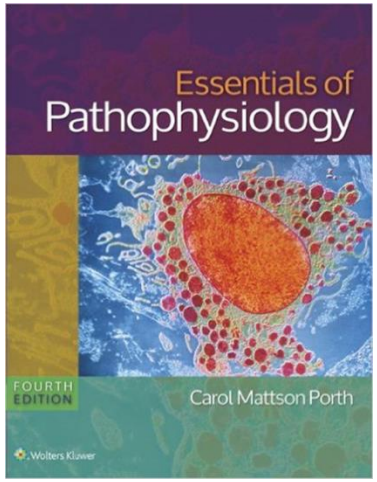
Pathophysiology of the respiratory system



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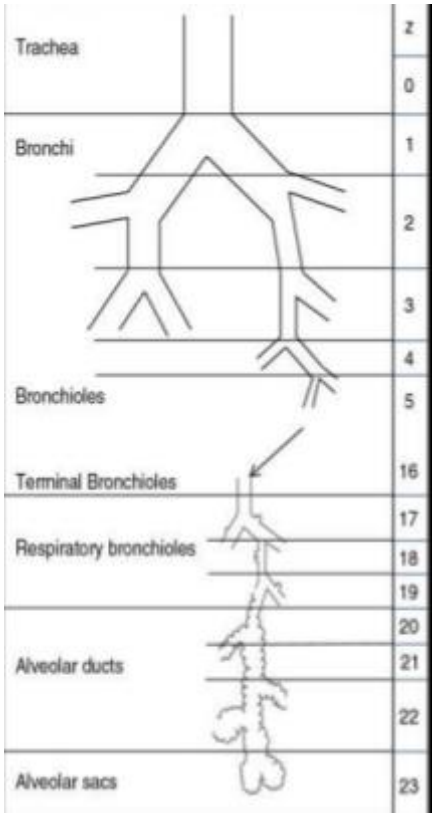
Lecture materials based on text book :
Essentials of pathophysiology 4 th edition by Porth

Conducting Airways

- **The conducting airways consist of: the nasal passages, mouth and pharynx, larynx, trachea, bronchi, and bronchioles.**
- **Functions: conduit for airflow, conditioning the inspired air (warming, filtering, and moistening the inspired air as it moves through these structures).**

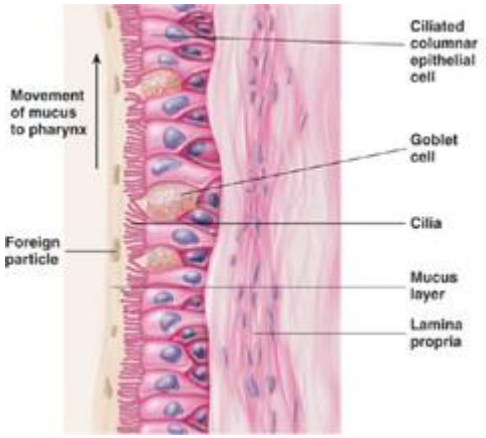


Mucociliary blanket of the airways

** **The mucus produced by the epithelial cells in the conducting airways forms a layer, called the *mucociliary blanket*.**

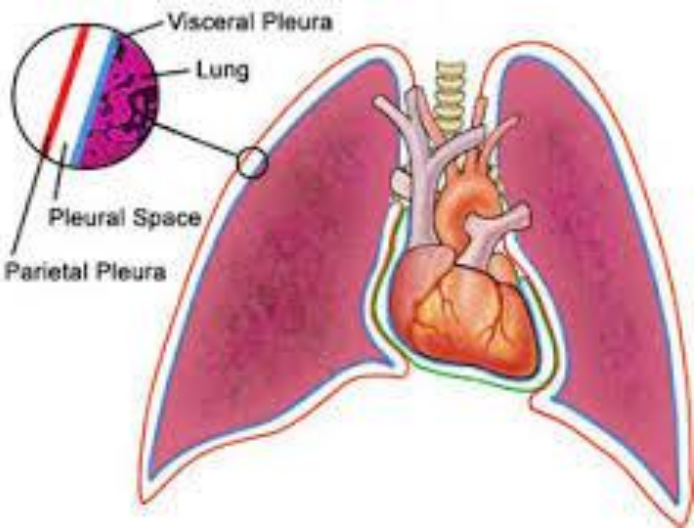
This layer protects the respiratory system by entrapping dust, bacteria, and other foreign particles that enter the airways.

The cilia, which are in constant motion, move the mucociliary blanket with its entrapped particles in an escalator-like fashion toward the oropharynx. At this point, the mucociliary blanket is expectorated or swallowed.



Pleura

- A thin, transparent, double-layered serous membrane ,called the *pleura,* lines the thoracic cavity and encases the lungs.
- The outer parietal layer lines the pulmonary cavities and adheres to the thoracic wall.
- The inner visceral pleura closely covers the lung and is adherent to all its surfaces.
- A thin film of serous fluid separates the two pleural layers, allowing the two layers to glide over each other and yet hold together.
- The pleural cavity is a potential space in which serous fluid or inflammatory exudate can accumulate. The term *pleural effusion* is used to describe an abnormal collection of fluid or exudate in the pleural cavity.

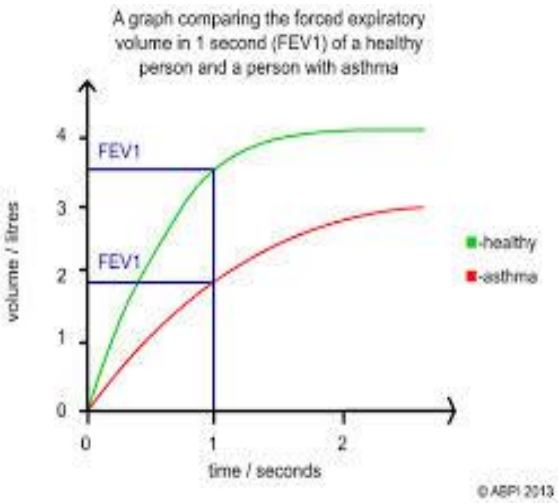


PULMONARY FUNCTION TESTS

****Forced vital capacity (FVC) is the maximum amount of air that can be rapidly and forcefully exhaled from the lungs after full inspiration.**

****Forced expiratory volume achieved in 1 second (FEV1.0) is the volume of air expired in the first second of FVC.**

****Percentage of FVC (FEV1.0/FVC%) is the volume of air expired in the first second, expressed as a percentage of FVC.**



The Common Cold

- The common cold is a viral infection of the upper respiratory tract.
- *Etiology :*
- The **rhinoviruses** are the most common cause of colds. Other viral causes include parainfluenza viruses, respiratory syncytial virus (RSV), human metapneumovirus (hMPV), coronaviruses, and adenoviruses.

Spreading of common cold virus

contaminated hands

- The “cold viruses” are spread rapidly from person to person. Children are the major reservoir of cold viruses. The fingers are the greatest source of spread, and the nasal mucosa and conjunctival surface of the eyes are the most common portals for entry of the virus.
- The most highly contagious period is during the first 3 days after the onset of symptoms, and the incubation period is approximately 5 days.

- Aerosol spread of colds through coughing and sneezing is much less important than the spread through direct mucous membrane contact by fingers picking up the virus from contaminated surfaces and carrying it to the nasal membranes and eyes.

Clinical Manifestations

** It begins with a sore and scratchy throat. This is followed by excessive production of nasal secretions and tearing of the eyes.
There are nasal congestion, sneezing and coughing.

**The affected person may experience headache, fatigue, hoarseness, myalgia and generalized malaise.

*In severe cases, there may be chills, fever, and exhaustion.

*The disease process is usually self-limited and lasts up to 10 days.

Influenza

** Influenza is one of the most important causes of acute upper respiratory tract infection in humans. Rates of infection are highest among children and older adults, but rates of serious illness and death are highest among people 65 years of age or older.

- The viruses that cause influenza belong to the orthomyxoviridae (RNA virus).

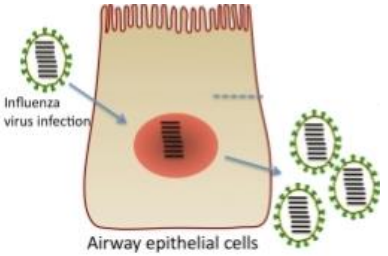
- Influenza is highly contagious.
- Transmission occurs by **inhalation of droplet** nuclei rather than touching contaminated objects.

- People become infectious starting 1 day before their symptoms begin and remain infectious through approximately 5-10 days after illness onset.

Pathogenesis

- The influenza viruses can cause three types of infections:

1. an uncomplicated upper respiratory infection **(rhinotracheitis),**
2. viral pneumonia
3. respiratory viral infection followed by a bacterial infection.



**Influenza initially causes upper airway infection. The virus first kills mucus-secreting, ciliated, and other epithelial cells, leaving holes between the underlying basal cells and allowing extracellular fluid to escape. This is the reason for the “runny nose” .

**If the virus spreads to the lower respiratory tract, the infection can cause severe shedding of bronchial cells.

****Also influenza infection promotes bacterial adhesion to epithelial cells.**

- Pneumonia may result from a viral pathogenesis or from a secondary bacterial infection.

Clinical Manifestations

- In the early stages, the symptoms of influenza often are indistinguishable from other viral infections.

***an abrupt onset of fever and chills

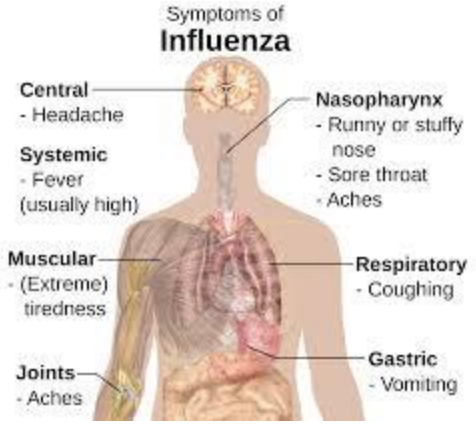
**malaise

**muscle aching

**headache

**profuse, watery nasal discharge

**nonproductive cough, and sore throat.



One distinguishing feature of an influenza viral infection is the **rapid onset of profound malaise**.

*** The symptoms of uncomplicated rhinotracheitis usually peak by days 3 to 5 and disappear by days 7 to 10.

Complications

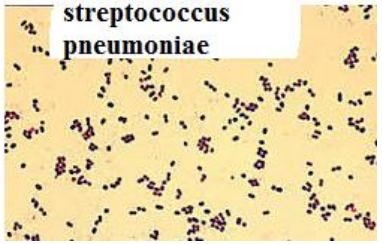
1. Viral pneumonia develops **mostly in older adults or in people with cardiopulmonary disease**. However, it has been reported in healthy people. It typically develops within 1 day after onset of influenza and is characterized by rapid progression of fever, tachypnea, tachycardia, and hypotension.

**The clinical course of influenza pneumonia progresses rapidly. It can cause hypoxemia and death within a few days of onset.

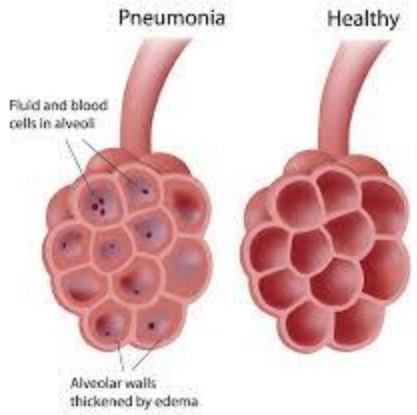
2. Sinusitis, otitis media, bronchitis, and bacterial pneumonia.

