Chronic obstructive pulmonary disease (COPD)

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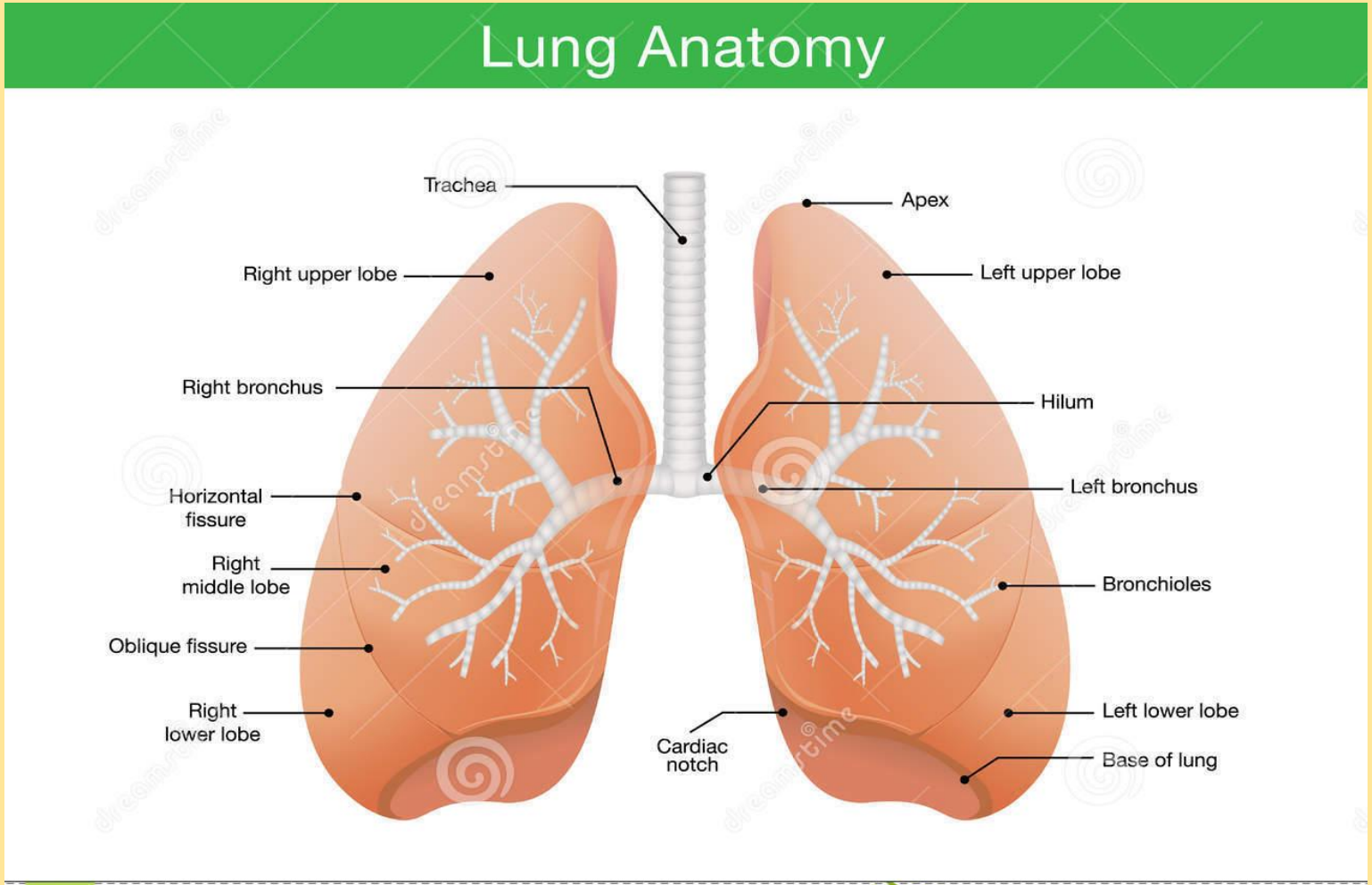
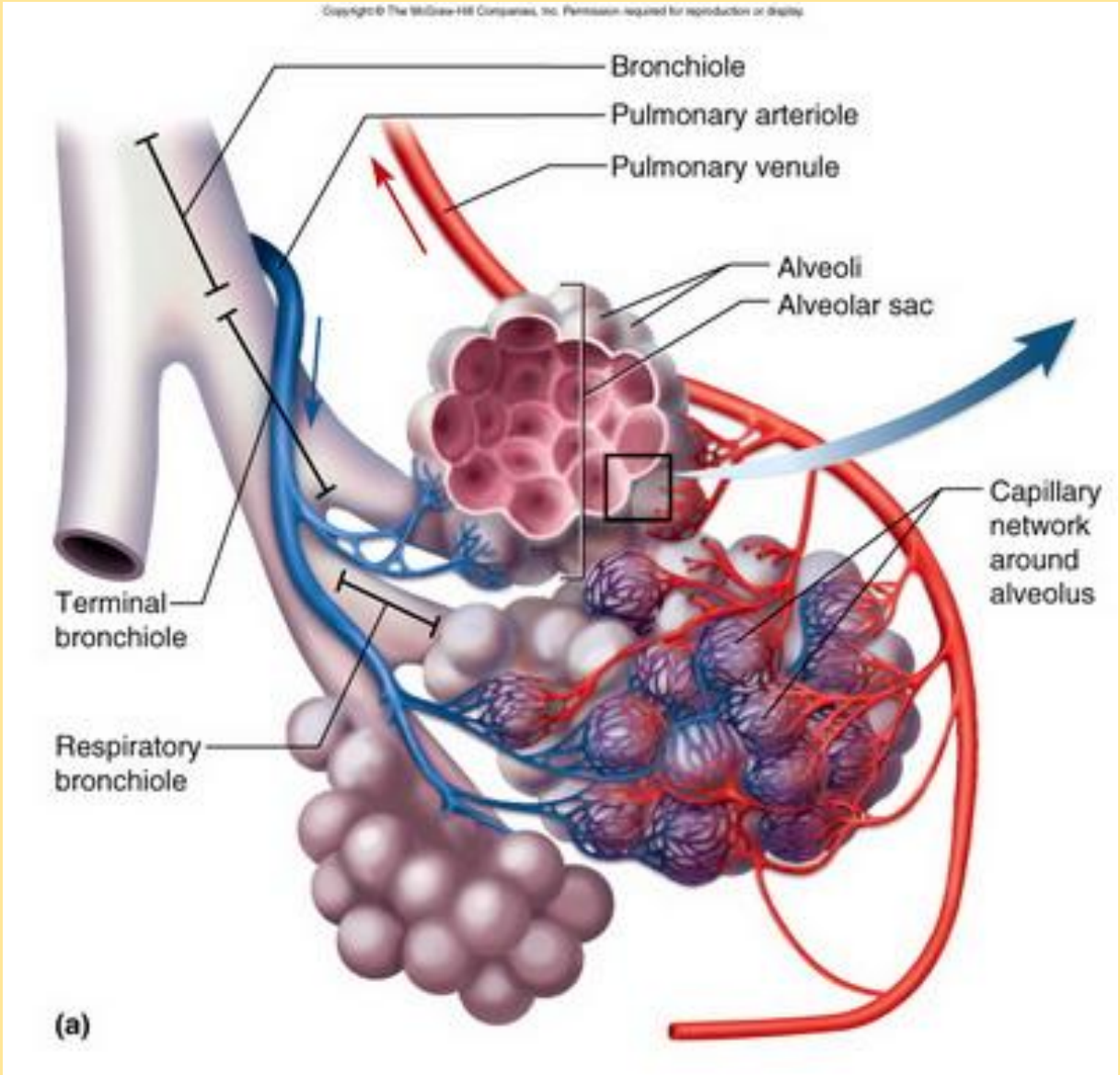
Lung anatomy & Physiology:



