
Global Initiative for Chronic Obstructive Lung Disease



POCKET GUIDE TO COPD DIAGNOSIS, MANAGEMENT, AND PREVENTION

A Guide for Health Care Professionals

UPDATED 2010

Global Initiative for Chronic Obstructive Lung Disease

Pocket Guide to COPD Diagnosis, Management, and Prevention

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Representatives from many countries serve as a network for the dissemination and implementation of programs for diagnosis, management, and prevention of COPD. The GOLD Executive Committee is grateful to the many GOLD National Leaders who participated in discussions of concepts that appear in GOLD reports, and for their comments during the review of the 2006 *Global Strategy for the Diagnosis, Management, and Prevention of COPD*.

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PREFACE

Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. The **Global Initiative for Chronic Obstructive Lung Disease** was created to increase awareness of COPD among health professionals, public health authorities, and the general public, and to improve prevention and management through a concerted worldwide effort. The Initiative prepares scientific reports on COPD, encourages dissemination and adoption of the reports, and promotes international collaboration on COPD research.

While COPD has been recognized for many years, public health officials are concerned about continuing increases in its prevalence and mortality, which are due in large part to the increasing use of tobacco products worldwide and the changing age structure of populations in developing countries. The **Global Initiative for Chronic Obstructive Lung Disease** offers a framework for management of COPD that can be adapted to local health care systems and resources. Educational tools, such as laminated cards or computer-based learning programs, can be prepared that are tailored to these systems and resources.

The **Global Initiative for Chronic Obstructive Lung Disease** program includes the following publications:

- *Global Strategy for the Diagnosis, Management, and Prevention of COPD*. Scientific information and recommendations for COPD programs. (Updated 2010)
- *Executive Summary, Global Strategy for the Diagnosis, Management, and Prevention of COPD*. (Updated 2010)
- *Pocket Guide to COPD Diagnosis, Management, and Prevention*. Summary of patient care information for primary health care professionals. (Updated 2010)
- *What You and Your Family Can Do About COPD*. Information booklet for patients and their families.

These publications are available on the Internet at www.goldcopd.org. This site provides links to other websites with information about COPD.

This Pocket Guide has been developed from the *Global Strategy for the Diagnosis, Management, and Prevention of COPD* (2010). Technical discussions of COPD and COPD management, evidence levels, and specific citations from the scientific literature are included in that source document.

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KEY POINTS

- **Chronic Obstructive Pulmonary Disease (COPD)** is a preventable and treatable disease with some significant extra-pulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.
- Worldwide, the most commonly encountered **risk factor** for COPD is **cigarette smoking**. **At every possible opportunity individuals who smoke should be encouraged to quit**. In many countries, air pollution resulting from the burning of wood and other biomass fuels has also been identified as a COPD risk factor.
- A **diagnosis** of COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease. The diagnosis should be confirmed by spirometry.
- A **COPD management program** includes four components: assess and monitor disease, reduce risk factors, manage stable COPD, and manage exacerbations.
- **Pharmacologic treatment** can prevent and control symptoms, reduce the frequency and severity of exacerbations, improve health status, and improve exercise tolerance.
- **Patient education** can help improve skills, ability to cope with illness, and health status. It is an effective way to accomplish smoking cessation, initiate discussions and understanding of advance directives and end-of-life issues, and improve responses to acute exacerbations.
- COPD is often associated with **exacerbations** of symptoms.

WHAT IS CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)?

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.

This definition does not use the terms chronic bronchitis and emphysema* and excludes asthma (reversible airflow limitation).

Symptoms of COPD include:

- Cough
- Sputum production
- Dyspnea on exertion

Episodes of acute worsening of these symptoms often occur.

Chronic cough and sputum production often precede the development of airflow limitation by many years, although not all individuals with cough and sputum production go on to develop COPD.

**Chronic bronchitis*, defined as the presence of cough and sputum production for at least 3 months in each of 2 consecutive years, is not necessarily associated with airflow limitation. *Emphysema*, defined as destruction of the alveoli, is a pathological term that is sometimes (incorrectly) used clinically and describes only one of several structural abnormalities present in patients with COPD.

RISK FACTORS: WHAT CAUSES COPD?

Worldwide, cigarette smoking is the most commonly encountered risk factor for COPD.

The genetic risk factor that is best documented is a severe hereditary deficiency of alpha-1 antitrypsin. It provides a model for how other genetic risk factors are thought to contribute to COPD.

