

PATHOLOGY OF DISEASES OF THE RESPIRATORY SYSTEM

Atelectasis

collapse of lung tissue → an airless lung parenchyma.

Inherited or acquired.

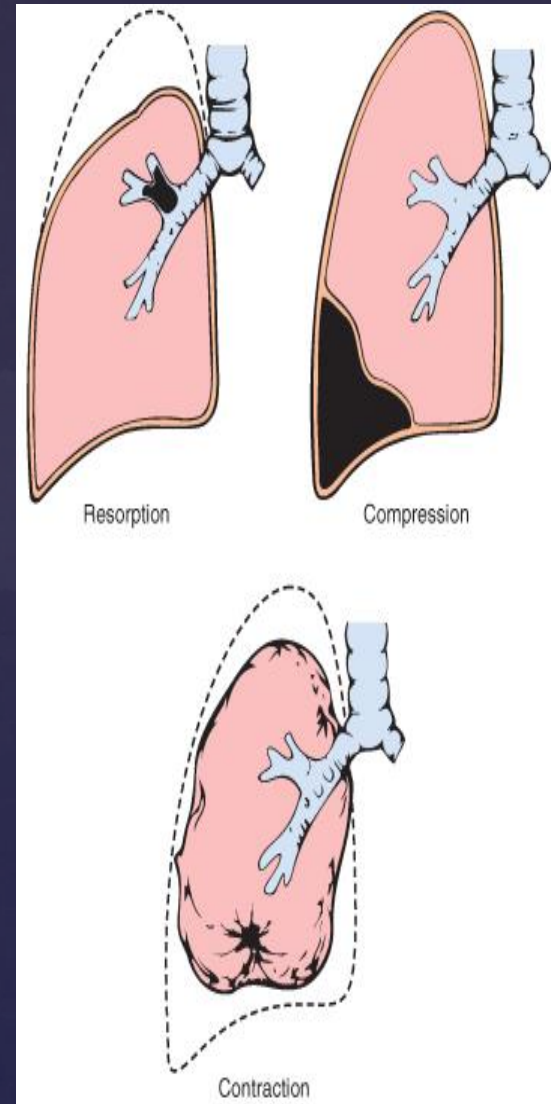
❖ **Acquired atelectasis** can be subdivided into:

1. Resorption (obstructive) atelectasis:

consequence of complete obstruction of an airway → resorption of the oxygen trapped in alveoli, without impairment of blood flow.
(reversible)

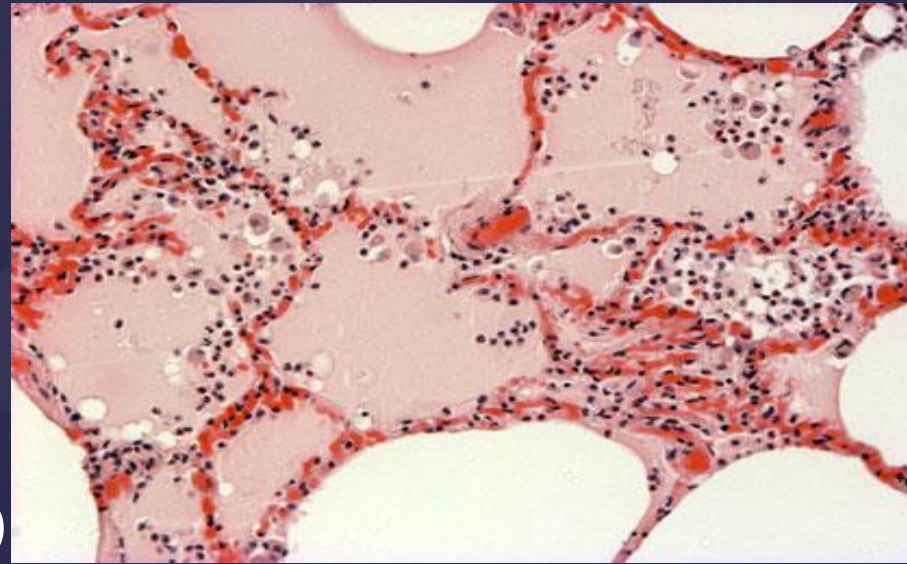
2. Compression atelectasis: pleural cavity (partially or completely) filled by fluid exudates (pleural effusion), tumor, blood (hæemothorax), or air (pneumothorax). (reversible)

