

Chronic Obstructive Pulmonary Disease (COPD)

Definition:

COPD is preventable & treatable disease characterized by airflow limitation that is not fully reversible.

The airflow limitation is usually progressive, associated with abnormal inflammatory response of the lung to noxious particles or gases associated with systemic manifestation.

Exacerbations and comorbidities contribute to the overall severity in individual patients.

Definition:

COPD is a term that applies to those patients with chronic bronchitis, or anatomical emphysema, with or without asthma, who have persistent obstruction of bronchial airflow.

Chronic bronchitis :

is defined clinically as productive cough every day, or most days for 3 months, two successive years or 2 months for 3 successive years .

What is chronic bronchitis?

Chronic bronchitis involves inflammation and swelling of the lining of the airways that leads to narrowing and obstruction of the airways.

The inflammation also stimulates production of mucous (sputum), which can cause further obstruction of the airways.

Obstruction of the airways, especially with mucus, increases the likelihood of bacterial lung infections.

Etiology and Pathogenesis of COPD:

Host factor

- 1.Hereditry**
- 2.Allergy**
- 3.Aging**

Exposure

- 1.Smoking**
- 2.Occupation**
- 3.Air pollution**
- 4.Infection**

Cigarettes contain many hazardous substances that damage the lungs when inhaled



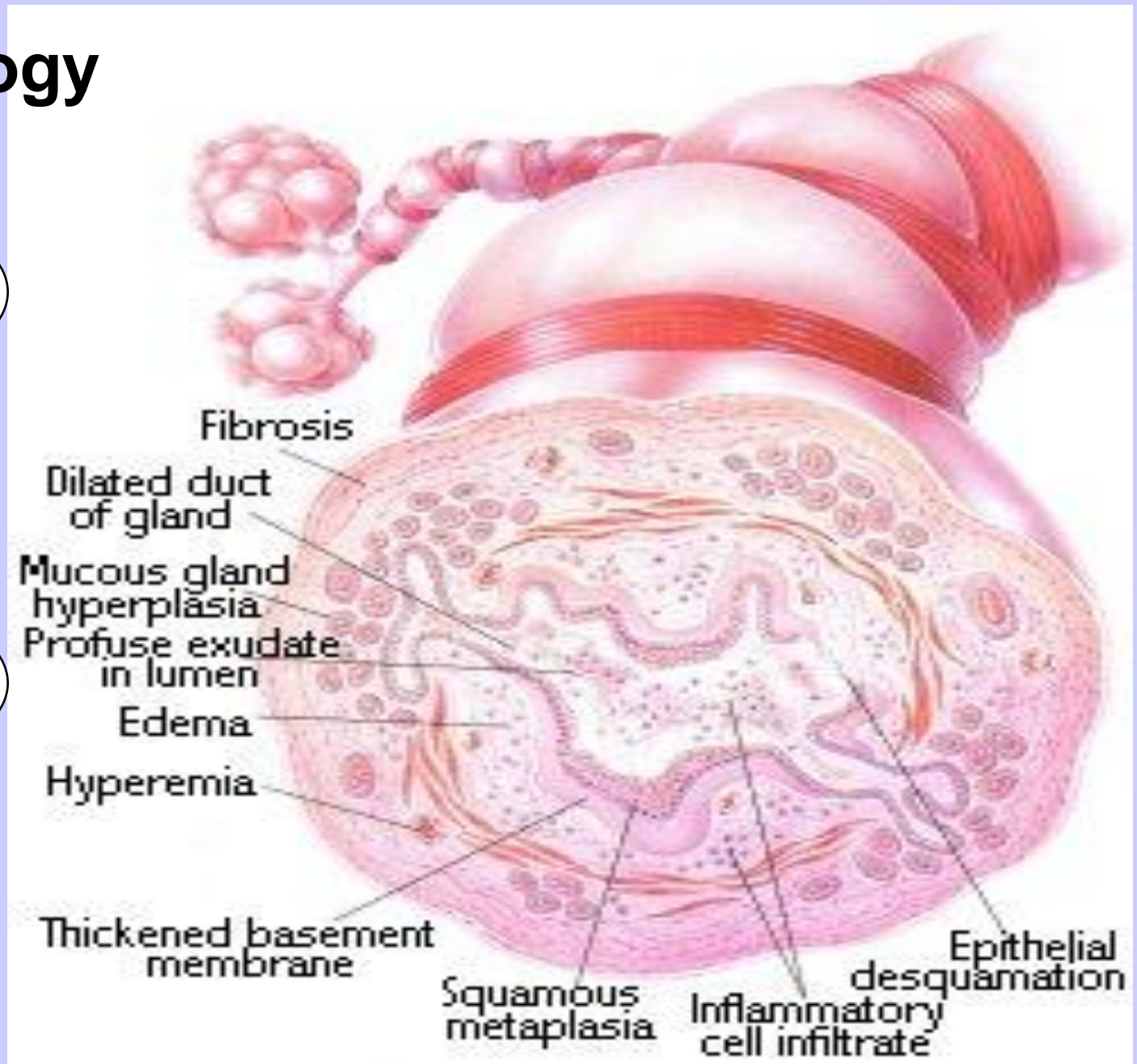
Others predisposing factors:

1. Pulmonary congestion
2. Sarcoidosis
3. Kyphoscoliosis

Pathology

Reversible

Irreversible



Pathology

1. Chronic simple bronchitis
2. Chronic mucopurulent bronchitis
3. Chronic obstructive bronchitis

Asthma

is a pulmonary disease in which there is obstruction to the flow of air out of the lungs, but unlike chronic bronchitis and emphysema, the obstruction in asthma usually is reversible. Between "attacks" of asthma the flow of air through the airways usually is good.

