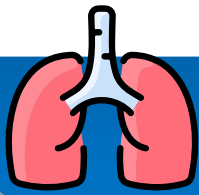




Round : Internal medicine.....

Group no : A4.....

Done By : Batool ALzubaidi.....



COPD

I. PATHOPHYSIOLOGY

- A. CHRONIC BRONCHITIS
- B. EMPHYSEMA

II. CLASSIC FINDINGS OF COPD

- A. CHRONIC BRONCHITIS
- B. EMPHYSEMA

III. COMPLICATIONS OF COPD

- A. CHRONIC BRONCHITIS
- B. EMPHYSEMA

IV. DIAGNOSTIC APPROACH TO COPD

- A. WORKUP FOR DYSPNEA SUSPECTED TO BE SECONDARY TO COPD
- B. ASSESS FOR THE PRESENCE OF AN OBSTRUCTIVE LUNG DISEASE LIKE COPD

V. TREATMENT OF COPD

- A. TREATMENTS THAT ↓ MORTALITY IN COPD
- B. TREATMENTS THAT IMPROVE SYMPTOMS IN COPD
- C. MANAGEMENT OF ACUTE COPD EXACERBATION

I. PATHOPHYSIOLOGY

A. CHRONIC BRONCHITIS

01:51

1. Pathophysiology:

- Chronic bronchiole inflammation → ↑Bronchospasm, ↑Mucus secretion and ↑Airway fibrosis → Narrowing of the bronchiole lumen → Inability for air to escape from alveoli → Airway obstruction → Air trapping → dynamic hyperinflation ensues
 - Dynamic hyperinflation → Triggers difficulty breathing via a short inspiration phase and a prolonged expiratory phase

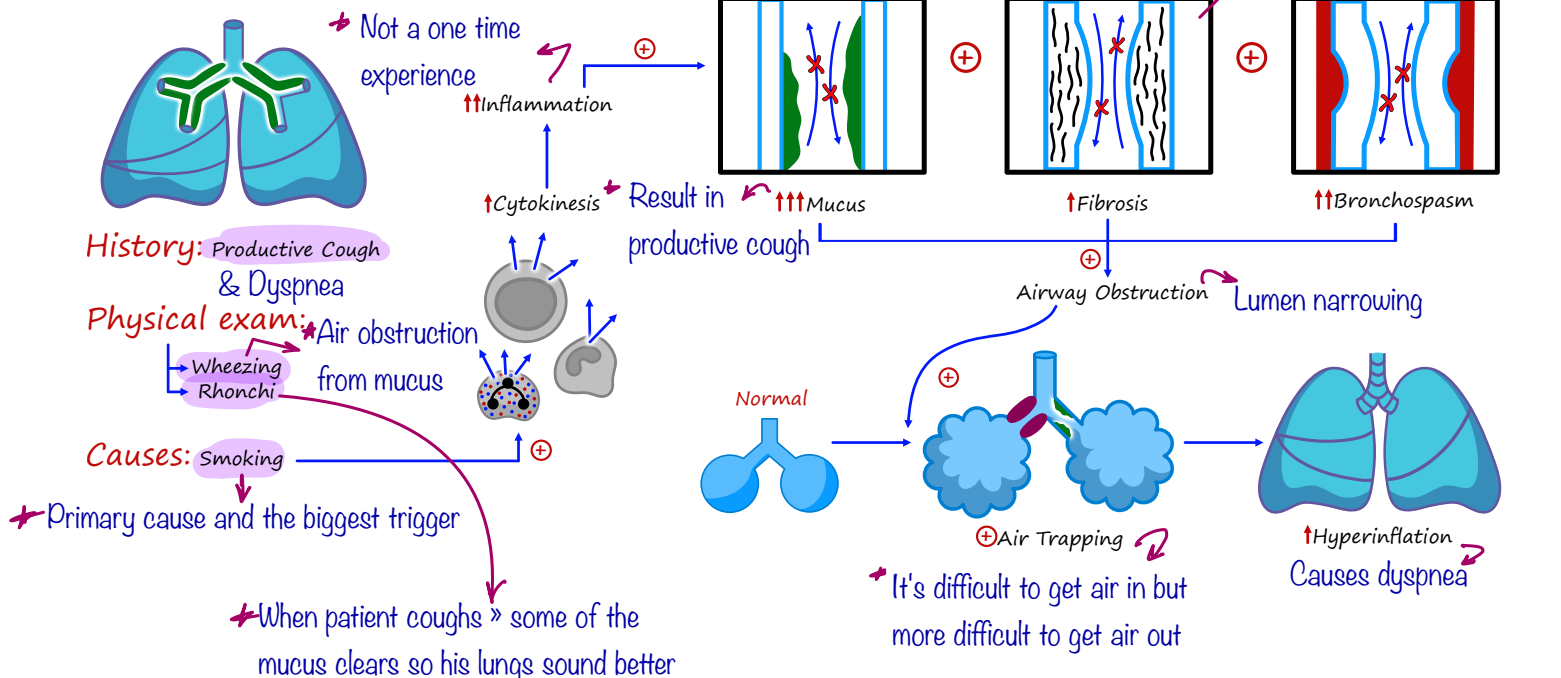
2. Causes of Chronic Bronchitis:

Smoking

- Chronic cigarette smoking → Chronic airway inflammation
 - This includes both active smoking and secondhand smoking exposure

Chronic Bronchitis

Pathophysiology



1. Pathophysiology:

- Chronic bronchiole inflammation → ↓ Alpha-1-Antitrypsin levels → ↑ Elastases present which remain unopposed → **Elastic tissue destruction of bronchioles and alveolar septa** → Bronchiole collapse and Airway enlargement (e.g. Acinus) → The airway enlargement leads to **impaired gas exchange** and The bronchiole collapse leads to **airway obstruction** → The combination of these leads to air trapping → **Dynamic hyperinflation**
 - Dynamic hyperinflation → Triggers difficulty in breathing via a short inspiration phase and a prolonged expiratory phase

2. Causes of Emphysema:

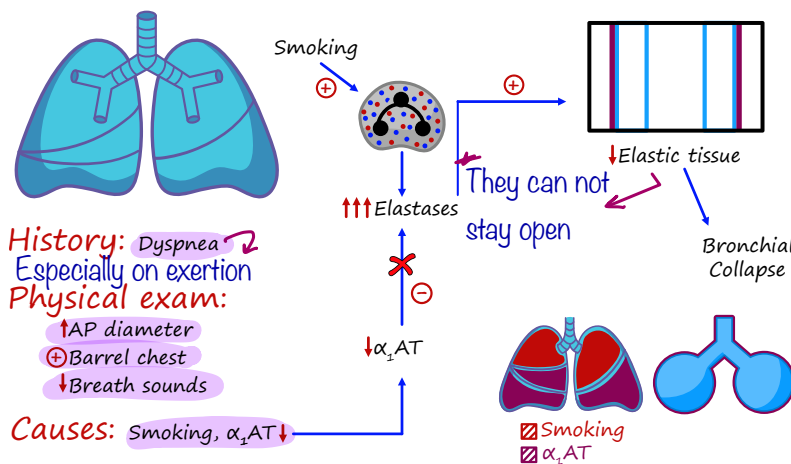
a) Smoking

- Chronic cigarette smoking → Chronic airway inflammation
 - This leads to upper lobe predominant emphysematous changes