3. Contraction atelectasis: local or generalized fibrotic changes in the lung or pleura prevent full expansion. (irreversible)



Pulmonary Oedema

accumulation of excess fluid in the interstitial (extracellular) spaces of lung tissue (alveolar lumen) as transudate or non-inflammatory fluid, Alveolar walls are thickened due to acute distention of capillaries and interstitial edema



Causes:

1. Hemodynamic:

- ↑ hydrostatic pressure
(↑ pulmonary venous pressure)
 - Left-sided heart failure (common)
 - Volume overload
 - Pulmonary vein obstruction
- ↓ oncotic pressure (less common)
 - Hypoalbuminemia

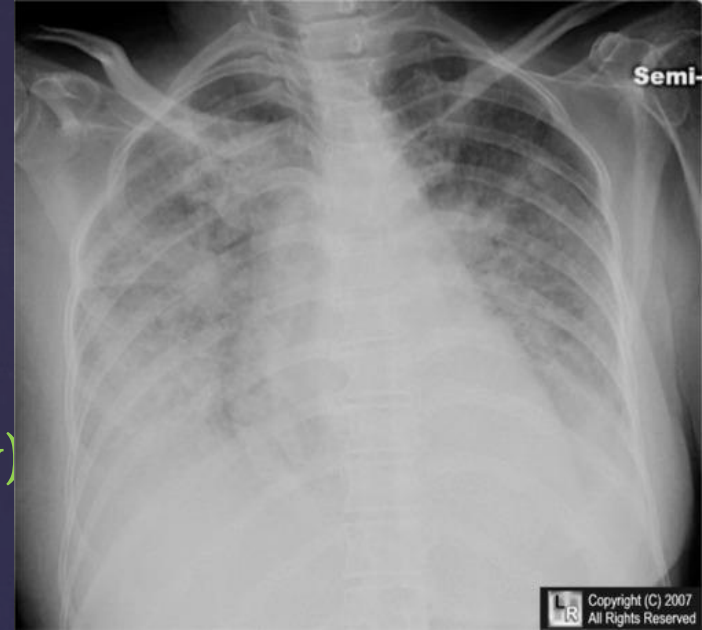
- Nephrotic syndrome
- Liver disease
- Protein-losing enteropathies
- Lymphatic obstruction (rare)

2. Microvascular injury (alveolar injury)

- Infections: pneumonia, septicemia
- Inhaled gases: oxygen, smoke
- Liquid aspiration: gastric contents
- Drugs and chemicals (bleomycin, amphotericin B, heroin)
- Shock, trauma
- Radiation
- Transfusion

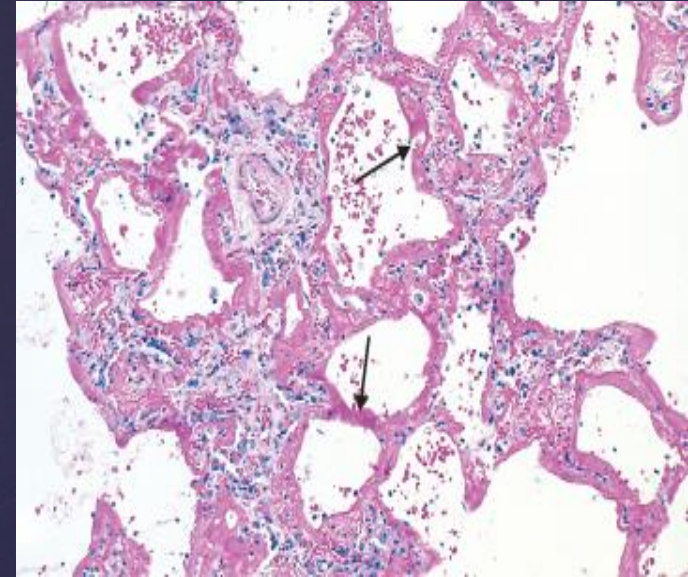
3. Unknown:

- High altitude
- Neurogenic (CNS trauma)



Acute Respiratory Distress Syndrome (ARDS) and Diffuse Alveolar Damage (DAD)

Clinical syndrome caused by diffuse alveolar capillary damage, with rapid onset of severe life-threatening respiratory insufficiency, cyanosis, and severe arterial hypoxemia refractory to oxygen therapy that may progress to extrapulmonary multisystem organ failure.



