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Case history

A 56-year-old man with a smoking history of 1 pack per day for the past 37 years presents with progressive shortness of breath and chronic productive cough of yellowish sputum, for the past 2 years.

On examination he appears cachectic and in moderate respiratory distress, especially after walking to the examination room, and has pursed-lip breathing.

Lung examination reveals a barrel chest and poor air entry bilaterally, with moderate diffuse expiratory wheezing. Heart and abdominal examination are within normal limits. Lower extremities exhibit scant pitting edema.

Burden of COPD

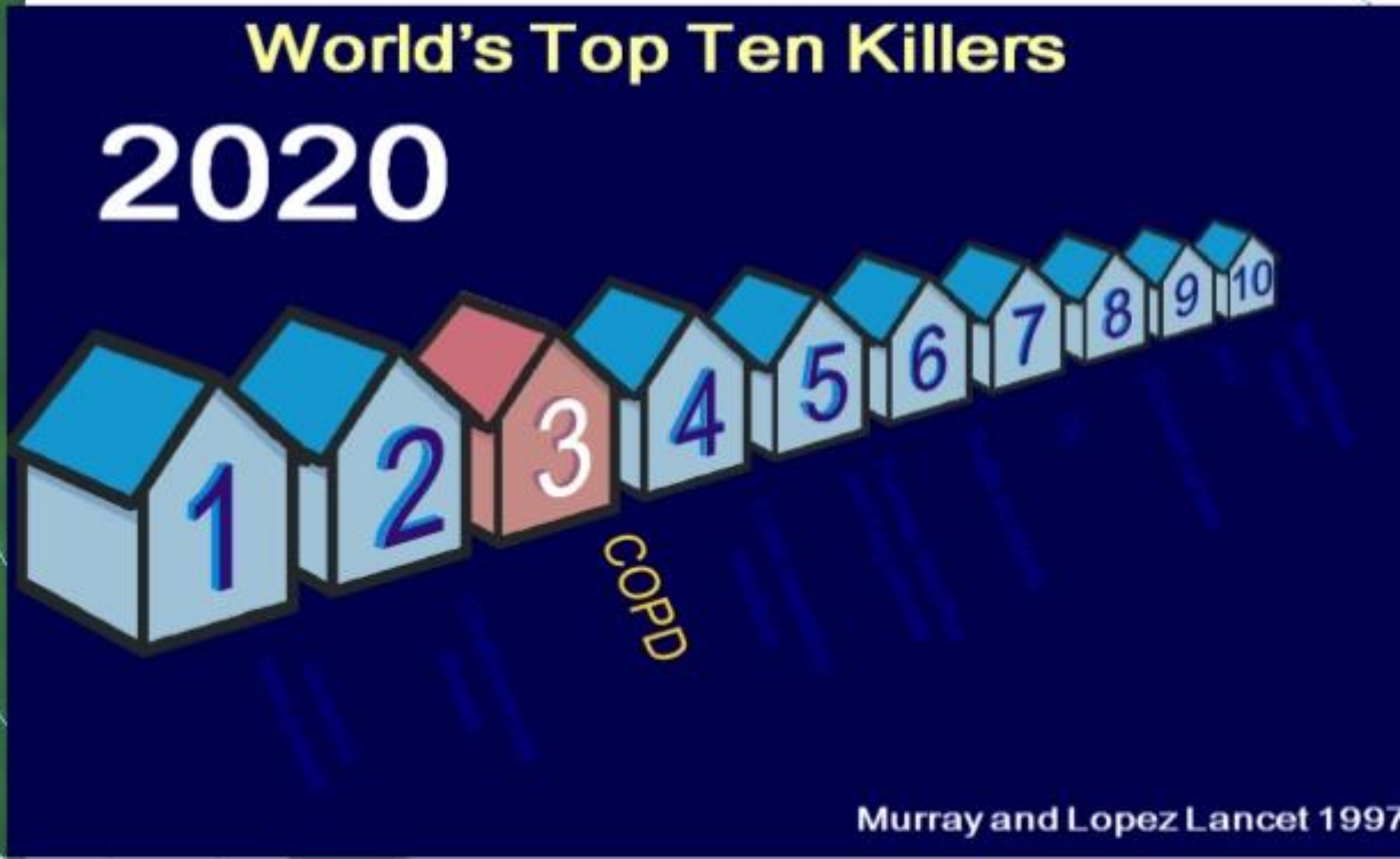
COPD is a leading cause of morbidity & mortality worldwide

The burden will increase in coming decades due to continued exposure to risk factors & the aging of the world's population

COPD is associated with significant economic burden

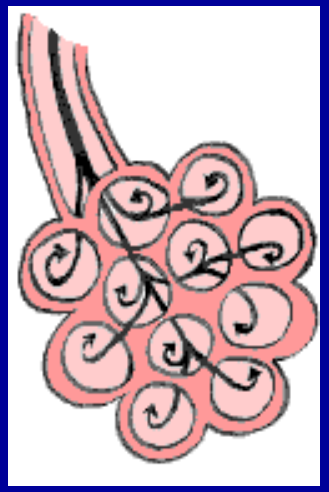
Mortality :

Global burden of Disease study: COPD rank

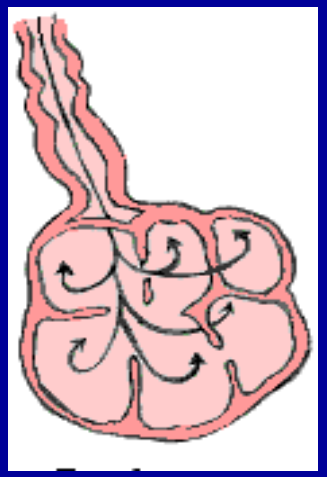


Definition of COPD

- COPD, a common preventable disease, characterized by airflow limitation which is persistent (not fully reversible) usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases.
- The most common respiratory diseases related to smoking.



Healthy Alveolus



COPD

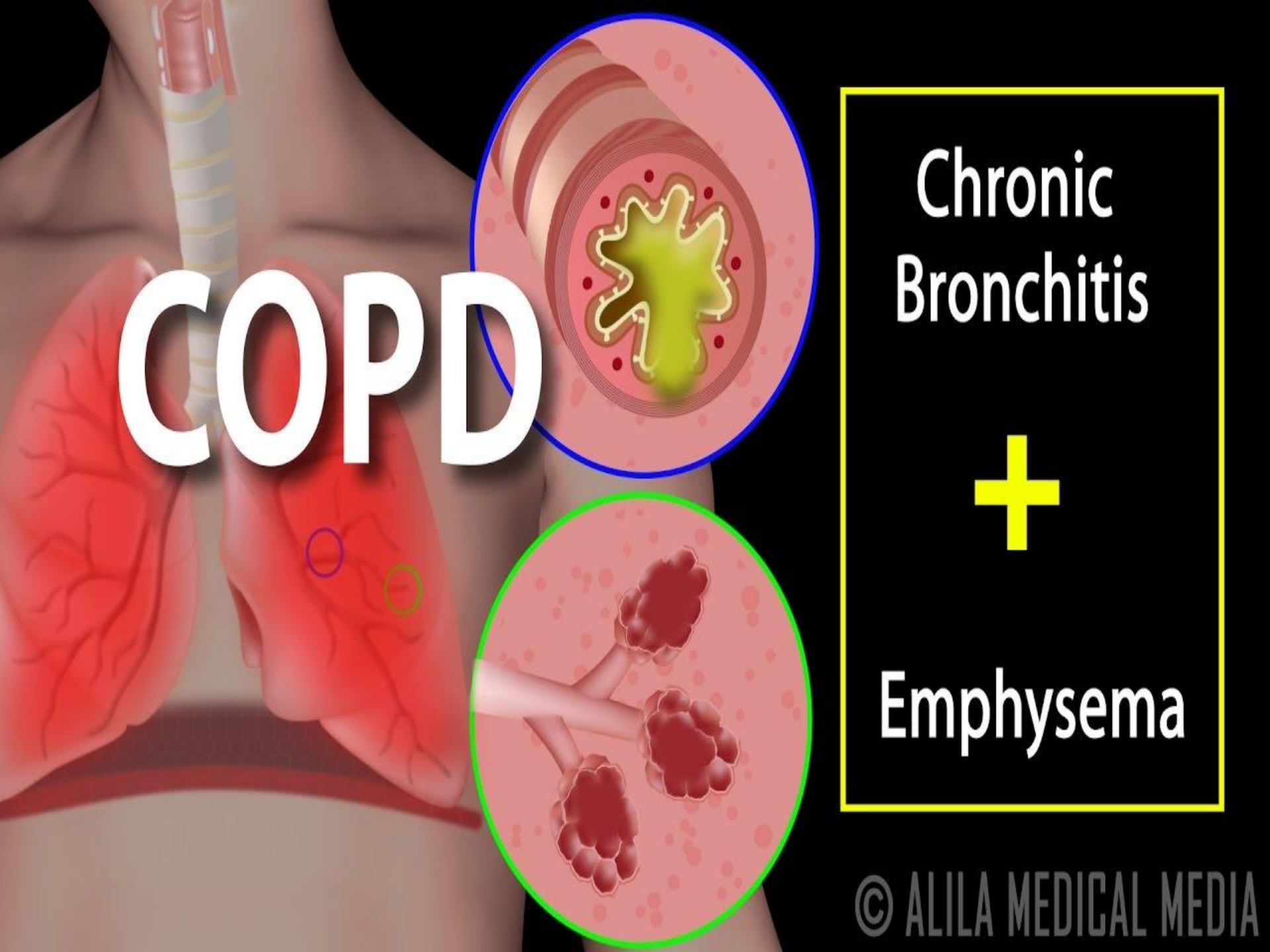
Which of the following diseases is included in the umbrella term COPD?

