COPD

By

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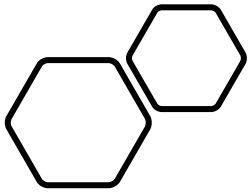


Basics Prevention Diagnosis treatment

Definition

- A **heterogeneous** lung condition:

❑ Characterized by **chronic** respiratory symptoms (dyspnea, cough, expectoration and/or exacerbations)

❑ Due to abnormalities of the **airways** (bronchitis, bronchiolitis) and/or **alveoli** (emphysema)

❑ That cause **persistent**, often **progressive**, airflow obstruction.

Pathogenesis

- The airways and air sacs are **elastic or stretchy**:

- When you **breathe in**, each air sac fills up with air, like a small balloon. - When you **breathe out**, the air sacs deflate, and the air goes out.

- In COPD, **less air flows** in and out of the airways because of one or more of the following:

- The airways and air sacs **lose** their **elastic** quality.

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

- The walls of the airways become **thick and inflamed**.

- The airways make **more mucus** than usual and can become clogged

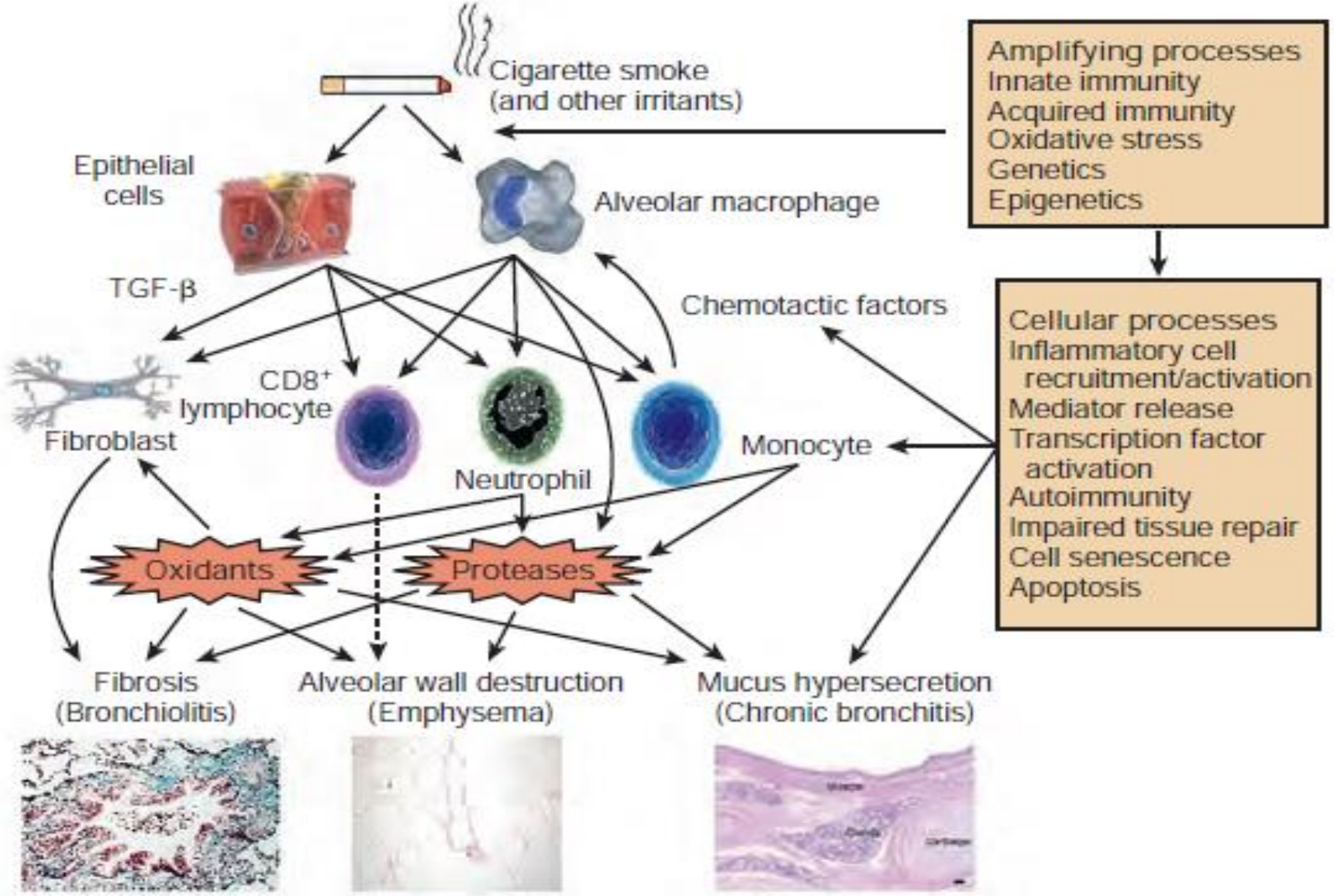
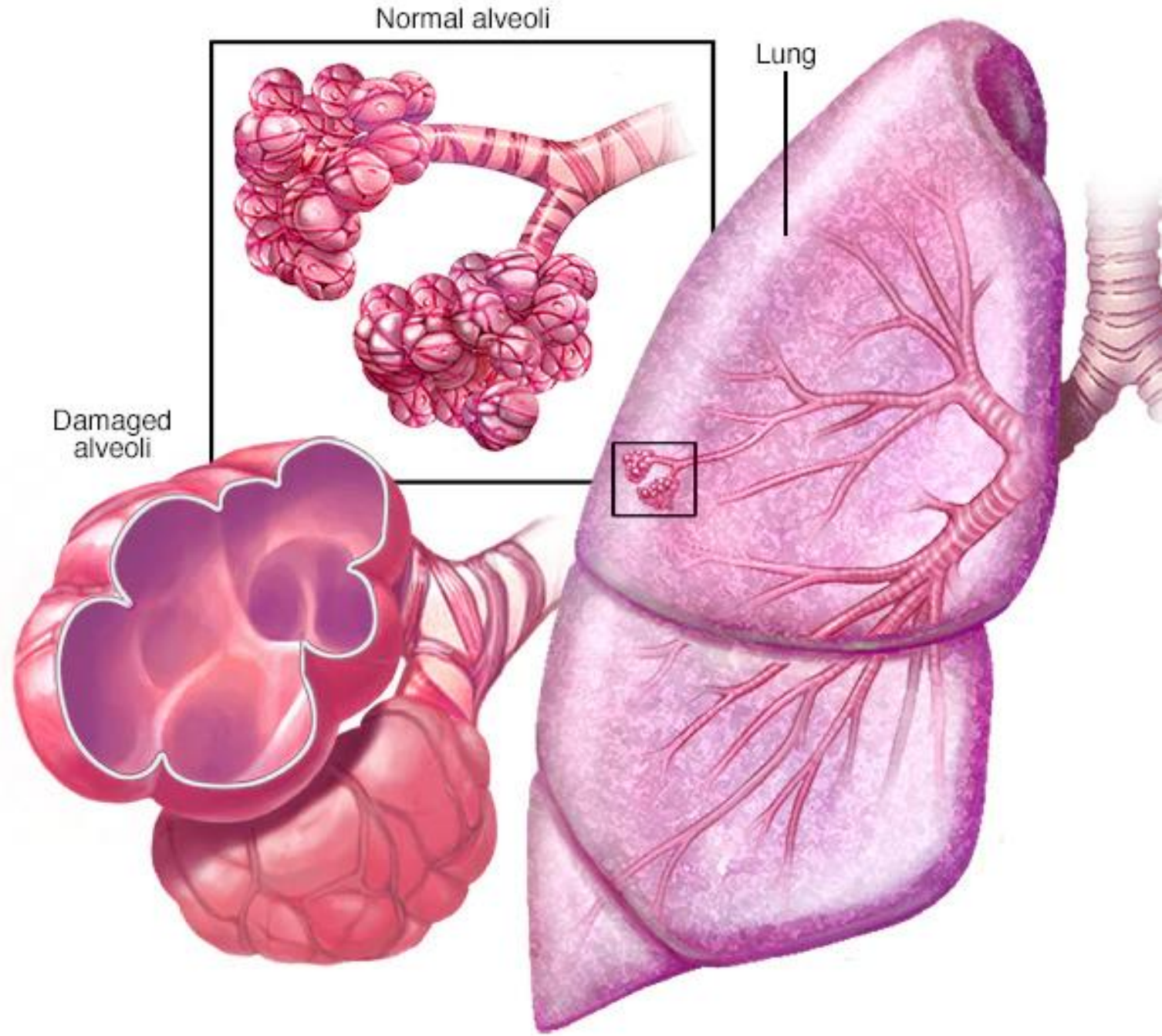
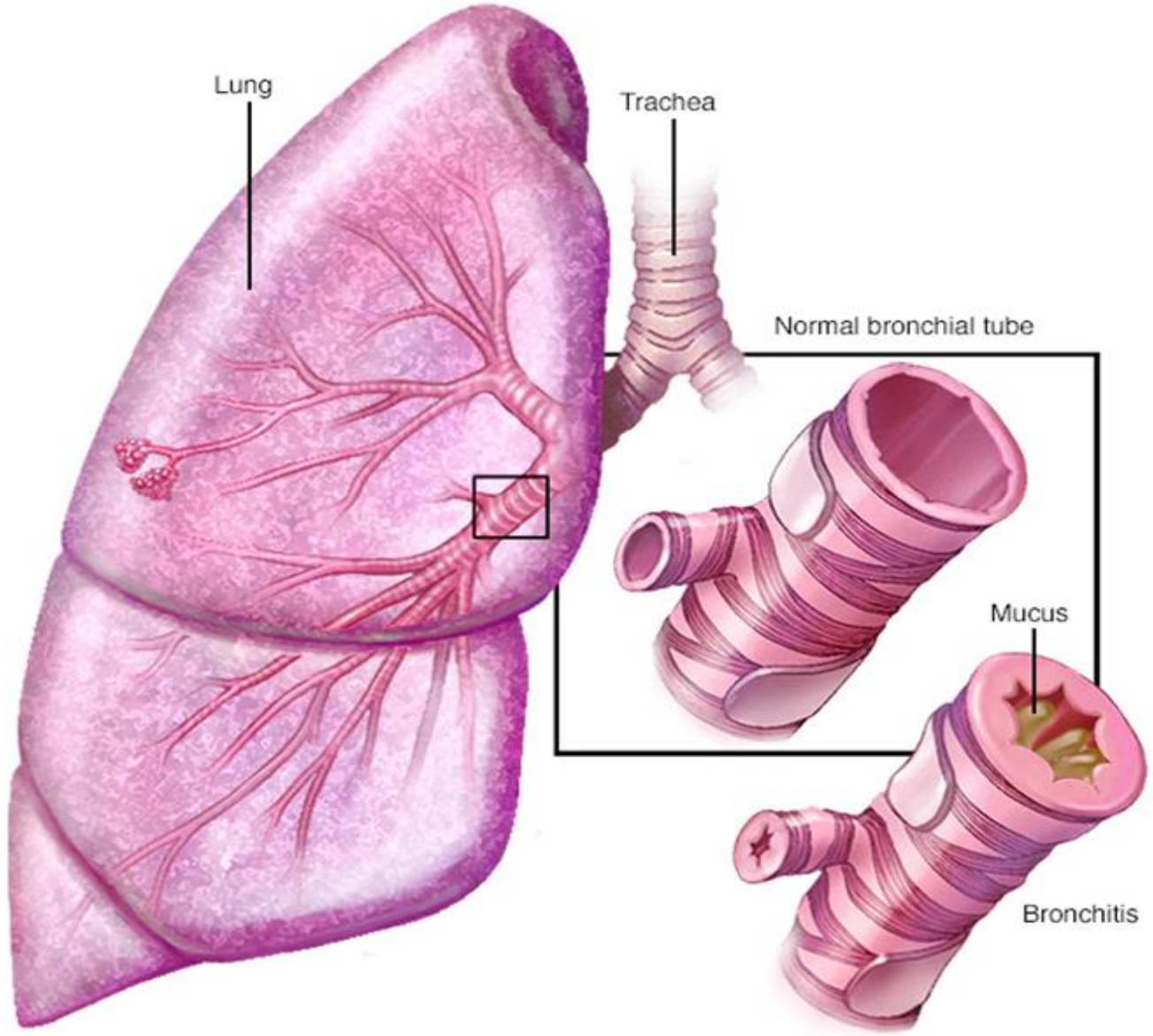


Figure 43-3 Overview of the pathogenesis of COPD. Cigarette smoke activates macrophages and epithelial cells to produce chemotactic factors that recruit neutrophils and CD8 cells from the circulation. These cells release factors that activate fibroblasts, resulting in abnormal repair processes and bronchiolar 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.

