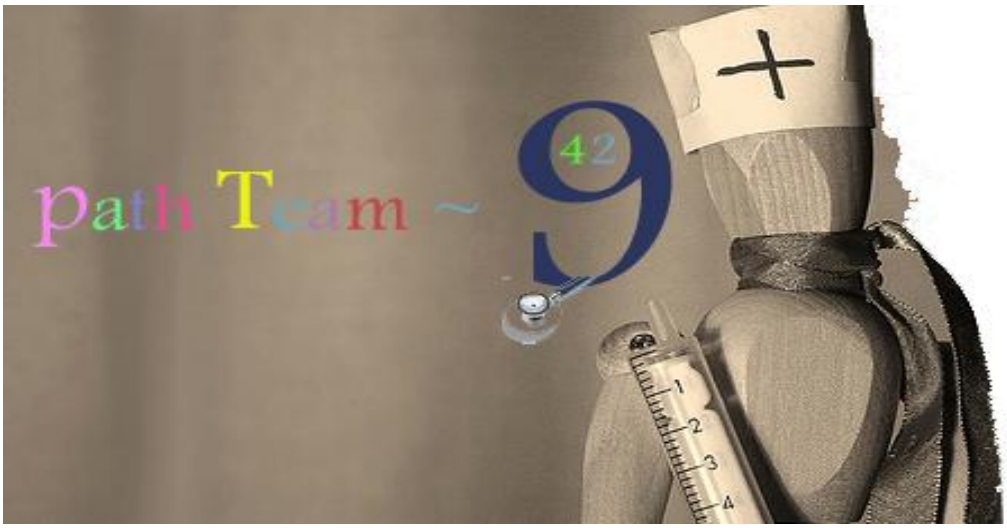

Chronic Obstructive Pulmonary Diseases

هذه المذكرة عبارة عن هاند أوت الدكتور**عمار الركابي** بعد تنسيقها.
شاملة لكل ما هو مطلوب في هذه الجزئية

وأيضا:
توجد بعض الجداول والصور التوضيحية مضافة من محاضرات الدكتورة **مها عرفة**.

Path Team – 429

Work by:

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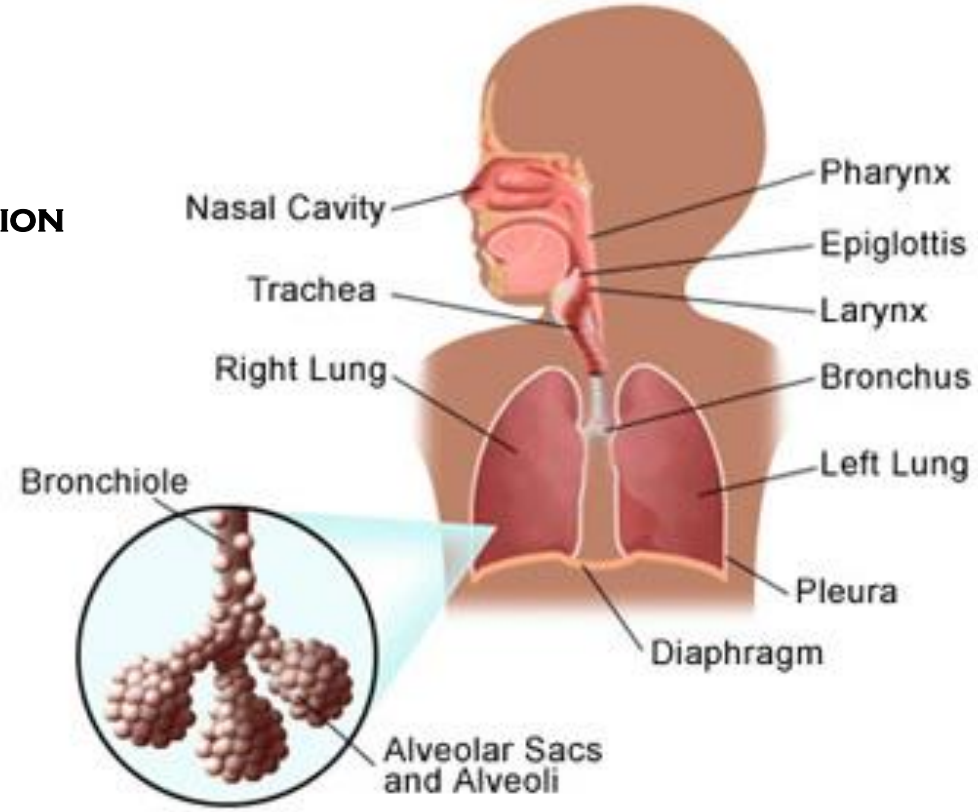
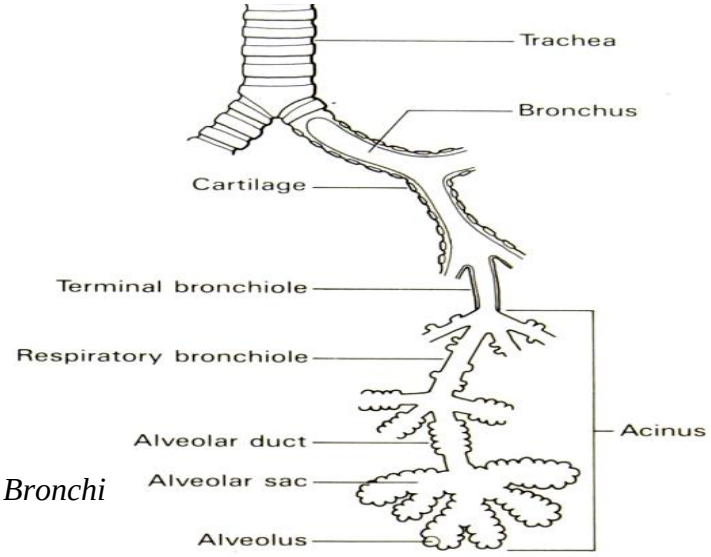
INTRODUCTION



