RESPIRATORY SYSTEM

Points to examine in respiratory system:

1. Movement of chest.
2. Mediastinal shifting.
3. Percussion.
4. Auscultation:
   a. Breath sounds:
      - Vesicular breath sound (VBS).
      - Bronchial breath sound (BBS).
   b. Vocal resonance.
   c. Any adventitious/ added sound(s).

Findings of respiratory system in some common diseases:

Condition	Mediastinal shifting	Percussion	Breath sound	VR/ VF	Added sound(s)
Pleural effusion	Opposite side	Dull	VBS↓	↓	-
Pneumothorax	If at all, to opposite	Tympanitic	VBS↓	↓	-
COPD	-	Hyper-resonant (if hyperinflation present)	VBS↓	↓↓	Rhonchi (due to airway narrowing)
Asthma	-	Normal	Normal/ VBS↓	Normal/ ↓↓	Rhonchi
Consolidation	-	Impaired/ dull	BBS	↑	Crepts (wet sound)
Collapse	Same side	Impaired/ dull	BBS	↑	-
Fibrosis	Same side	Impaired/ dull	VBS↓	↓↓	Crepts

It is defined as accumulation of abnormal amount of fluid in the pleural space.

Types and causes:

1. ***Transudative pleural effusion:***
   a. LHF/ CCF.
   b. Chronic liver disease.
   c. Nephrotic syndrome.
2. ***Exudative pleural effusion:***
   a. Infection in the pleural space:
      - TB.
   b. Malignant effusion:
      - Bronchogenic CA.
      - Breast CA.
      - Lymphoma.
   c. Autoimmune/ connective tissue disorder:
      - Rheumatoid arthritis.
      - SLE.
   d. Acute pancreatitis.
   e. Pulmonary embolism/ infarction.

Clinical features:

Due to effusion	Due to underlying etiology
• Chest discomfort/ dull ache in the affected side. • Shortness of breath (SOB). • Sharp pleuritic chest pain (if there is an underlying acute pleurisy).	1. LHF: SOB, PND, Orthopnea. 2. Chronic liver disease: Abdominal swelling, GI bleeding. 3. Nephrotic syndrome: Generalized swelling. 4. Bacterial infection: Fever, cough, dyspnea. 5. TB: Weight loss, loss of appetite, low grade fever, haemoptysis. 6. Malignancy: Rapid weight loss, loss of appetite. A lump with features of primary malignancy. 7. Connective tissue disorders: a. Red eye/ gritty eye. b. SOB. c. Skin rash. d. Arthralgia. e. Hematuria.

Signs due to effusion:

Inspection	Affected side: • Movement restricted. • Bulging.
Palpation	• Expansion restricted. • Shift: To the opposite side, in case of massive effusion.
Percussion	At and below the level of effusion: Dull.
Auscultation	• VBS↓. • VR/VF ↓.

Signs due to underlying etiology:

Following findings may give clue about underlying etiology:

Findings	Underlying etiology
Clubbing	• Bronchogenic CA.
Lymphadenopathy	• TB.
JVP↑	• CCF (Pulsatile). • SVC obstruction (Non-pulsatile). • Malignancy (Breast CA, Lymphoma).
Jaundice	• Chronic liver disease. • Metastatic disease of liver.
Cardiovascular signs of LVF may be present.	
GI signs of chronic liver disease may be present.	
Breast examination may show the lump.	

Investigation

1. Blood: Hb/ TC/ DC/ CRP or ESR.
2. Urea-creatinine Na+ K+.
3. Liver function test.
4. Chest X Ray:
   It helps to diagnose effusion and in many cases, the underlying etiology.
5. Diagnostic aspiration of pleural fluid.
   a. Physical character:
      - Hemorrhagic: Malignant cause.
      - Turbid: Infection.

www.FirstRanker.com www.FirstRanker.com

b. Chemical character:
   - Paired serum sample for protein and LDH:

     Diagnostic criteria:
     - Exudative (if it fulfils any 1 criteria):
       - $Fluid\ protein / Serum\ protein > 0.5$
       - $Fluid\ LDH / Serum\ LDH > 0.6$
       - $Fluid\ LDH > \frac{2}{3}rd\ of\ normal\ upper\ limit\ of\ serum\ LDH.$
     - Transudative: If it doesn't fulfil any criteria.

c. Microbiological character:
   - Gram stain, culture sensitivity.
   - AFB, Mycobacterial culture: Diagnostic yield is very low.

d. Cytological character:
   - Abnormal/ malignant cells: In malignant effusion.
   - Polymorphonuclear predominant: Polymorph. effusion.
   - Predominant lymphocytic: TB, Malignant effusion.

e. Markers of TB:
   - Adenosine Deaminase (ADA) level.
   - Interferon-γ level.

6. Pleural biopsy:
   Either under radiological evidence/ thoracoscopically.
7. Other relevant investigation(s):
   Depending on the underlying etiology.

Treatment

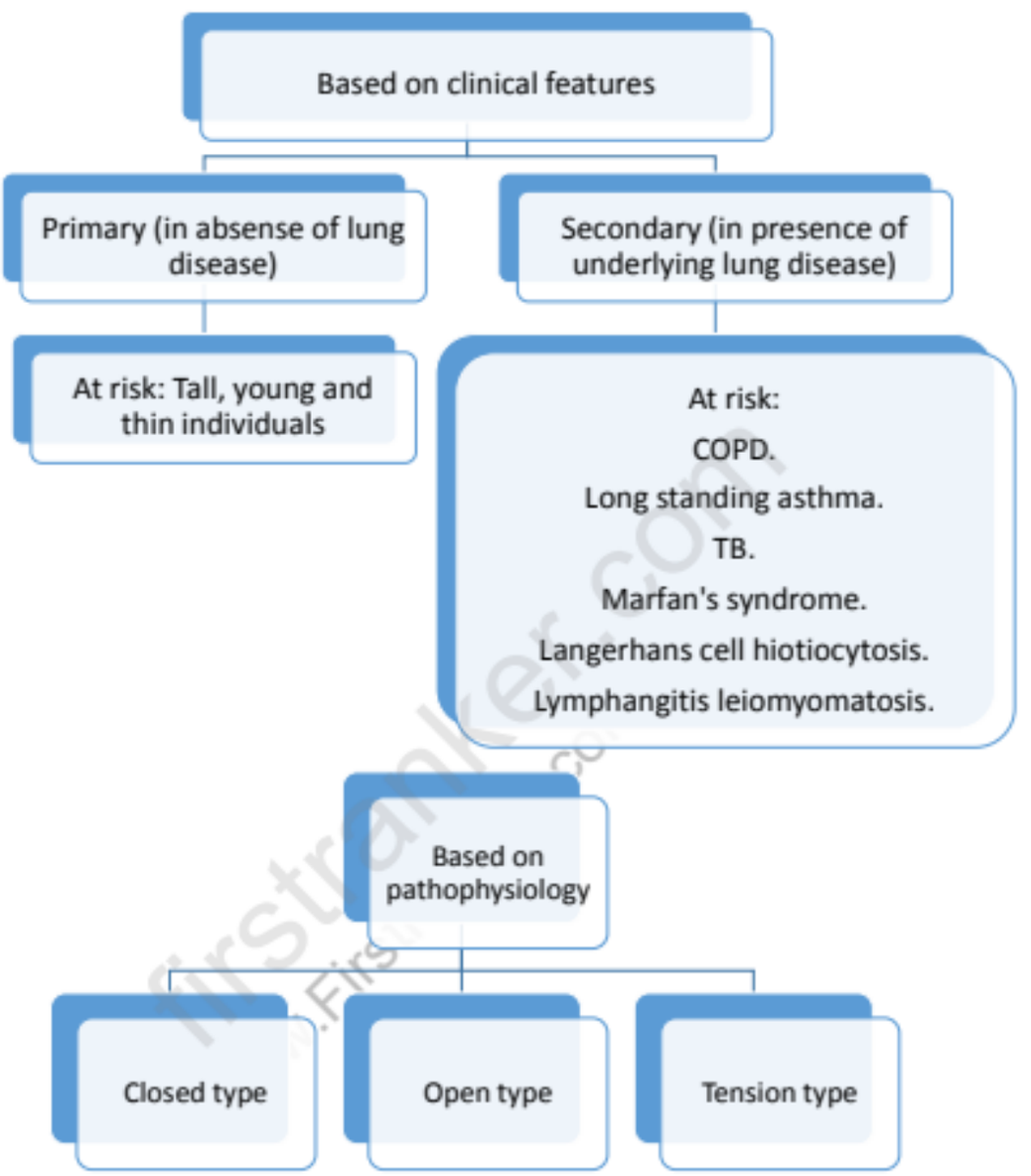
1. Treatment of effusion:
   a. If it is likely to be a Transudative effusion, then usually treating the cause is sufficient to get rid of the effusion. In most cases, effusion doesn't require drainage.
   b. In unilateral Exudative effusion, drainage of fluid by inserting an intercostal chest drain becomes essential under following circumstances:
      - Symptomatic effusion.
      - Moderate to severe effusion even it is asymptomatic.
      - Empyema.

c. In case of recurrent effusion:

- Pleurodesis can be attempted where adhesion is induced between visceral and parietal pleura by some sclerosing agent like TALK/ Bleomycin/ Tetracycline/ Betadine.
- Insertion of a long term indwelling pleural catheter.
- Treating the underlying cause.

It is defined as presence of air in the pleural space.

Types:



Closed type:

Air starts accumulating, but after a certain time, the punctured area of lung gets sealed off on its own.

Open type:

Air freely flows in and out of pleura during inspiration and expiration, respectively.

Here a pulmonary parenchymal flap acts as a one way valve, allowing air to move in during inspiration but does not allowing it to come out during expiration, leading to rapidly progressing accumulation of air, leading to compressive effect (tamponade effect) on heart and hemodynamic instability (reduced cardiac output).

Clinical features:

Symptoms

1. Dyspnea:
   It may be mild/ severe/ rapidly progressing, depending on the amount of air in the pleural space. At times, it is precipitated following a bout of severe cough.
2. Chest pain:
   A momentary sharp chest pain may occur (due to tear of the underlying lung tissue).
3. Sudden collapse/ blackout: In tension penumothorax.
4. History suggestive of underlying lung disease may be present.

Signs

1. Tachypnea.
2. Tachycardia.
3. Pulse oxymetry: Low SpO2 (where normal oxygen saturation is 95-96%).
4. Cyanosis: In case of severe hypoxia.
5. Hemodynamic instability: Suggestive of tension pneumothorax.

Chest examination

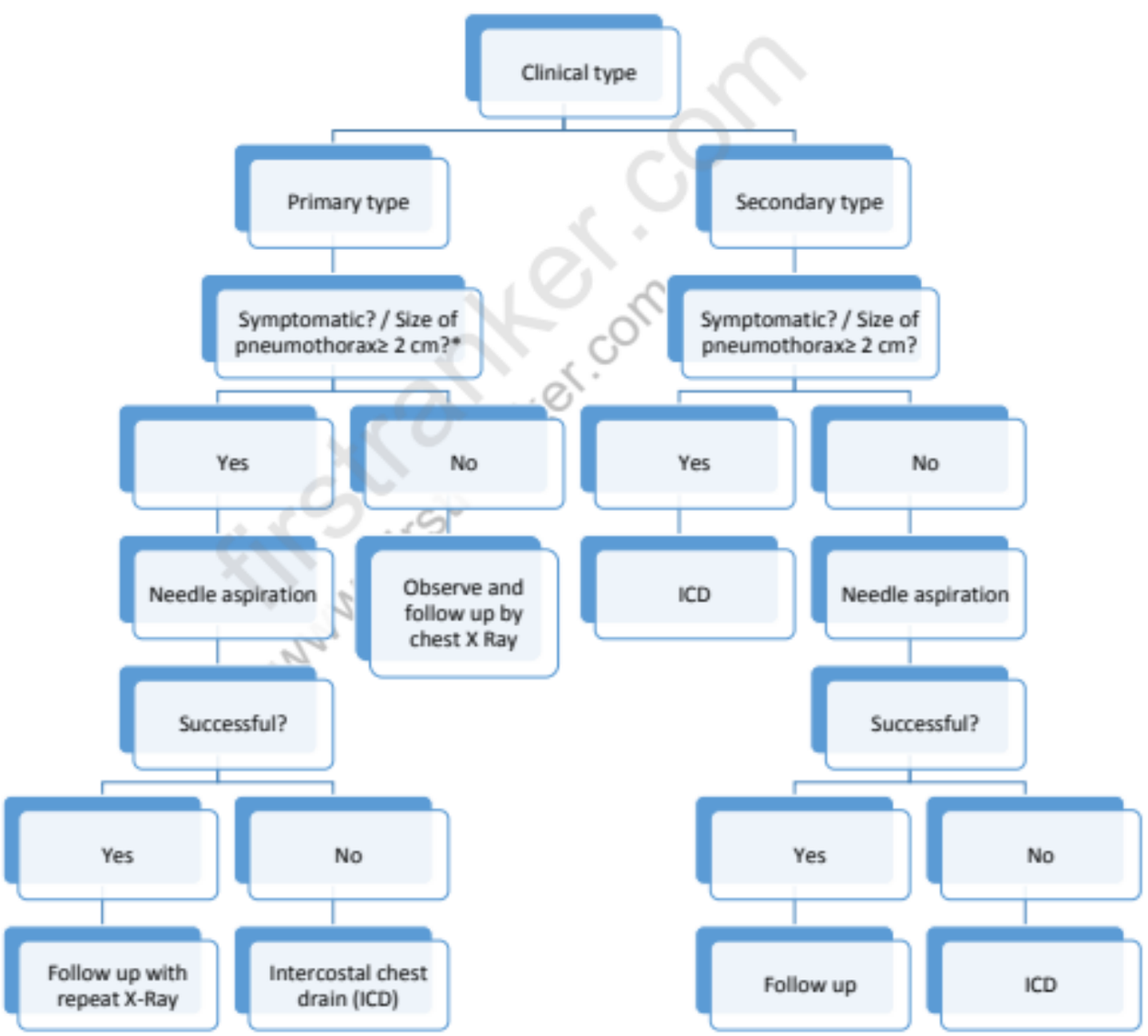
Inspection	Movement is restricted on the affected side.
Palpation	Mediastinal shifting towards the opposite side (usually in case of tension pneumothorax).
Percussion	Tympanitic.
Auscultation	Reduced VBS/ VR/ VF.
Added sound	1. Pneumothorax click: A clicking sound coinciding with each cardiac cycle due to movement of pleura against the surface of heart (in case of left sided pneumothorax only). 2. Coin percussion: A metallic sound is heard when the patient is

	asked to percuss a coin against another and the stetho is placed at a diametrically opposite point.

Investigation

1. Chest X Ray (CXR):
   a. Confirms diagnosis.
   b. An area of lung not traversed by bronchovascular margins.
   c. Collapsed border: Visible.
   d. Presence/ absence of tracheal shifting.
2. Other relevant investigations to assess the underlying disease.

Treatment protocol



Size of pneumothorax: Distance between rib margin and collapsed lung border at the level of hilum.

If it is ≥2 cm, it is suggestive of pneumothorax of 50% pleural space.

Indications of cardiothoracic referral:

1. Persistent air leak (bubbling> 5 days).
2. Second episode of ipsilateral pneumothorax.
3. First episode of contralateral pneumothorax.

Post-discharge advice:

1. Avoid air travel for 4-6 weeks.
2. Refrain from deep sea diving for rest of life.

It is defined as occlusion of pulmonary artery/ its branches by a variety of substances.

Types:

Air embolism

Amniotic fluid embolism

Blood clot/ thromboembolism

Cancer cells (RCC)

Fat embolism

Foreign body (in IV drug abusers).

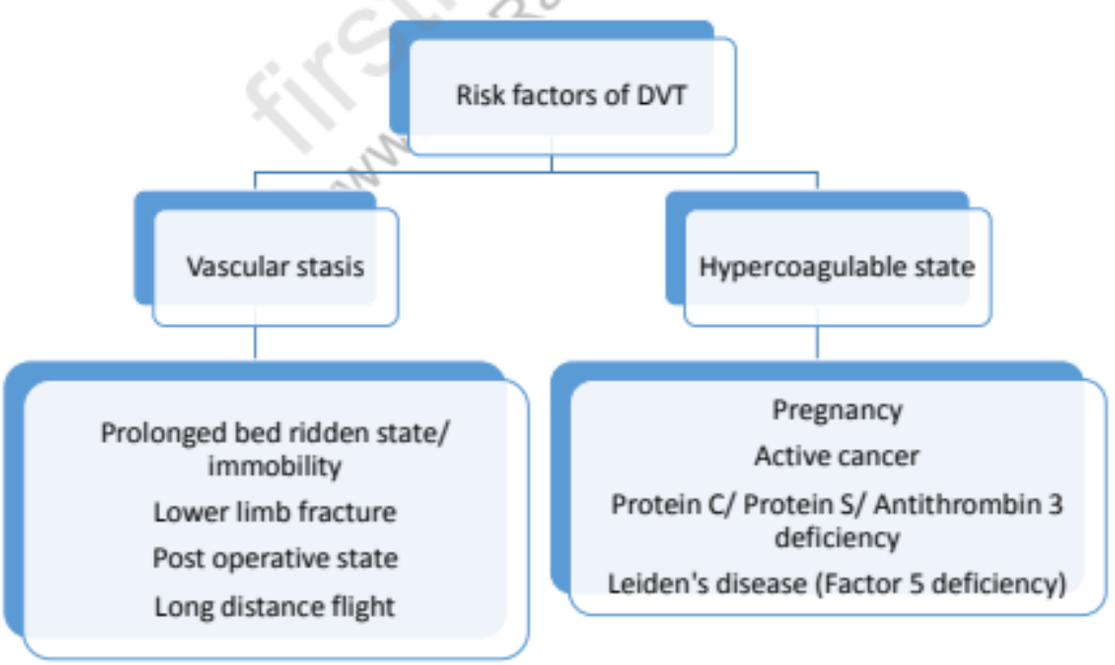
Pulmonary thromboembolism

It is defined as occlusion of pulmonary artery/ its branches by a thrombus which usually gets dislodged from a deep vein of lower limb.