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



- **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.



Epidemiology

- Represents an important public health challenge and is a major cause of chronic morbidity and mortality throughout the world.
- COPD is currently the **3rd leading** cause of death in the world.
- 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**.

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Proposed Taxonomy (Etiotypes) for COPD

Table 1.1

Classification	Description
Genetically determined COPD (COPD-G)	Alpha-1 antitrypsin deficiency (AATD) Other genetic variants with smaller effects acting in combination
COPD due to abnormal lung development (COPD-D)	Early life events, including premature birth and low birthweight, among others
Environmental COPD	
Cigarette smoking COPD (COPD-C)	• Exposure to tobacco smoke, including *in utero* or via passive smoking • Vaping or e-cigarette use • Cannabis
Biomass and pollution exposure COPD (COPD-P)	Exposure to household pollution, ambient air pollution, wildfire smoke, occupational hazards
COPD due to infections (COPD-I)	Childhood infections, tuberculosis-associated COPD, HIV-associated COPD
COPD & asthma (COPD-A)	Particularly childhood asthma
COPD of unknown cause (COPD-U)	

*Adapted from Celli et al. (2022) and Stolz et al. (2022)

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Clinical Indicators for Considering a Diagnosis of COPD

Table 2.1

Consider the diagnosis of COPD, and perform spirometry, if any of these clinical indicators are present:
(these indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of the presence of COPD; in any case, spirometry is required to establish a diagnosis of COPD)

Dyspnea that is	Progressive over time Worse with exercise Persistent
Recurrent wheeze	
Chronic cough	May be intermittent and may be unproductive
Recurrent lower respiratory tract infections	
History of risk factors	Tobacco smoke (including popular local preparations) Smoke from home cooking and heating fuels Occupational dusts, vapors, fumes, gases and other chemicals Host factors (e.g., genetic factors, developmental abnormalities, low birthweight, prematurity, childhood respiratory infections etc.)

- **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.

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

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.

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

Figure 3.4

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 counseling; 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*

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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.

Diagnosis

- **History**:

- **Early symptoms include**:
- Occasional shortness of breath, especially after exercise
- Mild but recurrent cough
- Needing to clear throat often, especially first thing in the morning
- Start making subtle changes, such **as avoiding stairs and skipping physical activities.**
- Symptoms can get progressively worse and harder to ignore

Diagnosis

-**As the lungs become more damaged** :

- Shortness of breath, after even **mild** exercise such as walking up a flight of stairs
- Wheezing, which is a type of higher pitched noisy breathing, especially during exhalations
- Chest tightness
- Chronic cough, with or without mucus
- Need to clear mucus from your lungs **every day**
- **frequent** colds, flu, or other respiratory infections
- lack of energy

Diagnosis

- In later stages of COPD, symptoms may also include:

- Fatigue
- Swelling of the feet, ankles, or legs
- Weight loss

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.”

Differential Diagnosis of COPD

Table 2.3

Diagnosis	Suggestive Features
COPD	Symptoms slowly progressive History of tobacco smoking or other risk factors
Asthma	Variable airflow obstruction Symptoms vary widely from day to day Symptoms worse at night/early morning Allergy, rhinitis, and/or eczema also present Often occurs in children Family history of asthma
Congestive heart failure	Chest X-ray shows dilated heart, pulmonary edema Pulmonary function tests indicate volume restriction, not airflow obstruction
Bronchiectasis	Large volumes of purulent sputum Commonly associated with bacterial infection Chest X-ray/HRCT shows bronchial dilation
Tuberculosis	Onset all ages Chest X-ray shows lung infiltrate Microbiological confirmation High local prevalence of tuberculosis
Obliterative bronchiolitis	Can occur in children Seen after lung or bone marrow transplantation HRCT on expiration shows hypodense areas
Diffuse panbronchiolitis	Predominantly seen in patients of Asian descent Most patients are male and nonsmokers Almost all have chronic sinusitis Chest X-ray & HRCT show diffuse small centrilobular nodular opacities & hyperinflation

These features tend to be characteristic of the respective diseases, but are not mandatory. For example, a person who has never smoked may develop COPD (especially in LMICs where other risk factors may be more important than cigarette smoking).

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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