❖ NORMAL ANATOMY

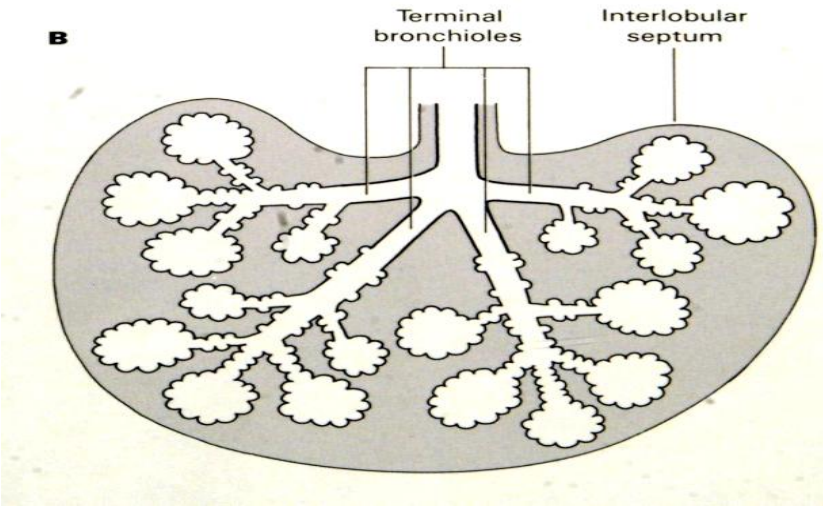
The *air conducting passages* consist of:

1- The nasal cavities.
2- Paranasal sinuses.
3- Nasopharynx.
4- Oropharynx.
5- Hypopharynx (epiglottis and larynx).
6- Tracheobronchial tree.



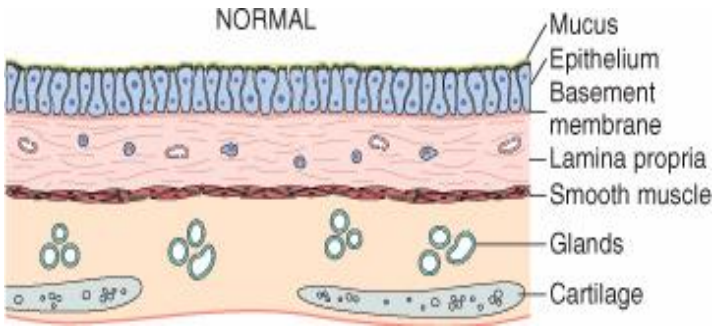
- **At *the carina*, the trachea branches into:**
  the *Mainstem Bronchi* → *Lobar Bronchi* → *Segmental Bronchi*
  (<u>supply</u> the *Intralobar Bronchopulmonary Segments*).

- ***Further branching* produces**:
  1- Subsegmental bronchi.
  2- Bronchioles.
  3- Terminal bronchioles.
  4- Respiratory bronchioles.
  5- Alveolar ducts
  6- Alveolar sacs.



<u>The pulmonary arteries</u> follow the airways
<u>The pulmonary veins</u> run through the C.T septa.
<u>Lymphatic channels</u>
are *present* along the bronchovascular structures but are also *found in* the pleura and C.T septa.

❖ Histology



With the exception of **the *Oropharynx*** and **portions of** the *Nasopharynx* and *Nypopharynx*
(which are lined by squamous epithelium)

The ***Upper Respiratory Tract*** and ***The Large Airways*** are lined by:
pseudostratified ciliated columnar epithelium interspersed with mucus-secreting goblet cells and neuroendocrine cells.

- **Mucus-secreting glands** lie beneath the epithelial surface and the cartilaginous plates help to maintain patency.
- **Cartilage, submucosal glands and goblet cells** are lost at the level of the **bronchioles**

  BRONCHIOLES are lined by ciliated cuboidal epithelium and **Clara cells**
  (CLARA CELLS secrete a non-mucoid watery substance that contains lysozyme and immunoglobulins).

- The majority of the **alveolar surface** is **lined by** the ***Type I Pneumocytes***
  which are interspersed with the *Surfactant* producing ***Type II (cuboidal / granular) pneumocytes.***
- **The interstitium** contains:
  collagen – elastin – mast cells – *occasional* inflammatory cells – Connective Tissue cells
  (primarily smooth muscle and fibroblasts).
- **Alveolar macrophages** are derived from blood monocytes
  are loosely attached to the alveolar wall or lie free within the alveolar space.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