3. Secondary bacterial pneumonia characterized by fever, shaking chills, pleuritic chest pain, and productive cough.

- The most common causes of secondary bacterial pneumonia are: *S. pneumoniae* and *H. influenzae*. This form of pneumonia commonly produces less tachypnea and is usually milder than primary influenza pneumonia.



Pneumonia



**Inflammation of parenchymal structures of the lung in the lower respiratory tract, such as the alveoli and the bronchioles.

*** Pneumonia is mostly caused by infectious agents (bacteria, viruses and fungi)

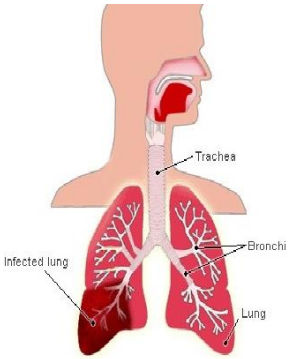
##Few cases of pneumonia are resulted from noninfectious agents such as inhalation of irritating fumes or aspiration of gastric contents. This type of pneumonia can result in severe pneumonia.

- Pneumonia remains an important immediate cause of death among older adults and in people with debilitating diseases.

CLASSIFICATION

1. According to the **setting of the disease**:
   a. community-acquired
   b. hospital-acquired
2. According to the type of **causing agent** :
   a. typical
   b. atypical
3. According to **distribution of the infection**:
   a. lobar pneumonia (affects one lobe)
   b. bronchopneumonia (affects patches throughout both lungs).

Community-Acquired Pneumonia (CAP)



- ** CAP is acquired through inhalation of pulmonary pathogens into the lung.
- *** Pneumonia is due to infections from organisms found in the community rather than in the hospital or nursing home. It is mostly caused by bacteria like, ***S. pneumoniae (commonest)***.

- **Other common pathogens include *H. influenzae, S. aureus,* and gram-negative bacilli.**
- Less common agents are *Mycoplasma pneumoniae* and viruses (influenza virus, respiratory syncytial virus, adenovirus, and parainfluenza virus).

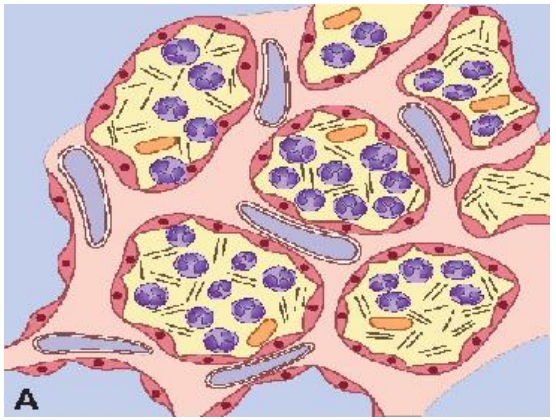
Hospital-Acquired Pneumonia

It is lower respiratory tract infection that was **not present or incubating on admission to the hospital. Usually, infections occurring 48 hours or more after admission

***has a high mortality rate

- ** mostly due to infection by bacteria present in the hospital environment.
- Bacteria causing this type of pneumonia are different from those responsible for community-acquired pneumonias, and many of them have **acquired antibiotic resistance and are more difficult to treat**.

Typical pneumonias (bacterial pneumonia)



*** result from infection by bacteria that multiply extracellularly in the alveoli and cause inflammation and exudation of fluid into the alveoli.

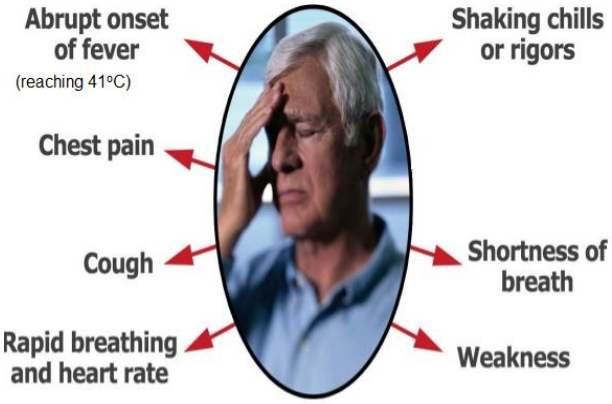
- ***Bacterial pneumonias remain an important cause of mortality among older adults and people with debilitating illnesses.

Pneumococcal Pneumonia as example of bacterial pneumonia

*** *Due to infection by Streptococcus pneumoniae. It is* the most common cause of bacterial pneumonia.

- The pathologic process of pneumococcal pneumonia starts by attachment and colonization of bacteria to the mucus and cells of nasopharynx

Clinical Signs and symptoms of pneumococcal pneumonia



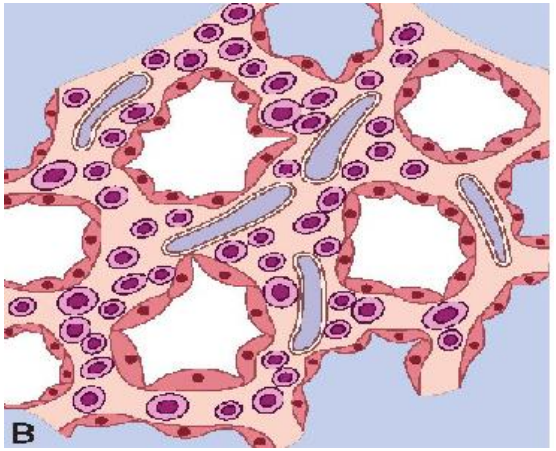
** The onset usually is sudden and is characterized by malaise, severe shaking chills, and fever.

** During the initial or congestive stage, coughing of watery sputum.

** As the disease progresses, the character of the sputum changes; it may be blood tinged or rust colored to purulent.

**Pleuritic pain, a sharp pain that is more severe with respiratory movements.

Atypical pneumonias



- are caused by viral and

mycoplasma infections that involve the alveolar septum and the interstitium of the lung.

- They produce less striking symptoms and physical findings than bacterial pneumonia. For example, there are lack of alveolar infiltration and lobar consolidation on the chest imaging. There are moderate amount of sputum, leukocytosis.

Clinical features of atypical pneumonia

Varies from condition similar to common cold to serious fatal out come

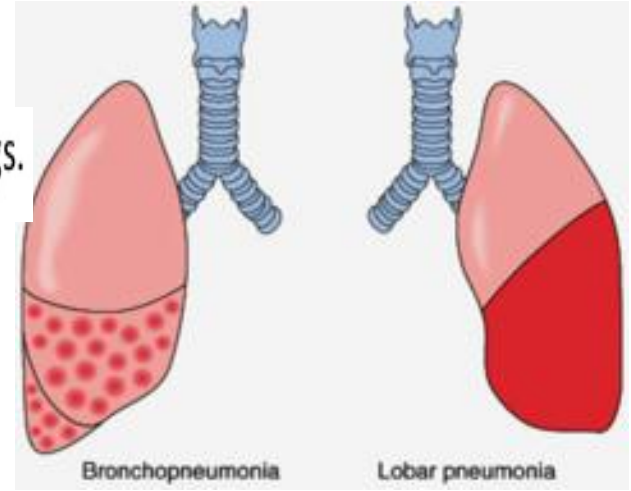
*** fever, headache, and muscle aches and pains.

***Cough, when present, characteristically dry and nonproductive

Lobar pneumonia versus bronchopneumonia

bronchopneumonia

affects patches throughout both lungs.



Lobar pneumonia

Lobar pneumonia : affects a section (lobe) of a lung.

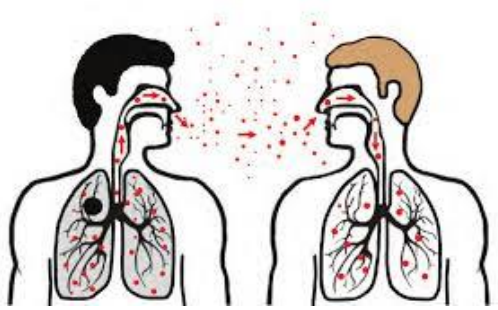
Tuberculosis (TB)

The bacteria is discovered in 1882 by Robert Koch Who received the Nobel Prize in physiology or medicine for this discovery in 1905.

** an infectious disease caused by the mycobacterium, *M. tuberculosis*. which is acid fast bacilli that thrive in an oxygen-rich environment.

**can infect practically any organ of the body. The lungs are most frequently involved.

** infection is transmitted through inhalation of respiratory droplets which are coming from infected persons during coughing, sneezing and talking.

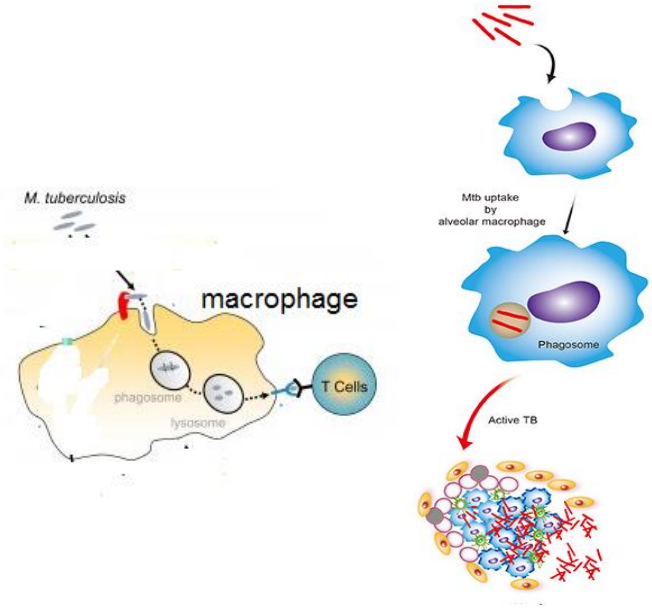


Pathogenesis of tuberculosis

When bacilli enter the lung and reaching the alveoli, they evoked cell mediated immune response.

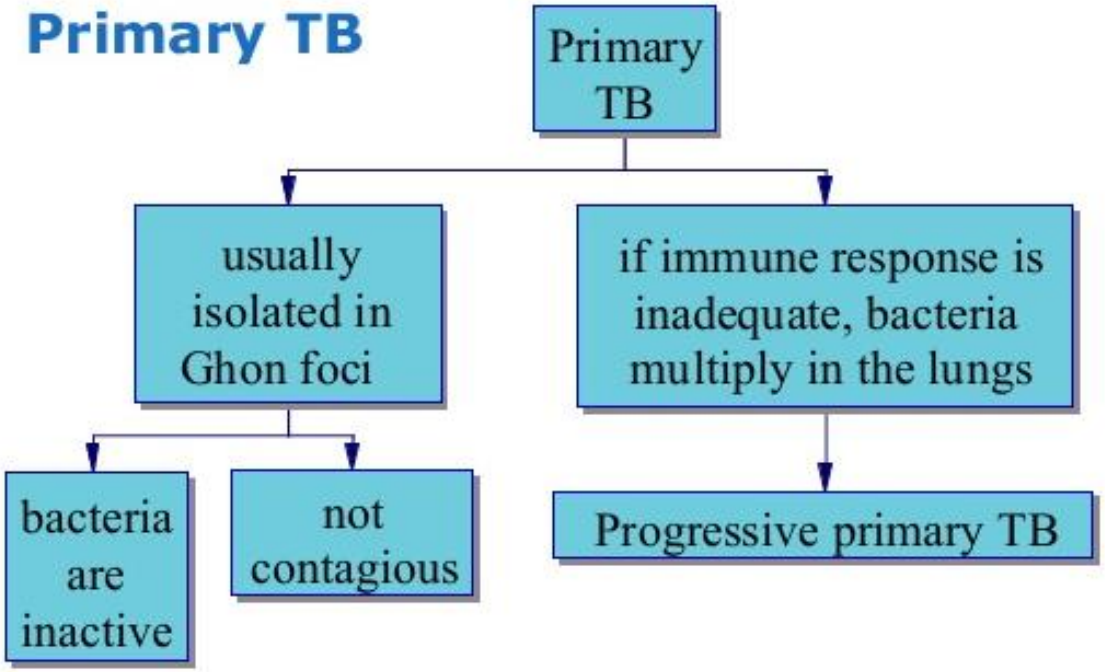
1. bacilli are engulfed by alveolar Macrophages which can not kill the bacilli at the beginging but initiate a cell mediated immune response.

