

# **RESPIRATORY SYSTEM DISEASES – PART 2**

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# **Obstructive lung diseases**

# CHRONIC OBSTRUCTIVE PULMONARY DISEASES (COPD)

- ❑ Chronic obstructive pulmonary disease (COPD) is a **progressive** disease characterized by airflow limitation that is not fully reversible and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases.
- ❑ COPD described as either **chronic bronchitis** or **emphysema**. Also include bronchiectasis and asthma.
- ❑ Initial symptoms include:
  - chronic cough.
  - sputum production.
  - dyspnea.

# Aetiology/Risk factors for COPD

- Tobacco smoke
- Air pollutants
- Occupational dust and fumes
- Genetics:  $\alpha$ 1-anti-trypsin deficiency (Emphysema)
- Repeated lung infections.
- Allergy

# Global Initiative for Chronic Obstructive Lung Disease



Table 2.4. Classification of airflow limitation severity in COPD (Based on post-bronchodilator FEV<sub>1</sub>)

GOLD 1:	Mild	FEV <sub>1</sub> ≥ 80% predicted
GOLD 2:	Moderate	50% ≤ FEV <sub>1</sub> < 80% predicted
GOLD 3:	Severe	30% ≤ FEV <sub>1</sub> < 50% predicted
GOLD 4:	Very Severe	FEV <sub>1</sub> < 30% predicted

**FEV1** is the amount of air you can force from your lungs in one second.

# CHRONIC BRONCHITIS

- The presence of persistent cough and sputum production for more than 3 consecutive months in at least 2 consecutive years.
- More frequent among middle aged men and 95% of the cases are found in chronic smokers.

# Pathogenesis :

The most common cause is exposure to tobacco smoke (95%)

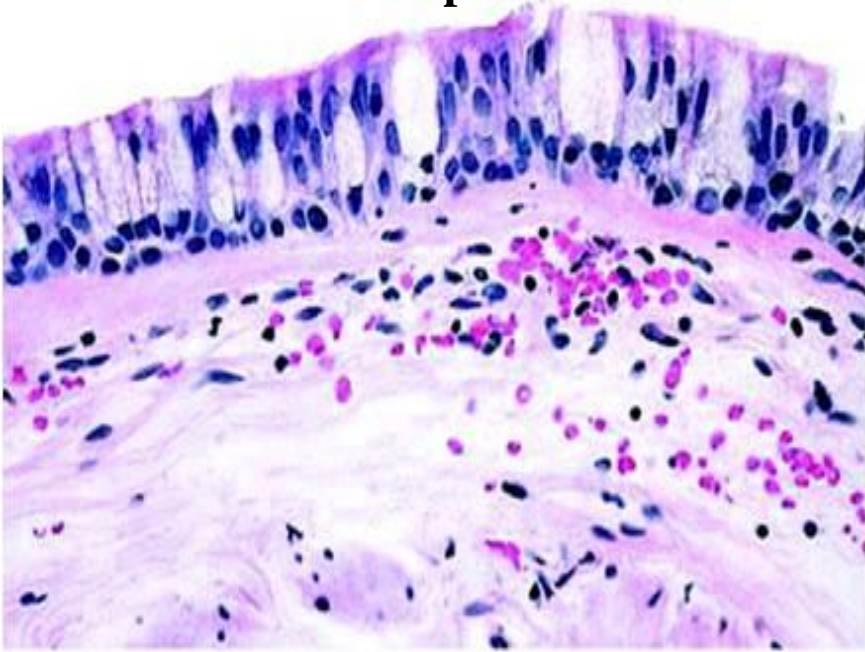
- Smoke (or any trigger factor )
- Cell injury and necrosis
- Inflammation and fibrosis of the Bronchial wall
- Hypertrophy of the submucosal glands
- Hypersecretion of mucus
- Partial or complete obstruction of bronchial airflow

# Morphology

1. **Bronchial epithelium: Squamous metaplasia**
2. **Mucosal goblet cells: Hyperplasia**
3. **Submucosal glands: Hypertrophy**
4. **Macrophage infiltration**
5. **Smooth muscle: Hypertrophy**
6. **Increased Reid Index: Ratio between the thickness of the submucosal mucous secreting gland and the thickness between the epithelium and cartilage.**