A. Emphysema

B. Chronic bronchitis

C. Lung cancer

D. A and B


COPD
Chronic Bronchitis
+
Emphysema
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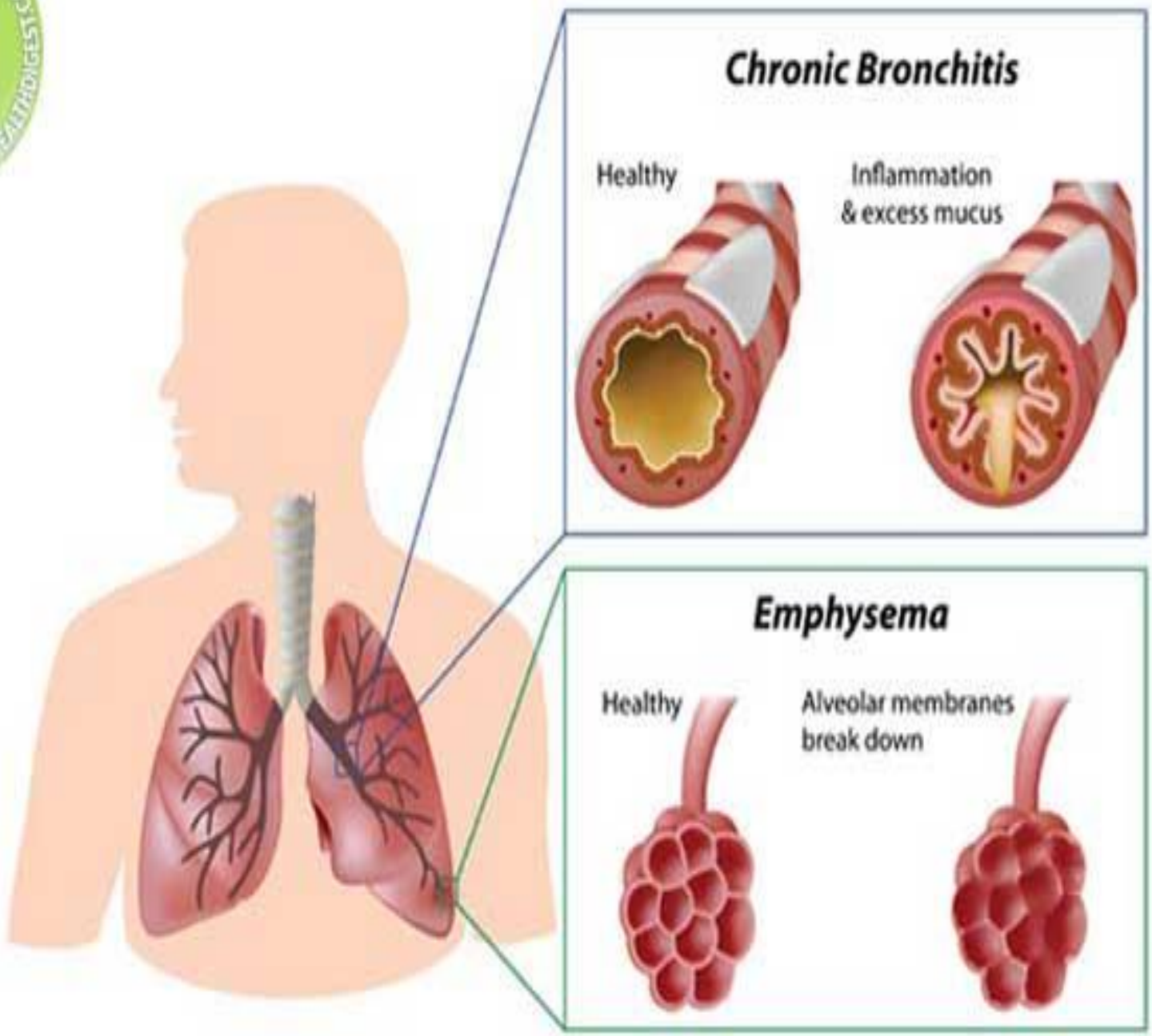
Pathologic Types

B- Chronic Bronchitis

Presence of chronic **productive cough** for 3 months / year for 2 successive years **after exclusion** of other causes of chronic productive cough

Chronic Obstructive Pulmonary Disease (COPD)



A- Emphysema

- Abnormal permanent enlargement of the air space distal to the terminal bronchioles

- Accompanied by destruction of the alveolar wall.

Alveoli with emphysema

Microscopic view of normal alveoli

Pathophysiological changes in emphysema

- Hyperinflation of alveoli
- Destruction of alveolar walls
- Destruction of alveolar capillary walls
- Loss of lung elasticity
- Narrowed airways

COPD is almost always caused by ……..?