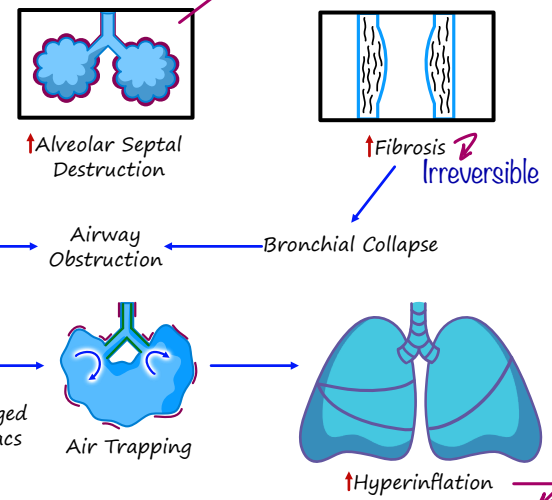
b) Alpha-1 Antitrypsin Deficiency

- Defective Alpha-1-Antitrypsin production by the liver → ↓ Alpha-1 Antitrypsin in the blood and therefore in the airways → ↑ Elastase activity → ↑ Bronchiole and alveolar septa destruction
 - This leads to lower lobe predominant emphysematous changes in a young (< 40 y.o) patient with associated liver disease

Emphysema



Pathophysiology



✚ Function of α₁AT » degradation or inhibition of elastases

✚ Deficiency » inhibition of this pathway » accumulation elastases » destruction of elastic tissue and fibrosis

✚ Much greater in this type because of enlarged sacs on top of the air trapping



II. CLASSIC FINDINGS OF COPD

A. CHRONIC BRONCHITIS

00:33

1. Productive Cough

- Chronic bronchiole inflammation → ↑↑ Mucus secretion
 - **Productive cough for > 3 months for 2 consecutive years** is a classic finding in chronic bronchitis

2. Wheezing

- Chronic bronchiole inflammation → ↑↑ Mucus secretion plugs up the bronchioles → Narrowing of the bronchiole lumen → **Loud expiratory noise** as air moves across the **narrowed airways**

3. Rhonchi

- Chronic bronchiole inflammation → ↑↑ Mucus secretion plugs up the bronchioles → Narrowing of the bronchiole lumen → **Snoring sounds heard during breathing**
 - Coughing clears the mucus from the airways which removes the snoring sound

B. EMPHYSEMA

08:16

1. Dyspnea on Exertion

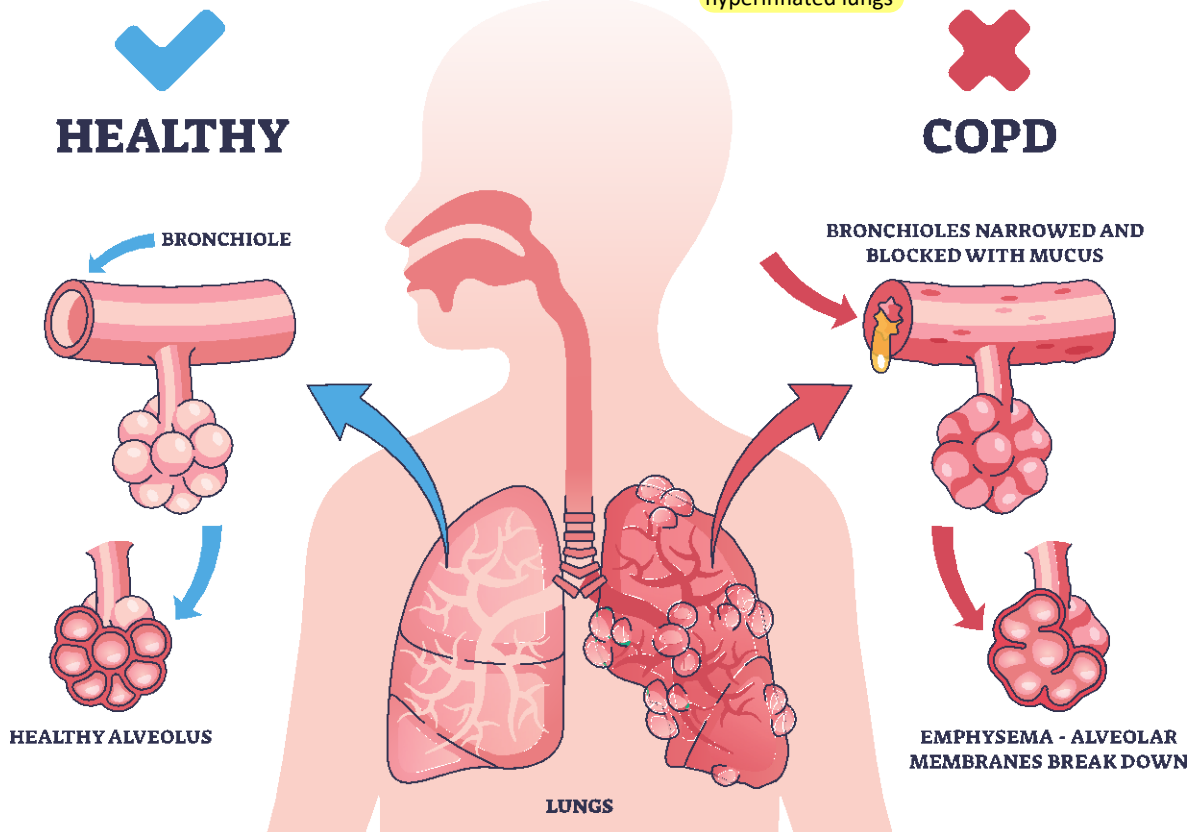
- The airway enlargement leads to **impaired gas exchange**, and the bronchiole collapse leads to **airway obstruction** → The combination of those two leads to **air trapping** → **Dynamic hyperinflation** → Triggers difficulty in breathing via a short inspiration phase and a prolonged expiratory phase
 - *Clinical Example: Try taking a deep breath, and then, without exhaling, try taking another deep breath on top of that. This is what it is like to have Emphysema* 😱

2. Wheezing

- Chronic bronchiole inflammation → Elastic tissue destruction of bronchioles → Bronchiole collapse during expiration → Narrowing of the bronchiole lumen → **Loud expiratory noise** as air moves across the **narrowed airways**