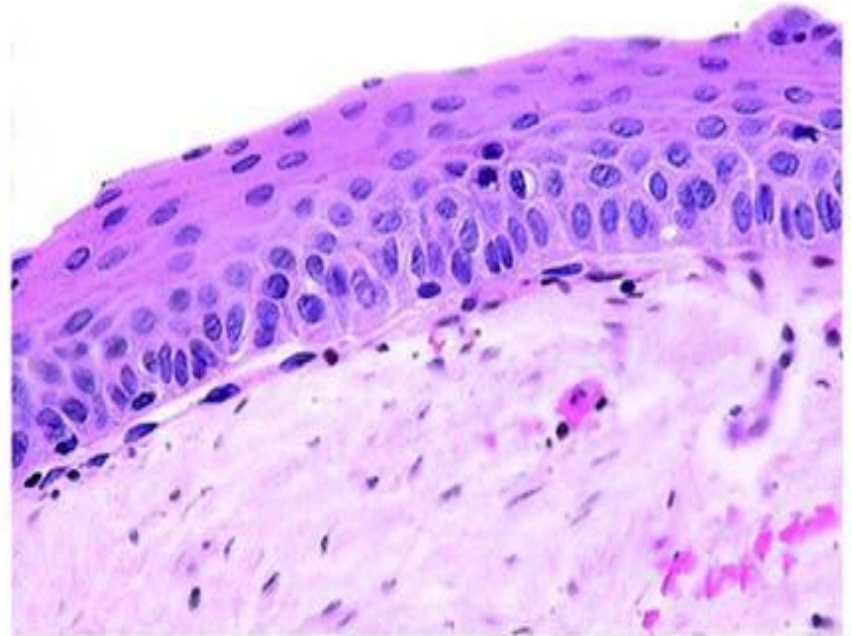
**A**

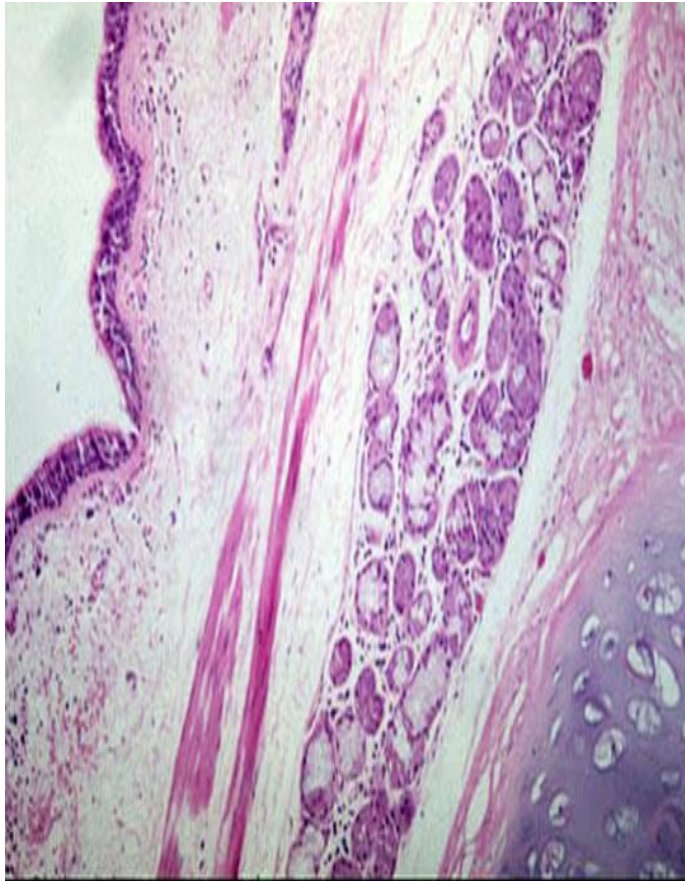
**Normal Columnar Epithelium**



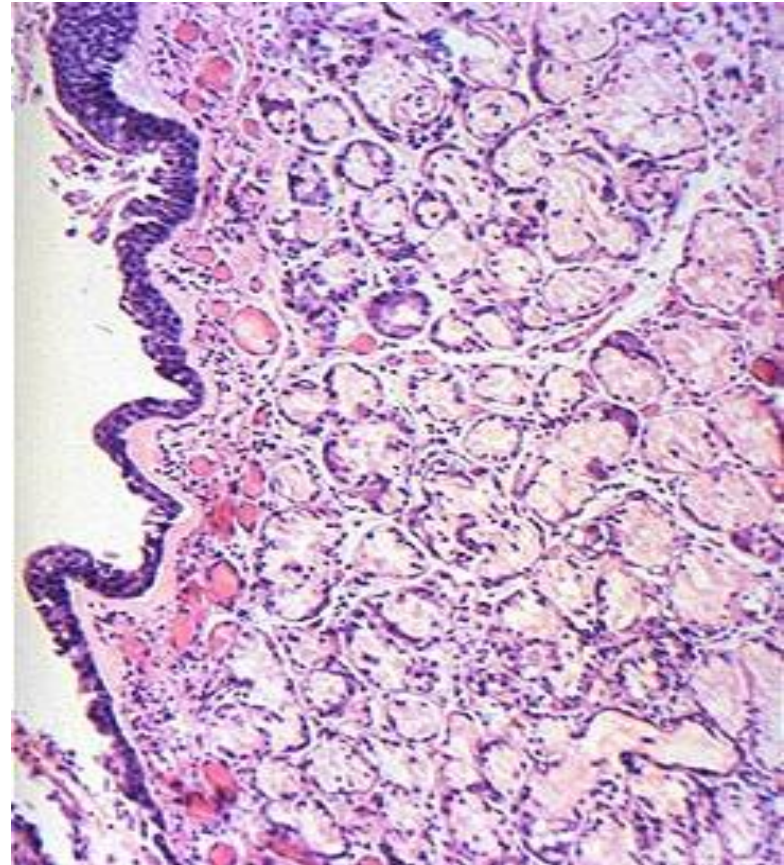
**B**

**Squamous Metaplasia**



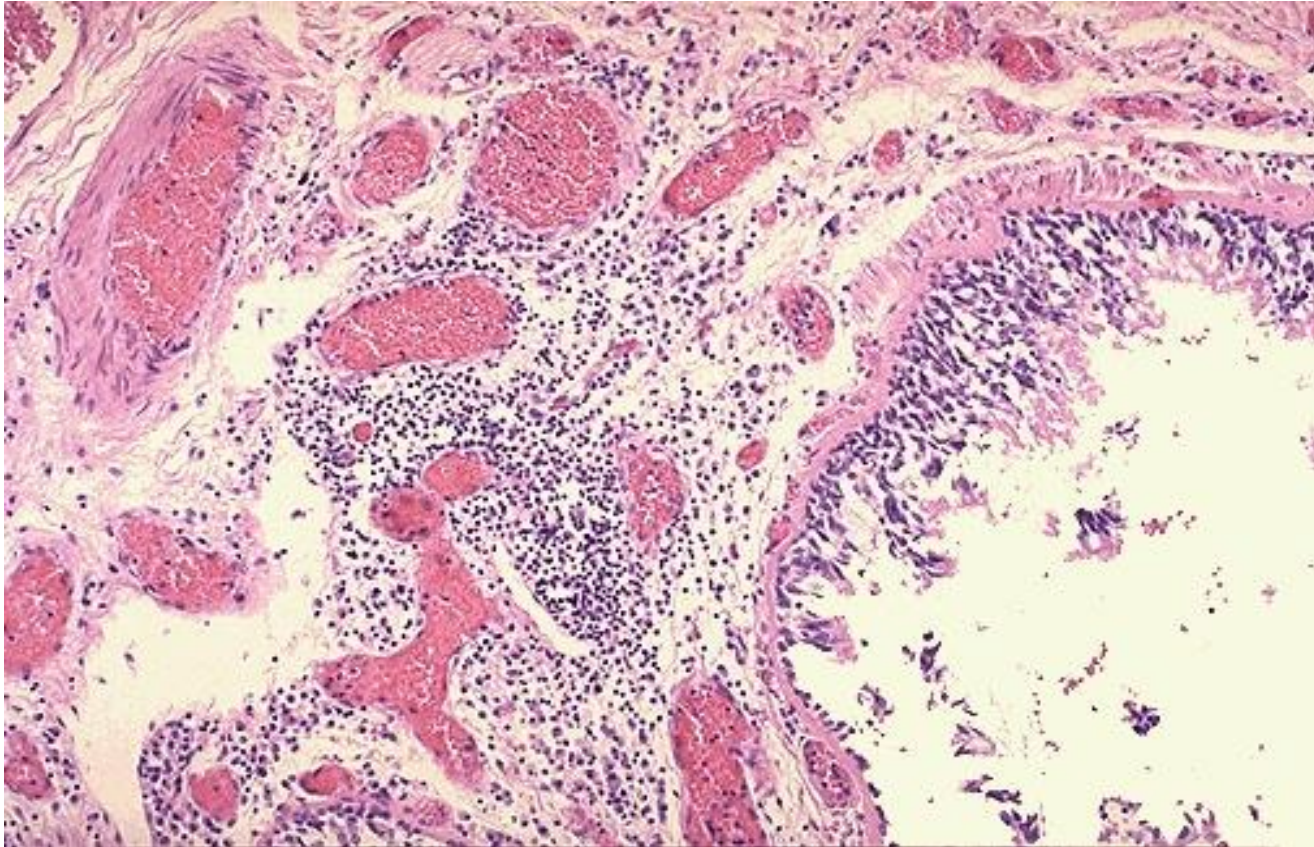


**Reid Index:**  
**Normal**

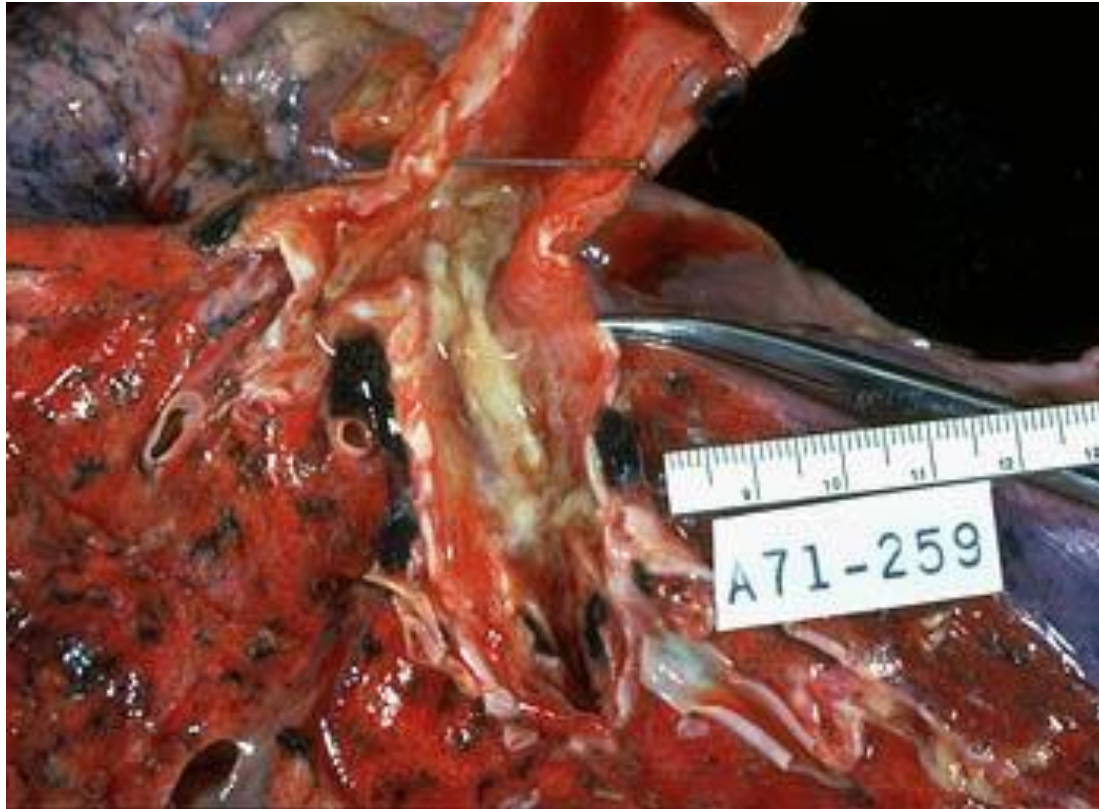


**Reid index:**  
**Increased in Chronic Bronchitis**

## Chronic Bronchitis



## Muco-purulent Secretions in Bronchus



## **Clinical presentation:**

1. Chronic cough.
2. Sputum production.
3. Dyspnea.
4. Wheezing
5. Cyanosis of the lips or fingernail → blue bloaters

## **Diagnosis**

1. It is diagnosed based on clinical grounds → The presence of persistent productive cough for more than 3 consecutive months in at least 2 consecutive years.
2. Pulmonary function test (PFT) → Spirometry
3. X-ray
4. CT scan
5. Blood gas analysis