General considerations

1- COPD is a group of disorders characterized by **airflow obstruction.**

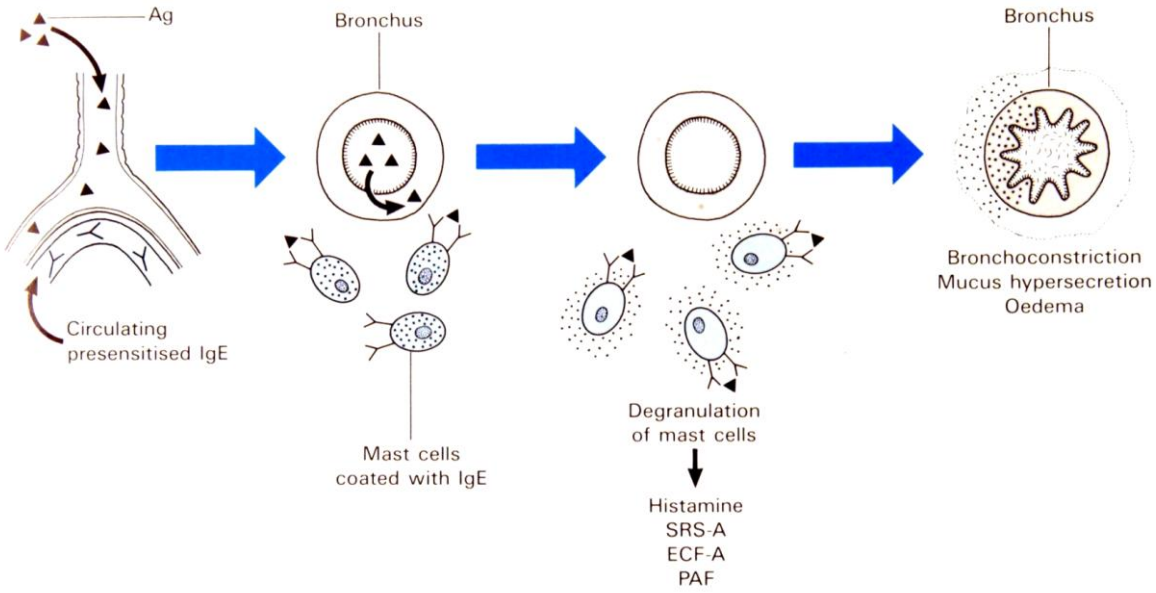
2- Characteristics include a marked decrease in the forced expiratory volume at 1st second (FEV_1).

Restrictive Pulmonary Disease: a group of disorders characterized by reduced lung capacity **due to** either, chest wall or skeletal abnormalities such as kyphoscoliosis or to interstitial and infiltrative parenchymal fibrotic disease.

Pathologic Findings in Chronic Obstructive Pulmonary Disease

Name of Disorders	Pathologic findings
Bronchial asthma	- Bronchial smooth muscle hypertrophy. - Hyperplasia of bronchial submucosal glands and goblet cells. - Airways plugged by viscid mucus **containing:** Curschmann spirals – Eosinophils – Charcot-leyden crystals.
Chronic bronchitis	- Hyperplasia of bronchial submucosal glands leading to increased ***Reid index:*** ratio of the thickness of the gland layer to that of the bronchial wall.
Pulmonary, emphysema	- Abnormal dilation of *air spaces* with destruction of alveolar walls. - Reduced lung elasticity.
Bronchiectasis	- Abnormally dilated *bronchi* which are filled with rnucusand neutrophils. . - Inflamamtion and necrosis of bronchial walls and alveolar fibrosis.

Bronchial Asthma



1] Types include: extrinsic and intrinsic asthma :

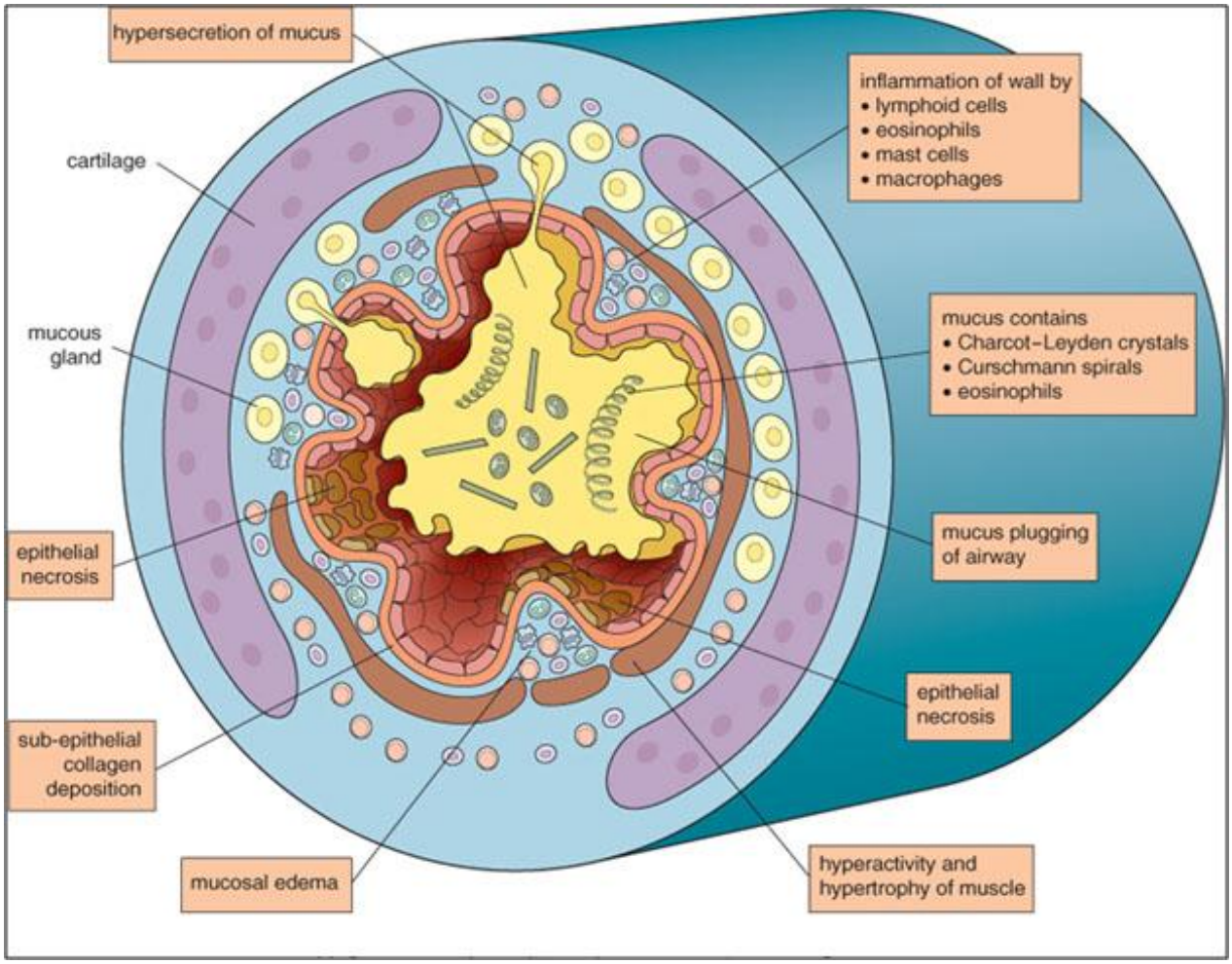
(a) **Extrinsic (immune)-asthma:**
is mediated by a type I hypersensitivity response involving ***IgE*** bound to mast cells.
The disease begins in childhood and usually in patients with a family history of allergy.

