

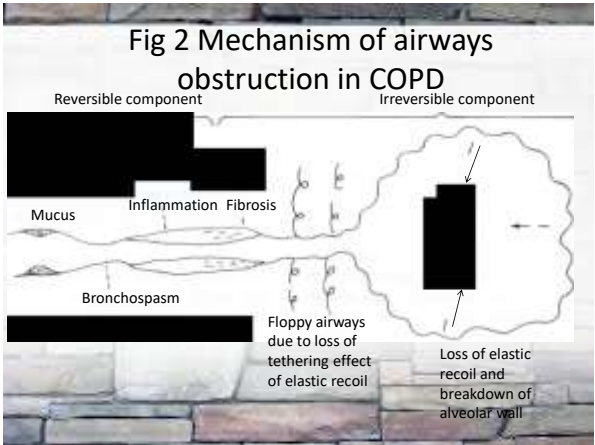
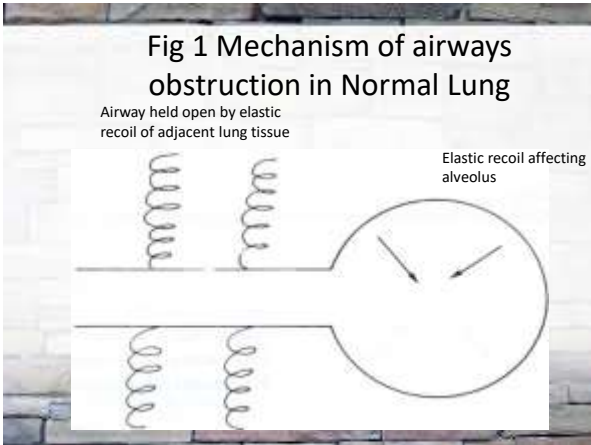
# Obstructive disorders

## INTRODUCTION

Airways obstruction increases airflow resistance and the work of breathing, as indicated by decreased peak flow rates.

**Causes:**

- ❑ reversible factors, e.g. inflammation, bronchospasm or mucus plugging.
- ❑ irreversible factors, e.g. fibrotic airway walls or floppy airways as a result of loss of the elastic recoil that normally supports them (Figure 1)
- ❑ localized lesions, e.g. upper airway tumour or foreign body.



## COPD Classification

- Chronic Bronchitis
  - Emphysema
- Now also includes
- Bronchopulmonary Dysplasia
  - Cystic fibrosis
  - Asthma
  - Bronchiectasis

## COPD

- COPD is the only chronic disease that is showing progressive upward trend in both **mortality** and **morbidity**
- It is expected to be the **third** leading cause of death by 2020

## Definition

- Chronic obstructive pulmonary disease (COPD) is a **preventable and treatable disease** state characterised by airflow limitation that is not fully reversible.
- The airflow limitation is usually **progressive** and is associated with an **abnormal inflammatory response** of the lungs to noxious particles or gases, primarily caused by cigarette smoking.
- Although COPD affects the lungs, it also produces significant **systemic consequences**.

## Risk Factors

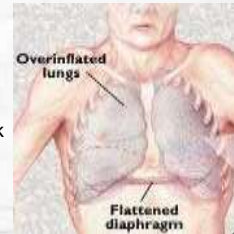
- Smoke from home cooking and heating fuel
- Occupational dust and chemicals
- Gender: More common in men. **M:F ratio is 5%:2.7%**
- Increasing age
- Others: Infection, nutrition and deficiency of  $\alpha_1$  antitrypsin

## Key Indicators for COPD Diagnosis

<b>Chronic cough</b>	Present intermittently or every day often present throughout the day; seldom only nocturnal
<b>Chronic sputum production</b>	Present for many years, worst in winters. Initially mucoid – becomes purulent with exacerbation
<b>Dyspnoea that is</b>	Progressive ( <b>worsens over time</b> ) Persistent ( <b>present every day</b> ) Worse on exercise Worse during respiratory infections
<b>Acute bronchitis</b>	Repeated episodes
<b>History of exposure to risk factors</b>	Tobacco smoke ( <b>including beedi</b> ) occupational dusts and chemical smoke from home cooking and heating fuel