(It should be noted that venous thrombosis of upper limb is rare.)

Risk factors of PE:

These are actually the risk factors of lower limb deep vein thrombosis (DVT).



Symptoms

1. Dyspnea: Sudden onset. Severity depends on size of thrombus.
2. Pleuritic chest pain: Sudden onset. It occurs due to spread of inflammation to the adjacent pleura.
3. Hemoptysis: Small amount of hemoptysis may occur.
4. Sudden collapse: In case of a massive PE occluding pulmonary trunk/ one of the major branches of pulmonary artery.
5. Patient may have pain and swelling in one of the lower limb (due to DVT).
6. You should always look for symptoms of the underlying factor(s).

Signs

1. Tachypnea.
2. Tachycardia.
3. Pulse oxymetry: Low oxygen saturation.
4. Hemodynamically unstable: In case of a massive PE.
5. Lower limb examination: May show signs of DVT.
6. Respiratory system: Often normal/ unremarkable on examination.

Investigation

1. *Estimation of D-Dimer:* It is a fibrin degradation product (FDP) which gets released into bloodstream due to *ineffective fibrinolysis*. It is a very good screening test as a negative result virtually excludes PE/DVT but a positive result does not always confirm the diagnosis as it may also rise in other conditions (like repeated venepuncture/ septicaemia/ DIC etc.).
2. *V/Q Scan:* It shows the area of ventilation-perfusion mismatch. It may be false +Ve in pre-existing lung diseases which can also cause V/Q mismatch.
3. *CT-Pulmonary angiogram:* Best test for diagnosing PE.

Associated investigations:

1. Chest X Ray.
2. ECG.
3. Echocardiogram: To assess RV dysfunction.
4. Relevant investigations: If a hypercoagulable state is suspected.

1. Anticoagulation:
   Initially heparin (Unfractionated/ Low molecular weight/ Fondaparinux) + Warfarin till target INR is achieved.
   Note:
   The therapeutic range for oral anticoagulant therapy is defined in terms of an international normalized ratio (INR).

$$INR = \frac{Patient\ prothrombin\ time}{Mean\ of\ normal\ prothrombin\ time}$$

   The target INR should be 2-2.5 in case of PE/ DVT.
   Duration of therapy:
   It depends on the underlying risk factors.
2. Thrombolysis:
   It is usually done in patients of PE who are hemodynamically unstable.
   Alteplase is the drug of choice.
3. In selected group of patients, insertion of IVC filter may be attempted.

It is defined as chronic infection of airways leading to abnormal, permanent dilation of bronchi/ bronchioles.

Causes

1. Post infective causes:
   a. TB.
   b. Necrotizing pneumonia.
   c. Whooping cough pneumonia.
2. Immune related:
   a. Immunodeficiency: Primary and secondary.
   b. Hyperimmune reaction (Allergic bronchopulmonary aspergillosis).
3. Impaired mucociliary clearance:
   a. Cystic fibrosis.
   b. Primary ciliary dyskinesia.
   c. Kartagener's syndrome.
4. Insult to the bronchus:
   a. Obstruction (Foreign body/ Lymph node etc.).
   b. Repeated gastric aspiration.
   c. Toxic fumes.

Clinical features:

1. Chronic productive cough: often mucopurulent/ purulent; may be foul smelling. Volume and appearance of cough suddenly changes during infective spells/ exacerbations.
2. Breathlessness.
3. Hemoptysis: It may be massive (due to rupture of bronchial artery as well as erosion of bronchial wall).

Signs:

1. Pulse oximetry: Low oxygen saturation.
2. Clubbing may be present.
3. Respiratory system:
   Often coarse crepitations are present, which may be diffuse/ localized depending upon the extent of distribution of bronchiectasis.

Initial investigations

1. Hb, TC, DC, CRP/ ESR.
2. Sputum: Gram stain and culture sensitivity.
3. Blood culture: During infective exacerbations.
4. CXR: May show certain changes suggestive of bronchiectasis.
5. High resolution CT scan (HRCT): Confirms the diagnosis.

Special investigations

The nature of special investigation(s) depends on the underlying disease.

Treatment

1. Antibiotic therapy:
   It is of 2 types:
   a. Short term (during exacerbation).
   b. Long term (for prophylaxis).
2. Bronchodilators (in case of airway obstruction).
3. Clearance of airway secretion:

   Bronchial toileting

   a. Postural drainage.
   b. Chest physiotherapy.
   c. Special breathing exercise.
   d. Cough assist device.
4. Surgery: Removal of the bronchiectatic area.
5. Vaccine:
   a. Influenza vaccine: Yearly.
   b. Pneumococcal vaccine: Single dose.

COPD (Chronic obstructive pulmonary disease)

It is defined as a chronic progressive disease of the airway characterized by *fixed irreversible airway obstruction* with/ without alveolar damage.

Etiology:

Cigarette smoking.

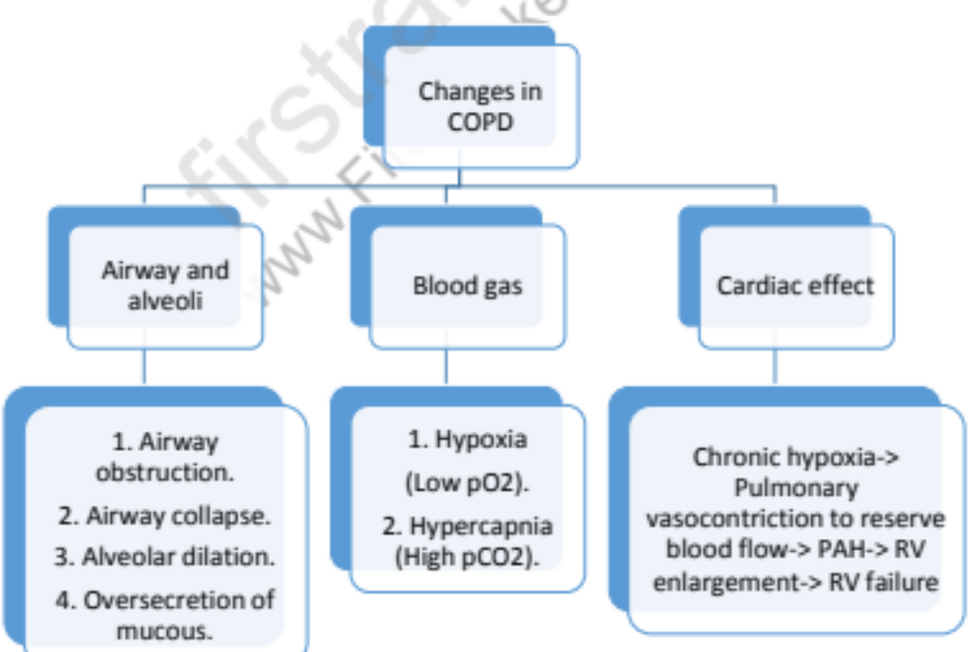
Contributory factors:

- Outdoor air pollutant.
- Occupational pollutant.
- Indoor air pollutant (biomass fuel).

Types:

1. *Chronic bronchitis:*
   Pathologically characterized by *over-secretion of bronchial mucous* which manifests as *chronic productive cough* lasting most of the days for at least 3 months in a year for at least 2 consecutive years.
2. *Emphysema:*
   Pathologically characterized by *abnormal permanent dilation of alveoli* with *destruction of their walls* without obvious fibrosis.

Pathophysiological changes:



- In some patients with COPD, there is a tendency to retain CO2 as they hypoventilate. In these patients, chronically elevated CO2 is no longer the main stimulus of respiratory centre. *In this state, hypoxia becomes the principal driving force of respiration.* Any attempt to completely correct this hypoxia will therefore take away the main respiratory driving force.
- In the other group of COPD patients, usually retention of CO2 doesn't occur.

Symptoms:

In chronic stable COPD patients, the main symptoms are:

- Breathlessness:
  Chronic, slowly progressive; leading to reduced exercise tolerance. There is no diurnal variation/ no seasonal variation (Asthma) and no orthopnea/ PND (CHF).
- Chronic cough:
  Often productive; mucoid/ mucopurulent. May be dry also.

Volume of this cough increases and becomes more purulent during infective exacerbation.

- Swelling: When RVF develops.

Signs

1. Tachypnea +/- Tachycardia.
2. Pulse oxymetry: Reduced oxygen saturation. If hypoxia is severe, then cyanosis may be evident.
3. Raised JVP with edema (in RVF).
4. Respiratory system:
   a. Inspection: Rate of respiration increased. Expansion of chest may be restricted.
   b. Palpation: No shifting.
   c. Percussion: Hyper-resonant at both sides.
   d. Auscultation:
      - Signs of reduced air entry (Reduced VBS/ VR/ VF).
      - Added sounds:

- ✓ Rhonchi/ wheeze may be present (due to bronchospasm).

- ✓ A localized area with crepitation may be present (due to infection of underlying parenchyma).

5. CVS:
   a. Signs of PAH: Accentuated P2 +/- Palpable P2. Mid-systolic murmur due to functional pulmonary stenosis.
   b. Signs of RV enlargement: Left parasternal heave. Apical impulse shifted outwards.

Investigation

1. Spirometry:
   a. $\frac{FEV1}{FVC} < 0.7.$
   b. Pre and post-bronchodilator challenge FEV1: No evidence of reversibility.
   c. Classification of COPD:

Severity	FEV1*
Mild	>80%
Moderate	50-79%
Severe	30-49%
Very severe	<30%

   [* These FEV1 values are used to classify COPD in a background of FEV1/FVC value of <0.7.]
2. CXR:
   a. Prominent bronchovascular markings.
   b. Hyperinflated lung.
   c. Always look for any pneumothorax.
3. Blood:
   a. Polycythemia (in response to hypoxia, there is an increased erythropoietin production, leading to polycythemia).
   b. Arterial blood gas (ABG): To document the baseline gas status.
4. Sputum: Gram stain+ culture sensitivity.

Treatment

In a patient of chronic stable COPD

Principles of treatment:

1. Smoking cessation.

2. Pulmonary rehabilitation.
3. Pharmacotherapy.
4. Long term oxygen therapy.
5. Surgery.
6. Vaccination.

Smoking cessation

- Nicotine replacement therapy (in different forms).
- Drugs (Bupropion/ Varinicline/ Nortryptiline).

Pulmonary rehabilitation

- Special breathing techniques.
- Graded physical exercises.

Pharmacotherapy

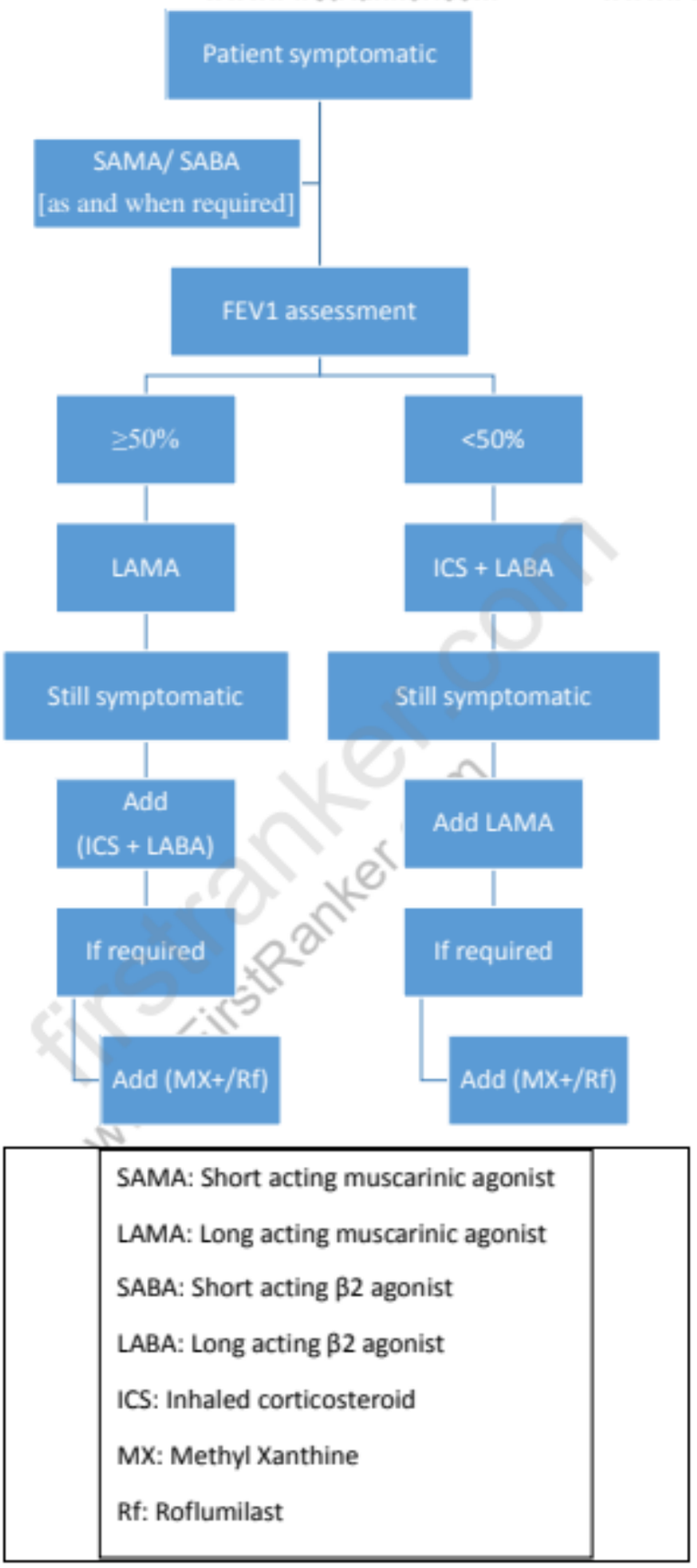
Inhalers:

- β2-agonist:
  - Short acting: Levo-salbutamol.
  - Long acting: Salmeterol/ Formoterol.
- Corticosteroid: Fluticasone/ Beclomethasone/ Budesonide.
- Anticholinergic (Muscarinic agonist):
  - Short acting: Ipratropium.
  - Long acting: Tiotropium.

Oral:

- Methyl xanthine: Aminophylline/ Theophylline.
- PDE inhibitor: Roflumilast.

Stepwise treatment approach



SAMA: Short acting muscarinic agonist

LAMA: Long acting muscarinic agonist

SABA: Short acting β2 agonist

LABA: Long acting β2 agonist

ICS: Inhaled corticosteroid

MX: Methyl Xanthine

Rf: Roflumilast

Criteria:

1. Arterial blood gas:
   paO_2< 50 mm Hg, when done in a stable state and at least twice, at least 3 weeks apart.
   (Normal value: 95-100 mm Hg)
2. Arterial blood gas:
   paO_2: (55-60) mm Hg, with evidence of (PAH/ RVF/ Polycythemia/ Any hypoxic condition).

Duration of long term oxygen therapy: At least 15 hours a day.

Surgery

Various options are:

1. Bullectomy
2. Lung volume reduction surgery.

Vaccination

1. Influenza vaccine: Yearly.
2. Pneumococcal vaccine: Single dose + Booster after 5 years.

Acute exacerbation of COPD

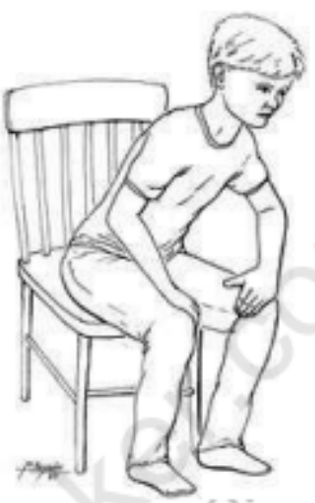
It is an acute emergency characterized by progressive worsening of COPD symptoms and commonly precipitated by an underlying respiratory infection.

Symptoms

1. Worsening dyspnoea
2. Productive cough: Often increased in volume and more purulent in appearance (in comparison to regular sputum).
3. In some patients, symptoms of CO_2 retention (CO_2 Narcosis) may occur. [CO_2 Narcosis→ Metabolic encephalopathy→ Hepatic/ uremic in nature→ Confusion/ Convulsion/ Coma/ Delirium].

Signs

1. Tachypnoea
2. Tachycardia
3. Pulse oximetry: Low SpO_2 (±Cyanosis)
4. Attitude:
   Patient sits up in a ***tripod position*** with outstretched hand supporting upper part of the body and breaths ***pursed lip.*** It is an attempt to prevent the collapsibility of the airway by increasing intra-airway pressure.



5. Flapping tremor may be present in CO_2 narcosis.

Respiratory system

1. Signs of COPD are present
2. Widespread rhonchi and localized crepts may be present.

Investigation

Same as COPD.

