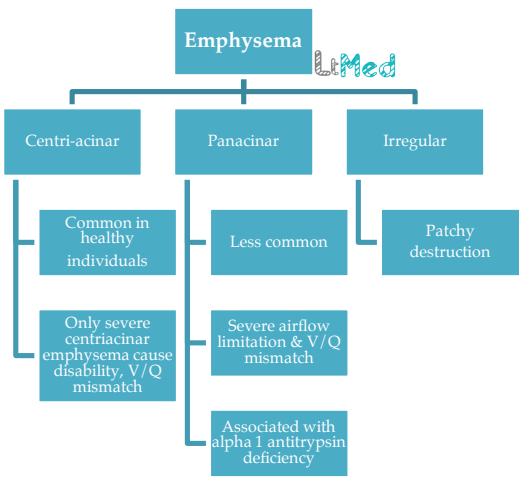
Chronic Obstructive Pulmonary Disease (COPD)

- **Definition:** a chronic disease characterized by progressive airflow limitation and not fully reversible with the presence of inflammatory response against toxic gases or particles.
  - *Airflow obstruction along with pulmonary parenchymal destruction.*
- **Epidemiology:**
  - *Smoking is the most common cause found in 90% of COPD cases in developed countries.*
  - Only 10-20% of all heavy smokers will develop COPD.
  - By 2020, it is predicted that COPD is going to be third most common cause of death & fifth most common cause of disability worldwide.
  - Mortality is higher in females.
- **Causes:**
  - *Smoking: the most common cause of COPD.*
  - Alpha 1 antitrypsin deficiency: only 2% of emphysema cases.
- **Pathophysiology:**
  - Microscopically:
    - Squamous cells will replace the normal columnar cells.
    - Bronchi & bronchioles infiltration with acute & chronic inflammatory cells with lymphoid follicles in severe cases.
    - Persistent inflammation will result *in scaring & fibrosis of bronchi & bronchioles causing narrowing and airflow limitation.*
  - If airflow limitation is combined with *loss of lung elastic recoil* and small airway collapse during expiration (*emphysema*).
    - *Emphysema is a secondary result of persistent inflammation and destruction.*
    - The resulting *V/Q mismatch* will decrease the PaO2 and increase the respiratory effort.
      - Later, if the patient fail to maintain the respiratory efforts the PaCO2 will increase, which will stimulate the respiratory center on the long-term (*those patients depend on hypoxemia to drive their ventilation and they become insensitive to CO2*).

Most common consistent pathological finding seen in COPD is *increased numbers of goblet mucus secreting cells.*

Emphysema: abnormal permanent enlargement of alveoli beyond the terminal bronchioles without obvious fibrosis.



Clinical correlation: Take full caution when giving supplemental O2 to COPD patient as that may *suppress their respiratory center and lead to death in-patient with chronic hypercabnia!*

- In summary, 3 mechanisms responsible for COPD:
  1. *Loss of elasticity of emphysema.*
  2. *Airway inflammation and scarring.*
  3. *Mucus plug.*

- **Signs & Symptoms:**
  - **Chronic Bronchitis (Blue Bloaters):**
    - Productive cough (white or clear sputum) >3months per year for 2 consecutive years.
    - Cyanosis and mild dyspnea.
    - Weight gain and obesity.
  - **EmPhysema (Pink Puffers):**
    - Minimal cough.
    - Dyspnea and pursed lips.
    - Weight loss.

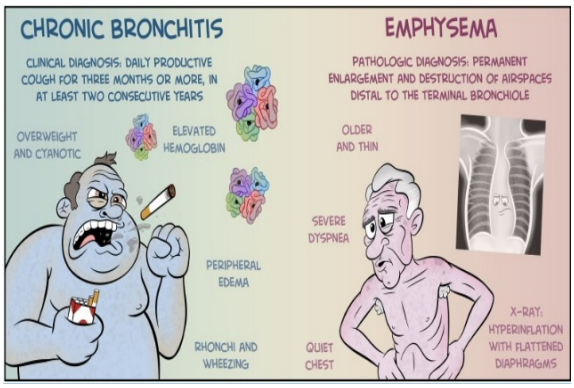


Figure 1: Chronic bronchitis vs. Emphysema

- **Diagnosis:**

Classification of COPD by impairment of lung function

Stage	Severity	Spirometry (postbronchodilator)
GOLD 1	Mild	$FEV_1 \geq 80\%$ predicted $FEV_1/FVC < 0.7$
GOLD 2	Moderate	$50\% \leq FEV_1 < 80\%$ predicted $FEV_1/FVC < 0.7$
GOLD 3	Severe	$30\% \leq FEV_1 < 50\%$ predicted $FEV_1/FVC < 0.7$
GOLD 4	Very severe	$FEV_1 < 30\%$ predicted $FEV_1/FVC < 0.7$