- Air travels down in the trachea through two large tubes (bronchi). they divide many times into many smaller tubes (bronchioles) that end in clusters of tiny air sacs (alveoli).
- The air sacs have very thin walls full of tiny blood vessels (capillaries). The oxygen in the air passes into these blood vessels and enters bloodstream. At the same time, carbon dioxide — a waste product of metabolism — is exhaled.


Bronchiole
Pulmonary arteriole
Pulmonary venule
Alveoli
Alveolar sac
Capillary network around alveolus
Terminal bronchiole
Respiratory bronchiole
(a)

- COPD is a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing.
- COPD is estimated to be as the third leading cause of death in the United States.
- The term COPD includes chronic bronchitis & Emphysema

- **Chronic bronchitis** is defined clinically as the presence of a chronic productive cough for 3 months during each of 2 consecutive years (other causes of cough being excluded).

- **Emphysema** is defined pathologically as an abnormal, permanent enlargement of the air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis.

Other causes of chronic cough:

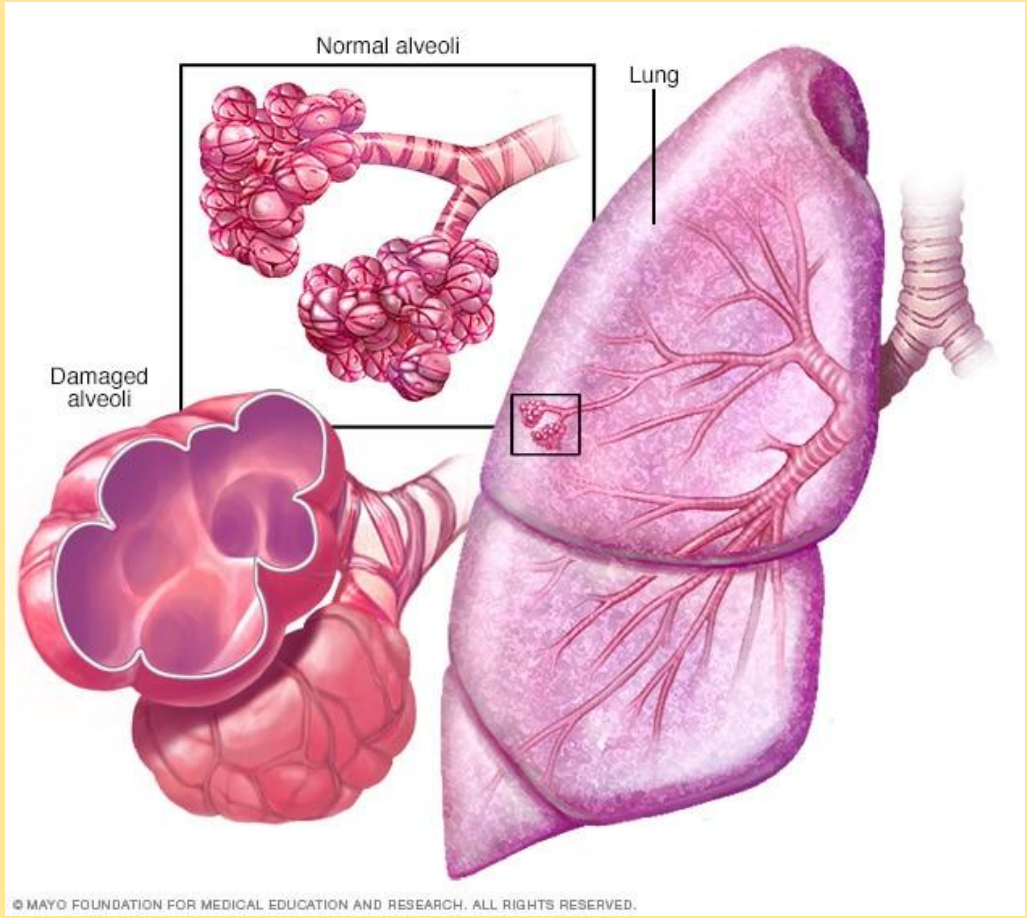
- Rhinitis & sinusitis
- Bronchial asthma
- Gastroesophageal reflux disease (GERD)
- Bronchiectasis
- Bronchogenic carcinoma
- Chronic aspiration
- Congestive heart failure (CHF)
- Foreign body of the airway
- Psychogenic cough
- Tuberculosis

How your lungs are affected?

