Reviewed By Tarfa Alsharidi

MED439 KING SAUD UNIVERSITY

Respiratory failure

Objectives:

1. Definition of Respiratory Failure.
2. To differentiate between type 1 and type 2 respiratory failure in mechanisms and clinical manifestations.
3. To recognize the common causes of respiratory failure.
4. To apply invasive and non-invasive modalities of management in respiratory failure.

Extra objectives from the doctor slides

5. Definition, Pathology and Pathophysiology of asthma
6. Factors that triggers Asthma
7. Manifestation and How To assess the severity of Asthma and the Treatment
8. COPD (Definition, Risk Factors, types, Treatment and Prevention)

Three questions will come from this lecture

439 Team Leader:
Nourah Alklaib

439 Team Member:
Mona Alomairini

Editing File

Color Index

- Slides / Reference Book
- Doctor notes
- OnlineMeded / Amboss
- Important
- Extra

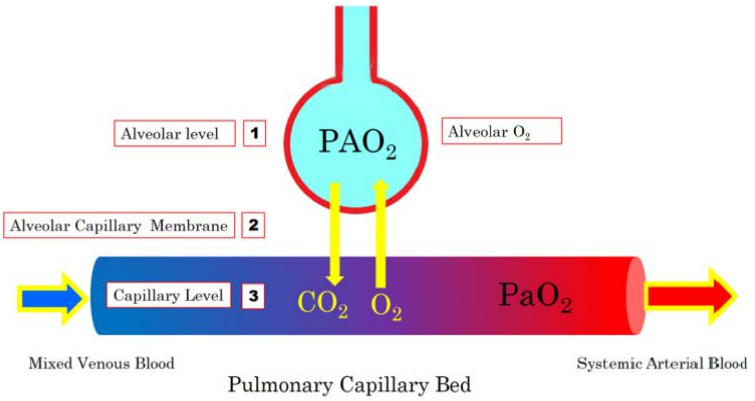
Definition of respiratory failure.

Is a clinical condition that happens when the respiratory system fails to maintain gas exchange:

- PaO2 is < 60 mmHg (around SpO2 <90%) and/or
- PaCO2 > 50 mmHg

Is classified according to blood gas abnormalities:

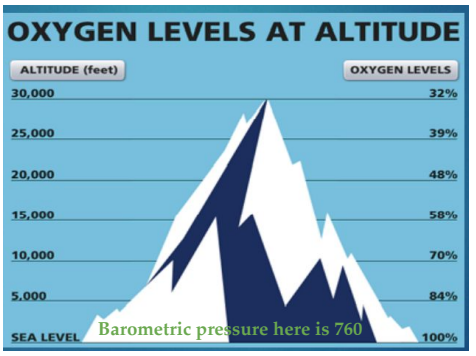
- Type I (Hypoxemic)
- Type II (Hypercapnic)



To differentiate between type 1 and type 2 respiratory failure in mechanisms and clinical manifestations.

	Type I (Hypoxemic)	Type II (Hypercapnic)
definition	Occurs when normal gas exchange is seriously impaired, causing hypoxemia (arterial oxygen tension [PaO2] <60 mm Hg or arterial oxygen saturation [SaO2] <90%). Usually associated with tachypnea and hypocapnia; however, progression can lead to hypercapnia as well. In this type, the gas exchange is impaired at the level of alveolar-capillary membrane.	Occurs with acute elevation of carbon dioxide (arterial carbon dioxide tension n [PaCO2] >50 mm Hg), producing a respiratory acidosis (pH <7.35) (Hypoxemia is common) Mechanism: It is due to pump failure either a failure of the lung or something outside the lung i.e. when the alveolar ventilation is insufficient to excrete the CO2 being produced.
PaO2	< 60 mmHg	Normal or < 80 mmHg
PaCO2	Normal or < 33 mmHg	> 50 mmHg
Clinical findings	➢ Restless ➢ Confusion ➢ Delirium ➢ Tachypnea ➢ Tachycardia ➢ Cyanosis ➢ Tremor	➢ Dyspnea ➢ Headache ➢ Peripheral and conjunctival hyperemia ➢ Impaired level of consciousness ➢ Papilledema ➢ Asterixis

