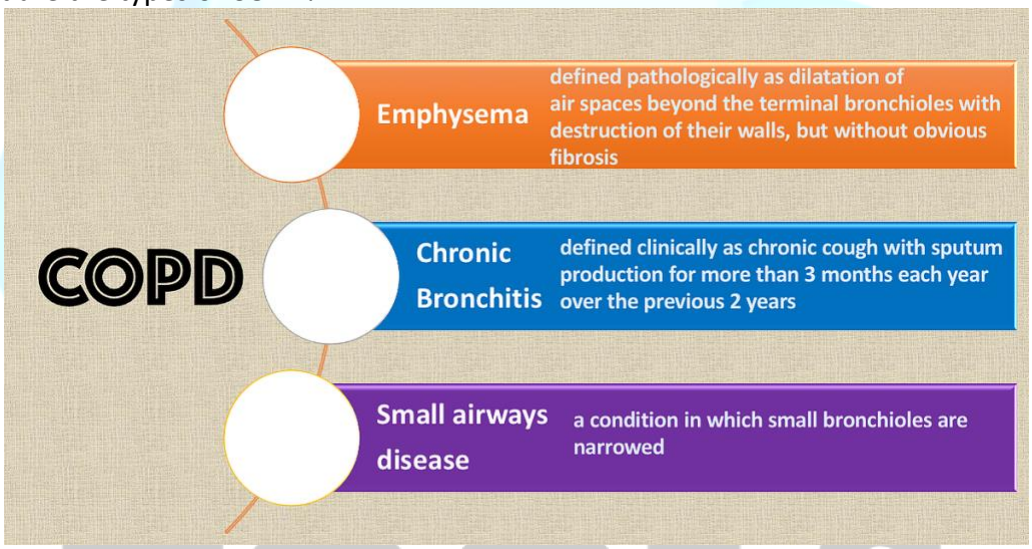
   Increased transmural pressure gradient across the wall of bronchiole favouring collapse during exhalation. (Increasing intrathoracic pressure due to forced exhalation exerting pressure on the bronchiolar walls via the neighboring lung parenchyma).

   Loss of elasticity of lung parenchyma causing less opening traction on the airways

Loss of rigidity of bronchiolus wall due to chronic inflammation
Increase in gas velocity in the narrowed bronchiolus, which lowers the pressure inside the bronchioles via Bernoulli's law.
In advanced stage, the limitation of expiratory flow occurs even during resting expiration. The inspiratory muscles work at a mechanical disadvantage, in part due to changes in the shape of the diaphragm and chest wall. Other features of the abnormal pulmonary mechanics in COPD patients include a greatly increased work of breathing, which in severe disease may account for more than 20% of the patient's total oxygen consumption.

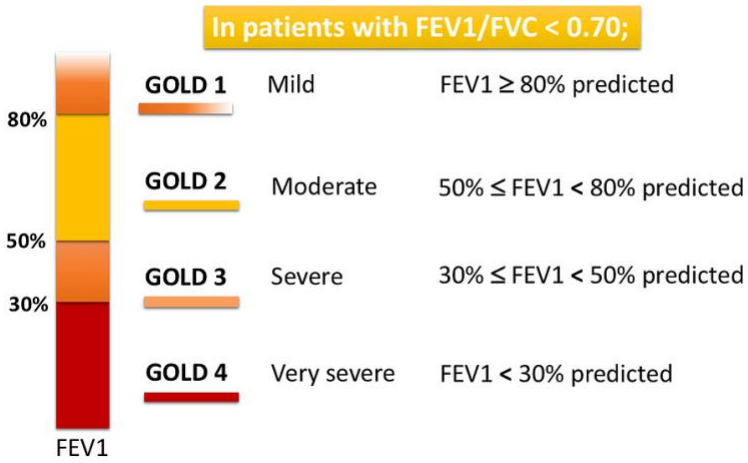
The consequences of these changes is incomplete expiration and gas trapping within the lungs, with a positive alveolar pressure at the end of expiration: the auto-PEEP (positive end-expiratory pressure). In patients breathing spontaneously auto-PEEP must be overcome before inspiratory flow can begin, further adding to the work of breathing. In mechanically ventilated patients auto-PEEP may promote unsuspected increases in intrathoracic pressure, reducing venous return and cardiac output. Auto-PEEP and the work of breathing can be reduced, and the mechanical efficiency of the respiratory muscles improved, by therapies that reduce airflow limitation and reduce gas trapping.

3. What are the types of COPD?



4. Grading of severity of COPD
Specific spirometric cut-points are used for purposes of simplicity. Spirometry should be performed after the administration of an adequate dose of at least one short-acting inhaled bronchodilator in order to minimize variability.

It should be noted that there is only a weak correlation between FEV1, symptoms and impairment of a patient's health status. For this reason, formal symptomatic assessment is required.



5. Define exacerbation of COPD.
   COPD exacerbation is defined as an event in the natural history of the disease characterized by a change in the patient's baseline dyspnea, cough and/or sputum that is beyond normal day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD. During exacerbations there is increased hyperinflation and gas trapping, with reduced expiratory flow, thus accounting for the increased dyspnoea. There is also worsening of V/Q abnormalities (ventilation-perfusing mismatch), which can result in hypoxemia.

6. How do you manage COPD infective exacerbation?
   Assess the severity of symptoms
   Investigations: Spirometry (if possible), blood gases, chest X-ray
   Controlled oxygen therapy is the cornerstone; repeat blood gases after 30-60 minutes. (adequate oxygenation: PaO2 >60 mm Hg and SaO2 > 90%)
   Bronchodilators:
   Increase doses and/or frequency
   Combine β2-agonists with anticholinergics
   Use spacers or air-driven nebulizers
   Consider adding intravenous mathylxanthines, if needed
   Add oral or intravenous glucocorticoids
   Consider antibiotics, if signs of bacterial infection are present
   Noninvasive mechanical ventilation
   At all times:
   Monitor fluid balance and nutrition
   Consider subcutaneous heparin
   Identify and treat associated conditions (e.g. heart failure, arrhythmias)
7. Describe the classic spirometry changes in COPD/Restrictive lung disease.

	Vital Capacity*	FEV_1	FEV_1/Vital Capacity
Obstructive Diseases			
COPD	Normal/decreased	Decreased	Decreased
Asthma	Normal/decreased	Decreased†	Decreased†
Restrictive diseases	Decreased	Decreased	Normal
Neuromuscular diseases	Decreased	Decreased	Decreased

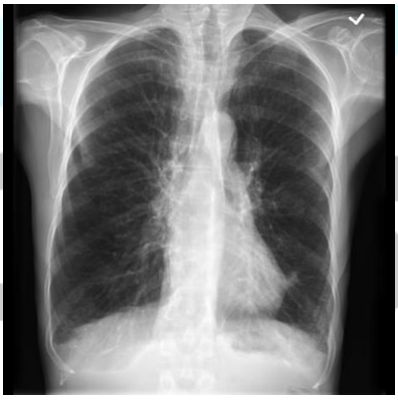
8. Concerns during oxygen supplementation for COPD patient
   It is abundantly clear that the rise in CO2 levels seen in COPD patients with oxygen therapy is unrelated to the abolition of the "hypoxic drive" as was conventionally believed. The inhibition of ventilation is transient and insignificant; the PCO2 levels rise through other mechanisms. Theories and hypotheses apart, the bottom line is that supplemental oxygen should never be denied to hypoxic patients. The chances of death due to uncorrected hypoxia is overwhelmingly higher than possible harm from the administration of supplemental oxygen. Needless to add, most patients, regardless of their CO2 levels, do not need an oxygen saturation of more than 92%.

   https://criticalcareblogspot.com/2018/12/02/the-hypoxic-drive-an-urban-legend/

9. Broad outline of management of COPD

New	0: At Risk	I: Mild	II: Moderate	III: Severe	IV: Very Severe
Characteristics	• Chronic symptoms • Exposure to risk factors • Normal spirometry	• $FEV_1/FVC < 70\%$ • $FEV_1 \geq 80\%$ • With or without symptoms	• $FEV_1/FVC < 70\%$ • $50\% \leq FEV_1 < 80\%$ • With or without symptoms	• $FEV_1/FVC < 70\%$ • $30\% \leq FEV_1 < 50\%$ • With or without symptoms	• $FEV_1/FVC < 70\%$ • $FEV_1 \geq 30\%$ or presence of chronic respiratory failure or right heart failure
	Avoidance of risk factor(s); influenza vaccination				
		Add short-acting bronchodilator when needed			
			Add regular treatment with one or more long-acting bronchodilators *Add* rehabilitation		
				Add inhaled glucocorticosteroids if repeated exacerbations	
					Add long-term oxygen if chronic respiratory failure *Consider* surgical treatments

10. A 65 yr old man; h/o 20 pack years smoking; MET >4; posted for inguinal hernia repair.
    CXR - as shown here.



    How do you evaluate perop?
    How do you optimize prep?
    Plan of anesthesia?
    What is incentive spirometry?
    What are your postop concerns?