COPD risk is related to the total burden of inhaled particles a person encounters over their lifetime:

- **Tobacco smoke**, including cigarette, pipe, cigar, and other types of tobacco smoking popular in many countries, as well as environmental tobacco smoke (ETS)
- **Occupational dusts and chemicals** (vapors, irritants, and fumes) when the exposures are sufficiently intense or prolonged
- **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
- **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

In addition, any factor that affects lung growth during gestation and childhood (low birth weight, respiratory infections, etc.) has the potential for increasing an individual's risk of developing COPD.

DIAGNOSING COPD

A diagnosis of COPD should be considered in any patient who has dyspnea, chronic cough or sputum production, and/or a history of exposure to risk factors for the disease, especially cigarette smoking (Figure 1).

Figure 1: Key Indicators for Considering a COPD Diagnosis

Consider COPD, and perform spirometry, if any of these indicators are present in an individual over age 40. These indicators are not diagnostic themselves, but the presence of multiple key indicators increases the probability of a diagnosis of COPD.

- **Dyspnea** that is: Progressive (worsens over time).
Usually worse with exercise.
Persistent (present every day).
Described by the patient as an “increased effort to breathe,” “heaviness,” “air hunger,” or “gasping.”
- **Chronic cough:** May be intermittent and may be unproductive.
- **Chronic sputum production:** Any pattern of chronic sputum production may indicate COPD.
- **History of exposure to risk factors:**
 - Tobacco smoke (including popular local preparations).**
 - Occupational dusts and chemicals.
 - Smoke from home cooking and heating fuel.

The diagnosis should be confirmed by spirometry* (Figure 2, page 9 and Appendix I, page 24).

*Where spirometry is unavailable, the diagnosis of COPD should be made using all available tools. Clinical symptoms and signs (abnormal shortness of breath and increased forced expiratory time) can be used to help with the diagnosis. A low peak flow is consistent with COPD but has poor specificity since it can be caused by other lung diseases and by poor performance. In the interest of improving the accuracy of a diagnosis of COPD, every effort should be made to provide access to standardized spirometry.

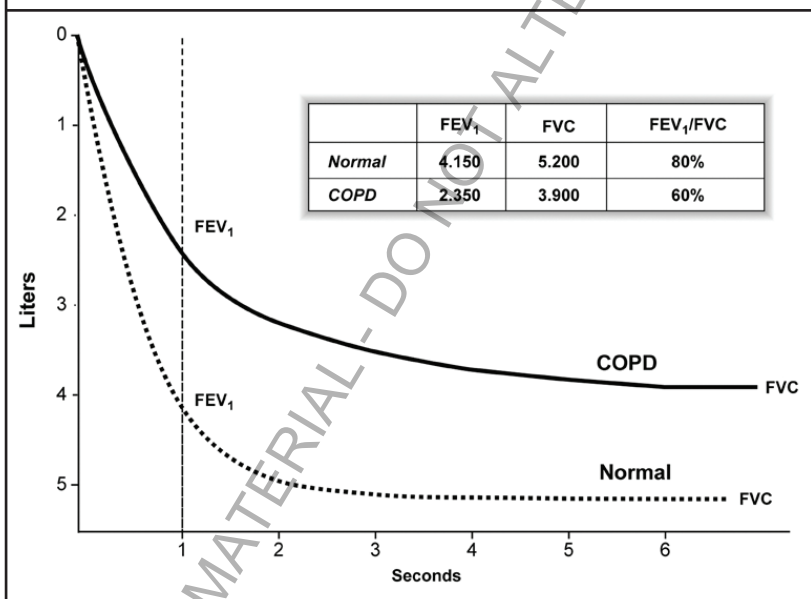
When performing spirometry, measure:

- Forced Vital Capacity (FVC) and
- Forced Expiratory Volume in one second (FEV_1).

Calculate the FEV_1/FVC ratio.

Spirometric results are expressed as % Predicted using appropriate normal values for the person's sex, age, and height.

Figure 2: Normal Spirogram and Spirogram Typical of Patients with Mild to Moderate COPD*



***Postbronchodilator FEV_1 is recommended for the diagnosis and assessment of severity of COPD.**

Patients with COPD typically show a decrease in both FEV_1 and FEV_1/FVC . The degree of spirometric abnormality generally reflects the severity of COPD. However, both symptoms and spirometry should be considered when developing an individualized management strategy for each patient.

Stages of COPD

Stage I: Mild COPD - Mild airflow limitation ($FEV_1/FVC < 70\%$; $FEV_1 \geq 80\%$ predicted) and sometimes, but not always, chronic cough and sputum production.