(b) **Intrinsic (non-immune) asthma** :
includes asthma associated with chronic bronchitis as well as other asthma variants such as exerciseor cold-induced asthma.
It begins in adult life and is not associated with a history of allergy.

2] Clinical presentation:

➔ There is marked episodic dyspnea and wheezing expiration caused by narrowing of the airways.
➔ Bronchial asthma is related to increased sensitivity of air passages to stimuli which leads to spasm in the bronchial muscular wall.

3] Pathological changes:

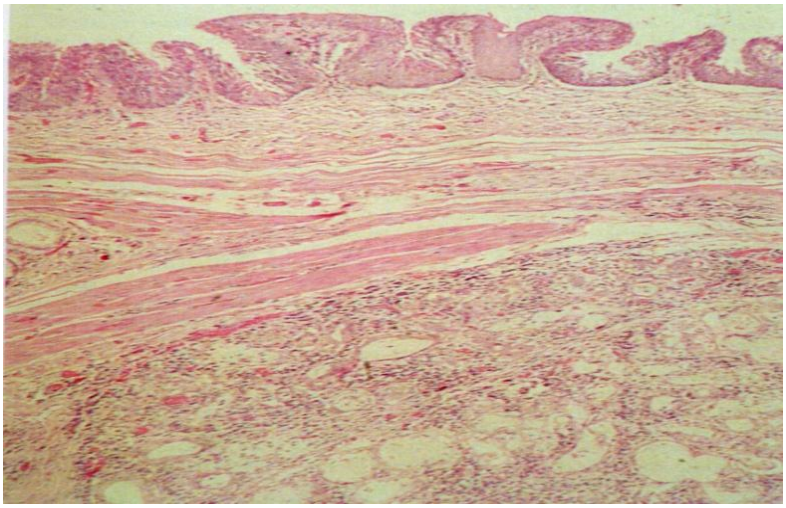


- Morphologic manifestations include:
  - ✓ Bronchial smooth muscle hypertrophy.
  - ✓ Hyperplasia of goblet cells.
  - ✓ Thickening and hyalinization of basement membranes.
  - ✓ Proliferation of eosiniophils and intrabronchial mucous plugs with whorl-like accumulations of epithelial cells (Curschmann spirals).
  - ✓ Crystalloids of eosinophil-derived proteins (Charcot-Leydencrystals).

- Complications include:
  - ✓ Superimposed infection.
  - ✓ Chronic bronchitis.
  - ✓ Pulmonary emphysema.

  → Bronchial asthma may also lead to **status asthmaticus** which is a prolonged bout of bronchial asthma that can last for days and responds poorly to therapy. Death can result from status asthmatics.

Chronic Bronchitis



1] Clinical presentation:

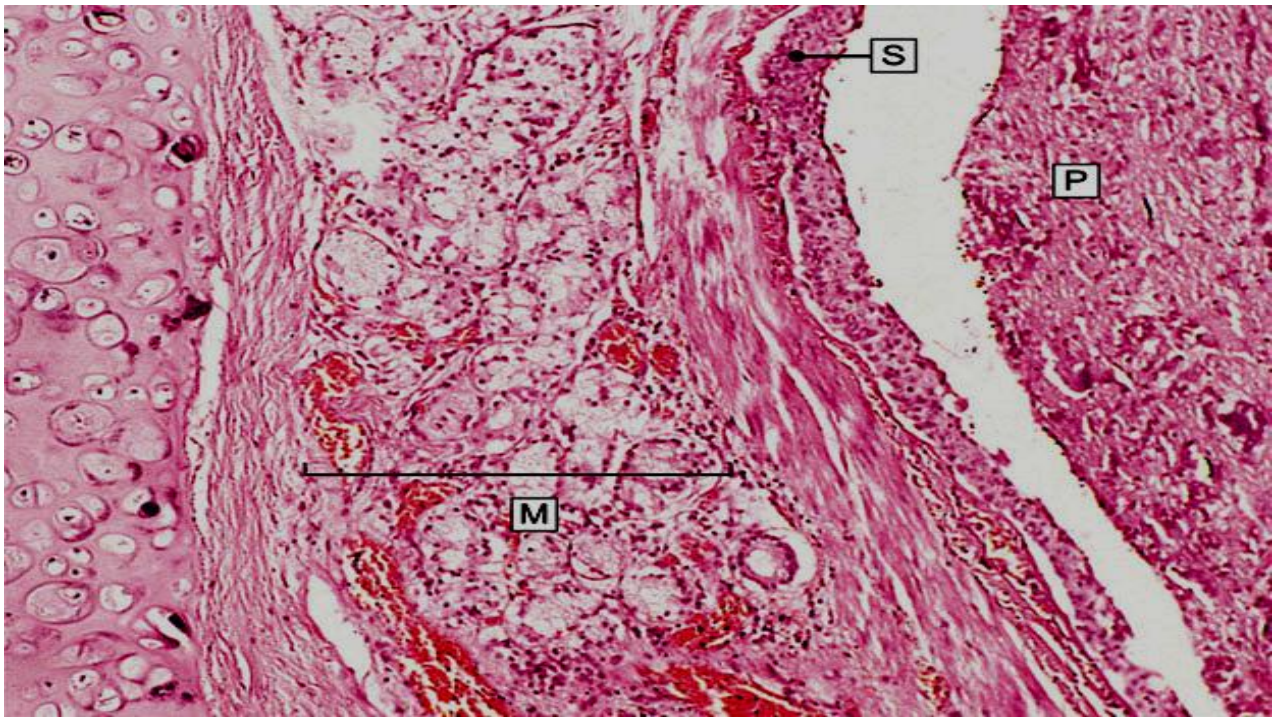
- The clinical definition is of chronic bronchitis is:
  a productive cough that occurs during at least 3 consecutive months over at least 2 consecutive years.