    Key points:
    The key management principles for anesthesia for patients with reactive airway disease
    Preoperative optimization of bronchodilation.

- Minimal (or no) instrumentation of the airways.
- Instrument the airways when necessary only after appropriate depth of anesthesia with a bronchodilating anesthetic (propofol, ketamine, sevoflurane).
- Maintenance of anesthesia with a bronchodilating anesthetic.
- Appropriate warming and humidification of inspired gases. An endotracheal tube bypasses nearly the entire natural airway humidification system. So

humidification of inspired gases and use of low gas flows will be needed to keep airway secretions moist.

- Regional anesthesia is suitable for operations that do not invade the peritoneum and for surgical procedures performed on the extremities. Lower intraabdominal surgery can also be performed using a regional technique. General anesthesia is the usual choice for upper abdominal and intrathoracic surgery.

Advantages of regional anesthesia in COPD patients

- Avoids airway manipulation, which may cause bronchospasm
- Reduces the requirement of sedatives/opioids with ventilatory depressant effects
- Excellent analgesia
- Increase in functional residual capacity (FRC); normalization of FRC and closing capacity (CC) ratio
- Preservation of phrenic nerve activity (Inhibition of phrenic nerve function via spinal reflex arcs appears to be responsible for the diaphragmatic muscle dysfunction seen after upper abdominal and thoracic surgery)
- Reduced incidence of deep vein thrombosis
- Reduces postoperative pulmonary complications
- Avoiding positive pressure ventilation is advantageous in patients with "bullae"
- When combined with general anesthesia, significant reduction in postoperative intubation time (i.e. early extubation) and requirement for mechanical ventilation after major abdominal and thoracic surgery.

https://www.onlineanesthesiatools.com/copd2

11. A 52 yr old man; known COPD; on bronchodilators/steroids via inhalers; report to ER with % severe breathlessness with fever - 2 days.

Your initial management plan?
Indications for ICU admission?
Outline your initial mechanical ventilator settings

Key:

Indications for ICU admission

- Severe dyspnea that responds inadequately to initial emergency therapy
- Changes in mental status (lethargy, confusion, coma)
- Persistent or worsening hypoxia ($PaO_2 < 40$ mmHg) and/or severe worsening acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation
- Need for invasive mechanical ventilation
- Hemodynamic instability – need for vasopressors

Noninvasive Ventilation (NIV)

Noninvasive mechanical ventilation in exacerbations improves respiratory acidosis, increases pH, decreases the need for endotracheal intubation, and reduces $PaCO_2$, respiratory rate, severity of breathlessness, the length of hospital stay and mortality.

The common indications for noninvasive ventilation are:

Moderate to severe dyspnea with use of accessory muscles of respiration and paradoxical abdominal breathing, or retraction of the intercostal spaces.
Moderate to severe acidosis (pH ≤ 7.35) and/or hypercapnia (PCO_2 ≥ 45 mm Hg)

The presence of any of the following is considered as a contraindication to noninvasive ventilation:

Respiratory arrest
Cardiovascular instability (hypotension, arrhythmias, myocardial infarction)
Change in mental status; uncooperative patient
High aspiration risk
Viscous or copious secretions
Recent facial or gastroesophageal surgery
Craniofacial trauma
Fixed nasopharyngeal abnormalities
Burns
Extreme obesity.

Invasive Mechanical Ventilation

The indications for invasive mechanical ventilation are:

- Unable to tolerate NIV or NIV failure
- Respiratory or cardiac arrest
- Respiratory pauses with loss of consciousness or gasping for air
- Diminished consciousness, psychomotor agitation inadequately controlled by sedation
- Massive aspiration
- Persistent inability to remove respiratory secretions
- Heart rate < 50 per min with loss of alertness
- Severe hemodynamic instability without response to fluids and vasoactive drugs
- Severe ventricular arrhythmias
- Life threatening hypoxemia.

Weaning COPD patient from mechanical ventilation

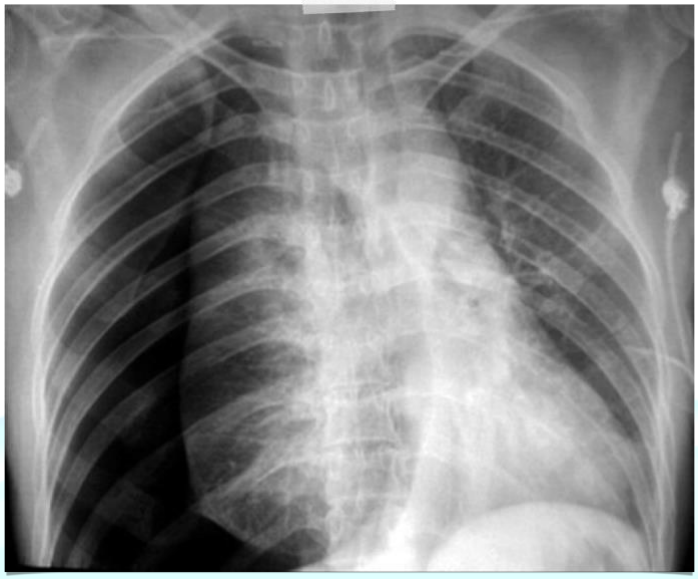
Weaning or discontinuation from mechanical ventilation can be particularly difficult and hazardous in COPD patients. The most influential determinant of dependency on mechanical ventilation in these patient is the balance between the respiratory load and the ability of the respiratory muscles to cope with the load. Hence weaning may become a prolonged process and challenging. The best method of weaning (pressure support or T-piece trial) is still a matter of debate. In COPD patients that failed extubation, noninvasive ventilation facilitates weaning and prevents reintubation, but does not reduce mortality.

12. What advice would you give a smoker 24 hours before a scheduled procedure under general anesthesia and why?

It is known that circulating catecholamine levels return to normal within 1 hour and carboxyhaemoglobin clearance occurs within 24 hours, thus massively improving oxygen delivery to all tissues including the myocardium, reducing the risk of perioperative ischaemic event. As oxygen carriage improves, physiological reserve to cope with perioperative periods of inadvertent hypoxia improves.

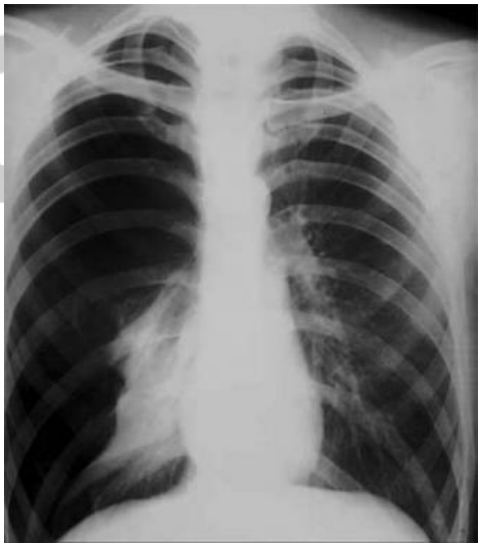
Postoperatively, ongoing smoking is known to be associated with poor tissue healing, including wounds, anastomoses, flaps. Blood hypercoagulability will start to improve as carbon monoxide levels fall, reducing risk of postoperative thrombotic events.
Hence, it is advisable to stop smoking for the remaining 24 hours preoperatively and, ideally, to stop thereafter.

Case 2: Pneumothorax



You can find hypertranslucency, with absence of lung markings, collapsed lung margin on right side, with shift of mediastinum.
This is Right sided pneumothorax.

1. How do you define pneumothorax?
   Pneumothorax is defined as the presence of air or gas in the pleural cavity (ie, the potential space between the visceral and parietal pleura of the lung), which can impair oxygenation and/or ventilation. The clinical results are dependent on the degree of collapse of the lung on the affected side. If the pneumothorax is significant, it can cause a shift of the mediastinum and compromise hemodynamic stability. Air can enter the intrapleural space through a communication from the chest wall (ie, trauma) or through the lung parenchyma across the visceral pleura.



