# COPD

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BASICS

PREVENTION

DIAGNOSIS

TREATMENT

# Definition

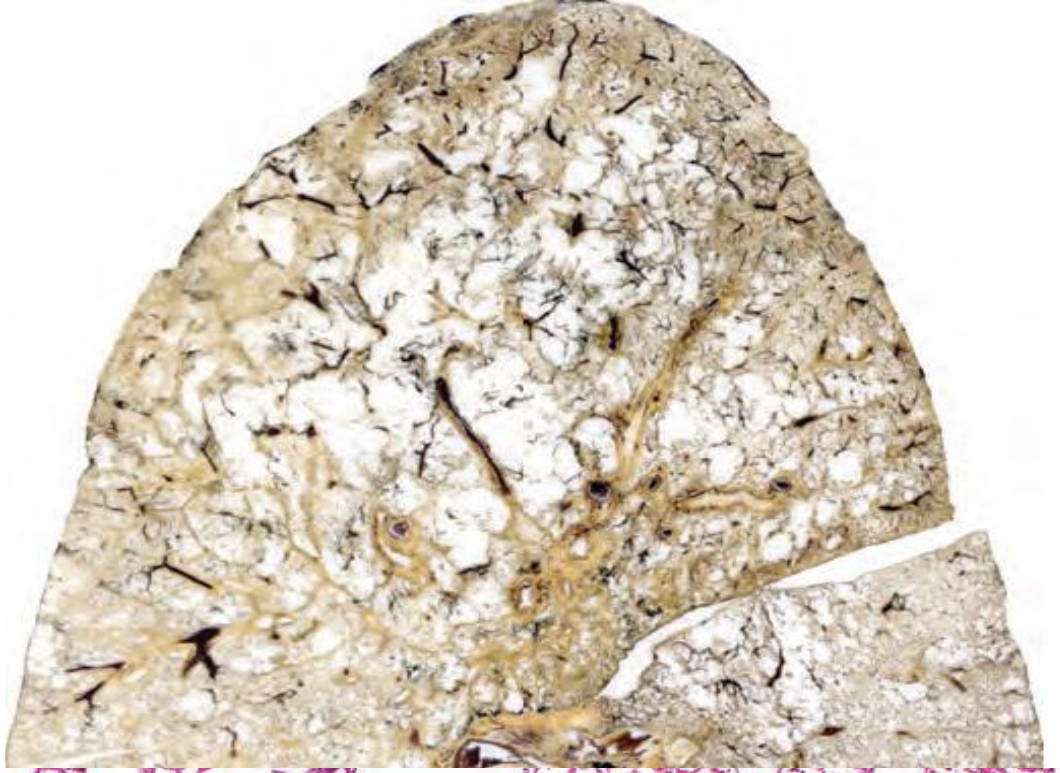
- is a **common**, **preventable** and **treatable** disease.

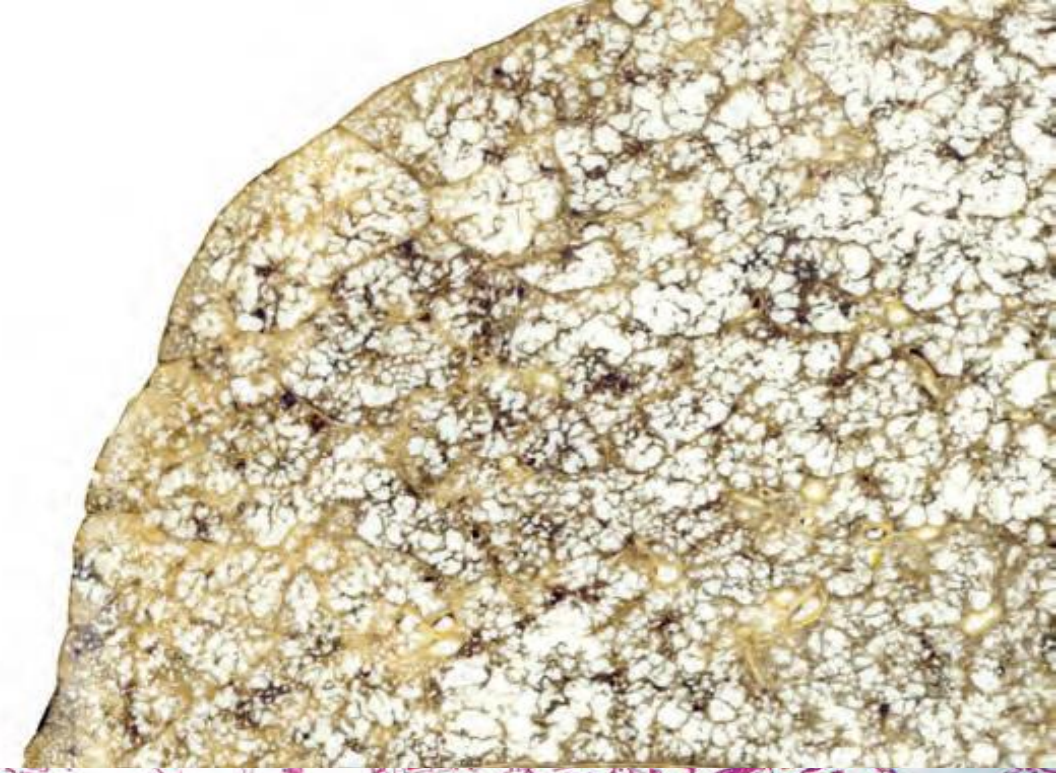
- That is characterized by **persistent** respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually **caused** by significant exposure to noxious particles or gases.

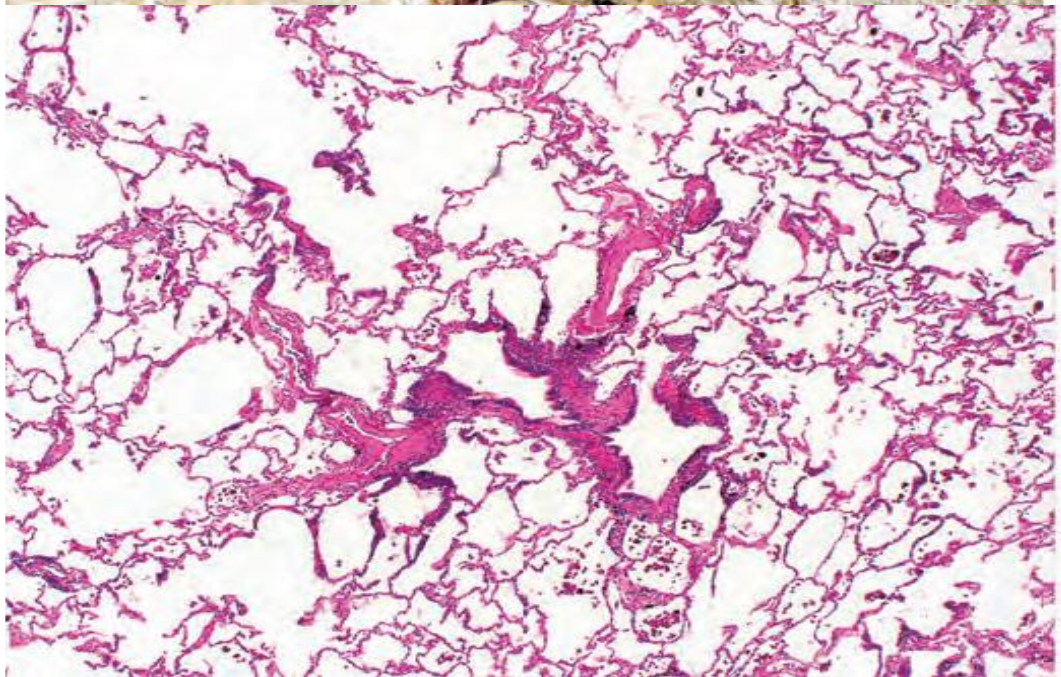
- The chronic airflow limitation that is characteristic of COPD is caused by a mixture of **small airways disease** (e.g., obstructive bronchiolitis) and **parenchymal destruction** (emphysema), the relative contributions of which **vary** from person to person.