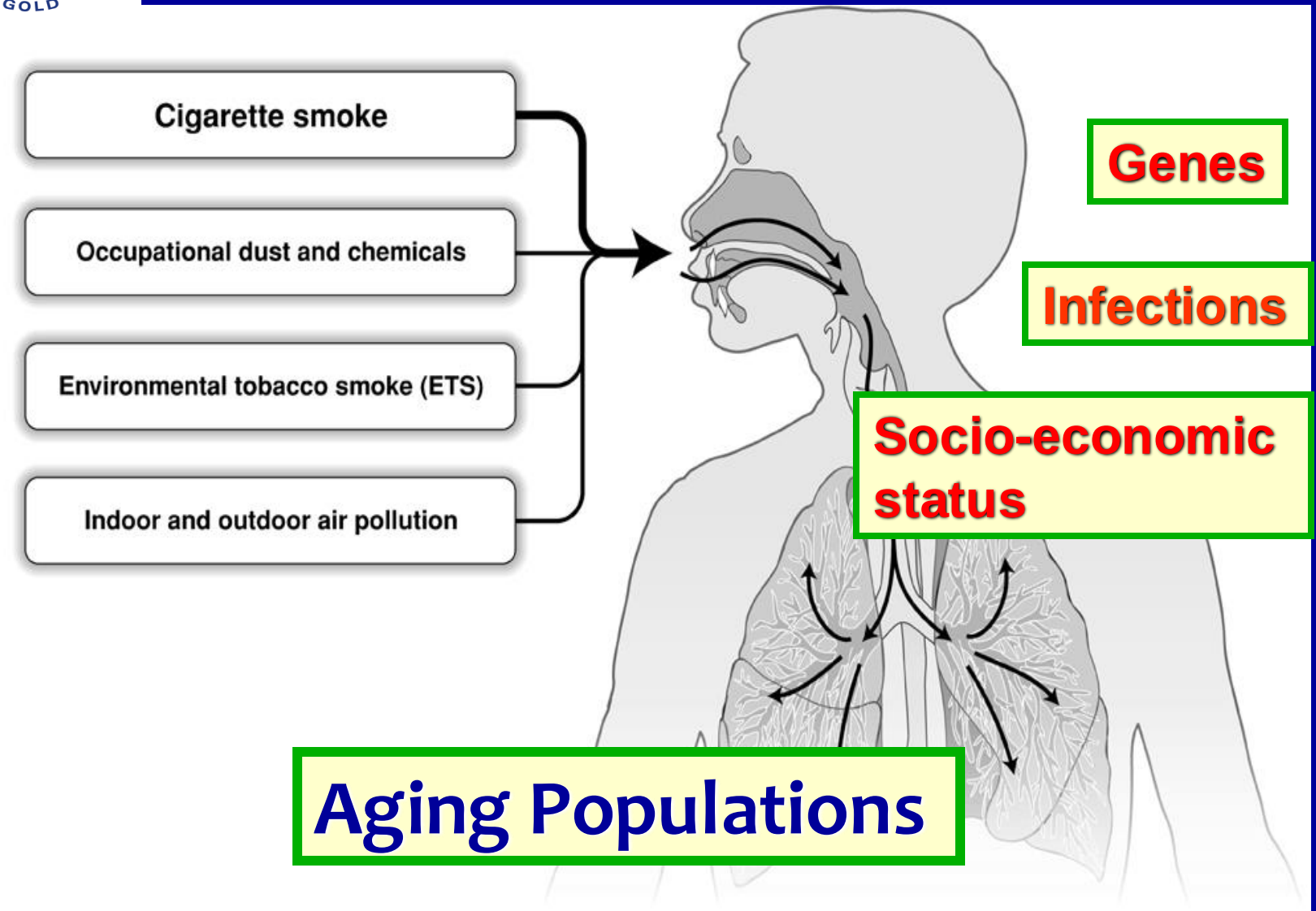
A. Pollution

B. Dust

C. Smoking

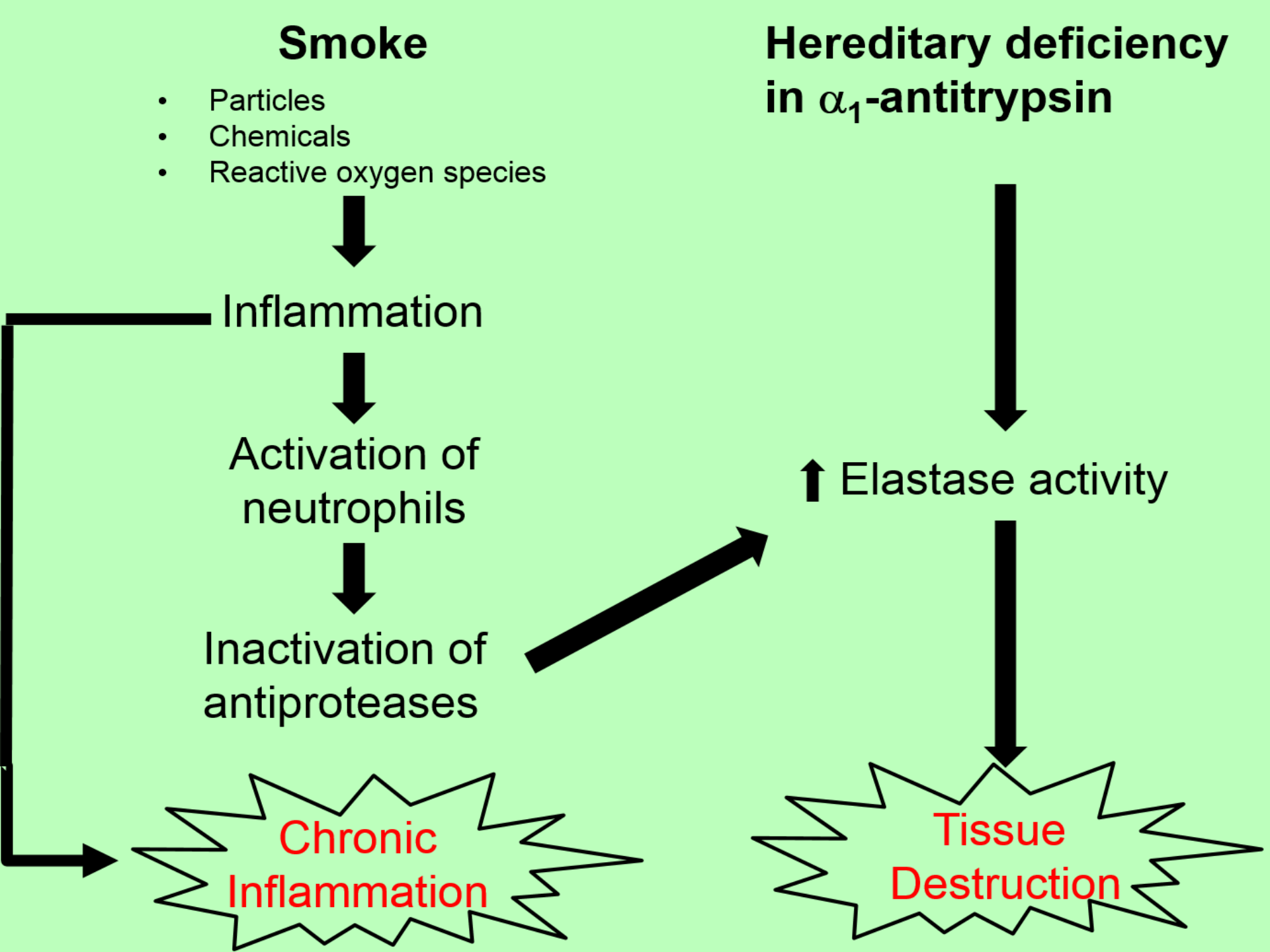
D. Exposure to asbestos

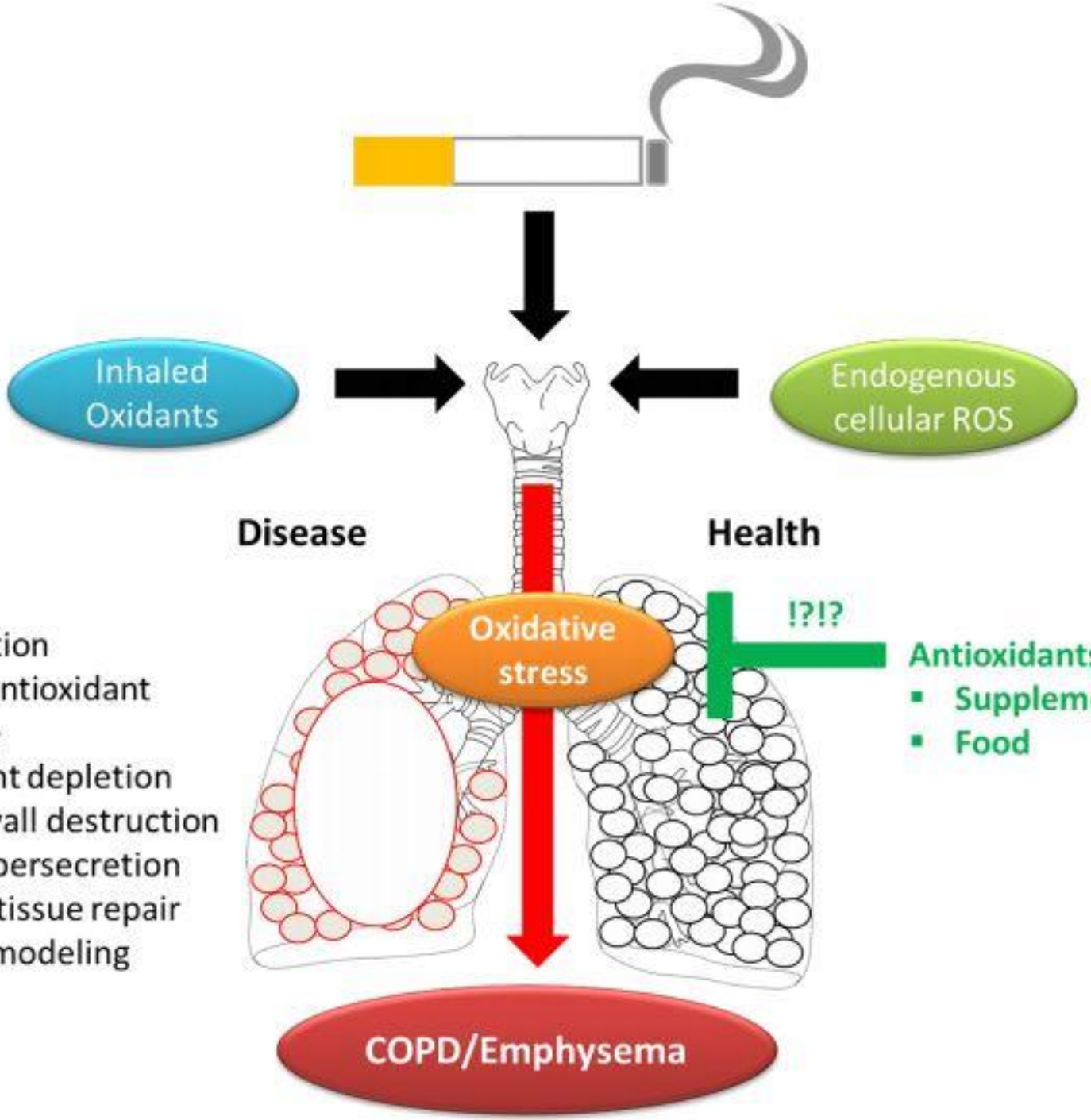
Risk Factors for COPD



Risk Factors for COPD

- **Tobacco smoking** is the **main risk factor** for COPD, although other **inhaled noxious particles** and gases may contribute.
- **Smoking causes COPD by these mechanisms:**
  - Inflammation
  - Imbalance of proteases and anti- proteases
  - Oxidative stress.


Smoke
Particles
Chemicals
Reactive oxygen species
Hereditary deficiency in α_1-antitrypsin
Inflammation
Activation of neutrophils
Inactivation of antiproteases
Elastase activity
Chronic Inflammation
Tissue Destruction



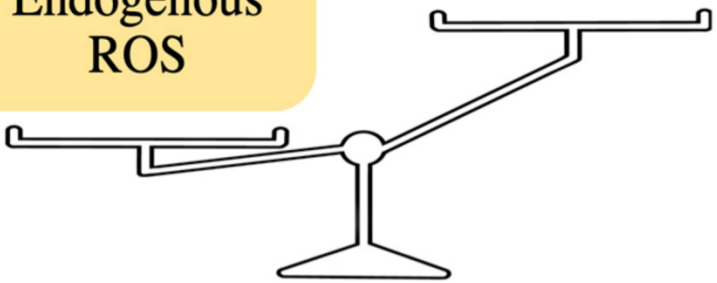
- Inflammation
- Oxidant/Antioxidant imbalance
- Antioxidant depletion
- Alveolar wall destruction
- Mucus hypersecretion
- Defective tissue repair
- Airway remodeling

Cigarette smoke
Aerobic metabolism
Airway inflammation

Exogenous & Endogenous ROS

Anti-oxidant defense
ex. TRX

Cellular aging



COPD pathogenesis

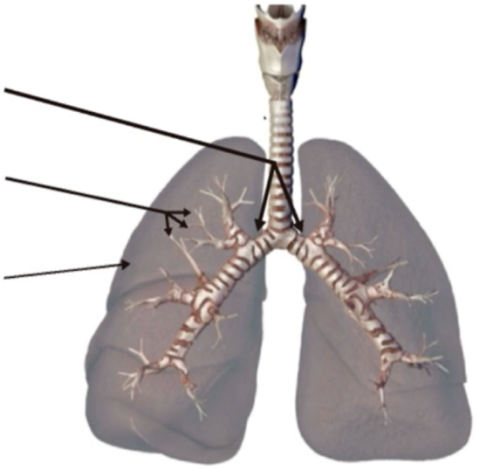
Chronic inflammation

Excessive oxidative stress

Protease anti-protease imbalance

Airway inflammation (chronic bronchitis)

Alveolar destruction (emphysema)



Non-Smoker

Smoker

- 80-90% of COPD patient are smoker.
- 10-20% of smokers develop COPD.

Risk Factors for COPD

- **Infection**
  - Major contributing factor in **exacerbation** and progression of COPD
- **Heredity**
  - **α-Antitrypsin (AAT) deficiency** (produced by liver and found in lungs); accounts for **< 1%** of COPD cases
  - Emphysema results from destruction of lung tissues by **proteolytic enzymes** from neutrophils and macrophages

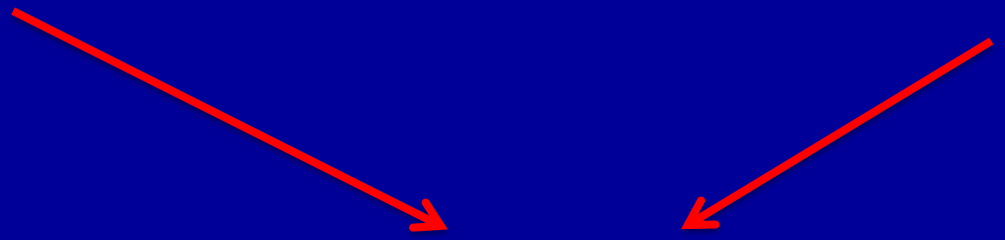
Mechanisms of Airflow Limitation in COPD