Inflammation in Asthma & COPD

Asthma

sensitizing agent

COPD

Noxious agent

Asthmatic

airway inflammation

CD₄ T lymphocyte &
eosinophil

COPD

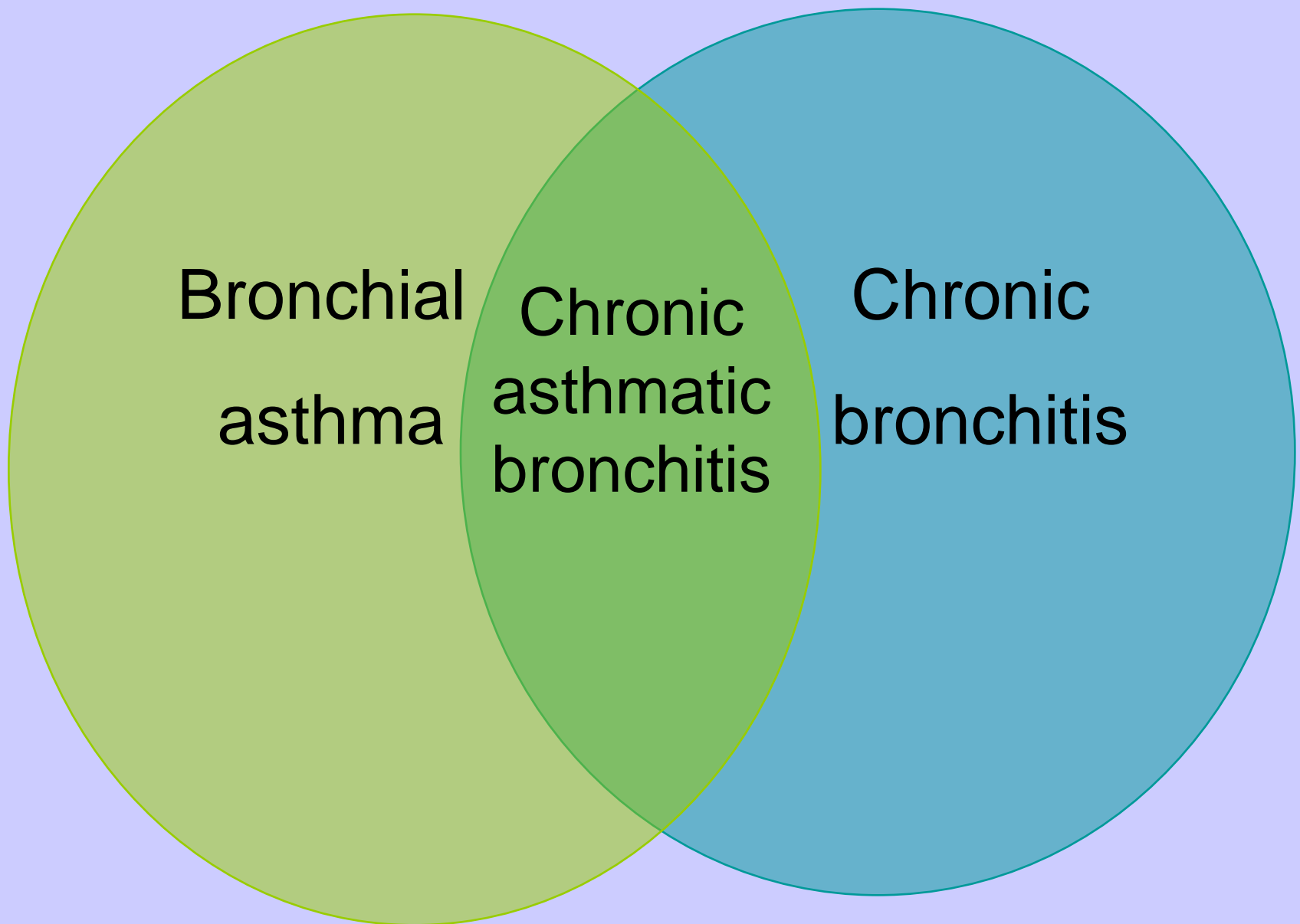
airway inflammation

CD₈ T lymphocyte &
macrophages & neutrophil

Airflow
limitation

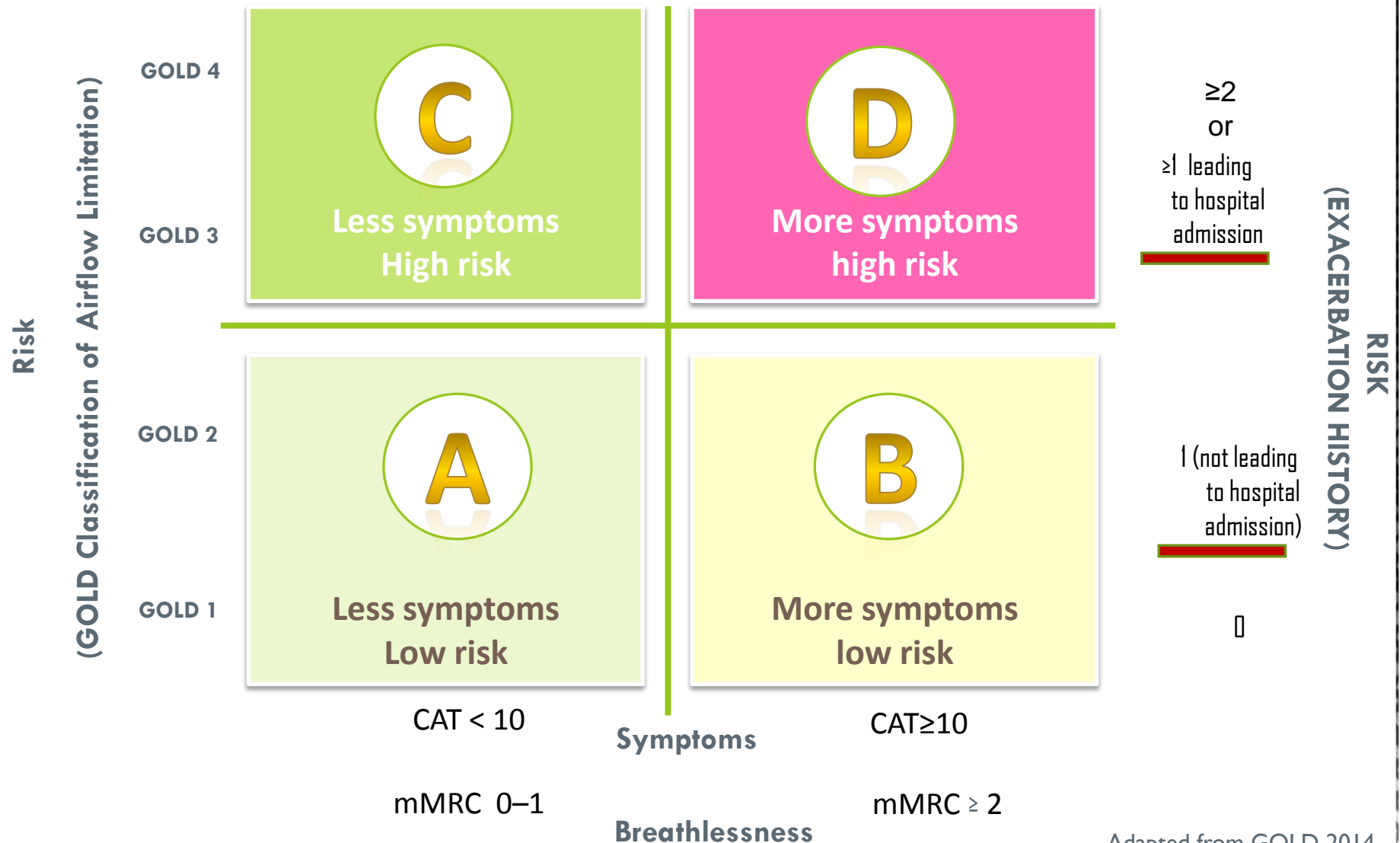
Reversible

Irreversible



GOLD 2014

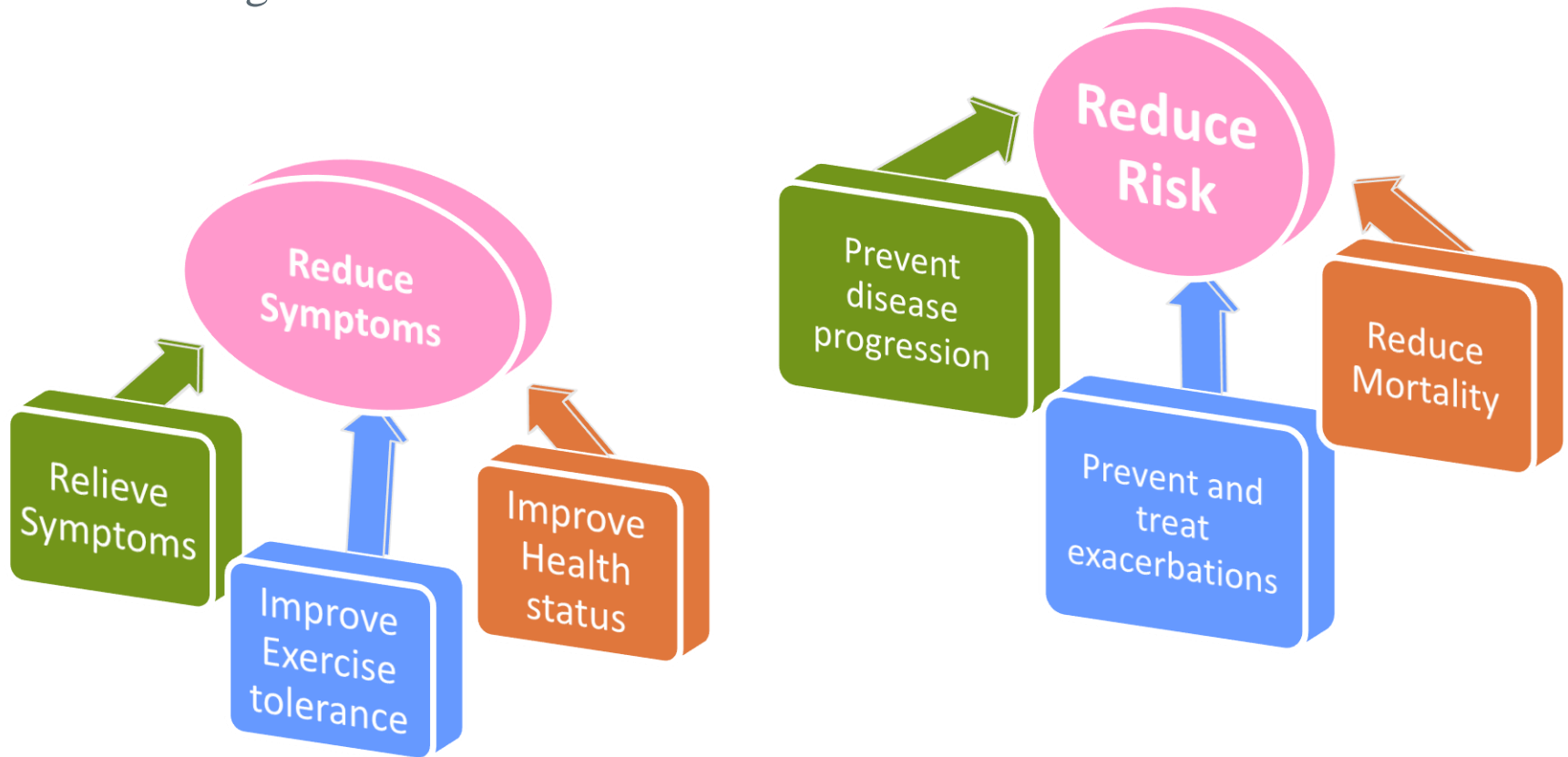
Combined assessment of COPD



Adapted from GOLD 2014

COPD Management:

- Disease Management should now be focusing on 2 key areas
 1. Reducing Symptoms
 2. Reducing Risk



Emphysema