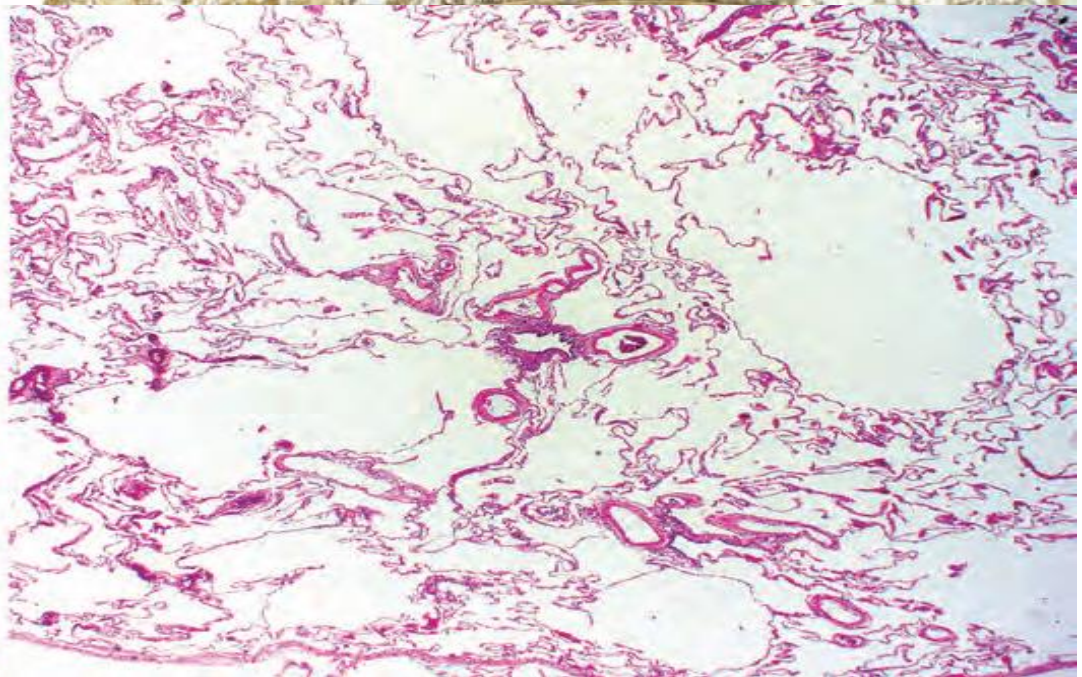
- **Chronic bronchitis** is defined in clinical terms as the presence of cough and sputum production for most days over 3 months for 2 consecutive years.

- **Emphysema** is defined as enlargement of the airspaces distal to the terminal bronchioles, due to destruction of the alveolar walls









# Epidemiology

- Represents an important public health challenge and is a major cause of chronic morbidity and mortality throughout the world.
- COPD is currently the **4th leading** cause of death in the world but is projected to be the 3rd leading cause of death by 2020. More than 3 million people died of COPD in 2012 accounting for 6% of all deaths globally.
- COPD burden is projected to increase in coming decades because of continued exposure to COPD risk factors and aging of the population

- more common in **older people**, especially those aged 65 years and older.

- The Burden of Obstructive Lung Disease (BOLD) Initiative estimates a worldwide population prevalence of COPD for stages II or higher as equivalent to **10.1 ± 4.8%** overall with 11.8 ± 7.9% for men and 8.5 ± 5.8% for women.

- Its associated mortality in **women** has more than doubled over the past 20 years and **now matches that in men**.

The risk of developing COPD is related to the following factors

- **Tobacco smoke**

including **cigarette**, pipe, cigar, **water pipe** and other types of tobacco smoking popular in many countries, as well as environmental tobacco smoke (ETS)

- **Indoor air pollution**:

from **biomass fuel** used for **cooking and heating in poorly vented** dwellings, a risk factor that particularly affects women **in developing countries**.

**- Occupational exposures:**

including organic and inorganic dusts, chemical agents and fumes, are under appreciated risk factors for COPD.

**- Outdoor air pollution**

Also contributes to the lungs' total burden of inhaled particles, although it appears to have a **relatively small effect** in causing COPD.

- **Genetic factors**

such as severe hereditary deficiency of **alpha 1 antitrypsin** (AATD).

**- Age and sex**

Aging and female  sex increase COPD risk

- **Lung growth and development**

Any factor that affects lung growth during gestation and childhood (low birth weight, respiratory infections, etc.) has the potential to increase an individual's risk of developing COPD.

**- Socioeconomic status :**

Strong evidence that the risk of developing COPD is **inversely related to socioeconomic** status. It is not clear, however, whether this pattern reflects exposures to indoor and outdoor air pollutants, crowding, poor nutrition, infections, or other factors related to low socioeconomic status

**- Asthma and airway hyper reactivity**

Asthma may be a risk factor for the development of airflow limitation and COPD.

**- Chronic bronchitis**:

May increase the frequency of total and severe exacerbations

**- Infections**

A history of severe childhood respiratory infection has been associated with reduced lung function and increased respiratory symptoms in adulthood