2. The bacilli multiply inside the infected macrophages which eventually degraded.



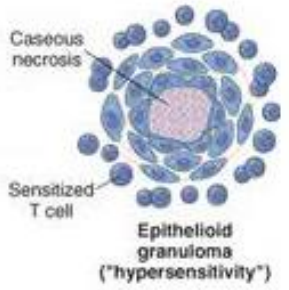
3. The bacilli sensitize the T lymphocytes which stimulate the macrophages to increase their concentration of lytic enzymes and ability to kill the mycobacteria and at the same time damage the lung tissues leading to formation of Ghon focus.

Primary TB


Primary TB
usually isolated in Ghon foci
if immune response is inadequate, bacteria multiply in the lungs
bacteria are inactive
not contagious
Progressive primary TB

Pathogenesis(cont.)

- Ghon focus contains the tubercle bacilli, modified macrophages, and other immune cells. The focus is necrotized in the center (caseation).

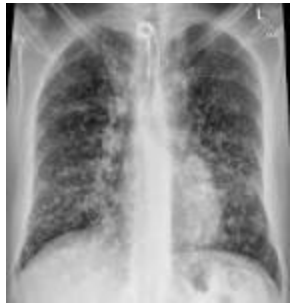


- Some bacilli reaching the lymph nodes causing caseation there also. The combination of the primary lung tissue with lymph nodes lesion is called Ghon complex which in healthy person with good immunity will heal and calcified.

Pathogenesis(cont.)

- In 95% of infected person with good immunity, the end result is calcified focus which contains few bacilli.
- In 5 % of newly infected people of poor immunity develop progressive primary tuberculosis with continued destruction of lung tissue and spread to multiple sites within the lung.

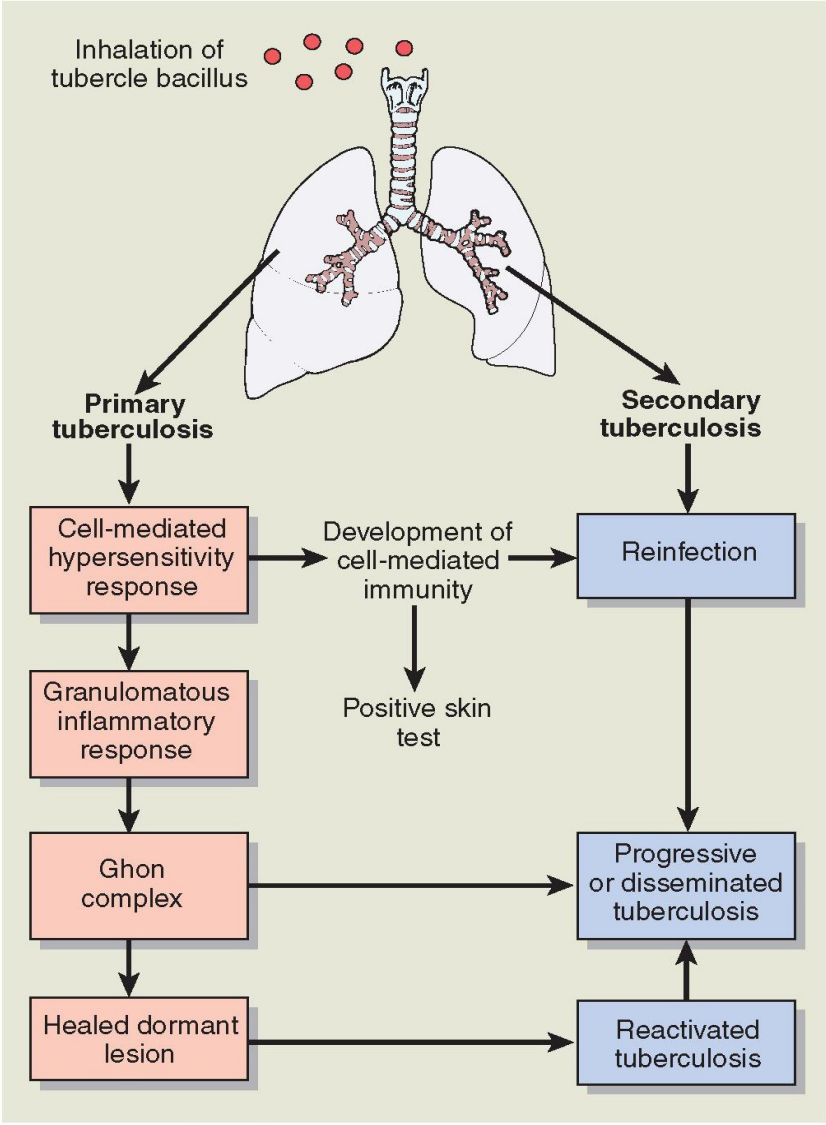
***In rare cases the bacilli spread through blood to different organs of the body (like brain, meninges, liver, kidney, and bone marrow) causing *Miliary tuberculosis.*

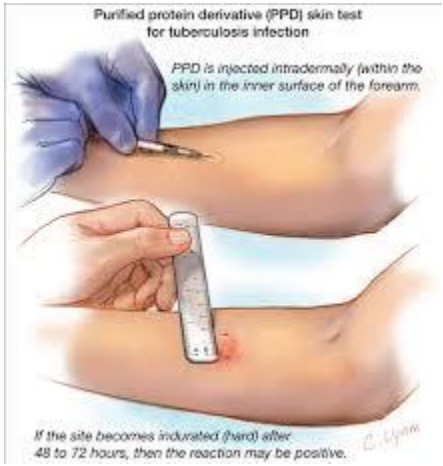


Pathogenesis of tuberculosis infection

Initial TB Infection

- Macrophages begin a cell-mediated immune response
- Results in a granulomatous lesion or **Ghon focus** containing
  - Macrophages
  - T cells
  - Inactive TB bacteria





The tuberculosis skin test is also known as the tuberculin test

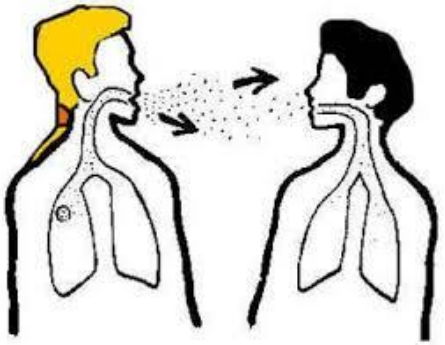
Clinical Manifestations

- the symptoms are usually insidious and nonspecific, with **fever, weight loss, fatigue, and night sweats**. Sometimes the onset of symptoms is abrupt, with high fever, pleuritis, and lymphadenitis.

chest pain because of pleuritis

lymphadenitis

- As the disease spreads, the organism gains access to the sputum, allowing the person to infect others.



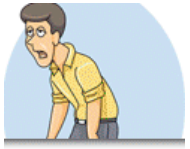
Secondary Tuberculosis

** is resulted from either reinfection from inhaled droplet or reactivation of a previously healed primary lesion (when immunity is impaired).


Coughing


Cough with blood


Weight loss


Tiredness


Night sweats

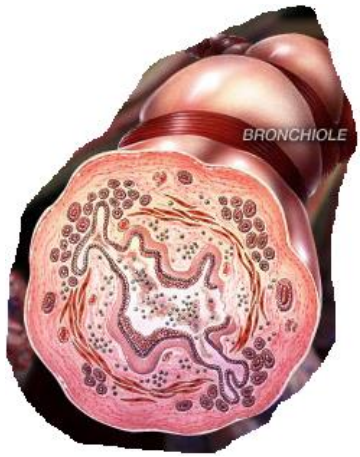

Fever

Signs and symptoms: low-grade fevers, fatigue, and weight loss. A cough initially is dry but later becomes productive with purulent and sometimes blood-tinged sputum.

Dyspnea and orthopnea develop as the disease advances. Also night sweats, anemia occur.

Asthma and chronic obstructive pulmonary disease

Physiology of airways:



Factors affect the movement of air through airways

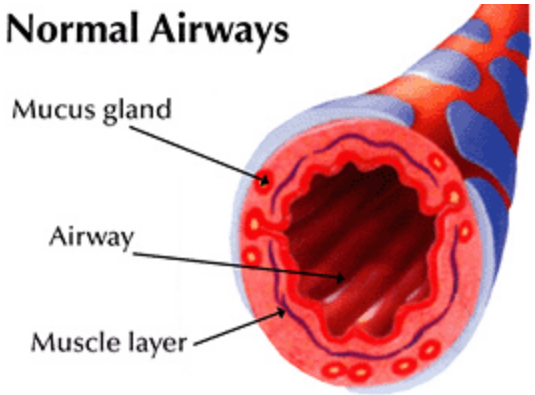
- 1. Parasympathetic stimulation, through

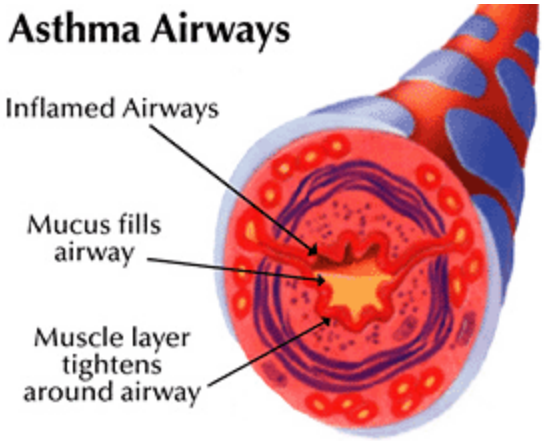
the vagus nerve and cholinergic receptors, produces bronchial constriction, whereas sympathetic stimulation, through β2-adrenergic receptors, increases bronchial dilation.