3. ↓ Breath Sounds Bilaterally

- The airway enlargement leads to impaired gas exchange, and the bronchiole collapse leads to airway obstruction → The combination of those two leads to air trapping → **Dynamic hyperinflation** → Difficulty in taking a deep breath leads to **less air movement in the lungs** and **less ability to auscultate the breath sounds**
 - Oftentimes these patients are referred to as having a **Barrel Chest**
 - The chest cage expands to accommodate the enlarged or hyperinflated lungs



III. COMPLICATIONS OF COPD

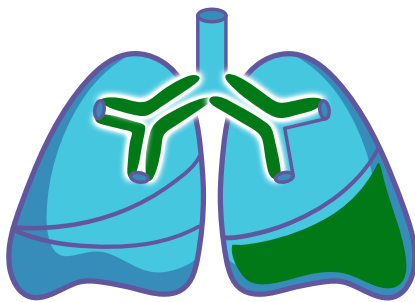
A. CHRONIC BRONCHITIS

1. Pneumonia

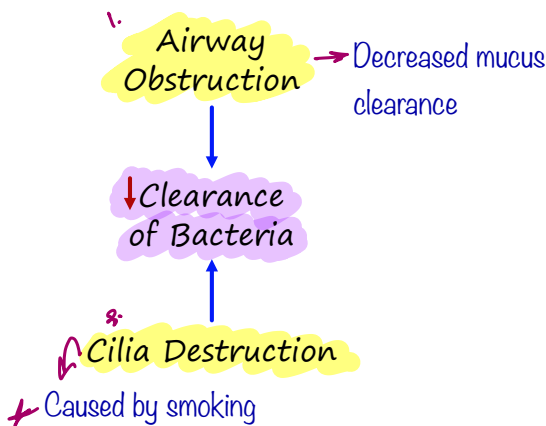
18:27

- **Pathophysiology:**
 - Chronic bronchiole inflammation → ↑↑ Mucus secretion and Poor mucus clearance due to ciliary dysfunction → Airway obstruction → ↓ Clearance of bacteria from the airways
- **Presentation:**
 - **Productive cough**
 - A more productive cough than usual
 - **Fevers and Leukocytosis**
 - Potentially a sign of more significant inflammation in the setting of pulmonary infection
 - **Acute Respiratory Failure in the setting of PNA**
 - Potentially due to the consolidation of alveoli worsening gas exchange
 - Assess for ↑RR and ↑WOB
 - Assess for Hypoxia
 - CXR with lobar consolidation

Chronic Bronchitis



Pneumonia

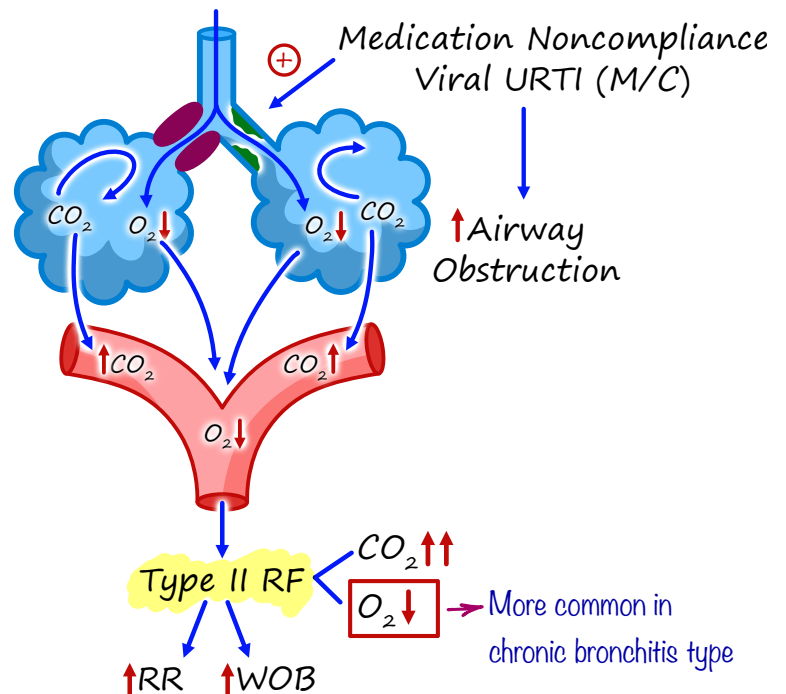


2. Respiratory Failure (Acute COPD Exacerbation)

20:22

- **Pathophysiology:**
 - Acute on Chronic bronchiole inflammation → ↑↑ Mucus secretion and Bronchospasm → Worsening airway obstruction → Worsening air trapping and dynamic hyperinflation → Severe Hypoventilation
 - The acute trigger is URTI or being noncompliant with bronchodilators → Impaired clearance of CO₂ » will accumulate in the blood » hypercapnia
- **Presentation:**
 - ↑RR and ↑WOB
 - Wheezing on auscultation
 - **ABG with Respiratory Acidosis suggests Type II RF**
 - ↓PaO₂ (Hypoxemia) → Air trapping » difficulty to take a breath » decrease in tidal volume » decrease in O₂ delivery » decrease in blood » hypoxemia
 - ↑PaCO₂ (Hypercapnia)
 - ↓pH (Acidosis)

Respiratory Failure Acute COPD Exacerbation [AECOPD]



✚ Hypoxemia is a potent vasoconstrictor

3. Cor Pulmonale

24:55

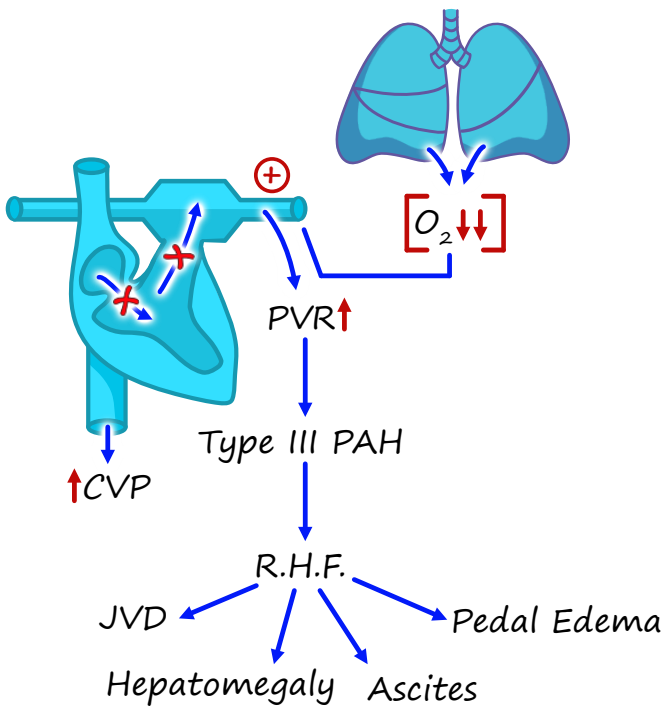
• Pathophysiology:

- Chronic respiratory failure → **Chronic Hypoxic Pulmonary Vasoconstriction** → ↑ Pulmonary vascular resistance → **Increases after load**
- ↑ Pulmonary artery pressures → **Right heart strain** arises (e.g. RVH) → **Right heart failure** develops from chronic right heart strain

• Presentation:

- JVD (related to SVC backflow)
- Hepatomegaly (related to IVC backflow)
- Ascites (related to IVC backflow)
- Lower extremity edema (related to IVC backflow)

Cor Pulmonale



4. Polycythemia

27:42

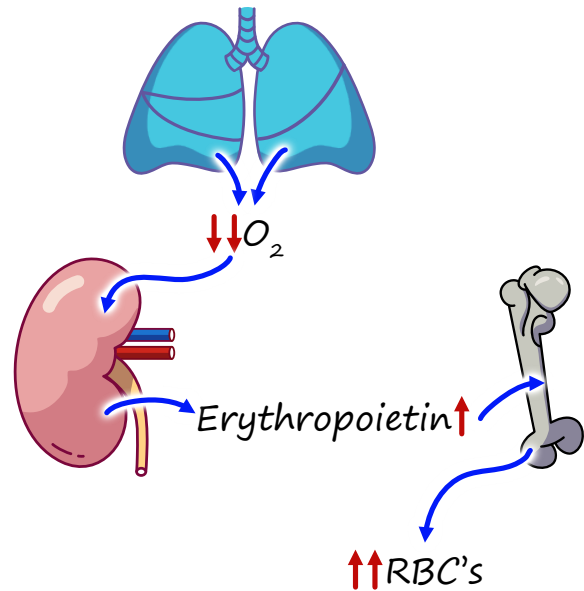
• Pathophysiology:

- Chronic respiratory failure → **Chronic Hypoxemia** → ↑ Renal production of Erythropoietin → Bone marrow increases its production of RBCs

• Presentation:

- ↑ Hgb and ↑ Hematocrit

Polycythemia

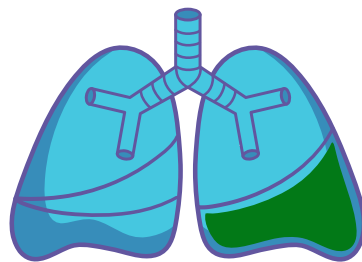


B. EMPHYSEMA

1. Pneumonia

- **Pathophysiology:**
 - Chronic bronchiole inflammation → ↑Elastic tissue breakdown of bronchioles → bronchiole collapse → Airway obstruction → ↓Clearance of bacteria from the airways
- **Presentation:**
 - **Productive cough**
 - A more productive cough than usual
 - **Fevers and Leukocytosis**
 - Potentially a sign of more significant inflammation in the setting of pulmonary infection
 - **Acute Respiratory Failure in the setting of PNA**
 - Potentially due to the consolidation of alveoli worsening gas exchange
 - Assess for ↑RR and ↑WOB
 - Assess for Hypoxia
 - CXR with lobar consolidation

Emphysema



Pneumonia

Airway Obstruction

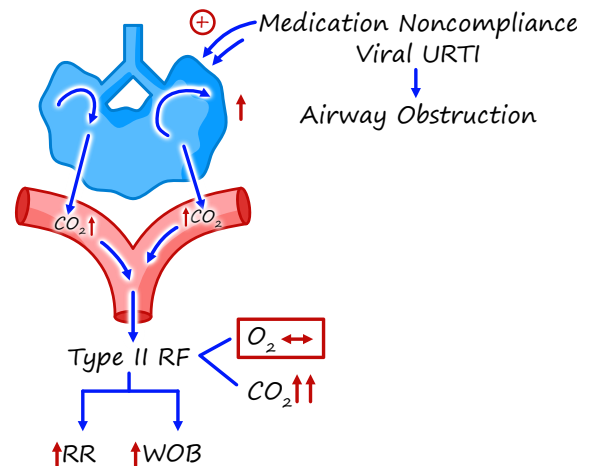
↓ Clearance of Bacteria

↑ Ciliary Dysfunction

2. Respiratory Failure (Acute COPD Exacerbation)

- **Pathophysiology:**
 - Acute on Chronic bronchiole inflammation → Worsening airway obstruction → Worsening air trapping and dynamic hyperinflation → **Severe Hypoventilation**
 - The acute trigger is **URTI** or being **noncompliant** with **bronchodilators**
- **Presentation:**
 - ↑RR and ↑WOB
 - Wheezing on auscultation
 - **ABG with Respiratory Acidosis suggests Type II respiratory failure**
 - ↑PaCO₂ (Hypercapnia)
 - ↓pH (Acidosis)
 - **Normal PaO₂**
 - These patients rarely exhibit hypoxemia → If present, points to a sign of extremely advanced emphysema → This is why **Emphysema patients do not experience Cor Pulmonale and Polycythemia**

Respiratory Failure Acute COPD Exacerbation [AECOPD]

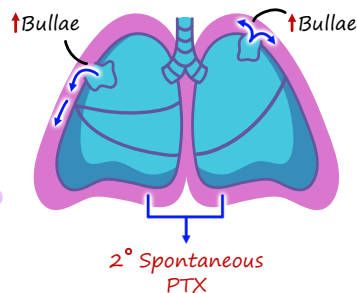


3. Pneumothorax

33:17

- **Pathophysiology:**
 - Airway enlargement leads to impaired gas exchange, and the bronchiole collapse leads to airway obstruction → The combination of these two leads to air trapping → Dynamic hyperinflation → **Bullae form** as a result of alveolar wall destruction and air trapping → Bullae may **rupture** into pleural space
 - Example of a **Secondary Spontaneous Pneumothorax**