- Your lungs rely on the natural elasticity of the bronchial tubes and air sacs to force air out of your body. COPD causes them to lose their elasticity and overexpand, which leaves some air trapped in your lungs when you exhale.

- The pathological foundation for chronic bronchitis is due to excessive tracheobronchial mucus production in response to the inflammatory signals.

Emphysema:


Normal alveoli
Lung
Damaged alveoli
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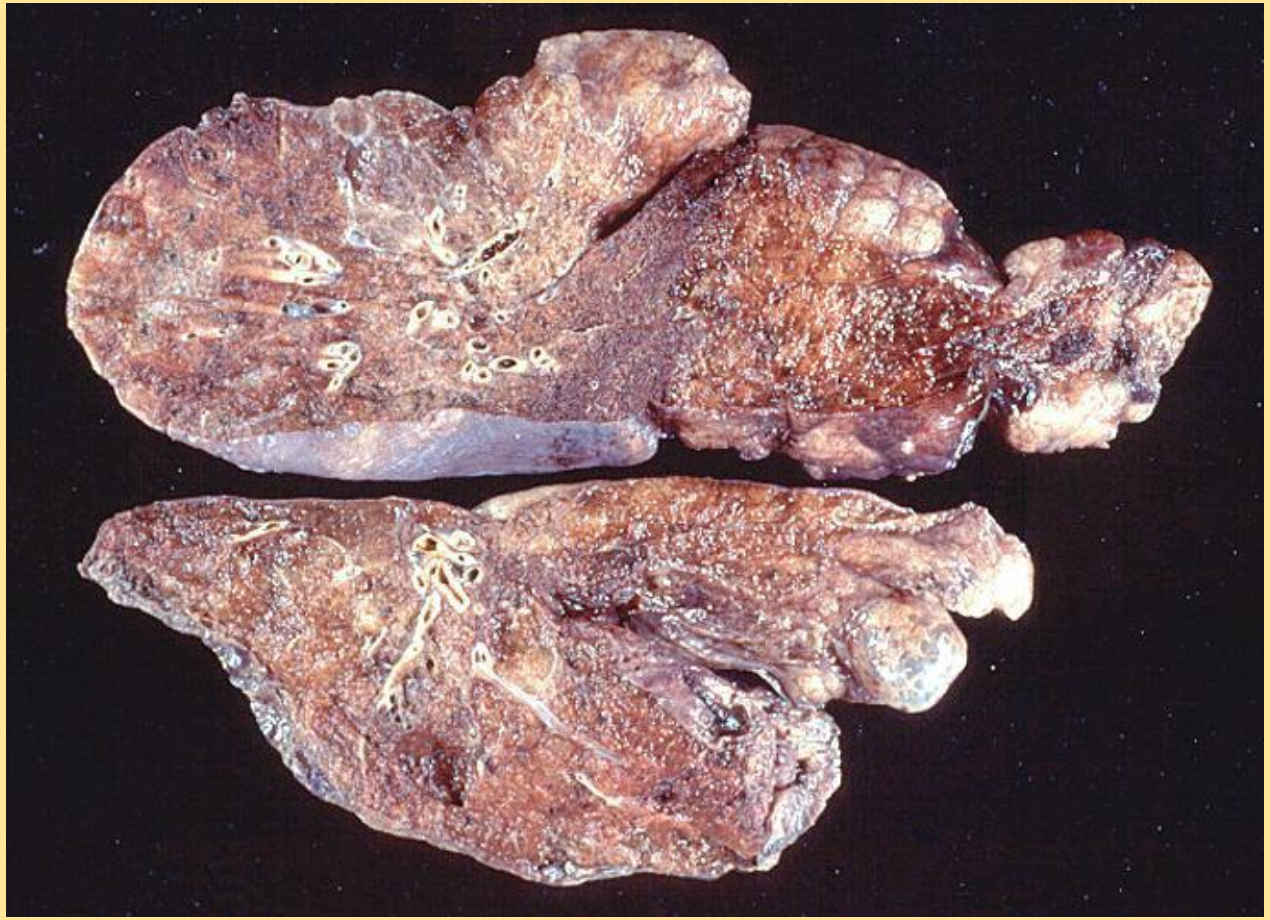
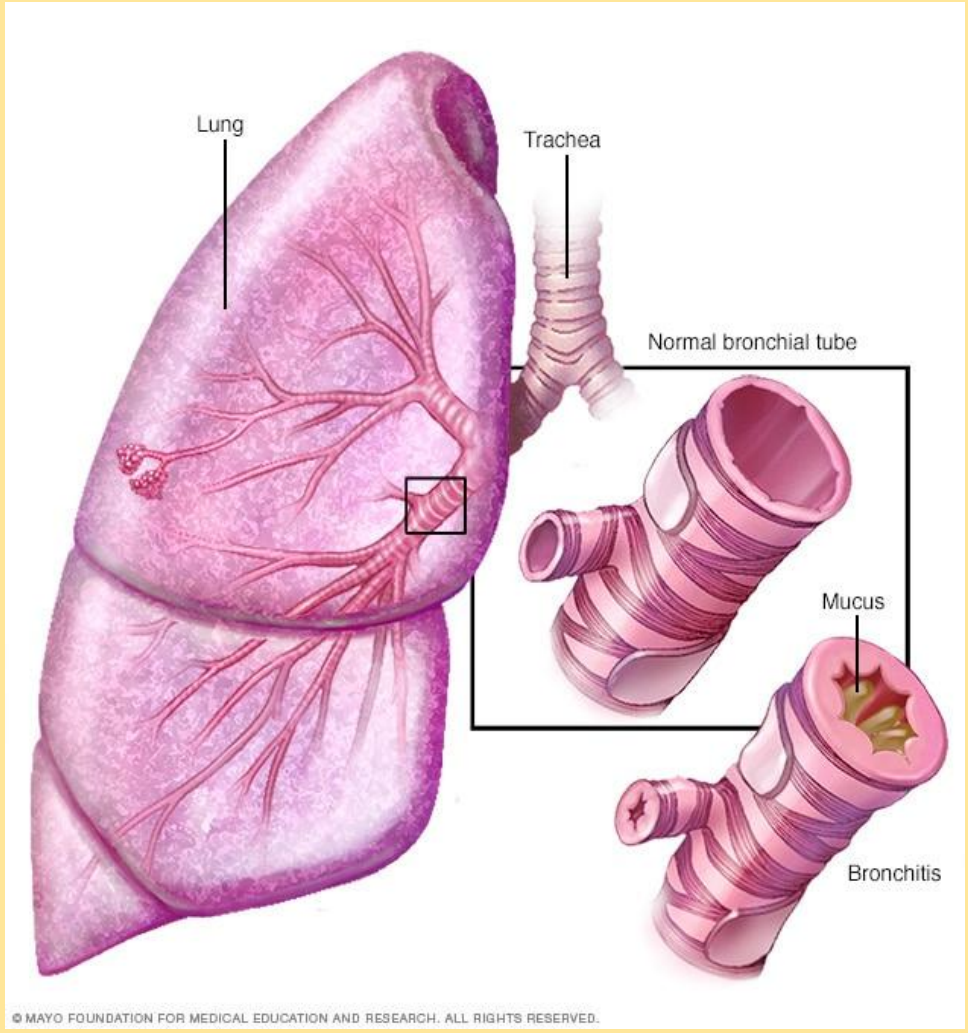
Gross pathology of advanced emphysema. Large bullae are present on the surface of the lung