- 2. Histamine, that act directly on bronchial smooth muscle cells to produce constriction.
- 3. decreased the diameter of the airway by accumulation of mucus inside them

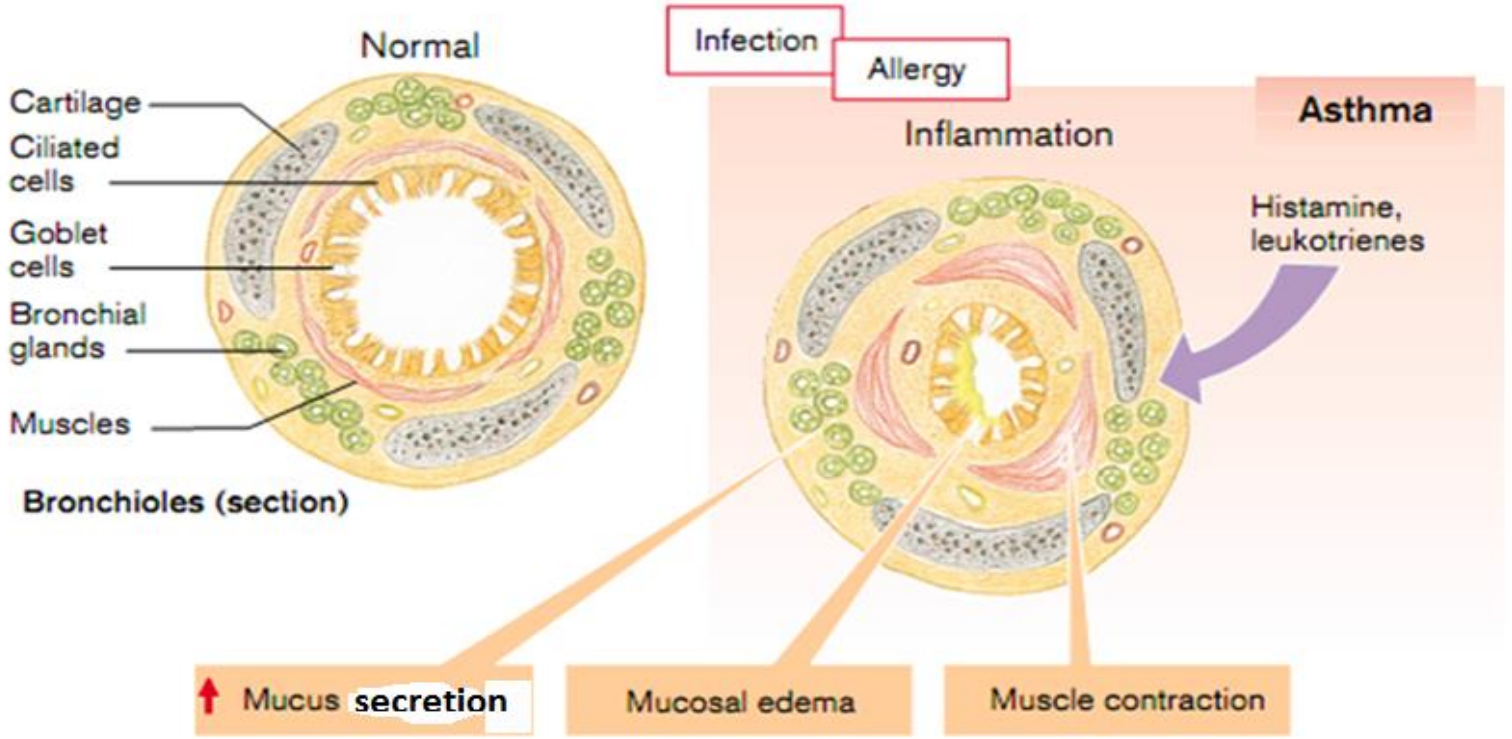
Asthma

** is chronic inflammatory disorder of the airways characterized by **episodic of airway obstruction**, bronchial hyperresponsiveness, airway inflammation, and, in some people, airway remodeling.





Normal airway versus asthmatic airway



Types and pathogenesis of asthma

*** **Two types of asthma:**

1. **Extrinsic or allergic asthma or atopic asthma: due to hypersensitivity reaction to external allergen.**

2. Non-atopic asthma: occurs with out involvement of external allergens.

**** Both types of asthma leads to BRONCHOCONSTRICTION.**

**** Bronchoconstriction can be triggered by many factors like:**

- **Respiratory tract infection**
- **Exercise**
- **Ingestion of aspirin**
- **Emotional upset**
- **Exposure to respiratory irritants**

Etiology and Pathogenesis

***The Airways of asthmatic people show:

1. Exaggerated hyperresponsiveness to a variety of stimuli.
2. Presence of inflammatory cells (like eosinophils, lymphocytes, and mast cells).
3. Damage to the bronchial epithelium.

pathogenesis of asthma (cont.)

** When causing agents enter the airways, they cause activation of macrophages, mast cells, eosinophils and basophils.

These cells release inflammatory mediators which cause:
1. Bronchoconstriction
2. Increase mucus production
3. Increased vascular permeability

** In some patients there are:
a. Injury to epithelial cells
b. Smooth cells hypertrophy
c. Blood vessels proliferation

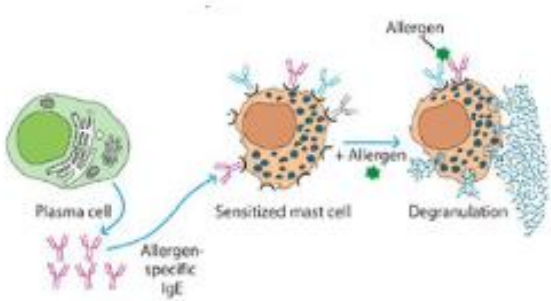
Asthma and allergy

** In response to allergens, T lymphocytes (T_H2, helper T lymphocyte type 2) stimulate B cells and these B

Converted to plasma cells that

Can produce

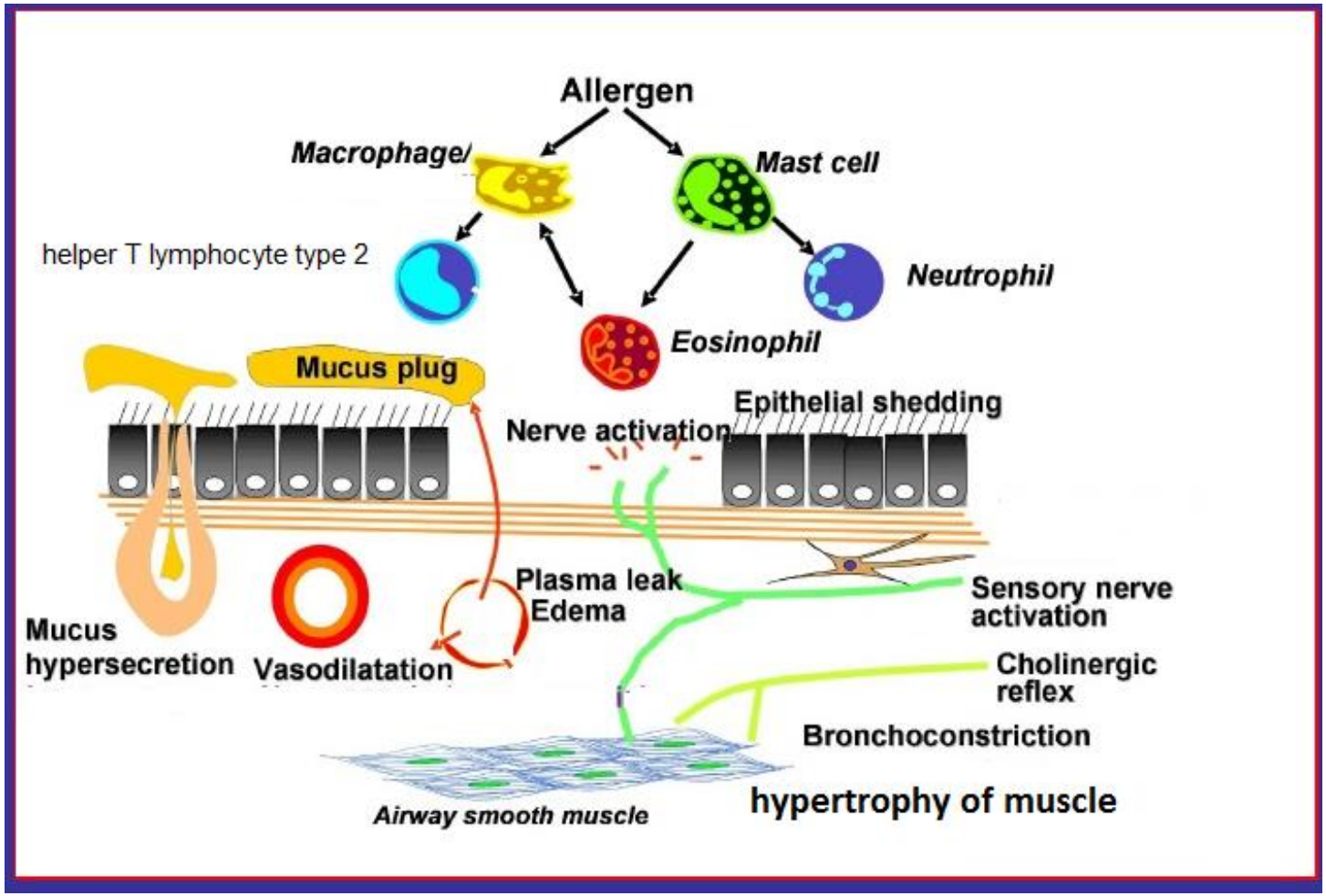
Immunoglobuline E (IgE).



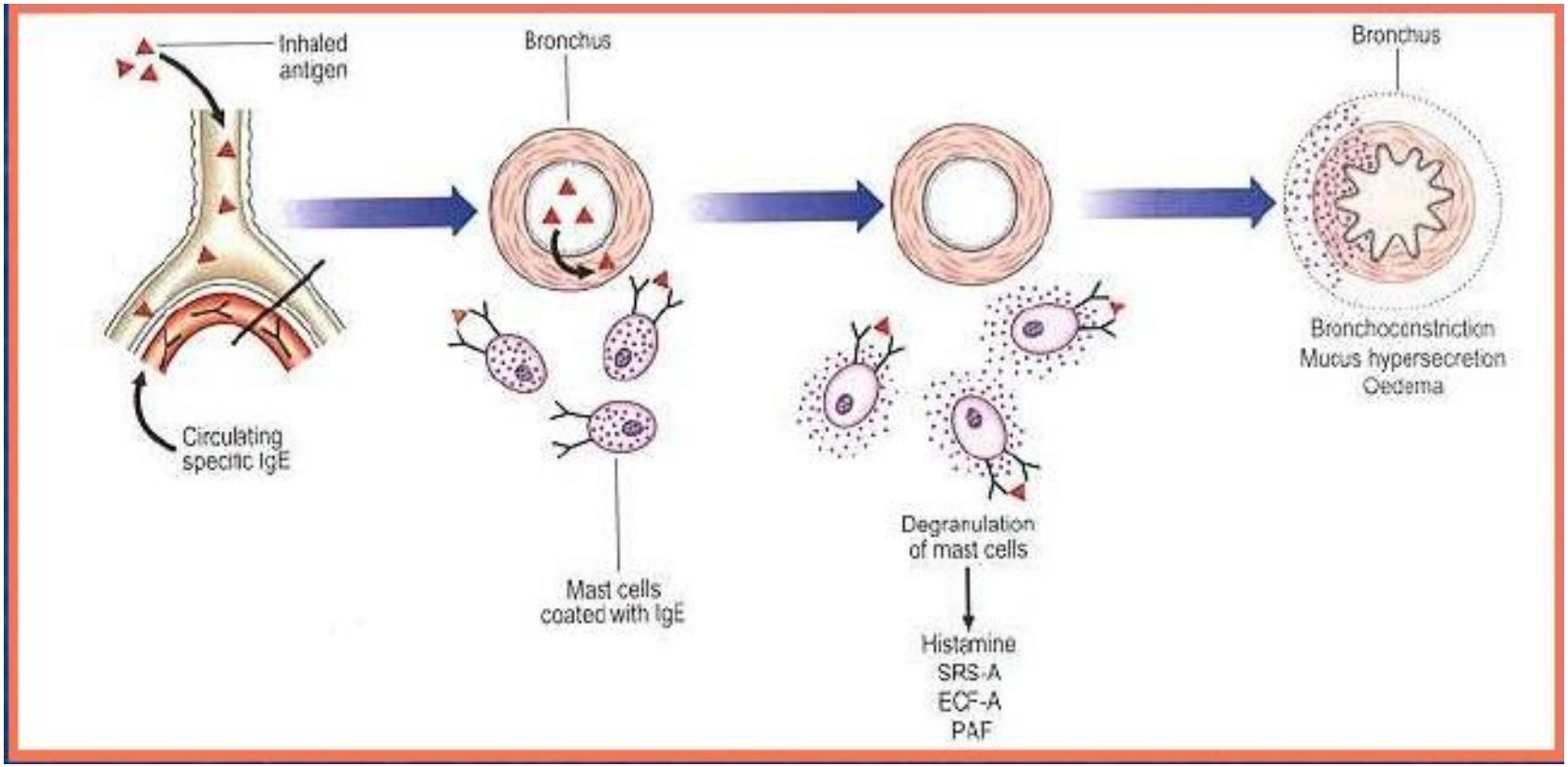
** the IgE bind to mast cells

Making them to release histamine, leukotrienes, interleukins and prostaglandins.

