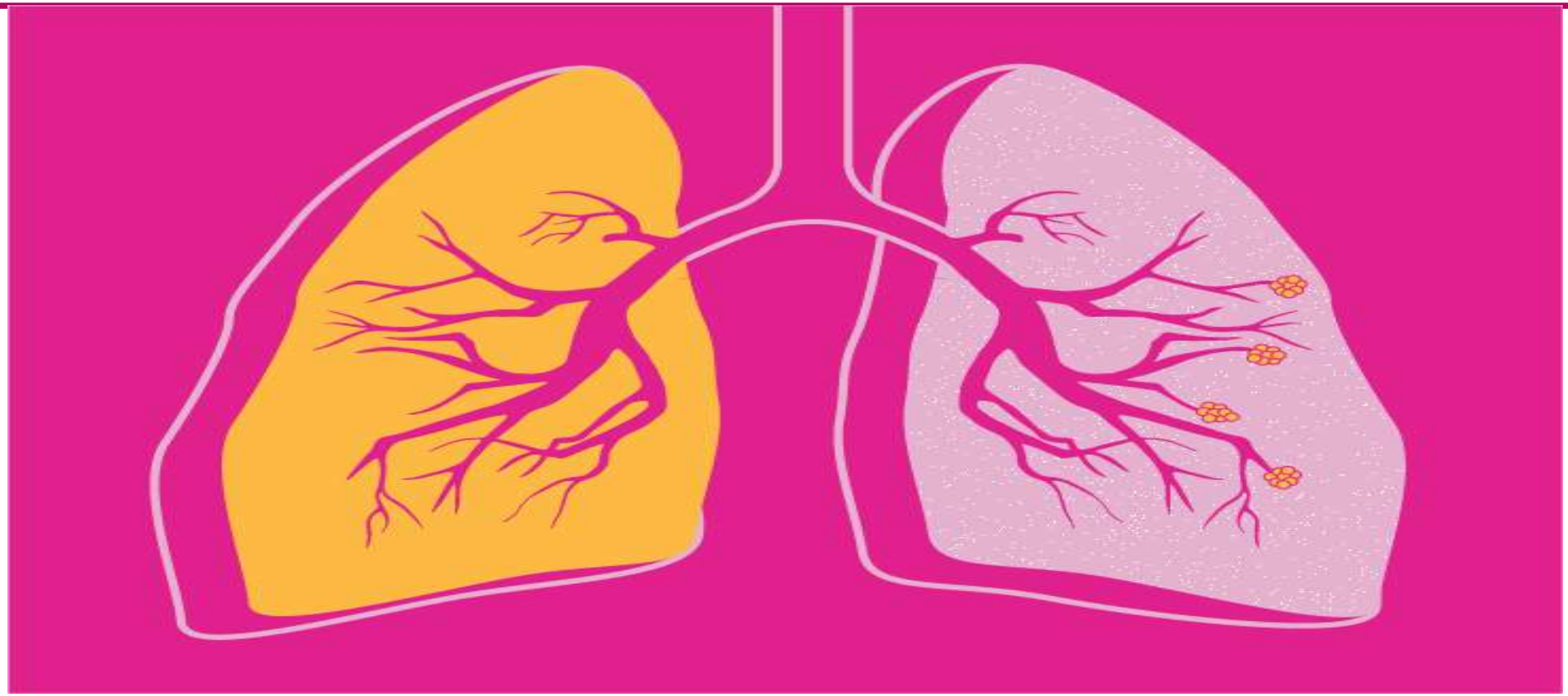


COPD

DEFINITION

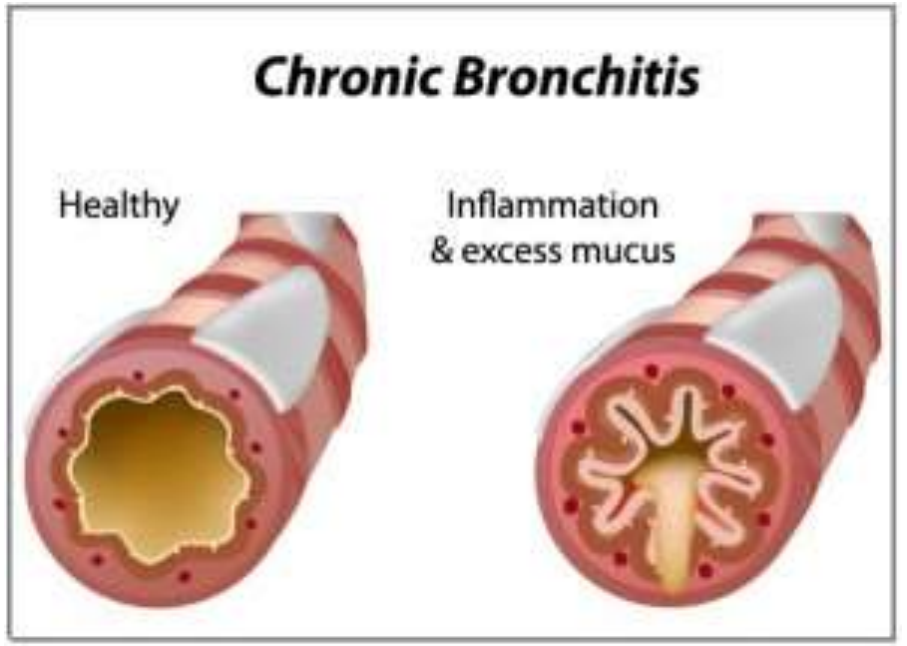
Chronic obstructive pulmonary disease (COPD) is a disease state characterised by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases (GOLD, 2009).

The term chronic obstructive lung disease (COLD), chronic obstructive airway disease (COAD) are synonymous with COPD.

It has conventionally included the subsets of chronic bronchitis and emphysema.