A pathological term indicating enlargement of the air spaces distal to terminal bronchioles due to dilatation and / or destruction of the alveolar walls

Types:

False emphysema

True emphysema

Types:

False pulmonary Emphysema

- 1. Senile**
- 2. Compensatory**
- 3. Localized**
- 4. Unilateral (Macleod's syndrome)**

True pulmonary emphysema

- 1. Primary (type A) :**
- 2. Secondary (type B) :**
 - *Chronic obstructive bronchitis**
 - *Bronchial asthma**
 - *Others**

The airways and air sacs in the lung are normally elastic—that is, they try to spring back to their original shape after being stretched or filled with air, just the way a new rubber band or balloon would. This elastic quality helps retain the normal structure of the lung and helps to move the air quickly in and out.

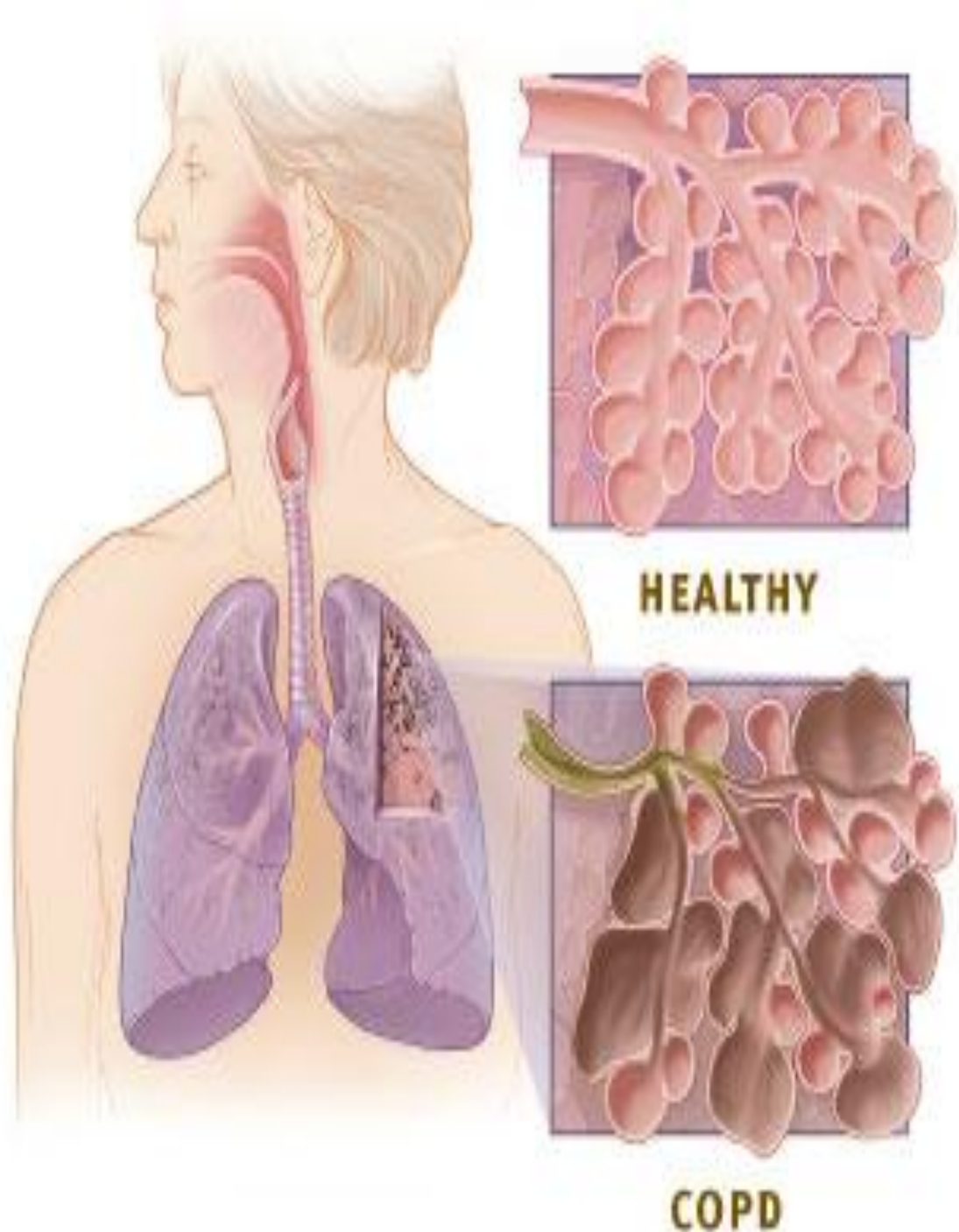
In COPD, much of the elastic quality is gone, and the airways and air sacs no longer bounce back to their original shape. This means that the airways collapse, like a floppy hose, and the air sacs tend to stay inflated. The floppy airways obstruct the airflow out of the lungs, leading to an abnormal increase in the lungs' size. In addition, the airways may become inflamed and thickened, and mucus-producing cells produce more mucus, further contributing to the difficulty of getting air out of the lungs.