Chronic bronchitis:



Etiology

Cigarette smoking:

- The primary cause of COPD is exposure to tobacco smoke.

  Tobacco smoking accounts for as much as 90% of COPD risk.

- Cigarette smoking induces macrophages to release neutrophil chemotactic factors and elastases, which lead to tissue destruction.

Environmental factors:

- COPD may occur in individuals who have never smoked
- Long-term exposure to traffic-related air pollution may be a factor in COPD.
- people exposed to fumes from burning fuel for cooking and heating in poorly ventilated homes

Alpha1-antitrypsin deficiency:

The main purpose of this amino-acid, is to protect the lung parenchyma from elastolytic breakdown.

Severe AAT deficiency predisposes to unopposed elastolysis with the clinical sequela of an early onset of emphysema.

Clinical presentation:

History:

- Most patients with COPD seek medical attention late, as they often ignore the symptoms because they start gradually and progressively.

Signs and symptoms:

- Cough, usually worse in the mornings and **productive** of a **small amount** of **colorless** sputum
- Breathlessness: The most significant symptom, but usually does not occur until the sixth decade of life
- Wheezing: May occur in some patients, particularly during exertion and exacerbations

Findings in severe disease include the following:

- Tachypnea and respiratory distress with simple activities
- Use of accessory respiratory muscles and indrawing of lower intercostal spaces (Hoover sign)
- Cyanosis
- Elevated jugular venous pulse (JVP)
- Peripheral edema

Complications:

- Respiratory infections >> COPD exacerbation
- Pulmonary hypertension >> Heart failure
- Rupture emphysematous bulla >> Pneumothorax
- Lung Cancer (Smoking is the same major risk factor)
- Depression

Diagnosis & workup:

Arterial Blood Gas Analysis (ABG):

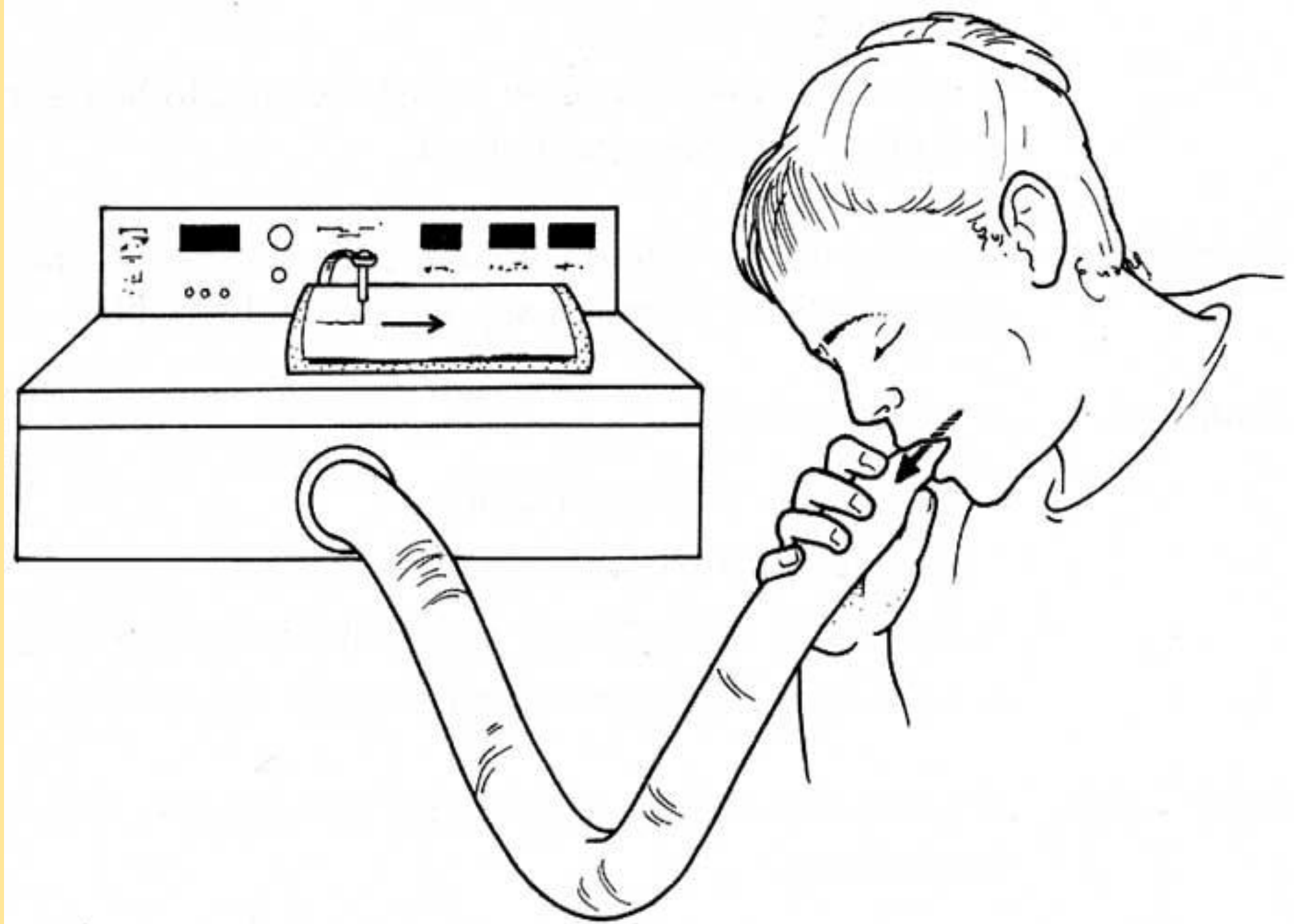
- Patients with mild COPD have mild to moderate hypoxemia (decreased Oxygen) without hypercapnia (increased CO2). As the disease progresses, hypoxemia worsens and hypercapnia may develop.