Causes:

1. Infection

Viral, *Mycoplasma*, and *Pneumocystis* pneumonia; miliary tuberculosis, Gastric aspiration

2. Physical/Injury

Mechanical trauma, Fractures with fat embolism, Burns, Ionizing radiation

3. Inhaled Irritants

Oxygen toxicity, Smoke

4. Chemical Injury

Heroin overdose, Acetylsalicylic acid, Barbiturate overdose

5. Hematologic Conditions

Multiple transfusions, Disseminated intravascular coagulation

6. *Pancreatitis*

7. *Uremia*

8. *Cardiopulmonary Bypass*

9. *Hypersensitivity Reactions*

Organic solvents, Drugs

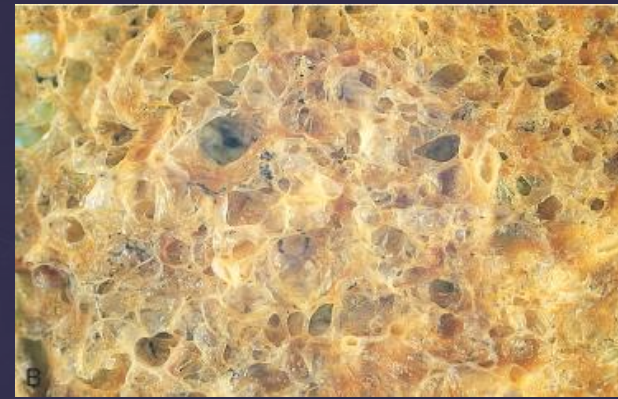
Obstructive Pulmonary Diseases

Include emphysema, chronic bronchitis, asthma, and bronchiectasis. Both emphysema and chronic bronchitis are often grouped together under the term "chronic obstructive pulmonary disease-COPD".

Clinical Term	Anatomic Site	Major Pathologic Changes	Etiology	Signs/Symptoms
Chronic bronchitis	Bronchus	Mucous gland hyperplasia, hypersecretion	Tobacco smoke, air pollutants	Cough, sputum production
Bronchiectasis	Bronchus	Airway dilation and scarring	Persistent or severe infections	Cough, purulent sputum, fever
Asthma	Bronchus	Smooth muscle hyperplasia, excess mucus, inflammation	Immunologic or undefined causes	Episodic wheezing, cough, dyspnoea
Emphysema	Acinus (alveoli)	Airspace enlargement; wall destruction	Tobacco smoke	Dyspnoea

Emphysema

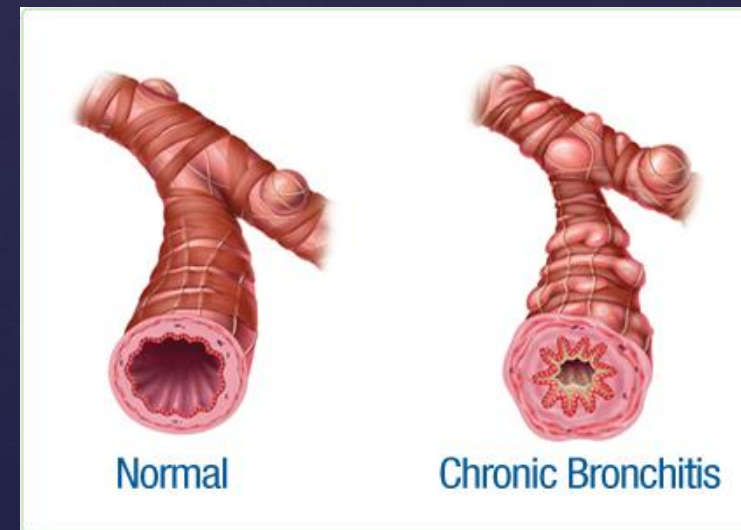
Condition of the lung characterized by abnormal permanent enlargement of the airspaces distal to the terminal bronchiole, accompanied by destruction of their walls without obvious fibrosis.