1-The airways and air sacs lose their elasticity (like an old rubber band).

2-The walls between many of the air sacs are destroyed.

3-The walls of the airways become thick and inflamed (swollen).

4-Cells in the airways make more mucus (sputum) than usual, which tends to clog the airways.



What is emphysema?

1. There is **permanent** enlargement of the alveoli due to the destruction of the walls between alveoli in emphysema.
2. The destruction of the alveolar walls **reduces the elasticity** of the lung overall.
3. Loss of elasticity leads to the collapse of the bronchioles, **obstructing airflow out** of the alveoli.
4. Air becomes "**trapped**" in the alveoli and reduces the ability of the lung to shrink during exhalation.

5. The reduced expansion of the lung during the next breath reduces the amount of **air that is inhaled**.
6. As a result, **less air for the exchange** of gasses gets into the lungs.
7. This trapped air also can **compress adjacent** less damaged lung tissue, preventing it from functioning to its fullest capacity.

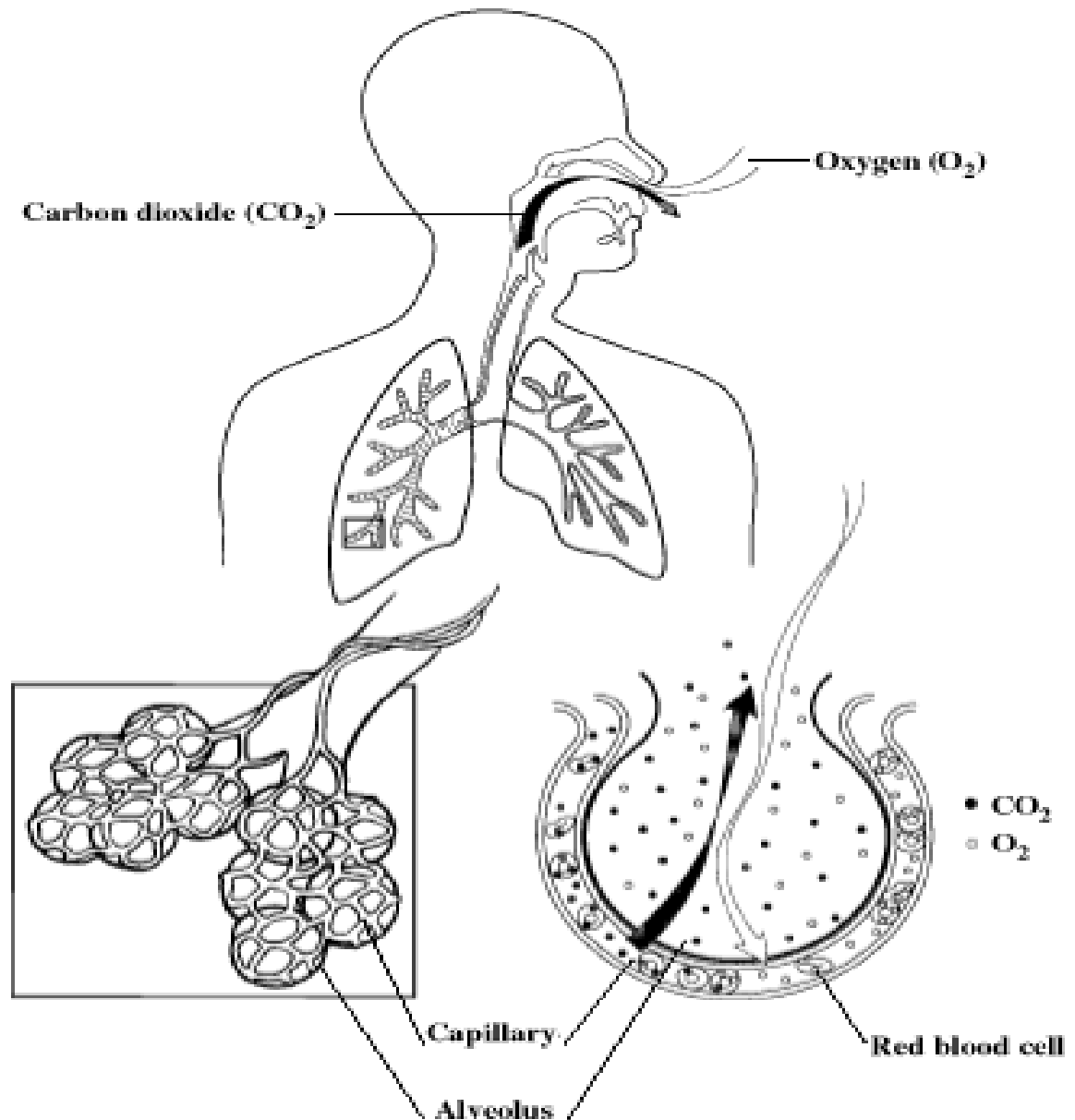
Pathophysiology

Respiratory abnormalities.

Cardiovascular abnormalities.

Respiratory abnormalities.