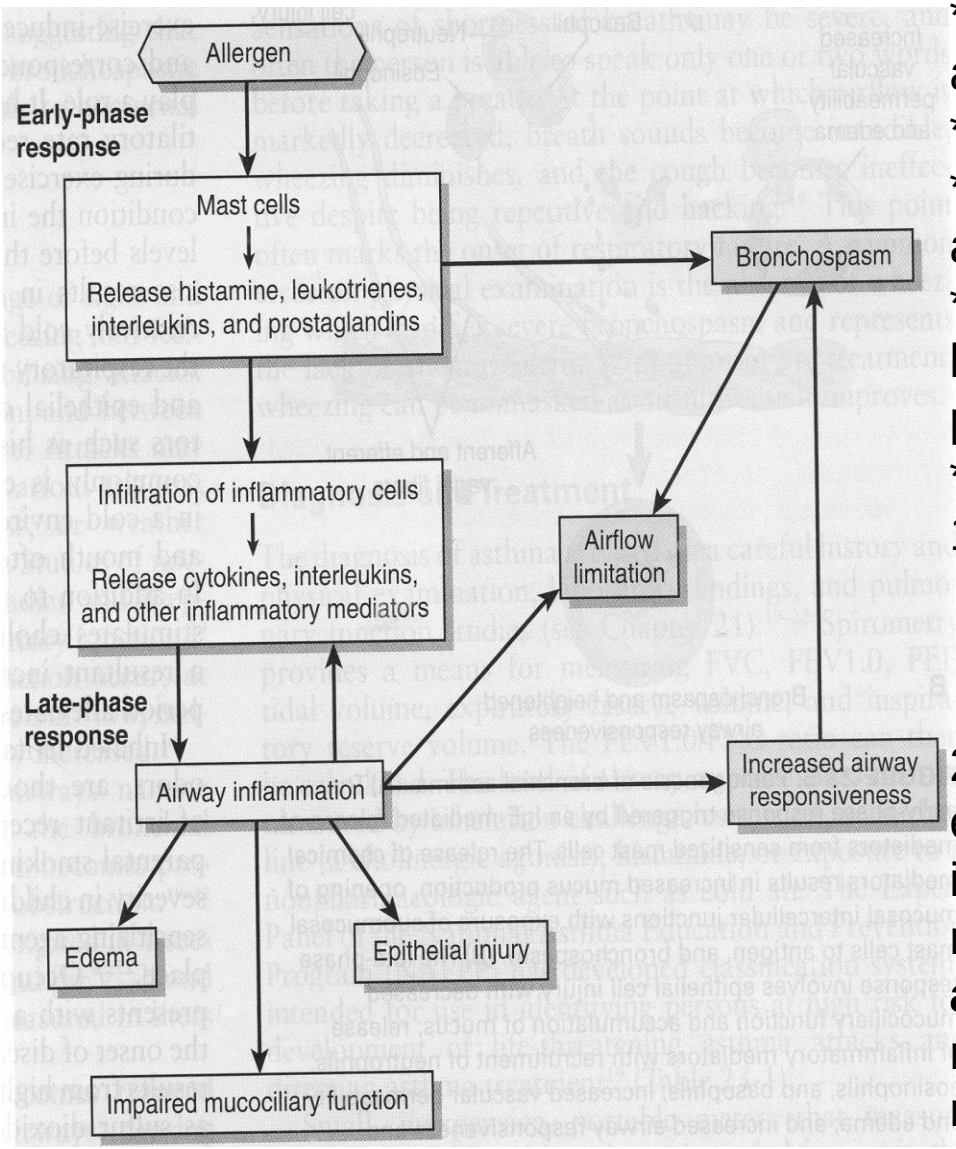
Changes in airway in Asthma



Atopic asthma



Atopic asthma



**Induced by exposure to extrinsic antigen (allergen)

** starts in childhood

** seen in persons with family history of atopic allergy

** other allergic disorders like eczema, hay fever and urticaria are seen in persons with this type of asthma.

** It has two responses:

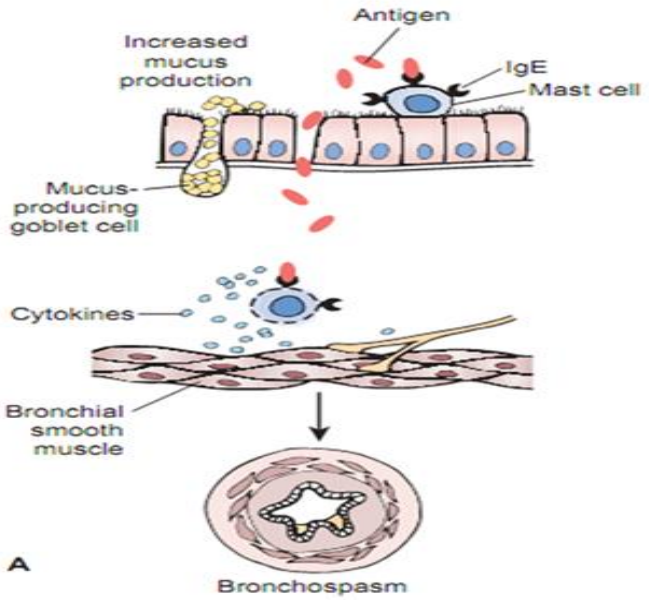
1. Early (acute) phase response (10-20 minutes after exposure to allergens). It is due to release of mediators from IgG coated mast cells.

2. Late phase response (4-8 hours after exposure to allergen). Due to release of inflammatory mediators from mast cells, macrophages and epithelial cells**. Also activated basophils, eosinophils and neutrophils play role in this response.**

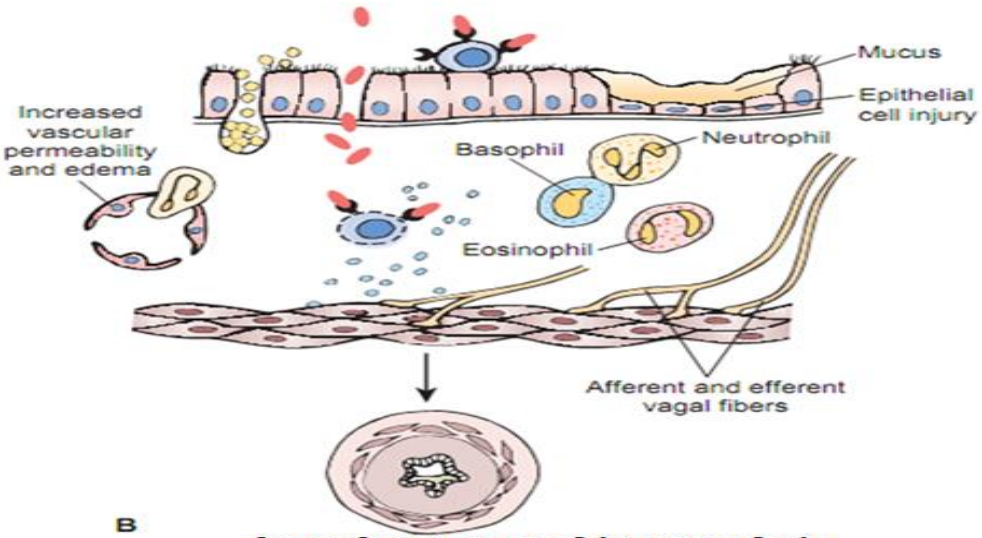
Two responses to allergens causing asthma:

****In early response: as result of mediators release there are bronchospasm, increased mucus production and vascular permeability.**

****In late response: there are increased in airway responsiveness and it is due to release mediators from activated basophils, eosinophils and neutrophils.**



early response



bronchospasm and increased air way responsiveness (late response)

Note: Repeated attacks of asthma lead to remodeling the airways and decrease their responsiveness to bronchodilators (obstruction become partially reversible).

Clinical Manifestations of asthma

**Asthma attacks may occur:

- either spontaneously
- or in response to various triggers like respiratory infections, emotional stress, or weather changes.

- Asthma is often worse at night, referred to as *nocturnal asthma*. This may be related to variation in hormones and respiratory function.
- The greatest decrease in respiratory function occurs at about 4:00 am, at this time cortisol levels are low and eosinophil activity increased.

Clinical Manifestations of asthma
signs and symptoms

***The attacks of asthma differ from person to person, and between attacks in the same person.

***During the attacks, the airways narrow due to one or more of these factors:

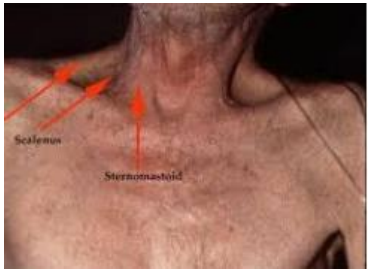
a. airways smooth muscle contraction (bronchoconstriction)

b. edema of the bronchial mucosa

c. mucus plugging

- *# during* mild attack, there is feeling of chest tightness, a slight increase in respiratory rate with prolonged expiration, and mild wheezing. A cough may accompany the wheezing.

signs and symptoms of severe asthma attack

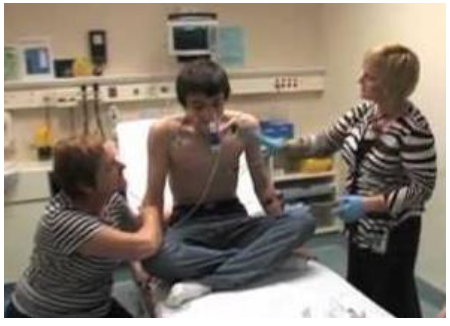


** In more severe attacks, there is loud wheezing and patients use their respiratory accessory muscles

As the condition progresses, fatigue develops, the skin becomes moist, and anxiety and apprehension are obvious.

Sensations of shortness of breath may be severe, and often the person is able to speak only one or two words before taking a breath.

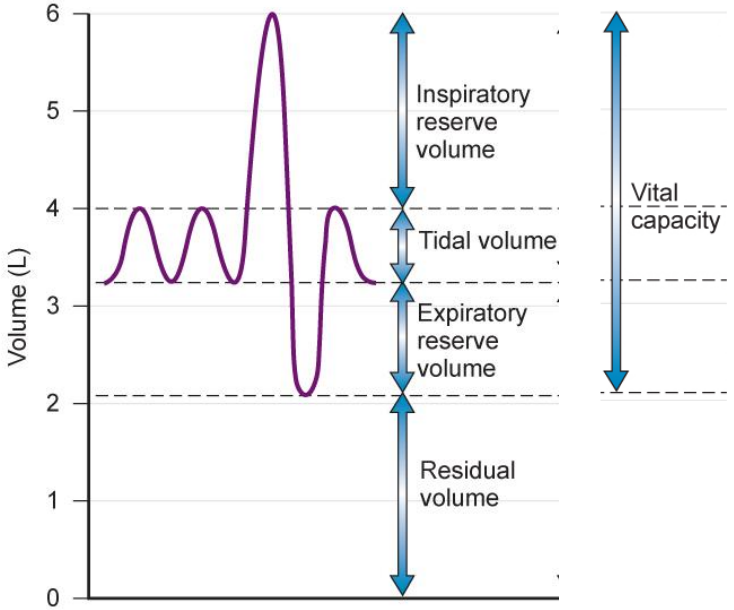
*** When airflow is markedly decreased, breath sounds become inaudible with diminished wheezing, and the cough becomes ineffective. This may mark the onset of respiratory failure.