- At this stage, the individual may not be aware that his or her lung function is abnormal.

Stage II: Moderate COPD - Worsening airflow limitation ($FEV_1/FVC < 70\%$; $50\% \leq FEV_1 < 80\%$ predicted) with shortness of breath typically developing on exertion.

- This is the stage at which patients typically seek medical attention because of chronic respiratory symptoms or an exacerbation of their disease.

Stage III: Severe COPD - Further worsening of airflow limitation ($FEV_1/FVC < 70\%$; $30\% \leq FEV_1 < 50\%$ predicted), greater shortness of breath, reduced exercise capacity, and repeated exacerbations which have an impact on patients' quality of life.

Stage IV: Very Severe COPD - Severe airflow limitation ($FEV_1/FVC < 70\%$; $FEV_1 < 30\%$ predicted) or $FEV_1 < 50\%$ predicted plus chronic respiratory failure. Patients may have *Very Severe (Stage IV) COPD* even if the FEV_1 is $> 30\%$ predicted, whenever this complication is present.

- At this stage, quality of life is very appreciably impaired and exacerbations may be life-threatening.

"At Risk for COPD"

A major objective of GOLD is to increase awareness among health care providers and the general public of the significance of COPD symptoms. The classification of severity of COPD now includes four stages classified by spirometry—*Stage I: Mild COPD*; *Stage II: Moderate COPD*; *Stage III: Severe COPD*; *Stage IV: Very Severe COPD*. A fifth category—*Stage 0: At Risk*—that appeared in the 2001 report is no longer included as a stage of COPD, as there is incomplete evidence that the individuals who meet the definition of "At Risk" (chronic cough and sputum production, normal spirometry) necessarily progress on to *Stage I: Mild COPD*. Nevertheless, the importance of the public health message that chronic cough and sputum are not normal is unchanged and their presence should trigger a search for underlying cause(s).

Differential Diagnosis: A major differential diagnosis is asthma. In some patients with chronic asthma, a clear distinction from COPD is not possible using current imaging and physiological testing techniques. In these patients, current management is similar to that of asthma. Other potential diagnoses are usually easier to distinguish from COPD (**Figure 3**).

Figure 3: Differential Diagnosis of COPD

Diagnosis	Suggestive Features*
COPD	Onset in mid-life. Symptoms slowly progressive. Long smoking history. Dyspnea during exercise. Largely irreversible airflow limitation.
Asthma	Onset early in life (often childhood). Symptoms vary from day to day. Symptoms at night/early morning. Allergy, rhinitis, and/or eczema also present. Family history of asthma. Largely reversible airflow limitation.
Congestive Heart Failure	Fine basilar crackles on auscultation. Chest X-ray shows dilated heart, pulmonary edema. Pulmonary function tests indicate volume restriction, not airflow limitation.
Bronchiectasis	Large volumes of purulent sputum. Commonly associated with bacterial infection. Coarse crackles/clubbing on auscultation. Chest X-ray/CT shows bronchial dilation, bronchial wall thickening.
Tuberculosis	Onset all ages. Chest X-ray shows lung infiltrate or nodular lesions. Microbiological confirmation. High local prevalence of tuberculosis.
Obliterative Bronchiolitis	Onset in younger age, nonsmokers. May have history of rheumatoid arthritis or fume exposure. CT on expiration shows hypodense areas.
Diffuse Panbronchiolitis	Most patients are male and nonsmokers. Almost all have chronic sinusitis. Chest X-ray and HRCT show diffuse small centrilobular nodular opacities and hyperinflation.

*These features tend to be characteristic of the respective diseases, but do not occur in every case. For example, a person who has never smoked may develop COPD (especially in the developing world, where other risk factors may be more important than cigarette smoking); asthma may develop in adult and even elderly patients.

COMPONENTS OF CARE: A COPD MANAGEMENT PROGRAM

The goals of COPD management include:

- Relieve symptoms
- Prevent disease progression
- Improve exercise tolerance
- Improve health status
- Prevent and treat complications
- Prevent and treat exacerbations
- Reduce mortality
- Prevent or minimize side effects from treatment

Cessation of cigarette smoking should be included as a goal throughout the management program.