(Radiograph of a patient with a complete right-sided pneumothorax due to a stab wound.)

2. What are the signs and symptoms of pneumothorax?

The presentation of pneumothorax ranges from completely asymptomatic to life-threatening respiratory distress:

Respiratory distress (considered a universal finding) or respiratory arrest

Tachypnea (or bradypnea as a preterminal event)

Asymmetric lung expansion: Mediastinal and tracheal shift to contralateral side (large tension pneumothorax)

Distant or absent breath sounds: Unilaterally decreased/absent lung sounds common, but decreased air entry may be absent even in advanced state of pneumothorax

Minimal lung sounds transmitted from unaffected hemithorax with auscultation at midaxillary line

Hyperresonance on percussion: Rare finding; may be absent even in an advanced state

Decreased tactile fremitus

Adventitious lung sounds: Ipsilateral crackles, wheezes

Cardiovascular findings may include the following:

Tachycardia: Most common finding; if heart rate is faster than 135 beats/min, tension pneumothorax likely

Pulsus paradoxus

Hypotension: Inconsistently present finding; although typically considered a key sign of tension pneumothorax, hypotension can be delayed until its appearance immediately precedes cardiovascular collapse

Jugular venous distention: Generally seen in tension pneumothorax; may be absent if hypotension is severe

Cardiac apical displacement: Rare finding

3. What are the physical findings specific to each type of pneumothorax?

Common findings among the types of pneumothoraces include the following:

- Spontaneous and iatrogenic pneumothorax: Tachycardia most common finding; tachypnea and hypoxia may be present.
- Tension pneumothorax: Variable findings; respiratory distress and chest pain; tachycardia; ipsilateral air entry on auscultation; breath sounds absent on affected hemithorax; trachea may deviate from affected side; thorax may be hyperresonant; jugular venous distention and/or abdominal distention may be present
- Pneumomediastinum: Variable or absent findings; subcutaneous emphysema is the most consistent sign; Hamman sign—a precordial crunching noise synchronous with the heartbeat and often accentuated during expiration—has a variable rate of occurrence, with one series reporting 10%

4. What is the pathophysiology of tension pneumothorax?

Tension pneumothorax occurs anytime a disruption involves the visceral pleura, parietal pleura, or the tracheobronchial tree. This condition develops when injured tissue forms a one-way valve, allowing air inflow with inhalation into the pleural space and prohibiting air outflow. The volume of this nonabsorbable intrapleural air increases with each inspiration because of the one-way valve effect. As a result, pressure rises within the affected hemithorax. In addition to this mechanism, the positive pressure used with mechanical ventilation therapy can cause air trapping.

As the pressure increases, the ipsilateral lung collapses and causes hypoxia. Further pressure increases cause the mediastinum to shift toward the contralateral side and impinge on and compress both the contralateral lung and impair the venous return to the right atrium. Hypoxia results as the collapsed lung on the affected side and the compressed lung on the contralateral side compromise effective gas exchange. This hypoxia and decreased venous return caused by compression of the relatively thin walls of the atria impair cardiac function. Kinking of the inferior vena cava is thought to be the initial event restricting blood to the heart. It is most evident in trauma patients who are hypovolemic with reduced venous blood return to the heart.

Arising from numerous causes, this condition rapidly progresses to respiratory insufficiency, cardiovascular collapse, and, ultimately, death if unrecognized and untreated.

5. What are the signs and symptoms of tension pneumothorax?

Symptoms of tension pneumothorax may include chest pain (90%), dyspnea (80%), anxiety, fatigue, or acute epigastric pain (a rare finding).

Tension pneumothorax is classically characterized by hypotension and hypoxia. On examination, breath sounds are absent on the affected hemothorax and the trachea deviates away from the affected side. The thorax may also be hyperresonant; jugular venous distention and tachycardia may be present. If on mechanical ventilation, the airway pressure alarms are triggered.

6. Which findings on chest radiographs suggest pneumothorax?

Finding of pneumothorax on chest radiographs may include the following:

- A linear shadow of visceral pleura with lack of lung markings peripheral to the shadow may be observed, indicating collapsed lung
- An ipsilateral lung edge may be seen parallel to the chest wall
- In supine patients, deep sulcus sign (very dark and deep costophrenic angle) with radiolucency along costophrenic sulcus may help to identify occult pneumothorax; the anterior costophrenic recess becomes the highest point in the hemithorax, resulting in an unusually sharp definition of the anterior diaphragmatic surface due to gas collection and a depressed costophrenic angle
- Mediastinal shift toward the contralateral lung may also be apparent
- Airway or parenchymal abnormalities in the contralateral lung suggest causes of secondary pneumothorax; evaluation of the parenchyma in the collapsed lung is less reliable

7. Describe the ultrasound (USG) findings suggest pneumothorax?

Features of the ultrasonographic examination for the diagnosis of pneumothorax include:

a) absence of lung sliding (high sensitivity and specificity)

In the absence of pleural disease, visceral pleura move against parietal pleura while breathing. This movement of the 2 pleura is detected by the ultrasound as lung sliding, which is a "kind of twinkling synchronized with respiration" seen in real-time and time-motion modes. That is, lung sliding refers to normal pleural movement in patients without pneumothorax.

b) absence of comet-tail artifact (high sensitivity, lower specificity)

Comet-tail artifacts are vertical air artifacts that arise from the visceral pleural line (or in the case of parietal emphysema or shotgun pellets may arise above the pleural line).

c) presence of lung point (high specificity, lower sensitivity)

Lung point is the location that lung-sliding and absent lung-sliding alternately appear; it has been shown in multiple studies to allow determination of the size of a pneumothorax. Zhang et al obtained a 79% sensitivity in lung point's ability to determine pneumothorax size.

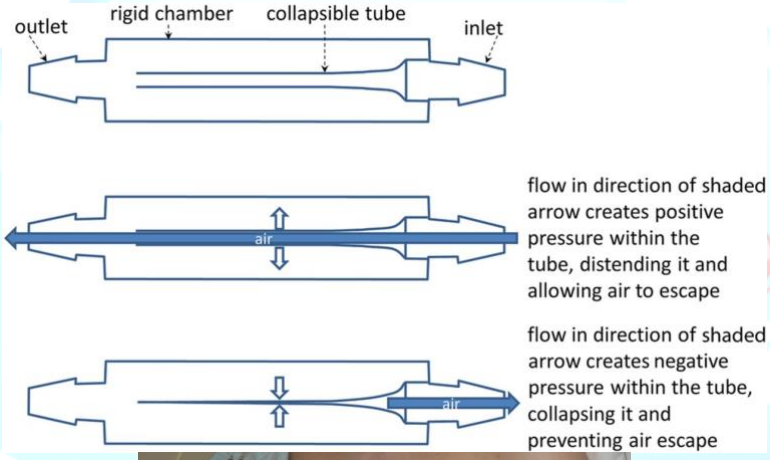
8. What are the options for restoring air-free plural space in pneumothorax?

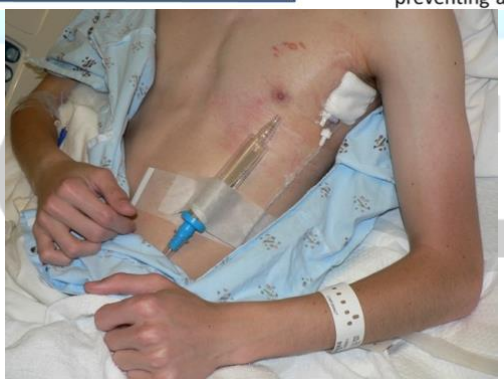
Several options are available to restore an air-free pleural space, including

i. observation without oxygen,
ii. administering supplemental oxygen,
iii. simple aspiration,
iv. chest tube placement,
v. one-way valve insertion, and
vi. thoracostomy with continuous suction.

9. How are chest tubes placed during treatment of pneumothorax?

A tube inserted into the pleural space is connected to a device with one-way flow for air removal. Examples of such devices are Heimlich valves or water seal canisters, and tubes connected to wall suction devices.





A Heimlich valve is a one-way, rubber flutter valve that allows for complete evacuation of air that is not under tension. The proximal end attaches to the chest tube or catheter, and the distal end connects to a suction device or is left open to the atmosphere. Heimlich valves do not require suction and thus eliminate the chance of a tension pneumothorax; they also allow greater mobility and less discomfort for the patient.