## Physical signs

- Large barrel shaped chest (**hyperinflation**)
- Prominent accessory respiratory muscles in neck and use of accessory muscle in respiration
- Low, flat diaphragm
- Diminished breath sound



## Pharmacotherapy for COPD

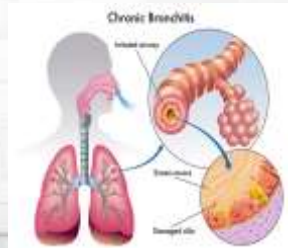
- Anti-inflammatory
  - Corticosteroids
- Bronchodilators
  - Beta-adrenergic agonist: Proventil
  - Methylxanthines: Theophylline
  - Anticholinergics: Atrovent
- Antihistamines

## Chronic Bronchitis

- Recurrent or chronic productive cough for a minimum of 3 months for 2 consecutive years.
- Risk factors
  - Cigarette smoke
  - Air pollution

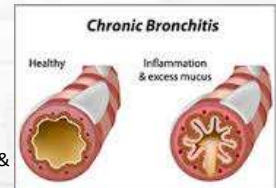
## Chronic Bronchitis Pathophysiology

- Chronic inflammation
- Hypertrophy & hyperplasia of bronchial glands that secrete mucus
- Increase number of goblet cells
- Cilia are destroyed



## Chronic Bronchitis Pathophysiology

- Narrowing of airway
  - Starting w/ bronchi smaller airways
- ↑airflow resistance
- ↑work of breathing
- Hypoventilation & CO<sub>2</sub> retention → hypoxemia & hypercapnea



## Chronic Bronchitis Pathophysiology

- Bronchospasm often occurs
- End result
  - Hypoxemia
  - Hypercapnea
  - Polycythemia (increase RBCs)
  - Cyanosis
  - Cor pulmonale (enlargement of right side of heart)

## Chronic Bronchitis: Clinical Manifestations

- In early stages
  - Clients may not recognize early symptoms
  - Symptoms progress slowly
  - May not be diagnosed until severe episode with a cold or flu
  - Productive cough
    - Especially in the morning
    - Typically referred to as "cigarette cough"
  - Bronchospasm
  - Frequent respiratory infections

## Chronic Bronchitis: Clinical Manifestations

- Advanced stages
  - Dyspnea on exertion → Dyspnea at rest
  - Hypoxemia & hypercapnea
  - Polycythemia
  - Cyanosis
  - Bluish-red skin color
  - Pulmonary hypertension → Cor pulmonale

## Chronic Bronchitis: Clinical Manifestations



## Chronic Bronchitis: Staging

The global initiative for chronic obstructive lung disease (GOLD) staging system is a commonly used severity staging system based on air flow limitation. According to this, there are 4 key stages:

- **Stage I:** mild,  $FEV_1 > 80\%$  of normal
- **Stage II:** moderate,  $FEV_1 = 50-79\%$  of normal
- **Stage III:** severe,  $FEV_1 = 30-49\%$  of normal
- **Stage IV:** very severe,  $FEV_1 < 30\%$  of normal or  $< 50\%$  of normal with presence of chronic respiratory failure present

The  $FEV_1:FVC$  ratio should be  $< 0.70$  for all stages.

## Chronic Bronchitis: Radiographic features

Findings of chronic bronchitis on **chest radiography** are nonspecific and include increased [bronchovascular markings](#) and [cardiomegaly](#).