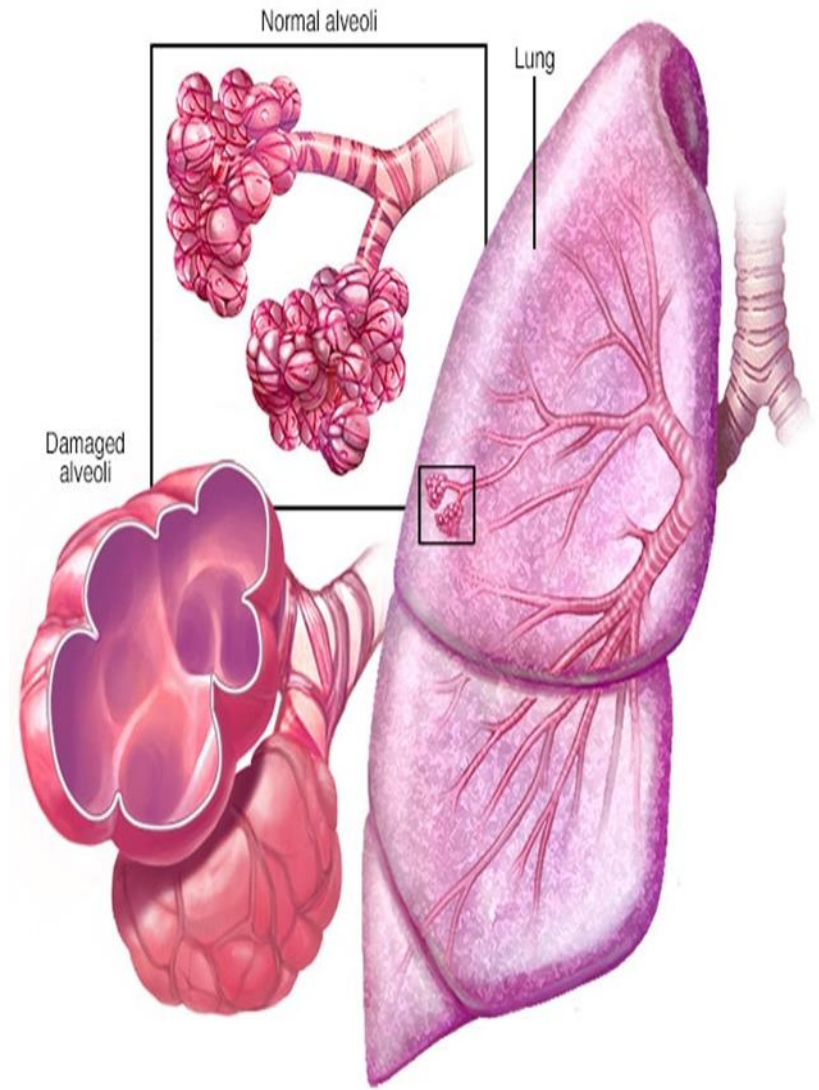
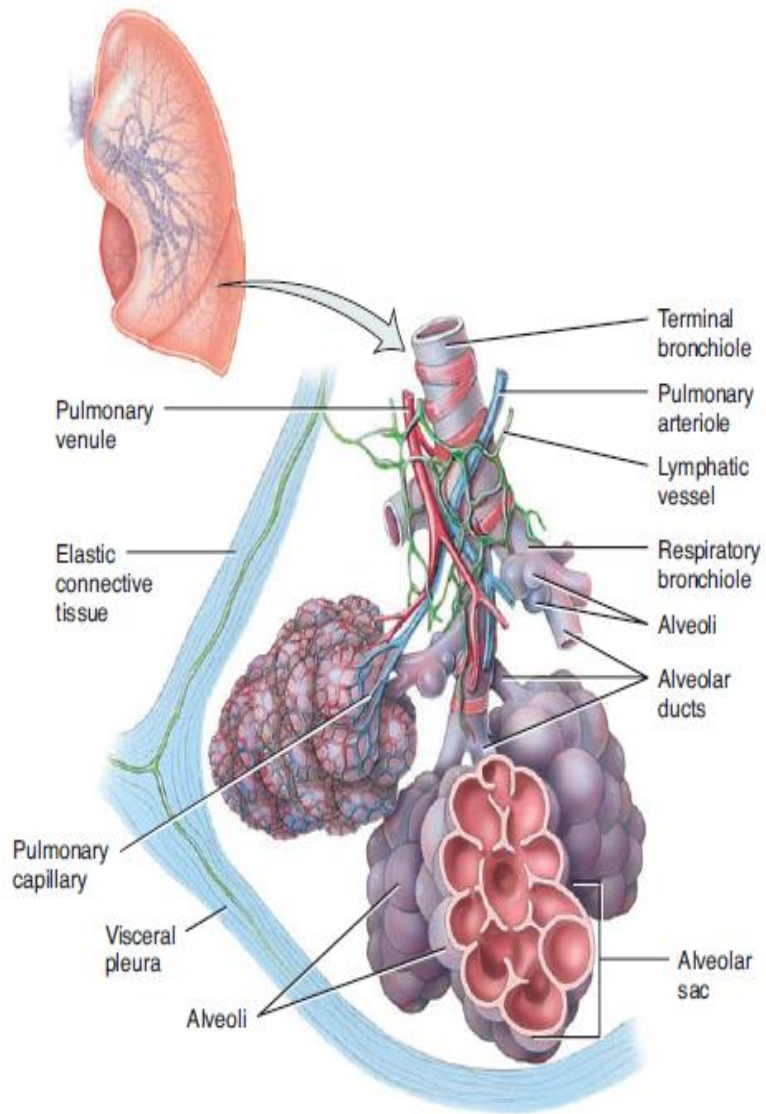
# Normal Arterial Blood Gas Values

	Normal	What is it?
pH	7.35 - 7.45	pH
PaCO <sub>2</sub>	35 - 45 mm Hg	Partial pressure of CO <sub>2</sub> in arterial blood
PaO <sub>2</sub>	80 - 100 mm Hg	Partial pressure of O <sub>2</sub> in arterial blood
SaO <sub>2</sub>	93 - 100%	Oxygen saturation of arterial blood
HCO <sub>3</sub> <sup>-</sup>	22 - 26 mEq/L	Serum bicarbonate
%MetHb	< 2.0%	Percent methemoglobinemia
%COHb	< 3.0%	Percent carboxyhemoglobinemia

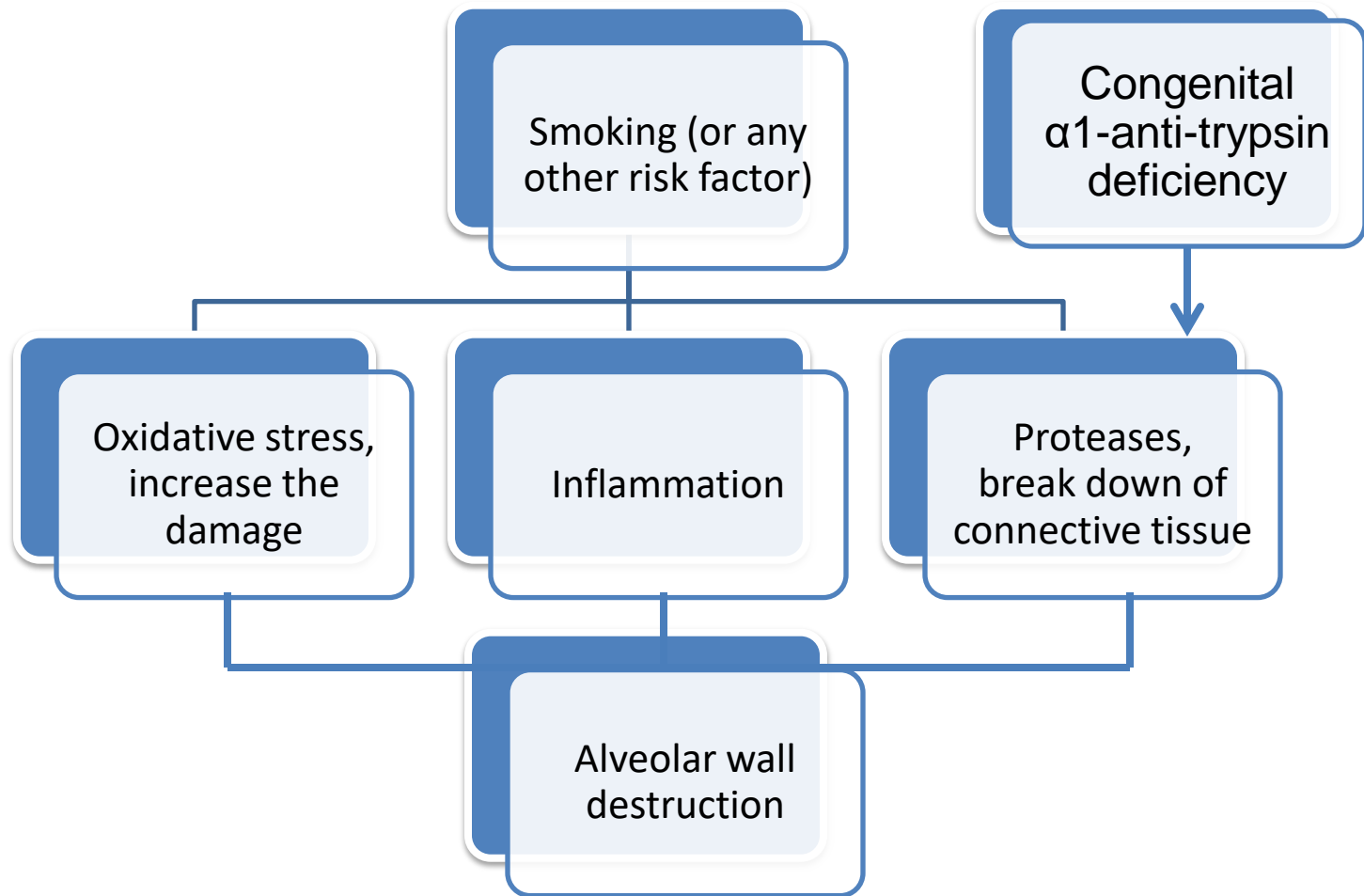
All of this assumes patient is breathing room air at sea level