  - Chest X-ray:
    - Hyperinflation.
    - Subpleural blebs and parenchymal bullae in emphysema.
  - Pulmonary function test (PFT):
    - ↓ FEV1/FVC ration <80%.
    - Normal or ↑ TLC.
    - ↓ DL_{CO2} in emphysema.

In the setting of fever and CXR infiltrates consider sputum culture and gram staining.

  - Arterial blood gases (ABGs):
    - ↑PCO2 in acute or chronic respiratory acidosis with hypoxemia.
  - Others:
    - CBC:
      - ✓ Leukocytosis in the context of acute exacerbation.
      - ✓ Secondary polycythemia due to hypoxia.
    - Echocardiogram: to asses the cardiac function.
    - Alpha 1 antitrypsin deficiency: if premature onset of the disease <40 Y/O, or lifelong non-smoker.

S.pneumoniae & H.influenzae are the most common organisms to cause COPD exacerbations.

- **Management:**
  - Non-pharmacological treatment:
    - Smoking & supplemental O2 are the most effective interventions to improve the survival in COPD patients.
      - Criteria for oxygen use if:
        - $PO_2 < 55$ mmHg or O_2 saturation $< 88\%$.
        - If the patient has right sided heart failure or elevated hematocrit:
          - $PO_2 < 66$ mmHg or O_2 saturation $< 90\%$.
        - The main aim is to raise the O_2 saturation $> 90\%$.
    - BiPAP in cases of nocturnal hypoxia and acute exacerbations.
    - Surgical treatment for bullae or blebs, lung volume reduction, or single lung transplantation.
  - Pharmacological treatment:
    - Bronchodilators (short acting beta 2 agonist and anti-muscarinic):
      - Inhaled anti-muscarinic agents are the most effective pharmacological treatment in COPD patients.
    - Theophyllines.
    - Corticosteroids.
    - Antibiotics.

Management of acute exacerbation:
1. Supplemental O2.
2. Bronchodilator (albuterol).
3. Antimuscarenic (ipratropium).
4. IV + - inhaled steroids.
5. Antibiotics.
6. In severe cases: BiPAP or intubation.

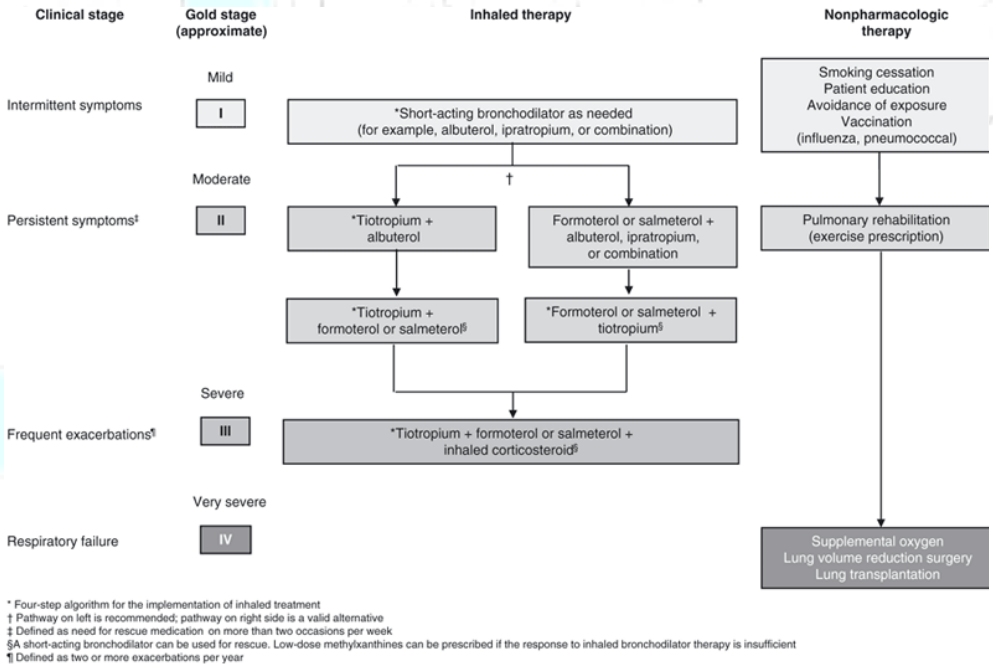


Figure 2: Management of COPD.