Mechanisms of Hypoxemia



1. Low ambient oxygen

- High altitude leads to low inspired oxygen

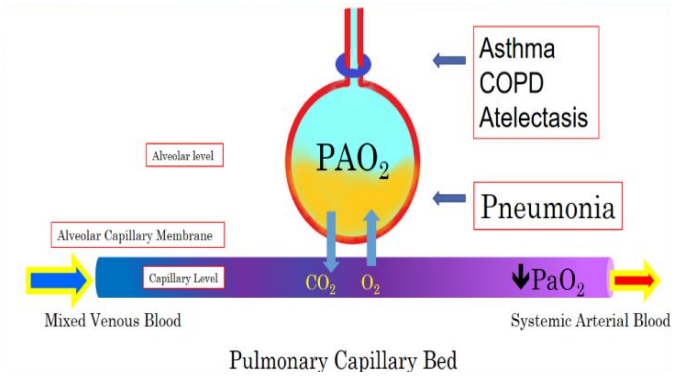
2. Alveolar hypoventilation

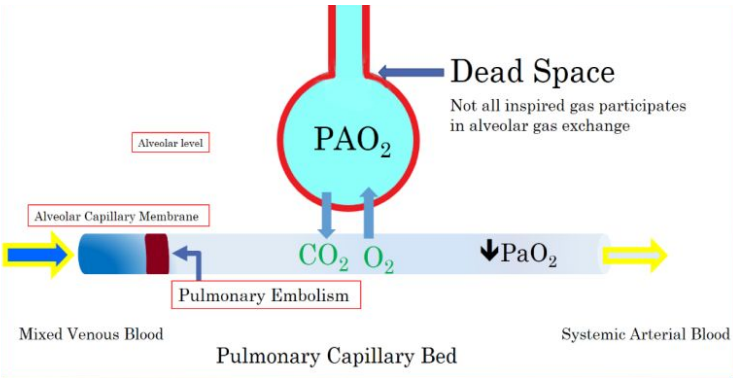
Due to in decreased minute ventilation This form can cause type I RF then develops Type II Respiratory Failure, for Example:

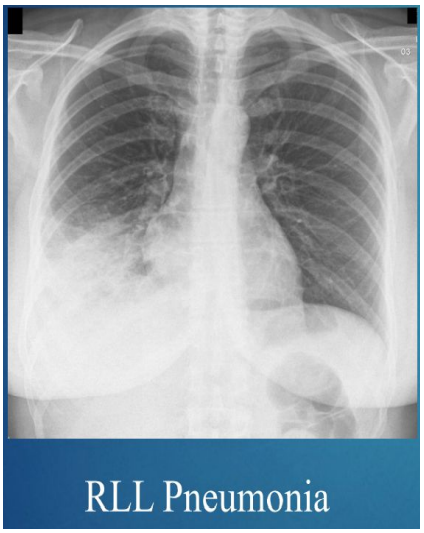
- Acute neuromuscular disease. (Guillain barre disease, Myasthenia gravis)
- Drug overdose, stroke (both acute),
- trauma or spinal injury especially C3-4-5 causing diaphragm paralysis

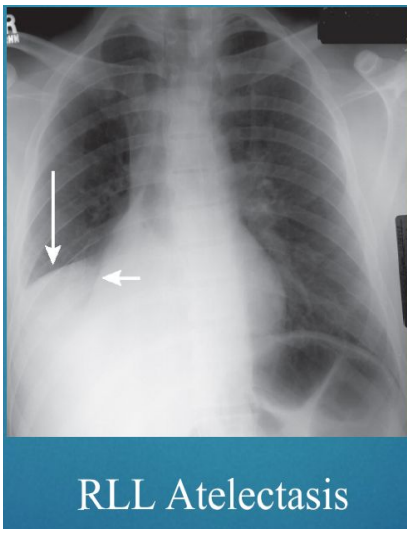
3. Ventilation-Perfusion Mismatch (V/Q) the most important mechanism

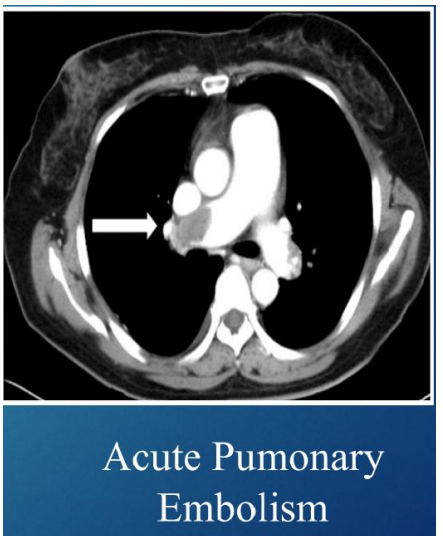
Parts of the lung receive oxygen but not enough to gas exchange
Examples of Hypoxemia due to V/Q mismatch :