Four pathologic variants are recognized: centriacinar, panacinar (panlobular), distal acinar (paraseptal) and irregular. Death in most patients with is due to (1) respiratory acidosis and coma, (2) right-sided heart failure, and (3) massive collapse of the lungs secondary to pneumothorax. Treatment options include bronchodilators, steroids, bullectomy, and in selected patients, lung volume reduction surgery and lung transplantation.

Chronic bronchitis

persistent cough with sputum production for at least 3 months in at least 2 consecutive years, in the absence of any other identifiable cause. It is so common among habitual smokers and inhabitants of smog-laden cities.



When persistent for years, it may

- (1) progress to chronic obstructive airway disease,
- (2) lead to cor pulmonale and CHF
- (3) cause atypical metaplasia and dysplasia of the respiratory epithelium, with malignant transformation.

The key pathological change is **mucus hypersecretion** caused by hyperplasia of submucous glands and goblet cells.

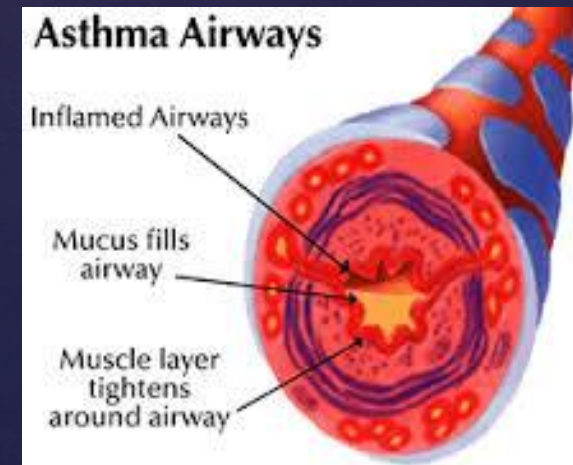
Histologically:

1. Goblet cell metaplasia with mucus plugging of the lumen.
2. Clustering of alveolar macrophages.
3. Inflammatory infiltration.
4. Fibrosis of the bronchiolar wall.



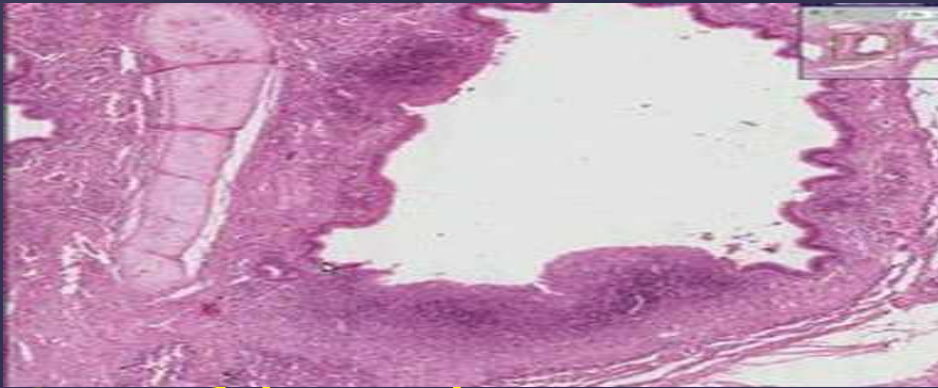
Asthma

chronic inflammatory disorder of the airways that causes recurrent episodes of wheezing, breathlessness, chest tightness, and cough, particularly at night and/or in the early morning, usually associated with reversible **bronchoconstriction and airflow limitation**.