Small Airways Disease

- **Airway inflammation**
- **Mucus hypersecretions**
- **Airway fibrosis,**

Parenchymal Destruction

- **Loss of alveolar attachments ► Decrease of elastic recoil**



Airflow limitation

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

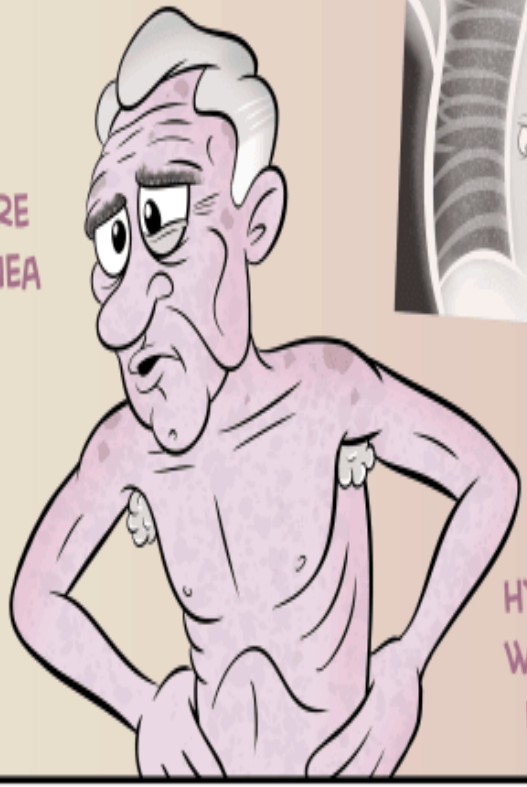
RHONCHI AND WHEEZING

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

Clinical Picture of COPD

- Cough
- Expectoration
- Chest Wheeze
- Progressive dyspnea

Global Strategy for Diagnosis, Management and Prevention of COPD

Modified MRC (mMRC)Questionnaire

PLEASE TICK IN THE BOX THAT APPLIES TO YOU
(ONE BOX ONLY)

mMRC Grade 0. I only get breathless with strenuous exercise. ☐

mMRC Grade 1. I get short of breath when hurrying on the level or walking up a slight hill. ☐

mMRC Grade 2. I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level. ☐

mMRC Grade 3. I stop for breath after walking about 100 meters or after a few minutes on the level. ☐

mMRC Grade 4. I am too breathless to leave the house or I am breathless when dressing or undressing. ☐

Signs of COPD

General examination

- Tripod position with pursed lip breathing
- ↑Respiratory Rate
- Cyanosis
- Flapping tremors
- Pitting lower limb edema

Signs of COPD

Inspection /Palpation

- Bilateral Diminished chest expansion
- Litten ‘s sign
- Barrel chest
- Tracheal tug

Normal Chest vs. Barrel-Shaped Chest

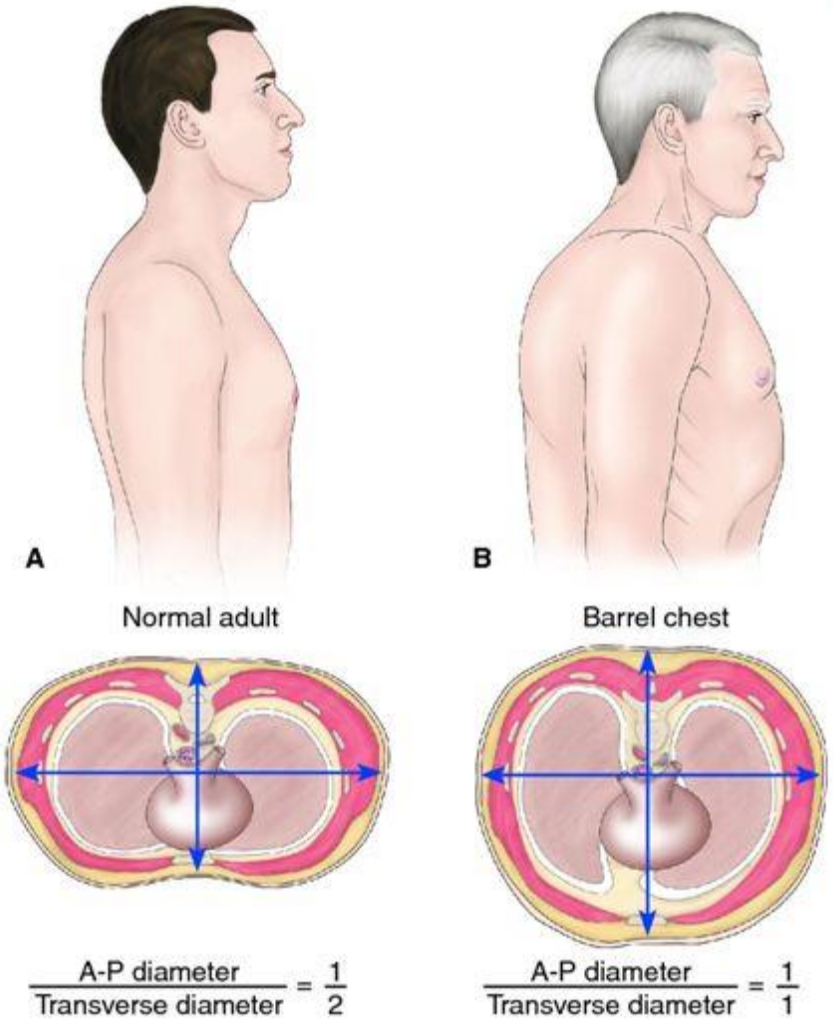


Figure 24-3

Litten's sign

Percussion :

- Hyperresonant chest
- Depressed diaphragm
- Resonant bare area of the heart

Auscultation:

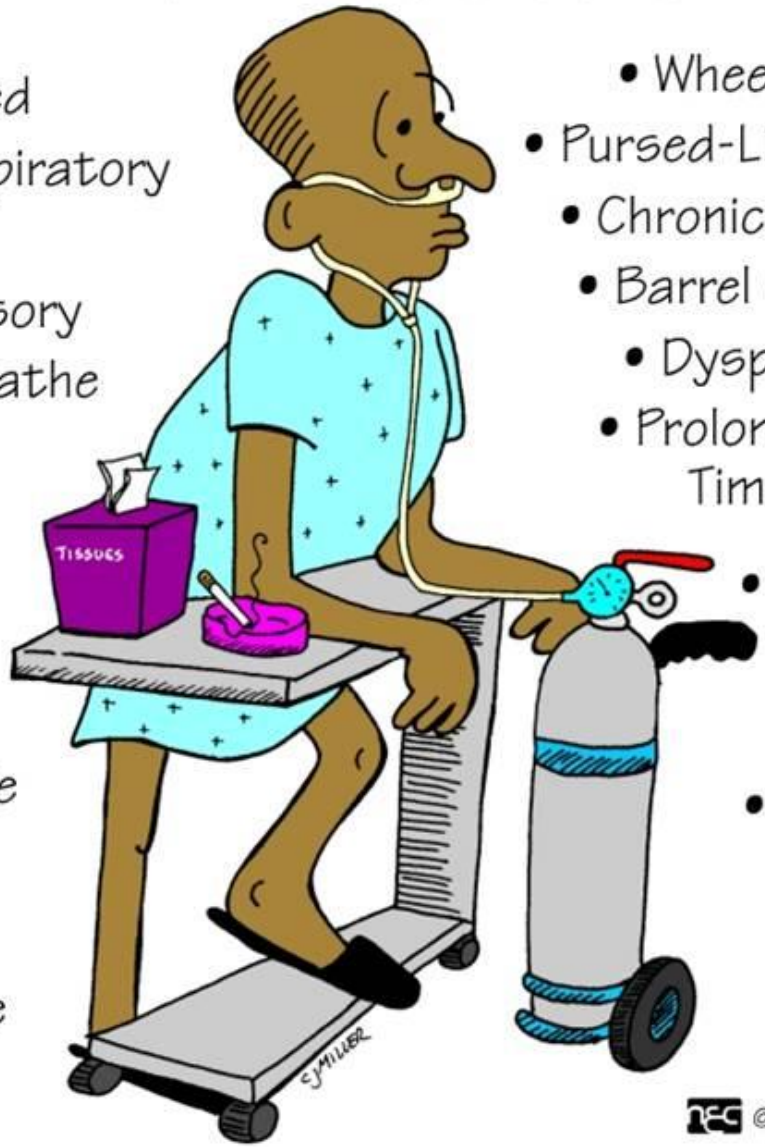
- Harsh vesicular BS with Prolonged expiration
- Wheezing during quiet breathing
- Crackle can be heard if infection exist.
- The heart sounds are best heard over the xiphoid area.

COPD

CHRONIC AIRFLOW LIMITATION

"EMPHYSEMA AND CHRONIC BRONCHITIS"

- Easily Fatigued
- Frequent Respiratory Infections
- Use of Accessory Muscles to Breathe
- Orthopneic



- Wheezing
- Pursed-Lip Breathing
- Chronic Cough
- Barrel Chest
- Dyspnea
- Prolonged Expiratory Time

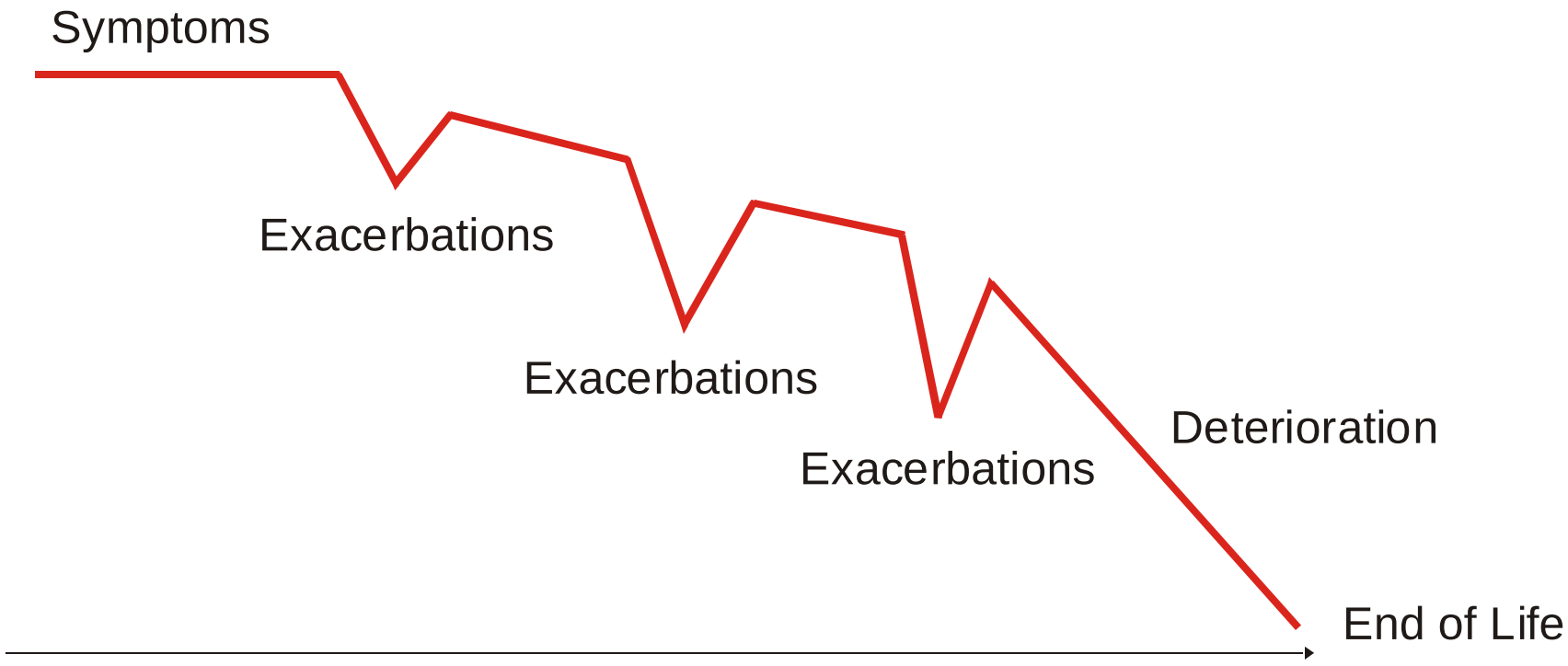
- Bronchitis - Increased Sputum

- Digital Clubbing

- Cor Pulmonale (Late in Disease)