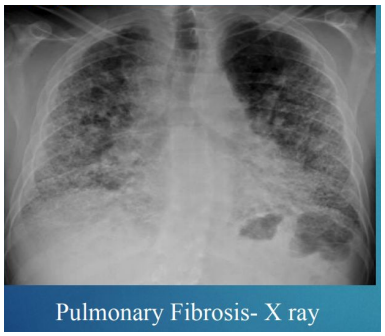

RLL Pneumonia


RLL Atelectasis

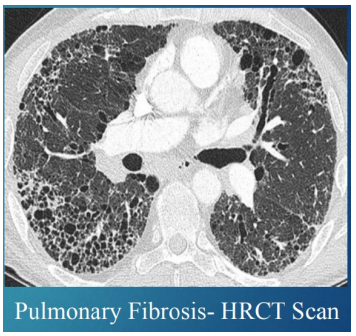

Acute Pumonary Embolism

4. Diffusion Abnormalities

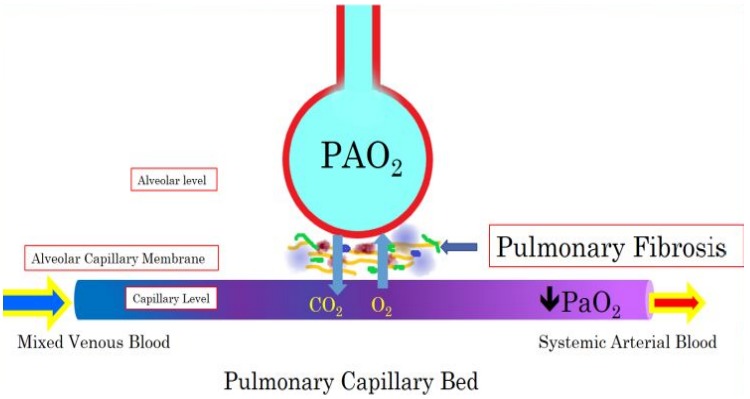
Oxygen transport across alveolar capillary membrane is impaired due to deposition of fibrotic tissue (pulmonary fibrosis)



Pulmonary Fibrosis- X ray

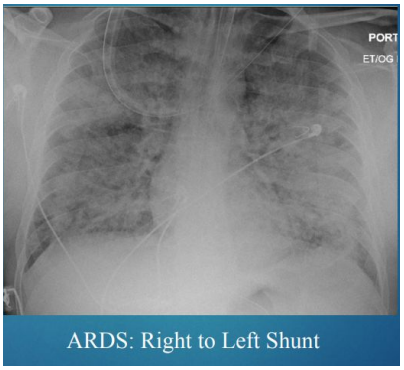


Pulmonary Fibrosis- HRCT Scan

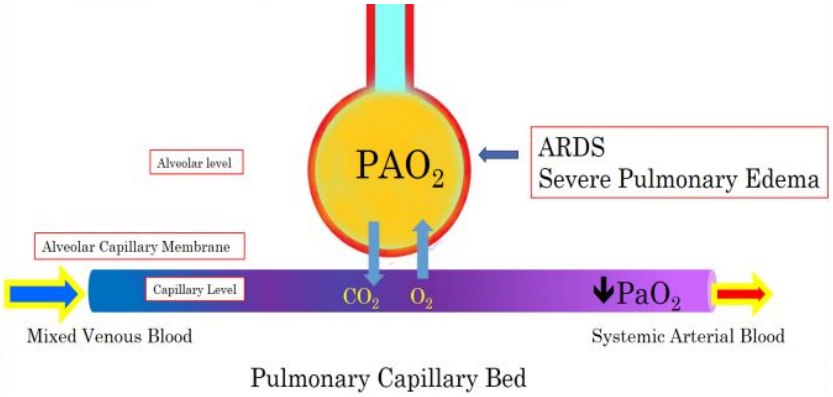


5. Shunt 2nd most important mechanism

The deoxygenated blood (mixed venous blood) bypasses the alveoli without being oxygenated, Alveoli is flooded with fluid. This mechanism is commonly seen in ICU patients they have to be intubated



ARDS: Right to Left Shunt



Clinical features

A clinical assessment of respiratory distress should be made on the following criteria:

- Use of accessory muscles of respiration
- Intercostal recession
- Tachypnoea
- Tachycardia
- Sweating
- Pulsus paradoxus (rarely present)
- Inability to speak, unwillingness to lie flat
- Agitation, restlessness, diminished conscious level
- Asynchronous respiration (a discrepancy in the timing of movement of abdominal & thoracic compartments)
- Paradoxical respiration (abdominal and thoracic compartments move in opposite directions)
- Respiratory alternans (breath-to-breath alteration in the relative contribution of intercostal/accessory muscles and the diaphragm)

To recognize the common causes of respiratory failure.