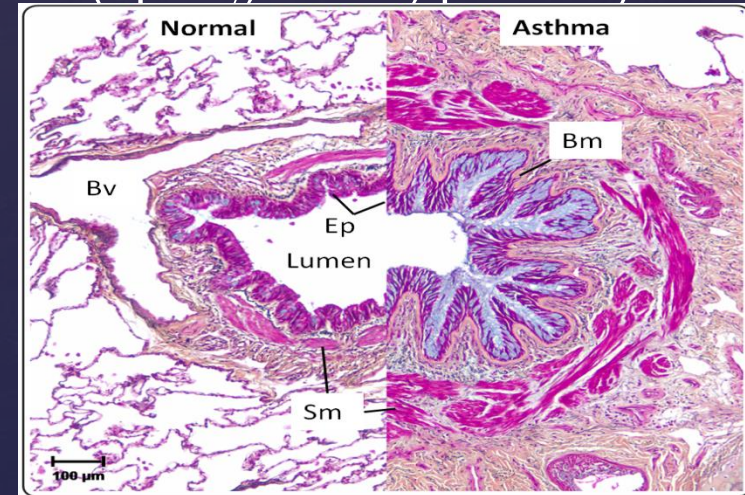
The inflammation → ↑ in airway responsiveness (bronchospasm) to a variety of stimuli, including extrinsic (allergic) and intrinsic (nonimmune). Asthma can be classified into:

1. **Atopic asthma**: seen mainly during childhood and precipitated by allergic reactions.
2. **Non-atopic asthma**: caused by viral infections of the respiratory mucosa.
3. **Drug-induced asthma**: e.g. aspirin-induced asthma.
4. **Occupational asthma**: precipitated by fumes (epoxy, resins, plastics).



Bronchiectasis

permanent dilation of bronchi and bronchioles caused by destruction of the muscle and elastic tissue, resulting → **chronic necrotizing infections**. It is encountered in:



1. **Congenital or hereditary conditions:** cystic fibrosis, lung sequestration, immotile cilia syndrome.
2. **Postinfectious conditions:** TB, Aspergillosis, Staph. pneumonia.
3. **Bronchial obstruction** by tumour, foreign bodies, mucus plugs.
4. **Autoimmune:** Rheumatoid arthritis, inflammatory bowel diseases, SLE.

Pulmonary Infections

Upper respiratory tract infections are the most common infections all over the body (pharyngitis and common cold).

Lower respiratory tract infections (pneumonias) Caused by:

1. Loss or suppression of the cough reflex (coma, anaesthesia).
2. Injury to the mucociliary apparatus (smoke, corrosives and gases).
3. Interference with the phagocytic or bactericidal action of alveolar macrophages (alcohol, smoke, anoxia).
4. Pulmonary congestion and edema.
5. Accumulation of secretions (cystic fibrosis).

Bacterial pneumonia: inflammation and consolidation of the lung tissue.

Two pathological patterns;

❖ **Bronchopneumonia** with lobular distribution

❖ **Lobar pneumonia:** four stages are seen:

❑ **Congestion** (Cellular exudates containing neutrophils lymphocytes and fibrin replaces the alveolar air)

❑ **red hepatization** (The lungs become hyperaemic-BV engorged with blood)

❑ **grey hepatization** (Leukocytes migrate into the congested alveoli)