**THESE GOALS CAN BE ACHIEVED THROUGH
IMPLEMENTATION OF A COPD MANAGEMENT PROGRAM
WITH FOUR COMPONENTS:**

1. **Assess and Monitor Disease**
2. **Reduce Risk Factors**
3. **Manage Stable COPD**
4. **Manage Exacerbations**

Component 1: Assess and Monitor Disease

A detailed medical history of a new patient known or thought to have COPD should assess:

- Exposure to risk factors, including intensity and duration.
- Past medical history, including asthma, allergy, sinusitis or nasal polyps, respiratory infections in childhood, and other respiratory diseases.
- Family history of COPD or other chronic respiratory disease.
- Pattern of symptom development.
- History of exacerbations or previous hospitalizations for respiratory disorder.
- Presence of comorbidities, such as heart disease, malignancies, osteoporosis, and musculoskeletal disorders, which may also contribute to restriction of activity.
- Appropriateness of current medical treatments.
- Impact of disease on patient's life, including limitation of activity; missed work and economic impact; effect on family routines; and feelings of depression or anxiety.
- Social and family support available to the patient.
- Possibilities for reducing risk factors, especially smoking cessation.

In addition to **spirometry**, the following **other tests** may be considered for the assessment of a patient with *Moderate (Stage II)*, *Severe (Stage III)*, and *Very Severe (Stage IV) COPD*.

- **Bronchodilator reversibility testing:** To rule out a diagnosis of asthma, particularly in patients with an atypical history (e.g., asthma in childhood and regular night waking with cough and wheeze).
- **Chest X-ray:** Seldom diagnostic in COPD but valuable to exclude alternative diagnoses such as pulmonary tuberculosis, and identify comorbidities such as cardiac failure.
- **Arterial blood gas measurement:** Perform in patients with $FEV_1 < 50\%$ predicted or with clinical signs suggestive of respiratory failure or right heart failure. The major clinical sign of respiratory failure is cyanosis. Clinical signs of right heart failure include ankle edema and an increase in the jugular venous pressure. Respiratory failure is indicated by $PaO_2 < 8.0$ kPa (60 mm Hg), with or without $PaCO_2 > 6.7$ kPa (50 mm Hg) while breathing air at sea level.
- **Alpha-1 antitrypsin deficiency screening:** Perform when COPD develops in patients of Caucasian descent under 45 years or with a strong family history of COPD.

COPD is usually a progressive disease. Lung function can be expected to worsen over time, even with the best available care. Symptoms and lung function should be monitored to follow the development of complications, to guide treatment, and to facilitate discussion of management options with patients. Comorbidities are common in COPD and should be actively identified.

Component 2: Reduce Risk Factors

Smoking cessation is the single most effective—and cost-effective—intervention to reduce the risk of developing COPD and slow its progression.

- Even a brief, 3-minute period of counseling to urge a smoker to quit can be effective, and at a minimum this should be done for every smoker at every health care provider visit. More intensive strategies increase the likelihood of sustained quitting (**Figure 4**).
- Pharmacotherapy (nicotine replacement, bupropion/nortryptiline, and/or varenicline) is recommended when counseling is not sufficient to help patients stop smoking. Special consideration should be given before using pharmacotherapy in people smoking fewer than 10 cigarettes per day, pregnant women, adolescents, and those with medical contraindications (unstable coronary artery disease, untreated peptic ulcer, and recent myocardial infarction or stroke for nicotine replacement; and history of seizures for bupropion).

Figure 4: Strategy to Help a Patient Quit Smoking

1. **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.
2. **ADVISE:** Strongly urge all tobacco users to quit.
In a clear, strong, and personalized manner, urge every tobacco user to quit.
3. **ASSESS:** Determine willingness 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).
4. **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 if appropriate; provide supplementary materials.
5. **ARRANGE:** Schedule follow-up contact.
Schedule follow-up contact, either in person or via telephone.

Smoking Prevention: Encourage comprehensive tobacco-control policies and programs with clear, consistent, and repeated nonsmoking messages. Work with government officials to pass legislation to establish smoke-free schools, public facilities, and work environments and encourage patients to keep smoke-free homes.

Occupational Exposures: Emphasize primary prevention, which is best achieved by elimination or reduction of exposures to various substances in the workplace. Secondary prevention, achieved through surveillance and early detection, is also important.

Indoor and Outdoor Air Pollution: Implement measures to reduce or avoid indoor air pollution from biomass fuel, burned for cooking and heating in poorly ventilated dwellings. Advise patients to monitor public announcements of air quality and, depending on the severity of their disease, avoid vigorous exercise outdoors or stay indoors altogether during pollution episodes.

Component 3: Manage Stable COPD

Management of stable COPD should be guided by the following general principles:

- Determine disease severity on an individual basis by taking into account the patient's symptoms, airflow limitation, frequency and severity of exacerbations, complications, respiratory failure, comorbidities, and general health status.
- Implement a stepwise treatment plan that reflects this assessment of disease severity.
- Choose treatments according to national and cultural preferences, the patient's skills and preferences, and the local availability of medications.