Type I (Hypoxemic)

Common etiologies:

- Pulmonary
- Edema
- ARDS
- pneumonia
- COVID-19 pneumonia

Type II (Hypercapnic)

Common etiologies:

- drug overdose, acute
- neuromuscular disorder, chronic
- chest wall abnormalities,
- sleep apnea syndrome,
- COPD
- Insult to the brain
- Destruction of the lung (severe COPD, severe bronchiectasis)

17.16 How to interpret blood gas abnormalities in respiratory failure

	Type I		Type II	
	Hypoxia (PaO_2 <8.0 kPa (60 mmHg)) Normal or low $PaCO_2$ (≤6 kPa (45 mmHg))		Hypoxia (PaO_2 <8.0 kPa (60 mmHg)) Raised $PaCO_2$ (>6 kPa (45 mmHg))	
	Acute	Chronic	Acute	Chronic
H^+	→	→	↑	→ or ↑
Bicarbonate	→	→	→	↑
Causes	Acute asthma Pulmonary oedema Pneumonia Lobar collapse Pneumothorax Pulmonary embolus ARDS	COPD Lung fibrosis Lymphangitic carcinomatosis Right-to-left shunts	Acute severe asthma Acute exacerbation of COPD Upper airway obstruction Acute neuropathies/paralysis Narcotic drugs Primary alveolar hypoventilation Flail chest injury	COPD Sleep apnoea Kyphoscoliosis Myopathies/muscular dystrophy Ankylosing spondylitis

(ARDS = acute respiratory distress syndrome; COPD = chronic obstructive pulmonary disease)

Acute respiratory distress syndrome (ARDS)

Is a form of hypoxemic respiratory failure caused by an acute lung injury. The common end result is disruption of the alveolocapillary membrane, leading to increased vascular permeability and accumulation of inflammatory cells and protein-rich edema fluid within the alveolar space.

The ARDS Definition Task Force redefined ARDS as follows:

- Onset within 1 week of a known clinical insult or new or worsening respiratory symptoms;
- Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules;
- Respiratory failure not fully explained by cardiac failure or volume overload; and
- Impaired oxygenation with low PaO2 to fraction of inspired oxygen (FIO2) ratio (PaO2 /FIO2 ≤300 mm Hg)

Severity of ARDS stratified based on PaO2 /FIO2 :

- Mild: 200 mm Hg < PaO2 /FIO2 ≤300 mm Hg with positive end-expiratory pressure (PEEP) ≥5 cm H2O
- Moderate: 100 mm Hg < PaO2 /FIO2 ≤200 mm Hg with PEEP ≥5 cm H2O
- Severe: PaO2 /FIO2 ≤100 mm Hg with PEEP ≥5 cm H2O

To apply invasive and non-invasive modalities of management in respiratory failure.

Management of respiratory failure

Approach

- Use ABCDE approach.
- Provide immediate respiratory support tailored to the underlying cause and severity of respiratory failure.
- Identify and treat rapidly reversible causes of respiratory failure.
- Perform focused clinical evaluation.
- Obtain initial diagnostics: e.g., ABG, routine laboratory studies, ECG, CXR, POCUS
- Treat the underlying cause of respiratory failure.

Respiratory support

- **Airway management:** Secure the airway if clinical features of airway compromise are present.
- **Rescue breaths:** indicated immediately for patients in respiratory arrest
- **Oxygen therapy:** Indicated for SpO2 < 95%
  - Nasal Cannula
  - Face Mask
  - Non- Rebreather Mask
  - Venturi Mask
- **Mechanical ventilation**
  - Non-Invasive Mechanical Ventilation: CPAP or BIPAP
  - Invasive Mechanical Ventilation: endotracheal or tracheostomy tube

Noninvasive Oxygen Therapy	Invasive Oxygen Therapy
➢ **Nasal cannulas:** most commonly used, Each additional liter of flow increases FIO2 by approximately 4%. Flow rates should be limited to ≤5 L/min. ➢ **Humidified high-flow nasal cannula:** deliver heated & humidified oxygen at higher flows and concentration up to 50 L/min & 100% FIO2. ➢ **Venturi masks:** allow the precise administration of oxygen. Usual FIO2 values delivered are 24%, 28%, 31%, 35%, 40%, 50%. ➢ **Nonrebreathing masks:** achieve higher oxygen concentrations (80-90%). A one-way valve prevents exhaled gases from entering the reservoir bag, maximizing the FIO2 . ➢ **Noninvasive positive-pressure ventilation (NPPV):** Includes continuous positive airway pressure (CPAP) & bilevel positive airway pressure (BPAP) ventilation, Use limited to patients who are conscious, cooperative, able to protect their airway, hemodynamically stable	➢ **Oral and nasopharyngeal airways:** ➢ **Bag-valve-mask ventilation:** Ineffective respiratory efforts can be augmented with simple bag-valve-mask ventilation. ➢ **Laryngeal mask airway (LMA):** it is a temporary airway and should not be used for prolonged ventilatory support. ➢ **Endotracheal intubation:** Indications are airway protection, inadequate oxygenation with less invasive methods, prevention of aspiration, excessive pulmonary secretions, and hyperventilation as a treatment for increased intracranial pressure

To be able to identify the severity of asthma and to understand the basics of management.