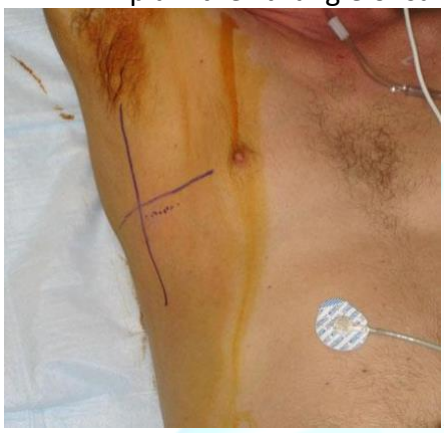
10. Describe the initial emergency department (ED) care of pneumothorax.

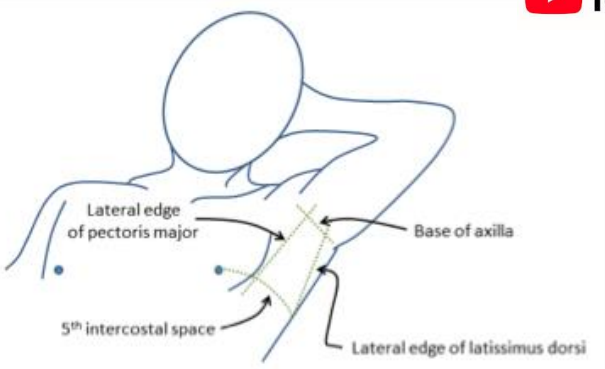
Immediate attention to the ABCs (airway, breathing, circulation) while assessing vital signs and oxygen saturation is paramount, particularly in patients with thoracic trauma.

Evaluate the patency of the airway and the adequacy of the ventilatory effort. Assess the circulatory status and the integrity of the chest wall. Carefully evaluate the cardiovascular system, because a tension pneumothorax and pericardial tamponade can cause similar findings.

ED care depends on the hemodynamic stability of the patient. All patients should receive supplemental oxygen to increase oxygen saturation and to enhance the reabsorption of free air.

11. Explain the “triangle of safety”.





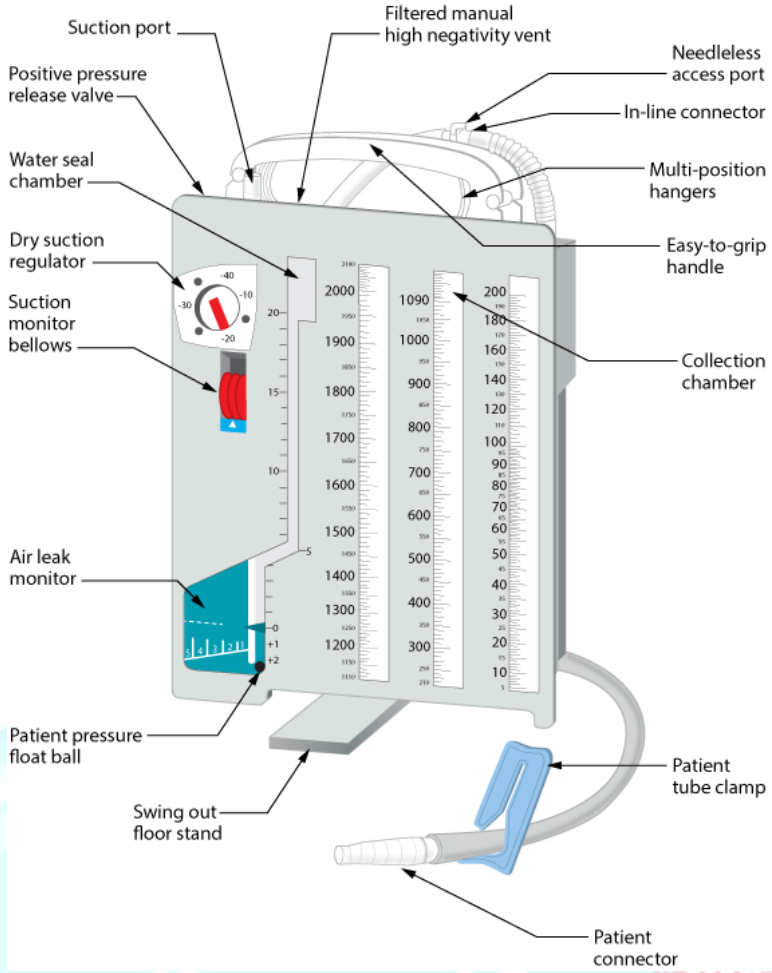
The triangle of safety is an anatomical region in the axilla that forms a guide as to the safe position for intercostal catheter (ICC) placement. With the arm abducted, the apex is the axilla, and the triangle is formed by the:

a) lateral border of the pectoralis major anteriorly
b) lateral border of the latissimus dorsi posteriorly
c) inferiorly, by a horizontal line from the nipple (commonly the 5th intercostal space)

As with all intercostal approaches, placing the catheter closer to the superior border of the rib below in the intercostal space should avoid injury to the intercostal neurovascular bundle under the costal groove of the rib above.

12. Explain underwater seal chest drainage system.

A system that allows drainage of the pleural space using an airtight system to maintain subatmospheric intrapleural pressure; the underwater seal acts a one-way valve.



Most commonly, drainage systems use three chambers which are based on the three-bottle system.

i. The first chamber allows fluid that is drained from the chest to be collected.

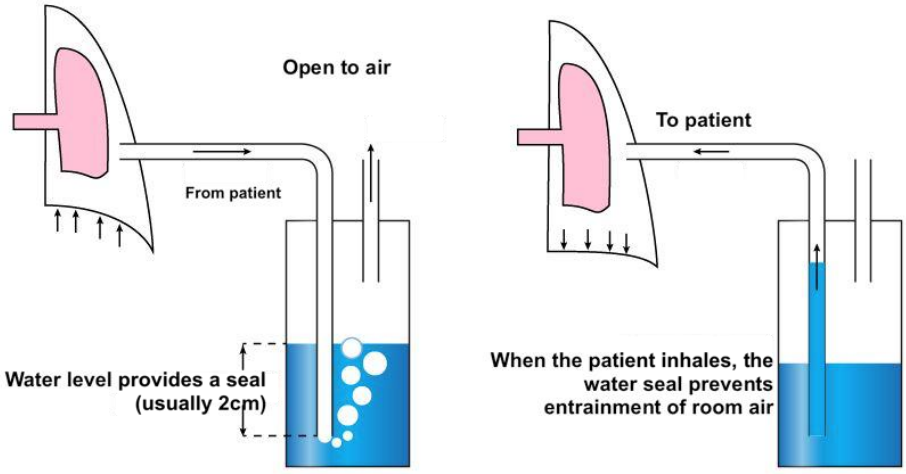
ii. The second chamber functions as a "water seal", which acts as a one way valve allowing gas to escape, but not reenter the chest. Air bubbling through the water seal chamber is usual when the patient coughs or exhales but may indicate, if continual, a pleural or system leak that should be evaluated critically. It can also indicate a leak of air from the lung.

iii. The third chamber is the suction control chamber. The height of the water in this chamber regulates the negative pressure applied to the system.

A gentle bubbling through the water column minimizes evaporation of the fluid and indicates that the suction is being regulated to the height of the water column. In this way, increased wall suction does not increase the negative pressure of the system.

13. Explain the working principle of single-chamber underwater seal system.

It is a single bottle, open to air. The patient's chest tube is submerged under a level of water (usually about 2cm) which acts as a one-way valve. When the patient's pleural pressure exceeds the level of water (i.e. it is greater than 2cm H2O), the air in the tube will bubble out and escape into the atmosphere. When the patient takes a breath in, the negative intrapleural pressure will suck drain water up the tube, but no additional air can enter.