- Thin in Appearance

Disease Trajectory of a Patients with COPD



Diagnosis of COPD

Symptoms & Signs

cough

sputum

shortness of breath

Risk Factors

Tobacco

Occupation

Indoor/Outdoor pollution

SPIROMETRY

Spirometry

- Diagnosis
- Assessing severity
- Assessing prognosis
- Monitoring progression

Spirometry

Classification of Severity of Airflow Limitation in COPD (Based on Post - Bronchodilator FEV_1)		
In patients with post - bronchodilator FEV1/ FVC< 0.70		
GOLD 1	Mild	FEV1 ≥ 80% predicted
GOLD 2	Moderate	≤ 50% FEV1 < 80% predicted
GOLD 3	Severe	≤ 30% FEV1 < 50% predicted
GOLD 4	Very severe	FEV1 < 30 % predicted

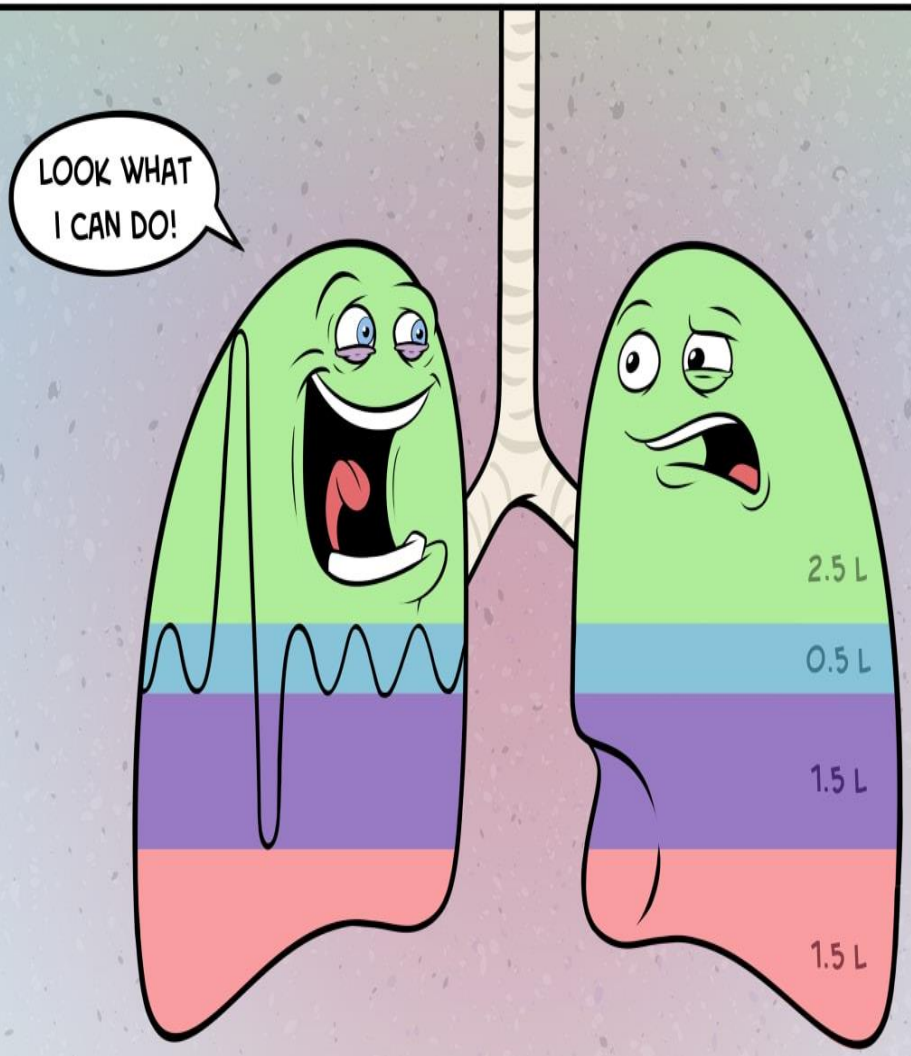
LUNG VOLUMES AND CAPACITIES

IRV
INSPIRATORY RESERVE VOLUME

V_T
TIDAL VOLUME

ERV
EXPIRATORY RESERVE VOLUME

RV
RESIDUAL VOLUME



IC
INSPIRATORY CAPACITY

FRC
FUNCTIONAL RESIDUAL CAPACITY

VC
VITAL CAPACITY

TLC
TOTAL LUNG CAPACITY

Chest X-ray

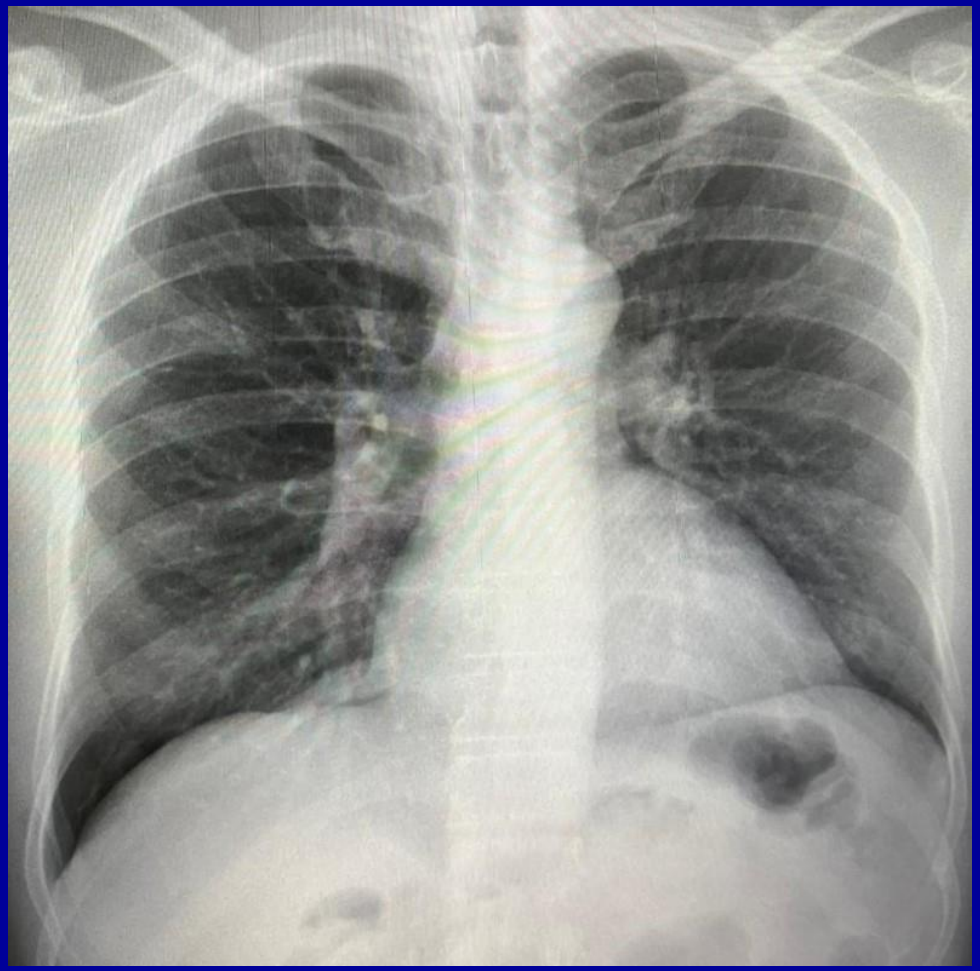
Radiological signs of Hyperinflation:

- ✓ **Low flat diaphragm with indentation**
- ✓ **Intercostal space becomes widen**
- ✓ **Horizontal pattern of ribs**
- ✓ **A long thin heart shadow**
- ✓ **Decreased Bronchovascular markings of lung (airways & vessels)**