Pulmonary function test during asthma attacks

*** Forced expiratory volume in 1 second [FEV_1), FEV_1/FVC and the peak expiratory flow (PEF) rate are decreased.

- **The trapped air behind the occluded and narrowed airways, causing hyperinflation of the lungs. This produces an increase in the residual volume (RV) along with a decrease in the inspiratory reserve capacity [IRC) and forced vital capacity (FVC).



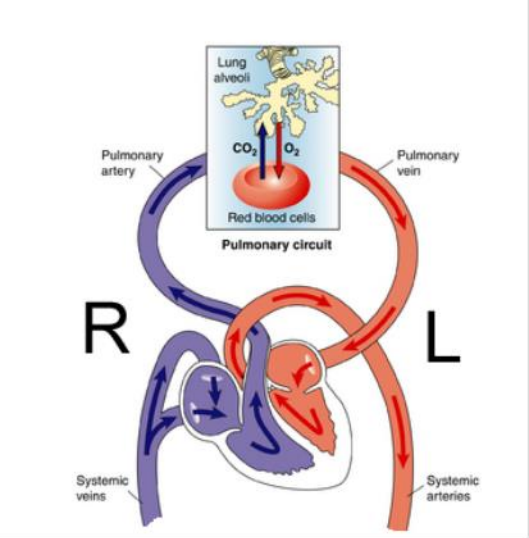
Severe or Refractory Asthma

*** some people with asthma have severe or refractory asthma. These people need high medication requirements to control their asthma or those who continue to have persistent symptoms despite high medication use.
***These people are at increased risk for fatal or near-fatal asthma.

Blood gases in severe asthma

**In severe asthma hypoxemia and hypercapnia develop due to ineffective ventilation of alveoli.

- **hypoxemia causes an increase in pulmonary vascular resistance leading to a rise in pulmonary arterial pressure and increased work demands on the right heart**.



Chronic Obstructive Pulmonary Disease (COPD)

Definition: chronic and recurrent obstruction of airflow in the airways

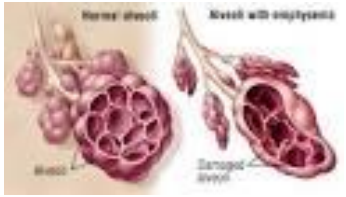
It consists of two types of obstructive airway disease:

1. emphysema: enlargement of airspaces and destruction of lung tissue,
2. chronic obstructive bronchitis: obstruction of small airways, and a chronic productive cough with increased mucus production.

***People with COPD often have overlapping features of both disorders.

Stop smoking (Quit smoking)

Emphysema



***In emphysema there are

1. loss of lung elasticity
2. abnormal enlargement of the airspaces distal to the terminal bronchioles
3. destruction of the alveolar walls and capillary bed

Causes of emphysema

1. smoking, which causes lung injury,
2. deficiency of α1-antitrypsin (antiprotease enzyme). It is inherited condition.

Role of deficiency of α1-antitrypsin in emphysema

*** Normally α1-antitrypsin enzyme protects the lung from injury caused by protease enzymes like elastase (digests elastin fibers) which is produced mainly by neutrophils.

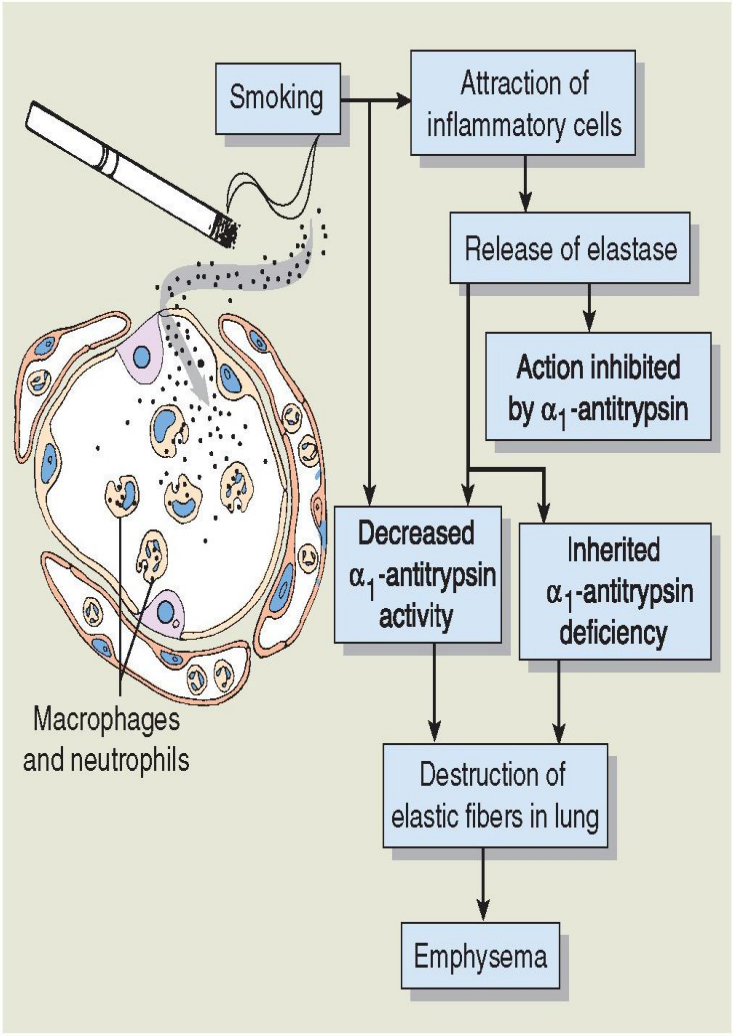
***Normally elastase assists in removing bacteria during acute respiratory inflammation.

** If elastase action is not terminated by α1-antitrypsin, it will cause damages to healthy lung tissue.

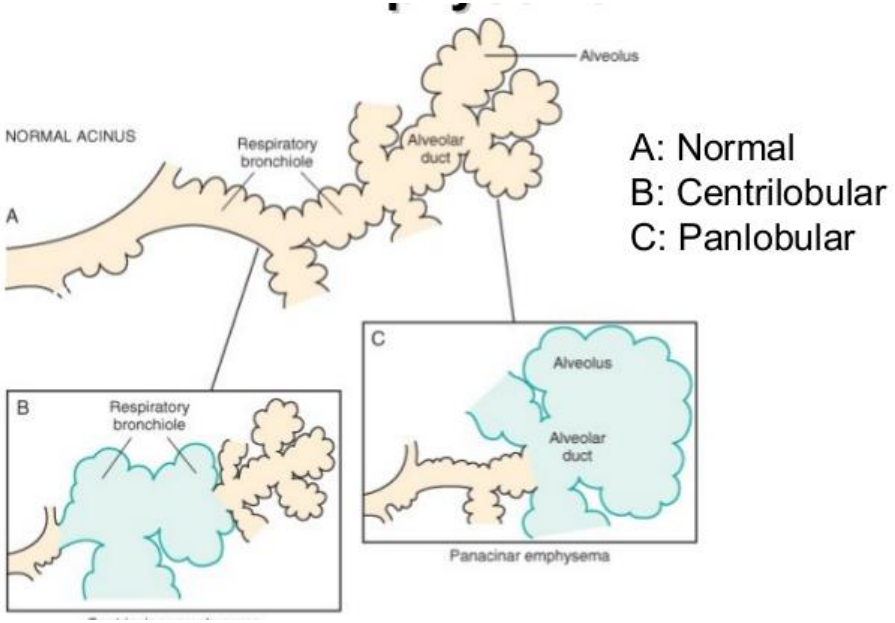
** Ephysema caused by deficiency of α1-antitrypsin is resulted from break down of elastin and other components in alveolar wall.

Role of Cigarette smoke in emphysema

***Cigarette smoke and other irritants stimulate the movement of inflammatory cells like neutrophils into the lungs, resulting in increased release of elastase and other proteases.
*** Cigarette smoke decreases α1-antitrypsin activity and production and this will end in more proteases like elastase which will end in digestion of proteins in alveolar wall resulting in emphysema.



Types of emphysema



1. Centriacinar emphysema: is the most common type of emphysema and is seen predominantly in male smokers. It affects the bronchioles in the central part of the respiratory lobule, with initial preservation of the alveolar ducts and sacs.

2. Panacinar emphysema : is more common in people with α1-antitrypsin deficiency. It initially affects the alveoli and later extends to involve the more central bronchioles

Chronic Bronchitis

***Airway obstruction of the major and small airways.

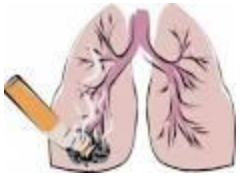
***A clinical diagnosis of chronic bronchitis requires the history of a chronic productive cough for at least 3 consecutive months in at least 2 consecutive years.

***It is seen most commonly in middle-aged men and is associated with chronic irritation from smoking and recurrent infections.

Chronic Bronchitis (cont.)

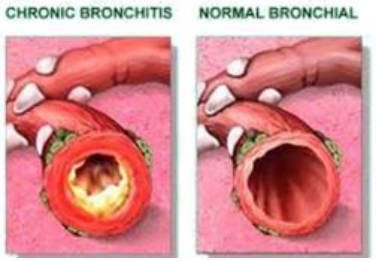
*** At the beginning: due to irritation of airways by cigarette smoke and repeated infection, there is hypersecretion of mucus in the large airways, associated with hypertrophy of the submucosal glands in the trachea and bronchi.

**Mucus hypersecretion (a protective reaction against tobacco smoke and other pollutants) causes airway obstruction.



*** The changes in bronchial tree in chronic bronchitis involve:

1. marked increase in goblet cells and excess mucus production
2. plugging of the airway lumen
3. inflammatory infiltration
4. fibrosis of the bronchiolar wall.



clinical manifestations of COPD

*** Early manifestation include fatigue, exercise intolerance, cough, sputum production, or shortness of breath (dyspnea).

**The productive cough usually occurs in the morning

**Dyspnea becomes more severe as the disease progresses.

**Frequent exacerbations of infection and respiratory insufficiency.

**The late stages of COPD are characterized by recurrent respiratory infections and chronic respiratory failure.

clinical manifestations of COPD (cont.)

** Obstruction of airflow is greater on expiration than inspiration, resulting in increased work of breathing and feeling of dyspnea (in early stages it is felt when patient does exercise like walking but in late stages it occurs at rest).

- ** Breathing becomes less effective in maintaining normal blood gases (arterial PO2 and PCO2) and this leads to Hypoxemia, hypercapnia, and cyanosis.

- **Death usually occurs during an exacerbation of illness associated with infection and respiratory failure.