Treatment

1. *Airway protection:*
   Frequent oropharyngeal suction, intubation if required.
2. *Breathing:*
   a. Free flow oxygen
   b. Controlled oxygen: Ideally via venture mask
   c. During acute stage, a target saturation of 88-92% should be maintained, particularly if pCO_2 level is high; so that the hypoxia is not overcorrected.

www.FirstRanker.com www.FirstRanker.com

c. Assisted ventilation may be required (non-invasive/ invasive).

3. *Circulatory support with IV fluids.*
4. *Drugs:*

Antibiotics

Oral/ IV, depending on patient's ability to swallow a tablet and severity of infection, usually a 7-10 days course is given. The antibiotics routinely administered in acute exacerbation of COPD are:

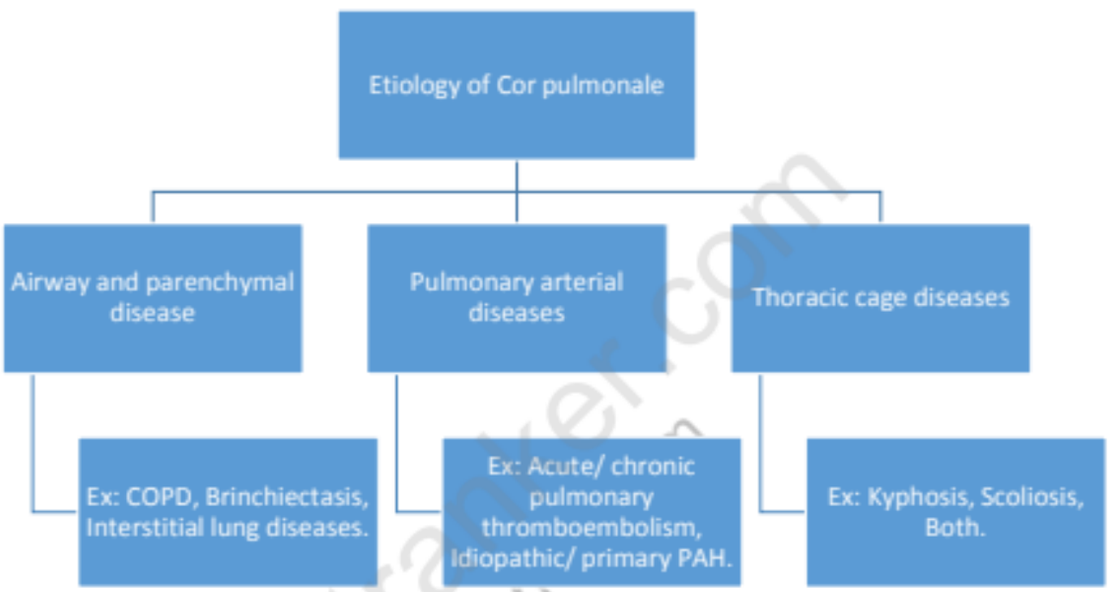
Drugs	Description
Aminophylline infusion	Usually given when in spite of maximum medical therapy, patient remains significantly symptomatic. Maximum for 24-48 hours.
Bronchodilator (Nebulization with Salbutamol + Budesonide+ Ipratropium)	Initially it may be repeated few times every 15-30 minutes till the acute stage is over and then every 4-6 hourly, till the patient is stable enough to use inhaler.
Corticosteroid (Oral/ IV short course steroid: usually Hydrocortisone is given)	It is continued for 5-7 days and is now routinely recommended in acute exacerbations of COPD.

Cor pulmonale

Introduction

It is a clinical condition characterized by right ventricular enlargement (RVE) from acute/ chronic lung pathology.

Etiology



Clinical features

1. Signs and symptoms of underlying disease
2. Symptoms and signs of PAH:
   - Symptoms:
     Exertional chest pain (also called Right ventricular angina).
   - Signs:
     - ✓ Loud P2 ± Palpable P2
     - ✓ MSM due to functional PS
     - ✓ EDM due to PR (occurs very lately).
3. ***Signs of RVE:***
   - Apex: Shifted outwards.
     As RV doesn't have an apex, in case of a right ventricular apical impulse, it is diffuse in nature.

- Left parasternal heave
- PSM due to a functional TR
- Visible/ palpable epigastric pulsation.

4. Symptoms and signs of RVF:
   - Symptoms:
     Swelling.
   - Signs:
     - ✓ Raised JVP
     - ✓ Bilateral edema
     - ✓ Soft tender hepatomegaly.

Investigation

1. To assess the underlying disease
2. To assess the RV:
   a. ECG
   b. ECHO.

Treatment

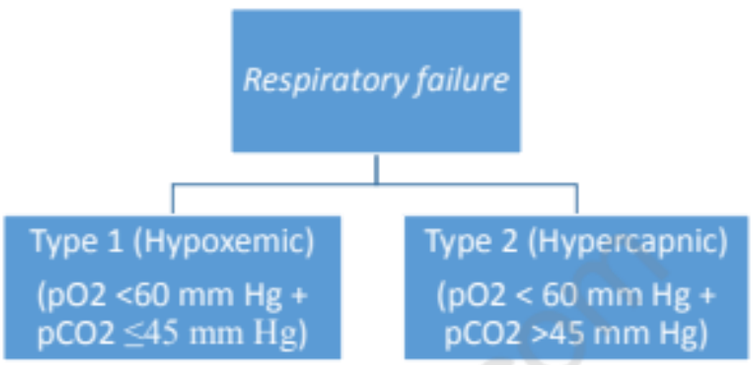
1. Treatment of the underlying disease
2. For RVF:
   a. Salt and fluid restriction
   b. Diuretics.

Respiratory failure

Introduction

It is defined as an arterial blood gas (ABG) value of <60 mm Hg.

Types



Causes

Causes of type 1 failure	Causes of type 2 failure
1. Pneumonia 2. Pulmonary edema (LVF) 3. Pulmonary embolism 4. ARDS 5. Acute asthma.	• Centre failure/ CNS diseases: 1. Brainstem depression: ✓ Drug overdose (Opiate/ barbiturate) ✓ Deep coma. 2. Brainstem structural damage: ✓ CVA ✓ Head injury ✓ Intracerebral space occupying lesion (IC-SOL). • Pump failure/ respiratory diseases: 1. COPD (Most common) 2. Neuromuscular diseases: ✓ Motor neuron disease ✓ Guillain-Barré syndrome ✓ Myasthenia gravis. 3. Chest wall diseases: ✓ Kyphosis ✓ Scoliosis ✓ Both.

Treatment

Treatment of type 1 failure	Treatment of type 2 failure
1. High flow oxygen 2. Assisted ventilation: ✓ Invasive (It breaths for the patient) ✓ Non-invasive (It gives respiratory support to the patient by recruiting more alveoli): Also called CPAP (Continuous positive airway pressure).	1. Controlled oxygen (in COPD) 2. Assisted ventilation: ✓ Invasive ✓ Non-invasive: Also called BiPAP (Bilevel positive airway pressure)

* Star topics: CPAP and BiPAP

CPAP:

The Continuous Positive Airway Pressure (CPAP) machine gives a predetermined level of pressure. It releases a gust of compressed air through a hose which is connected to the nose mask. This continuous air pressure keeps the upper airway open.

BiPAP:

The Bilevel Positive Airway Pressure (BiPAP) machine delivers two levels of pressure:

- Inspiratory Positive Airway Pressure (IPAP) is a high level of pressure, applied when the patient inhales.
- Expiratory Positive Airway Pressure (EPAP) is a low level of pressure exerted during exhalation.

Advantage of BiPAP over CPAP:

- Patients using the CPAP have to exert extra force against the air flow while exhaling, as the airway pressure remains constant.
- With the BiPAP, the airway pressure is set at high and low levels, making it easier for the patient to breath.

Bronchial asthma

Introduction

It is a chronic inflammatory disease of airway characterized by *reversible airway obstruction* due to hyperactivity of the airway.

Etiology

1. *Underlying factors:*
   Some of the individuals are genetically predisposed to develop airway hyper-reactivity/ inflammation spontaneously/ when exposed to external stimulus (allergen).
   These individuals are called *atopic*- often they will have H/O recurrent allergic rhinitis/ dermatitis/ hay fever and usually having a high serum IgE level.
2. *Triggering factors:*
   These factors quite often initiate/ precipitate an asthmatic attack. Some of the common substances are:
   a. Indoor allergen: Dust/ mites/ fungus (aspergillus)/ dog.
   b. Outdoor allergen: Dust/ smoke.
   c. Occupational: Isocyanate/ flour.
   d. Drugs: Aspirin/ β-blocker.
   e. Physical exercise.
   f. Chemical: Perfume/ tobacco smoke.

Clinical features

Symptoms

1. Breathlessness:
   a. May start gradually/ suddenly.
   b. Often intermittent, with asymptomatic spells within 2 episodes.
   c. Aggravated by exposure to allergen.
   d. Shows *diurnal variation*: increasing symptoms at early morning as *bronchial tone follows a circadian rhythm* and is maximum during these hours.
   e. Often shows *seasonal variation* (aggravates during season changes).

2. Wheeze:
   Usually intermittent and occurs along with dyspnea.
3. Cough:
   a. May be intermittent/ chronic.
   b. Usually *dry*, but may be productive, characteristically thick, white sputum which becomes purulent and increases in volume during infective exacerbations.

Sign

Examination may be entirely normal in asymptomatic intervals.

1. Tachypnoea
2. Tachycardia
3. Pulse oximetry: Low SpO_2 ± Cyanosis
4. Respiratory system:
   - Movement/ expansion may be restricted.
   - Signs of reduced air entry:
     Reduced VBS, VR, VF.
     Note: In COPD/ Asthma, often VBS will have a prolonged expiratory phase.
   - Added sound:
     Diffuse polyphonic wheeze may be present.

Investigation

Preliminary investigations:

1. Spirometry: $\frac{FEV1}{FVC} < 0.7$
2. Bronchodilator reversibility: +Ve
   Pre-bronchodilator FEV1 increases by 15%/200 ml, following bronchodilator challenge by short acting β2 agonist.
3. Chest X Ray: Often normal.
4. Blood: Hb, TC, DC, ESR/ CRP (Eosinophilia may be present).
5. Sputum: Gram stain and culture sensitivity.

Special investigations:

1. Bronchial provocation test:
   Helpful if clinically asthma is suspected but spirometry is inconclusive. Here, bronchoconstriction is provoked with certain substances like Methacholine/ Histamine/ Mannitol and degree of fall of pre-bronchoconstriction FEV1 is measured.
2. Detection of allergen by skin test.
3. Examination of antibodies against common allergens (also called precipitins) in the serum.

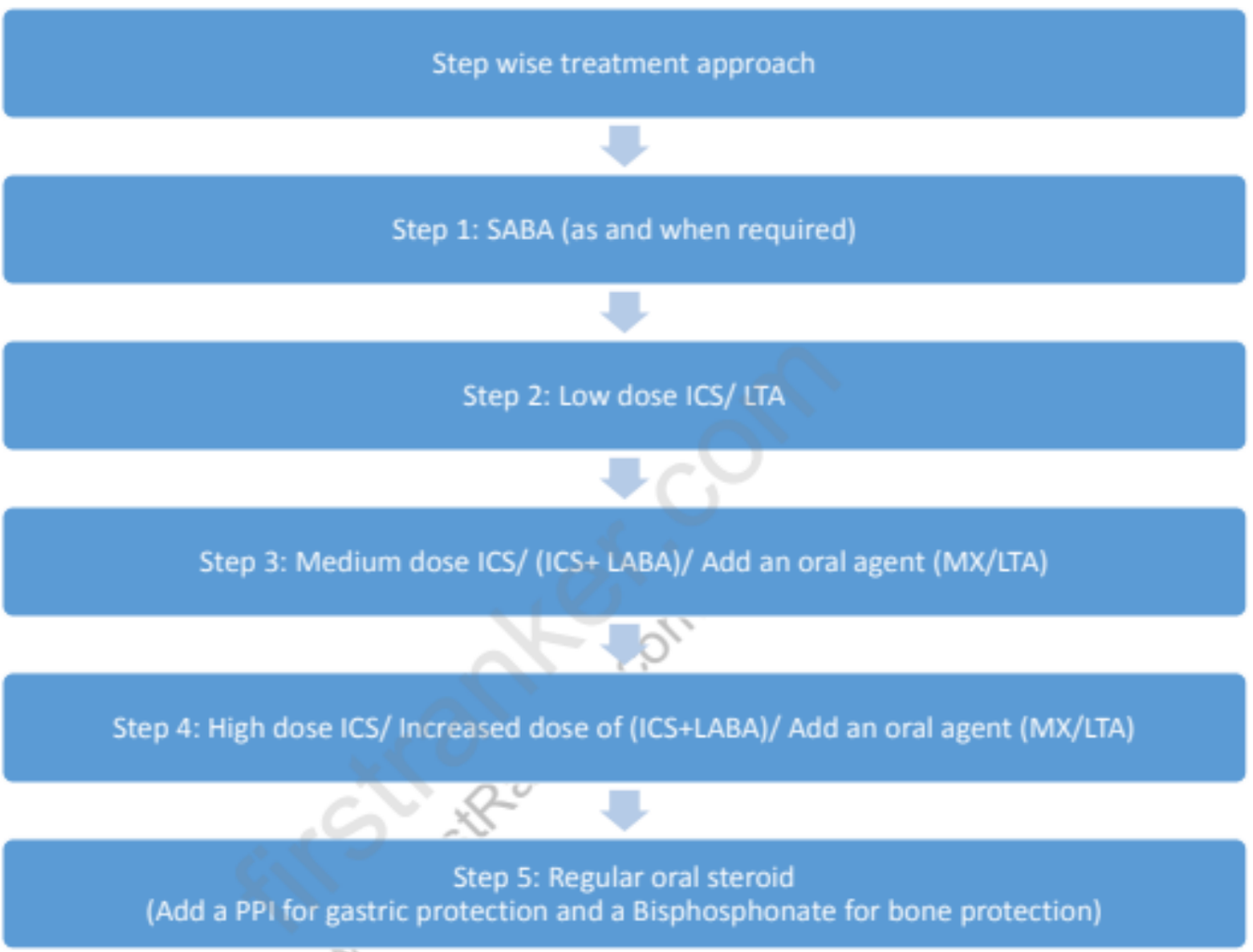
Evidence of airway inflammation:

1. Sputum eosinophil count
2. Fractional excretion of nitric oxide (NO)
3. Estimation of serum IgE.

Treatment

1. Patient education:
   - Avoid any suspected allergen (if any)
   - Protective devices (if feasible)
   - Show the technique to use inhaler.

2. Pharmacotherapy:
   - Inhaler:
     - ✓ Reliever: Short acting β2 agonist
     - ✓ Preventer: Long acting β2 agonist, Inhaler corticosteroid.
   - Oral:
     - ✓ Theophylline (Methyl Xanthine)
     - ✓ Mast cell stabilizer (Sodium cromoglycate)
     - ✓ Leukotriene antagonist (Montelukast, Zafirlukast, Zileuton)
     - ✓ Long term oral corticosteroid
     - ✓ Anti-IgE agent (Omalizumab).



[SABA/LABA: Short/long acting β2 agonist, ICS: Inhaled corticosteroid, MX: Methyl xanthine, LTA: Leukotriene antagonist]

Treatment outcomes

1. Well controlled asthma:
   It is defined as:
   I. No symptoms (or ≤2 per week)
   II. No reliever use (or ≤2 per week)
   III. No nocturnal symptoms
   IV. No restriction of activity of daily living.

2. Acute exacerbation of asthma:
   It is an acute emergency presentation of asthma with increasing symptoms often precipitated by an underlying infection. They are more frequent in patients with poorly controlled asthma.
   - Clinical features:
     1. Worsening dyspnea
     2. Wheeze
     3. Often productive cough.
   - Sign:
     1. Tachypnoea
     2. Tachycardia
     3. Low SpO_2 ± Cyanosis
     4. Signs of reduced air entry
     5. Widespread wheeze.
   - Investigation:
     1. Blood: Hb, TC, DC, CRP
     2. ABG: Usually hypoxia with type 1 respiratory failure
     3. Blood culture: If patient is febrile
     4. Sputum: Gram stain and culture sensitivity
     5. Chest X Ray
     6. ECG
     7. ECHO (In selected patients).
   - Treatment:
     A. Airway:
        Protected by frequent suction, intubation if required.
     B. Breathing:
        - ✓ High flow oxygen (target saturation > 95%)
        - ✓ Assisted ventilation: Non-invasive (CPAP)/ Invasive.

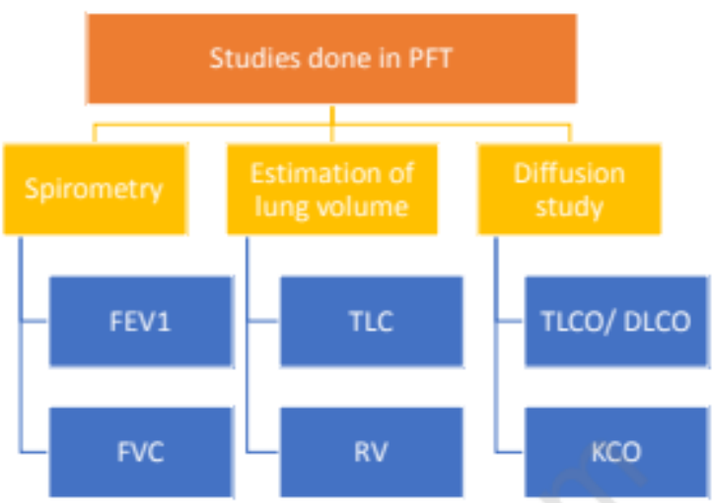
     C. Circulatory support by IV fluid
     D. Drugs:
        - ✓ Antibiotic: Short course
        - ✓ Aminophylline infusion:
          If patient remains symptomatic after optimal therapy.