Chest X-ray - Emphysema

Chest X-ray - Chronic Bronchitis

Thickened and increased of the lung markings

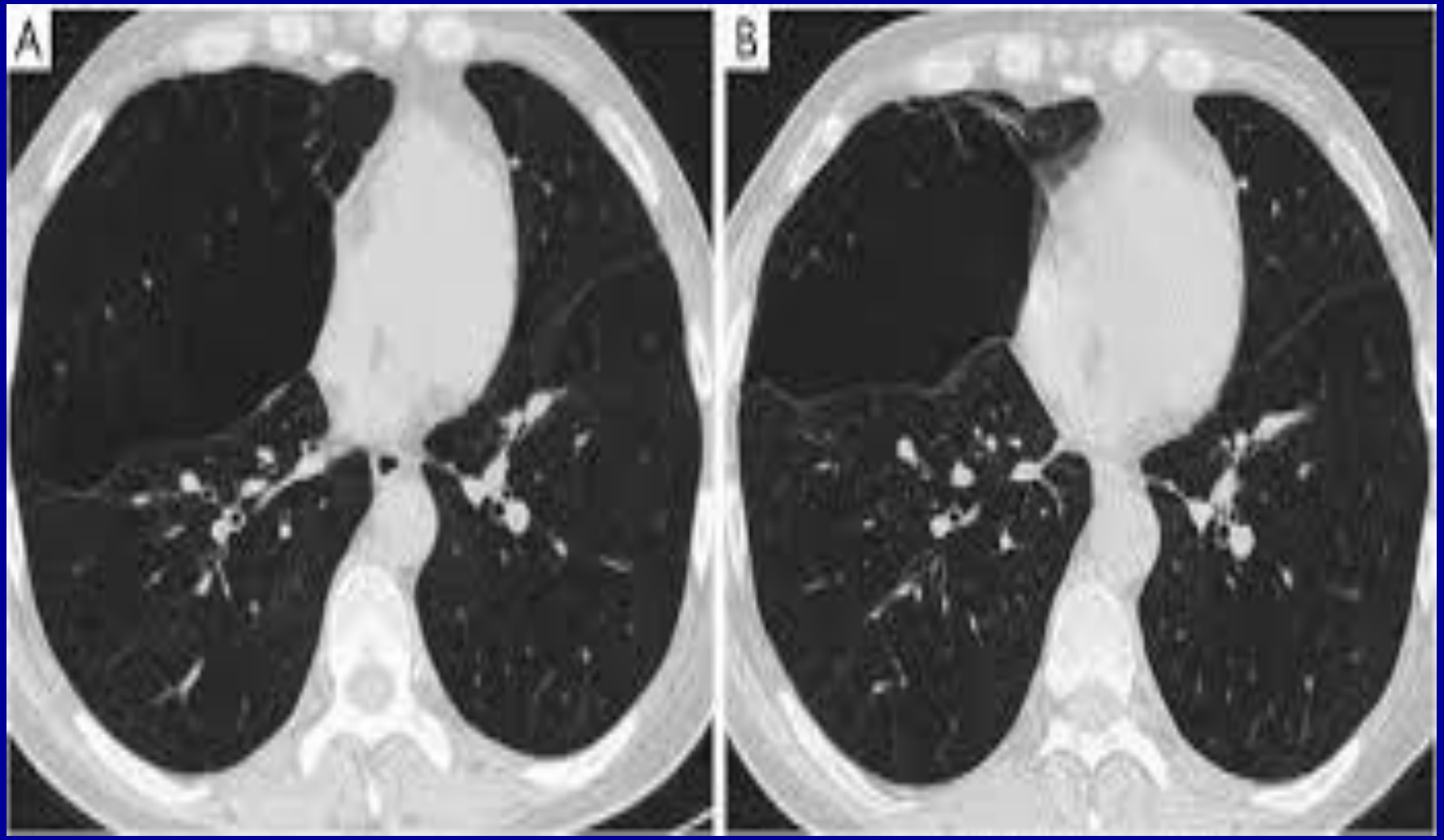


CT Chest

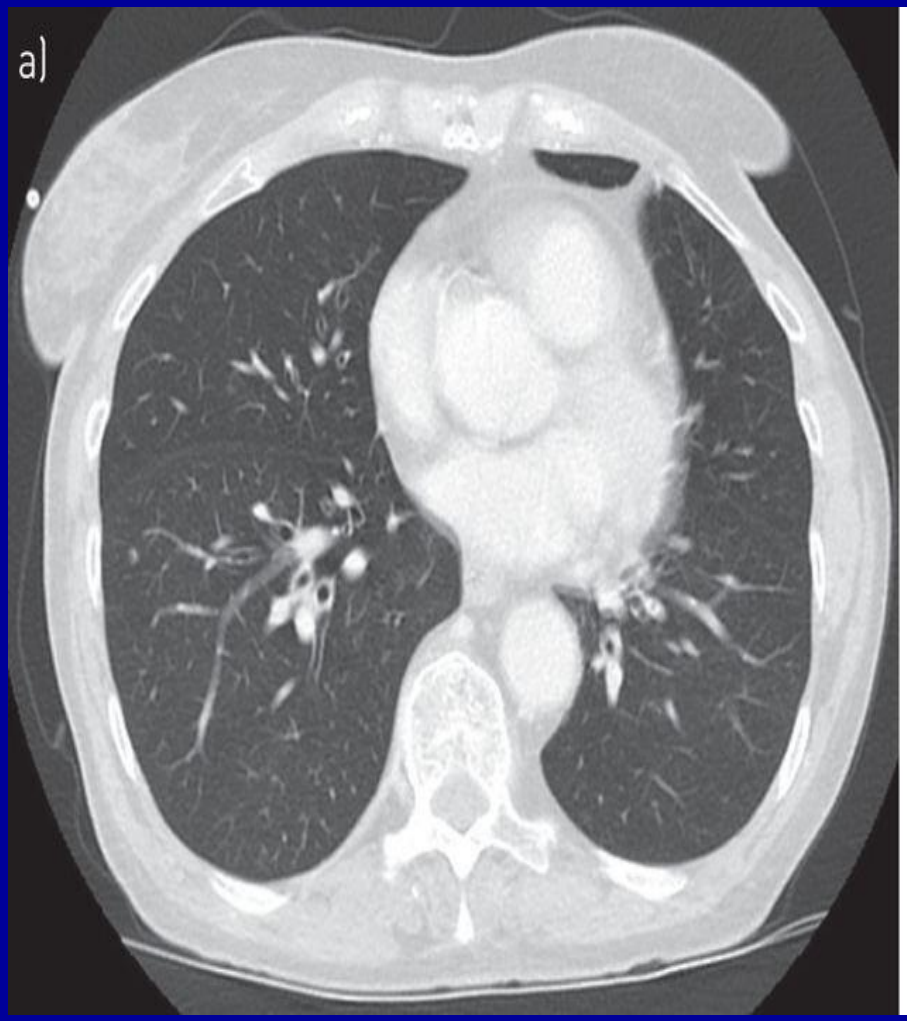
- Greater sensitivity and specificity for emphysema than CXR

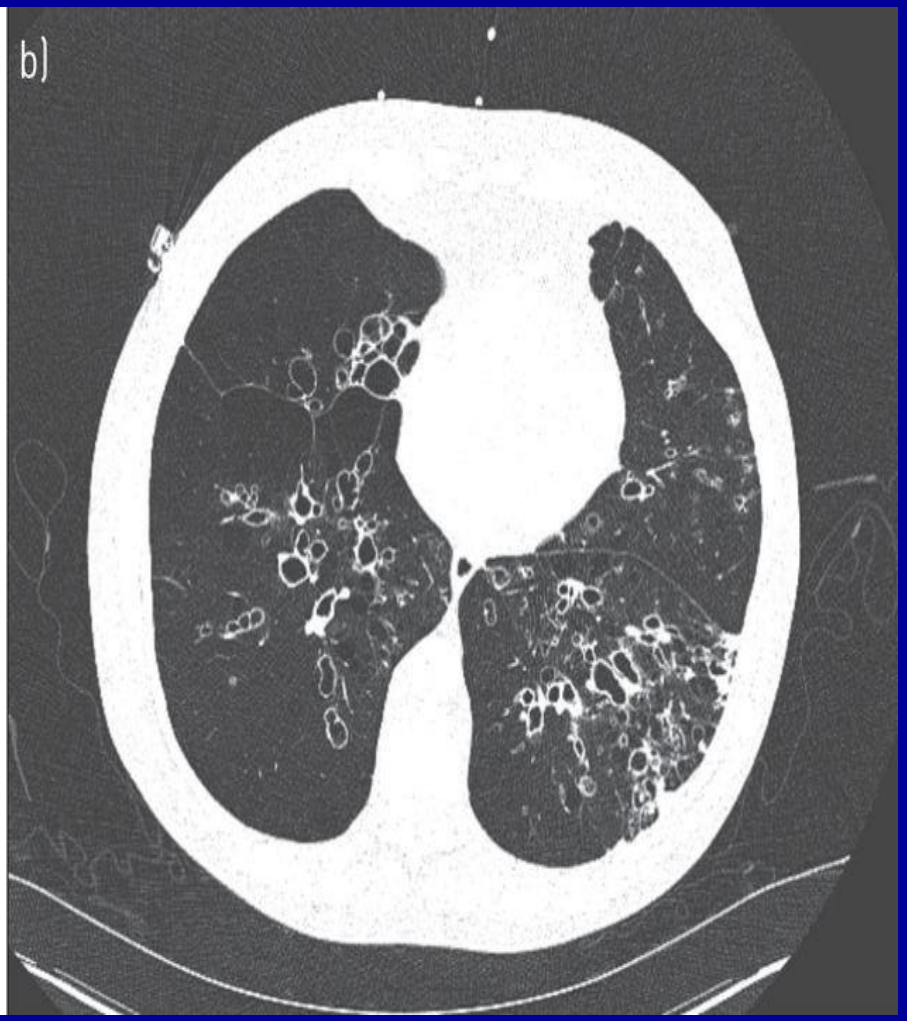
- Especially for the diagnosis of bronchiectasis and evaluation of bullous disease

CT Chest



CT Chest





Diagnostic studies

- ABGs
  - ↓ PaO_2
  - ↑ $PaCO_2$ (especially in chronic bronchitis)
  - ↓ pH (especially in chronic bronchitis)
  - ↑ Bicarbonate level found in late stages COPD