Effect of severe hypoxemia in COPD

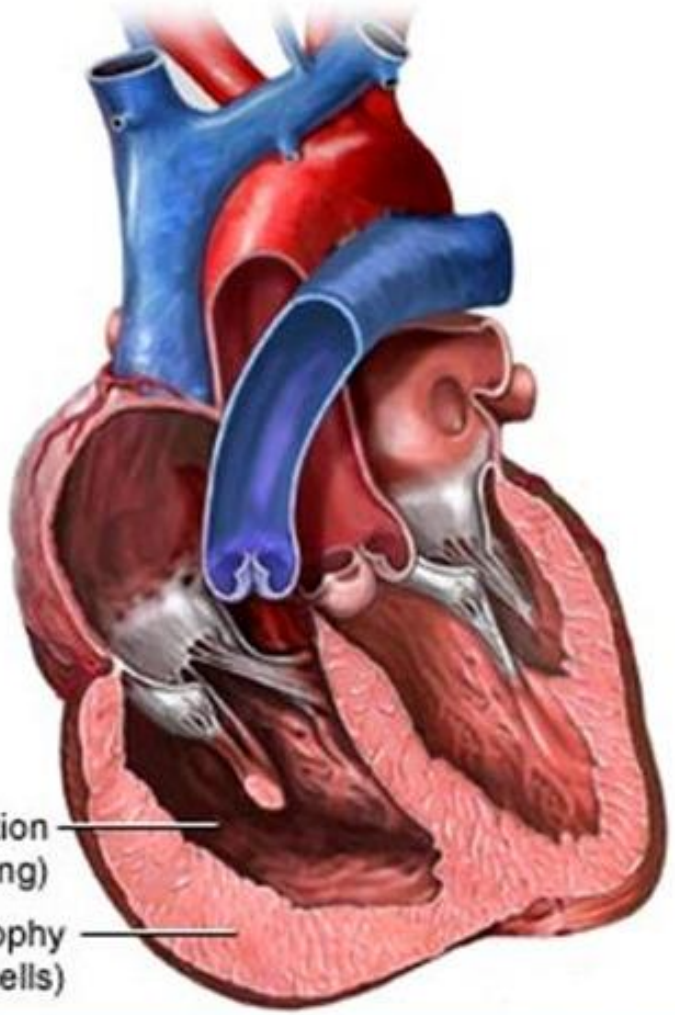
**When arterial PO2 levels fall below 55 mm Hg, it cause severe vasoconstriction of the pulmonary vessels which leads to elevation in pulmonary artery pressure (pulmonary hypertension) and this increases the work of the right ventricle.

*** Because of more work put on right ventricle, **right-sided heart failure** with peripheral edema develop (*i.e.,* cor pulmonale).

**Hypoxemia also stimulates red blood cell production, causing polycythemia.

COPD - Cor Pulmonale

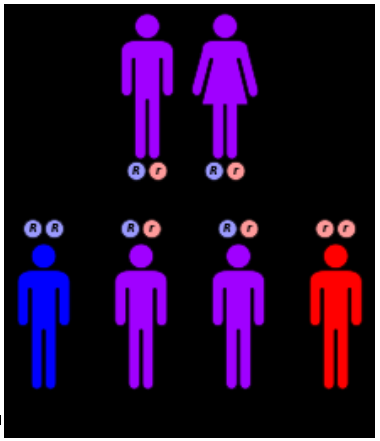
Cor pulmonale, or right-sided heart failure, is an enlargement of the right ventricle due to high blood pressure in the arteries of the lungs usually caused by chronic lung disease



Cystic Fibrosis

**It causes severe chronic respiratory disease in children.

It is inherited disease transmitted as **autosomal recessive disorder (defective gene on is chromosome 7).



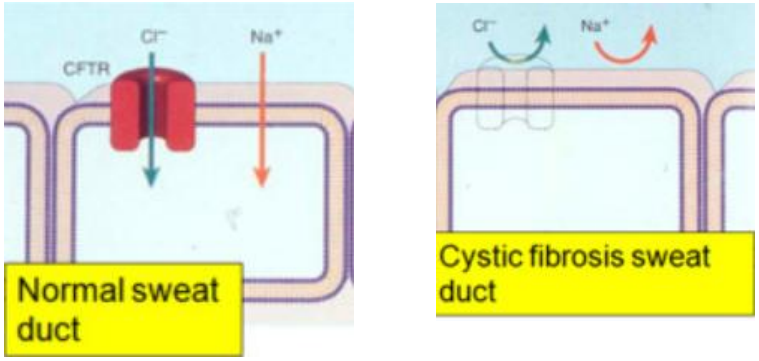
**It involves the exocrine glands in the epithelial lining of the respiratory, gastrointestinal and reproductive tracts.

Etiology and Pathogenesis

- The defective gene makes the epithelial membrane relatively impermeable to the chloride ion.

Lumen of sweat gland

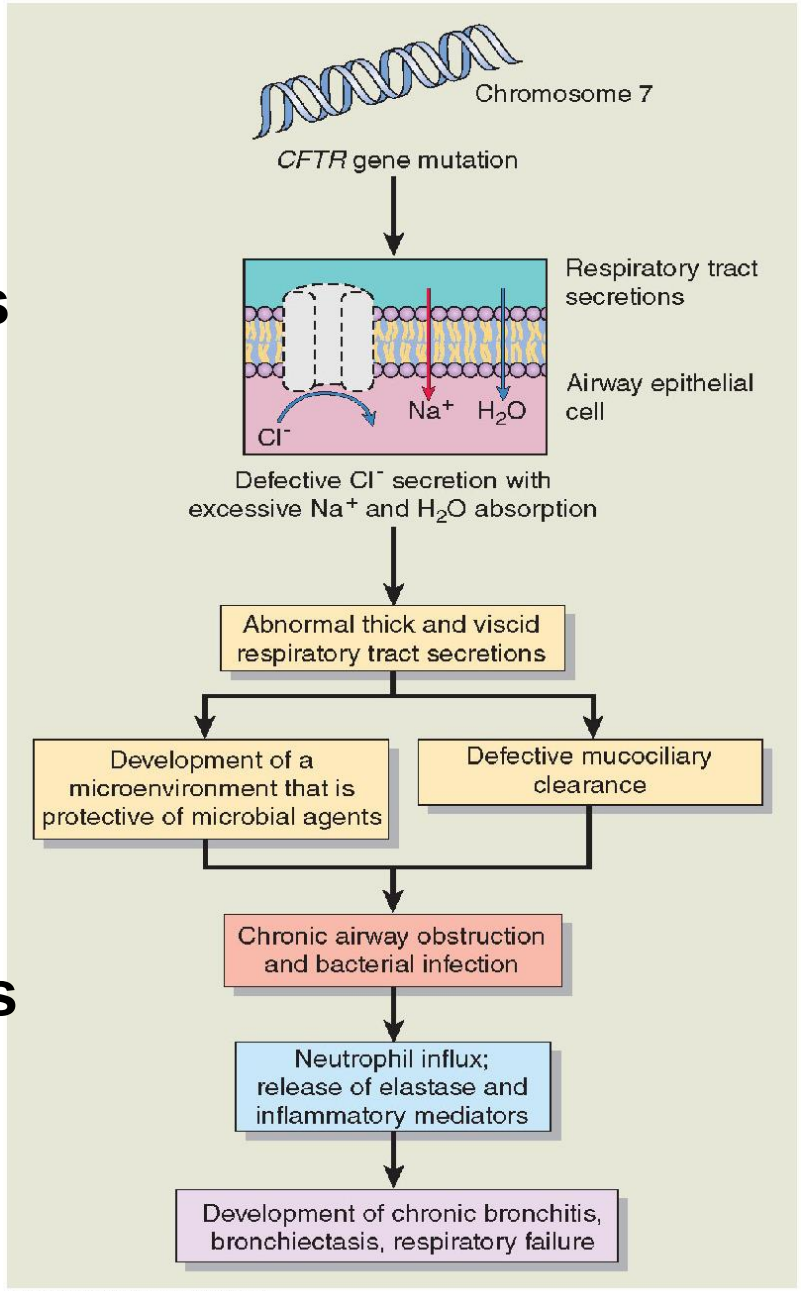
- In sweat gland: because of no reabsorption of Na and Cl ions from the ducts of sweat glands, the concentration of NaCl in sweat is high.



- Also transport abnormalities take place in the pancreatic (causing pancreatitis) and biliary ducts (causing cholelithiasis) and in the vas deferens in boys (causing azoospermia and sterility).

Cystic fibrosis affecting the lungs

In airway epithelium, the Cl ions are not secreted into airways lumen and this leads increased absorption of Na+ and water from the airways lumen. This leads to formation more viscid mucus which makes the mucociliary function ineffective to move the viscid mucus towards the larynx and thus this viscid mucus accumulates and obstructs the airways causing recurrent pulmonary infections.



Clinical Manifestations

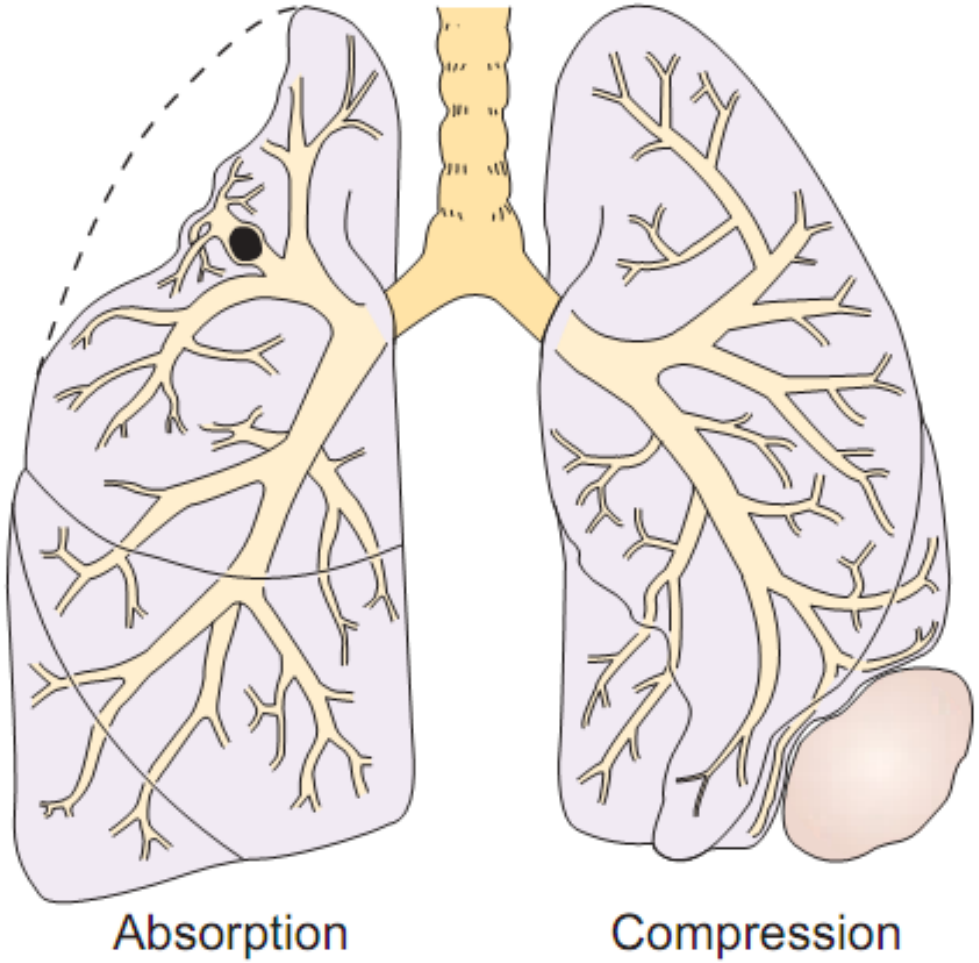
** In the lung (airways) due to accumulation of viscid mucus in the bronchi, impaired mucociliary clearance, and lung infections this will leads to: Chronic bronchiolitis and bronchitis at the beginning and then after many years and due to repeated infection to structural changes in the bronchial wall causing bronchiectasis

Clinical Manifestations (cont.)

**In exocrine pancreas: Because of impaired secretion from pancreas this will leads to steatorrhea, diarrhea, and abdominal pain and discomfort. In the newborn, meconiumn ileus may cause intestinal obstruction.