Pneumothorax



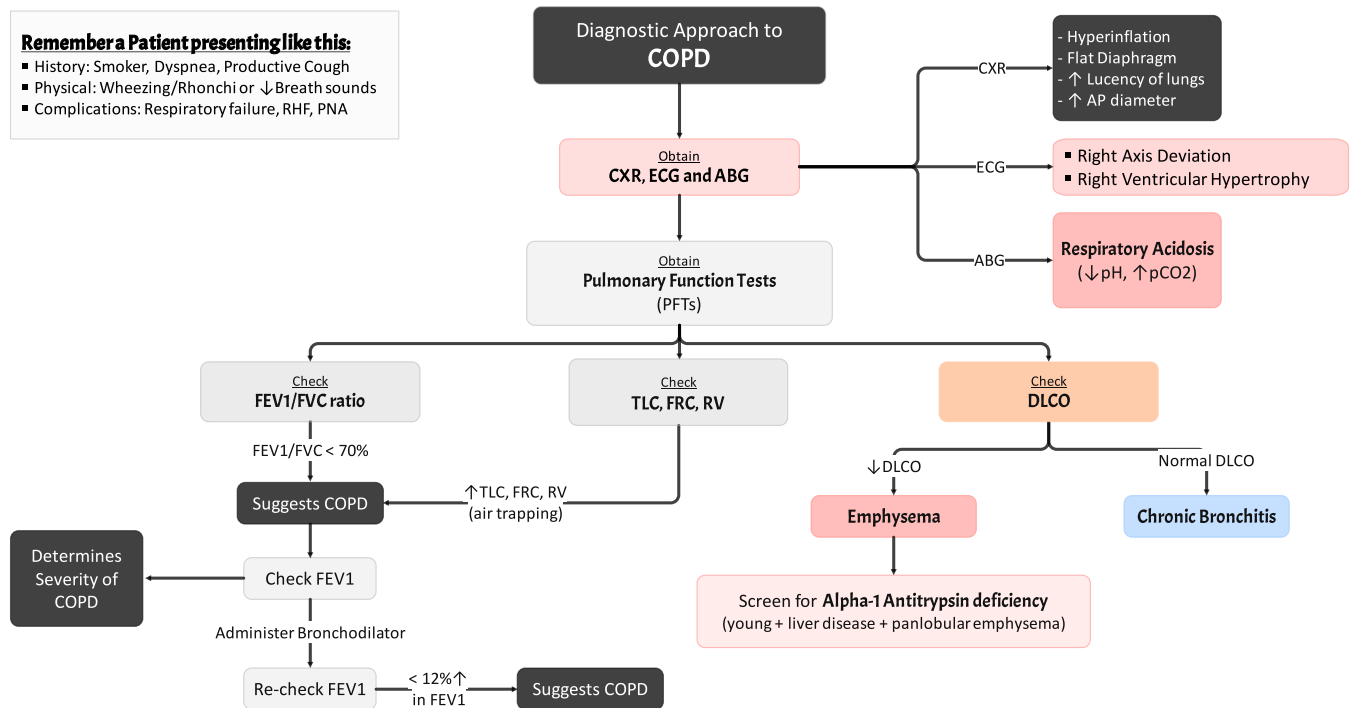
- **Presentation:**
 - Pleuritic chest pain
 - ↓Tactile fremitus on the affected side
 - Hyperresonance to percussion on the affected side
 - ↓Breath sounds on the affected side
 - **↑Risk of Tension Pneumothorax**
 - JVD
 - Hypotension/shock
 - Tracheal deviation to the **contralateral side**



IV. DIAGNOSTIC APPROACH TO COPD

Remember a Patient presenting like this:

- History: Smoker, Dyspnea, Productive Cough
- Physical: Wheezing/Rhonchi or ↓ Breath sounds
- Complications: Respiratory failure, RHF, PNA



A. WORKUP FOR DYSPNEA SUSPECTED TO BE SECONDARY TO COPD

1. Obtain CXR

- Indications:**
 - Rule out other pulmonary causes of dyspnea (e.g. PNA, Pulmonary edema, Pleural Effusions, Pneumothorax, etc.)
- Findings Supportive of COPD:**
 - Hyperinflation of the lungs causing hyperlucency
 - Flattening of the diaphragm
 - ↑ AP diameter on lateral CXR view → Barrel chest

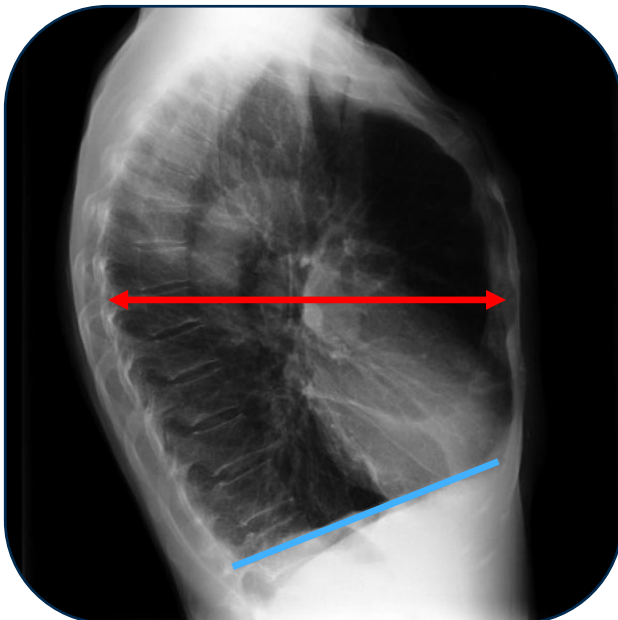


FIGURE 1. LATERAL VIEW ON CXR INDICATING ↑ AP DIAMETER & DIAPHRAGM FLATTENING

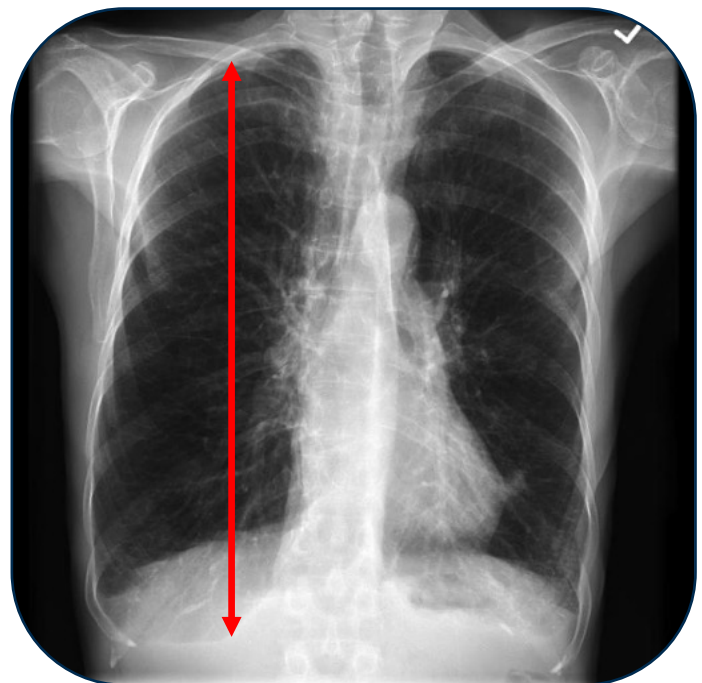


FIGURE 2. ANTERIOR VIEW ON CXR INDICATING HYPERINFLATION

✦ Increased darkness of lungs



2. Obtain 12 Lead ECG

- **Indications:**
 - Rule out cardiac causes of dyspnea (e.g., Acute Myocardial Infarction)
- **Findings Supportive of COPD:**
 - COPD may lead to Pulmonary HTN causing **Right Heart Strain** made evident by:
 - Right axis deviation (RAD)
 - Right Ventricular Hypertrophy (RVH)
 - Right Bundle Branch Block (RBBB)