Patient education can help improve skills, ability to cope with illness, and health status. It is an effective way to accomplish smoking cessation, initiate discussions and understanding of advance directives and end-of-life issues, and improve responses to acute exacerbations.

Pharmacologic treatment (Figure 5) can control and prevent symptoms, reduce the frequency and severity of exacerbations, improve health status, and improve exercise tolerance.

Bronchodilators: These medications are central to symptom management in COPD.

- Inhaled therapy is preferred.
- Give "as needed" to relieve intermittent or worsening symptoms, and on a regular basis to prevent or reduce persistent symptoms.
- The choice between β_2 -agonists, anticholinergics, methylxanthines, and combination therapy depends on the availability of medications and each patient's individual response in terms of both symptom relief and side effects.
- Regular treatment with long-acting bronchodilators, including nebulized formulations, is more effective and convenient than treatment with short-acting bronchodilators.
- Combining bronchodilators of different pharmacologic classes may improve efficacy and decrease the risk of side effects compared to increasing the dose of a single bronchodilator.

Figure 5. Formulations and Typical Doses of COPD Medications*

Drug	Inhaler (µg)	Solution for Nebulizer (mg/ml)	Oral	Vials for Injection (mg)	Duration of Action (hours)
β₂-agonists					
<i>Short-acting</i>					
Fenoterol	100-200 (MDI)	1	0.05% (Syrup)		4-6
Levalbuterol	45-90 (MDI)	0.21, 0.42			6-8
Salbutamol (albuterol)	100, 200 (MDI & DPI)	5	5mg (Pill), 0.024%(Syrup)	0.1, 0.5	4-6
Terbutaline	400, 500 (DPI)		2.5, 5 (Pill)		4-6
<i>Long-acting</i>					
Formoterol	4.5-12 (MDI & DPI)	0.01±			12+
Arformoterol		0.0075			12+
Indacaterol	150-300 (DPI)				24
Salmeterol	25-50 (MDI & DPI)				12+
Anticholinergics					
<i>Short-acting</i>					
Ipratropium bromide	20, 40 (MDI)	0.25-0.5			6-8
Oxitropium bromide	100 (MDI)	1.5			7-9
<i>Long-acting</i>					
Tiotropium	18 (DPI), 5 (SMI)				24+
Combination short-acting β₂-agonists plus anticholinergic in one inhaler					
Fenoterol/Ipratropium	200/80 (MDI)	1.25/0.5			6-8
Salbutamol/Ipratropium	75/15 (MDI)	0.75/0.5			6-8
Methylxanthines					
Aminophylline			200-600 mg (Pill)	240 mg	Variable, up to 24
Theophylline (SR)			100-600 mg (Pill)		Variable, up to 24
Inhaled glucocorticosteroids					
Beclomethasone	50-400 (MDI & DPI)	0.2-0.4			
Budesonide	100, 200, 400 (DPI)	0.20, 0.25, 0.5			
Fluticasone propionate	50-500 (MDI & DPI)				
Combination long-acting β₂-agonists plus glucocorticosteroids in one inhaler					
Formoterol/Budesonide	4.5/160, 9/320 (DPI)				
Salmeterol/Fluticasone propionate	50/100, 250, 500 (DPI) 25/50, 125, 250 (MDI)				
Systemic glucocorticosteroids					
Prednisone			5-60 mg (Pill)		
Methyl-prednisolone			4, 8, 16 mg (Pill)		
Phosphodiesterase-4 inhibitors					
Roflumilast			500 mcg (Pill)		24

MDI=metered dose inhaler; DPI=dry powder inhaler

*Not all formulations are available in all countries; in some countries, other formulations may be available.

±Formoterol nebulized solution is based on the unit dose vial containing 20 µgm in a volume of 2.0ml

Inhaled Glucocorticosteroids: Regular treatment with inhaled glucocorticosteroids does not modify the long-term decline in FEV₁ but has been shown to reduce the frequency of exacerbations and thus improve health status for symptomatic patients with an FEV₁ < 50% predicted and repeated exacerbations (for example, 3 in the last three years). The dose-response relationships and long-term safety of inhaled glucocorticosteroids in COPD are not known. Treatment with inhaled glucocorticosteroids increases the likelihood of pneumonia and does not reduce overall mortality.