r»

➔ Chronic bronchitis is clearly linked to **cigarette smoking** and is also associated with air pollution, infection and genetic factors.

➔ It may lead to cor pulmonale. (Heart failure induced by pulmonary diseases).

2] Pathological changes:



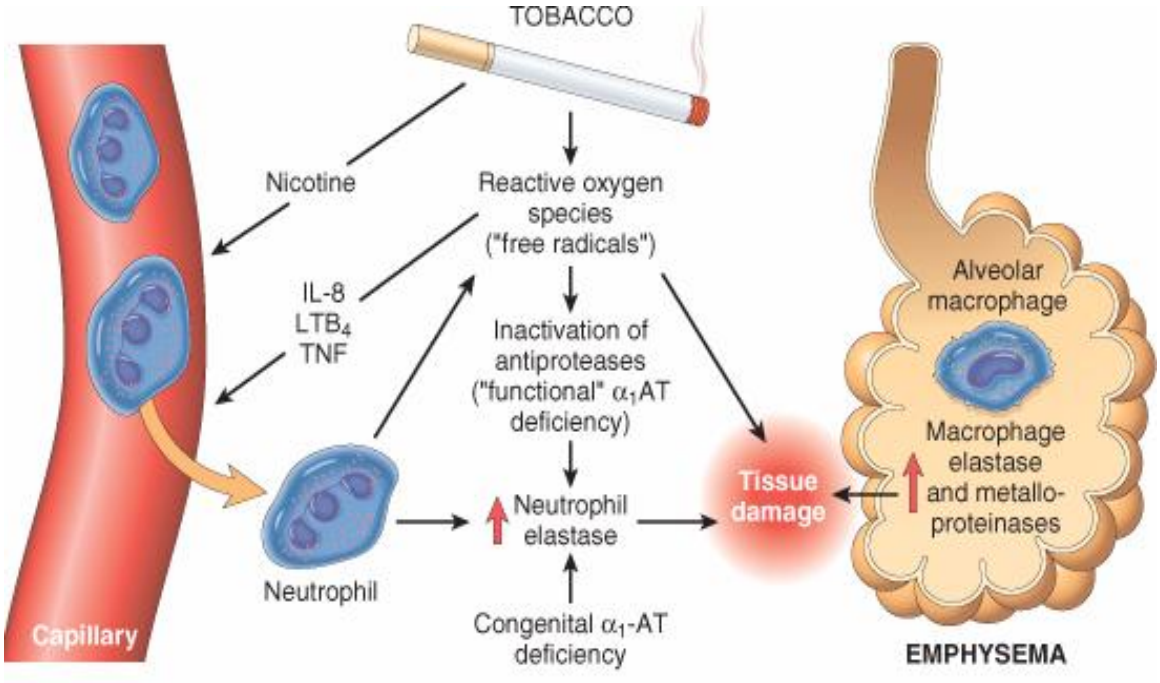
✓ Typical characteristics include:
hypersecretion of mucus due to marked hyperplasia of mucus-secreting submucosal glands.

Emphysema

1] General considerations, definitions and clinical features:

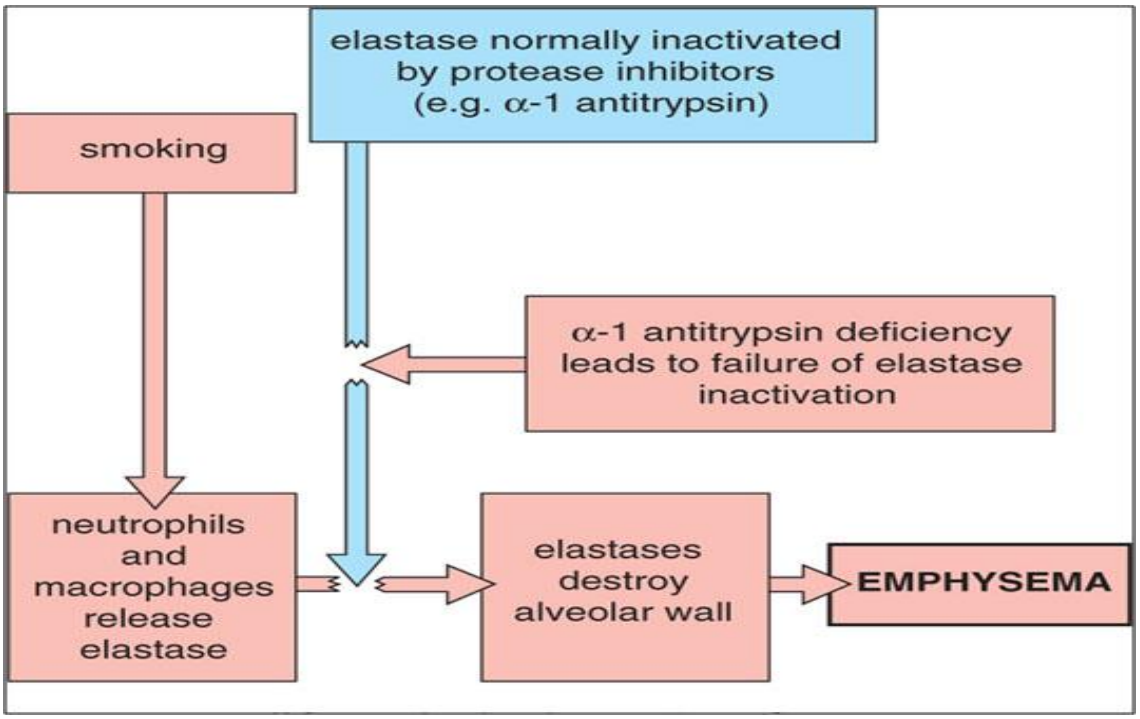
(a) **Emphysema**: is dilation of air spaces from and beyond the respiratory bronchioles with destruction of alveolar walls.

(b) The disease is strongly associated with **cigarette smoking**.



2] Clinical characteristics:

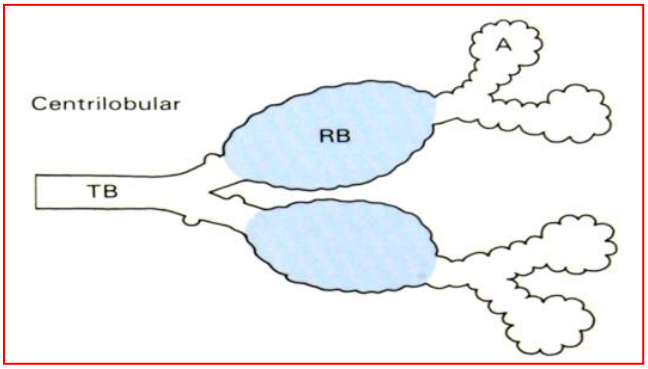
- Include increased anteroposterior diameter of the chest (Barrell chest).
- Increased total vital capacity, hypoxia, cyanosis and respiratory acidosis.



3] Types of emphysema:

(a) Centrilobular emphysema (Centralacinar Emphysema):
Dilatation of the respiratory bronchioles

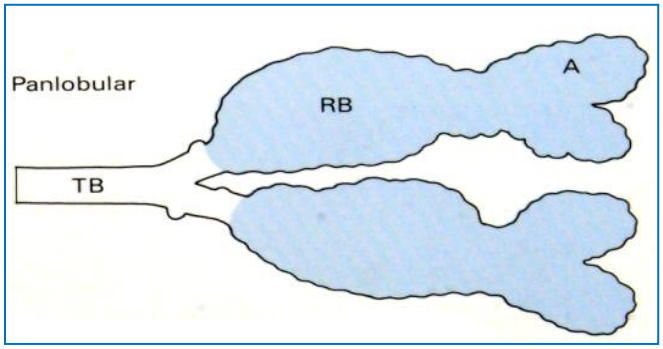
- most often localized to the upper part of the pulmonary lobes.