1. Disturbed Ventilation
2. Disturbed diffusion
3. Disturbed perfusion



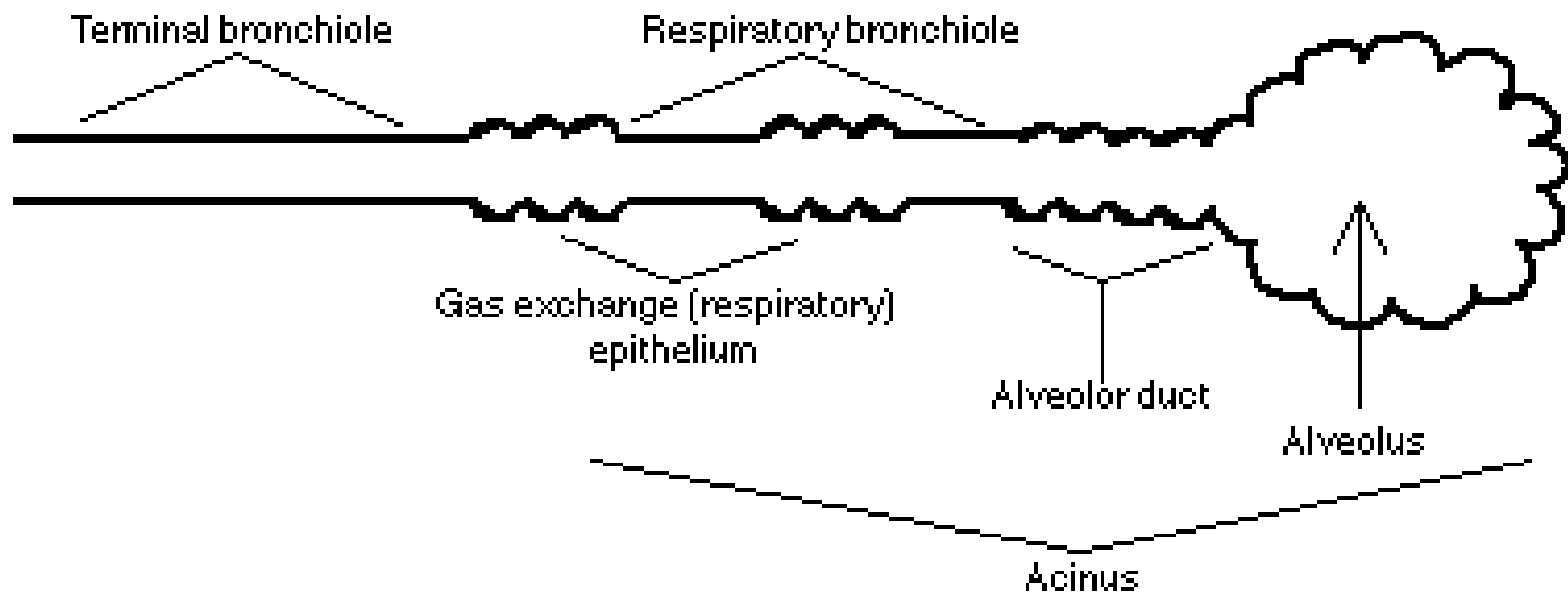
Transfer of oxygen of inhaled air into the blood and of waste carbon dioxide of blood into the lungs occur in the alveolus.

Cardiovascular abnormalities.

1. Pulmonary Hypertension

2. Hypoxic cor pulmonale

3. Left ventricular failure



* There are three orders of respiratory bronchioles, each with an increasing amount of respiratory epithelium.