# Emphysema

- Definition = Permanent dilatation of air spaces distal to terminal bronchioles with destruction of the walls of air spaces.”
  - Co-exists with Chronic Bronchitis
  - Permanent & irreversible
  - Get dilated non- functioning alveoli



# Pathogenesis :



# **Morphology**

## **Gross:**

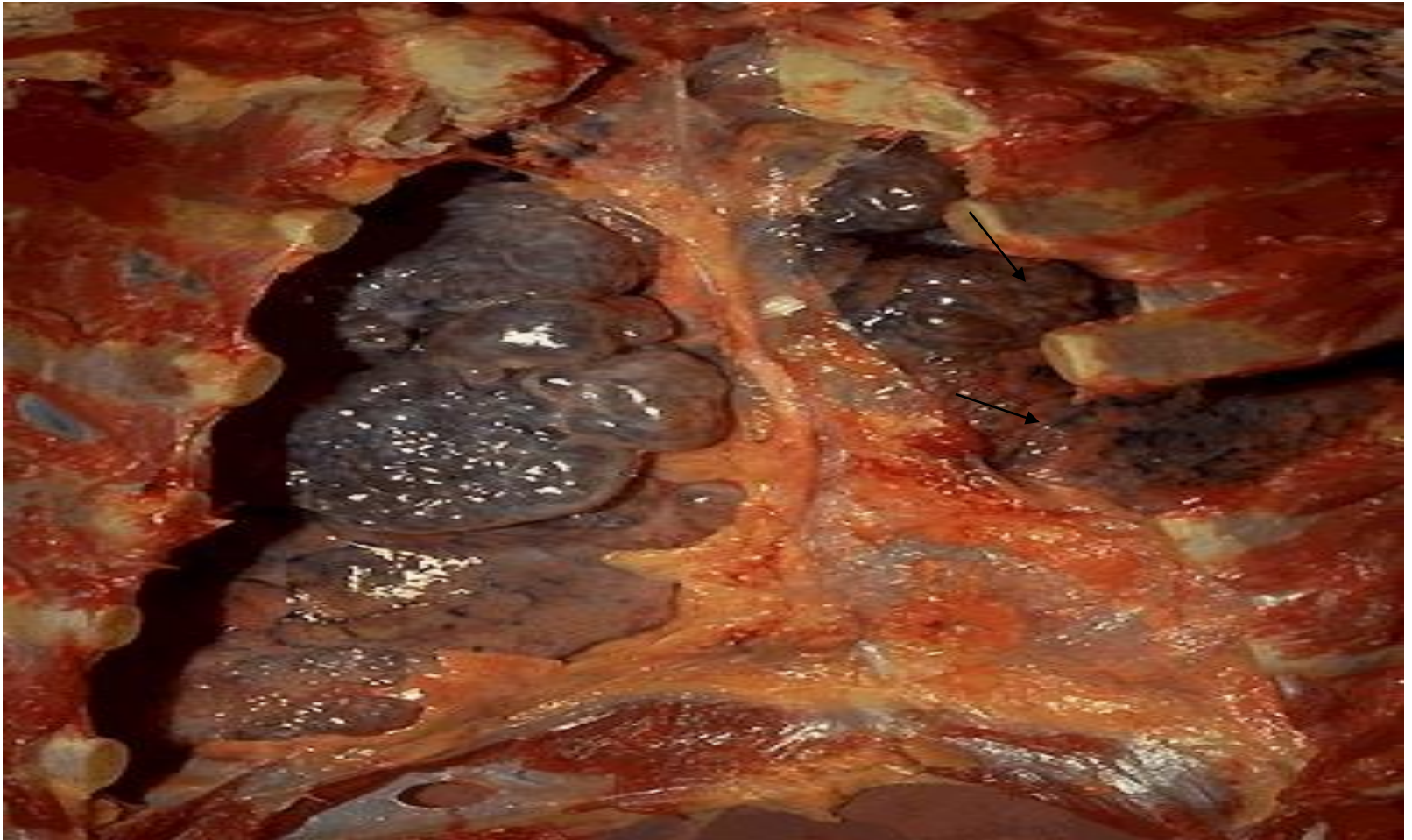
**Large, voluminous and pale.**

**Bullae: Air filled air sacs:**

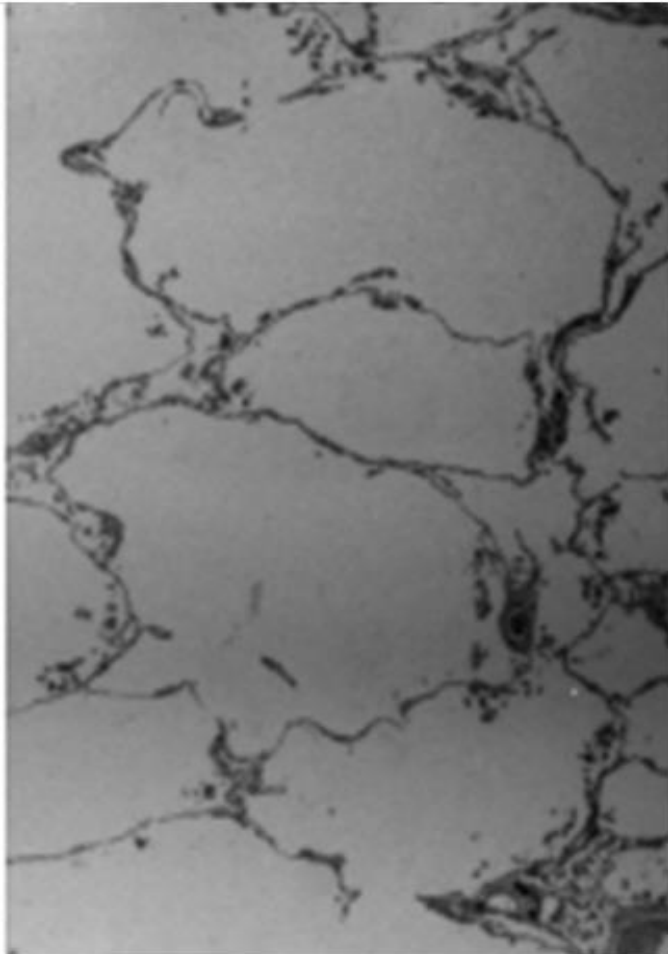
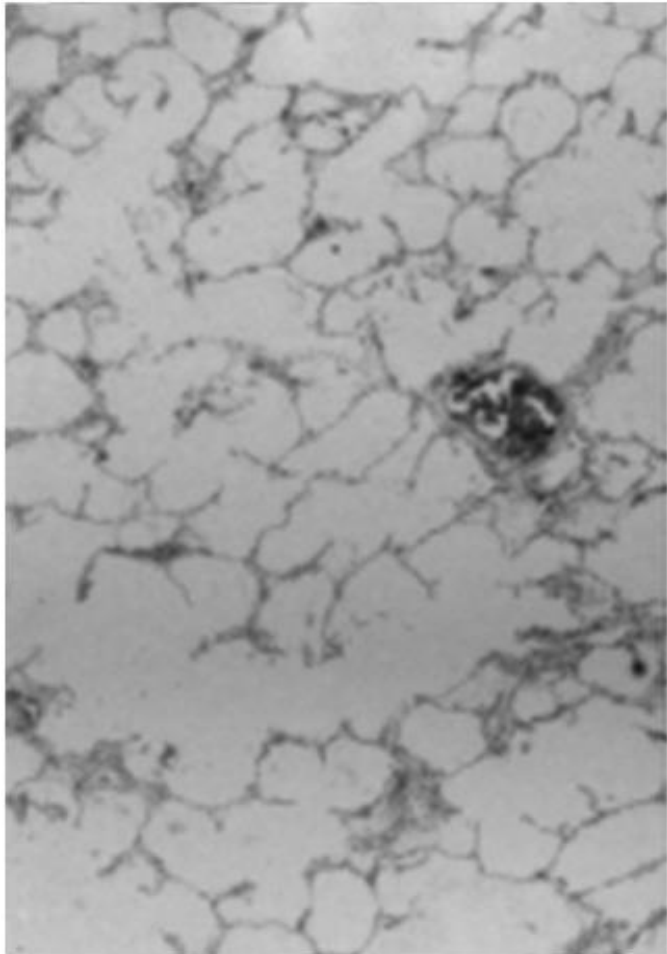
## **Microscopic:**