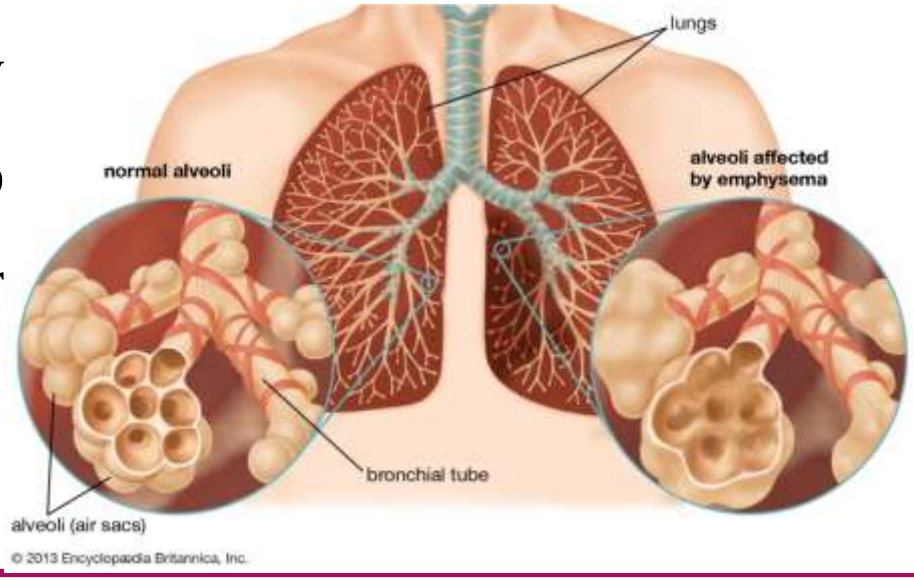
Chronic bronchitis

Chronic bronchitis is a condition with chronic or excess mucus secretion into the bronchial tree that occurs on most days during a period of at least 3 months of the year for at least 2 consecutive years.



Emphysema

Emphysema is a condition of the lung characterized by abnormal, permanent enlargement of the air spaces distal to the terminal bronchiole, accompanied by destruction of their walls, yet without obvious fibrosis.