14. Enumerate the safety guidelines for ICD underwater seal systems
- The first tube connecting drain to drainage bottles must be wide to decrease resistance
- The volume capacity of this tube should exceed half of patient's maximum inspiratory volume (otherwise water may enter chest)
- The volume of water in bottle B should exceed half patient's maximum inspiratory volume to prevent indrawing of air during inspiration
- Drain should always stay at least 45cm below patient (prevention of removed fluid or water refluxing into patient)
- Clamp the drain tube when moving
- Water level above tube in the manometer bottle determines the amount of suction applied before air drain through tube (safety suction limiting device)
- If suction is turned off then tubing must be unplugged -> so air can escape into atmosphere (otherwise a tension pneumothorax)
- Should not be applied following pneumonectomy

Case 3: Acute Pulmonary Edema

1. What is cardiogenic pulmonary edema?

Cardiogenic pulmonary edema (CPE) is defined as pulmonary edema due to increased capillary hydrostatic pressure secondary to elevated pulmonary venous pressure. CPE reflects the accumulation of fluid with a low-protein content in the lung interstitium and alveoli as a result of cardiac dysfunction.

2. What are the pathophysiologic mechanisms that cause cardiogenic pulmonary edema (CPE)?

Pulmonary edema can be caused by the following major pathophysiologic mechanisms:

a) Imbalance of Starling forces - Ie, increased pulmonary capillary pressure, decreased plasma oncotic pressure, increased negative interstitial pressure
b) Damage to the alveolar-capillary barrier
c) Lymphatic obstruction
d) Idiopathic (unknown) mechanism

3. Explain the pathophysiology of cardiogenic pulmonary edema (CPE)?

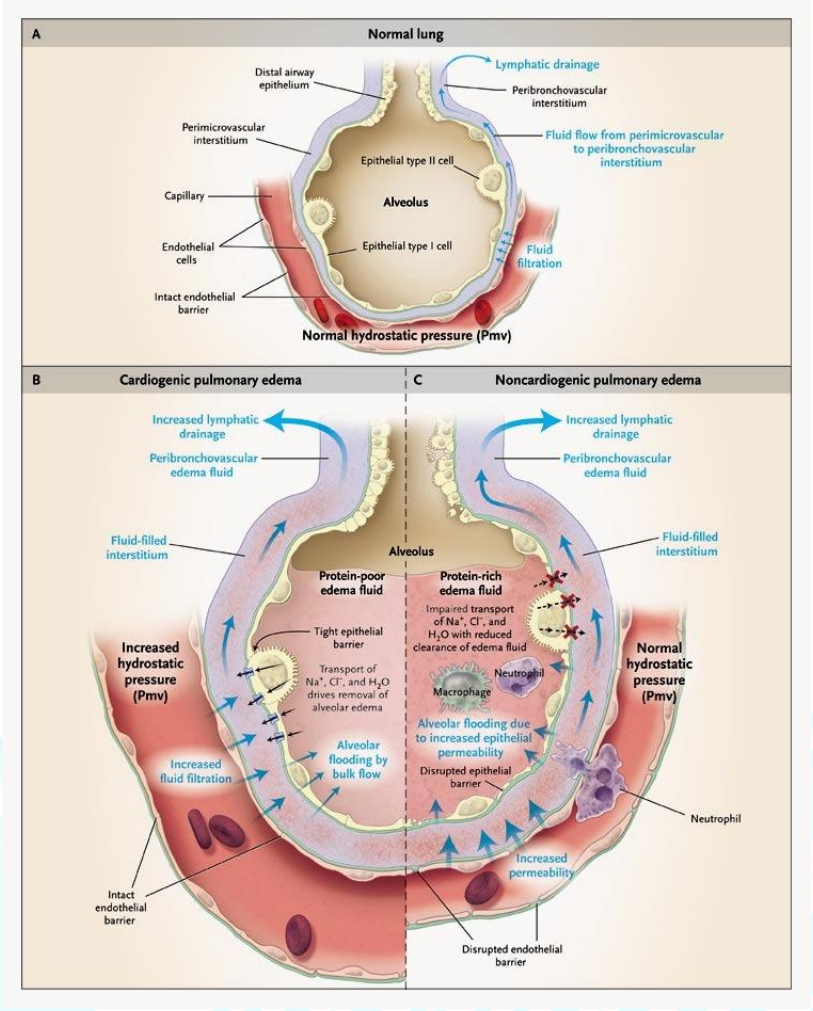
Pulmonary capillary blood and alveolar gas are separated by the alveolar-capillary membrane, which consists of 3 anatomically different layers: (1) the capillary endothelium; (2) the interstitial space, which may contain connective tissue, fibroblasts, and macrophages; and (3) the alveolar epithelium.

Exchange of fluid normally occurs between the vascular bed and the interstitium. Pulmonary edema occurs when the net flux of fluid from the vasculature into the interstitial space is increased. The Starling relationship determines the fluid balance between the alveoli and the vascular bed. Net flow of fluid across a membrane is determined by applying the following equation:

Q = K(Pcap - Pis) - l(Pcap - Pis),

where Q is net fluid filtration; K is a constant called the filtration coefficient; Pcap is capillary hydrostatic pressure, which tends to force fluid out of the capillary; Pis is hydrostatic pressure in the interstitial fluid, which tends to force fluid into the capillary; l is the reflection coefficient, which indicates the effectiveness of the capillary wall in preventing protein filtration; the second Pcap is the colloid osmotic pressure of plasma, which tends to pull fluid into the capillary; and the second Pis is the colloid osmotic pressure in the interstitial fluid, which pulls fluid out of the capillary.

The net filtration of fluid may increase with changes in different parameters of the Starling equation. CPE predominantly occurs secondary to LA outflow impairment or LV dysfunction. For pulmonary edema to develop secondary to increased pulmonary capillary pressure, the pulmonary capillary pressure must rise to a level higher than the plasma colloid osmotic pressure. Pulmonary capillary pressure is normally 8-12 mm Hg, and colloid osmotic pressure is 28 mm Hg. High pulmonary capillary wedge pressure (PCWP) may not always be evident in established CPE, because the capillary pressure may have returned to normal when the measurement is performed.



Reference: Ware, L. B., & Matthay, M. A. (2005). Acute Pulmonary Edema. New England Journal of Medicine, 353(26), 2788–2796.

4. Which physical findings are characteristic of cardiogenic pulmonary edema (CPE)?

Physical findings in patients with CPE are notable for tachypnea and tachycardia. Patients may be sitting upright, they may demonstrate air hunger, and they may become agitated and confused. Patients usually appear anxious and diaphoretic.
Hypertension is often present, because of the hyperadrenergic state. Hypotension indicates severe LV systolic dysfunction and the possibility of cardiogenic shock. Cool extremities may indicate low cardiac output and poor perfusion.
Auscultation of the lungs usually reveals fine, crepitant rales, but rhonchi or wheezes may also be present. Rales are usually heard at the bases first; as the condition worsens, they progress to the apices.

5. What is brain-type natriuretic peptide (BNP) and N-terminal proBNP (NT-proBNP) testing, and why is it performed in the workup of cardiogenic pulmonary edema?

Brain-type natriuretic peptide (BNP) and N -terminal proBNP (NT-proBNP) are derived from pre-proBNP, a 134–amino acid precursor synthesized by cardiac myocytes. A number of triggers including wall stretch, ventricular dilation, and/or increased pressures, stimulate a 26–amino-acid signal peptide sequence to be cleaved from the precursor's N -terminus to produce proBNP (which has a 108–amino acid sequence). This hormone is further cleaved by

a membrane-bound serine protease (corin) into the inactive NT-proBNP fragment and the active BNP (32–amino acid sequence) fragment.
NT-proBNP and BNP testing are clinically available and have exhibited parallel changes across broad ranges of patient age, ejection fraction, diastolic CHF, and renal function.

6. What is the role of Ultrasonography in evaluation of CPE?

In cases in which there is a moderate to high pretest probability of acute CPE, ultrasonography can be useful in strengthening a working diagnosis. Findings of B-lines on ultrasonography have been reported to have a sensitivity of 94.1% and a specificity of 92.4% for acute CPE.

Analysis of critical care ultrasonography (CCUS) findings revealed that a low B-line ratio was predictive of miscellaneous cause vs CPE or ARDS. In the further differentiation of CPE from ARDS, moderately or severely decreased left ventricular function, left-sided pleural effusion (> 20 mm), and a large inferior vena cava minimal diameter (> 23 mm) were predictive of CPE.

7. Describe Kerley lines

Septal lines, also known as Kerley lines, are seen when the interlobular septa in the pulmonary interstitium become prominent. This may be because of lymphatic engorgement or edema of the connective tissues of the interlobular septa. They usually occur when pulmonary capillary wedge pressure reaches 20-25 mmHg. Kerley lines are named after Sir Peter James Kerley (1900-1979), an Irish radiologist.