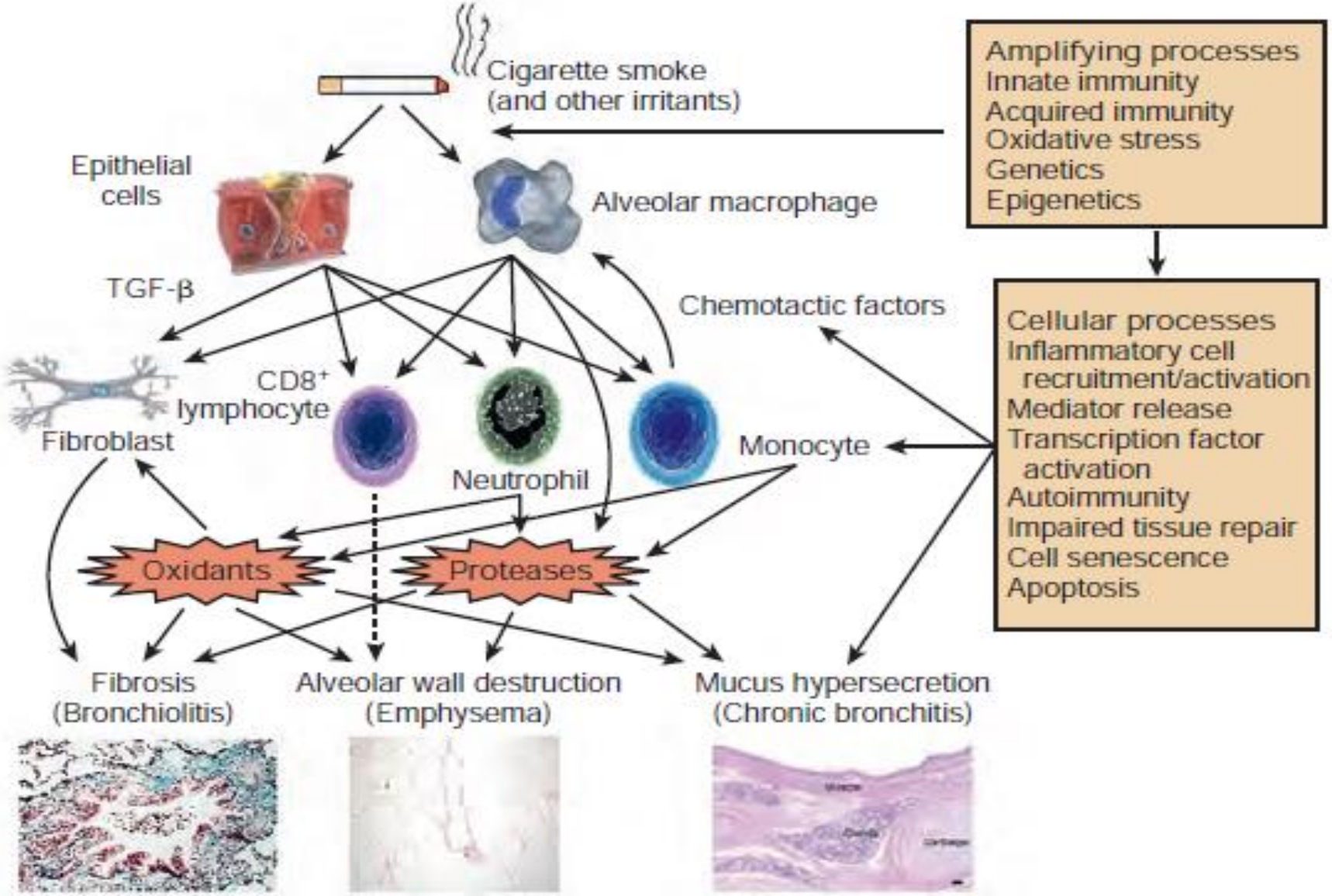



**Figure 43-3 Overview of the pathogenesis of COPD.** Cigarette smoke activates macrophages and epithelial cells to produce chemotactic factors that recruit neutrophils and CD8 cells from the circulation. These cells release factors that activate fibroblasts, resulting in abnormal repair processes and bronchiolar fibrosis. Imbalance between proteases released from neutrophils and macrophages and antiproteases leads to alveolar wall destruction (emphysema). Proteases also cause the release of mucus. An increased oxidant burden resulting from smoke inhalation or release of oxidants from inflammatory leucocytes causes epithelial and other cells to release chemotactic factors, inactivates antiproteases, directly injures alveolar walls, and causes mucus hypersecretion. Several processes are involved in amplifying the inflammatory responses in COPD.

- In emphysema, the final outcome of the inflammatory responses is **elastin breakdown** and subsequent loss of alveolar integrity.

- In chronic bronchitis, these inflammatory changes lead to **ciliary dysfunction and increased goblet cell size and number** which leads to the excessive mucus secretion. These changes are responsible for decreased airflow, hypersecretion, and chronic cough.

- In both conditions, changes are progressive and usually not reversible.

# Primary prevention

- **Avoidance of tobacco exposure** (both active and passive measures) and toxic fumes are of invaluable importance in primary prevention of COPD.
- All smokers should be offered interventions aimed at smoking cessation, including pharmacotherapy and counselling.
- Although smoking cessation may be associated with minor short-term adverse effects such as weight gain and constipation, its long-term benefits are unquestionable.

# Screening

- No data to show conclusively that screening spirometry is effective in directing management decisions or in improving COPD outcomes in patients who are identified before the development of significant symptoms.
- However, if COPD is **diagnosed at an early** stage and risk factors are eliminated, the rate of decline in lung function will dramatically decrease.
- **Screening** can be done by **asking** about smoking history and environmental or occupational exposure. In high-risk populations a screening spirometry should be obtained to document airway obstruction

# Secondary prevention

- **Smoking cessation** has the **greatest** capacity to influence the natural history of COPD.
- Effective resources and time are dedicated to smoking cessation, long term quit **success rates of up to 25%** can be achieved.
- **A five step program** for intervention provides a helpful strategic framework to guide health care providers interested in helping their patients stop smoking

# Brief strategies to help the patient willing to quit (5As)

- **ASK:**

Systematically identify all tobacco users at every visit. Implement an office wide system that ensures that, for EVERY patient at EVERY clinic visit, tobacco use status is queried and documented.

- **ADVISE:**

Strongly urge all tobacco users to quit. In a clear, strong, and personalized manner, urge every tobacco user to quit.

- **ASSESS:**

Determine willingness and rationale of patient's desire to make a quit attempt. Ask every tobacco user if he or she is willing to make a quit attempt at this time (e.g., within the next 30 days).

- **ASSIST:**

Aid the patient in quitting. Help the patient with a quit plan; provide practical counselling ; provide intra treatment social support; help the patient obtain extra treatment social support; recommend use of approved pharmacotherapy except in special circumstances; provide supplementary materials.

- **ARRANGE:**

Schedule follow up contact.

Schedule follow up contact, either in person or via telephone

# Counselling:

Counselling delivered by physicians and other health professionals significantly increases quit rates over self initiated strategies. **Even brief (3minute)** periods of counselling urging a smoker to quit improve smoking cessation rates . There is a relationship between counselling intensity and cessation success.

# Vaccination

- **Influenza vaccine**:

- Can reduce serious illness (such as lower respiratory tract infections requiring hospitalization) and **death i**n COPD patients.

- **Pneumococcal vaccine**:

- The 23valent pneumococcal polysaccharide vaccine (PPSV23) has been shown to reduce the incidence of community acquired pneumonia in COPD patients aged < 65 years with an FEV1 < 40% predicted and in those with comorbidities.

- In the general population of adults ≥ 65 years the 13 valent conjugated pneumococcal vaccine (PCV13) has demonstrated significant efficacy in reducing bacteremia and serious invasive pneumococcal disease

# Diagnosis

- **History**:

- Often asymptomatic

- However, as the disease progresses, dyspnoea, wheezing, cough and sputum production typically become more prominent.

- Patients may also **modify their activities** to avoid dyspnoea so that the progression of pulmonary limitation may be rather insidious.

- As with **dyspnoea**, patients may attribute cough to other factors such as smoking and therefore may not complain about this symptom unless prompted.

- **Sputum**, when present, tends to be mucoid, clear to white in appearance, and more purulent with exacerbations.

# Physical examination

- **Early** in the course of the disease, **no specific** abnormalities may be noted on physical examination.

-**Wheezing** may or may not be present and does not necessarily relate to the severity of airflow obstruction.

- **Prolonged expiratory** time is a more consistent finding in COPD, particularly as the disease progresses.