3. Obtain ABG

- **Indications:**
 - **Respiratory Failure**
 - Differentiate Primary - Type I (Hypoxic) from Type 2 (Hypercapnic) Respiratory Failure
 - Determine the severity of COPD exacerbation
- **Findings Supportive of COPD:**
 - **Respiratory Acidosis**
 - $\uparrow \uparrow \text{PaCO}_2$ and $\downarrow \text{pH}$ suggest Type II respiratory failure
 - Hypoxemia ($\downarrow \text{PaO}_2$) may be seen in Chronic Bronchitis

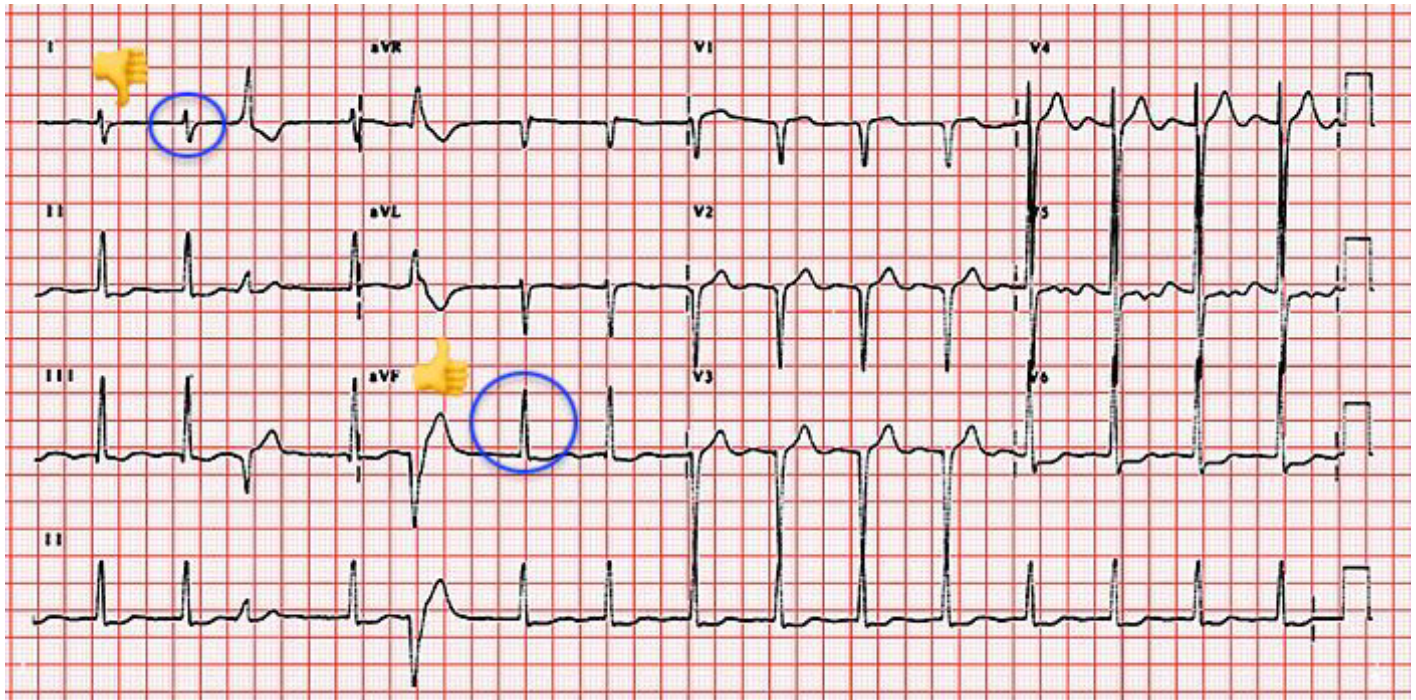


FIGURE 3. ECG INDICATING RIGHT AXIS DEVIATION

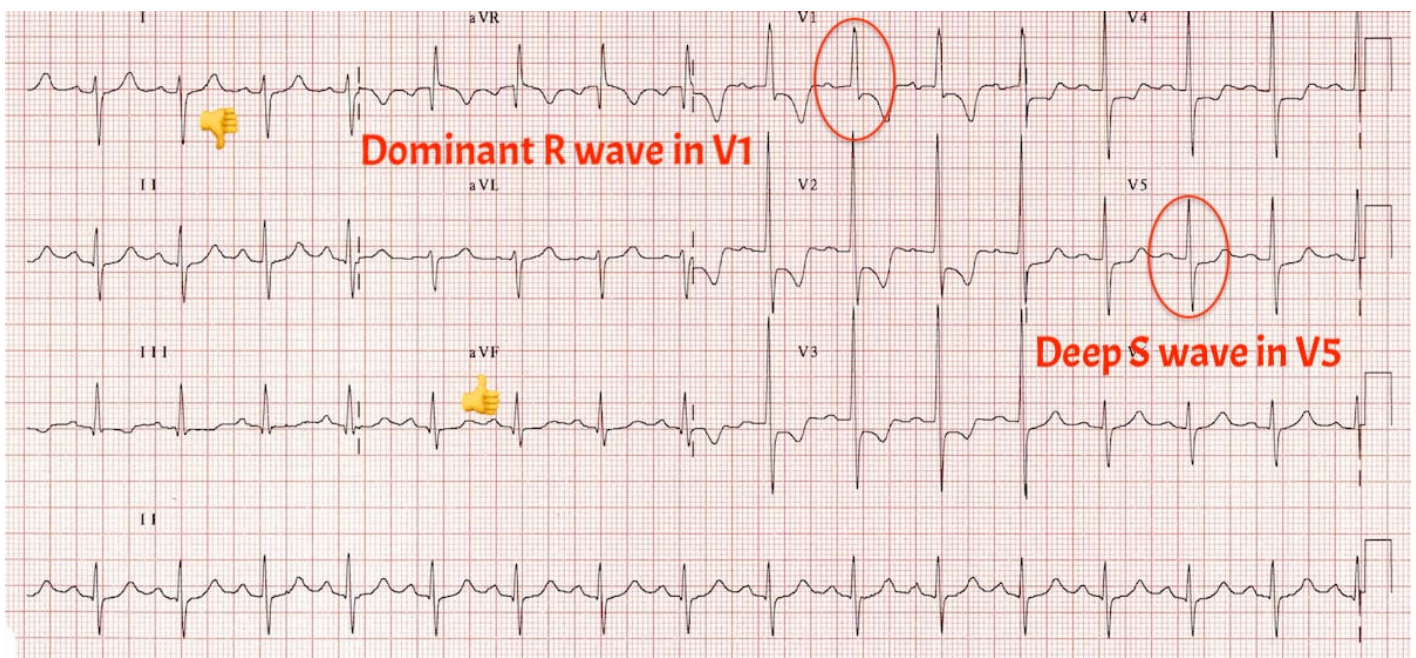


FIGURE 4. ECG INDICATING VENTRICULAR HYPERTROPHY



B. ASSESS FOR THE PRESENCE OF AN OBSTRUCTIVE LUNG DISEASE LIKE COPD

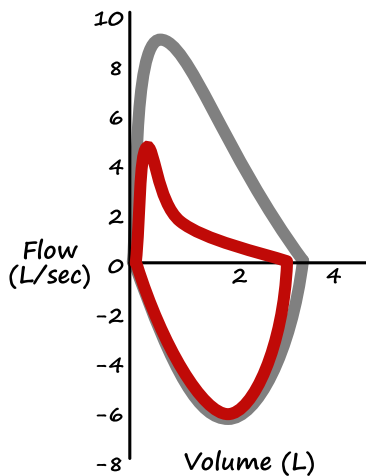
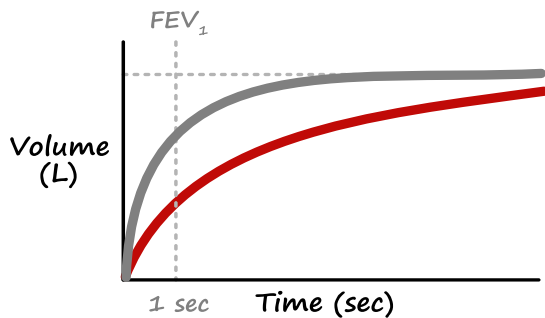
1. Obtain **Pulmonary Function tests** (PFTs)

- **Parameters of PFTs**

- FEV₁/FVC ratio
- FEV₁ post Bronchodilator administration
- FEV₁ Post Methacholine administration
- Diffusion limitation of carbon monoxide (DLCO)
- Lung Volumes (e.g. TLC, RV, and FRC)

- **Indications:**

- Determine if the patient has an Obstructive vs Restrictive lung disease



- **Findings Supportive of COPD:**