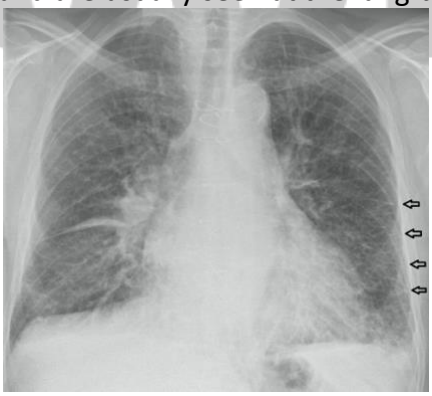
Classification

a) Kerley A lines

These are 2-6 cm long oblique lines that are <1 mm thick and course towards the hila. They represent thickening of the interlobular septa that contain lymphatic connections between the perivenous and bronchoarterial lymphatics deep within the lung parenchyma. On chest radiographs they are seen to cross normal vascular markings and extend radially from the hilum to the upper lobes. HRCT is the best modality for the demonstration of Kerley A lines.

b) Kerley B lines

These are thin lines 1-2 cm in length in the periphery of the lung(s). They are perpendicular to the pleural surface and extend out to it. They represent thickened subpleural interlobular septa and are usually seen at the lung bases.



c) Kerley C lines

Kerley C lines are short lines which do not reach the pleura (i.e. not B or D lines) and do not course radially away from the hila (i.e. not A lines).

d) Kerley D lines

Kerley D lines are exactly the same as Kerley B lines, except that they are seen on lateral chest radiographs in the retrosternal air gap.

Case 4: ARDS

1. How is acute respiratory distress syndrome (ARDS) defined?

ARDS is defined by timing (within 1 week of clinical insult or onset of respiratory symptoms); radiographic changes (bilateral opacities not fully explained by effusions, consolidation, or atelectasis); origin of edema (not fully explained by cardiac failure or fluid overload); and severity based on the PaO2/FiO2 ratio on 5 cm of continuous positive airway pressure (CPAP).

The 3 categories are mild (PaO2/FiO2 200-300), moderate (PaO2/FiO2 100-200), and severe (PaO2/FiO2 ≤100).

Ref: ARDS Definition Task Force, Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA. 2012 Jun 20. 307 (23):2526-33.

2. Describe the pathogenesis of acute respiratory distress syndrome (ARDS).

ARDS is associated with diffuse alveolar damage (DAD) and lung capillary endothelial injury. The early phase is described as being exudative, whereas the later phase is fibroproliferative in character.

ARDS causes a marked increase in intrapulmonary shunting, leading to severe hypoxemia. Although a high FiO2 is required to maintain adequate tissue oxygenation and life, additional measures, like lung recruitment with PEEP, are often required. Theoretically, high FiO2 levels may cause DAD via oxygen free radical and related oxidative stresses, collectively called oxygen toxicity. Generally, oxygen concentrations higher than 65% for prolonged periods (days) can result in DAD, hyaline membrane formation, and, eventually, fibrosis.

3. How is cardiogenic pulmonary edema distinguished from acute respiratory distress syndrome (ARDS)?

Because cardiogenic pulmonary edema must be distinguished from ARDS, carefully look for signs of congestive heart failure or intravascular volume overload, including jugular venous distention, cardiac murmurs and gallops, hepatomegaly, and edema.

4. What is the role of radiography in the evaluation of acute respiratory distress syndrome (ARDS)?

ARDS is defined by the presence of bilateral pulmonary infiltrates. The infiltrates may be diffuse and symmetric or asymmetric, especially if superimposed upon preexisting lung disease or if the insult causing ARDS was a pulmonary process, such as aspiration or lung contusion.

The pulmonary infiltrates usually evolve rapidly, with maximal severity within the first 3 days. Infiltrates can be noted on chest radiographs almost immediately after the onset of gas exchange abnormalities. They may be interstitial, characterized by alveolar filling, or both.

Initially, the infiltrates may have a patchy peripheral distribution, but soon they progress to diffuse bilateral involvement with ground glass changes or frank alveolar infiltrates.

5. What is the role of noninvasive positive-pressure ventilation (NIPPV) in the treatment of acute respiratory distress syndrome (ARDS)?

Because intubation and mechanical ventilation may be associated with an increased incidence of complications, such as barotrauma and nosocomial pneumonia, alternatives to mechanical ventilation such as a high-flow nasal cannula or noninvasive positive-pressure ventilation (NIPPV) may be beneficial in patients with ARDS. High-flow nasal cannula uses a system of heated humidification and large-bore nasal prongs to deliver oxygen at flows of up to 50-60 L/min. This is usually used in conjunction with an oxygen blender, allowing delivery of precise inspired oxygen concentrations. High-flow nasal cannula is usually well tolerated and allows the patient to talk, eat, and move around. NIPPV is usually given by full facemask. Sometimes, continuous positive airway pressure (CPAP) alone may be sufficient to improve oxygenation. In a 2015 study on hypoxemic, nonhypercapnic patients comparing standard oxygen, high-flow nasal cannula, and NIPPV, all three modes had the same incidence of need for intubation/mechanical ventilation, but high-flow nasal cannula resulted in improved 90-day mortality.

6. What are the goals of mechanical ventilation for the treatment of acute respiratory distress syndrome (ARDS)?

The goals of mechanical ventilation in ARDS are to maintain oxygenation while avoiding oxygen toxicity and the complications of mechanical ventilation. Generally, this involves maintaining oxygen saturation in the range of 85-90%, with the aim of reducing the fraction of inspired oxygen (FiO2) to less than 65% within the first 24-48 hours. Achieving this aim almost always necessitates the use of moderate-to-high levels of positive end-expiratory pressure (PEEP).

7. Explain the "***baby lung concept***" for mechanical ventilation in ARDS.

Mechanical ventilation does not cure ARDS but simply buys time by maintaining a gas exchange sufficient for survival. The "baby lung" concept originated as an offspring of computed tomography examinations which showed in most patients with acute lung injury/acute respiratory distress syndrome that the normally aerated tissue has the dimensions of the lung of a 5- to 6- year-old child (300–500 g aerated tissue).

Now, we know that ARDS lung is not stiff but small, with near normal intrinsic mechanical characteristics. The size of the baby lung determines tissue compliance and directly dictates the mechanical properties of the ARDS lung. The use of PEEP keeps open newly recruited and previously non-aerated regions, thus enlarging the baby lung and improving oxygenation.

The baby lung is more a functional concept than an anatomical one; in fact, in the prone position, the baby lung ''shifts'' from the ventral lung regions toward the dorsal lung regions while usually increasing its size. The "baby lung" may quickly change its location and/or its dimensions with PP and/or application of PEEP.

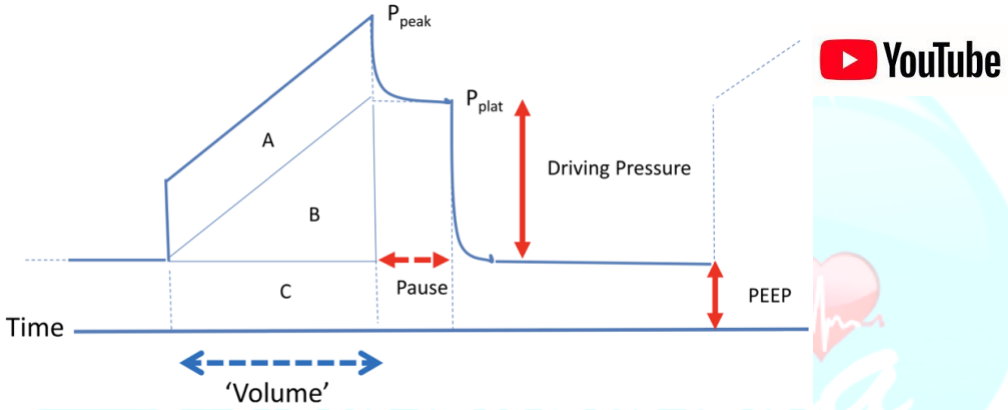
The practical message is straightforward, the smaller the baby lung, the greater is the potential for unsafe mechanical ventilation. Ventilator associated lung injury (VALI) is attributable in part to the application of physiological tidal volumes to the reduced area of non-consolidated alveoli.