A. Spirometry - Normal Trace B. Spirometry - Airflow Obstruction

Figure 2.5

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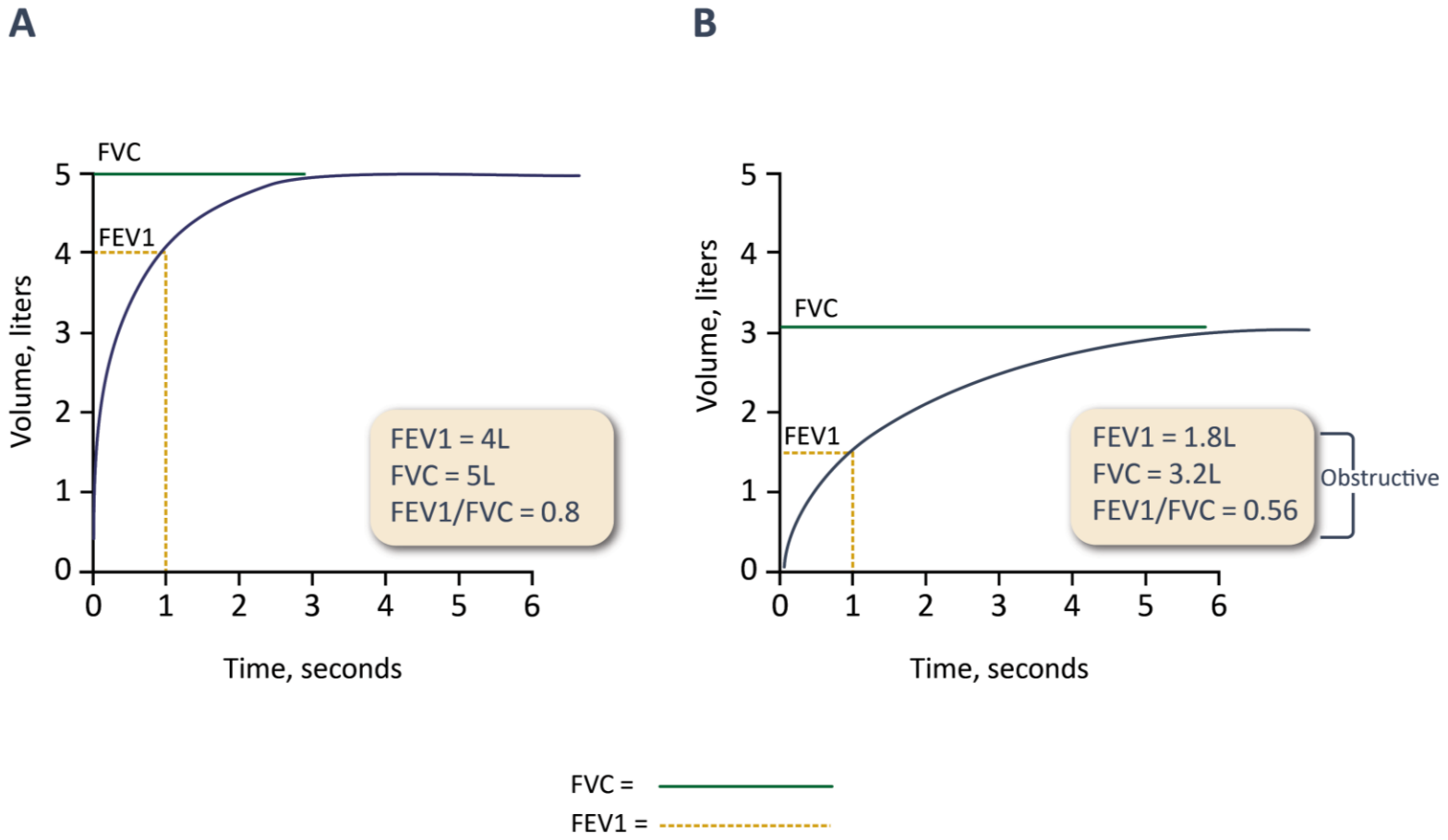


Spirometry

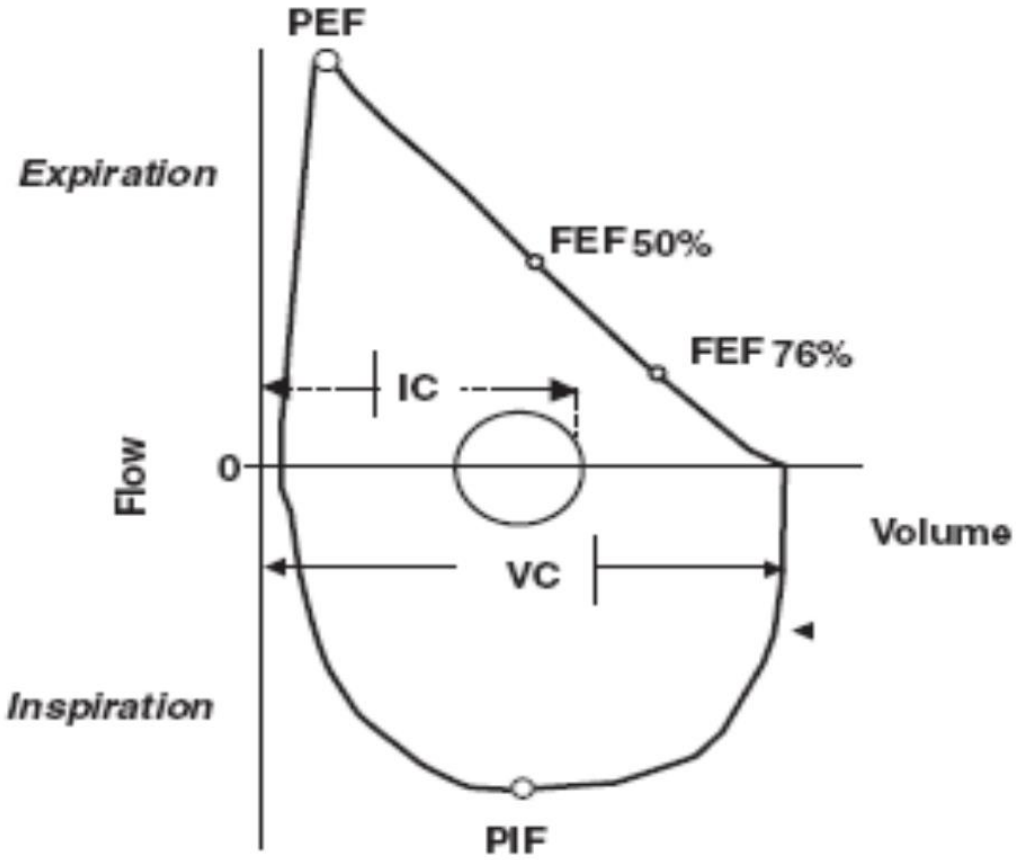
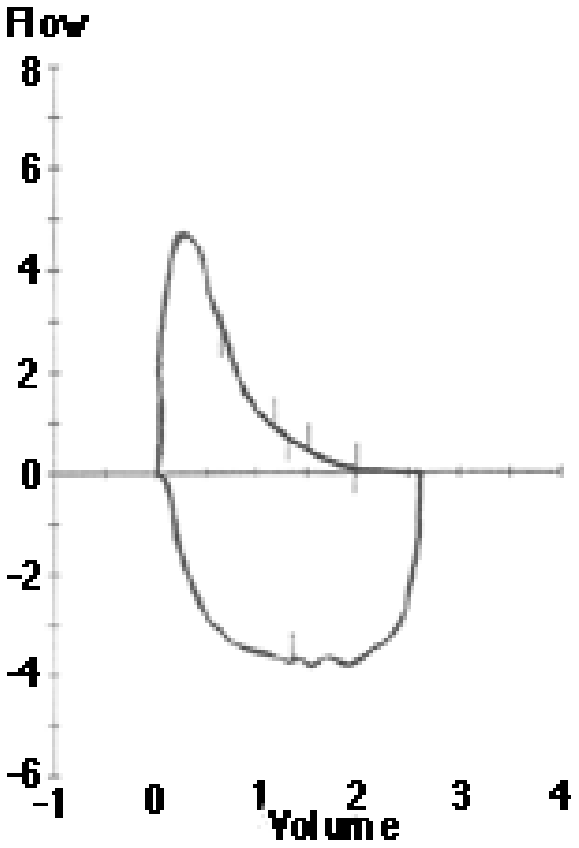
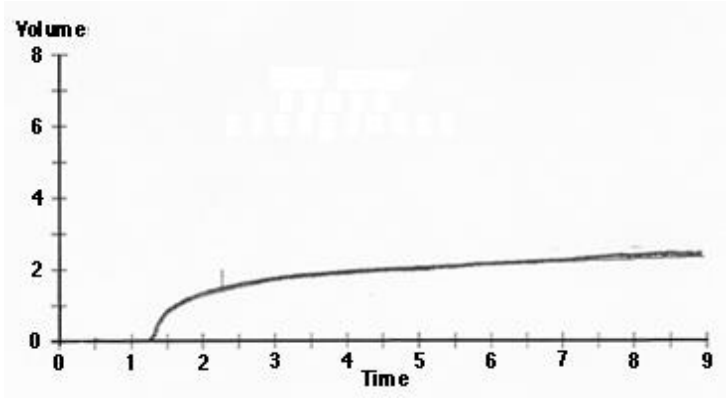
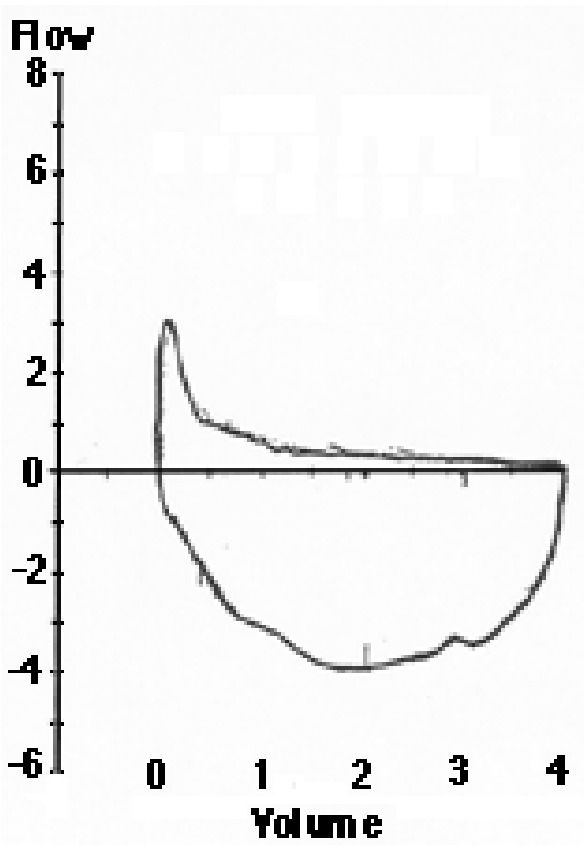