## Chronic Bronchitis: Diagnostic Tests

- PFTs
  - FVC:     ↓ Forced vital capacity
  - FEV1:   ↓ Forcible exhale in 1 second
  - FEV1/FVC = <70%
- ABGs
  - ↑ PaCO<sub>2</sub>
  - ↓ PaO<sub>2</sub>

## Chronic Bronchitis: Prophylaxis

- Control of atmospheric pollution
- Stop smoking
- Treat all acute infections promptly
- Maintain good general health

## Chronic Bronchitis: Principles of treatment

- Decrease the bronchial irritation to a minimum
- Control infections
- Improve breathing pattern
- Control bronchospasm
- Control / decrease the amount of sputum
- Oxygen therapy

## Chronic Bronchitis: PT treatment

- Postural drainage, airway clearance techniques
- Breathing control and reduction of the work of breathing (high side lying, relaxed sitting, forward lean sitting, relaxed standing, forward lean standing)
- Thoracic mobilization exercises
- Increased exercise tolerance

## Emphysema

Emphysema is a condition of the lung characterized by permanent dilatation of the air spaces distal to the terminal bronchioles with destruction of the walls of these airways.



## Emphysema

### Primary / Congenital:

Primary emphysema may be caused by  $\alpha_1$  antitrypsin (protease inhibitor) deficiency

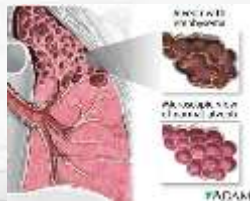
### Secondary to other factors:

- Obstructive airway disease (asthma, cystic fibrosis)
- Occupational lung diseases (pneumoconiosis)
- Risk factors (cigarette smoking)

## Emphysema: Pathophysiology

### Structural changes

- Hyperinflation of alveoli
- Destruction of alveolar & alveolar-capillary walls
- Small airways narrow
- Lung elasticity decreases



## Emphysema: Pathophysiology

### Mechanisms of structural change

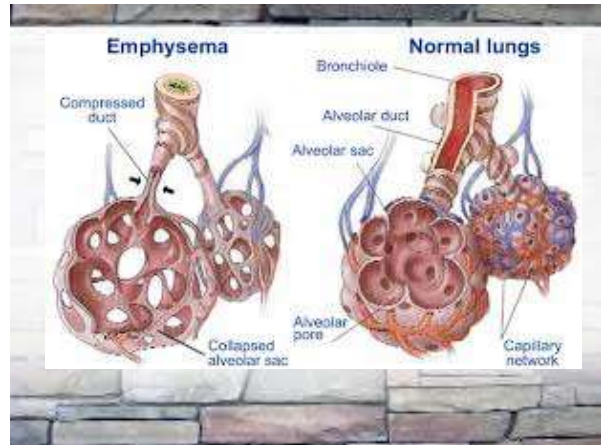
- Obstruction of small bronchioles
- Proteolytic enzymes destroy alveolar tissue
- Elastin & collagen are destroyed
  - Support structure is destroyed



## Emphysema: Pathophysiology

### The end result:

- Alveoli lose elastic recoil, then distend, & eventually blow out.
- Small airways collapse or narrow
- Air trapping
- Hyperinflation
- Decreased surface area for ventilation



## Emphysema: Clinical Manifestations

- Early stages
  - Dyspnea
  - Non productive cough
  - Diaphragm flattens
  - A-P diameter increases  
"Barrel chest"
  - Hypoxemia may occur
    - Increased respiratory rate
    - Respiratory alkalosis
  - Prolonged expiratory phase



## Emphysema: Clinical Manifestations

- Later stages
  - Hypercapnea
  - Purse-lip breathing
  - Use of accessory muscles to breathe
  - Underweight
    - No appetite & increase breathing workload
  - Lung sounds diminished





## Emphysema: Clinical Manifestations

- **Pulmonary function**
  - ↑ residual volume, ↑ lung capacity, DECREASED FEV<sub>1</sub>, vital capacity maybe normal
- **Arterial blood gases**
  - Normal in moderate disease
  - May develop respiratory alkalosis
  - Later: hypercapnia and respiratory acidosis
- **Chest x-ray**
  - Flattened diaphragm
  - hyperinflation