8. Enumerate the **Lung Protective Ventilation Strategies (LPVS).**
    - Avoid regional over distension (Baby lung concept)

- Avoid repeated opening/closing of airway (Open lung concept)
- Permissive Hypercapnia
- Permissive Hypoxemia

9. Initial ventilatory setting on conventional invasive ventilation in ARDS patient
   - Ventilator mode- PRVC >PCV >VCV/(HFOV- Whenever indicated)
   - Tidal volume- < 6ml/kg (adjusted according to plateau pressure)
   - Plateau pressure <30 cmH20
   - Rate- 6 to 35/min
   - I: E ratio- 1:1 to 1:3
   - PEEP and FiO2-Set according to predetermined combinations (PEEP range 5 to 24 cmH2O), limit FiO2 less than 0.6 whenever possible.
   - Oxygenation target
     PaO2-55- 80 mmHg / SpO2- 88-95%

10. What is "**driving pressure**:?



In patients without spontaneous breathing efforts (i.e., sedated and/or paralyzed on controlled mechanical ventilation), the driving pressure of the respiratory system is defined as the difference between plateau pressure and positive end-expiratory pressure (Pplat-PEEP), and can also be expressed as the ratio of tidal volume to respiratory system compliance (Vt/Crs). The potential importance of driving pressure in patients with ARDS was first recognized in 1998.

Driving pressure (DP): the change in airway pressure during a tidal breath.

◦ DP = Pplat – PEEP

◦ Crs = Vt/(Pplat – PEEP) = Vt/DP ◦ DP = Vt/Crs

Thus, DP represents the tidal volume corrected for the patient's respiratory system compliance. Hence, lower driving pressure may be associated with lower mortality may be due to a resultant reduction in cyclic lung stretch/inflation during mechanical ventilation. Driving pressure can be considered as a complementary tool to adjust tidal volume or PEEP, particularly in patients with severe ARDS.

Case 5: Pacemaker in-situ

Pacemakers are frequently used for a variety of brady- and tachyarrythmias. The North American Society of Pacing and Electrophysiology and the British Pacing and Electrophysiology Group initiated a generic code for describing pacemaker function. The code

consists of five letters or positions. The first three describe antibradycardia functions and are always stated. The fourth and fifth positions relate to additional functions and are often omitted if not present (e.g. VDD or 000MS).

Position 1 refers to the chamber paced (V/A/D for ventricle, atrium or dual).
Position 2 refers to the chamber sensed (V/A/D).
Position 3 refers to response to sensing (T/I/D for triggered, inhibited or dual).
Position 4 refers to programmability or rate modulation (P simple programmable, M multiprogrammable, R rate modulation).
Position 5 anti-tachycardia functions (P pacing, S shock, D dual).

Anesthetic Implications

- Patients with pacemakers should attend follow-up clinics. The most recent pacemaker check should confirm good function. A preoperative ECG will detect some problems but by no means all. Absence of all pacing spikes may represent appropriate sensing or total failure.
- Bipolar diathermy is safe with pacemakers. If conventional diathermy is necessary then the plate should be positioned so that most of the current passes away from the pacemaker and it should be used in short bursts. The pacing wires may act as aerials and cause heating where the wire contacts the endocardium. The pacemaker may detect the diathermy as ventricular activity and inhibit its output, but only for the duration of diathermy use. In an emergency a pacemaker can be changed to V00 (asynchronous - ventricular pacing) by placing a magnet over the box. However, this should only be done as a last resort as there is a risk of inducing ventricular fibrillation.
- Any pacemaker that has the facility to cardiovert or overpace should have this facility turned off for theatre to avoid erroneous discharge if diathermy noise is interpreted as a tachyarrhythmia. It is important that it is turned back on before the patient leaves the theatre area.

Case 6: Consolidation of the right middle lobe

Ans: Pneumonic infiltration of the right middle lobe with indistinct right heart border. Lateral view confirms consolidation of the right middle lobe.

Consolidation refers to the alveolar airspaces being filled with fluid (exudate/transudate/blood), cells (inflammatory), tissue, or other material.

The list of causes of consolidation is broad but for complete consolidation of a lobe, the most common cause is pneumonia.

General features of consolidation on CXR include:

- airspace opacification causing obscuration of pulmonary vessels
- air bronchograms

Specific lobar consolidation can be determined by the location of airspace opacification, pattern and the effect on adjacent structures (silhouette sign):

right upper lobe consolidation

may obscure right paratracheal stripe
may outline the horizontal fissure below

right middle lobe consolidation
may obscure the right heart border
may outline the horizontal fissure above
right lower lobe consolidation
may obscure the right hemidiaphragm
left upper lobe consolidation
may obscure the left heart border
may obscure the left paratracheal stripe
left lower lobe consolidation
may obscure the left hemidiaphragm
may obscure the descending aorta

It must be remembered that the homogeneity of the consolidation will be influenced by any underlying lung disease. For example, consolidation in background emphysematous lung is very often non-confluent.

Case 7: Mitral stenosis

Radiographs demonstrate cardiomegaly, in particular enlargement of the left atrial appendage and left atrium consistent with underlying mitral valve stenosis. There is also prominence the central pulmonary vasculature consistent with pulmonary hypertension. The vascular clarity is maintained. Lateral views again show left atrial enlargement. Follow up postoperative radiographs demonstrate bilateral pleural drainage catheters. Changes of interval median sternotomy and clipping of the left atrial appendage. There are new moderate right and small left pleural effusions with associated basilar atelectasis. The cardiomediastinal silhouette is partially obscured, but appears grossly stable in size. There is mild pulmonary edema.

Case 7: Foreign body ingestion and aspiration

- Eighty percent of all FB esophagus occur in children, with a peak incidence in the age group of 6 months to 3 years.
- The most common site for lodgment of an ingested FB is the cricopharynx.
- X-ray neck, AP and lateral view, is most commonly done for diagnosis.
- Esophageal FB can damage the esophagus leading to perforations and strictures. Apart from eroding into the trachea, the object can erode into the aorta, leading to exsanguinations and death. Other serious complications reported after FB ingestion include abscess formation and even sudden death.
- Plan of anesthesia: GA /ETT/IPPV
- Appropriate measures to prevent post-op nausea, vomiting, measures to reduce peri-laryngeal edema (steroids/adrenaline nebulisations postop)
- Watch for stridor / upper airway obstruction / other related complications in the post-operative period.

Case of Inhaled Foreign Body

- A foreign body in the upper airways may present as an emergency acute airway obstruction.

- Obstruction of lower airways follows several days after a historyof coughing. Peanut oil is an irritant and leads to mucosal oedema and chemical pneumonitis.
- Chest radiograph shows characteristic hyperinflation during expiration, but a foreign body is often not visible.
- Treat symptoms as indicated, e.g. dehydration, pneumonia, wheeze.

Anesthesia Management

- Inhalational induction is usual to avoid displacing the object further. Use 100% O2 with sevoflurane.
- Deep inhalational maintenance with sevoflurane. TIVA is becoming a more popular technique, usually supplementing the volatile agent.
- Apply topical anesthesia to the vocal cords (4% lidocaine, up to 3mg/ kg), and consider a drying agent (atropine 20 micrograms/kg IM 30min preoperatively or 10 micrograms/kg IV at induction, or glycopyrronium 5 micrograms/kg IM or IV).
- Prior to bronchoscopy, maintain the airway with a face mask or LMA.
- Rigid bronchoscopy: the Storz bronchoscope has an attachment for a T-piece. Check compatibility before the procedure.
- For foreign objects in the upper airways, maintain SV.
- If the foreign body is in the lower airway, then IPPV with a muscle relaxant is acceptable, since the object will be pushed distally by the bronchoscope until it can be grasped by forceps. Give assisted ventilation via a T-piece or high-frequency jet ventilation. Intubation will then be required once the scope is removed.

Post-operative

- If bronchoscopy is traumatic, give dexamethasone 0.25mg/kg IV, then two doses 8-hourly of 0.125mg/kg.
- Consider physiotherapy, bronchodilators, and antibiotics, as indicated.

Special considerations

- If tracheal/ball–valve obstruction suspected, IPPV is contraindicated.
- Intubation may assist lung ventilation and sizing of the bronchoscope if a tracheal foreign body is excluded.

Case 8: Pericardial effusion

Pericardial effusion. Whenever we encounter a large heart figure, we should always be aware of the possibility of pericardial effusion simulating a large heart. Especially in patients who had recent cardiac surgery an enlargement of the heart figure can indicate pericardial bleeding.