**Dilatation of air sacs**

**Destruction of acinar septal walls.**



Emphysematous lung with Large bullae formation



## **Clinical presentation:**

1. Chronic cough.
2. Sputum production.
3. Dyspnea.
4. Pursed-lip-breathing. The person with emphysema struggles to exhale completely, in an attempt to empty trapped air → **Pink puffers**
5. barrel chest

## **Diagnosis**

1. Pulmonary function test (PFT) → Spirometry
2. X-ray
3. CT scan
4. Blood gas analysis
5. It is diagnosed based on pathological grounds → biopsy

# CHRONIC BRONCHITIS

CLINICAL DIAGNOSIS: DAILY PRODUCTIVE COUGH FOR THREE MONTHS OR MORE, IN AT LEAST TWO CONSECUTIVE YEARS

OVERWEIGHT AND CYANOTIC



ELEVATED HEMOGLOBIN



PERIPHERAL EDEMA

RHONCHI AND WHEEZING



# EMPHYSEMA

PATHOLOGIC DIAGNOSIS: PERMANENT ENLARGEMENT AND DESTRUCTION OF AIRSPACES DISTAL TO THE TERMINAL BRONCHIOLE

OLDER AND THIN



SEVERE DYSPNEA

QUIET CHEST

X-RAY: HYPERINFLATION WITH FLATTENED DIAPHRAGMS



# Complications of COPD

COPD can cause many complications, including:

- **Respiratory infections.** People with COPD are more likely to catch infection and pneumonia.
- **Heart problems.** Can lead to cor pulmonale
- **Lung cancer.**
- **High blood pressure in lung arteries.** COPD may cause high blood pressure in the arteries that bring blood to your lungs (pulmonary hypertension).
- **Hypercoagulability:** Systemic inflammatory marker release → Stroke, pulmonary embolism & deep vein thrombosis
- **Depression.**
- **Weight loss**
- **osteoporosis**

# **BRONCHIECTASIS**

## **Definition:**

- Permanent destruction and dilatation of the bronchial airways proximal to the terminal bronchioles, secondary to a chronic necrotizing infection of the bronchi and the bronchioles. Bronchiectasis can occur at any age

## Pathogenesis :

- Repeated attacks of obstruction and Infection of the bronchi leading to permanent destruction and dilatation of the airways.
- Repeated infection destroy the bronchial walls and lead to permanent dilatation
- The affected bronchial walls are heavily infiltrated with inflammatory cells and the lumen is filled with inflammatory exudate.
- The mucosal surface may show squamous metaplastic changes.

# **Morphology:**

## **Gross:**

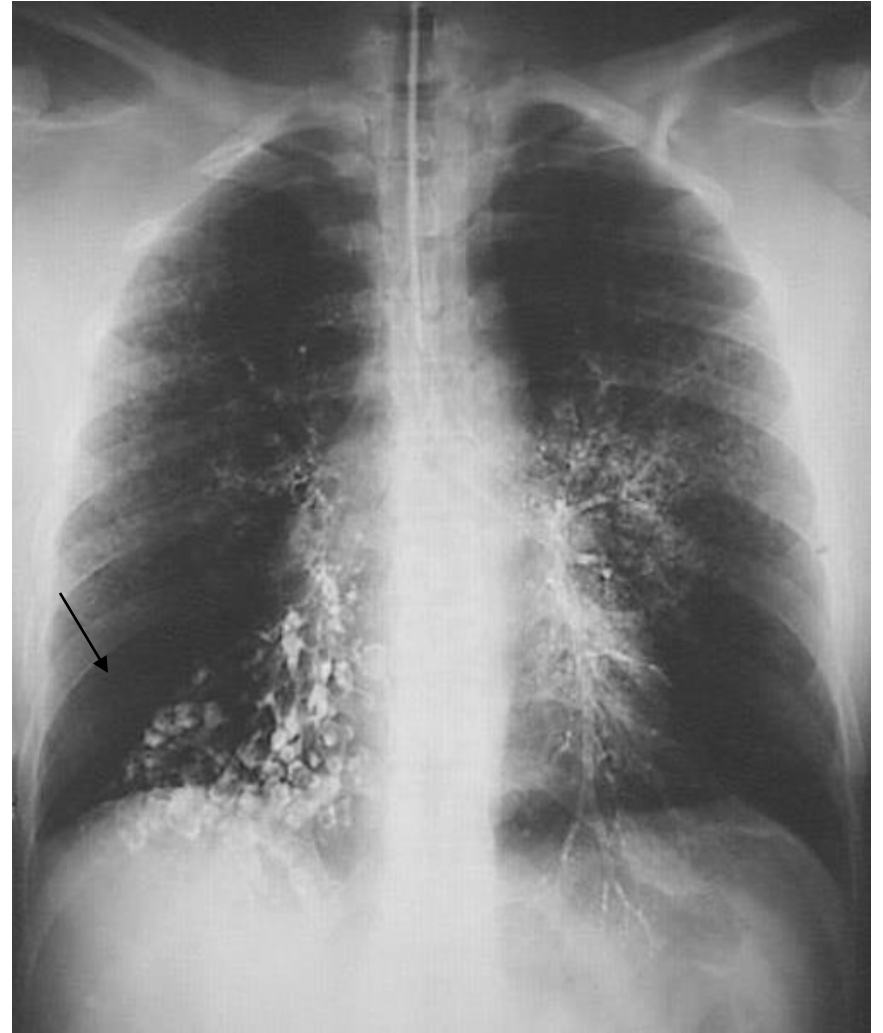
- **Lungs: Honey combed**
- **Bronchi: Dilated containing mucous and pus**

## **Microscopic:**

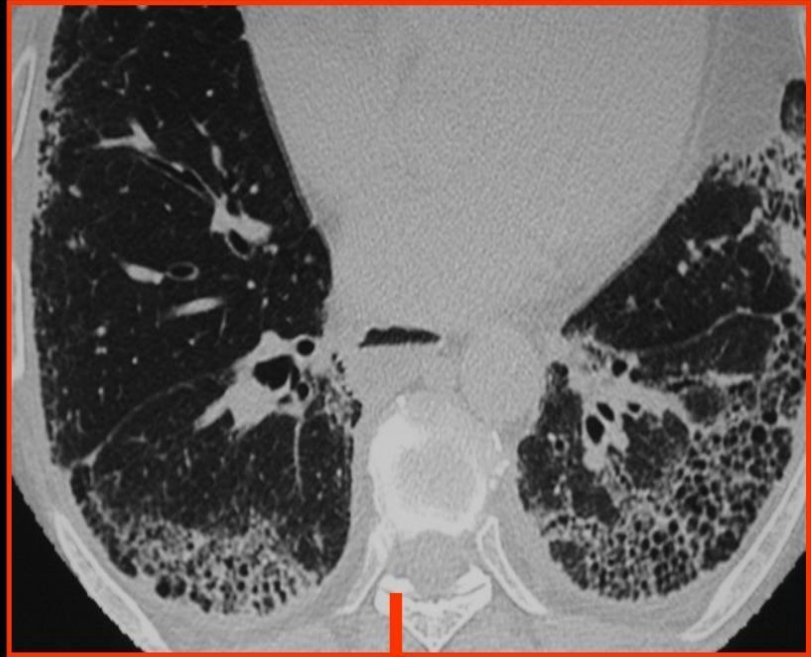
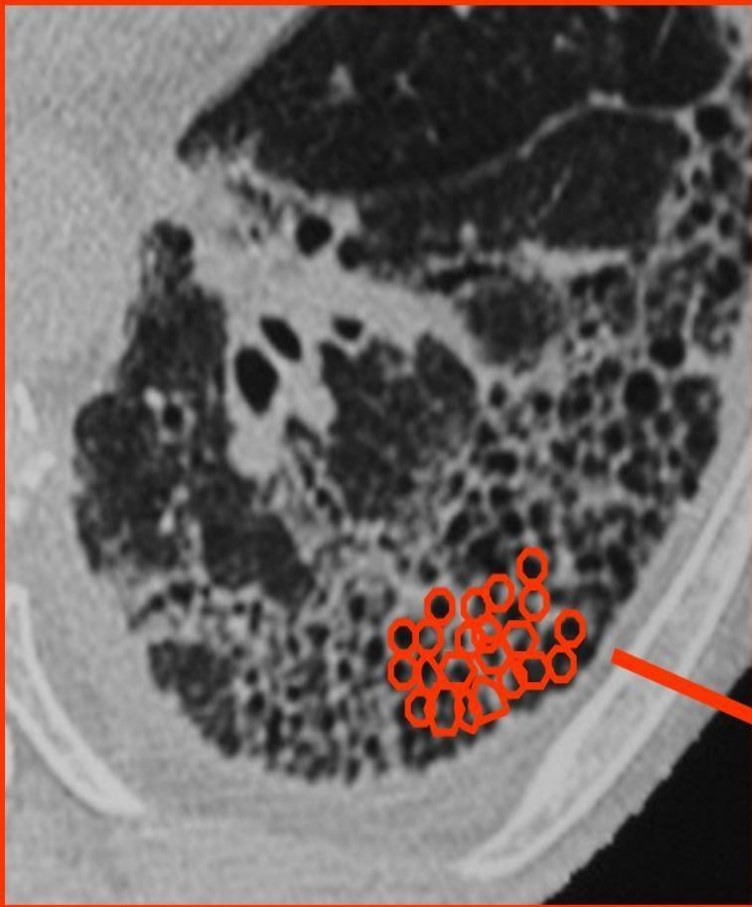
- **Ulceration of the bronchial wall, squamous metaplasia, and mucous gland hyperplasia.**
- **Inflammatory cell infiltration.**
- **Destruction of Smooth muscle and elastic tissue**