## Emphysema: Radiographic features

Except in the case of very advanced disease with bulla formation, chest radiography does not image emphysema directly, but rather infers the diagnosis due to associated features.  
Features:

- **hyperinflation**
  - flattened hemidiaphragm(s) : most reliable sign
  - increased and usually irregular radiolucency of the lungs
  - increased antero-posterior diameter of chest
- **vascular changes**
- **Small sized heart**



## Emphysema: Principles of treatment

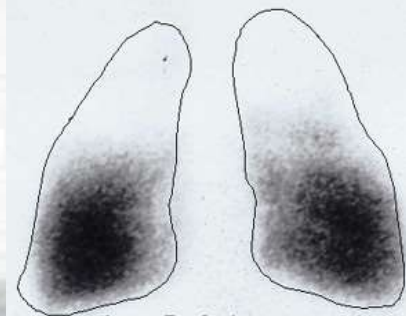
### Prevention and treatment of chest infections

- Antibiotics
- Rehousing
- Change occupation
- Stop smoking

### Improve lung function

- Steroids
- Bronchodilators
- Oxygen therapy
- Surgery

## Poor upper lung perfusion



Lung Perfusion

### Lung Volume Reduction Surgery

Many people who suffer with emphysema have portions of the lung which are more affected than others. This finding led to the development of a surgical approach to treat emphysema. Lung volume reduction surgery (LVRS) is a procedure which removes approximately 20-35% of the poorly functioning, space occupying lung tissue from each lung. By reducing the lung size, the remaining lung and surrounding muscles (intercostals and diaphragm) are able to work more efficiently. This makes breathing easier and helps patients achieve greater quality of life

## Emphysema: PT Treatment

- Re- education of breathing pattern
- Removal of secretions – postural drainage and additive techniques.
- To improve thoracic mobility – increase ROM of the joints of the thoracic cage.
- To increase exercise tolerance and function – breathing control taught in positions of relaxation progressing from half lying to standing.
- To regain fullest possible function.

Fig 9 (a) Patients with pink puffer characteristics of COPD.



Fig 9 Patients with blue bloater characteristics of COPD.



## Bronchopulmonary Dysplasia

BPD is a chronic lung disease of prematurity. Classically it follows a course of primary lung disease (RDS, MAS, etc) that requires exposure to mechanical ventilation and high oxygen concentration.

### Pathophysiology

- Alveolar stage of lung development begins at 36 weeks and continues postnatally. BPD occurs when insults result in defective repair, impede alveolarization, and cause vascular dysgenesis. Classically, BPD results from a primary lung disease (often RDS) requiring long-term mechanical ventilation or exposure to high oxygen concentration. It can occur without antecedent illness, particularly in the setting of extreme prematurity or sepsis.

### – Key contributors to BPD:

- Oxygen exposure: decreased alveolar septation (causing fewer, larger alveoli), increases lung fibrosis and inhibits lung growth.
  - Mechanical ventilation: Barotrauma and bacterial colonization can cause lung injury
  - Inflammation: Those who later develop BPD have been shown to have an early exaggerated inflammatory response in the lungs.
- **Risk Factors**
    - Lung immaturity, low birth weight, prematurity, exposure to high oxygen concentration,

## Bronchopulmonary Dysplasia

### Clinical Presentation

- Progressive, idiopathic pulmonary deterioration in susceptible neonates;
- oxygen dependence,
- retractions,
- diffuse rales/wheeze,
- hypoxemia,
- hypercapnea,
- compensatory metabolic alkalosis
- May develop right sided heart failure
- Poor weight gain

## Bronchopulmonary Dysplasia

### Differential Diagnosis

- Cardiovascular anomalies
- Airway obstruction
- Tracheomalacia
- Immunodeficiency
- Aspiration, reflux
- Cystic fibrosis

**Treatment**

- Maintain oxygenation
- PPV
- Fluid restriction
- Diuretic therapy
- Bronchodilation
- Corticosteroids

**Progression of  
Bronchopulmonary Dysplasia**