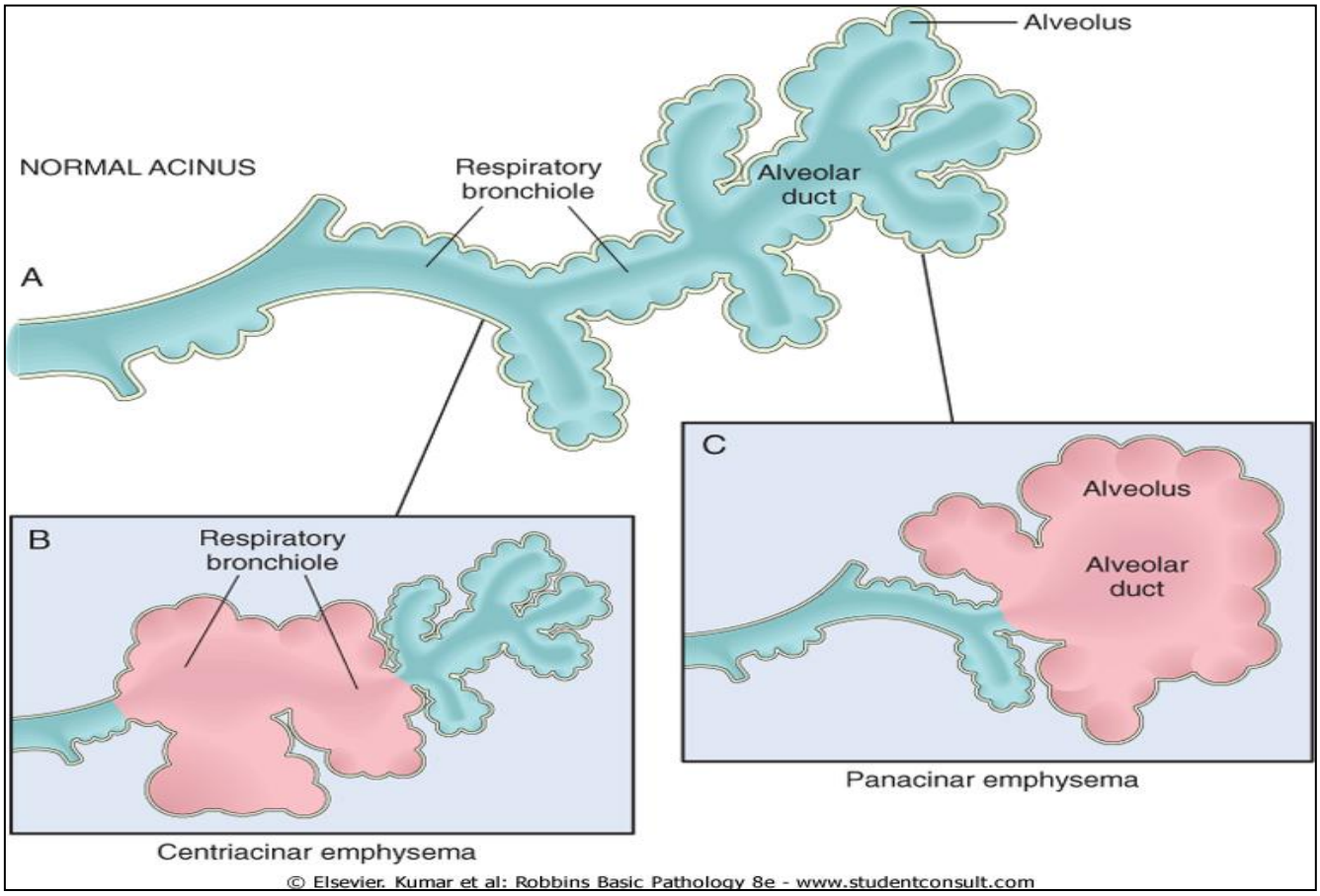
(b) Panacinar emphysema:

(1) Dilatation of the entire respiratory acinus, including the alveoli, alveolar ducts, respiratory bronchioles and terminal bronchioles.

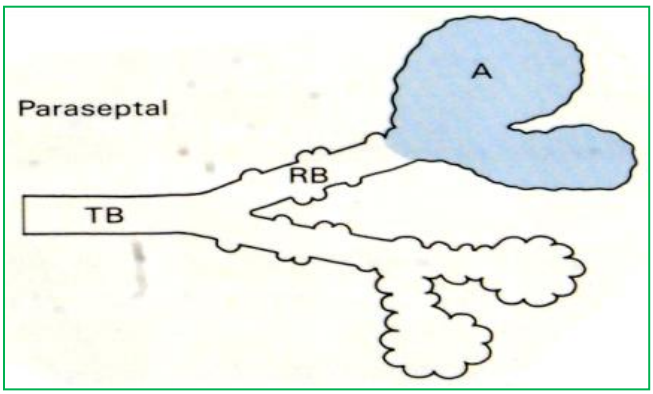
- The disease is most often distributed uniformly throughout the lung.



(2) It is associated with loss of elasticity and sometimes, genetically determined deficiency of *alpha l-antitrypsin* (alpha 1 - protease inhibitor).



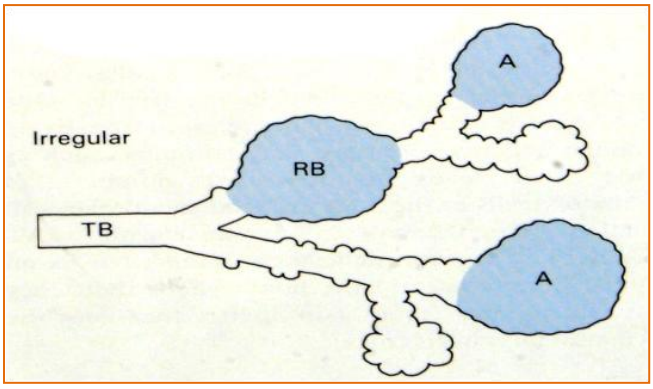
(c) Paraseptal emphysema:



1- Dilatation involves mainly the distal part of the acinus.
   - including the alveoli
     and to a lesser extent, the alveolar ducts.
   - It tends to localize
     subjacent to the pleura and interlobar septa.

2- It is associated occasionally with
   large subpleural bullae or blebs.

(d) Irregular emphysema:



✓ Is the most common subtype and *characterized by*
irregular involvement of the acinus
with scarring within the walls of enlarged air spaces.

✓ This type is usually a complication of
various inflammatory processes
including chronic pulmonary TB.

4] Complications:

A. Emphysema is often complicated by or coexistent with ***chronic bronchitis.***

B. ***Interstitial emphysema***
in which air spaces may enter into the interstitial tissues of the chest from a tear in the airways
may sometimes occur.

C. ***Other complications*** of emphysema may include rupture of a surface bleb
(markedly dilated and emphysematous alveolus) with resultant pneumothorax.