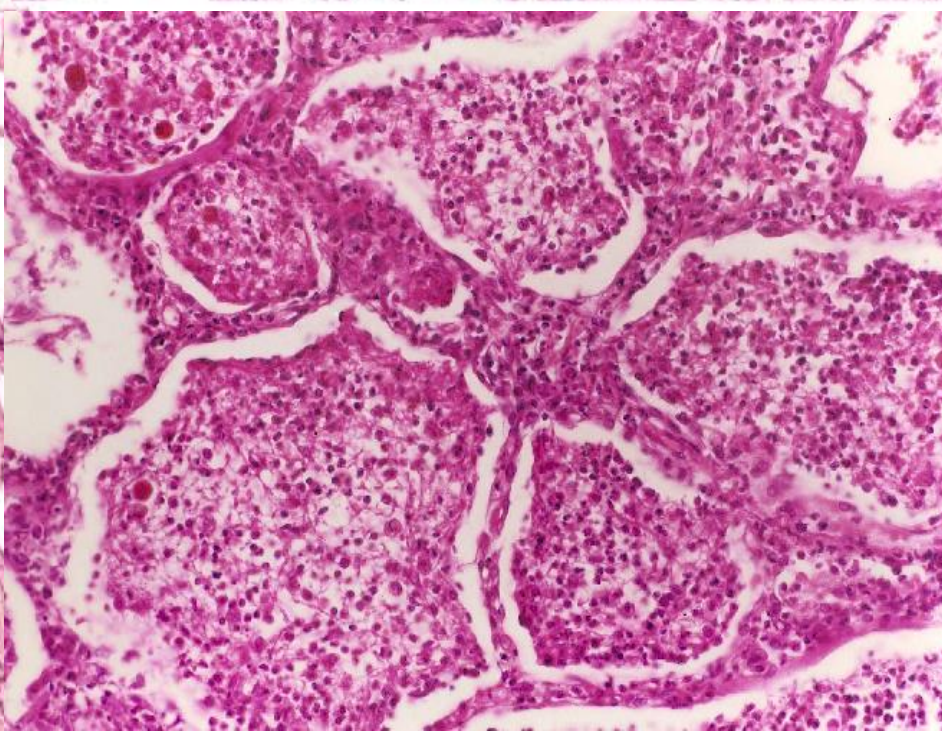
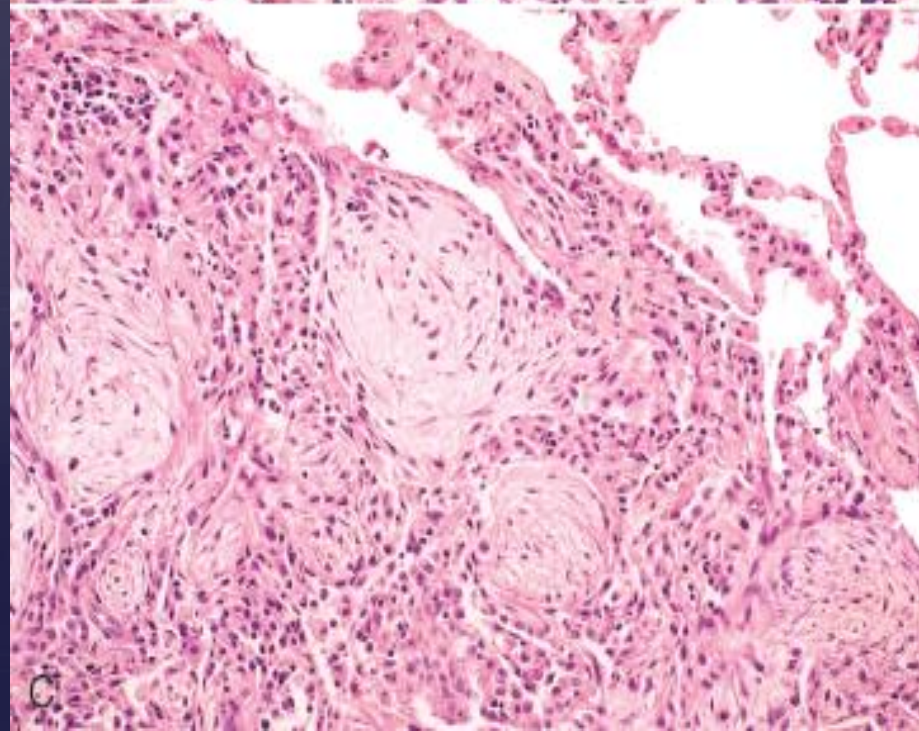
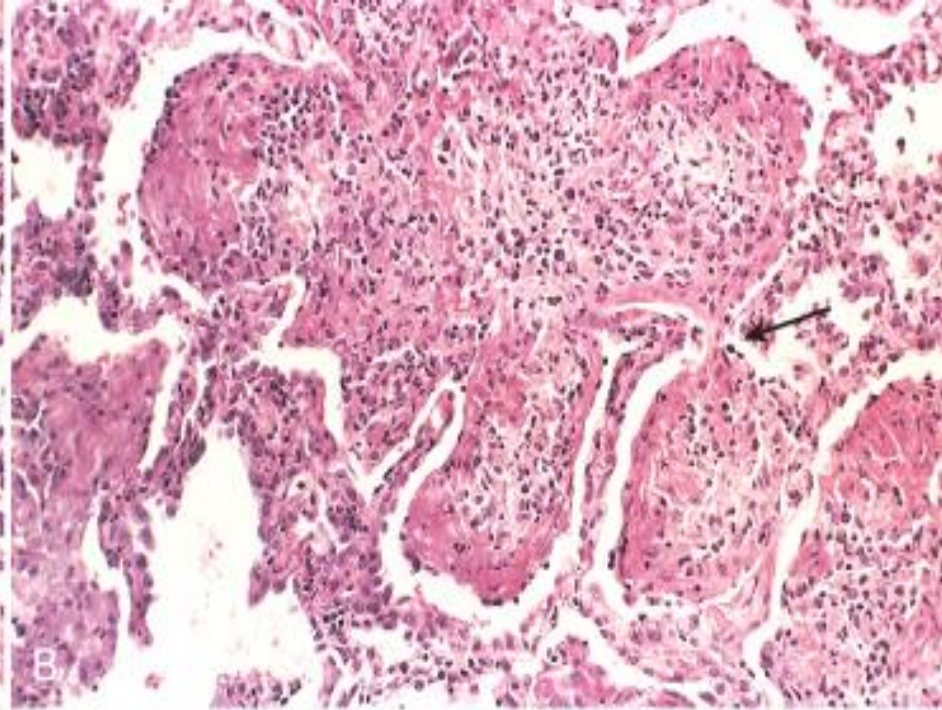