An inhaled glucocorticosteroid combined with a long-acting β_2 -agonist is more effective than the individual components in reducing exacerbations and improving lung function and health status. Combination therapy increases the likelihood of pneumonia and has no significant effects on mortality. In patients with an FEV₁ less than 60%, pharmacotherapy with long-acting β_2 -agonist, inhaled glucocorticosteroid and its combination decreases the rate of decline of lung function. Addition of a long-acting β_2 -agonist/inhaled glucocorticosteroid to an anticholinergic (tiotropium) appears to provide additional benefits.

Oral Glucocorticosteroids: Long-term treatment with oral glucocorticosteroids is not recommended.

Phosphodiesterase-4 inhibitors: In patients with *Stage III: Severe COPD* or *Stage IV: Very Severe COPD* and a history of exacerbations and chronic bronchitis, the phosphodiesterase-4 inhibitor, roflumilast, reduces exacerbations treated with oral glucocorticosteroids. These effects are also seen when roflumilast is added to long-acting bronchodilators; there are no comparison studies with inhaled glucocorticosteroids.

Vaccines: *Influenza vaccines reduce serious illness and death in COPD patients by 50%. Vaccines containing killed or live, inactivated viruses are recommended, and should be given once each year. Pneumococcal polysaccharide vaccine is recommended for COPD patients 65 years and older, and has been shown to reduce community-acquired pneumonia in those under age 65 with FEV₁ < 40% predicted.*

Antibiotics: Not recommended except for treatment of infectious exacerbations and other bacterial infections.

Mucolytic (Mucokinetic, Mucoregulator) Agents: Patients with viscous sputum may benefit from mucolytics, but overall benefits are very small. Use is not recommended.

Antitussives: Regular use contraindicated in stable COPD.

Non-Pharmacologic Treatment includes rehabilitation, oxygen therapy, and surgical interventions.

Rehabilitation: Patients at all stages of disease benefit from exercise training programs improvements in exercise tolerance and symptoms of dyspnea and fatigue. Benefits can be sustained even after a single pulmonary rehabilitation program. The minimum length of an effective rehabilitation program is 6 weeks; the longer the program continues, the more effective the results. Benefit does wane after a rehabilitation program ends, but if exercise training is maintained at home the patient's health status remains above pre-rehabilitation levels.

The goals of pulmonary rehabilitation are to reduce symptoms, improve quality of life, and increase participation in everyday activities.

Oxygen Therapy: The long-term administration of oxygen (>15 hours per day) to patients with chronic respiratory failure increases survival and has a beneficial impact on pulmonary hemodynamics, hematologic characteristics, exercise capacity, lung mechanics, and mental state.

Initiate oxygen therapy for patients with *Stage IV: Very Severe COPD* if:

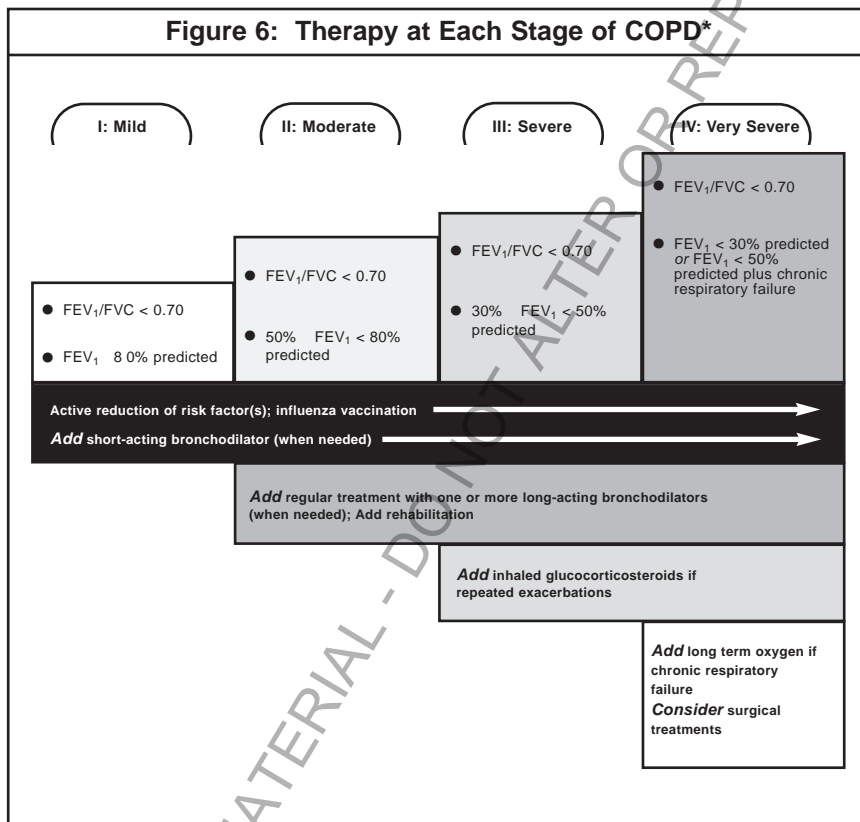
- PaO₂ is at or below 7.3 kPa (55 mm Hg) or SaO₂ is at or below 88%, with or without hypercapnia; or
- PO₂ is between 7.3 kPa (55 mm Hg) and 8.0 kPa (60 mm Hg) or SaO₂ is 88%, if there is evidence of pulmonary hypertension, peripheral edema suggesting congestive heart failure, or polycythemia (hematocrit > 55%).