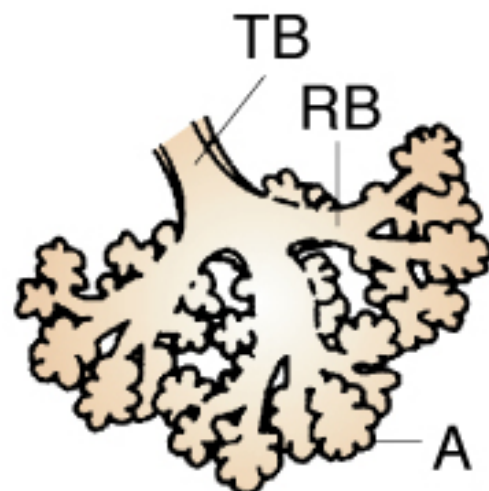
Alveoli

Alveolar Ducts

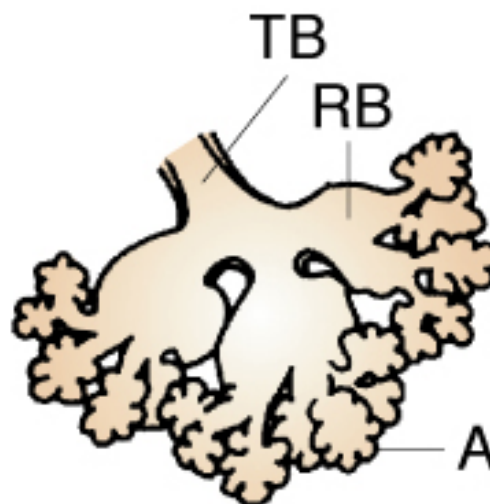
**Respiratory
Bronchioles**



Normal



Centriacinar



Panacinar

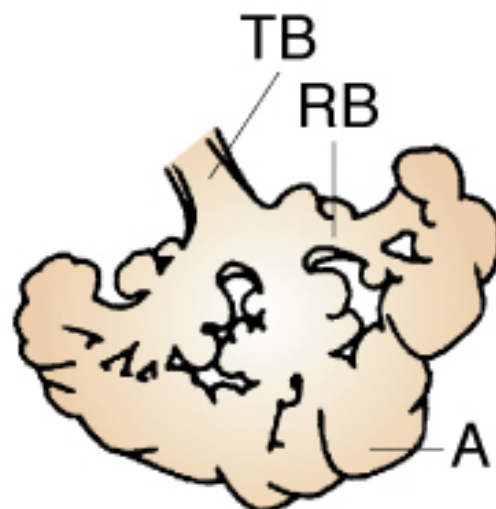
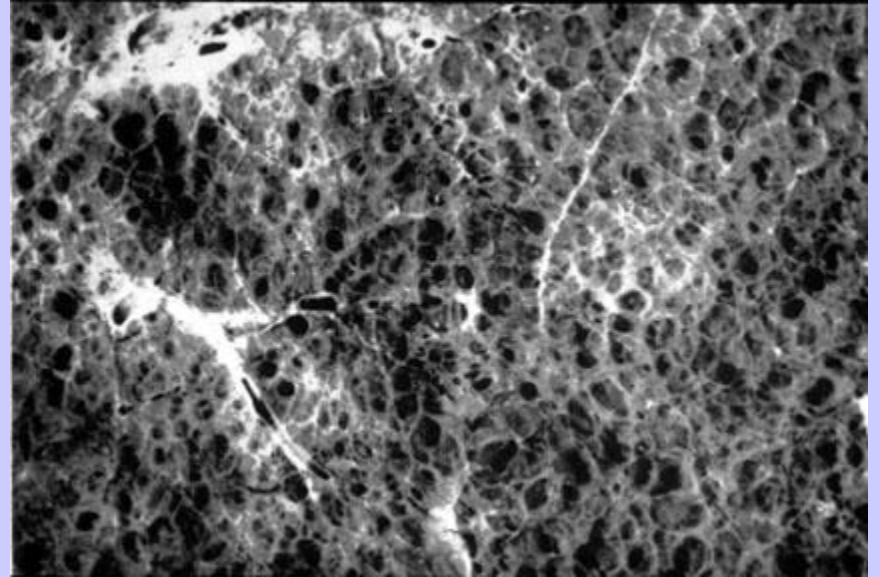


Figure 31-11 Centriacinar and panacinar emphysema. In centriacinar emphysema, the destruction is confined to the terminal (TB) and respiratory bronchioles (RB). In panacinar emphysema, the peripheral alveoli (A) are also involved. (West J.B. [1997]. *Pulmonary pathophysiology* [5th ed., p. 53]. Philadelphia: Lippincott-Raven)



PAE



CLE

Pathology :

Panacinar

Type A

Emphysema is the dominant

The respiratory center is more sensitive due to absence of hypercapnia

this result in severe hypoxia make the patients , keeping pCO_2 near normal.

(puffering)

Centrilobular

Type B

Bronchitis is the dominant

The respiratory center is less sensitive due to presence of hypercapnia this result in absence of severe dyspnea

Hypoxia results in cor pulmonale RVF.

cyanosis (Blue) & edematous (Bloater)

Classification

	Type A Pink Puffer	Type B Blue bloater
Clinical: Dyspnea Sputum Weight Cyanosis Cor pulmonale Chest ex.	More marked Scanty mucoid Decreased Mild or absent Late hyperinflation	Less marked Copious, purulent Normal or decreased Marked Comon Increase airway resistance
Chest X ray	hyperinflation	Exaggerated bronchovascular marking
Respiratory function test: RV Diffusion PO2 PCO2	Marked increase Marked decrease Slight decrease Normal or low	Moderate increase Variable Marked decrease High



What are the signs and symptoms of COPD?

The signs and symptoms of chronic obstructive pulmonary disease (COPD) include :

1. Cough
 2. Sputum (mucus) production
 3. Shortness of breath, especially with exercise
 4. Wheezing (a whistling or squeaky sound when you breathe)
- Chest tightness

A cough that doesn't go away and coughing up lots of mucus are common signs of COPD. These often occur years before the flow of air in and out of the lungs is reduced. However, not everyone with a cough and sputum production goes on to develop COPD, and not everyone with COPD has a cough .

The severity of the symptoms depends on how much of the lung has been destroyed. If you continue to smoke, the lung destruction is faster than if you stop smoking.

Signs

1.Vital signs

Pulse , RR

2.Head examination:

Eye, Cyanosis

3.Neck examination

4.Upper limb

5.Lower limb

6.Abdominal examination

7.Local chest examination :

Inspection

Palpation

Percussion

Auscultation



An exacerbation of COPD is:

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

Assessment of COPD

Assess risk of exacerbations

- ***History of exacerbations** (Two or more exacerbations in the preceding year).
- ***Spirometry** (GOLD 3 or GOLD 4 categories).

Complication Of COPD:

1. Respiratory :

- Failure
- Infection
- Bronchial obstruction & collapse & bronchiectasis
- Pneumothorax

2.CVS:

- Cor pulmonale
- LVF
- Embolism

3.Polythemia

4.Edema

5.Proteinuria& nephrotic syndrome

6. Peptic ulcer

7. Complication of chronic cough

Differential diagnosis

1. Asthma
2. Congestive heart failure
3. Bronchiectasis
4. Pulmonary tuberculosis

Investigation:

1- x ray chest

Barrel shaped chest



Respiratory function tests:

Ventilation

Diffusion

Perfusion



Assessment of COPD

Assess degree of airflow limitation (Spirometry)

Spirometric classification of airflow limitation (in patients with $FEV_1/FVC < 0.70$)