5] Postulated causes:

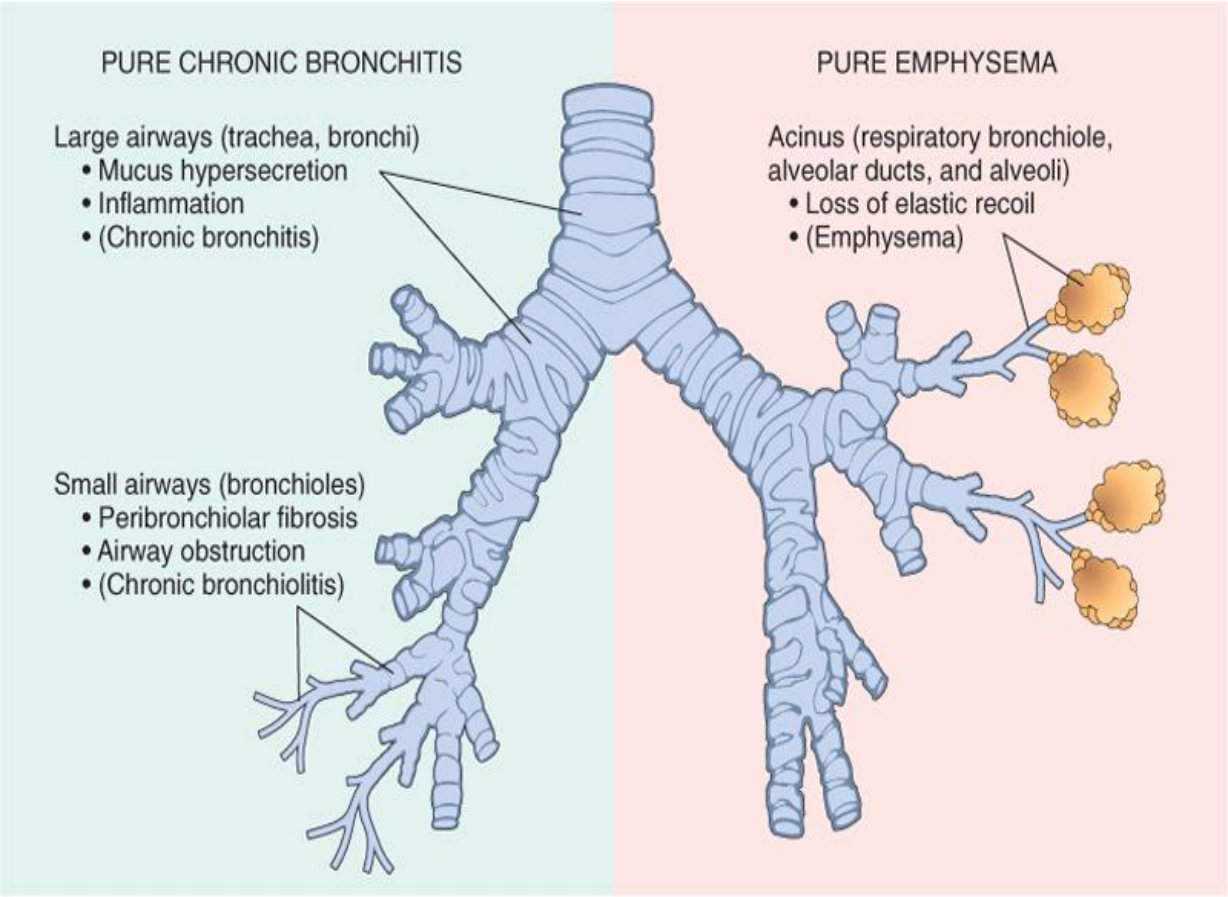
✓ Emphysema may result from.action of proteolytic enzymes such as elastasc on the alveolar wall.

✓ Elastase can induce destruction of elastin unless neutralized by the antiproteinase antielastase activities of alpha 1-antitrypsin which can be deficient in cases of emphysema.

(a) Cigarette smoking: attracts neutrophils and macrophages, which are sources of elastase (anenzyme which destroys elastic fibers from thewall of alveoli).

(b) Hereditary alpha 1 antitrypsin deficiency: accounts for a small subgroup of cases of panacinar emphysema.

✓ It is caused by variants in the *pi* (proteinase inhibitor)gene, localized to chromosome 14.



Emphysema and Chronic Bronchitis

	Predominant Bronchitis	Predominant Emphysema
Appearance	"Blue bloaters"	"Pink Puffers"
Age	40-45	50-75
Dyspnea	Mild, late	Severe, early
Cough	Early, copious sputum	Late, scanty sputum
Infection	Common	Occasional
Respiratory Insufficiency	Repeated	Terminal
Cor pulmonale	Common	Rare, terminal
Airway resistance	Increased	Normal or slightly increased
Elastic recoil	Normal	Low
Chest radiography	Prominent vessels, large heart	Hyperinflation, small heart

Bronchiectasis

1] Definition:

✓ This condition is *characterized by*
permanent and abnormal bronchial dilatation which is *caused by*
chronic infection with inflammation and necrosis of the bronchial wall.

2] Predisposing factors:

- Include ***bronchial obstruction,*** most often by tumor.
- Other predisposing factors include ***chronic sinusitis*** accompanied by postnasal drip.
- The disease is rarely a manifestation of **Kartagener syndrome**
(sinusitis, bronchiectasis and situs inversus, sometimes with hearing loss and male infertility)

- caused by a defect in the motility of respiratory, auditory and sperm cilia that is referred to as primary ciliary dyskinesia, an ucommon autosomal recessive syndrome.
- In this condition, there is a structural defect in dynein arms of the cilia which can be seen by electron microscopy.

Impaired ciliary activity predisposes to infection in the sinuses and bronchi and disturbs embryogenesis, sometimes resulting in situs inversus.
Male infertility is an important manifestation of ciliary dyskinesia.

3] Pathological features:

- often involves the lower lobes of both lungs.
- Characteristics include production of copious purulent sputum, hemoptysis and recurrent pulmonary infection that may lead to lung abscesses.