- ✓ Bronchodilator:
  Nebulization with (Salbutamol + Budesonide+ Ipratropium): Initially repeated every 15-30 minutes. When acute stage is over, it is given every 4-6 hours till the patient is stable enough to use inhaler.
- ✓ Corticosteroids:
  Oral (Prednisolone)/ IV (Hydrocortisone) for 5-7 days.
- ✓ A single dose of IV $MgSO_4$.

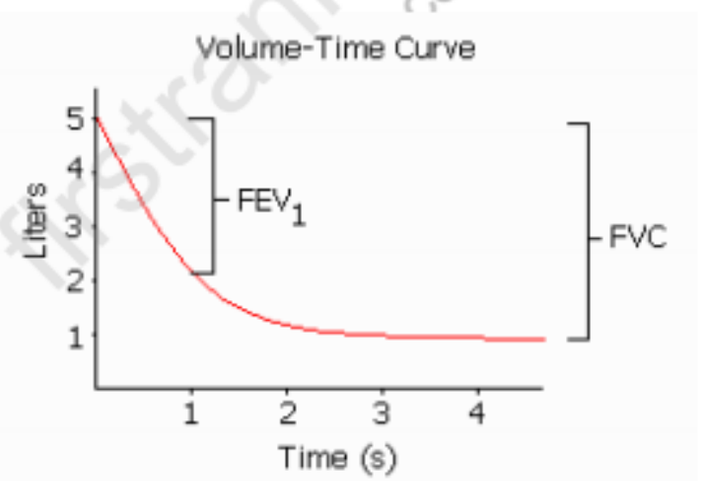
Star Topic: Grading of asthma

Points	Moderate	Severe	Life threatening
Peak expiratory flow rate (% of predicted)	50-75%	33-50%	<33%
Features:	Increasing symptoms	Increasing symptoms	- No chest sound - Cyanosed - Tachy/brady-arrhythmia - Hypotension - Confusion/ coma/ exhaustion - pO_2 <60 mm Hg - Saturation <92% - pCO_2: Normal/ near normal.
	Absence of severe/ life threatening features	Respiratory rate >30/min Heart rate >110/min	

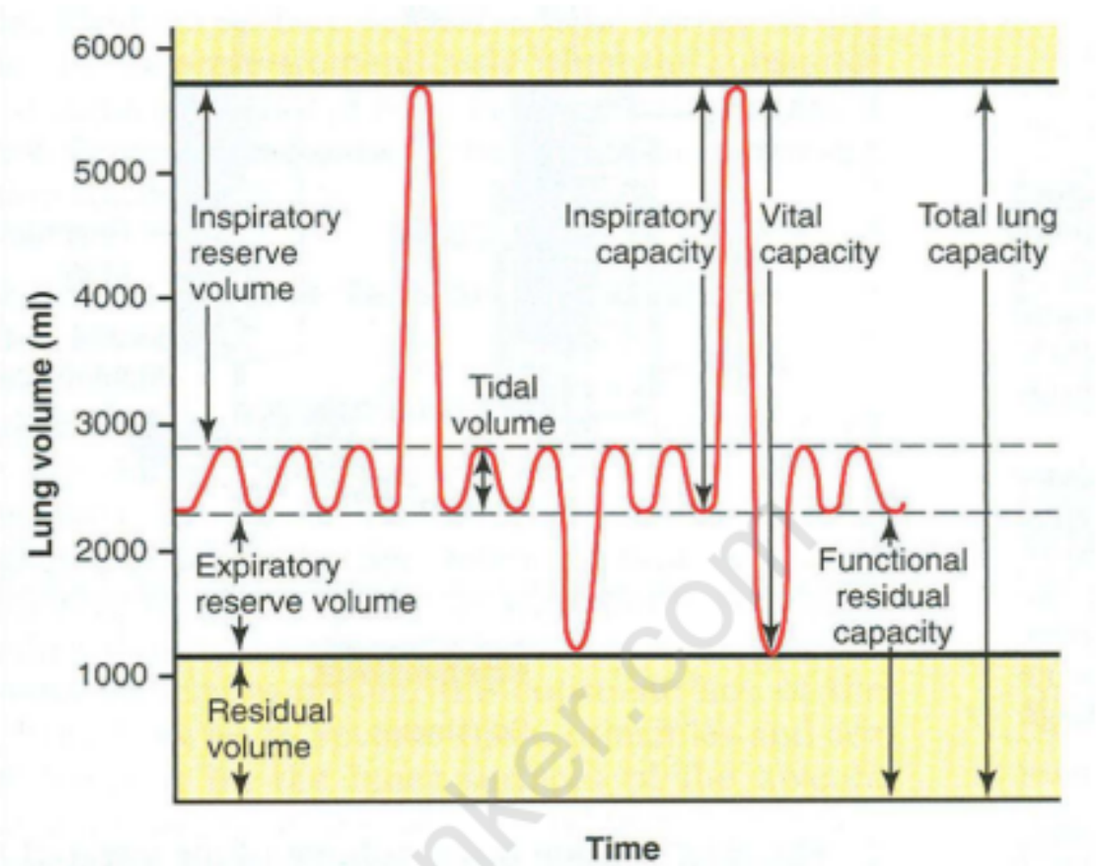
Pulmonary function test (PFT)



- *FEV1 (Forced expiratory volume in 1 second):* FEV1 is the volume exhaled during the first second of a forced expiratory maneuver started from the level of total lung capacity.
- *FVC (Forced vital capacity):* FVC is the volume of air that can forcibly be blown out after full inspiration.



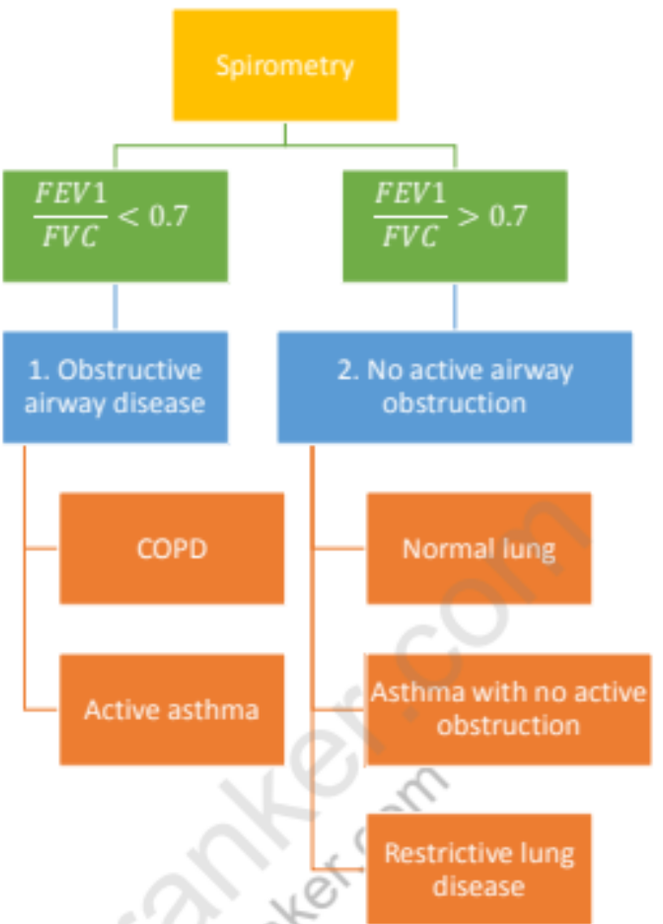
- *TLC (Total lung capacity):* TLC is the maximum volume of air present in the lungs.
- *RV (Residual volume):* RV is the volume of air remaining in the lungs after a maximal exhalation.



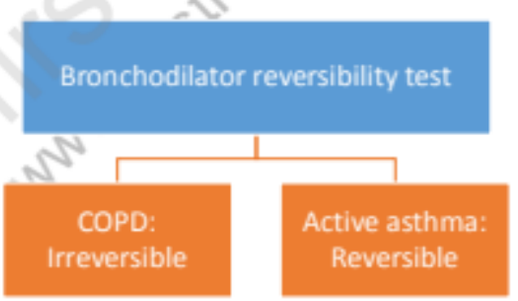
- *DLCO/TLCO (Diffusing capacity/ Transfer factor of the lungs for carbon monoxide):* DLCO is the carbon monoxide uptake from a single inspiration in a standard time (usually 10 seconds). The exhaled gas is tested to determine how much of the tracer gas was absorbed during the breath. DLCO measures the ability of the lungs to transfer gas from inhaled air to the red blood cells in pulmonary capillaries.
- KCO: Diffusing capacity of the lung per unit volume.

Basic clinical application of PFT in lung diseases

We can clearly differentiate among some common diseases through using the pulmonary function tests. In this section, we will show how the 3 components of PFT as discussed above (spirometry, lung volume and diffusion study) can be used to reach a diagnosis separately.

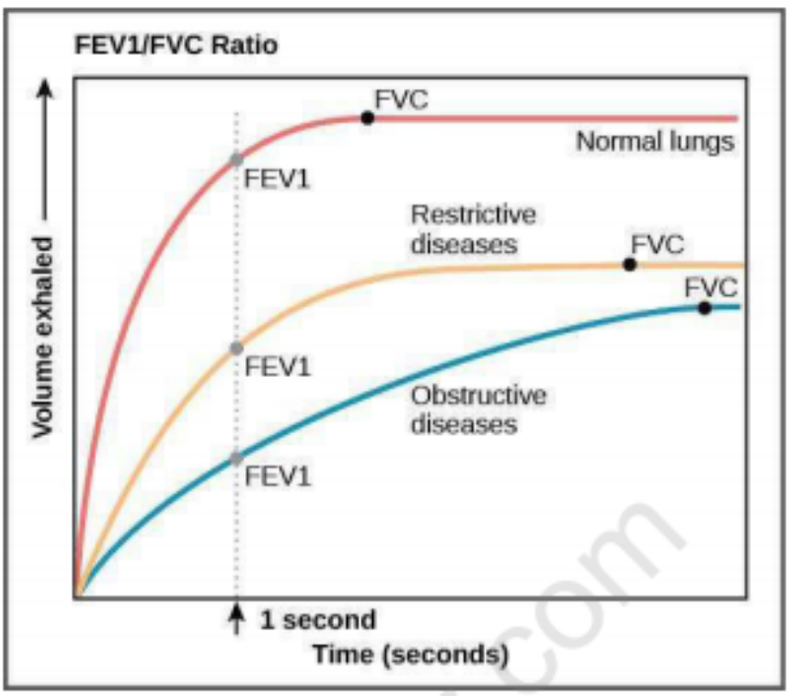


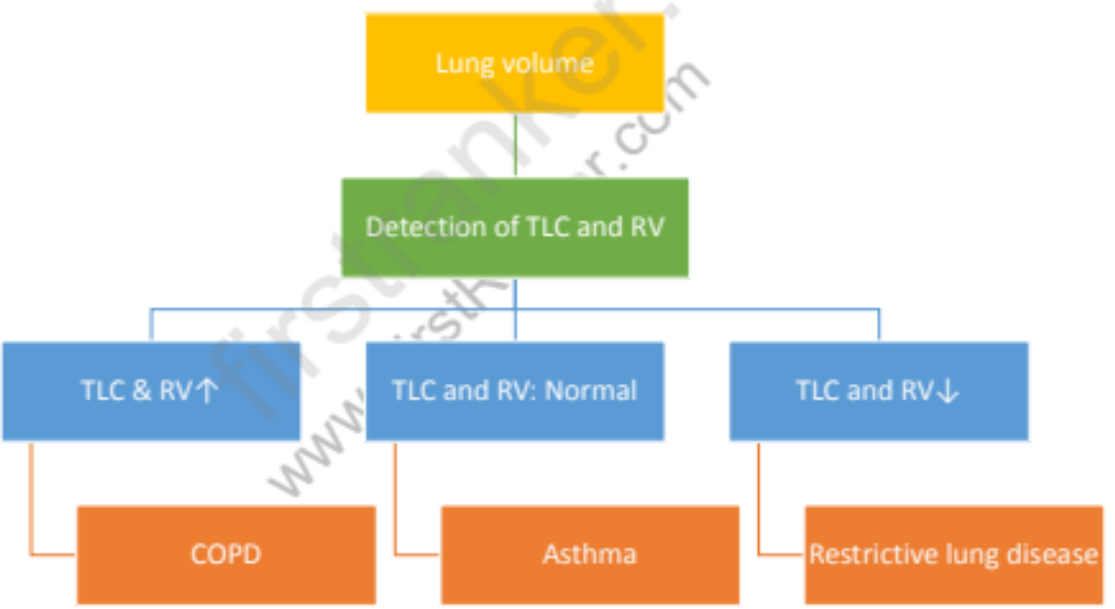
For group 1:

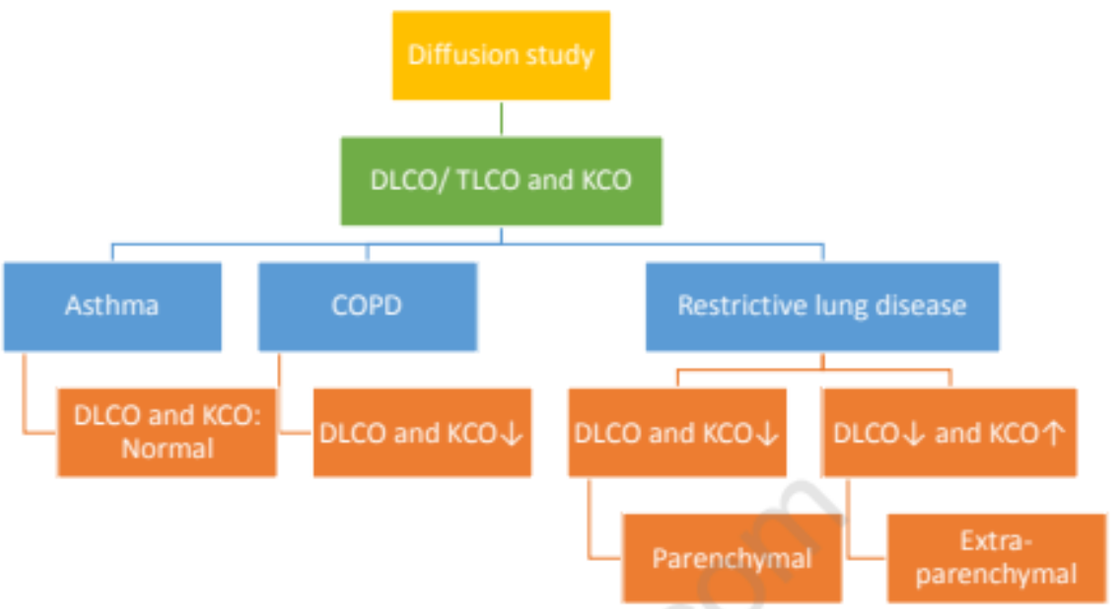


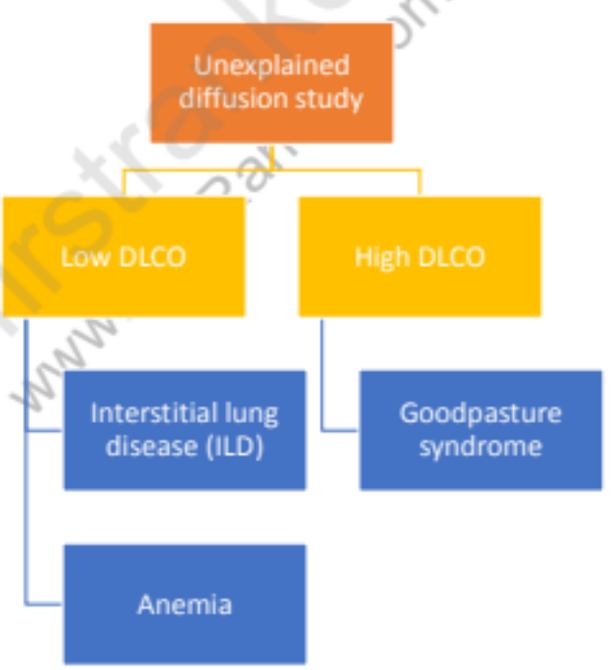
For group 2:

1. Asthma with no active ostruction: Detect by a bronchial provocation test.
2. Normal lung Vs. Restrictive lung disease:
   In normal lung: FVC and FEV1: both values will be high individually.
   In restrictive lung disease: FVC and FEV1: both values will be low individually.









Bronchogenic Carcinoma

Introduction

Malignancy of bronchial/ bronchiolar epithelium.

Risk factors

1. Cigarette smoking:
   a. Initiator (Ex: Polyaromatic hydrocarbon: PAH)
   b. Promoter (Ex: Phenol derivatives).
2. Occupational exposure:
   Ex: Asbestos (usually causes adenocarcinoma of lung).
3. Environmental toxins.