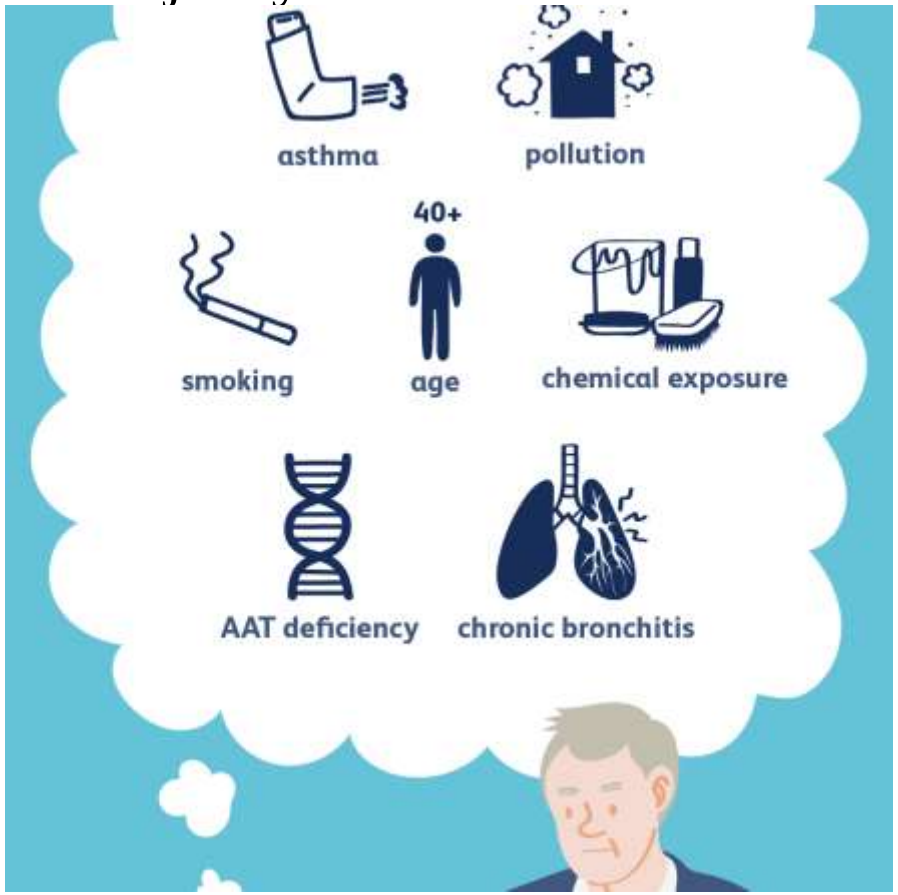
ETIOLOGY

The two most important etiologic factors responsible for majority of cases are:

- Cigarette smoking and
- Atmospheric pollution.