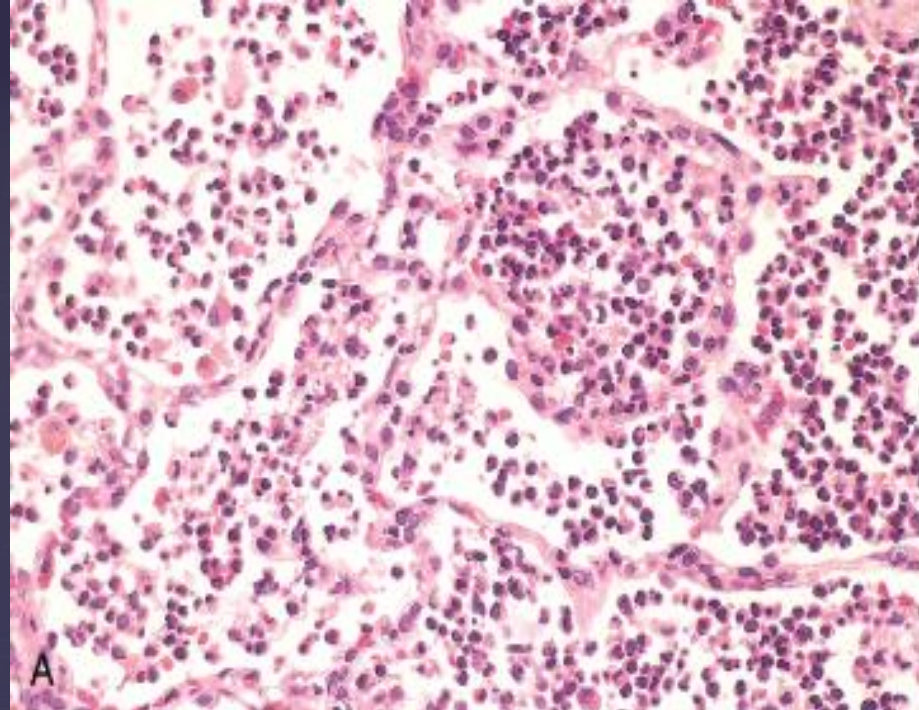
❑ **resolution** (resorption and restoration of the pulmonary architecture due to phagocytosis).