Pathophysiology

Intrathoracic effects of bronchogenic CA

Structures involved	Effects
Airway	• Obstruction • Narrowing
Parenchyma	• Collapse • Consolidation • Cavitation
Pleura	Effusion
Pericardium	
Ribs	• Erosion • Irritation of intercostal nerves (ICN)
Lymph node	Hilar/ mediastinal/ supraclavicular lymphadenopathy may result in compression of the following structures: 2 tubes: ✓ Esophagus ✓ Trachea. 2 nerves: ✓ Sympathetic trunk ✓ Recurrent laryngeal nerve. 2 great vessels: ✓ Superior vena cava ✓ Subclavian artery.

Extra-thoracic effects of bronchogenic CA

1. Metastasis to:
   - ✓ Brain
   - ✓ Bone
   - ✓ Liver.
2. Paraneoplastic syndromes.

Clinical features

Symptoms:

Category	Symptoms
Usual complaints	• Dyspnea • Productive cough • Chest pain (dull in nature in erosion of ribs/ sharp shooting in nature in irritation of ICN) • Hemoptysis.
Due to compression	• Dysphagia (more towards solid food) • Stridor • Hoarseness of voice (due to injury to recurrent laryngeal nerve: Adductor palsy) • Explosive nature of cough (also termed as 'bovine cough': due to RLN palsy) • Symptoms of SVC obstruction.
Due to extrathoracic effect	• Bone pain • Increased intracranial tension (ICT) due to brain metastasis: ✓ Headache ✓ Vomiting ✓ Convulsion.

Signs:

1. Pallor: may be present
2. Clubbing may be positive
3. Jaundice may be present (in case of liver metastasis)
4. Signs of IVC obstruction
5. Supraclavicular lymphadenopathy.

System specific signs	
System	Signs
Respiratory system	Signs of the following may be present: • Consolidation • Collapse • Cavitation • Pleural effusion.
GIT	• Firm to hard hepatomegaly (in case of liver metastasis) • Malignant ascites (in case of peritoneal metastasis).
CNS	Following signs may be present: • Papilledema • Gradual onset hemiparesis • Signs due to Pancoast tumor.

Paraneoplastic syndromes

Distant non-metastatic manifestations of bronchogenic CA which are usually due to either secretory nature of the tumor or immunologically mediated.

System involvement	Symptoms	
Endocrinal (most commonly seen in small cell lung carcinoma)	Hormone	Effect
	ACTH	Cushing syndrome
	PTHrP	Hypercalcemia
	ADH	SIADH
	Gonadotropin	Gynecomastia
CNS	• Lambert–Eaton myasthenic syndrome • Subacute cerebellar degeneration.	
Hematological	• DIC • Anemia.	
CVS	• Non-infective endocarditis • Migratory thrombophlebitis.	
Renal	Nephrotic/ nephritic syndrome	

Investigation

- Confirmation of diagnosis:
  1. Chest X Ray
  2. Contrast enhanced CT scan (CE-CT) of chest and abdomen
  3. PET scan (in selected cases)
  4. Histopathological confirmation by:
     - ✓ Bronchoscopic biopsy
     - ✓ CT guided biopsy
     - ✓ Lymph node biopsy
     - ✓ Cytological examination of pleural fluid.
- To evaluate and assess various effect of tumor:
  1. Blood: Hb, TC, DC, ESR/ CRP.
  2. Na+ K+ Urea Creatinine (Hyponatremia due to SIADH may occur)
  3. Liver function test (Alkaline phosphatase level may be elevated)
  4. Serum Ca++ level (may be elevated in case of bone metastasis/ Paraneoplastic syndrome: secretion of PTHrP by the tumor)
  5. CT/ MRI of brain
  6. Bone scan (if bone metastasis is suspected).
- Assessment of physical function:
  1. ECG
  2. Echocardiogram (if needed)
  3. Pulmonary function test.

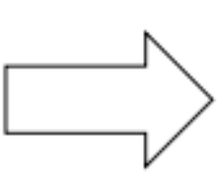
Treatment

Treatment depends on:

1. Staging of tumor
2. Overall fitness of patient.

Treatment options:

1. Surgery
2. Chemotherapy
3. Radiotherapy



1. Down staging of tumor
2. Slow down the progress of tumor
3. Control of symptoms

Therapeutic classification and treatment of choice:

Group	Feature	Treatment of choice
Small cell lung CA	More aggressive Limited role of surgery	▪ Chemotherapy: Carboplatin/ Cisplatin ▪ Radiotherapy
Non-small cell lung CA	Less aggressive Often resectable	▪ Surgery: Options are: 1. Lobectomy 2. Pneumonectomy (removal of a lung). The choice of surgery depends on the size of tumor and pre-operative fitness of patient. ▪ Chemotherapy: Carboplatin/ Cisplatin + Gemcitabine ▪ Radiotherapy.

Some conditions associated with bronchogenic CA

SVC Obstruction

Introduction

Occlusion of SVC due to extraluminal/ intraluminal compression.

Cause

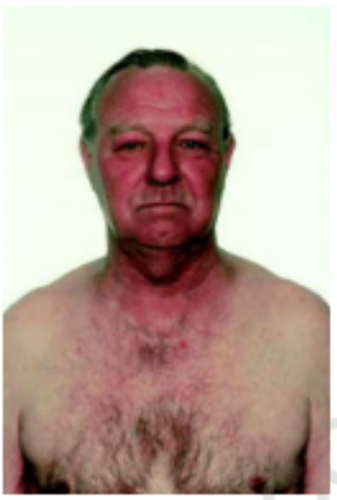
1. Malignancy:
   a. Bronchogenic CA
   b. Lymphoma.
2. Benign:
   a. Thymoma
   b. Retrosternal goitre
   c. Clot/ thrombus.

Clinical features

Symptoms:

The following symptoms are commonly seen in SVC obstruction and these occur because of impaired venous drainage:

1. Facial and neck swelling
2. Plethoric appearance (red, flushed) of face
3. Throbbing headache.



Signs:

1. JVP: Raised and non-pulsatile
2. Facial plethora and swelling may increase on lifting both the arms (Pemberton's sign).
3. Superficial venous prominence over the chest wall with direction of filling from above downwards may be seen.

Investigation

1. CT chest
2. Histopathological confirmation the underlying disease/ condition

Treatment

1. Supportive treatment:
   a. Oxygen
   b. Steroid: to reduce the edema surrounding the obstruction.
2. Specific treatment of the underlying cause
3. If the patient is in severe distress, then *SVC stenting* may be considered.

Pancoast tumor

Introduction

It is malignancy situated in the apex of the lung. Because of its location, it sometimes causes some unusual manifestations. When these manifestations occur, it is called *Pancoast syndrome*.

Clinical features

<table>
<tr><th>Cause</th><th colspan="2">Symptoms and signs</th></tr>
<tr><td>Compression of C8-T2 nerve root</td><td colspan="2">• Pain/ paresthesia along the ulnar border of arm and forearm
• Wasting of hypothenar muscles.</td></tr>
<tr><td rowspan="6">Compression of sympathetic trunk</td><td colspan="2">Horner’s syndrome</td></tr>
<tr><td>Symptom</td><td>Cause</td></tr>
<tr><td>Miosis</td><td>Due to involvement of dilator pupillae</td></tr>
<tr><td>Anhydrosis* of same side of face</td><td>Due to involvement of vasomotor fibers to face</td></tr>
<tr><td>Partial ptosis</td><td>Due to paralysis of Muller’s muscle</td></tr>
<tr><td>Loss of ciliospinal reflex^</td><td>---</td></tr>
<tr><td>Enophthalmos~</td><td>Due to paralysis of Orbitalis muscle</td></tr>
</table>

[*: Decreased sweating, ^: Dilation of the ipsilateral pupil in response to pain applied to neck, face and upper trunk, ~: Posterior displacement of the eyeball.]

Pneumonia

Types

1. Community acquired pneumonia
2. Hospital acquired pneumonia
3. Aspiration pneumonia

Community acquired pneumonia

Introduction

Pneumonia occurring outside hospital setting/ within 48 hours of admission, who hasn't been in hospital for last 14 days.

Organism

Typical pneumonia:

1. Strep. pneumoniae
2. H. influenzae
3. Moraxella
4. Klebsiella.

Atypical pneumonia:

1. Mycoplasma
2. Legionella
3. Chlamydia.

Clinical features

Symptoms:

1. Systemic symptoms:
    a. Fever ± chill and rigor
    b. Headache
    c. Myalgia
    d. Loss of appetite
    e. Confusion.
2. Respiratory symptoms:
    a. Productive cough

b. Often the sputum is purulent/ mucopurulent
c. Shortness of breath (if a significant part of lung parenchyma is affected)
d. Hemoptysis (blood stained sputum) may occur.

Signs:

1. Tachypnea
2. Tachycardia
3. Pulse oximetry: Low SpO_2
4. Respiratory system:
   Signs of consolidation:
   a. BBS: Unaltered
   b. VR/ VF: ↑
   c. No shifting.
   Signs of reduced air entry:
   a. VBS: ↓
   b. VR/ VF: ↓.
   Localized area with crepitation: Usually present.

Investigation

1. Blood:
   a. Hb, TC, DC, ESR/ CRP (Raised inflammatory marker)
   b. Na+ K+ Urea creatinine
   c. Blood culture and sensitivity
   d. Serum pro-calcitonin: Very sensitive marker of bacterial inflammation.
   e. Arterial blood gas: Done if the patient is hypoxic.
2. Sputum:
   a. Gram stain and culture-sensitivity
   b. Chest X Ray (Radiological evidence of resolution always lags behind clinical picture)
3. Special tests:
   In selected cases, special tests like CT chest, bronchoscopy etc. can be performed.

Treatment

Supportive treatment (particularly in critically ill patients)

A. Airway:
   Protected by frequent suction and ventilation if required.
B. Breathing:
   - Oxygen
   - Assisted ventilation:
     - ✓ Non-invasive
     - ✓ Invasive.
C. Circulation:
   - IV fluid
   - Vasopressor (Norepinephrine infusion in septicemic shock).
D. Drug:
   - Antipyretic
   - Nebulization, if required
   - Mucolytic agent.

Antimicrobials

Choice of antibiotic:

Commonly empirical therapy with:

CO-AMOXICLAV + MACROLIDE

OR

LEVOFLOXACIN

OR

3RD/4TH GENERATION CEPHALOSPORIN/ CARBAPENEM (in critically ill patients)

Complications of pneumonia

1. Septicemia
2. Respiratory failure
3. Lung abscess
4. Non-resolving pneumonia:

Common causes are:

a. Wrong diagnosis
b. Wrong choice of antibiotic
c. Unusual/ resistant organism
d. Underlying pulmonary disease (malignancy/ bronchiectasis).

Hospital acquired pneumonia

Introduction

Pneumonia occurring after 48 hours of hospital admission.

Common organism

1. Pseudomonas
2. Staph. aureus
3. Acinetobacter.

Clinical features and investigation

Same as community acquired pneumonia.

Treatment

Supportive treatment

Same as community acquired pneumonia.

Antimicrobial

Empirical antibiotic of choice:

PIPERACILLIN + TAZOBACTUM

OR

CARBAPENEM

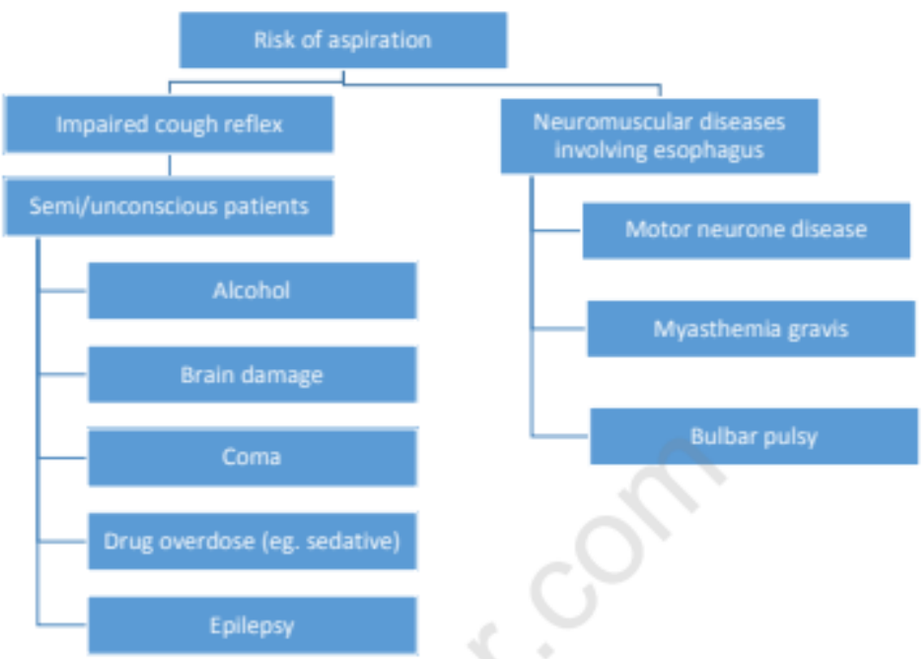
OR

POLYSTIN

Aspiration pneumonia

Pneumonia secondary to aspiration of gastric content/ oropharyngeal secretion.

Which people are at risk of aspiration pneumonia?



Clinical features

1. Background risk factor(s) of aspiration usually present
2. Often, sudden onset breathlessness with profuse respiratory secretion
3. Signs due to hypoxia
4. Signs of reduced air entry with crepitation.

Investigation

Often suggestive X Ray changes are present in the *right lower lobe*.

Treatment

Supportive treatment (as in community acquired pneumonia).

Antimicrobial

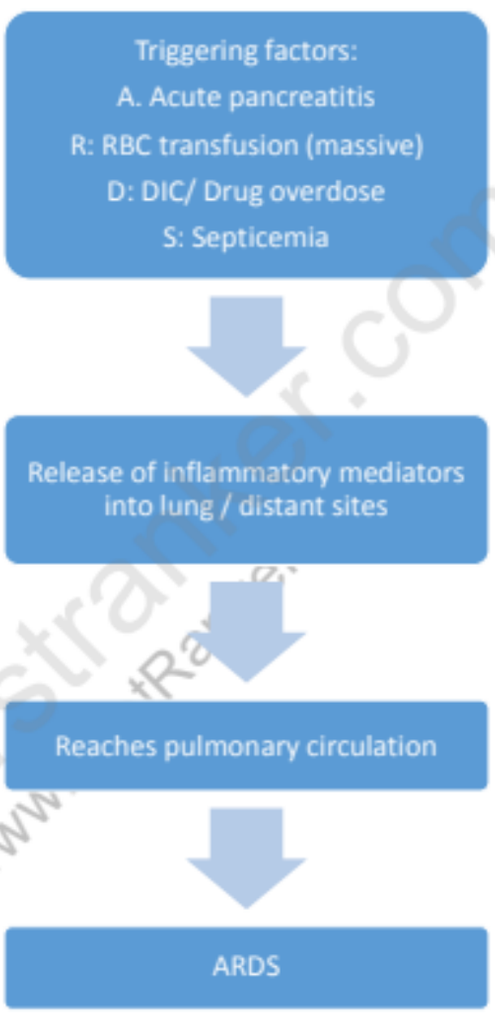
CO-AMOXICLAV + METRONIDAZOLE

Acute respiratory distress syndrome (ARDS)

Introduction

It is an acute complication of a wide variety of underlying diseases and it is characterized by *non-cardiogenic pulmonary edema.*

Pathogenesis



Clinical features

1. Rapidly progressing breathlessness
2. An underlying illness/ condition must be present
3. Often tachypnea/ tachycardia/ low SpO_2 is present
4. Often patient is hemodynamically unstable
5. Usually bilateral crepitations are present.

Treatment

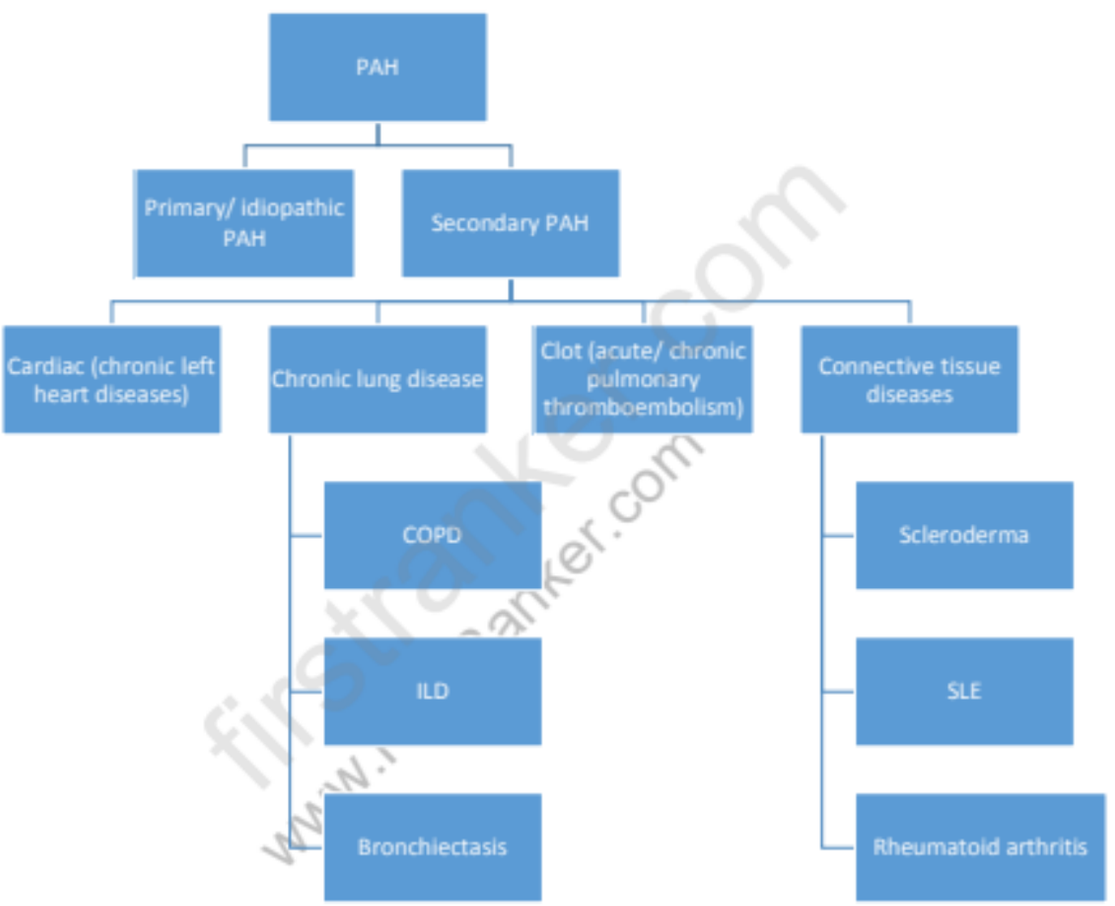
A. Airway protection:
   Often patient will require intubation
B. Breathing:
   Assisted ventilation (often invasive ventilation is required)
C. Circulation:
   IV fluid and vasopressors
D. Drugs for underlying conditions.