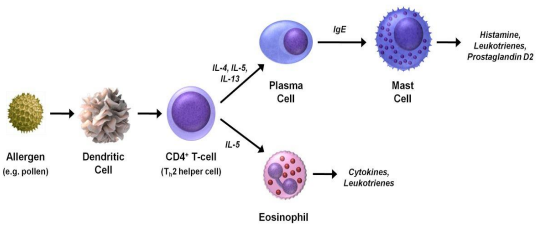
Asthma

Introduction :

- Asthma is a chronic lung disease due to **inflammation** of the airways resulted into airway obstruction. The obstruction is reversible.
- Asthma is the most common chronic disease particularly among children.

Symptoms :

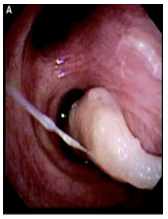
- Cough, wheeze, chest tightness, SOB, nocturnal symptoms.

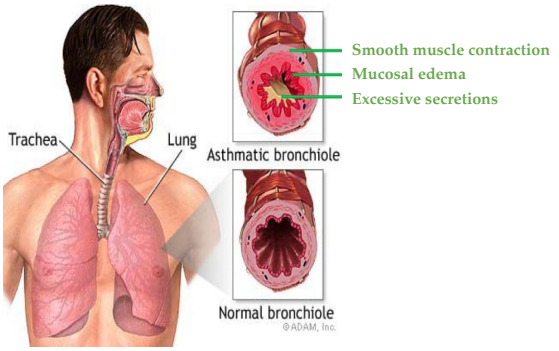


Pathophysiology:

Triad of:

- Airway inflammation (accumulation of Histamine, Leukotrienes, Prostaglandins)
- Airway hyperresponsiveness
- Reversible airflow obstruction leading to difficulty in exhaling air (air trapping)

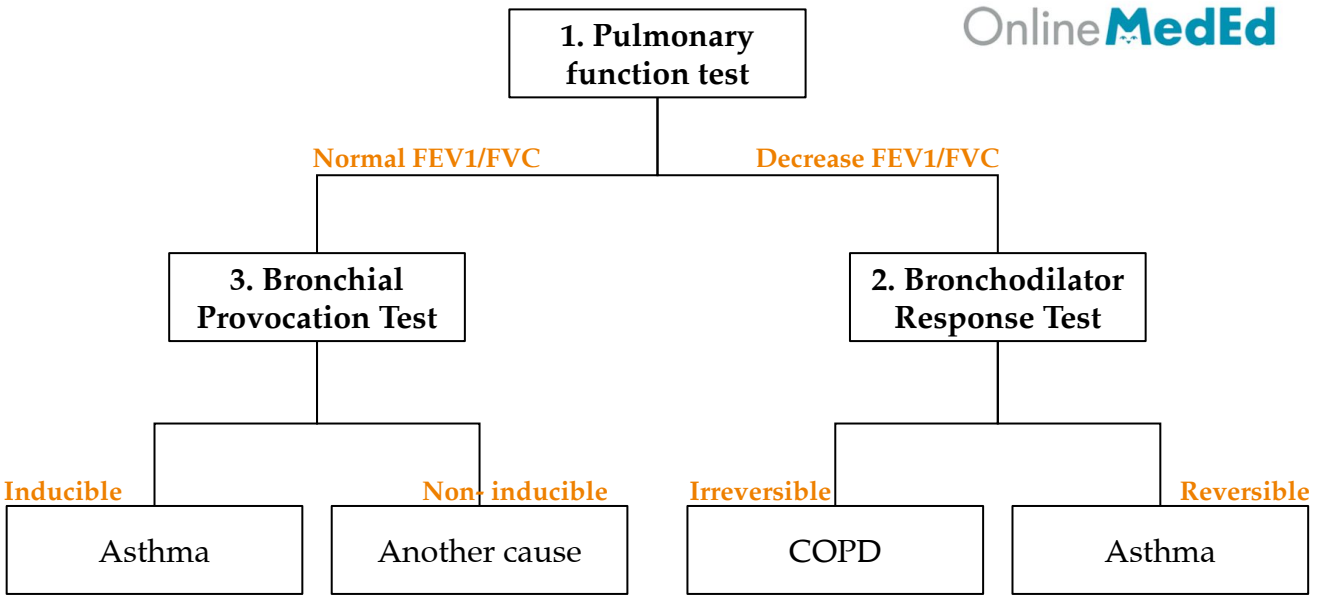




Triggers:

- Air pollutants (Tobacco smoke, perfumes, wood dusts, gases, paint)
- Pollen (Trees, flowers, weeds, plants)
- Animal dander (Birds, cats, dogs)
- Medication (Aspirin, anti-inflammatory drugs, B-blockers)
- Food (Eggs, nuts, wheat)

Diagnosis :



To be able to identify the severity of asthma and to understand the basics of management.