Figure 3 Flow volume curve for a normal subject showing the principal measures used.







Airflow obstruction

Mild on left

Severe on right

Role of Spirometry in COPD

Figure 2.6

- **Diagnosis**
- **Assessment of severity of airflow obstruction (for prognosis)**
- **Follow-up assessment**
  - Therapeutic decisions
    - Pharmacological in selected circumstances (e.g., discrepancy between spirometry and level of symptoms)
    - Consider alternative diagnoses when symptoms are disproportionate to degree of airflow obstruction
    - Non-pharmacological (e.g., interventional procedures)
  - Identification of rapid decline

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GOLD Grades and Severity of Airflow Obstruction in COPD (based on post-bronchodilator FEV1)

Figure 2.7

In COPD patients (FEV1/FVC < 0.7):

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

GOLD ABE Assessment Tool

Figure 2.10

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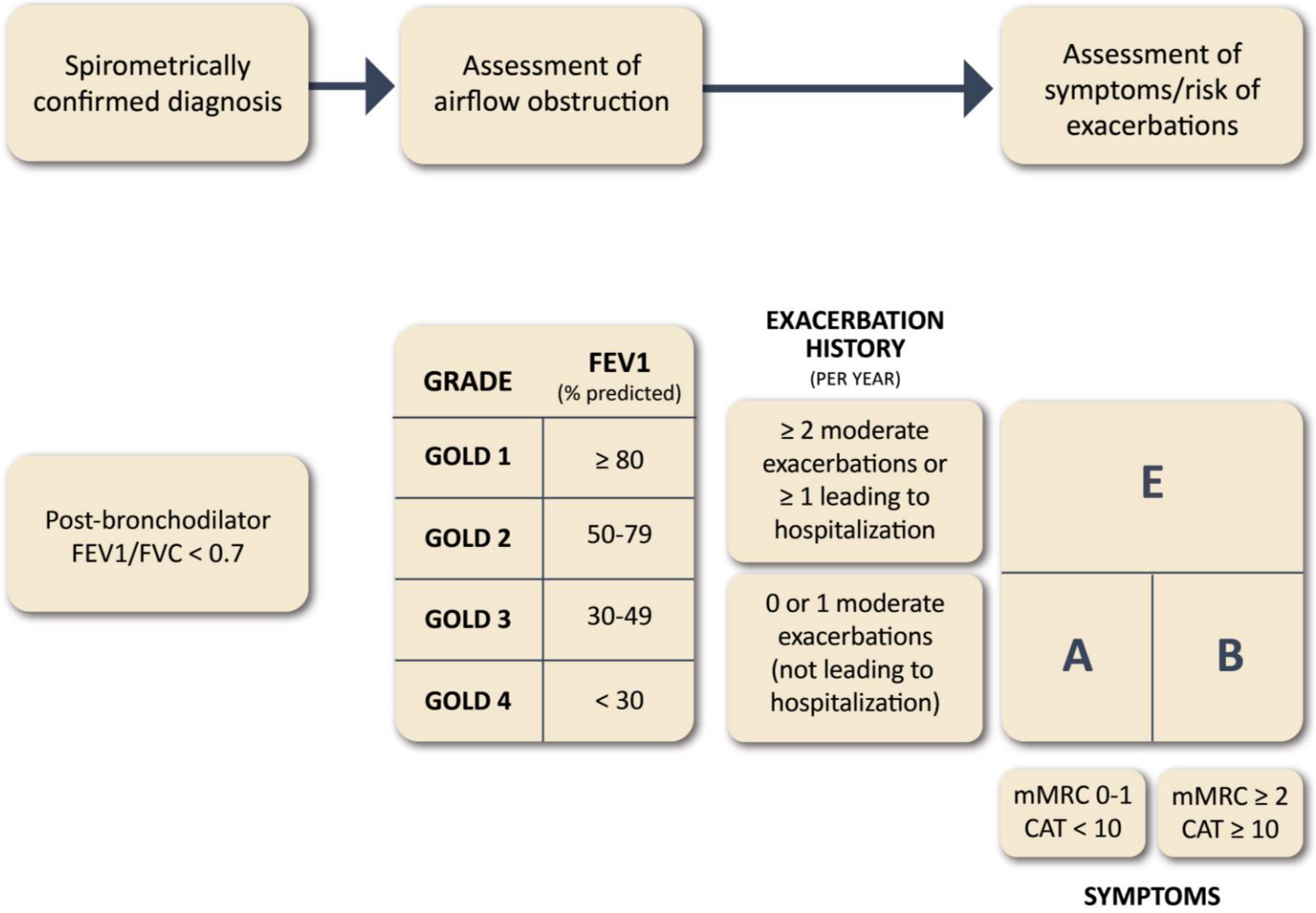