Pulmonary arterial hypertension (PAH)

Introduction

It is a condition characterized by pulmonary arterial pressure >20 mm Hg.

Causes



Clinical features

1. Symptoms and signs of underlying disease(s)
2. Symptoms and signs due to PAH:
   a. Exertional chest pain (also called right ventricular angina)
   b. Shortness of breath
   c. Accentuated P2 ± Palpable P2

d. MSM in case of a functional PS
e. EDM in case of a functional PR (rare).

3. Signs of RV enlargement:
   a. Apical impulse shifted outwards
   b. Diffuse apex impulse (in case of a RV apex)
   c. Left parasternal heave
   d. PSM due to a functional TR
   e. Epigastric pulsation.
4. Signs of RV failure:
   a. Raised JVP
   b. Soft tender hepatomegaly.

Investigations

1. ECG
2. Echo: Assess RV structure/ function and an approximate estimation of pulmonary arterial pressure
3. RV catheterization
4. Relevant investigations to assess underlying disease(s).

Treatment

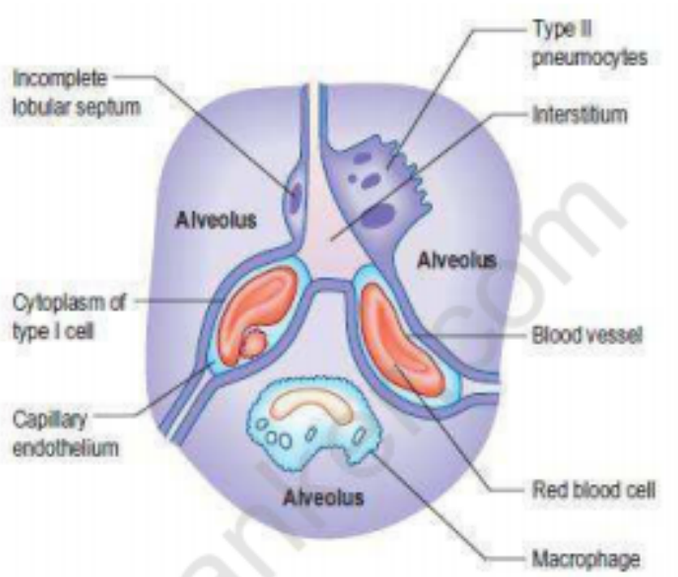
A. Anticoagulation agents (Significant PAH leads to a sluggish PA circulation, predisposing to formation of PA clot)
B. BP lowering agents:
   - Calcium channel blockers:
     - ✓ Nifedipine
     - ✓ Amlodipine.
   - PDE-5 inhibitors:
     - ✓ Sildenafil
     - ✓ Tadalafil.
   - PG analogue:
     - ✓ Iloprost (via inhalation route)
   - Endothelin receptor antagonists:
     - ✓ Bosentan
     - ✓ Ambrisentan.
C. Cause: Treat the underlying cause.

Interstitial Lung Disease (ILD)/

Introduction

It is a group of heterogenous conditions characterized by damage of interstitium of lung (alveolar epithelium + capillary basement membrane).

In many cases, these diseases also affect interlobular septa.



Types

1. Cryptogenic fibrosing alveolitis (Usual interstitial pneumonia/UIP)
2. Non-specific interstitial pneumonia (NIP)
3. Acute interstitial pneumonia
4. Desquamative interstitial pneumonia
5. Lymphoid interstitial pneumonia
6. Cryptogenic organizing pneumonia
7. Respiratory bronchiolitis associated interstitial lung disease (RB-ILD).

Causes

1. Idiopathic
2. Iatrogenic (Amiodarone)
3. Infection
4. Immunological (Connective tissue disease).

Symptoms

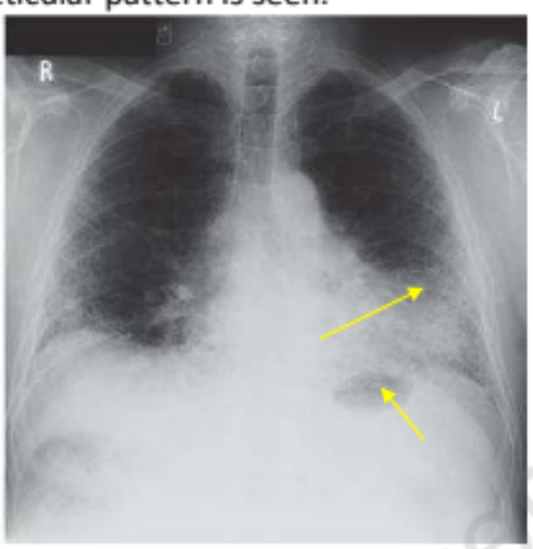
1. Shortness of breath:
   Onset, severity and progression depend on the underlying disease.
2. Chronic productive/ dry cough
3. Systemic:
   a. Fever
   b. Arthralgia
   c. Weight loss may occur.
4. In the long run: Swelling (due to RVF).

Signs

1. If breathless:
   a. Tachypnea
   b. Tachycardia
   c. Low SpO_2 ± Cyanosis
2. Clubbing:
   May occur in cases of chronic infection/ inflammation. It is seen particularly in UIP.
3. Chest:
   a. Signs of reduced air entry:
      VBS↓, VR↓, VF↓
   b. Crepitation:
      Fine inspiratory crepitation may be present (during opening up of alveoli).

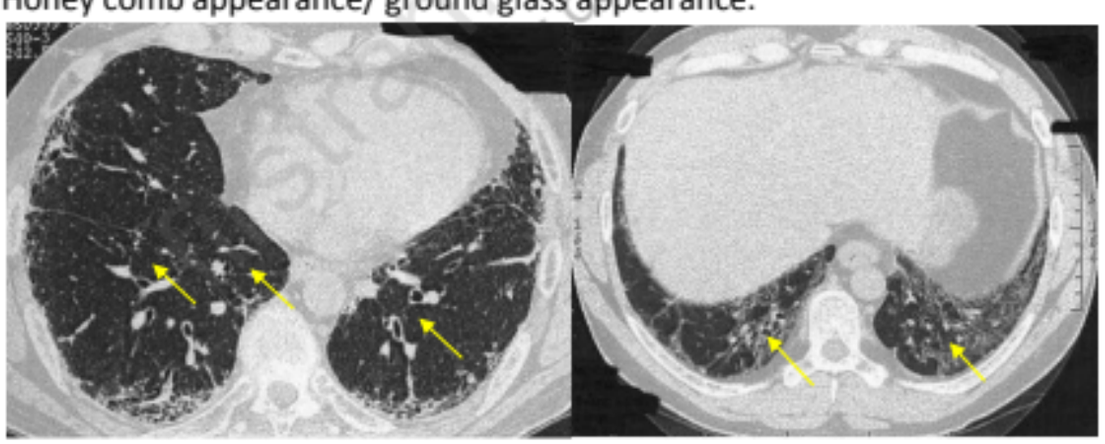
Investigation

1. Chest X Ray:
   Reticular pattern is seen.



Chest X-ray showing bilateral, predominantly lower zone and peripheral coarse reticulonodular shadowing and small lungs

2. High resolution CT scan (HRCT):
   Honey comb appearance/ ground glass appearance.



[HRCT showing honeycomb appearance in UIP (left) and extensive ground glass appearance in NIP (right)]

3. Pulmonary function test:
   Pattern: Restrictive parenchymal defect
4. If radiological appearance is inconclusive, then histopathological confirmation by lung biopsy is recommended.
5. Relevant investigation to diagnose any underlying disease.

Treatment

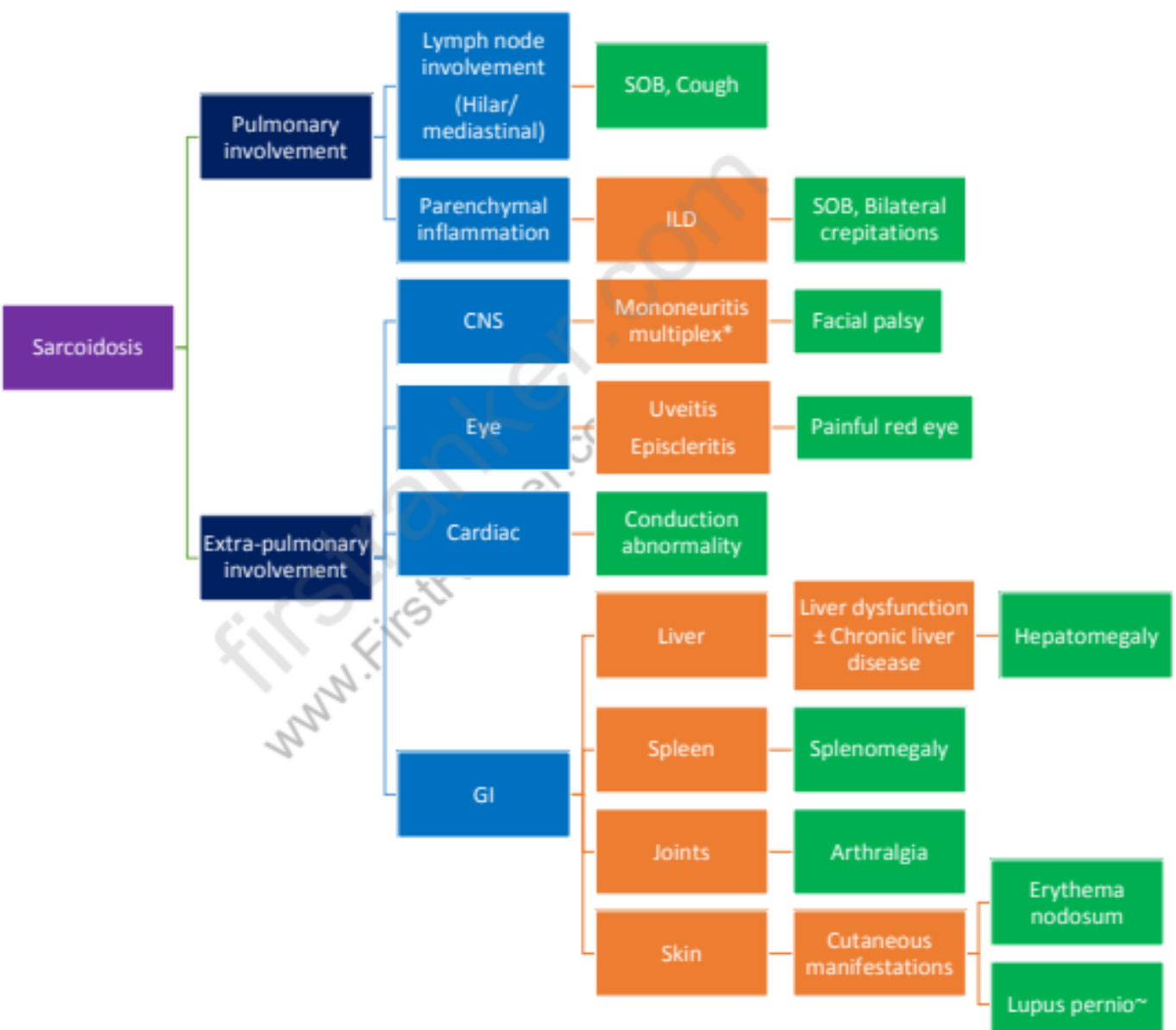
1. Corticosteroid (systemic)
2. Immunosuppressive drugs:
   a. Azathioprine
   b. N-Acetyl Cysteine (NAC)
   c. Pirfenidone.
3. Symptomatic:
   a. Long term oxygen therapy
   b. Treatment of RHF, if present.
4. Surgery:
   Lung transplantation.

Sarcoidosis

Introduction

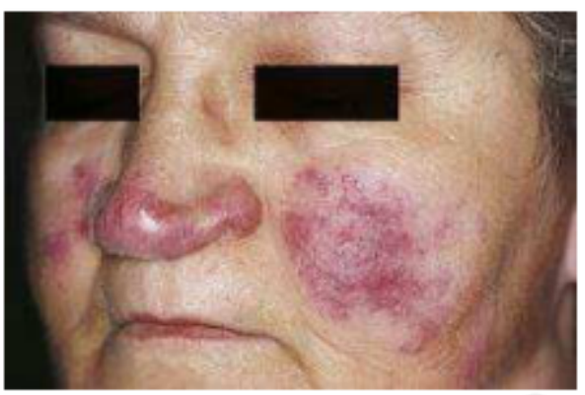
It is an immunologically mediated multisystem disease characterized by *non-caseating granulomatous inflammation*.

Involvement and clinical features



*Single/ multiple cranial nerve palsy, most common: CN7 palsy. ~Lupus pernio is a chronic raised indurated (hardened) lesion of the skin, often purplish in color. It

resembles frostbite as it is seen on tip of the nose and surrounding cheeks. It is pathognomonic of sarcoidosis.



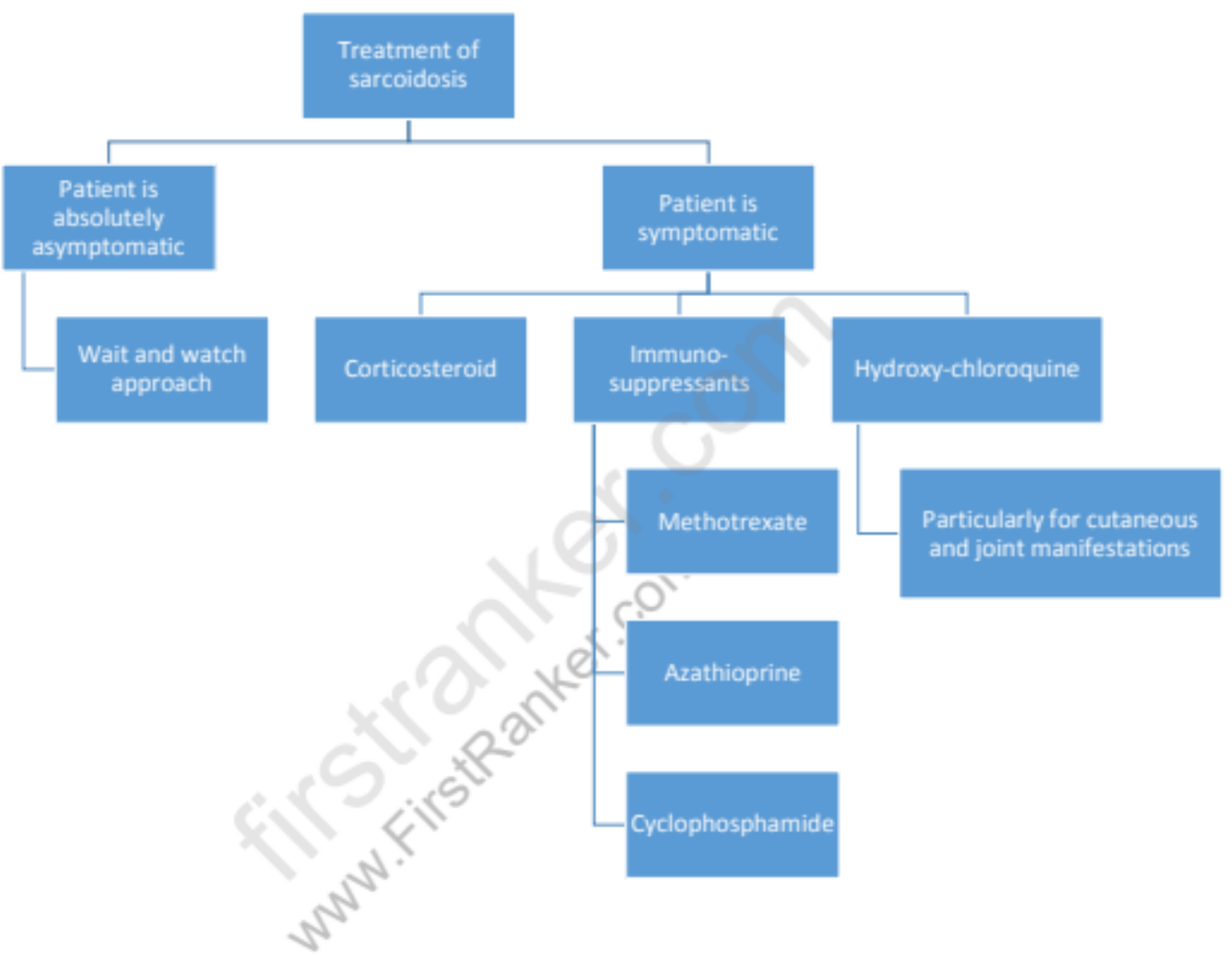
Investigations

1. Pulmonary:
   a. Chest X Ray
   b. HRCT (If CXR is abnormal)
      - Both will show reticulonodular pattern ± lymphadenopathy.
   c. Histopathological confirmation by biopsy from:
      - Lymph node
      - Parenchyma.
      - If radiological appearance is inconclusive.
   d. Pulmonary function test:
      Restrictive lung disease.