General management of **COPD**:
C: corticosteroids
O: oxygen supplement.
P: prevention of smoking and prophylaxis
D: dilators (beta agonist, & antimuscarenic).

- **Complications:**
  - Respiratory failure.
  - Pulmonary hypertension & cor pulmonale.
  - Nocturnal hypoxia:
    - Most deaths in COPD patients occur at night due to hypoxemia & cardiac arrhythmias.

- **Prophylaxis:**
  - Pneumococcal vaccine every 5 years.
  - Influenza vaccine annually.
- **Prognosis:**
  - The level of dyspnea is the best predictor of COPD prognosis.
  - 4 year mortality is measured by the BODE index:
    - 0-2 has 10% rate of mortality.
    - 7-10 has 80% rate of mortality.
  - Progressively decreasing FEV1 is indicator of poor prognosis.

If pharmacological treatment is insufficient referral to transplantation is needed.

	Emphysema	Chronic Bronchitis
Definition	Permanent abnormal enlargement of *air spaces*, distal to the terminal bronchioles due to destruction of **alveolar** walls.	Chronic **productive cough**, on most days of the week, lasting for >= 3 months per year, for at least 2 consecutive years.
Diagnosis	A **Pathological** Diagnosis	A **Clinical** Diagnosis
Microscopic/ histologic	Damage of the **alveoli** - Bullae Damage of the capillary bed	Damage of the **endothelium** - Focal squamous columnar to squamous metaplasia - Bronchial wall infiltration with acute and chronic inflammatory cells → collagen deposition → wall thickening - Ciliary abnormalities - Relatively undamaged pulmonary capillary bed
Pathophysiology	Alveolar wall destruction and loss of elasticity caused by: - High levels of protease (elastase) (produced by neutrophils & PMNs) - Low levels of Alfa1-antitrypsin	- Excess **mucus production** → Narrow airways + **productive cough** - Airway **inflammation and scarring** → 1) *Mucus gland* enlargement 2) *Smooth muscle* hyperplasia. → **Airway obstruction & increased resistance!**
Types	**Centriacinar:** - Most common type - Localizes to the respiratory bronchioles just distal to the terminal bronchiole - Sever form is associated with **smoking** - **more with the upper lung zones** **Panacinar:** - Alveolar ducts are diffusely enlarged - More severe - Asociated with *Alfa1-antitrypsin (AAT) deficiency*. - **Premature COPD** - **More in the lung bases** **Distal acinar emphysema / paraseptal emphysema:** - The least common form - Involves distal airway structures, alveolar ducts, and sacs, and is localized to fibrous septa or to the pleura - Leads to formation of bullae - Is not associated with airflow obstruction. - Can lead to **pneumothorax**	NA

Presentation	- **Dyspnea** (Most prominent symptom, with pursed lips & use of accessory muscles) - Wheezing - Little or no cough - **Chachicsic** appearance - **Barrel chest** - Hyperresonant chest - Distant heart sounds	- Less dyspnea (can be using the accessory muscles, but are never anxious) - Coarse rhonchi and wheezing - **Productive cough** (prominent symptom) - **Obesity** - **Cor Palmonale** S&Sx: - Edema - Cyanosis
DLCo	Decreased diffusing capacity for carbon monoxide (DLCo)	Normal diffusing capacity for carbon monoxide
Blood gases	Maintain almost normal levels of blood gases	Develop CO2 retention (air trapping) & hypoxemia (cyanosed)
PFT	Reduced FEV1	Reduced FEV1
CXR	- Decreased bronchovascular markings - A long, narrow heart shadow - Flattening of the diaphragm - Increased retrosternal air space - Hyperlucency of the lungs	- Increased bronchovascular markings - Cardiomegaly
Notes	- Not usually associated with fibrosis	- Females are more likely to develop bronchitis

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