- *GOLD 1** (Mild; $FEV_1 \geq 80\%$ predicted)
- *GOLD 2** (Moderate; $50\% \leq FEV_1 < 80\%$ predicted)
- *GOLD 3** (Severe; $30\% \leq FEV_1 < 50\%$ predicted)
- *GOLD 4*** (Very severe; $FEV_1 < 30\%$ predicted)

III- Arterial blood gases : O_2 & CO_2

IV- Sputum examination:

V- CBC

VI- ECG & Echocardiography

VII- CT chest

**VIII- Alpha 1 antitrypsin
measurement**

Treatment:

1- Prophylaxis

Quit smoking

Avoid pollution

Proper ttt of respiratory infection:
antibiotic, mucolytic & hydration

2- Non Pharmacological treatment: -

- Smoking cessation

- Physical activity &

- Flu/Pneumococcal vaccination

- Oxygen therapy: for $pO_2 \leq 55$ mm Hg or saturation $< 88\%$, \pm hypercapnia **Or** $pO_2 = 55 - 60$ mm Hg with pulmonary hypertension, peripheral edema suggesting heart failure, or polycythemia via **mask, nasal canula, tent.**

- Nutrition: carbohydrates, Proteins , Anabolic hormones

-Surgical treatment: bullectomy , lung volume reduction

-Pulmonary rehabilitation: Breathing techniques & Clearing of secretions

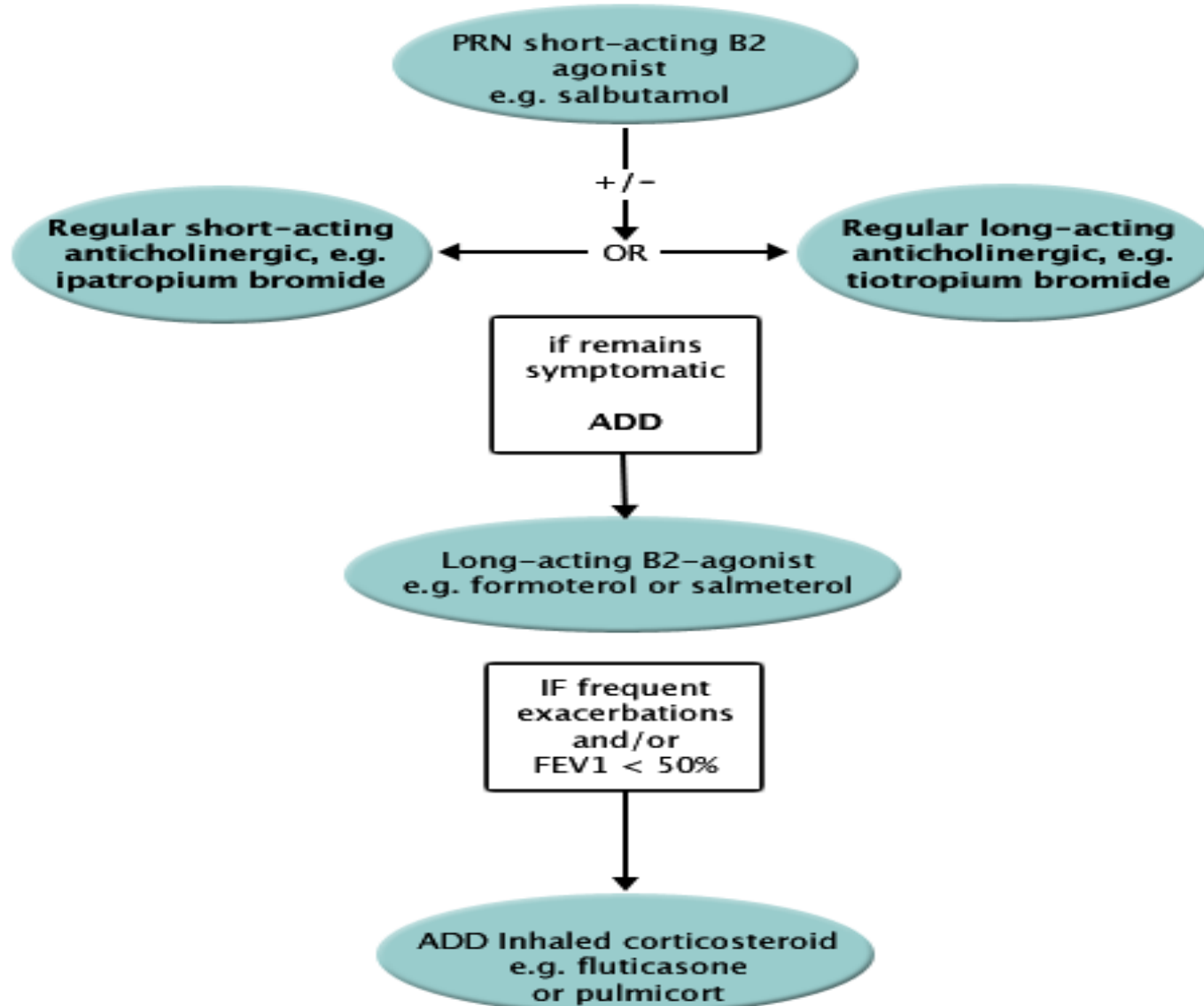
Mechanical ventilation : non invasive(CPAP& BiPAP) or invasive

-Treatment of complication:Respiratory failure, Heart failure, Polycythemia

3- Pharmacological treatment:

- Control of respiratory infection: *Antibiotics*
- Bronchodilators* : beta₂-agonists with or without anticholinergics.
- Methyl-xanthine
- Systemic Corticosteroids*

Pharmacologic Management of COPD



Thank you