Dilated bronchioles seen in bronchiectasis



X-ray show dilated bronchioles

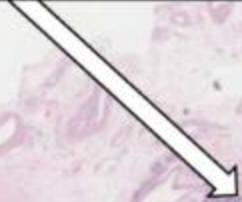


**HONEYCOMB LUNG**

**Lung: Bronchiectasis (low power)**

Histology of the *bronchi* shows grossly dilated lumina.

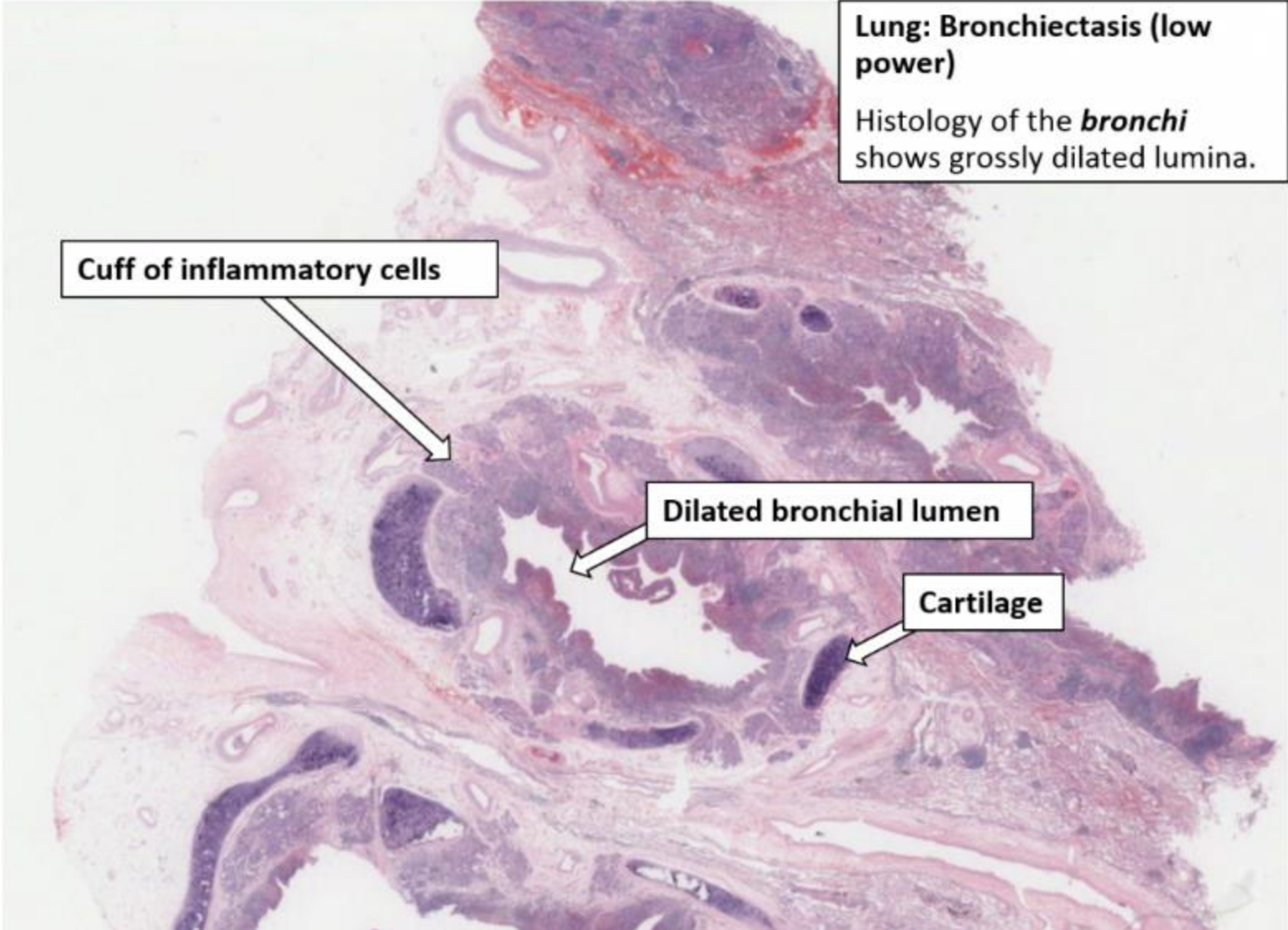
**Cuff of inflammatory cells**



**Dilated bronchial lumen**



**Cartilage**



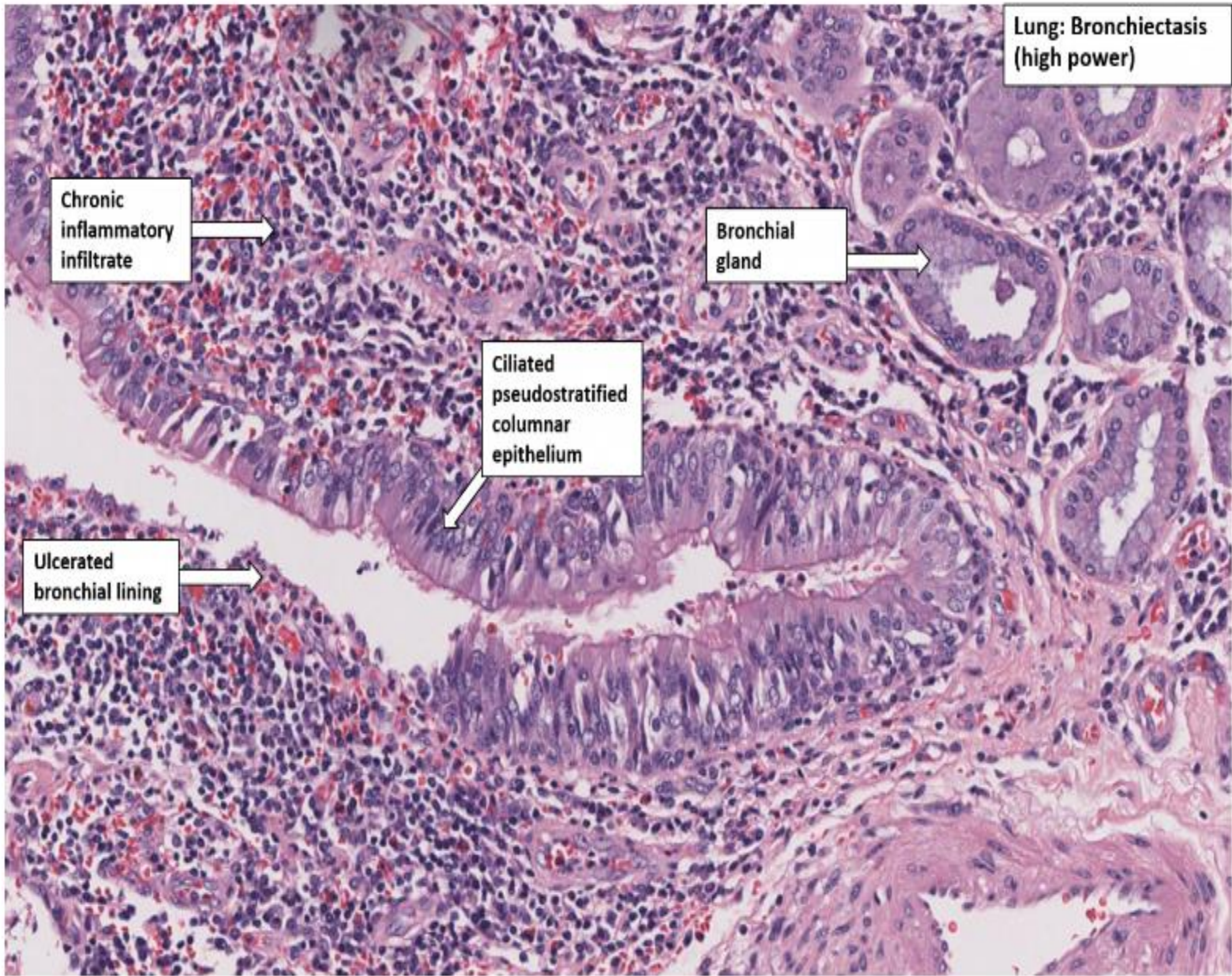
Lung: Bronchiectasis  
(high power)