- In very severe disease, patients develop physical signs indicative of hyperinflation, including a **barrel-shaped chest**, **decreased breath** sounds, **distant heart sounds**, and **increased resonance** to percussion.

- Patients may breathe in a “**tripod” position** in which the individual learns forward and supports his or her upper body with extended arms.

- Patients with severe disease may also use **pursed-lip breathing**, which involves exhaling through tightly pressed, pursed lips.

- With severe disease, other systemic manifestations may include signs of **cor pulmonale.**

**- Tar stains** on the fingers from cigarette smoking may be present.

Two commonly recognized COPD subtypes are the “pink puffers” and “blue bloaters.”

- **Pink puffers**, typically associated with significant **emphysema**, compensate by hyperventilation and often manifest muscle wasting and weight loss. Compared with blue bloaters, pink puffers are less hypoxemic and therefore appear “pink.”

- **Blue bloaters** typically have chronic bronchitis and tend to have decreased ventilation and greater *ventilation-perfusion* (V/Q) mismatch than pink puffers, leading to hypoxemia and hence **cyanosis** and to cor pulmonale with edema or “bloating.”

# Assessment

Your name:

Today's date:

CAT
COPD Assessment Test™

## How is your COPD? Take the COPD Assessment Test™ (CAT)

This questionnaire will help you and your healthcare professional measure the impact COPD (Chronic Obstructive Pulmonary Disease) is having on your wellbeing and daily life. Your answers, and test score, can be used by you and your healthcare professional to help improve the management of your COPD and get the greatest benefit from treatment.

For each item below, place a mark (X) in the box that best describes you currently. Be sure to only select one response for each question.

**Example:** I am very happy 0 X 2 3 4 5 I am very sad

| | | | SCORE |
|---|---|---|---|
| I never cough | 0 1 2 3 4 5 | I cough all the time | |
| I have no phlegm (mucus) in my chest at all | 0 1 2 3 4 5 | My chest is completely full of phlegm (mucus) | |
| My chest does not feel tight at all | 0 1 2 3 4 5 | My chest feels very tight | |
| When I walk up a hill or one flight of stairs I am not breathless | 0 1 2 3 4 5 | When I walk up a hill or one flight of stairs I am very breathless | |
| I am not limited doing any activities at home | 0 1 2 3 4 5 | I am very limited doing activities at home | |
| I am confident leaving my home despite my lung condition | 0 1 2 3 4 5 | I am not at all confident leaving my home because of my lung condition | |
| I sleep soundly | 0 1 2 3 4 5 | I don't sleep soundly because of my lung condition | |
| I have lots of energy | 0 1 2 3 4 5 | I have no energy at all | |
| | | TOTAL SCORE | |

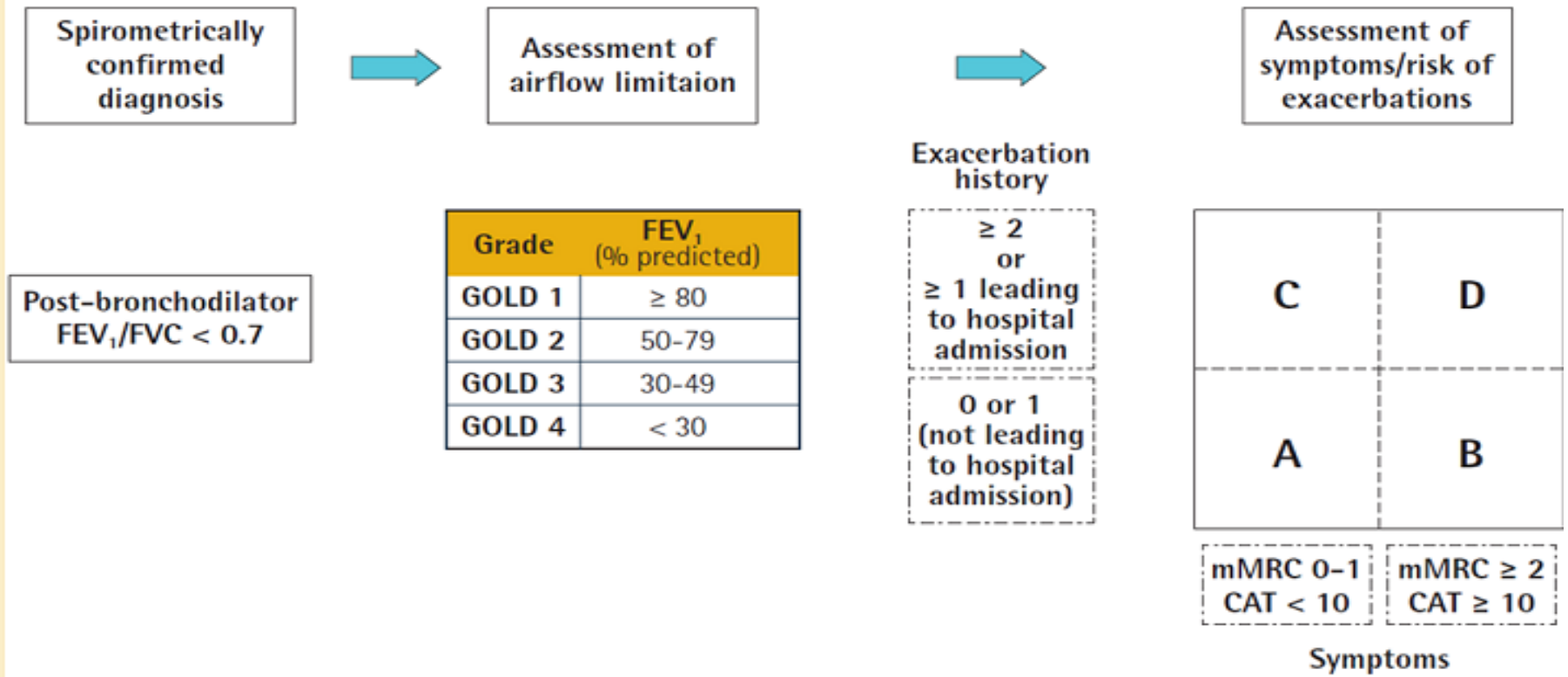
**Table 44-2** GOLD Classification of Severity of Airflow Limitation in COPD, Based on Post-Bronchodilator $FEV_1$

| 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 |


**The Modified Medical Research Council (MMRC) Dyspnoea Scale**

| Grade of dyspnoea | Description |
|---|---|
| 0 | Not troubled by breathlessness except on strenuous exercise |
| 1 | Shortness of breath when hurrying on the level *or* walking up a slight hill |
| 2 | Walks slower than people of the same age on the level because of breathlessness *or* has to stop for breath when walking at own pace on the level |
| 3 | Stops for breath after walking about 100 m *or* after a few minutes on the level |
| 4 | Too breathless to leave the house *or* breathless when dressing or undressing |

# THE REFINED ABCD ASSESSMENT TOOL



# ABCD Assessment Tool

## Example

- Consider two patients:
  - Both patients with $FEV_1$ < 30% of predicted
  - Both with CAT scores of 18
  - But, one with **0 exacerbations** in the past year and the other with **3 exacerbations** in the past year.

- Both would have been labelled **GOLD D** in the prior classification scheme.
- With the new proposed scheme, the subject with 3 exacerbations in the past year would be labelled **GOLD grade 4, group D**.
- The other patient, who has had no exacerbations, would be classified as **GOLD grade 4, group B**.