2. Other investigations:
   a. Hb, TC, DC, CRP/ESR
   b. Liver function test
   c. Serum Ca++:
      Hypercalcemia is quite common due to increased production of 1,25-$(OH)_2$-cholecalciferol [vitamin D_3], which causes increased absorption of Ca++ from gut.
   d. Serum angiotensin converting enzyme (ACE):
      Due to activation of macrophages in sarcoidosis and their release of ACE, serum ACE level is increased. Although serum ACE is a non-specific marker for sarcoidosis. It is an indicator of disease activity.

e. ECG: To look for conductive disturbances.
f. Biopsy of any affected organ.

Treatment



Extrinsic allergic alveolitis/ Hypersensitivity pneumonitis

Introduction

It is an inflammatory condition characterized by alveolar/ parenchymal inflammation usually due to *a hypersensitivity reaction against organic antigens*.

Common antigens

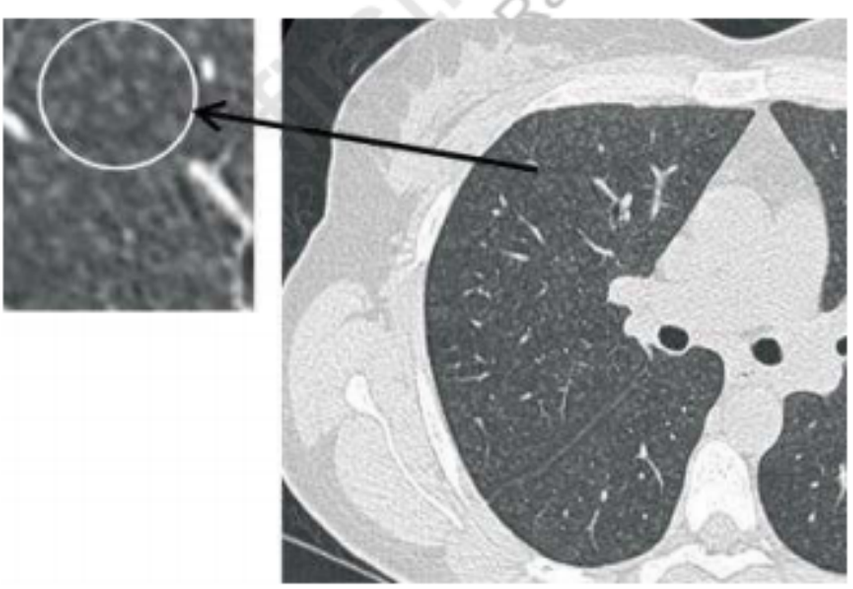
1. Fungus:
   Actinomycetes
   Aspergillus
2. Bird proteins.

Clinical features

1. Breathlessness: acute/ chronic
2. Dry cough
3. On examination: tachypnea, low SpO_2, bilateral crepts may be found.

Investigations

1. Chest X Ray
2. HRCT
   - Predominantly nodular pattern ± reticulation is seen.



HRCT of the chest in acute hypersensitivity pneumonitis showing widespread centrilobular nodules.

3. Blood: CBC

4. ABG: Hypoxic
5. Detection of serum precipitins against the organic antigens.

Treatment

1. Supportive:
   a. Oxygen: If the patient is hypoxic
   b. Systemic corticosteroid.

Cryptogenic organizing pneumonia

Introduction

It is an atypical type of pneumonia characterized by *formation of granulation tissue inside the alveoli*.

Clinical features

Symptoms

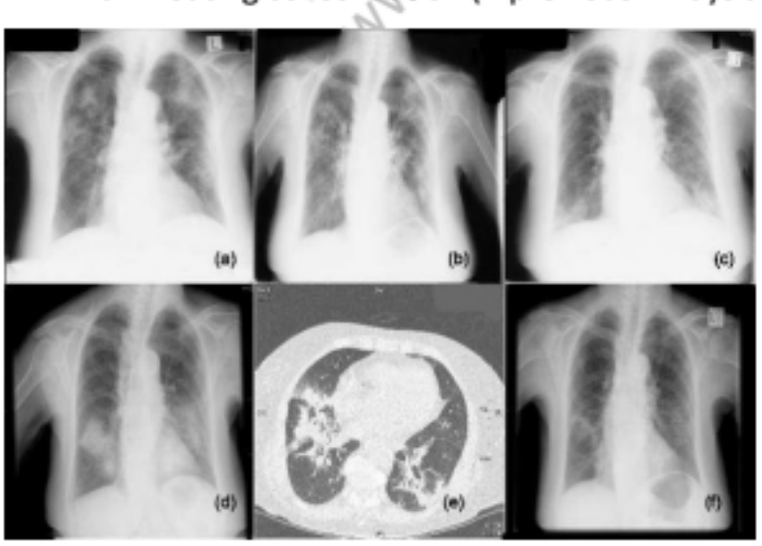
1. Shortness of breath
2. Dry/ productive cough
3. Systemic symptoms:
   a. Fever
   b. Malaise
   c. Loss of appetite.

Signs

1. Tachypnea, low SpO_2 ± cyanosis
2. Signs of consolidation/ reduced air entry
3. Crepitations usually present.

Investigations

1. Chest X Ray:
   a. Often peripheral consolidation is seen
   b. Floating consolidation (if previous X Rays are available)



Floating consolidations

This initial chest X-ray (a) at time of viral illness shows patchy bilateral mid and upper zone opacities, a second X-ray 1 month later (b) shows non resolution despite a course of oral Amoxiclav. The third X-ray 2 months later (c) shows bilateral basal opacities. A fourth shows worsening opacities bibasally another 2 months later (d). HRCT at this time shows bilateral dense consolidation with air bronchograms (e). Last chest X-ray (f) shows marked improvement after 6 weeks of steroids.

2. HRCT: Often confirms the diagnosis
3. In many cases, histopathological confirmation is required
4. Blood: CBC, ABG, Blood culture
5. Sputum: Gram stain and culture.

Treatment

1. Supportive: Oxygen if required
2. Antibiotic: Often prevent keeping CAP (Community acquired pneumonia) in mild.
3. Corticosteroid: Many patients need prolonged course of steroids.

Pericardial diseases

Acute pericarditis

Introduction

Acute inflammation of pericardium.

Etiology

1. Infection: May be viral (more common) or bacterial
2. Post irradiation
3. Immunologically mediated: Connective tissue diseases
4. Inflammatory:
   a. Post-MI: If pericarditis occurs within 34 days of AMI, it is called immediate post MI pericarditis.
   b. Dressler's syndrome: It occurs as a delayed complication of MI and it is also seen in post-CABG patients.

Clinical features

Symptom

Chest pain: Usually sharp, often aggravated on supine position and relieved when patient sits up and leans forward.

Occasionally, the inflammation spreads to the adjacent pleura causing pleuropericarditic pain which characteristically aggravates on deep inspiration/ sneezing/ coughing.

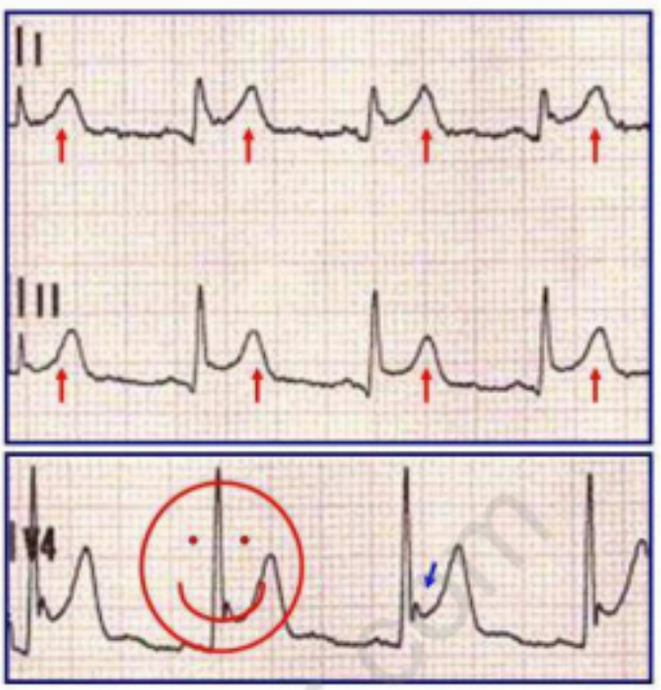
Signs

Pericardial rub: Often present and does not disappear on breath holding.

[Upon auscultation, this sound is heard as an extra heart sound of to-and-fro character, grating in nature, <u>*best heard between the apex and sternum*</u>.]

Investigation

1. Chest X Ray: Often normal
2. ECG:
   a. ST segment elevation with concavity upwards (scooped pattern)
   b. Diffuse/ global ST segment elevation (in all leads).



3. Echocardiogram: To rule out any pericardial effusion.

Treatment

1. In most of the cases: NSAIDS/ Paracetamol
2. In selective cases: Corticosteroid is quite helpful particularly in Dressler's syndrome.

Pericardial effusion

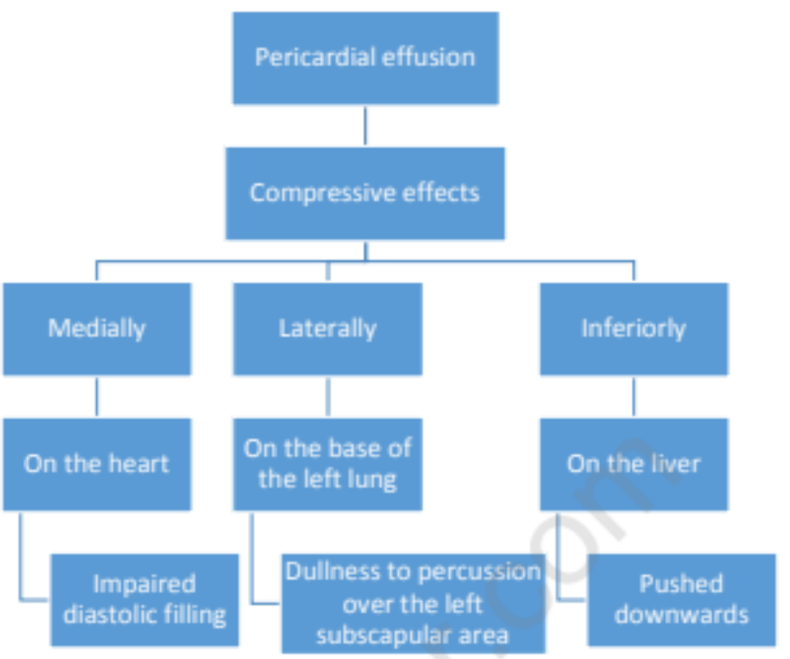
Introduction

Accumulation of fluid in the pericardial cavity.

Causes

1. Infection: TB
2. Autoimmune diseases/ connective tissue diseases
3. Malignant effusion: Breast/ lung/ lymphoma.
4. Trauma.

Effects on surrounding structures



Clinical features

Symptoms

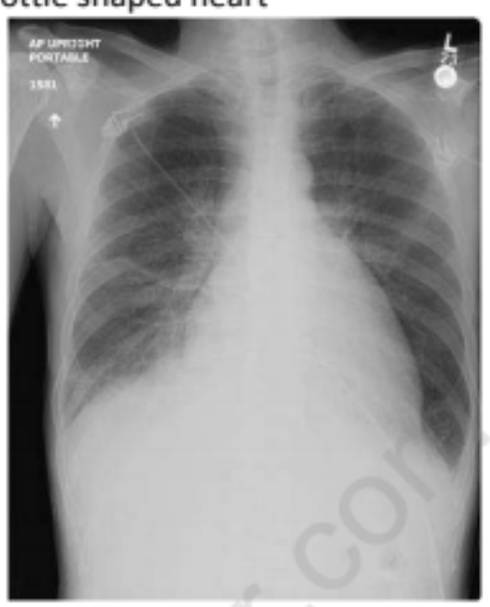
1. Chest discomfort/ heaviness
2. Swelling of the body
3. In significant effusion, symptoms due to low cardiac output (such as fatigability, exertional muscle pain etc.) are seen
4. Symptoms due to underlying disease.

Signs

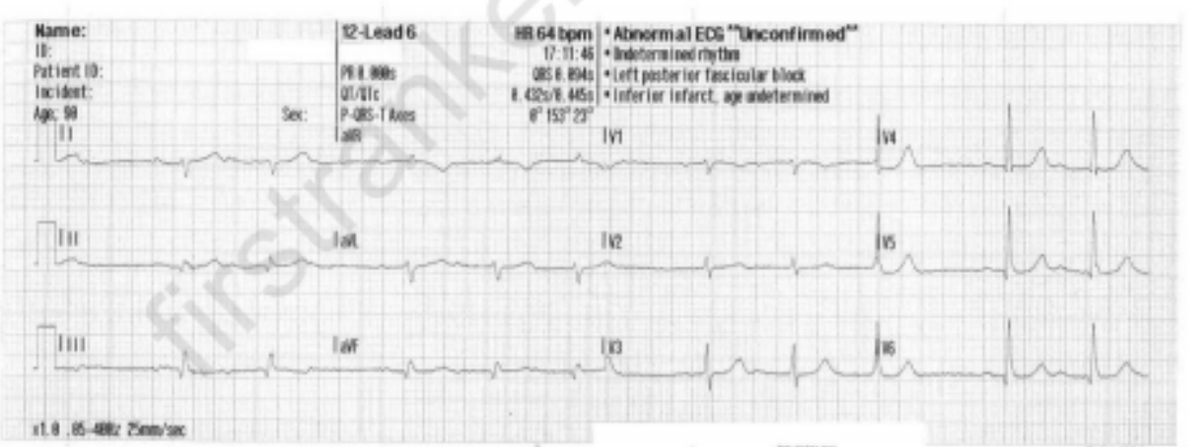
1. Edema
2. JVP↑
3. CVS: Muffled/ soft heart sound
4. Abdomen:
    - Hepatomegaly due to portal venous congestion
    - Liver may be palpable in spite of absence of hepatomegaly due to pushed down liver
    - Ascites may be present.
5. Signs due to underlying disease.

Investigation

1. Chest X Ray: Water-bottle shaped heart



2. ECG: Low amplitude complex



3. Echocardiogram confirms the diagnosis
4. Diagnostic aspiration of pericardial fluid with physical/ biochemical/ cytological/ microbiological character often helps to diagnose the underlying disease.

Treatment

1. Therapeutic pericardial aspiration (Pericardiocentesis) for symptomatic relief
2. Specific treatment for underlying cause.

Cardiac tamponade

Introduction

It is a condition where there is rapid accumulation of fluid in the pericardial cavity leading to significant compression of the heart.

Cause

Pericardial effusion

Clinical features

Symptoms

Symptoms occur due to severely impaired diastolic filling and subsequently reduced cardiac output:

1. Sudden collapse/ blackout
2. Chest discomfort
3. Weakness/ fatigability.

Signs

1. ↑ JVP
2. Hemodynamic instability: Hypotension with rapid but weak pulse
3. Heart sound: Muffled.

Investigation

1. Urgent chest X Ray
2. Urgent echocardiogram
3. Once the patient is stable, diagnostic pericardiocentesis.

Treatment

1. Immediate pericardiocentesis:
   Particularly when the patient is hemodynamically unstable.
2. When the patient is stable, further treatment of the underlying disease.

Constrictive pericarditis

Introduction

It is a condition characterized by thickening, fibrosis and adhesion between the pericardial layers which ultimately leads to *encasement of the heart*.

Etiology

1. TB
2. Post irradiation

Clinical features

Symptoms

1. Swelling
2. Fatigability
3. Exertional muscle pain.

Signs

1. ↑JVP
2. Edema
3. CVS: *Pericardial knock* may be present: It is a late diastolic abnormal sound due to *sudden vibration caused by the rigid pericardium when it reaches its elastic limit*.
4. Abdomen:
    - Hepatomegaly
    - Ascites.

Investigation

1. Chest X Ray
2. ECG
3. Echocardiogram
4. Cardiac MRI.

Treatment

1. Symptomatic treatment with diuretics
2. Pericardiectomy.

Cardiomyopathies

Dilated cardiomyopathy

Introduction

It is a primary (intrinsic) cardiac muscle disease characterized by *abnormal dilatation/ enlargement of the cardiac chambers*.

Etiology

1. Idiopathic: Probably underlying genetic factors are responsible
2. Chronic alcohol abuse: Alcohol acts as a toxin cardiac muscle
3. Long standing diabetes mellitus
4. Infiltration of cardiac muscles:
   Ex: Amyloidosis/ Hemochromatosis/ Sarcoidosis.