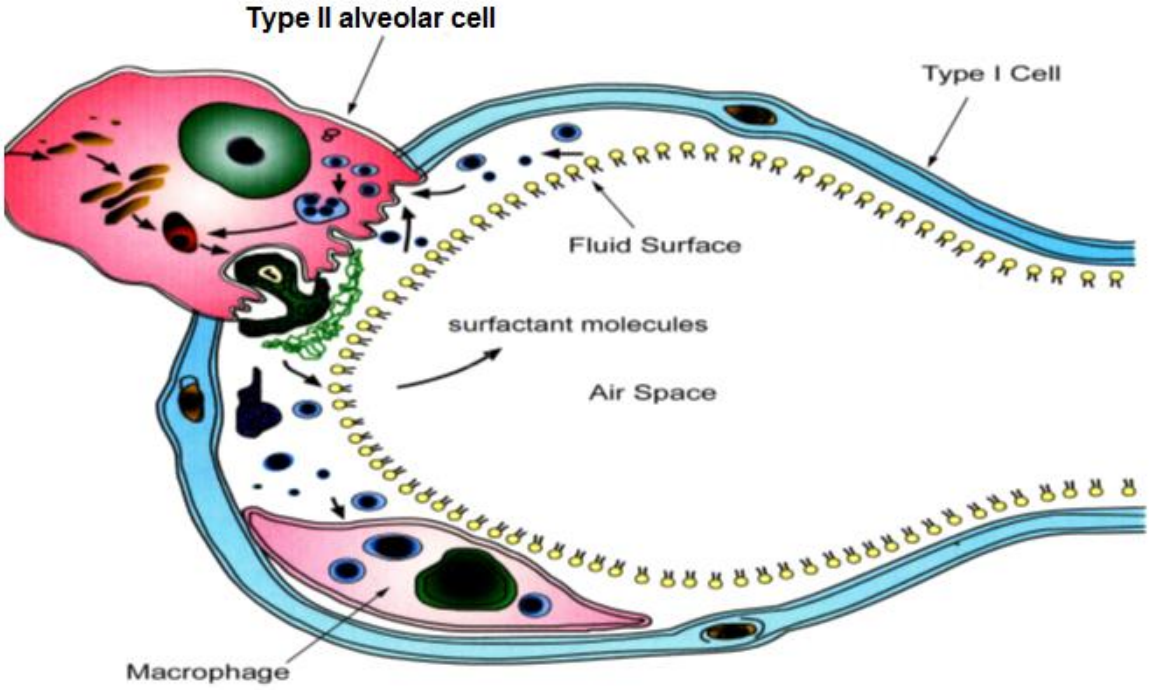
***If endocrine pancreas is involved in disease process, hyperglycemia may occur and many people with cystic fibrosis develop diabetes mellitus.

Atelectasis: means incomplete expansion of the whole lung or part of it.



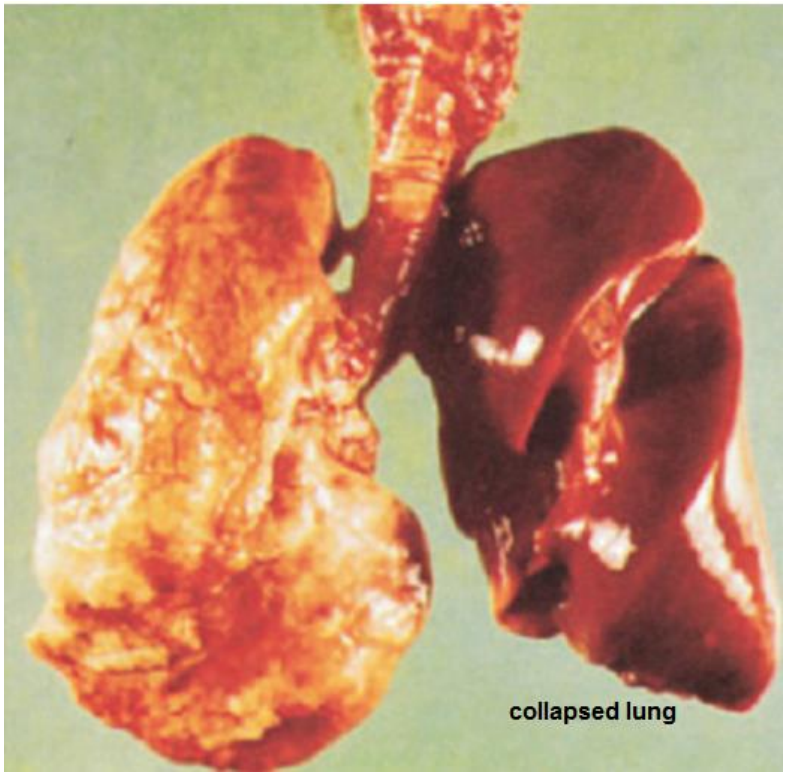
- Causes of atelectasis:
  1. airway obstruction and absorption of air from the involved lung area.
  2. compression of lung tissue
  3. deficiency of lung surfactant

Role of lung surfactant



The surfactant covers the thin layer of fluid lining the alveolar wall. The surfactant decreases the surface tension of the fluid liing the alveolar wall and thus helps in expansion of the alveoli and prevents their collapse.

Primary Atelectasis of newborn



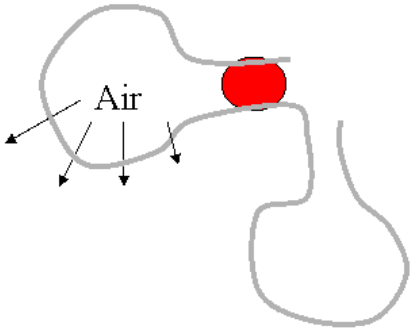
- The right lung of an infant (left side of photo) is pale and expanded by air, whereas the left lung is collapsed.

Acquired atelectasis

**occurs mainly in adults.
** It is caused commonly by:

1. airway obstruction (like mucus plug in the airway)
2. external compression of lung by fluid or air, tumor mass, exudate in the area surrounding the airway.

** When there is complete obstruction of an airway, the air from the dependent alveoli will be absorbed leading to collapse of that portion of the lung.



NOTE: Breathing high concentrations of oxygen increases the rate at which gases are absorbed from the alveoli and predisposes to atelectasis.

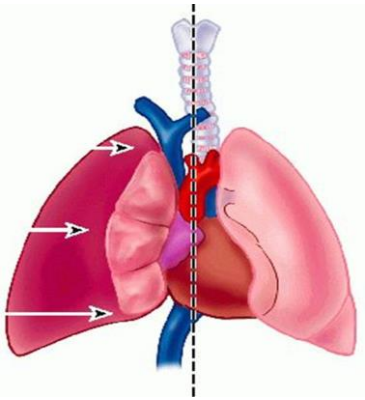
Clinical Manifestations of atelectasis

- ❑ tachypnea
- ❑ tachycardia
- ❑ dyspnea
- ❑ cyanosis
- ❑ signs of hypoxemia
- ❑ Diminished chest expansion
- ❑ decreased breath sounds
- ❑ intercostal retraction (pulling in of the intercostal spaces) over the involved area during inspiration.

proportional to the extent of lung collapse.

Note:
In large atelectasis caused by obstruction , the mediastinum and trachea shift to the affected side.

In compression atelectasis (like tension pneumothorax), the mediastinum shifts away from the affected lung.



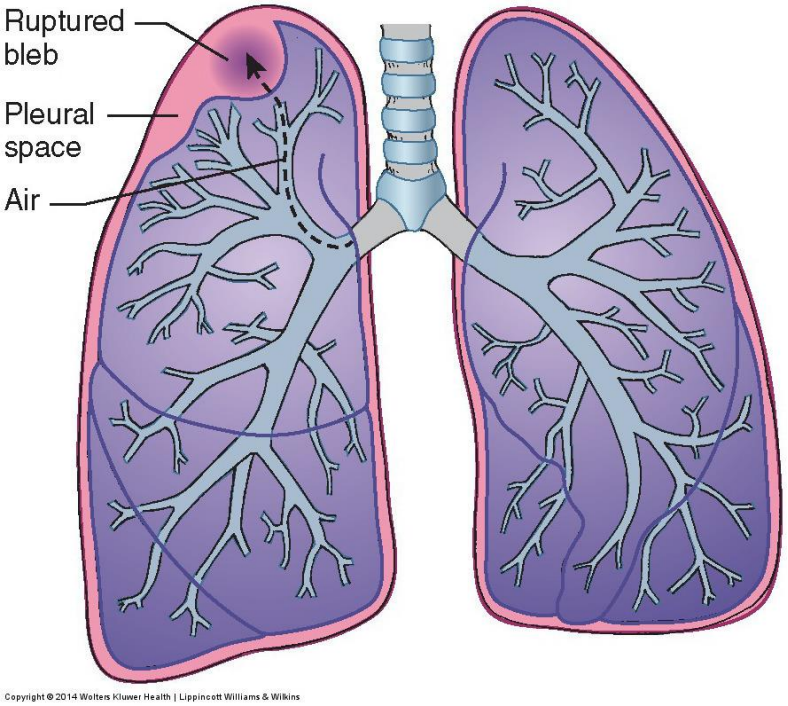
Pneumothorax

**Presence of air in the pleural space. Pneumothorax causes partial or complete collapse of the affected lung. It occurs either without obvious injury (Spontaneous pneumothorax) or after injury to chest (traumatic pneumothorax)

Spontaneous Pneumothorax.

Spontaneous pneumothorax is due to the rupture of an air-filled bleb, or blister, on the surface of the lung. Rupture of these blebs allows atmospheric air from the airways to enter the pleural cavity.

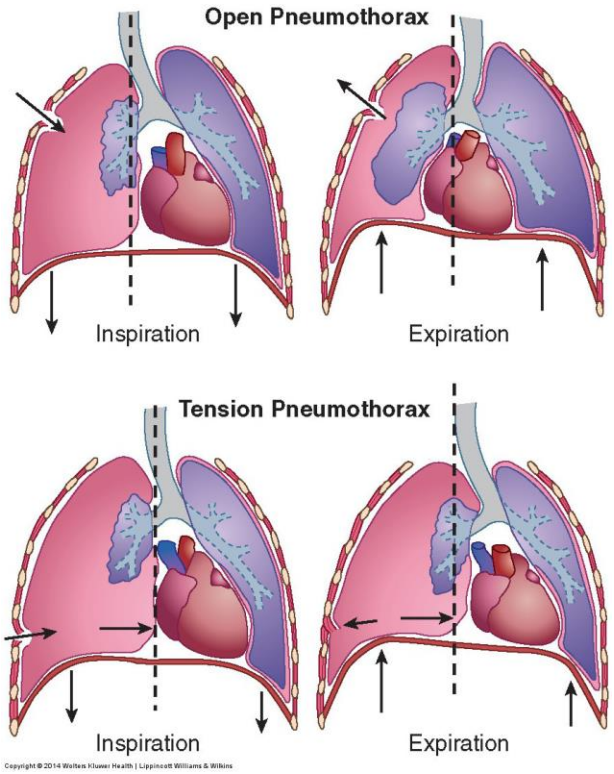
Blebs are small subpleural thin walled air containing spaces. If they rupture, they allow air to escape into pleural space resulting in a spontaneous pneumothorax.



Tension Pneumothorax

**In this type, the intrapleural pressure exceeds atmospheric pressure.

It is a life-threatening condition and occurs when injury to the chest or respiratory structures permits air to enter but not leave the pleural space. It causes compression atelectasis of the unaffected lung, a shift in the mediastinum to the opposite side of the chest, and compression of the vena cava, which results in a decrease in venous return to the heart and reduced cardiac output.



**It is mostly seen often in people with traumatic pneumothoraces.