# Spirometry

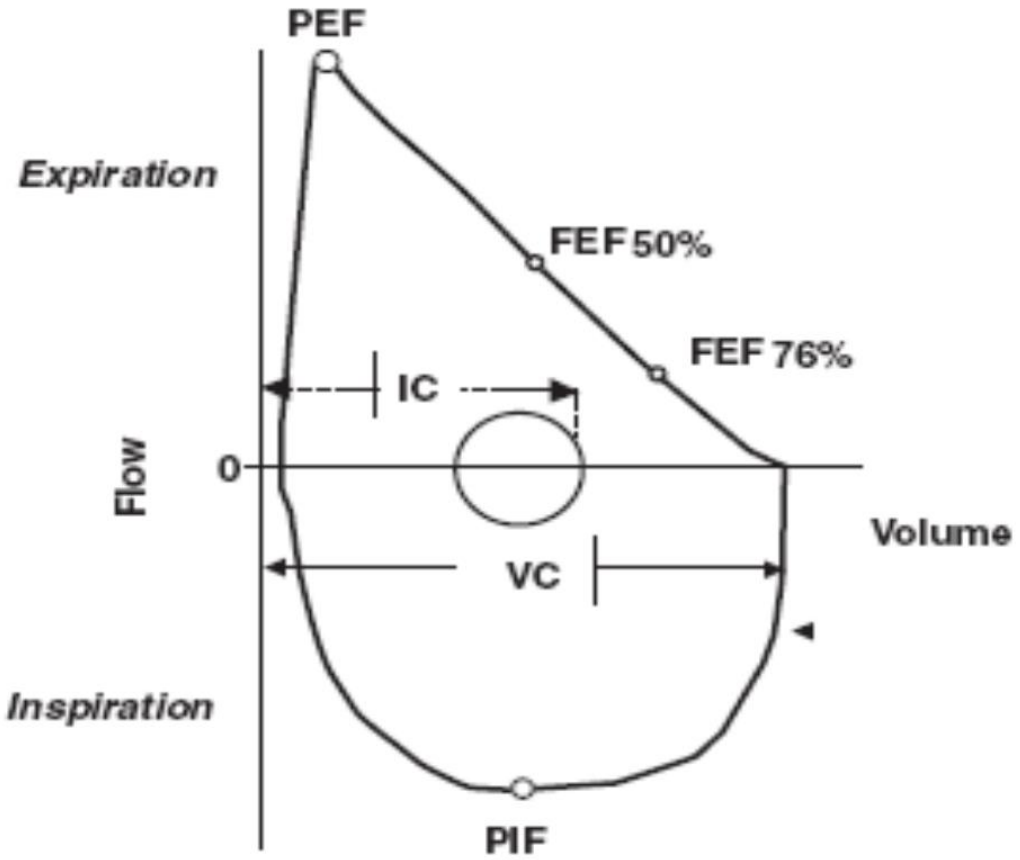
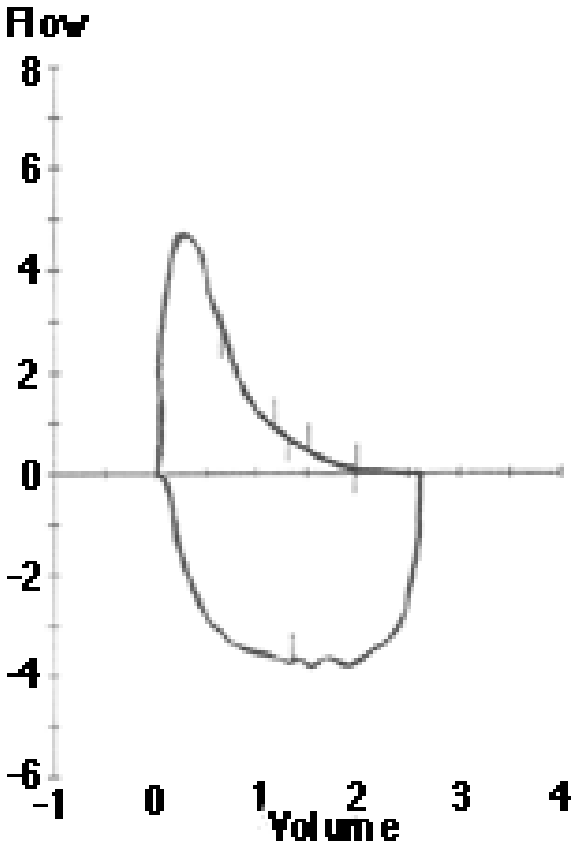
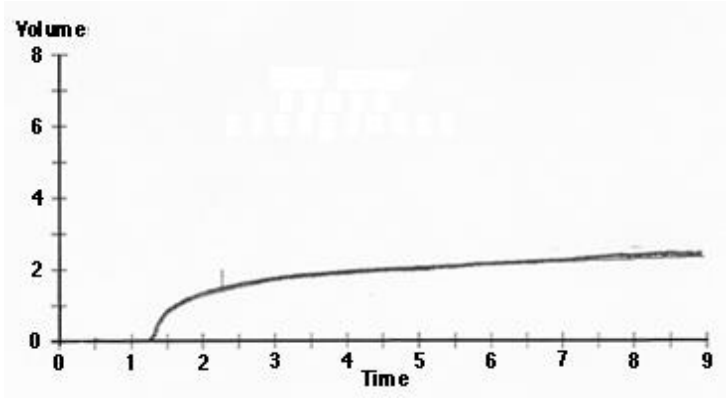
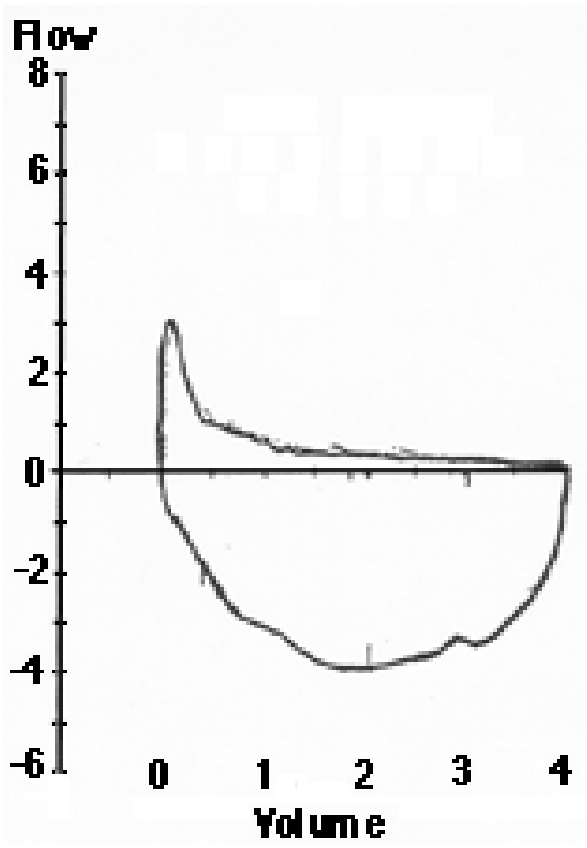