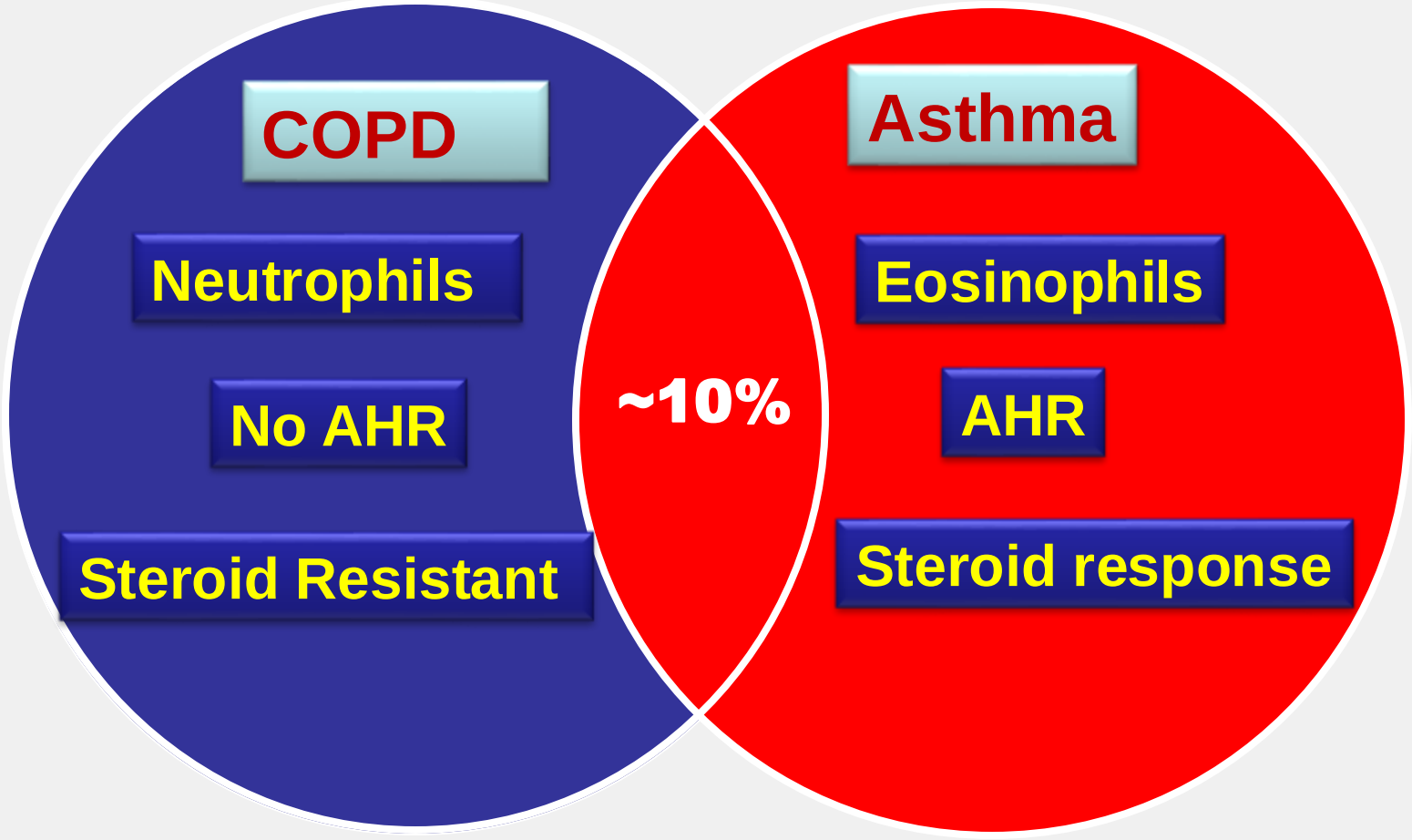
Complications of COPD

- Pulmonary hypertension
- Cor pulmonale (Rt. ventricular enlargement ± Heart Failure resulting from diseases that affect structure or function of the lungs)
- Pneumothorax
- Respiratory Failure
- Lunge cancer
- Bronchiectasis
- Psychological disorders eg depression.

Clinical Differences Between Asthma and COPD

Clinical features	Asthma	COPD
Age of onset	Usually early childhood, but may have onset at any age	Usually > 40 years old
Smoking history	May be non-, ex- or current smoker	Usually > 10 pack-years
Atopy	Often	Infrequent
Family history	Asthma or other atopic disorders commonly present	Not a usual feature
Clinical symptoms	Intermittent and variable	Persistent and gradually progressive worsening
Cough	Nocturnal cough or on exertion	Morning cough with sputum
Sputum production	Infrequent	Often
Reversibility of airflow obstruction	Characteristic of asthma	Airflow limitation may improve but never normalises