The goal of long-term oxygen therapy is to increase the baseline PaO₂ at rest to at least 8.0 kPa (60 mm Hg) at sea level, and/or produce SaO₂ at least 90%, which will preserve vital organ function by ensuring an adequate delivery of oxygen.

Surgical Treatments: Bullectomy and lung transplantation may be considered in carefully selected patients with *Stage IV: Very Severe COPD*. There is currently no sufficient evidence that would support the widespread use of lung volume reduction surgery (LVRS).

There is no convincing evidence that mechanical ventilatory support has a role in the routine management of stable COPD.

A summary of characteristics and recommended treatment at each stage of COPD is shown in **Figure 6**.



***Postbronchodilator FEV₁ is recommended for the diagnosis and assessment of severity of COPD.**

Component 4: Manage Exacerbations

An exacerbation of COPD is defined as *an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough, and/or sputum that is beyond normal day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD.*

The most common causes of an exacerbation are infection of the tracheobronchial tree and air pollution, but the cause of about one-third of severe exacerbations cannot be identified.

How to Assess the Severity of an Exacerbation

Arterial blood gas measurements (in hospital):

- $\text{PaO}_2 < 8.0 \text{ kPa}$ (60 mm Hg) and/or $\text{SaO}_2 < 90\%$ with or without $\text{PaCO}_2 > 6.7 \text{ kPa}$, (50 mmHg) when breathing room air indicates respiratory failure.
- Moderate-to-severe acidosis ($\text{pH} < 7.36$) plus hypercapnia ($\text{PaCO}_2 > 6-8 \text{ kPa}$, 45-60 mm Hg) in a patient with respiratory failure is an indication for mechanical ventilation.

Chest X-ray: Chest radiographs (posterior/anterior plus lateral) identify alternative diagnoses that can mimic the symptoms of an exacerbation.

ECG: Aids in the diagnosis of right ventricular hypertrophy, arrhythmias, and ischemic episodes.

Other laboratory tests:

- Sputum culture and antibiogram to identify infection if there is no response to initial antibiotic treatment.
- Biochemical tests to detect electrolyte disturbances, diabetes, and poor nutrition.
- Whole blood count can identify polycythemia or bleeding.

Home Care or Hospital Care for End-Stage COPD Patients?

The risk of dying from an exacerbation of COPD is closely related to the development of respiratory acidosis, the presence of serious comorbidities, and the need for ventilatory support. Patients lacking these features are not at high risk of dying, but those with severe underlying COPD often require hospitalization in any case. Attempts at managing such patients entirely in the community have met with limited success, but returning them to their homes with increased social support and a supervised medical care program after an initial emergency room assessment has been much more successful. However, detailed cost-benefit analyses of these approaches have not been reported.

Home Management

Bronchodilators: Increase dose and/or frequency of existing short-acting bronchodilator therapy, preferably with β_2 -agonists. If not already used, add anticholinergics until symptoms improve.

Glucocorticosteroids: If baseline $FEV_1 < 50\%$ predicted, add 30-40 mg oral prednisolone per day for 7-10 days to the bronchodilator regimen. Budesonide alone may be an alternative to oral glucocorticosteroids in the treatment of exacerbations and is associated with significant reduction of complications.

Hospital Management

Patients with the characteristics listed in **Figure 7** should be hospitalized. Indications for referral and the management of exacerbations of COPD in the hospital depend on local resources and the facilities of the local hospital.