Modified MRC Dyspnea Scale

Figure 2.8

PLEASE TICK IN THE BOX THAT APPLIES TO YOU | ONE BOX ONLY | Grades 0 - 4

mMRC Grade 0	mMRC Grade 1	mMRC Grade 2	mMRC Grade 3	mMRC Grade 4
I only get breathless with strenuous exercise	I get short of breath when hurrying on the level or walking up a slight hill	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	I stop for breath after walking about 100 meters or after a few minutes on the level	I am too breathless to leave the house or I am breathless when dressing or undressing
☐	☐	☐	☐	☐

Reference: ATS (1982) Am Rev Respir Dis. Nov;126(5):952-6.

CAT™ Assessment

Figure 2.9

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	

Reference: Jones et al. ERJ 2009; 34 (3); 648-54.

TOTAL SCORE: ☐

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Use of CT in Stable COPD

Figure 2.11

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Differential Diagnosis	• Frequent exacerbations with excessive cough with sputum production, raising concern for bronchiectasis or atypical infection • Symptoms out of proportion to disease severity based on lung function testing
Lung Volume Reduction	• Endobronchial valve therapy may be a therapeutic option for patients if they demonstrate postbronchodilator FEV1 between 15% to 45% and evidence of hyperinflation • Lung volume reduction surgery may be a therapeutic option for patients with hyperinflation, severe upper lobe predominant emphysema and low exercise capacity after pulmonary rehabilitation
Lung Cancer Screening	• Annual low-dose CT scan is recommended for lung cancer screening in patients with COPD due to smoking according to recommendations for the general population

Common Risk Factors for Development of Lung Cancer

Table 6.1

- Age > 55
- Smoking history > 30 pack years
- Presence of emphysema by CT scan
- Presence of airflow limitation FEV1/FVC < 0.7
- BMI < 25 kg/m^2
- Family history of lung cancer

Treatment

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

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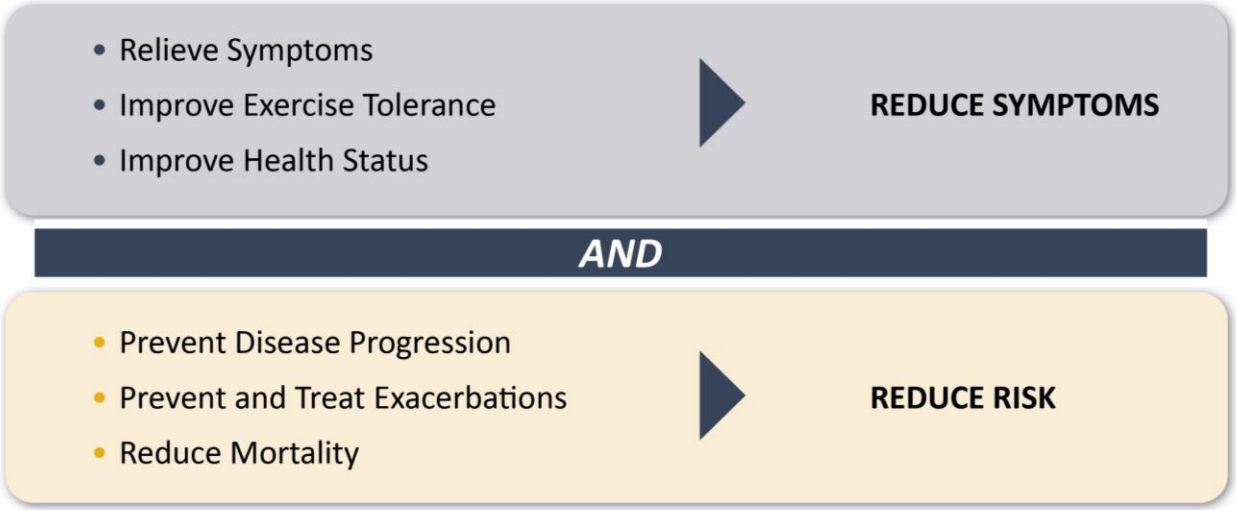
Goals for Treatment of Stable COPD

Figure 3.1



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

Non-Pharmacological Management of COPD*

Figure 3.12

Patient Group	Essential	Recommended	Depending on Local Guidelines
A	Smoking cessation (can include pharmacological treatment)	Physical activity	Influenza vaccination COVID-19 vaccinations Pneumococcal vaccination Pertussis vaccination Shingles vaccination RSV vaccination
B and E	Smoking cessation (can include pharmacological treatment) Pulmonary rehabilitation	Physical activity	Influenza vaccination COVID-19 vaccinations Pneumococcal vaccination Pertussis vaccination Shingles vaccination RSV vaccination