Chronic  
inflammatory  
infiltrate

Bronchial  
gland

Ciliated  
pseudostratified  
columnar  
epithelium

Ulcerated  
bronchial  
lining



## **Clinical presentation:**

1. Chronic cough.
2. Sputum production.
3. Dyspnea.
4. Wheezing
5. Repeated, purulent respiratory tract infections
6. Hemoptysis occurs in 50–70% of cases

## **Diagnosis**

1. Pulmonary function test (PFT) → Spirometry
2. X-ray
3. CT scan (especially with high-resolution images)
4. Blood gas analysis

# Asthma (Extrinsic)

- Asthma is a disease of increasing prevalence that is a result of genetic predisposition and environmental interactions; it is one of the most common chronic diseases of childhood.
- Asthma is primarily a chronic inflammatory disease of the airways of the lung for which there is no known cure or primary prevention.
- Asthma is characterized by either the intermittent or persistent presence of highly variable degrees of airflow obstruction from airway wall inflammation and bronchial smooth muscle constriction; in some patients, persistent changes in airway structure occur.
- Two key mechanisms: Bronchospasm & inflammation
- Bronchial smooth muscle constriction is prevented or treated most effectively with inhaled  $\beta$ 2-adrenergic receptor agonists.
- The inflammatory process in asthma is treated most effectively with corticosteroids.

# Etiology

**TABLE 28-1** List of Agents and Events Triggering Asthma

## Respiratory infection

Respiratory syncytial virus (RSV), rhinovirus, influenza, parainfluenza, *Mycoplasma pneumonia*

## Allergens

Airborne pollens (grass, trees, weeds), house-dust mites, animal danders, cockroaches, fungal spores

## Environment

Cold air, fog, ozone, sulfur dioxide, nitrogen dioxide, tobacco smoke, wood smoke