Treatment for Stable Patient (in general) :

- Exposure Risk
- Reduce exposure to indoor allergens
- Avoid tobacco smoke
- Avoid vehicle emission
- Identify irritants in the workplace
- **SABA** (Quick Reliever used in acute attacks, Begins to work immediately and peaks at 5- 10 minutes) (combined with ICS)
- **Inhaled Corticosteroids** 1st line (Mainstay treatment of asthma, Reduce airway inflammation)
- **Spacers** can help patients who have difficulty with inhaler use and can reduce potential for adverse effects from medication.
- **Nebulizers** (Machine produces a mist of medication that is used for small children or for severe asthma, No evidence that it is more effective than an inhaler used with spacers)

Acute Severe Asthma (Status Asthmaticus)

- Severe asthmatic attack unresponsive to repetitive courses of beta-agonist therapy
- A medical emergency that requires immediate recognition and treatment

Manifestation of severe asthma (History of):

- Past history of sudden severe exacerbation.
- Prior intubation and mechanical ventilation for asthma.
- Prior admission to ICU due to severe attack of asthma.
- Three or more emergency visits for asthma in the past year.
- Use of more than 2 canisters per month of inhaled short acting b2 agonist.
- Current use or recent withdrawal from systemic corticosteroids.

Clinical Features

- HR > 115/min.
- RR > 30/min.
- Pulsus paradoxus > 10 mmHg.
- Unable to speak. (Due to air trapping)
- Cyanosis.
- Silent chest (due low flow of air)

Indicates very severe state

- **Change in mental status.**
- **Cardiac arrhythmias and hypotension.**
- **Drowsiness, confusion and obtundation.**
- Peak expiratory flow meter <200 L/min.

Approach

- **Peak Expiratory Flow Rate (PEFR) :** assess severity and patient response to treatment
- **Arterial blood gases:** Acidemia, hypoxemia, hypercapnia, based on the findings of :

	PH	PCO2	PO2
1	↑	↓	N or ↓
2	N	N	↓
3	↓	↑	↓↓

To be able to identify the severity of asthma and to understand the basics of management.

Treatment of Severe Asthma:

1. **Oxygen:**
   - High concentration of oxygen to achieve **O2 Sat >92%**
   - Failure to achieve appropriate oxygenation and acidemia → assisted ventilation

2. **High doses of inhaled bronchodilator:**
   - Short acting B_2 agonist, (given hourly until the pt improves)
     - via nebulizer OR
     - via metered dose inhaler through a spacer device
   - An inhaled anticholinergics (Ipratropium bromide)
   - It has synergistic effect with B_2 agonist

3. **Systemic Corticosteroids**
   - Oral or IV hydrocortisone for those who are unable to swallow or in case of vomiting or disturb level of consciousness
   - It decreases mucus production
   - Improves oxygenation
   - Decreases bronchial hypersensitivity

4. **Intravenous Fluids (To correct dehydration and acidosis)** **to reduce and improve the mucus blogging.**
   - Normal saline infusion
   - Potassium supplement to treat hypokalemia induced by salbutamol

5. **IV $MgSO_4$** → Relaxes smooth muscles (if the pt isn't responding)

6. **Heliox** → Improves laminar flow (given in the ICU)

7. **Ketamine** → Anesthetic agent induced bronchodilation, It has anticholinergic effects (given in the ICU)

Non-invasive Mechanical Ventilation Treatment

Can be used in severe asthmatic attack but its efficacy is **not proven (IMP)**

Initial Goals:

- To correct hypoxemia
- To achieve adequate alveolar ventilation
- To minimize circulatory collapse
- To buy time for medical management to work

Indication:

- Coma
- Respiratory arrest
- Deterioration of arterial gas despite optimal therapy
- Exhaustion, confusion, drowsiness

Indication for ICU Admission

- Drowsiness
- Confusion
- Silent chest
- Worsening hypoxemia despite supplemental oxygen
- Acidemia and hypercapnia

To understand the basics of management of COPD.

Chronic Obstructive Pulmonary Disease (COPD):

A disease state that is characterized by a chronic progressive, irreversible airflow obstruction and associated with an abnormal inflammatory response to **SMOKING**
Includes : Emphysema, Chronic bronchitis, and Small airway disease

- COPD is the 4th leading cause of death in the United States
- COPD has higher mortality rate than asthma
- Leading cause of hospitalization in the US
- 2nd leading cause of disability

COPD exacerbation

- an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough and/or sputum that is beyond day to day variation and is acute in onset.