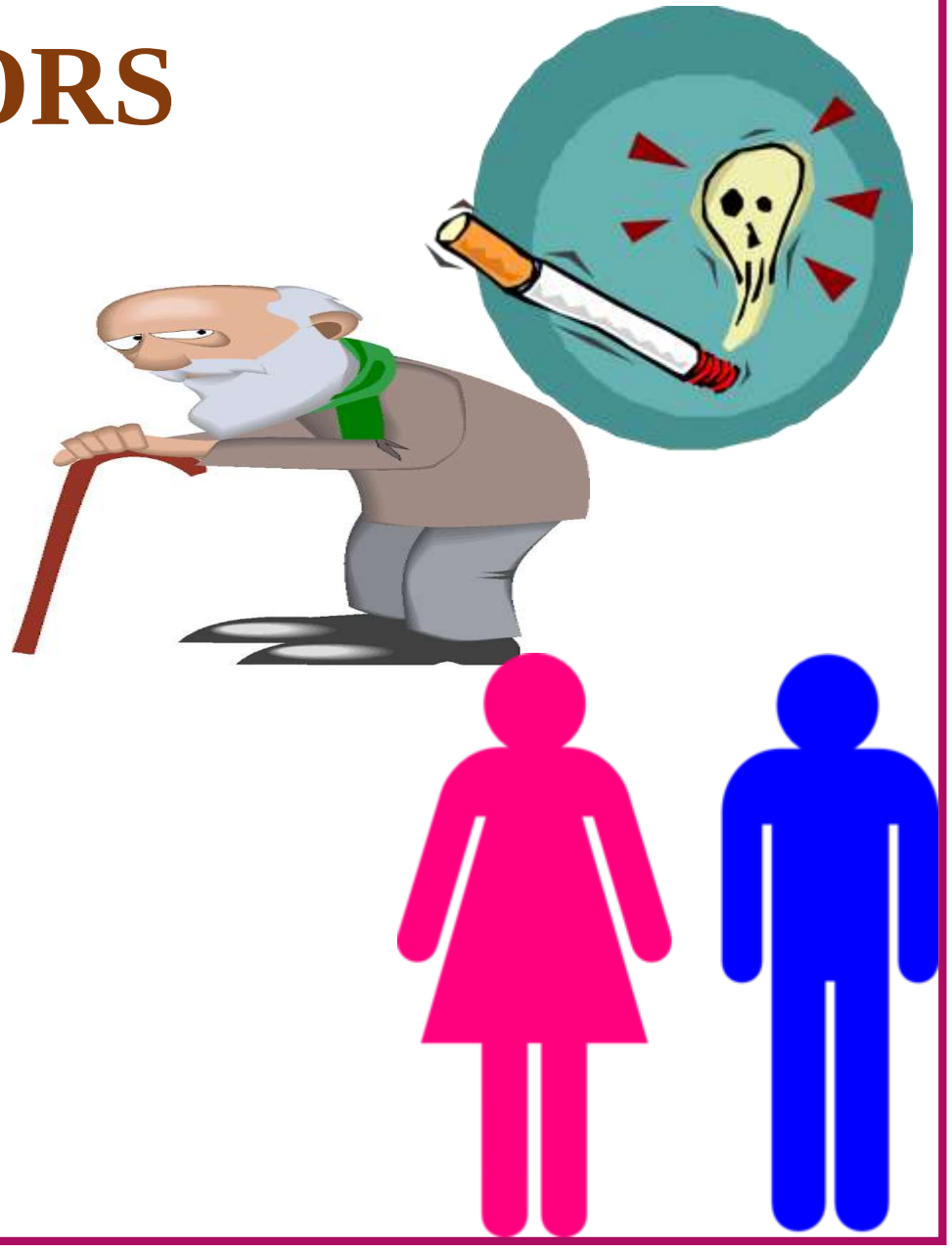
Other contributory factors are:

- Occupation,
- Infection,
- Familial and Genetic factors.



RISK FACTORS

- Smoking - Risk increases with increasing consumption but there is also large inter individual variation in susceptibility
- Age - Increasing age results in ventilatory impairment; most frequently related to cumulative smoking
- Gender - Male gender was previously thought to be a risk factor but this may be due to a higher incidence of tobacco smoking in men. Women have greater airway reactivity and experience faster declines in FEV1, so may be at more risk than men



- Occupation - The development of COPD has been implicated with occupations such as coal and gold mining, farming, grain handling and the cement and cotton industries
- Genetic factors - α1-Antitrypsin deficiency is the strongest single genetic risk factor, accounting for 1–2% of COPD. Other genetic disorders involving tissue necrosis factor and epoxide hydrolase may also be risk factors
- Air pollution - Death rates are higher in urban areas than in rural areas. Indoor air pollution from burning biomass fuel is also implicated as a risk factor, particularly in underdeveloped areas of the world
- Socio-economic status - More common in individuals of low socio-economic status
- Airway hyper-responsiveness and allergy - Smokers show increased levels of IgE, eosinophils and airway hyper-responsiveness but how these influence the development of COPD is unknown