## Emotions

Anxiety, stress, laughter

## Exercise

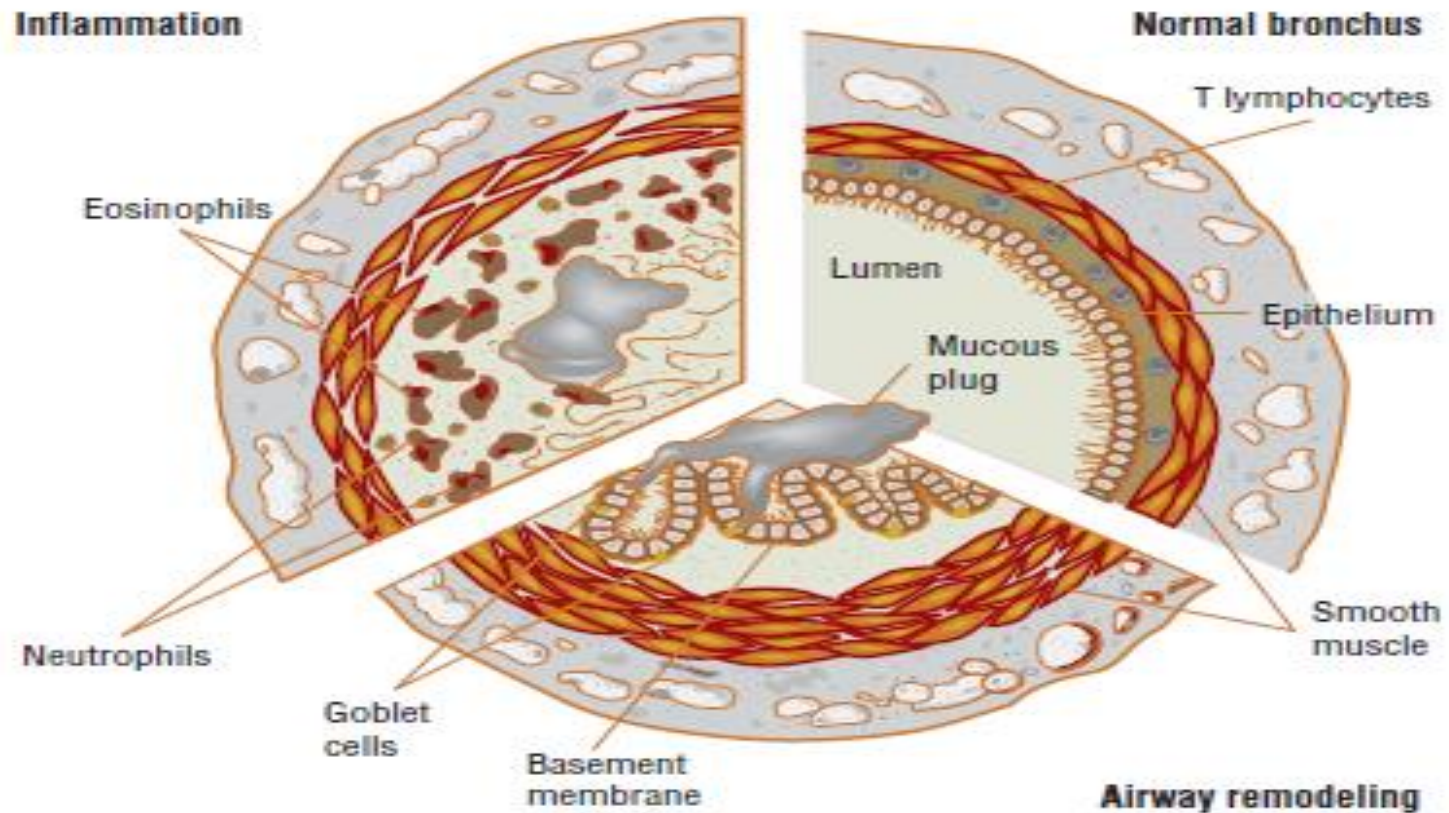
Particularly in cold, dry climate

## Drugs/preservatives

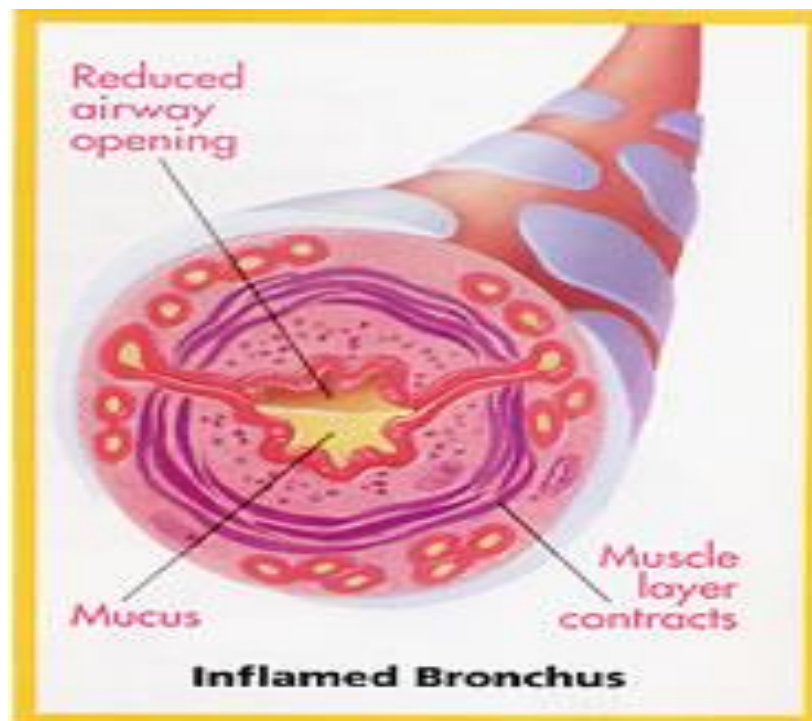
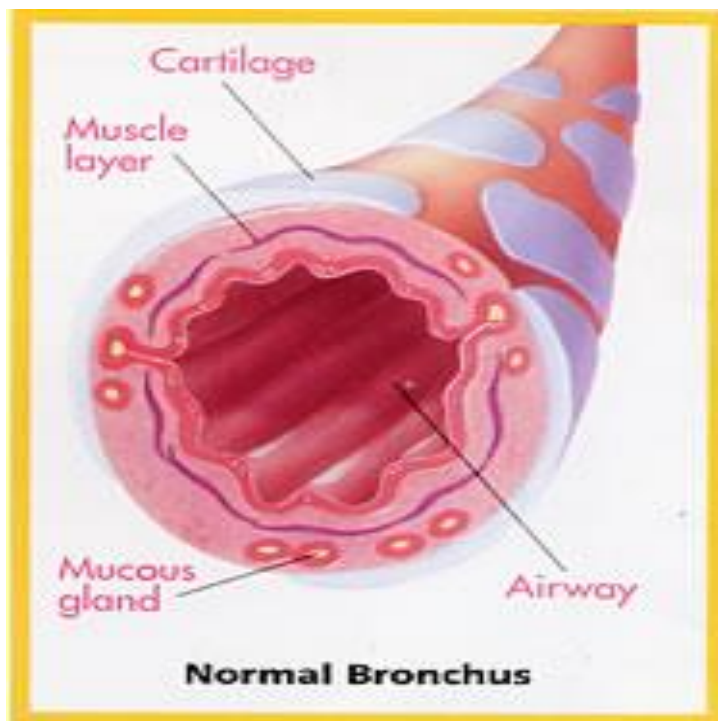
Aspirin, nonsteroidal antiinflammatory drugs (cyclooxygenase inhibitors), sulfites, benzalkonium chloride, nonselective  $\beta$ -blockers

## Occupational stimuli

Bakers (flour dust); farmers (hay mold); spice and enzyme workers; printers (arabic gum); chemical workers (azo dyes, anthraquinone, ethylenediamine, toluene diisocyanates, polyvinyl chloride); plastics, rubber, and wood workers (formaldehyde, western cedar, dimethylethanolamine, anhydrides)



**FIGURE 28-1.** Representative illustration of the pathology found in the asthmatic bronchus compared with a normal bronchus (*upper right*). Each section demonstrates how the lumen is narrowed. Hypertrophy of the basement membrane, mucus plugging, smooth muscle hypertrophy, and constriction contribute (*lower section*). Inflammatory cells infiltrate, producing submucosal edema, and epithelial desquamation fills the airway lumen with cellular debris and exposes the airway smooth muscle to other mediators (*upper left*).



# Pathogenesis of Asthma

- Inhaled allergens in allergic patients causes **type I hypersensitivity** response.
- After rapid activation, airway mast cells and macrophages release inflammatory mediators such as histamine that induce contraction of airway smooth muscle, mucus secretion, vasodilation, and exudation of plasma in the airways.
- Plasma protein leakage induces a thickened, engorged, edematous airway wall and narrowing of lumen with reduced mucus clearance.
- There is a variable degree of airflow obstruction (related to inflammation, bronchospasm, edema, and hypersecretion and bronchial hyper-responsiveness)

# CLINICAL PRESENTATION

## CHRONIC ASTHMA

- Symptoms include episodes of dyspnea, chest tightness, coughing (particularly at night), wheezing, or a whistling sound when breathing. These often occur with exercise but may occur spontaneously or in association with known allergens.
- Asthma can vary from chronic daily symptoms to only intermittent symptoms. Intervals between symptoms may be days, weeks, months, or years.

## ACUTE SEVERE ASTHMA

- Uncontrolled asthma can progress to an acute state in which inflammation, airway edema, mucus accumulation, and severe bronchospasm result in profound airway narrowing that is poorly responsive to bronchodilator therapy.

# Intrinsic Asthma

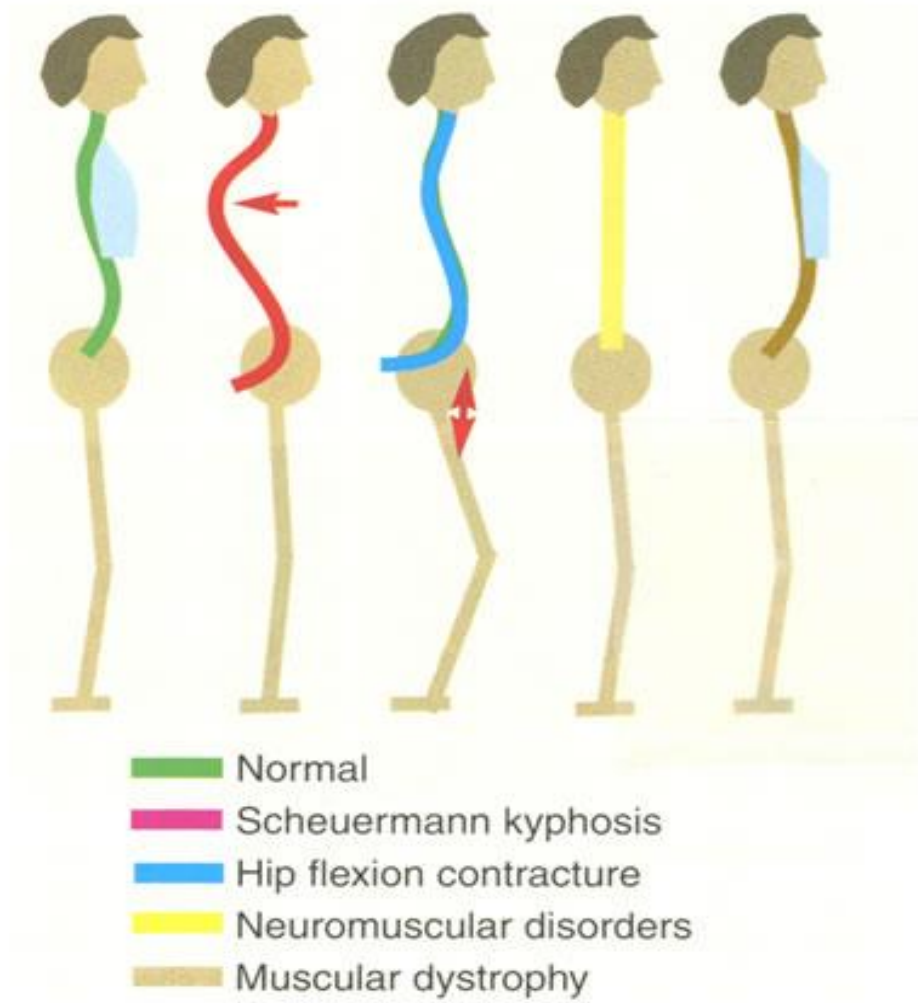
## *Non-allergic asthma*

- **Less than 10% of patients of Asthma**
- **Idiopathic**
- **Precipitating Factors**
  - Irritant exposure
    - (Air Pollution, Fumes, Perfumes, Household cleaning agents, Insecticides, paint, tobacco, cold air)
  - Infection
  - Gastro-Esophageal Reflux Disease (GERD)
- **Epidemiology**
  - More common in adults than children
  - Onset age over 40 years old

# **Restrictive lung diseases**

# Restrictive lung disorders

- **2 groups of diseases**
  - 1. Abnormalities of chest wall which limits lung expansion**
    - Includes:
      - » Kyphosis
      - » Scoliosis
      - » Muscular dystrophy
  - 2. Disease affecting lung tissue that provides supporting framework**
    - Includes:
      - » Idiopathic pulmonary fibrosis (autoimmune disease)
      - » Pulmonary edema
      - » Pulmonary embolism
      - » Acute respiratory distress syndrome (ARDS)



## Scoliosis



## Kyphosis



# Vascular disorders

## Pulmonary edema

- **Pathogenesis**
  - Fluid collection (edema) in all lung tissues
    - Affects gas exchange
    - Affects lung expansion
  - Key = pulmonary capillary pressure increases & fluid moves into alveoli
    - Capillaries rupture & get bloody sputum (hemoptysis)
- **medical emergency**
- **Etiology**
  - Left sided heart failure
  - Hypoproteinemia
  - Inhalation of toxic gases
  - Lymphatic blockage (e.g. from tumor)

**THANK YOU**