Cardiac tamponade is a life-threatening emergency. Cardiac tamponade is defined as an accumulation of fluid in the pericardial sac, creating an increased pressure within the pericardial space that impairs the ability of the heart to fill and to pump. As the pump function of the heart becomes impaired then there is a fall in cardiac output and systemic perfusion leading to life-threatening organ dysfunction. Clot or tumour compressing the pericardium may also have the same effect.

Beck's Triad: consists of hypotension, elevated jugular venous pressure and muffled heart sounds.

Pulsus paradoxus: an exaggerated fall in systemic arterial blood pressure during the inspiratory phase of spontaneous ventilation. This phenomenon occurs due to negative intrapleural pressures increasing venous return to the right side of the heart, resulting in a bulging of the interventricular septum towards the left heart chambers. A minor respiratory variation in venous return to the heart occurs in the normal heart, but this effect is exaggerated in cardiac tamponade.
(Note: *Reverse pulsus paradoxus* is observed to a certain degree in all sedated and mechanically ventilation patients with cyclical variation in pulse pressure used as an indicator of the fluid responsiveness of a patient.)

Echocardiography is the single most important investigation for cardiac tamponade. A continuous pericardial effusion produces a classical pattern, recognised as a "swinging heart". The heart oscillates from side to side within the pericardium, most impressively seen with large effusions, also changing the anatomical relationship between the heart and ECG electrodes with a resultant effect on the QRS complex (the cause of electrical alternans).

Treatment involves careful fluid resuscitation and inotropes, but this is not a substitute for definitive drainage via either percutaneous or open surgical techniques.

Anesthetic technique
All patients require adequate peripheral access and standard ASA monitoring prior to any anesthetic agent being administered.
Depends on the clinical condition and hemodynamic stability of the patient, any concomitant co-morbidities, the etiology of the effusion and the procedure being performed
Local anesthetic infiltration with supplemental sedation using ketamine, midazolam or fentanyl may be sufficient for pericardiocentesis and subxiphoid windows in the less stable patients who are co- operative.
If general anesthetic is required, hemodynamic goals remain the same:

maintain and augment preload
maintain afterload
maintain and augment contractility
maintain heart rate
maintain sinus rhythm to preserve atrial contribution to ventricular filling

<u>Induction</u>

- Avoid respiratory depressant drugs and positive pressure ventilation if possible.
- spontaneous respiration with a volatile agent is ideal if tolerated.
- use infusion of vasopressor to maintain blood pressures while ensuring adequate depth of anesthesia with volatile agent before manipulation of the airway.
- IV induction can be used for stable patients with no evidence of tamponade.
- surgical preparation and draping to facilitate emergency drainage is advisable if IV induction is to be used.
- ketamine and etomidate are recommended as they will have the least vasodilatory effects.

If endotracheal intubation and positive pressure ventilation is required

- use the lowest possible inspiratory pressures and PEEP to maintain minute volume and oxygenation
- choice of ETT will depend on the procedure: one lung ventilation may be needed to facilitate surgery in thoracotomy and VATS
- the time taken to insert a double lumen ETT may be detrimental in an unstable patient
- placing a single lumen ETT with a bronchial blocker may be advisable
- if the patient is very unstable, performing a subxiphoid window initially to relieve the tamponade and then performing the general anesthetic is another option
- Inotropic and vasopressor support should be anticipated and used as needed to maintain hemodynamic goals at induction of anesthesia.

Maintenance

- IV opioids, propofol, ketamine and volatile agents can all be used if they are tolerated
- Muscle relaxants should only be used once the patient can tolerate positive pressure ventilation
- Continuous infusions of inotropes and pressors may be needed and require continuation depending on the case.
- Intra-operative arrhythmias are common because of surgical handling of pericardium and heart and should be anticipated
- defibrillator and anti-arrhythmic drugs should be immediately available.
- Because intrinsic myocardial function is preserved in pericardial effusion and tamponade, once the tamponade is relieved, there is usually a dramatic improvement in hemodynamics and the patients do well post-operatively.
- Hypertension may present after drainage, this should be anticipated and controlled with IV agents especially in patients where increases in blood pressure will worsen intraoperative bleeding or outcome, e.g. chest trauma, coronary or myocardial perforation or aortic dissection.
- Occasionally, following drainage of large, chronic effusions, patients develop pulmonary oedema and global LV dysfunction.

Postoperative

- Patients should be transferred to a high dependency unit or ICU
- Ongoing care is necessary to monitor for recurrence of tamponade, ongoing bleeding and continuation of inotropic and vasopressor support
- Extubation will depend on the patient's pre-op condition and intraoperative course.

Note: ***Kussmaul's sign*** - increase in jugular venous pressure on inspiration (Constrictive pericarditis)

- increase in right atrial preload mediated by an increase in intra-abdominal pressure during inspiration independent of intrathoracic pressure changes
- increase in preload cannot be accommodated because of the fixed pericardial constriction which is detected clinically as increased pressure in the jugular vein.

Note: ***Friedreich's sign***: A prominent y-descent of the jugular venous pressure reflects the predominance of right ventricular filling in early diastole that is seen with constrictive pericarditis.

Case 10: Gas under the diaphragm

Perforation peritonitis is a frequently encountered surgical emergency in tropical countries like India, most commonly affecting young men in their prime of life. Imaging studies like X-ray Chest or abdomen in the upright position will reveal gas under the diaphragm. Paralytic ileus is characterized by marked distension of small gut. If the patient is too sick for an X-ray in the erect posture, then a left lateral decubitus X-ray of the abdomen is of help. It may show the presence of free air between the liver margin and the abdominal wall.

Prediction of prognosis:

The **Boey score** encompasses three factors – major medical illness, preoperative shock and longstanding perforation <24 h. The mortality rate increases progressively with an increasing number of risk factors: 0, 10, 45 and 100% in patients with none, one, two and all the three risk factors respectively.

Manheim Peritonitis Index (MPI): has eight parameters and a score ranging from 0 to 57. Patients with a score of >21 have a reportedly low mortality (0-23%) as compared to those with a score <29 (100%).

Management

- Quick restoration of the circulatory hemodynamics followed promptly by surgery
- Use of appropriate antibiotics
- Critical Care and support of different organ systems
- Maintenance of nutrition.

General anesthesia with endotracheal intubation and controlled ventilation is the technique of choice. De-nitrogenation of the lungs, breathing 100% oxygen through a face mask should be considered before induction of anesthesia. A rapid sequence induction and intubation using succinylcholine to facilitate tracheal intubation may be required. If the patient is having hyperkalemia or any other contraindication to succinylcholine, rocuronium can be employed for facilitating neuromuscular relaxation.

Options for induction drugs include ketamine, etomidate, slow administration of propofol, or titrated doses of thiopentone sodium.

Continued volume resuscitation and vasopressor infusions are helpful to counteract the hypotensive effect of anesthetic agent and positive pressure ventilation.

For maintenance of anesthesia, either inhalational agents or intravenous agents may be used with opioids. During surgery the hemodynamic state may be further complicated by blood loss or systemic release of bacteria and endotoxins. Intravascular volume resuscitation should be continued throughout the surgical procedure. Intraoperative CVP values may be increased by raised intra-thoracic and intra-abdominal pressure. Throughout the surgical procedure, cardiovascular parameters (heart rate, cardiac filling pressure, inotropic state, systemic arterial pressure) can be adjusted to optimize tissue oxygen delivery. Intraoperative hypothermia should be avoided as it has been found to be associated with impaired platelet and coagulation factor dysfunction.

Role of neuraxial blockade: Sepsis is considered to be a relative contraindication to regional neuraxial blockade. It should be undertaken with caution; since the hemodynamic effects of these techniques in the setting of sepsis can induce cardiovascular compromise which may be difficult to reverse. Recent blood tests confirming normal coagulation are

required. Epidural blockade has also been used in these patients, but is better to be used as adjunct rather than sole anesthetic.

In all critically ill patients, analgesia, sedation and mechanical ventilation are maintained at the conclusion of surgery. Stress ulcer and deep vein thrombosis prophylaxis are also recommended. Nutrition is one of the cornerstones of management in these patients. Though enteral route should be started as soon as feasible, parenteral nutrition should be considered if there is surgical contraindication for enteral administration.

Acute renal failure occurs in 23% of patients with severe sepsis. Renal replacement therapy may be initiated to correct acidosis, hyperkalemia or fluid overload. Continuous renal replacement therapy (CRRT) and sustained low efficiency dialysis (SLED) can be considered in hemodynamically unstable patients.