PATHOPHYSIOLOGY

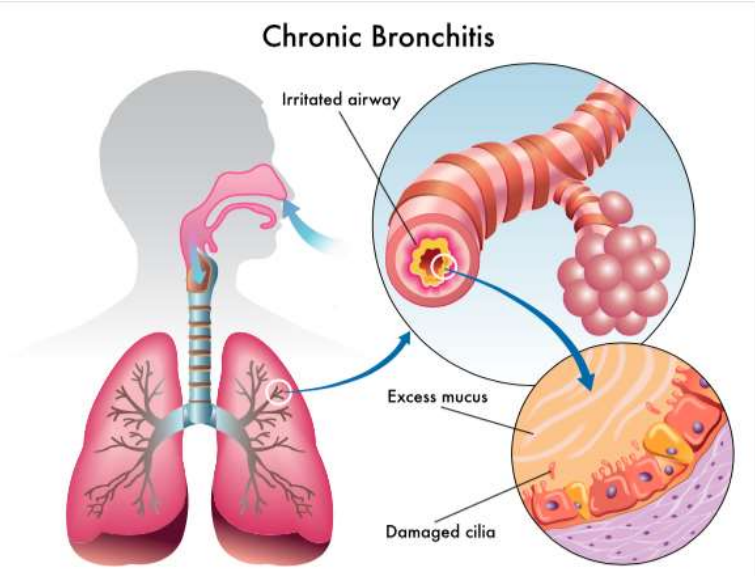
Chronic bronchitis

Irritants from cigarette and other smokes

Inhibits mucociliary clearance

Exaggerate the response of the protective mechanisms

Leads to inflammation of the bronchioles and alveoli



Trigger a degree of epithelial destruction

Macrophages and neutrophils infiltrate the epithelium

Hypertrophy and hyperplasia of mucus producing cells

Plugging of small bronchioles and alveoli with mucus and other particulate matter

Obstructed airway stimulate the vagus and causes bronchoconstriction

Accumulated pus and mucus leads to infections

Emphysema

- Alpha-1-antitrypsin (α-1-AT), also called α1-protease inhibitor (α-1-Pi), is a glycoprotein normally synthesised in the liver and is distributed in the circulating blood, tissue fluids and macrophages. The function of α1-AT is to inhibit proteases and hence its name α1-protease inhibitor.
- The proteases (mainly elastases) are derived from neutrophils. It has the capability of digesting lung parenchyma but is inhibited from doing so by anti-elastase effect of α1-AT.