**Figure 3** Flow volume curve for a normal subject showing the principal measures used.



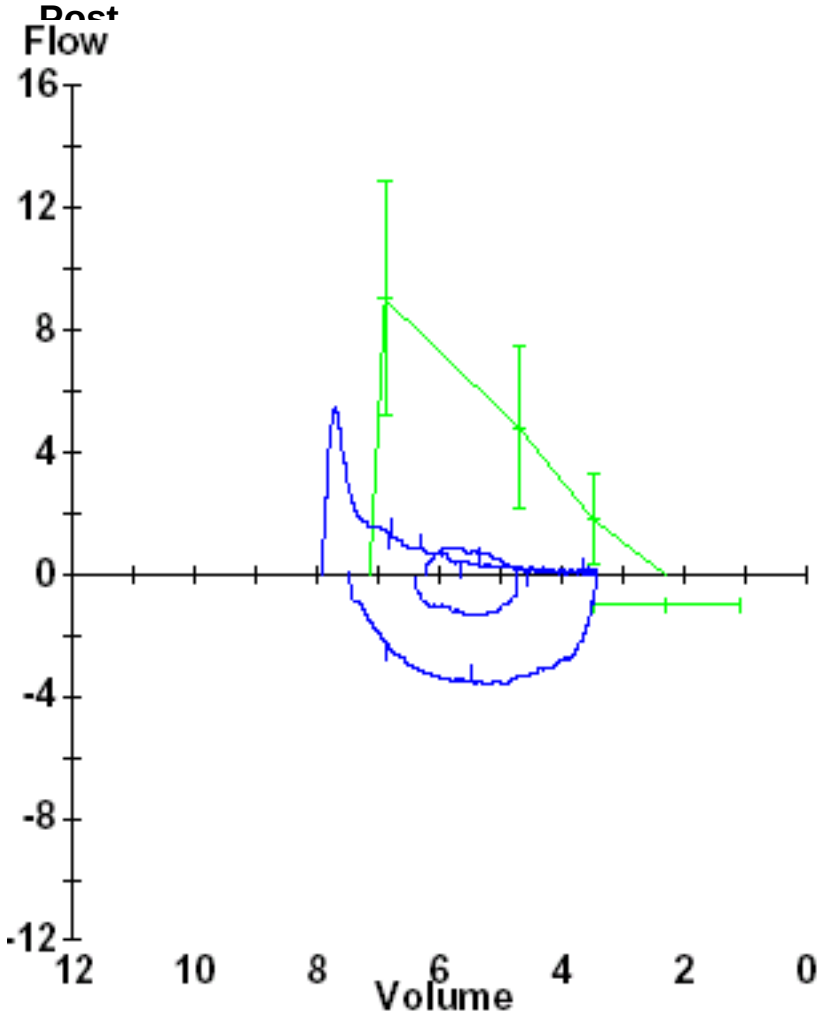

Airflow obstruction

Mild on left

Severe on right

ID: CSM4166 Date: 10/03/04 Gender: Male Age: 62
Weight(kg): 79.0 Height(cm): 184 BMI: 23.33
PB: 753 Temp: 23

| Spirometry | Ref | Pre Meas | Pre % Ref | Post Meas | Post % Ref |
|---|---|---|---|---|---|
| FVC | 4.86 | 4.48 | 92 | | |
| $FEV_1$ | 3.38 | (1.61) | (48) | | |
| $FEV_1$/FVC | 70.0 | (36.0) | | | |
| $FEF_{25-75}$% | 3.11 | (0.35) | (11) | | |
| PEF | 9.02 | 5.43 | 60 | | |
| **Lung Volumes** | | | | | |
| TLC | | | | | |
| RV | | | | | |
| RV/TLC | | | | | |
| FRC PL | | | | | |
| ERV | | | | | |
| VC | | | | | |
| **Resistance** | | | | | |
| Raw | | | | | |
| sRaw | | | | | |
| **Diffusion** | | | | | |
| $D_{LCO}$ | | | | | |
| $D_{LCO}$ /$V_A$ | | | | | |
| $V_A$ | | | | | |




**Comments:** The patient could not fully expire during to FVC or SVC, therefore the results for both vital capacities may be underestimated. See attached FV loops

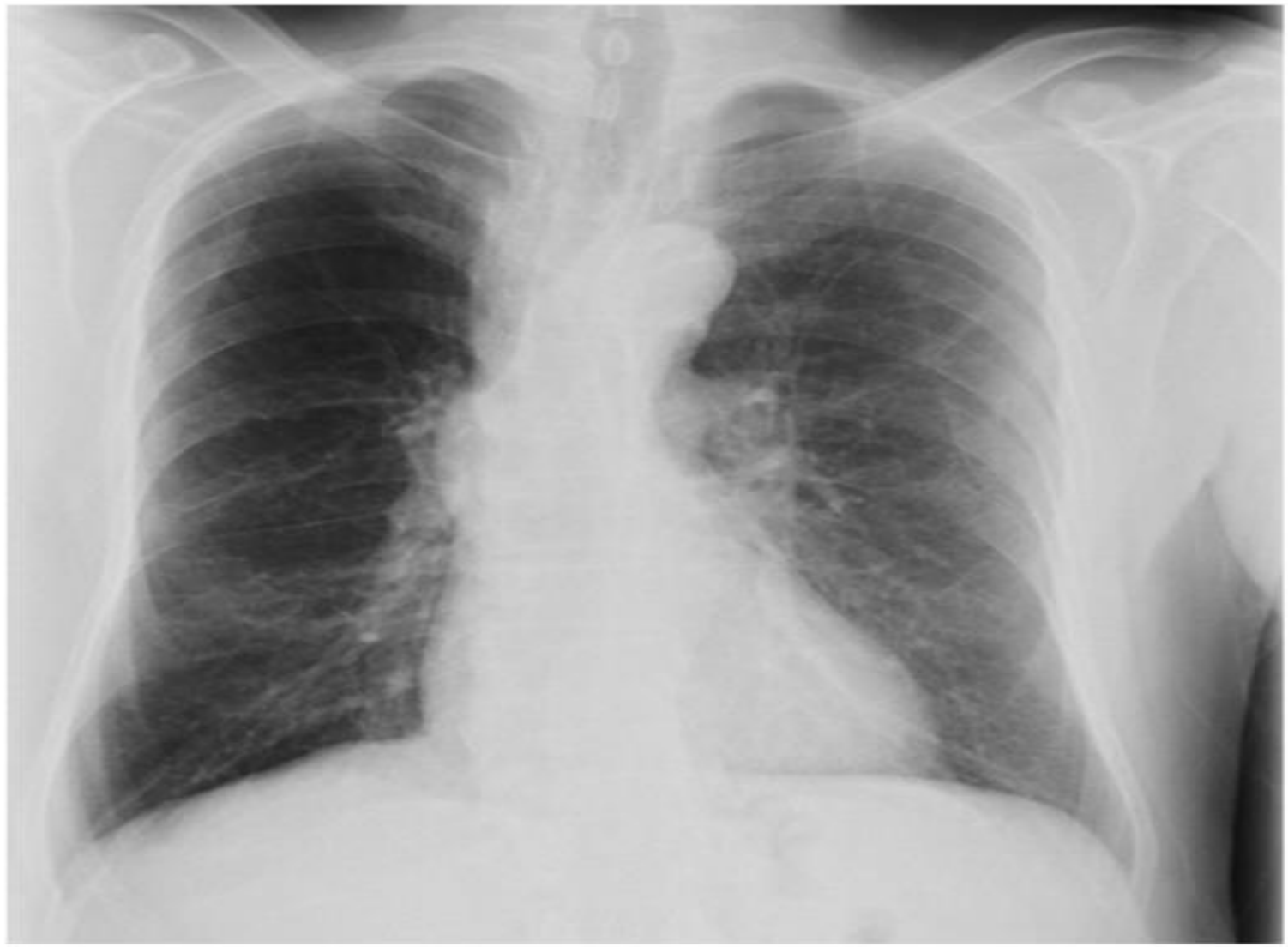


*Figure 1: COPD chest x-ray (AP view): hyperinflated lung, flattened diaphragm, increased intercostal spaces*