*Can include pharmacological treatment

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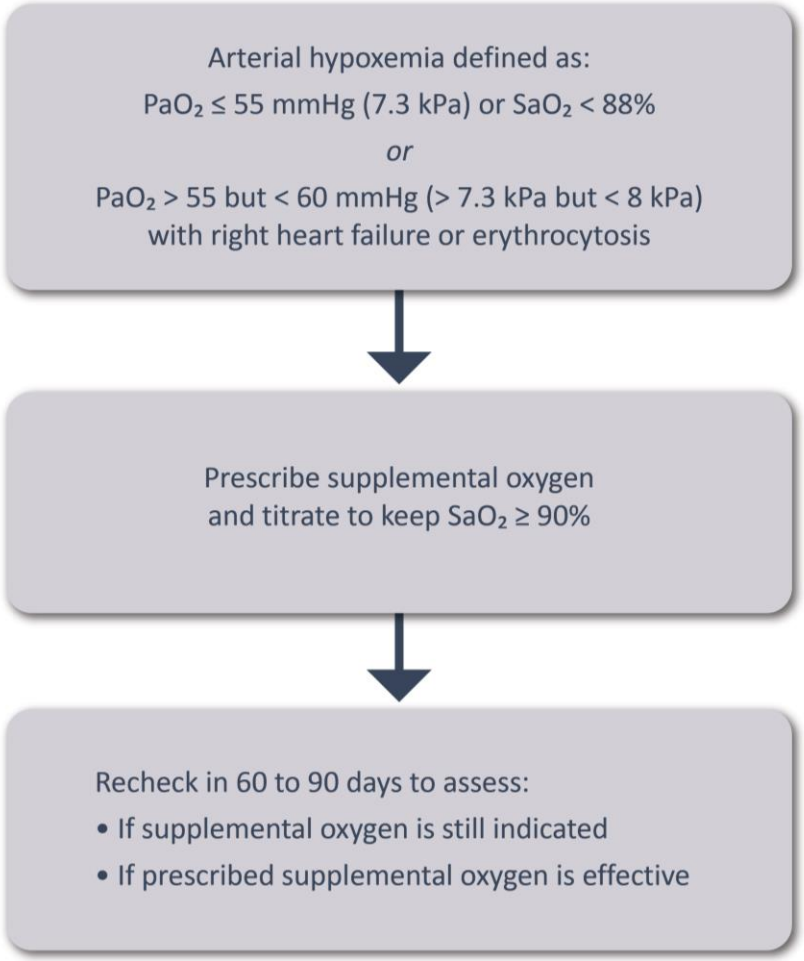
Prescription of Supplemental Oxygen to COPD Patients

Figure 3.15



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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

Initial Pharmacological Treatment

Figure 3.7

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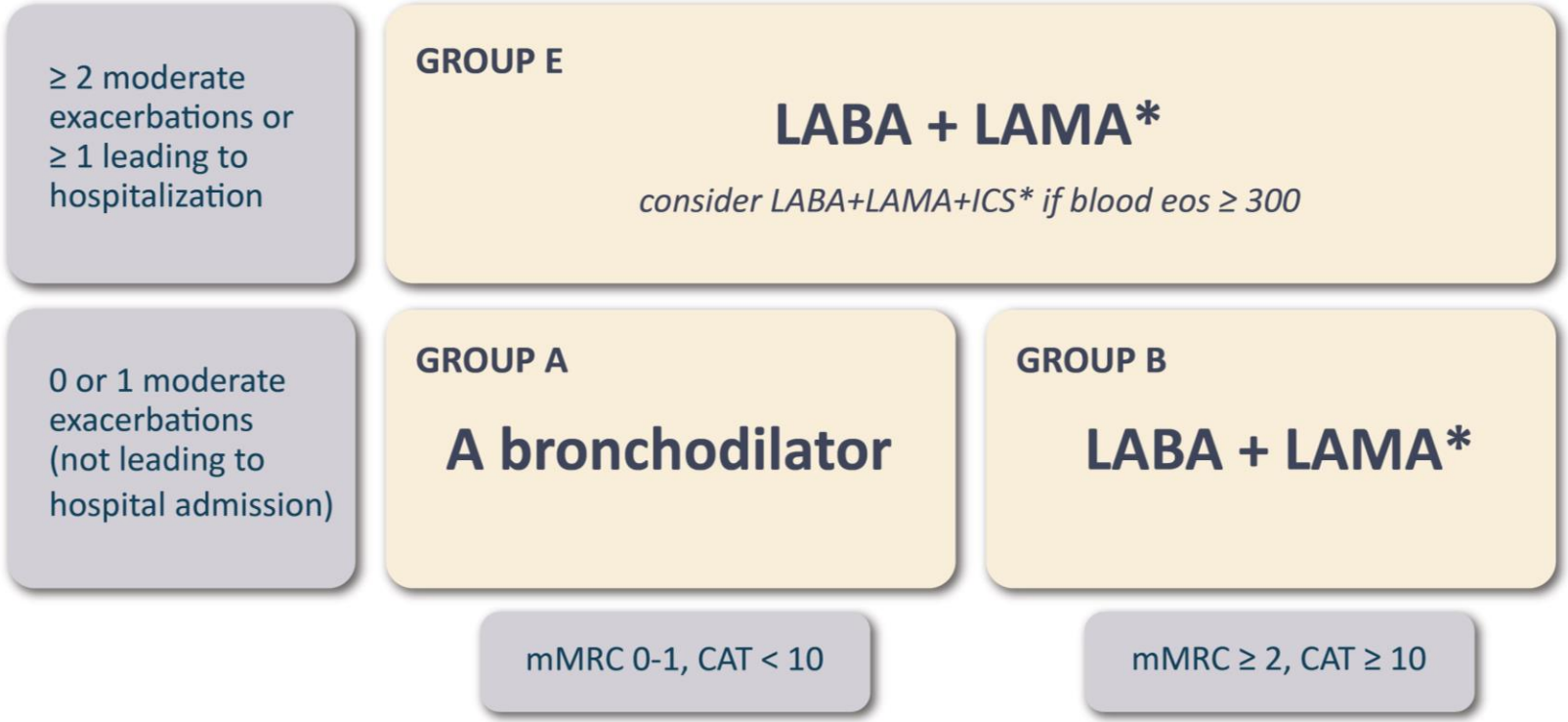

*Single inhaler therapy may be more convenient and effective than multiple inhalers; single inhalers improve adherence to treatment

Exacerbations refers to the number of exacerbations per year; eos: blood eosinophil count in cells per microliter; mMRC: modified Medical Research Council dyspnea questionnaire; CAT™: COPD Assessment Test™.

COPD Exacerbation

An event characterized by dyspnea and/or cough and sputum that worsen **over ⩽14 days**, which may be accompanied by **tachypnea** and/or tachycardia and is often associated with increased local and systemic **inflammation** caused by airway infection, pollution, or other insult to the airway.