Pathophysiology

1. Impaired diastolic relaxation
2. Systolic dysfunction.
   - Patient develops features of uni/bi-ventricular failure.

Clinical features

Symptoms

1. Shortness of breath/ orthopnea/ PND
2. Swelling.

Signs

1. Signs of LHF:
   - S3 is usually present
   - Gallop rhythm (tachycardia + S3/S4) may be audible
   - Bilateral fine inspiratory crepitations.
2. Signs of RHF:
   - Edema
   - JVP↑
   - Hepatomegaly.

Investigations

1. Chest X Ray: Cardiomegaly
2. ECG: Features of chamber enlargement
3. Echocardiogram: Confirms the diagnosis and can assess the degree of dysfunction.

Treatment

1. Treatment of heart failure
2. Treatment of underlying disease (if any).

Hypertrophic cardiomyopathy

Introduction

It is an intrinsic disease of cardiac muscle characterized by *asymmetrical septal hypertrophy leading to left ventricular outflow tract obstruction*.

Clinical features

Symptoms

1. Due to LVOT obstruction:
   a. Syncope/ transient blackout
   b. Sudden collapse
2. Due to LVF:
   SOB/ Orthopnea/ PND.

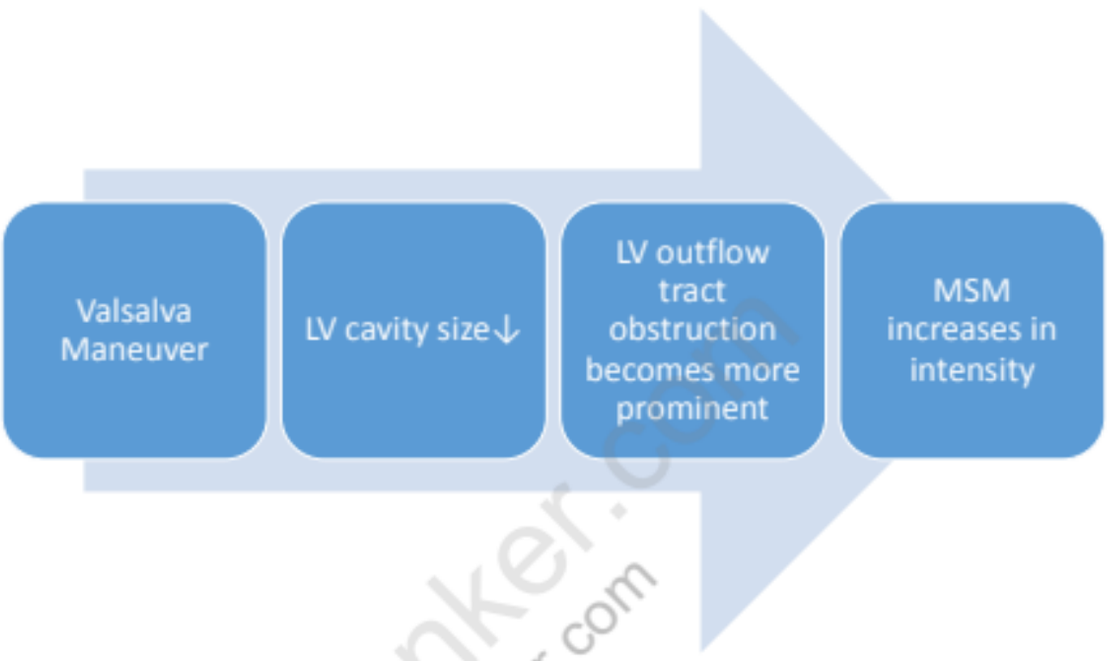
Signs

1. Apical impulse:
   - Forceful, well sustained
   - Double apical impulse (2 systolic impulses) may be felt on palpation.
2. Auscultation:
   - Signs of LHF may be present
   - MSM may be present over the aortic area, which accentuates and decreases in intensity during Valsalva maneuver* and squatting position, respectively.

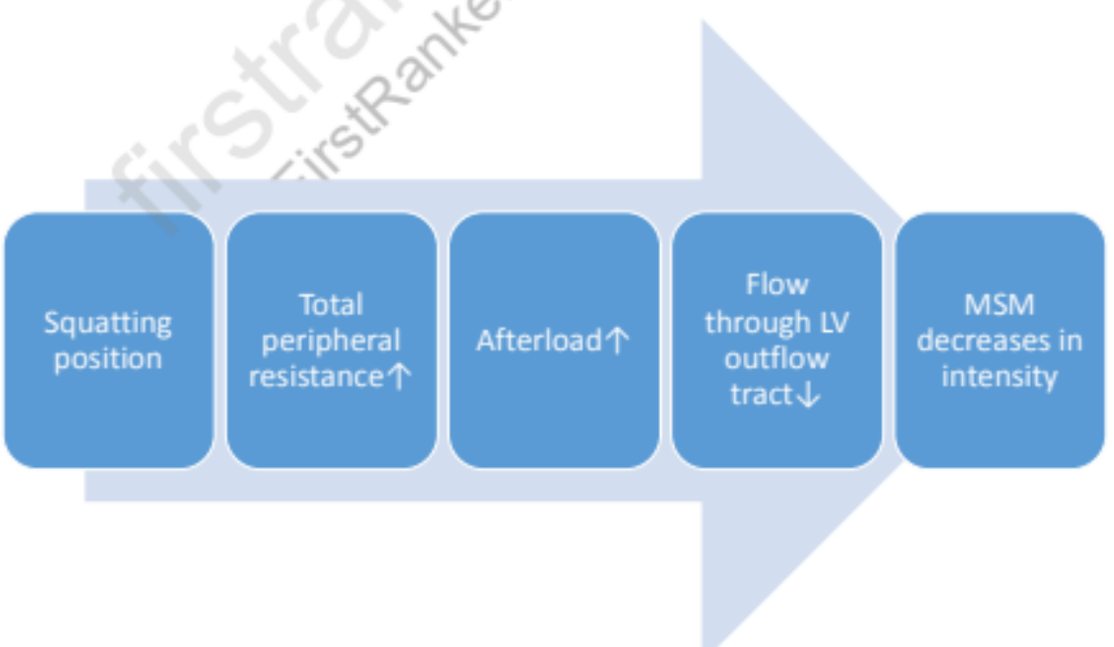
*Valsalva maneuver is performed by moderately forceful attempted exhalation against a closed airway, usually done by closing one's mouth, pinching one's nose shut while pressing out as if blowing up a balloon.

Mechanism:

1.



2.



Investigation

1. Chest X Ray
2. ECG
3. Echocardiogram.

Treatment

1. Symptomatic treatment of heart failure
2. Surgical: Septal de-bulking
3. Screening of other family members.

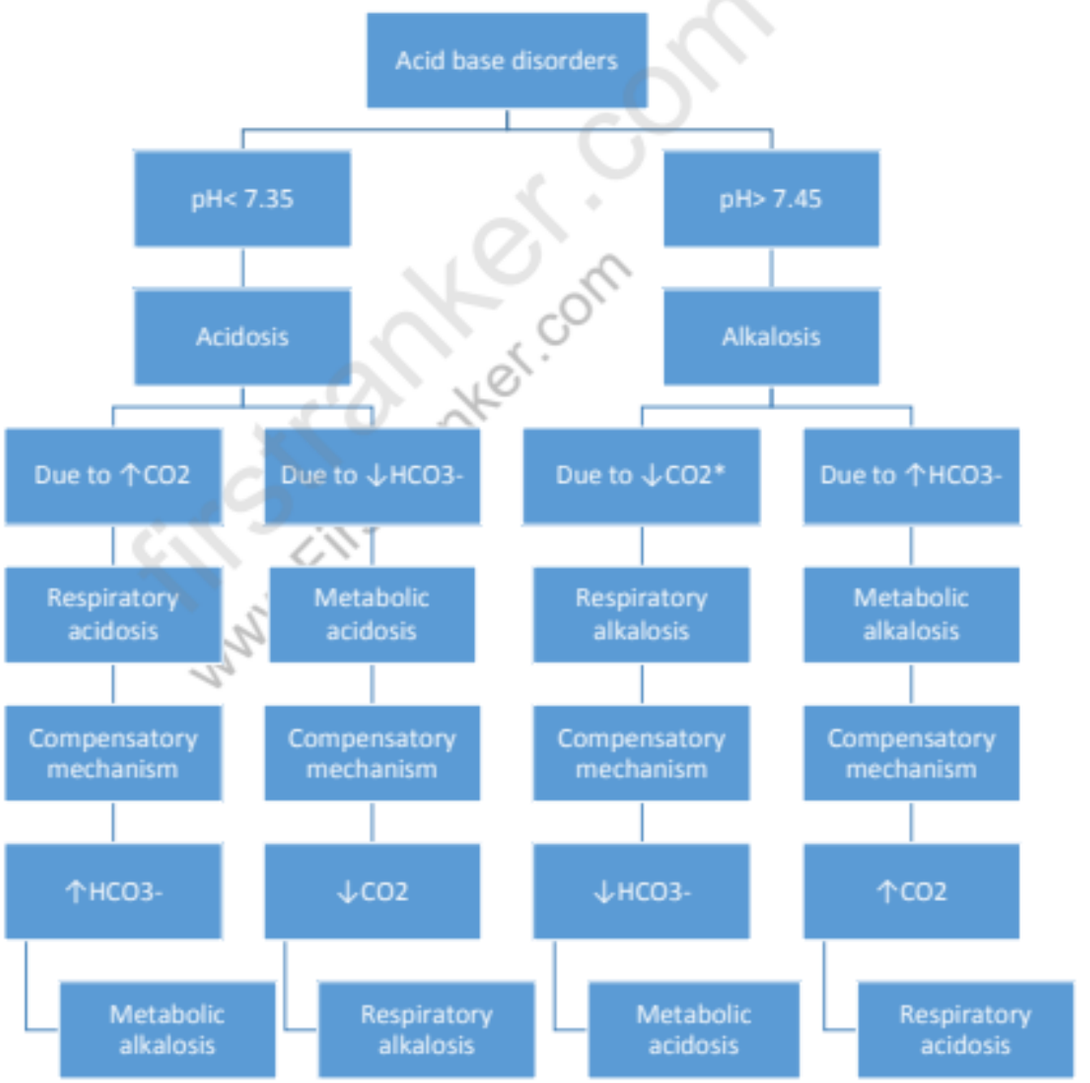
Special topic: Acid base balance

Normal ABG values

- pH: 7.35-7.45
- PaO_2: 80-100 mm Hg
- $PaCO_2$: 35-45 mm Hg
- SpO_2: 96-100%
- HCO_3^-: 22-26 mEq/L
- Base excess: -2 to +2.

ABG: Arterial blood gas,
Pa: Arterial tension,
Sp: Saturation

Classification of acid base disorders



[*Due to hyperventilation, excessive wash out of CO_2 causes ↓$PaCO_2$.]

pH, PCO_2 and HCO_3^- values in different acid base disorders

Condition	pH (N: 7.35-7.45)	PCO_2 (N: 35-45 mm Hg)	HCO_3^- (N: 22-26 mEq/L)
Respiratory acidosis	<7.35	>45	>22
Metabolic acidosis	<7.35	<35	<22
Respiratory alkalosis	>7.45	<35	<22
Metabolic alkalosis	>7.45	>45	>26

[Red color: From direct mechanism & Green color: From compensatory mechanism]

Steps of ABG

- Step 1: pH detection (acidosis/ alkalosis)
- Step 2: Primary process identification (respiratory/ metabolic)
- Step 3: Compensatory process identification (respiratory/ metabolic)
- Step 4: PO_2 measurement.

Respiratory acidosis

Introduction

Condition characterized by abnormal CO_2 retention.

Causes

Acute	Chronic
1. Acute exacerbation of COPD 2. GB syndrome 3. Myasthenia gravis 4. Acute brainstem damage.	1. COPD 2. Thoracic cage disease 3. Neuromuscular junction disease (MND/MG etc.) 4. Sleep related breathing disease: a. Obstructive sleep apnoea b. Obesity hypoventilation syndrome.

Clinical features

1. Features of underlying disease
2. Features due to high CO_2: CO_2 narcosis (Metabolic encephalopathy):
   a. Confusion
   b. Convulsion
   c. Coma
   d. Delirium
   e. Flapping tremor.

Investigation

1. To identify respiratory acidosis: ABG
   - The values will be as below:
     - ✓ pH↓
     - ✓ PCO_2 ↑
     - ✓ HCO_3- ↑.
   - In fully compensated cases, pH will return to normal.
   - Rate of compensation:
     - ✓ In acute respiratory acidosis, rise of HCO_3- will be 1 mEq/L for every 10 mm Hg of CO_2.

$$Expected\ HCO3-= 24 + \frac{(PC02 - 40)}{10}$$

     - ✓ In chronic respiratory acidosis, rise of HCO_3- will be 4 mEq/L for every 10 mm Hg of CO_2.

Treatment

1. Treatment of underlying disease
2. Assisted ventilation: Noninvasive/ Invasive.

Metabolic acidosis

Introduction

Condition characterized by low HCO_3- state either due to overconsumption or excessive loss of HCO_3-.

Anion gap

$$Anion\ gap = Measured\ cations - Measured\ anions$$
$$= [(Na\ +\ K) - (HCO3 + Cl)]$$

- Normal anion gap: 6-12 mEq/L
- Anion gap represents unmeasured anions in the body
- The unmeasured anions in our body are:
  a. Organic and inorganic acids
  b. Serum proteins (SO_4^{--}, PO_4^{---}).

Classification and causes

High anion gap metabolic acidosis	Normal anion gap metabolic acidosis
• Overproduction of H+: 1. Lactic acidosis: a. Severe hypoxia b. Hypoperfusion: ✓ Any shock ✓ Acute pancreatitis ✓ Septicemia 2. Ketoacidosis: a. Diabetic ketoacidosis b. Starvation c. Alcoholics • Underexcretion of H+: 1. Acute kidney injury 2. Chronic kidney disease.	• Excess loss of HCO_3^-: 1. Renal loss: Renal tubular acidosis 2. GI loss: a. Severe diarrhoea b. Pancreatic fistula c. Uretero-sigmoidostomy. *Here, there is a compensatory rise of Cl- concentration: *hyperchloremic metabolic acidosis*.

Clinical features

1. Due to underlying disease
2. Compensatory hyperventilation leading to rapid breathing pattern: acidotic/ Kussmaul's breathing.

Investigation

1. ABG: To identify metabolic acidosis (pH↓, HCO_3^-↓, PCO_2↓)

$$Expeceted\ PCO2\ in\ mm\ Hg = [(1.5 \times HCO3-) + 8] \pm 2.$$

(±2) is for normal compensation.

2. Anion gap should be calculated.

Treatment

1. Treatment of the underlying cause
2. In selective cases, when HCO_3^- is too low, $NaHCO_3$ administration.

Respiratory alkalosis

Introduction

Condition characterized by excess wash out of CO_2.

Primary problem

Hyperventilation.

Causes (4Ps)

1. Physical exercise
2. Pregnancy
3. Panic attack
4. Pulmonary causes*:
   a. Pulmonary embolism
   b. Pneumothorax
   c. Pulmonary interstitial disease
   d. Pulmonary edema
5. High altitude.

*In these pulmonary diseases, because of continuous tachypnea, respiratory muscles become fatigue and PCO_2 level will gradually start to fall.

Clinical features

1. Due to the underlying disease
2. Due to hypocapnia:

- ✓ Laziness/ light headacheness
- ✓ Tingling
- ✓ Perioral numbness/ paresthesia.

Investigation

1. ABG: To identify respiratory alkalosis (pH↑, PCO_2↓ , HCO_3^-↓, ***PO_2***↓)

Type of respiratory alkalosis	For every 10 mm Hg ↓ in PCO_2 level	Fall of HCO_3^-	Expected HCO_3^-
Acute		2 mEq/L	$24 - \frac{2(40 - PCO_2)}{10}$
Chronic		5 mEq/L	$24 - \frac{5(40 - PCO_2)}{10}$

2. Relevant investigations to assess the underlying condition.

Treatment

1. Treatment of the underlying disease
2. If type 1 respiratory failure occurs, then attempts to correct hypoxia by:
   a. High flow oxygen
   b. Assisted ventilation: Noninvasive/ invasive.
3. In panic attack:
   a. Reassure the patient
   b. Rebreathing into the bag.

Metabolic alkalosis

Introduction

Condition characterized by abnormal retention of HCO_3^-.

Causes

1. ↑Cl- loss/ ↑H+ loss: (Hypochloremic metabolic alkalosis)
   a. Severe vomiting
   b. Repeated gastric suction.

2. Abnormal generation of HCO_3^-:
   a. Diuretic use
   b. Hypokalemia
   c. Mineralocorticoid excess.
3. Post-hypercapnic metabolic alkalosis
   If a chronically elevated arterial PCO_2 is returned to normal quickly (as if the patient is intubated and ventilated), then the patient is in the situation of having *an elevated HCO_3^-* (due to renal compensation) *without there being the physiological need for it anymore*. The elevated bicarbonate is typically slow to fall as return to normal requires renal excretion of the excess bicarbonate.

Clinical features

Due to the underlying disease

Investigation

1. ABG: To identify metabolic alkalosis (pH↑, HCO_3^-↑, PCO_2↑)
   $$Expected\ PCO2\ in\ mm\ Hg = [(0.7 \times HCO3 -) + 20] \pm 5$$
   ±5 is for normal compensation.
2. Investigation to assess the underlying disease.