Complications include:

➤ pleuritis

➤ abscess formation,

➤ bacteremia.



Tumors

most frequently diagnosed major cancer in the world and the most common cause of cancer mortality.

Risk Factors: Include:

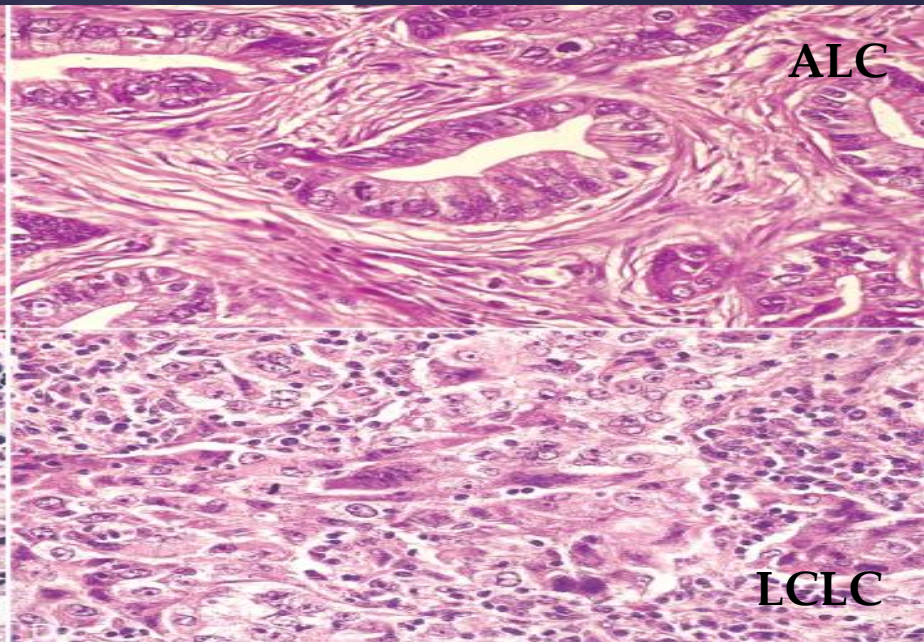
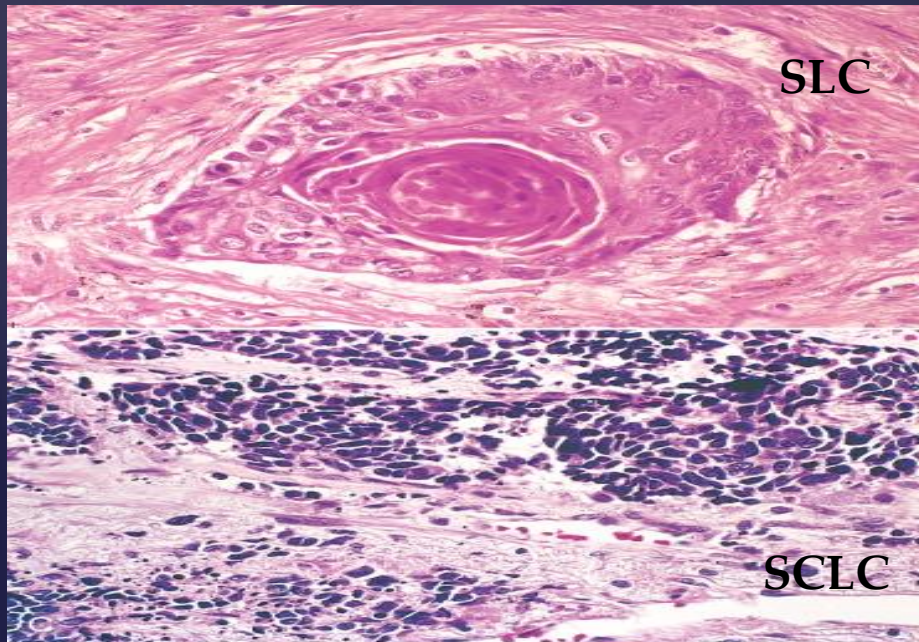
1. **Cigarette smoking:** ↑about 10 folds. The development of cancers is proportionate to the amount of daily smoking, tendency to inhale and the duration of smoking.
2. **Industrial hazards:** ionizing radiation and asbestos.
3. **Atmospheric pollutants.**

Pathological variants

- A. **Small cell carcinoma** (20-25%): most aggressive and rapidly growing.
small cells with a high nucleo-cytoplasmic ratio that proliferate rapidly
neuroendocrine carcinoma
usually develop peribronchially and infiltrate the bronchial submucosa

B- Non-Small cell carcinoma

1. **Squamous cell carcinoma** (25-40%): produce keratin in the same way as normal squamous epithelial cells, develop from a precancerous lesion preceded by squamous metaplasia and squamous cell hyperplasia.
2. **Adenocarcinoma** (25-40%): glandular tumor cells producing mucous, grow in the peripheral part of the lung. A special variant is adenocarcinoma in situ, previously (bronchoalveolar carcinoma)
3. **Large cell carcinoma** (10-15%): large and there is no special differentiation. A variant is the large cell neuroendocrine carcinoma.



THANK YOU