Chest radiography:

- Chest radiographs in emphysema reveal signs of hyperinflation
- Chronic bronchitis is associated with increased bronchovascular markings.
- High-resolution CT (HRCT) scanning is more sensitive than standard chest radiography and is highly specific for diagnosing emphysema

Pulmonary function tests:

- Pulmonary function tests are essential for the diagnosis and assessment of the severity of disease, and they are helpful in following its progress.



Wendolyn Hill

Nursing Assessment:

- Assess patient’s exposure to risk factors.
- Assess the patient’s past and present medical history.
- Assess the signs and symptoms of COPD and their severity.
- Assess the patient’s knowledge of the disease.
- Assess the patient’s vital signs.
- Assess breath sounds and pattern.

Alarming signs (Please inform the doctor)

- Disturbed conscious level
- Tachypnea (increased respiratory rate) or change in pattern of respiration.
- Desaturation (Oxygen saturation getting worse < 90 %)
- Cyanosis (Bluish discoloration of the skin or mucous membrane)
- Hypotension
- Tachycardia or arrhythmia with some drugs (e.g: Salbutamol)
- Acute chest pain (Rupture emphysematous bulla >> Pneumothorax)

Treatment:

Smoking Cessation:

Smoking cessation is the most important therapeutic intervention for COPD.

- Chest physiotherapy and postural drainage to improve pulmonary ventilation.

Management of inflammation:

- Systemic steroids are used in acute exacerbations.

  (Prednisone ‘Hostacortin’ & Methyl prednisolone ‘Medrol’)

- Inhaled corticosteroids, less systemic side effects:

  Fluticasone (Flixotide) & Budesonide (Pulmicort)

Management of Infection

- Antibiotic therapy for those with exacerbations that were characterized by at least 2 of the following: increases in **dyspnea**, **sputum production**, and **sputum purulence**
- Empiric antimicrobial therapy is recommended in patients with an acute exacerbation and evidence of an infectious process, such as **fever**, **leukocytosis** (increased WBC count), or an **infiltrate on chest radiograph**.

Management of Sputum Viscosity and Secretion:

- Proper hydration helps to cough up secretions or tracheal suctioning when the patient is unable to cough.
- The oral agent *N* -acetylcysteine has mucokinetic properties and is used to treat patients with COPD.

Oxygen Therapy and Hypoxemia

- COPD is commonly associated with progressive hypoxemia.
- Long-term low flow oxygen therapy improves survival.
- Avoid using high flow Oxygen as this may lead to CO2 retention, keep your target Saturation 90 – 92 %.
- Non invasive mechanical ventilation may be needed in acute exacerbation, and invasive endotracheal intubation in severe cases.

- Bronchodilators:

Act to decrease muscle tone in small and large airways.

- Salbutamol (Ventolin)
- salmeterol (Metrovent)
- formoterol (Foradil).
- Ipratropium (Atrovent)
- Tiotropium bromide (Spiriva)
- Xanthine derivatives (Theophylline ‘Quibron’ & aminophylline)

Combination drugs:

- Salbutamol/ipratropium (Combivent)
- Budesonide/formoterol (Symbicort)
- Fluticasone and salmeterol (Seretide Diskus)


THANK YOU!