COPD risk factors

- Smoking: most common cause
- Environmental exposure
  - Chemicals. Dust, fumes
  - Second hand smoke

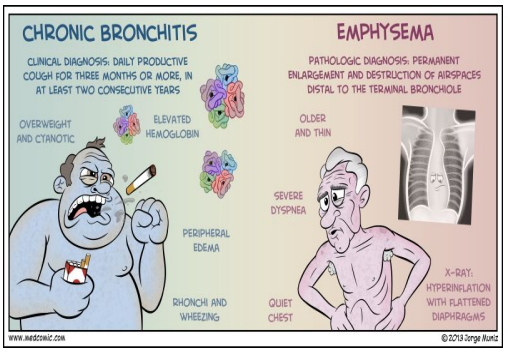
★ **Alpha-1 antitrypsin (AAT) deficiency** is a serine protease inhibitors, Inhibit neutrophil elastase which break down elastin which is more concentrated in the lower lobes.

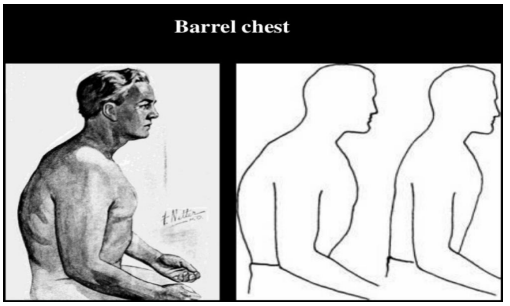
- Synthesized and secreted by hepatocytes
- PiZZ phenotype is associated with low plasma concentration of AAT i.e. associated with development of emphysema

Emphysema	Chronic Bronchitis
Abnormal permanent enlargement of the airspace distal to the terminal bronchiole accompanied by destruction of their without obvious fibrosis *Spaces in parenchyma > 1 mm = abnormal.	Cough for 3 months in a year for 2 consecutive year.

COPD Clinical features:

- Wheezing
- Dyspnea–progressive
- Chronic cough, with or without clear/white sputum.
- Cyanosis
- Clubbing
- Right heart failure: Edema, JVD , Hepatosplenomegaly
- Barrel chest
- Hyperresonance on percussion
- Pursed lips to prolong expiration
- Weight loss
- Accessory muscle Hypertrophy





To understand the basics of management of COPD.

OXYGEN THERAPY

For COPD with severe hypoxemia:

- It improves quality of life.
- **Together with smoking cessation,** It improves survival.
- which is indicated in patient with **PaO2 60 mmHg (<8 kPa)**

Treatment of Acute Attack of COPD:

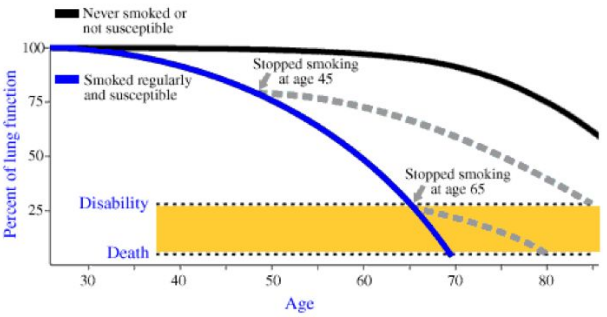
Assess severity of symptoms, blood gases, pulse oximetry, CXR

1. Oxygen therapy
   a. Low flow of oxygen to keep the SO2 ≈90% to avoid oxygen induced hypercapnia
2. Inhaled bronchodilators
3. Inhaled corticosteroids
4. Oral Antibiotics (if there is sign of infection)
5. Inhaled anticholinergic
6. Theophylline therapy
7. Consider non-invasive mechanical ventilation (NIV)

At all times :
monitor fluid balance
identify and treat underlying cause
Consider thromboembolism prophylaxis : LMW heparin or SC heparin

Prevention

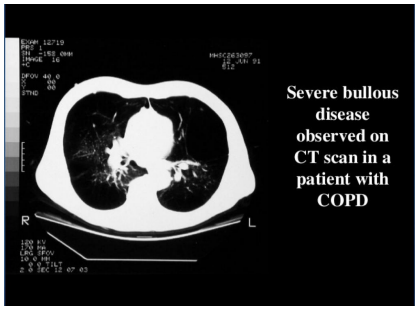
- Smoking cessation It improves survival.
- Annual Influenza Vaccine
- Polyvalent Pneumococcal polysaccharide vaccine
- COVID-19 Vaccination
- Pic: the peak lung function age is 25 then declines gradually, in smokers if they quit at 40 lung function will not return to the normal expected range