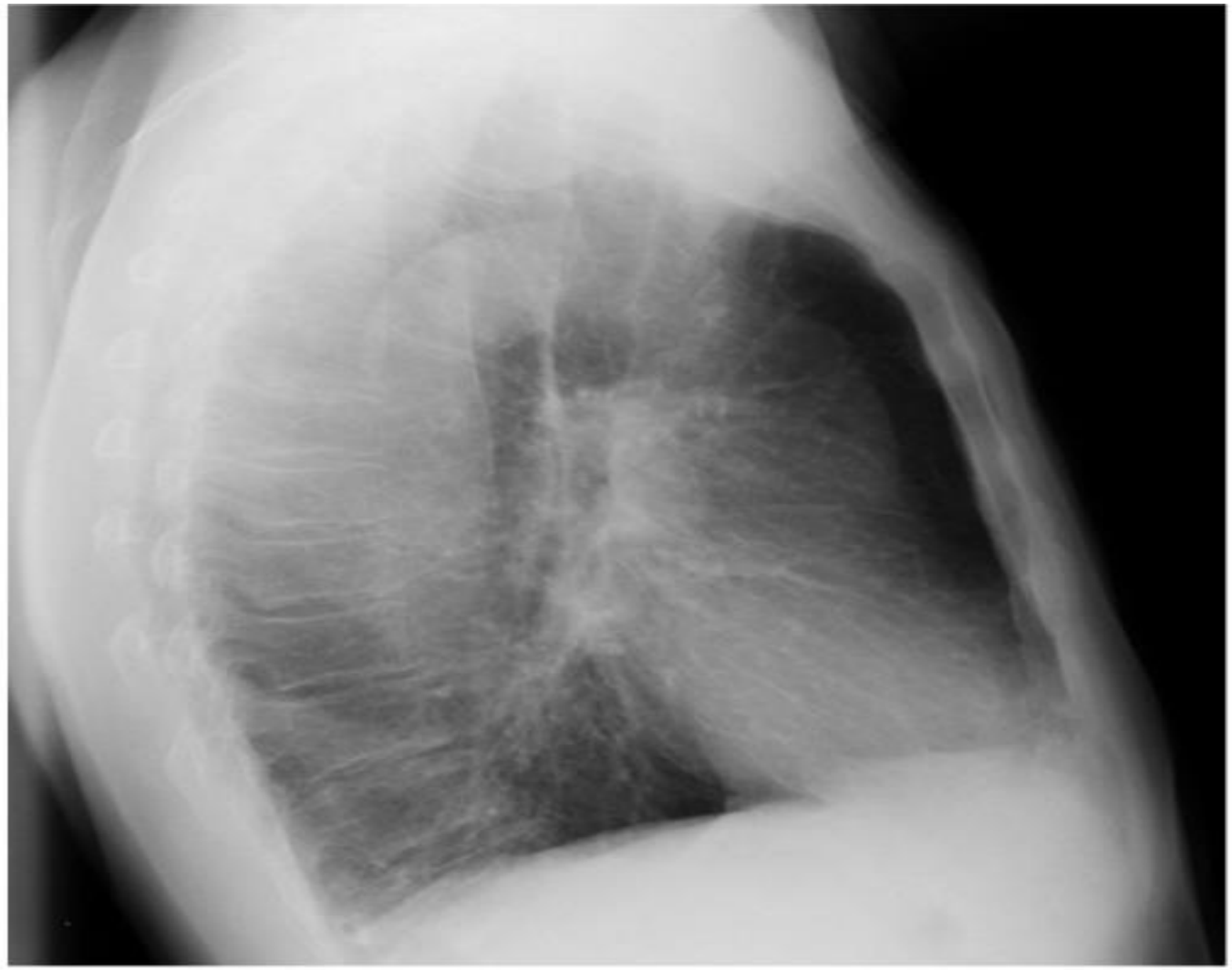


*Figure 2: COPD chest x-ray (lateral view): hyperinflated lung, flattened diaphragm, increased antero-posterior diameter (barrel chest) in lateral view*

# Differentiating COPD from Asthma

| | Asthma | COPD |
|---|---|---|
| Onset | Anytime (often childhood or youth) | Later in life |
| Etiology | Allergic, family history | Smoking, other noxious exposures |
| Course | Intermittent | Chronic progressive |
| Clinical features | Wheeze, episodic dyspnea, cough | Persistent dyspnea, productive cough |
| Pattern of Symptoms | Variable day to day, more at night/early morning | Less variable, more on exertion |
| Inflammatory cells and mediators | Eosinophils, mast cells, Th-2 type | Neutrophils, macrophages, Th-1 type |
| Response to Bronchodilators | Largely reversible | Partially reversible or irreversible |
| Response to steroids | Substantial | Partial |

# Other differential diagnosis :

- Congestive heart failure
- Bronchiectasis
- GERD
- Bronchiolitis
- T.B

# Other tests

- CBC ,ABG, Chest CT, sputum culture ….

## 1st test to order

| Test | Result |
| --- | --- |
| **spirometry**<br>• COPD is classified based on the patient's FEV1 and its percentage of the predicted FEV1. In cases where FVC may be hard to measure, FEV6 (forced expiratory volume at 6 seconds) can be used.[19] | **FEV1/FVC ratio <0.70; total absence of reversibility is neither required nor the most typical result** |
| **pulse oximetry**<br>• Checked as part of vital signs on acute presentation. A good pulse wave should be picked up by the device. In patients with chronic disease, an oxygen saturation of 88% to 90% may be acceptable.<br>• If <92% arterial or capillary blood gases should be checked.[1] | **low oxygen saturation** |
| **ABG**<br>• Checked in patients who are acutely unwell, especially if they have an abnormal pulse oximetry reading. Should also be performed in stable patients with FEV1 <35% predicted or with clinical signs suggestive of respiratory failure, or if peripheral arterial oxygen saturation is <92%.<br>• Hypercapnia, hypoxia, and respiratory acidosis are signs of impending respiratory failure and possible need for intubation. | **PaCO2 >50 mmHg and/ or PaO2 of <60 mmHg suggests respiratory insufficiency** |
| **CXR**<br>• Seldom diagnostic, but useful in ruling out other pathologies.<br>• Increased anteroposterior ratio, flattened diaphragm, increased intercostal spaces, and hyperlucent lungs may be seen.<br>[Fig-1]<br><br>[Fig-2]<br>• May also demonstrate complications of COPD, such as pneumonia and pneumothorax. | **hyperinflation** |
| **FBC**<br>• This test may be considered to assess severity of an exacerbation and may show polycythaemia (haematocrit >55%), anaemia, and leucocytosis.[1] | **raised haematocrit, possible increased WBC count** |
| **ECG**<br>• Risk factors for COPD are similar to those for ischaemic heart disease, so comorbidity is common. | **signs of right ventricular hypertrophy, arrhythmia, ischaemia** |