Smoking

Increased inflammatory cell recruitment

Infiltration of neutrophils in lungs

Degradation of ECM

Increased level of protease

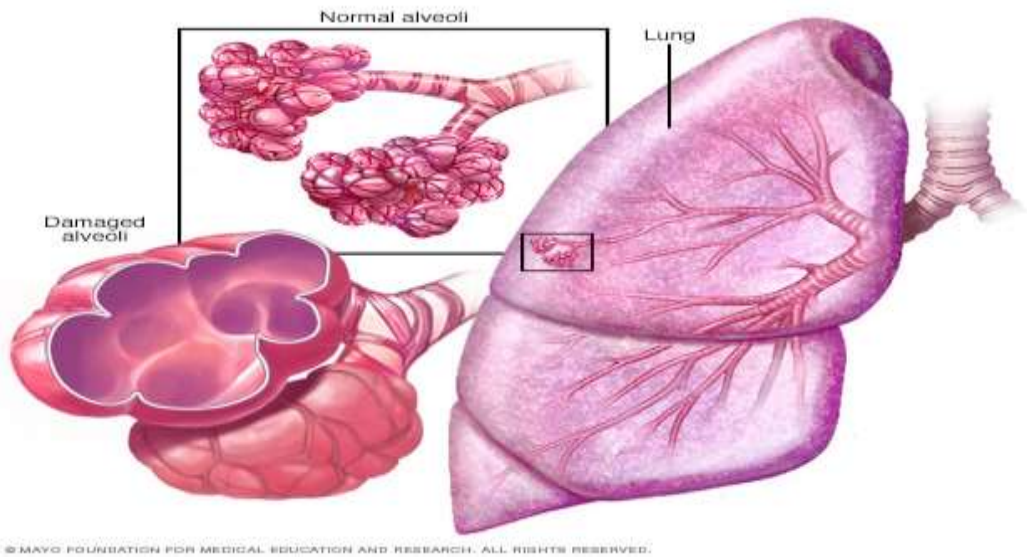
Digestion of lung parenchyma

Elastic damage of lungs

Emphysema

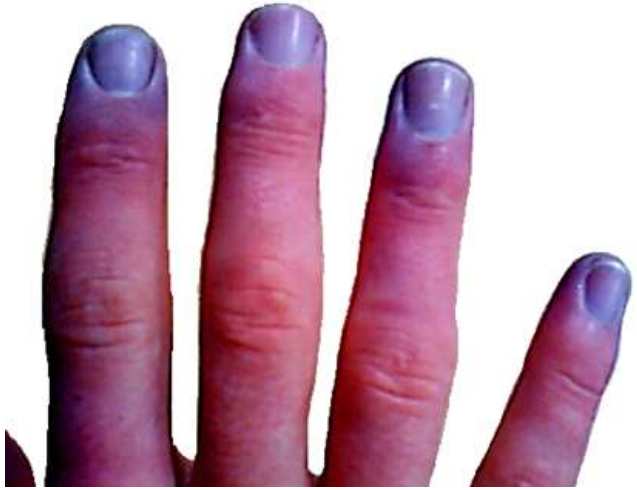
Oxidant in cigarette smoke has inhibitory influence on α-1-antitrypsin

Decreased level of α-1-antitrypsin

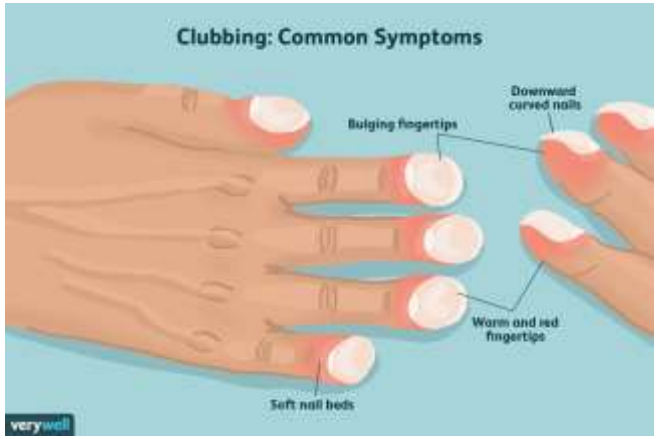


CLINICAL MANIFESTATIONS

- Bronchospasm resulting in wheeze and dyspnoea.
- Hypoxia
- Hypercapnia
- Productive cough
- Dyspnoea
- Cyanosis



- Peripheral oedema
- Pulmonary hypertension
- Clubbing
- Barrel chest



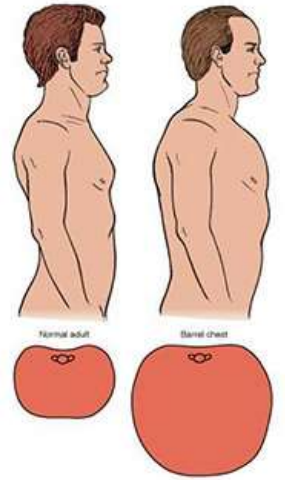


Figure 25-31 Profile and anteroposterior diameter of normal adult chest and barrel chest.

- Signs of infection include an increase in the volume of thick and viscous sputum, which is yellow or green in colour and may contain bacterial pathogens, squamous epithelial cells, alveolar macrophages and saliva, but pyrexia may not be present.

The clinical features of emphysema are different from those of bronchitis.

- Increasing dyspnoea even at rest,
- Minimal cough
- Scanty and mucoid sputum
- Tachypnoea
- Cor pulmonale

The patient with emphysema is sometimes referred to as a '**pink puffer**' because he or she hyperventilates to compensate for hypoxia by breathing in short puffs. As a result, the patient appears pink with little carbon dioxide retention and little evidence of oedema.

COMPLICATIONS

- Flu
- Pneumonia
- Heart diseases
- Lung cancer
- Anemia of chronic disease

DIAGNOSIS

- Spirometry
- Chest x ray
- Blood test
- Sputum test
- ECG
- Echocardiogram

THANK YOU