Figure 7: Indications for Hospital Admission for Exacerbations

- | | |
|---|--|
| <ul style="list-style-type: none">• Marked increase in intensity of symptoms, such as sudden development of resting dyspnea• Severe underlying COPD• Onset of new physical signs (e.g., cyanosis, peripheral edema) | <ul style="list-style-type: none">• Failure of exacerbation to respond to initial medical management• Significant comorbidities• Frequent exacerbations• Newly occurring arrhythmias• Diagnostic uncertainty• Older age• Insufficient home support |
|---|--|

Antibiotics: Antibiotics should be given to patients:

- With the following three cardinal symptoms: increased dyspnea, increased sputum volume, increased sputum purulence
- With increased sputum purulence and one other cardinal symptom
- Who require mechanical ventilation

APPENDIX I: SPIROMETRY FOR DIAGNOSIS OF COPD

Spirometry is as important for the diagnosis of COPD as blood pressure measurements are for the diagnosis of hypertension. Spirometry should be available to all health care professionals.

What is Spirometry?

Spirometry is a simple test to measure the amount of air a person can breathe out, and the amount of time taken to do so.

A **spirometer** is a device used to measure how effectively, and how quickly, the lungs can be emptied.

A **spirogram** is a volume-time curve.

Spirometry measurements used for diagnosis of COPD include (see Figure 2, page 9):

- **FVC** (Forced Vital Capacity): maximum volume of air that can be exhaled during a forced maneuver.
- **FEV₁** (Forced Expired Volume in one second): volume expired in the first second of maximal expiration after a maximal inspiration. This is a measure of how quickly the lungs can be emptied.
- **FEV₁/FVC**: FEV₁ expressed as a percentage of the FVC, gives a clinically useful index of airflow limitation.

The ratio FEV₁/FVC is between 70% and 80% in normal adults; a value less than 70% indicates airflow limitation and the possibility of COPD.

FEV₁ is influenced by the age, sex, height and ethnicity, and is best considered as a percentage of the predicted normal value. There is a vast literature on normal values; those appropriate for local populations should be used^{1,2,3}.

Why do Spirometry for COPD?

- Spirometry is needed to make a firm diagnosis of COPD.
- Together with the presence of symptoms, spirometry helps stage COPD severity and can be a guide to specific treatment steps.
- A normal value for spirometry effectively excludes the diagnosis of clinically relevant COPD.
- The lower the percentage predicted FEV₁, the worse the subsequent prognosis.
- FEV₁ declines over time and faster in COPD than in healthy subjects. Spirometry can be used to monitor disease progression, but to be reliable the intervals between measurements must be at least 12 months.

What You Need to Perform Spirometry

Several types of spirometers are available:

- relatively large bellows or rolling-seal spirometers (usually only available in pulmonary function laboratories). Calibration should be checked against a known volume e.g. from a 3-litre syringe on a regular basis.
- smaller hand-held devices, often with electronic calibration systems.

A hard copy of the volume time plot is very useful to check optimal performance and interpretation, and to exclude errors.

Most spirometers require electrical power to permit operation of the motor and/or sensors. Some battery operated versions are available that can dock with a computer to provide hard copy.

It is essential to learn how your machine is calibrated and when and how to clean it.

How to Perform Spirometry

Spirometry is best performed with the patient seated. Patients may be anxious about performing the tests properly, and should be reassured. Careful explanation of the test, accompanied by a demonstration, is very useful. The patient should:

- Breathe in fully.
- Seal their lips around the mouthpiece.
- Force the air out of the chest as hard and fast as they can until their lungs are completely "empty."
- Breathe in again and relax.

Exhalation must continue until no more air can be exhaled, must be at least 6 seconds, and can take up to 15 seconds or more.

Like any test, spirometry results will only be of value if the expirations are performed satisfactorily and consistently. Both FVC and FEV₁ should be the largest value obtained from any of 3 technically satisfactory curves and the FVC and FEV₁ values in these three curves should vary by no more than 5% or 100 ml, whichever is greater. The FEV₁/FVC is calculated using the maximum FEV₁ and FVC from technically acceptable (not necessarily the same) curves.

Those with chest pain or frequent cough may be unable to perform a satisfactory test and this should be noted.

Where to find more detailed information on spirometry

1. **American Thoracic Society**
<http://www.thoracic.org/adobe/statements/spirometry1-30.pdf>
2. **Australian/New Zealand Thoracic Society**
<http://www.nationalasthma.org.au/publications/spiro/index.htm>
3. **British Thoracic Society**
<http://www.brit-thoracic.org.uk/copd/consortium.html>
4. **GOLD**
A spirometry guide for general practitioners and a teaching slide set is available:
<http://www.goldcopd.org>

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