Overlap between COPD and Asthma



Differential diagnosis of COPD patients

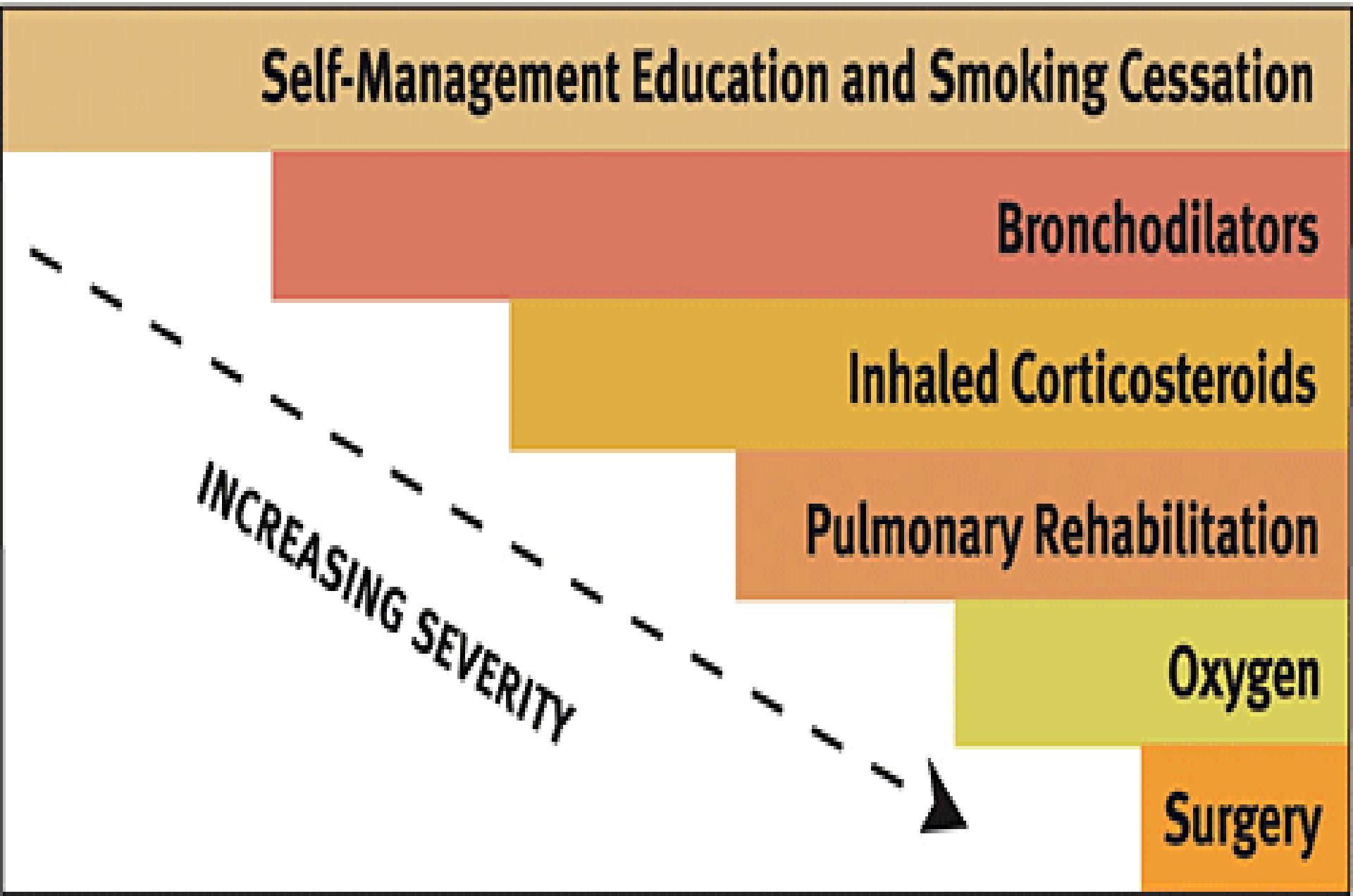
Respiratory

- Asthma
- Bronchiectasis
- ILD
- TB
- Recurrent PE
- OSAS

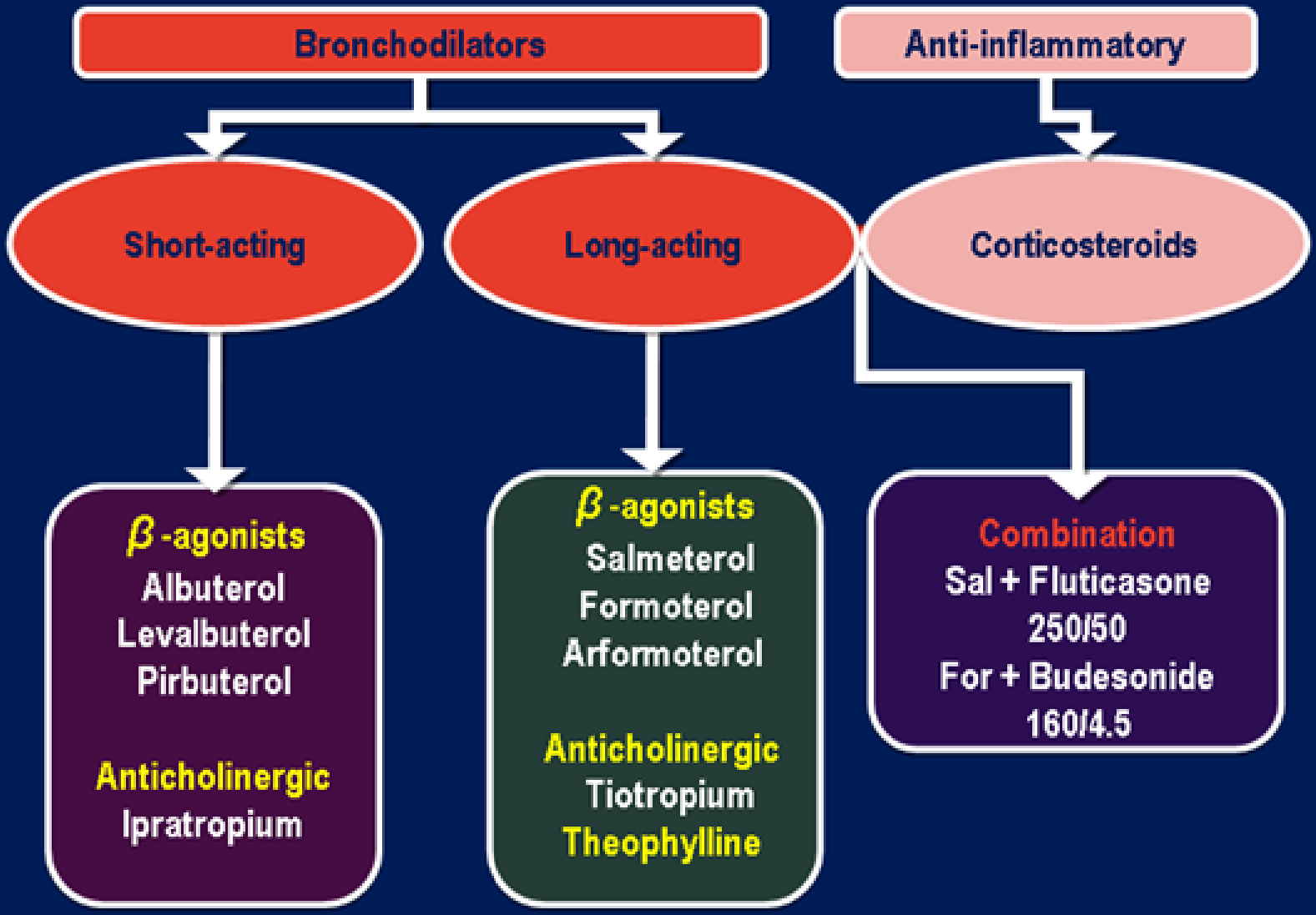
Non-Respiratory

- Cardiac disease
- Vocal cord dysfunction
- Psychogenic

TREATMENT OPTIONS FOR COPD



Existing Pharmacologic Treatment Options in COPD



Non-Pharmacological treatment in COPD

Other Therapeutic Options

- Smoking cessation has the greatest capacity to influence the natural history of COPD
- Nicotine replacement reliably increase long-term smoking abstinence rates.
- Influenza and pneumococcal vaccination should be offered depending on local guidelines.
- Alpha-1 antitrypsin augmentation therapy: not recommended for patients with COPD that is unrelated to the genetic deficiency.
- Mucolytics: Patients with viscid sputum may benefit from Mucolytics; overall benefits are very small.

Oxygen Therapy

Chronic long term Oxygen therapy at home

LTOT

- Improved prognosis
- Increased exercise tolerance
- Decreased hematocrit
- Reduced pulmonary hypertension

Oxygen Therapy

- Long-term oxygen therapy is generally introduced in Stage IV: for patients who have:
  - $PaO_2 < 55$ mm Hg or $SaO_2 < 88\%$ or
  - PaO_2 55-60 mm Hg or SaO_2 of 88-90%, if there is evidence of
    - Pulmonary hypertension,
    - Peripheral edema suggesting cor-pulmonale or
    - Polycythemia (hematocrit > 55%) .

- The primary goal is to increase PaO_2 to at least 60 mm Hg and SaO_2 at least 90%. This will preserve vital organ function without respiratory center depression.

Surgical Treatment

Lung volume reduction surgery (LVRS) is more effective among patients with upper-lobe predominant emphysema and low exercise capacity.

In appropriately selected patients with very severe COPD, lung transplantation has been shown to improve quality of life and functional capacity.

COPD Exacerbations

COPD exacerbation is:

"An acute event characterized by a worsening of the patient's respiratory symptoms beyond normal day-to-day variations that leads to a change in medication."

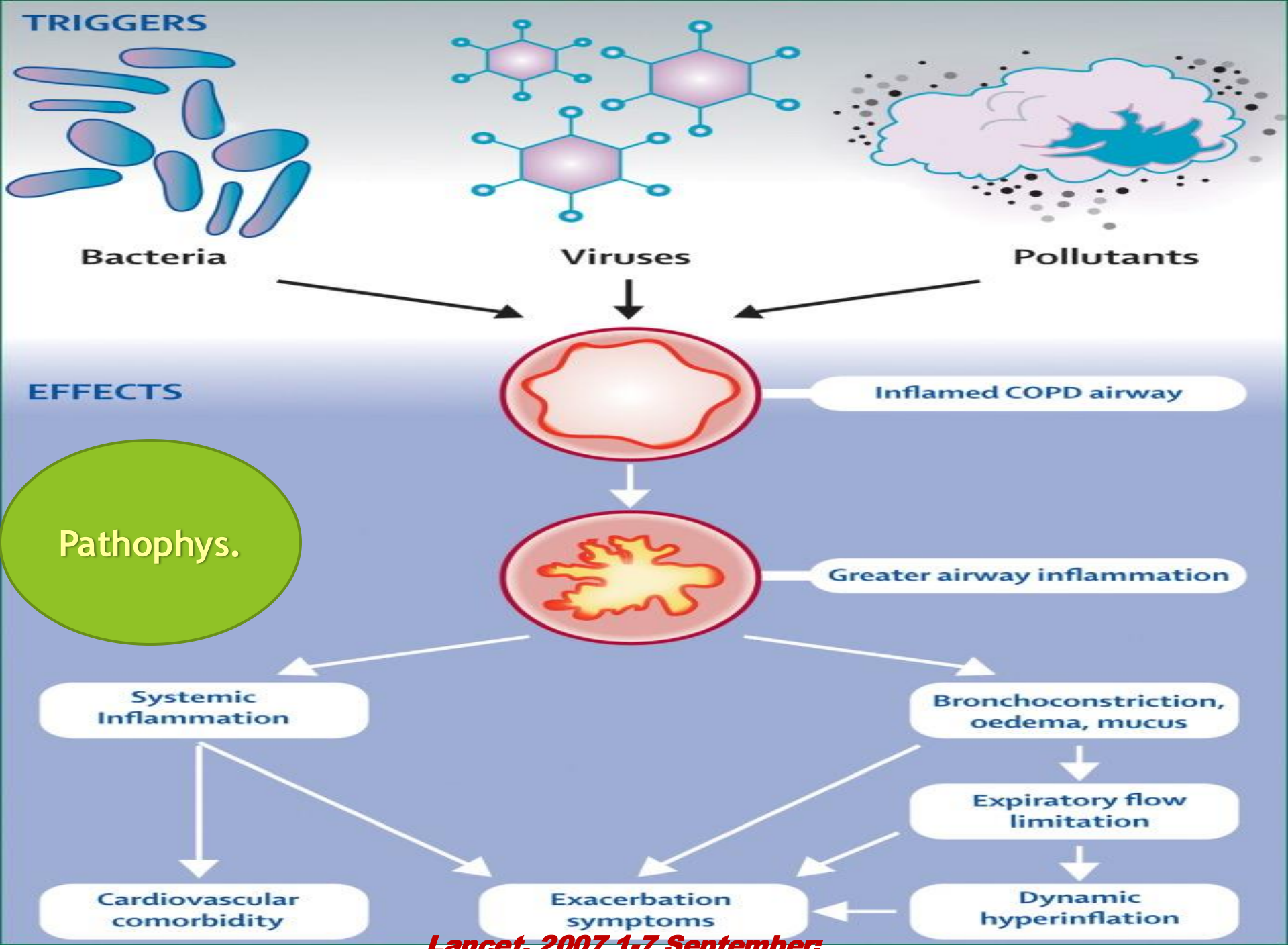
Acute exacerbation of COPD

- Acute exacerbation COPD is defined as acute clinical deterioration in patient's respiratory status

- The cardinal symptoms of COPD exacerbation includes:
  - Worsening dyspnea.
  - Increase in sputum production.
  - Increase in sputum purulence.

Assessing severity of exacerbation

- **Severe exacerbation;** have all three cardinal symptoms of acute exacerbation.
- **Moderate exacerbation;** exhibit two of the cardinal symptoms.
- **Mild exacerbation;** have one of these clinical findings plus at least one of the minor criteria:
  - Upper respiratory tract infection in the past 5 days.
  - Fever without other apparent cause.
  - Increased wheezing.
  - Increased cough.
  - Increase in respiratory rate or heart rate by 20 %.



Pathophys.

Lancet. 2007 1-7 September;

Acute exacerbation of COPD

Causes

Most common bacterial and viral pathogens isolated from patients with COPD exacerbations

Bacteria

- ✓ Haemophiles influenzae
- ✓ Moraxella catarrhalis
- ✓ Streptococcus pneumoniae
- ✓ Pseudomonas aeruginosa

Viruses

- ✓ Rhinovirus
- ✓ Coronavirus
- ✓ Influenza
- ✓ Parainfluenza
- ✓ Adenovirus
- ✓ Respiratory syncytial virus

Manage Exacerbations

- Oxygen: titrate to improve the patient's hypoxemia with a target saturation of 88-92% (controlled O2 therapy).
- Bronchodilators: Short-acting inhaled beta$_2$-agonists with short-acting anticholinergics are preferred.

- Systemic Corticosteroids: A dose of 30-40 mg Prednisolone daily for 7 days is recommended to:
  - Shorten recovery time,
  - Improve lung function (FEV_1) and hypoxemia
  - ↓ Risk of early relapse, treatment failure,

Manage Exacerbations

- Antibiotics should be given to patients with:
  - Three cardinal symptoms: increased dyspnea, increased sputum volume, and increased sputum purulence.

- Aminophylline: Intravenous infusion every 8 hours to be transformed into oral long acting preparation after improvement.

Hospital management:

Indications:

- **Failure of response to outpatient management and still dyspnic (Inability to walk between rooms. Or Inability to eat or sleep.**
- **Co-morbid condition e.g pneumonia or embolism.**
- **Altered mentation.**
- **Worsening hypoxaemia or worsening hypercapnia**

Intensive care unit (ICU) management:

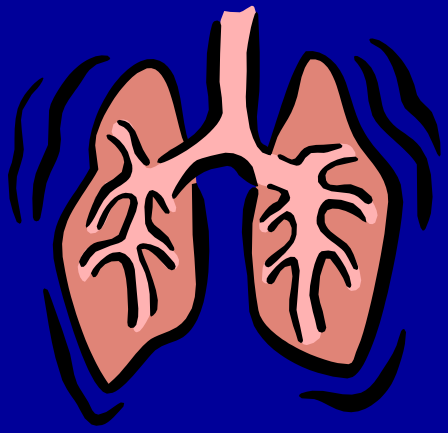
Any of the followings:

- Confusion, or respiratory muscle fatigue.

- Persistent or worsening hypoxaemia despite supplemental O_2 or severe worsening of respiratory acidosis (PH ≤ 7.30).

- Required assisted M.V.

Questions???




Thank you