Confounders or Contributors to be Considered in Patients Presenting with Suspected COPD Exacerbation

Figure 4.1

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Most frequent	**Pneumonia**	• Chest radiograph
	Pulmonary embolism	• Clinical probability assessment (Hemoptysis, surgery, fracture, history of cancer, DVT) • D-dimer • CT angiography for pulmonary embolism
	Heart failure	• Chest radiograph • NT Pro-Brain Natriuretic Peptide (Pro-BNP) and BNP • Echocardiography
Less frequent	**Pneumothorax, pleural effusion**	• Chest radiograph • Thoracic ultrasound
	Myocardial infarction and/or cardic arrhythmias (atrial fibrillation/flutter)	• Electrocardiography • Troponin

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Diagnosis and Assessment

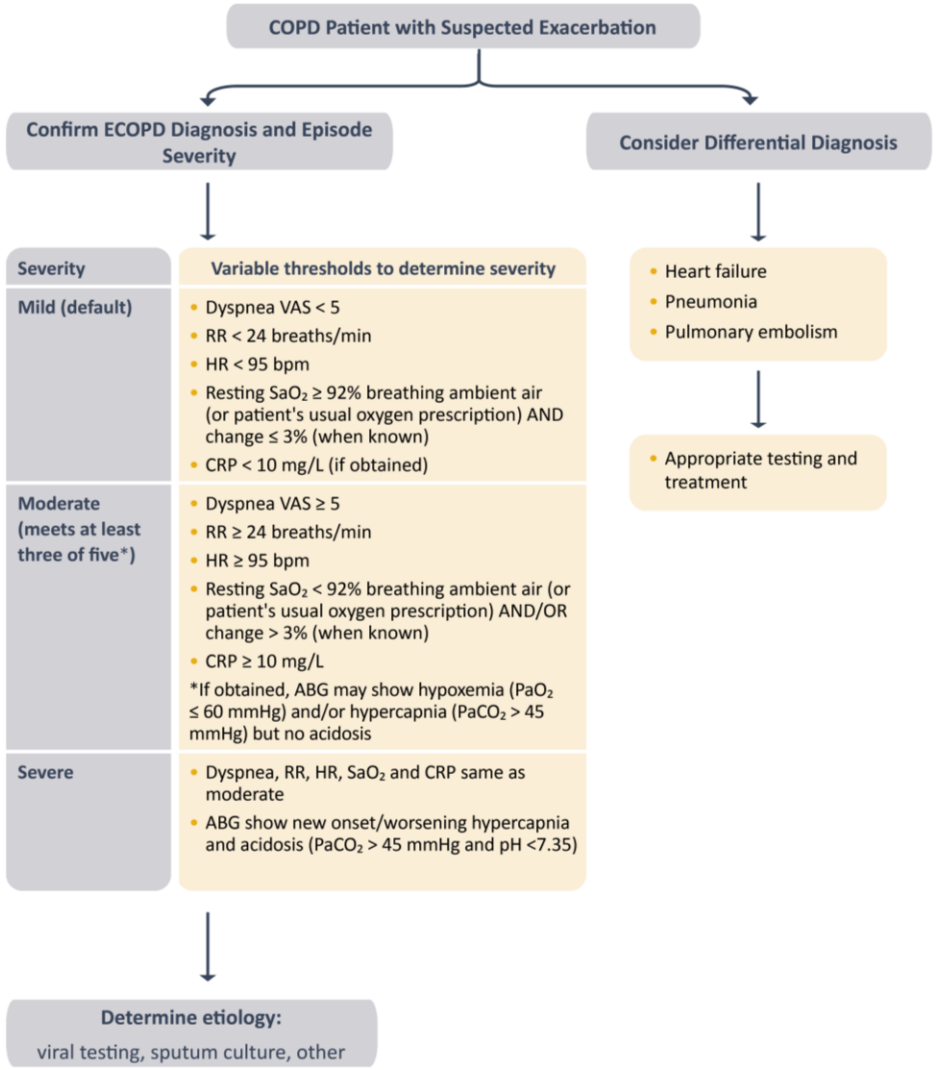
Figure 4.2

1.	Complete a thorough clinical assessment for evidence of COPD and potential respiratory and non-respiratory concomitant diseases, including consideration of alternative causes for the patient's symptoms and signs: primarily pneumonia, heart failure, and pulmonary embolism.
2.	**Assess:** a. Symptoms, severity of dyspnea that can be determined by using a VAS, and documentation of the presence of cough. b. Signs (tachypnea, tachycardia), sputum volume and color, and respiratory distress (accessory muscle use).
3.	Evaluate severity by using appropriate additional investigations such as pulse oximetry, laboratory assessment, CRP, arterial blood gases.
4.	Establish the cause of the event (viral, bacterial, environmental, other).

Abbreviations: COPD = chronic obstructive pulmonary disease; CRP = C-reactive protein; VAS = visual analog scale.

Classification of the Severity of COPD Exacerbations

Figure 4.3



Adapted from: The ROME Proposal, Celli et al. (2021) Am J Respir Crit Care Med. 204(11): 1251-8.

Abbreviations: VAS visual analog dyspnea scale; RR respiratory rate; HR heart rate; SaO_2 oxygen saturation; CRP C-reactive protein; ABG arterial blood gases; PaO_2 Arterial pressure of oxygen.

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Potential Indications for Hospitalization Assessment*

Figure 4.4

- Severe symptoms such as sudden worsening of resting dyspnea, high respiratory rate, decreased oxygen saturation, confusion, drowsiness
- Acute respiratory failure
- Onset of new physical signs (e.g., cyanosis, peripheral edema)
- Failure of an exacerbation to respond to initial medical management
- Presence of serious comorbidities (e.g., heart failure, newly occurring arrhythmias, etc.)
- Insufficient home support

*Local resources need to be considered

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Key Points for the Management of Stable COPD During COVID-19 Pandemic

Figure 6.1

Protective Strategies	• Follow basic infection control measures • Wear a face covering • Consider shielding/sheltering-in-place • Have the COVID-19 vaccinations in line with national recommendations
Investigations	• Only essential spirometry at times of high prevalence of COVID-19
Pharmacotherapy	• Ensure adequate supplies of medications • Continue unchanged including ICS
Non-Pharmacological Therapy	• Ensure annual influenza vaccination • Maintain physical activity

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Thank you