- **Obstructive Lung disease pattern that is reversible:**

- **FEV₁/FVC ratio < 70%** → Suggests Obstructive Lung Disease

- **FEV₁** helps to determine the **severity of COPD**

- ↓↓↓ FEV₁ → Supports ↑↑↑ Severity of COPD

- **↑↑ in FEV₁ of < 12% from baseline after bronchodilator** →

Supports an **irreversible** obstructive lung disease like **COPD**

- Bronchodilators (e.g. Albuterol) reduce resistance to expiratory flow

- **DLCO**

- **Emphysema → ↓DLCO**

- Emphysema **decreases alveolar surface area** → There is less surface area available for the diffusion of gases

- If the patient has lower lobe predominant emphysematous changes, liver disease, and is < 40 y of age → **Obtain Alpha-1 Antitrypsin level screening**

- **Chronic Bronchitis → Normal DLCO**

- Chronic Bronchitis does not affect the surface area or thickness of the respiratory membrane

DLCO

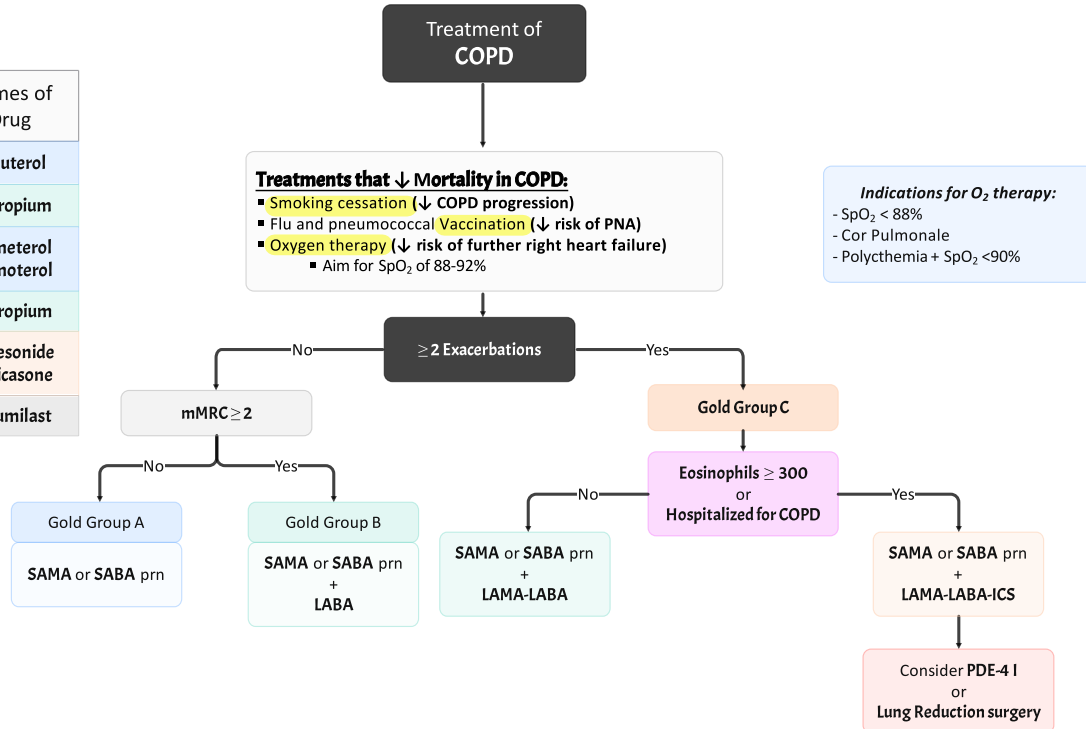
$$\downarrow \text{DLCO} \propto \frac{\text{TSA of R.M.} \downarrow}{\text{Thickness of R.M.}}$$

Emphysema → TSA ↓



V. TREATMENT OF COPD

Type of Drug	Names of Drug
SABA	Albuterol
SAMA	Ipratropium
LABA	Salmeterol Formoterol
LAMA	Tiotropium
ICS	Budesonide Fluticasone
PDE4-I	Roflumilast