## Other tests to consider

| Test | Result |
| --- | --- |
| **sputum culture**<br>• Presence of purulent sputum is sufficient to commence empirical antibiotics. Sputum culture indicated if empirical antibiotics fail.[1] | **infecting organism** |
| **PFTs**<br>• Useful for resolving diagnostic uncertainties and preoperative assessment.[1] Requires specialist laboratory facilities.<br>• Decreased diffusing capacity of the lung for carbon monoxide (DLCO) is supportive of emphysema over chronic bronchitis. | **obstructive pattern, decreased DLCO** |
| **chest CT scan**<br>• Provides better visualisation of type and distribution of lung tissue damage and bulla formation than CXR. [Fig-3]<br>• In contrast to smoking-related COPD, alpha-1 antitrypsin deficiency mainly affects lower fields.<br>• Useful in excluding other underlying pulmonary disease and for pre-operative assessment. | **hyperinflation** |
| **alpha-1 antitrypsin level**<br>• Low level in patients with alpha-1 antitrypsin deficiency. Test is done if there is high suspicion for alpha-1 antitrypsin deficiency, such as a positive family history and atypical COPD cases (young patients and non-smokers). | **should be normal in patients with COPD** |
| **exercise testing**<br>• Can be of value in patients with a disproportional degree of dyspnoea compared with spirometry.[21] It can be performed on a cycle or treadmill ergometer, or by a simple timed walking test (e.g., 6 minutes). Exercise testing is of use in selecting patients for rehabilitation. | **poor exercise performance or exertional hypoxaemia is suggestive of advanced disease** |
| **sleep study**<br>• Obstructive sleep apnoea, a common finding in patients with COPD, is associated with increased risk of death and hospitalisation in patients with COPD.[20] | **elevated apnoea-hypopnoea index and/or nocturnal hypoxaemia** |
| **respiratory muscle function**<br>• Respiratory muscle function may be tested if dyspnoea or hypercapnia are disproportionately increased with respect to FEV1, as well as in patients with poor nutrition and those with corticosteroid myopathy.[22] | **reduced maximal inspiratory pressure** |

# Treatment

- Reducing risk factor exposure
- Appropriate assessment of disease
- Patient education
- Pharmacological and non-pharmacological management of stable COPD
- Prevention and treatment of acute COPD exacerbations

# Nonpharmacological treatment(stable COPD)

- **Smoking cessation**
- **Education , self management and pulmonary rehabilitation**
- **Vaccinations**
- **Nutrition**
- **End of life and palliative care**
- **Treatment of hypoxia**
- **Treatment of hypercapnia**
- Intervention bronchoscopy and surgery

# Pharmacological treatment

- Inhaled B2 agonist(short acting)(SABA)
- Inhaled B2 agonist(long acting)(LABA)
- Inhaled anticholinergic(short acting)(SAMA)
- Inhaled anticholinergic(long acting)(LAMA)
- Inhaled corticosteroid (ICS)
- Combination inhalers
- Methylxanthine
- Phosphodiastrase-4 inhibitor

# Treatment of stable COPD

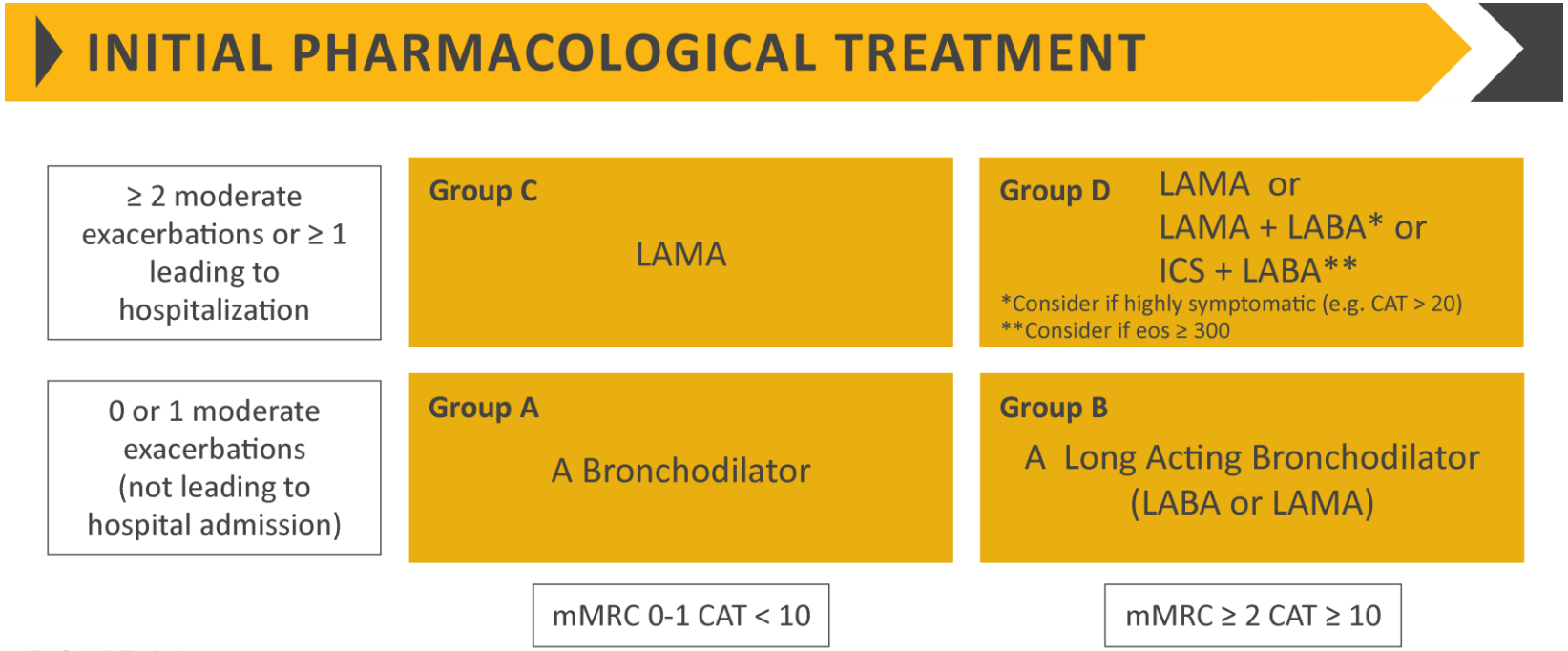



FIGURE 4.1

**Definition of abbreviations:** eos: blood eosinophil count in cells per microliter; mMRC: modified Medical Research Council dyspnea questionnaire; CAT™: COPD Assessment Test™.

# COPD exacerbation

- COPD exacerbations are defined as an acute worsening of respiratory symptoms that result in additional therapy.

(Increasing SOB , cough or sputum production or colour )

- They are classified as:

• Mild (treated with short acting bronchodilators only, SABDs)

• Moderate (treated with SABDs plus antibiotics and/or oral corticosteroids)

•Severe (patient requires hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure.

# Thank you