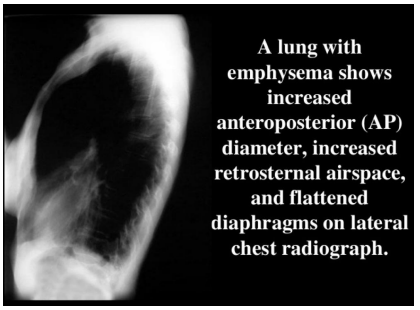


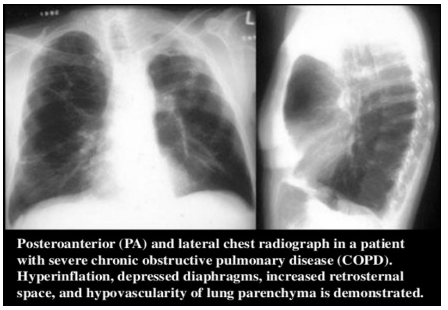
Changes in FEV1 with Aging (Smoker vs Non-Smoker)

Rehabilitation program

- Decreased symptoms (fatigue, dyspnea)
- Decreased anxiety and depression improved quality of life
- Decreased hospitalization
- Increase exercise capacity


Severe bullous disease observed on CT scan in a patient with COPD


A lung with emphysema shows increased anteroposterior (AP) diameter, increased retrosternal airspace, and flattened diaphragms on lateral chest radiograph.


Posteroanterior (PA) and lateral chest radiograph in a patient with severe chronic obstructive pulmonary disease (COPD). Hyperinflation, depressed diaphragms, increased retrosternal space, and hypovascularity of lung parenchyma is demonstrated.

To understand the basics of management of COPD.

Comt. Treatment of Acute Attack of COPD:

Non-Invasive Mechanical Ventilation (NIV)

Indication for Non-Invasive Mechanical Ventilation (NIV)	Non-Invasive Mechanical Ventilation
At least one of the following: • Respiratory acidosis: PCO2 ≥ 45mmHg (6 ≥ kPa) and pH < 7.35 • Severe dyspnea with clinical degree suggestive of respiratory muscle fatigue [1] • Persistent hypoxemia despite supplemental oxygen therapy	It reduce: • Intubation rate • Mortality in COPD patients with decompensated respiratory acidosis.

Mechanical Ventilation:

Indication	Initial Goals
• Unable to tolerate NIV or NIV failure • Status post- **respiratory or cardiac arrest** • Diminished consciousness, psychomotor agitation inadequately controlled by sedation (**Coma**) • Exhaustion, confusion, drowsiness • Massive aspiration or persistent vomiting • persistently unable to remove respiratory secretions • severe hemodynamic instability without response to fluids and vasoactive drugs • Sever ventricular or supraventricular arrhythmia • Life threatening hypoxemia in patients unable to tolerate NIV • Deterioration of arterial gas despite optimal therapy	• To correct hypoxemia • To achieve adequate alveolar ventilation • To minimize circulatory collapse • To buy time for medical management to work

Indication for ICU Admission

- Severe dyspnea that respond inadequately to initial emergency therapy
- Change in mental status (confusion, coma)
- Persistent or worsening hypoxemia PO2 < 50 mmHg and/ OR worsening respiratory acidosis pH < 7.25
- Need for mechanical ventilation e.g: apnea or respiratory arrest
- Hemodynamic instability
- need for vasopressor

1. Sign of respiratory muscle fatigue or Increase work of breathing or both : use of accessory muscles, paradoxical motion of the abdomen or retraction of the intercostal spaces

Lecture Quiz

1:C / 2:C

Q1: A 65-year-old male presents to the emergency department with worsening shortness of breath and confusion. He has a history of chronic obstructive pulmonary disease (COPD). On examination, he is tachypneic, cyanotic, and his oxygen saturation is 85% on room air. Arterial blood gas analysis reveals a pH of 7.28, PaO2 of 55 mmHg, and PaCO2 of 70 mmHg. What is the most likely type of respiratory failure in this patient?

A. Acute respiratory distress syndrome (ARDS)
B. Hypoxemic respiratory failure
C. Hypercapnic respiratory failure
D. Combined hypoxemic and hypercapnic respiratory failure

Q2: A 70-year-old male is admitted to the hospital with pneumonia and develops acute respiratory failure. He is initially started on supplemental oxygen, but his condition deteriorates further. His arterial blood gas analysis reveals a pH of 7.25, PaO2 of 58 mmHg, and PaCO2 of 78 mmHg. What is the next step in management for this patient?

A. Administer diuretics
B. Initiate non-invasive positive pressure ventilation (NIPPV)
C. Perform endotracheal intubation and initiate mechanical ventilation
D. Administer antibiotics