A. TREATMENTS THAT ↓ MORTALITY IN COPD

- Treatment:**
 - Smoking Cessation
 - Vaccination (e.g. influenza and pneumococcal)
 - Oxygen therapy
- Purpose:**
 - Smoking Cessation:**
 - Smoking cessation → ↓ Airway inflammation → ↓ Progressive airway destruction → ↓ COPD progression
 - Vaccination:**
 - Influenza and pneumococcal vaccination → Lowers risk of LRTIs → **Lowers the risk of COPD exacerbations**
 - Oxygen Therapy:**
 - Oxygen therapy → **Reduces hypoxic pulmonary vasoconstriction** → Reduces progression to pulmonary HTN → Lowers risk of Cor Pulmonale progression
 - This is only indicated in the following:
 - SpO₂ < 88%
 - Cor Pulmonale
 - Polycythemia + SpO₂ < 90%
 - Higher threshold for oxygen therapy given polycythemia
- Monitoring:**
 - Monitor PFTs for improvement or stability
 - Monitoring SpO₂ for a goal of 88-92% while on oxygen therapy



B. TREATMENTS THAT IMPROVE SYMPTOMS IN COPD

● Treatment:

- Short-Acting Beta-2 Agonist (SABA):
 - Albuterol
- Short-Acting Muscarinic Antagonist (SAMA):
 - Ipratropium
- Long-Acting Beta-2 Agonist (LABA):
 - Salmeterol
 - Formoterol
- Long-Acting Muscarinic Antagonist (LAMA):
 - Tiotropium
- Inhaled Corticosteroids (ICS):
 - Budesonide
- PDE-4 Inhibitor:
 - Roflumilast

● Indications:

GOLD Group	≥ 2 Exacerbations/ year	mMRC ≥ 2	Eosinophils > 300 or Hospitalized for COPD	Therapy
Gold Group A	(-)	(-)	N/A	-SABA or SAMA prn
Gold Group B		(+)	N/A	-SABA or SAMA prn -Add LABA
Gold Group C	(+)	N/A	(-)	-SABA or SAMA prn -LABA daily -Add LAMA daily
			(+)	-SABA or SAMA prn -LABA + LABA daily -Add ICS daily

● Purpose:

- **SAMA, SABA, LABA, LAMA** → Promotes bronchodilation → Improves expiratory airflow
- **SAMA and LAMA** → Promotes reduction in mucus in bronchioles → Improves expiratory airflow
- **ICS** → Promotes reduction in bronchiole inflammation → Improves expiratory airflow

● Monitoring:

- Improvement in symptoms (e.g., dyspnea)
- Improvement in PFTs (e.g., FEV1)
- Reduced frequency of exacerbation

Stage	FEV1	mMRC 0-1	mMRC ≥ 2
I (mild)	$\geq 80\%$	[A] ▪ SAMA or SABA prn	[B] ▪ Add LABA or LABA
II (moderate)	$< 80\%$		
III (severe)	$< 50\%$	[C] ▪ Add ICS ▪ Consider adding PDE4-I	[D] ▪ ICS + LABA + LABA + SAMA or SABA prn ▪ Consider adding PDE4-I ▪ Oxygen therapy (Ind: Cor pulmonale, $SpO_2 < 88\%$ or $PaO_2 < 55\text{mmHg}$)
IV (very severe)	$< 30\%$		



C. MANAGEMENT OF ACUTE COPD EXACERBATION

1. Bronchodilators

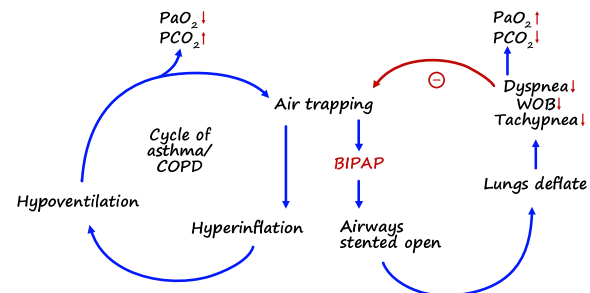
- **Treatment:**
 - Combination of **SABA** (e.g. Albuterol) and **SAMA** (Ipratropium)
 - These are often referred to as “**Duonebs**” and are often dosed every 6 hours
- **Indications:**
 - **First-line** therapy in a **COPD exacerbation**
- **Purpose:**
 - Promote bronchodilation and assist in expiratory airflow and resolution in air trapping
- **Monitoring:**
 - Improvement in wheezing, dyspnea, RR and WOB

2. Reduce Airway Inflammation

- **Treatment:**
 - **Systemic Corticosteroids**
 - If able to swallow pills → **Oral Prednisone** usually 40-60mg (depending on severity) daily x 5-7 days
 - If unable to swallow pills due to respiratory distress → **IV Methylprednisolone** usually 40-125 mg (depending on severity) every 6 hrs x 5-7 days
- **Indications:**
 - COPD exacerbation that is not improving with bronchodilator therapy
- **Purpose:**
 - Systemic Corticosteroids → Achieve higher than normal serum steroid levels → Promote massive reduction in airway inflammation → Massive reduction in bronchoconstriction → **Significantly improves expiratory airflow**
- **Monitoring:**
 - Improvement in wheezing, dyspnea, RR and WOB
 - Improvement in PEFR from baseline/pre-treatment

3. Reduce Work of Breathing

- **Treatment:**
 - Noninvasive Positive Pressure Ventilation (e.g. BIPAP)
 - Invasive Positive Pressure Mechanical Ventilation (e.g. Intubation)
- **Indications:**
 - Severe tachypnea/wheezing, ↑ WOB
 - ↑↑ PCO₂ (Hypercapnia) suggests severe obstruction and poor CO₂ clearance
 - Hypercapnia can also lead to severe lethargy, and potentially, comatose states, which can impair the protection of the airway
- **Purpose:**
 - **Positive Pressure Ventilation** (e.g. BIPAP or Intubation) → Stents bronchioles open given higher airway pressure delivery → Allows air to easily escape the lungs → The lungs properly deflate → **Allows the patient to easily be able to take a deep breath** → Allows CO₂ removal and improvement in tachypnea and WOB
- **Monitoring:**
 - Improvement in tachypnea, dyspnea, and WOB
 - Improvement in PCO₂ on serial ABGs
 - Improvement in mental status if severely hypercapnic



4. Reduce Bacterial Colonization

- **Treatment:**
 - **Antibiotics**
 - Azithromycin or Doxycycline for mild-moderate exacerbations
 - Fluoroquinolones (e.g. Levofloxacin) for severe exacerbations
- **Indications:**
 - **COPD Exacerbation**
 - Increased productive cough
 - Fever and leukocytosis
 - Respiratory failure requiring ventilation support
- **Purpose:**
 - Reduce bacterial load or clear infection → reduce airway inflammation → Reduce COPD exacerbation
- **Monitoring:**
 - Improvement in productive cough
 - Improvement in tachypnea, dyspnea, and WOB leading to less ventilation support
 - Improvement in fever and leukocytosis

Mechanism of Treatment	Treatment
Bronchodilation	SAMA + SABA
↓ Airway Inflammation	Systemic Steroids (IV or PO)
↓ Work of breathing	BIPAP
↓ Bacterial colonization	Azithromycin or Doxycycline

