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# Global Initiative for Chronic Obstructive Lung Disease



## **POCKET GUIDE TO COPD DIAGNOSIS, MANAGEMENT, AND PREVENTION**

A Guide for Physicians and Nurses

**UPDATED JULY, 2003**

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**BASED ON THE NHLBI/WHO WORKSHOP REPORT, GLOBAL STRATEGY FOR THE DIAGNOSIS,  
MANAGEMENT, AND PREVENTION OF COPD**

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NATIONAL INSTITUTES OF HEALTH  
NATIONAL HEART, LUNG, AND BLOOD INSTITUTE

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# **Global Initiative for Chronic Obstructive Lung Disease**

**Pocket Guide to COPD Diagnosis, Management, and Prevention**  
**U.S. Department of Health and Human Services**  
**Public Health Service**  
**National Institutes of Health**  
**National Heart, Lung, and Blood Institute**



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# PREFACE

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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 recent and 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 is conducted in collaboration with the U.S. National Heart, Lung, and Blood Institute and the World Health Organization. Publications include:

- NHLBI/WHO Workshop Report: *Global Strategy for the Diagnosis, Management, and Prevention of COPD*. Scientific information and recommendations for COPD programs. (Updated July 2003)
- Executive Summary, NHLBI/WHO Workshop Report: *Global Strategy for the Diagnosis, Management, and Prevention of COPD*. (Updated July 2003)
- *Pocket Guide to COPD Management and Prevention*. Summary of patient care information for primary health care professionals. (Updated July 2003)
- *What You and Your Family Can Do About COPD*. Information booklet for patients and their families.

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These publications are available on the Internet at <http://www.gold-copd.com>. This site provides links to other websites with information about COPD.

This Pocket Guide has been developed from the NHLBI/WHO Workshop Report, *Global Strategy for the Diagnosis, Management, and Prevention of COPD*. (Updated 2003) Technical discussions of COPD and COPD management, evidence levels, and specific citations from the scientific literature are included in the Workshop Report.

**Acknowledgements:** Grateful acknowledgement is given for the educational grants from Altana Pharma, Andi-Ventis, AstraZeneca, Aventis, Bayer, Boehringer Ingelheim, Chiesi, GlaxoSmithKline, Merck, Sharp & Dohme, Mitsubishi-Tokyo, Nikken Chemicals, Novartis, Pfizer, Schering-Plough, and Zambon. The generous contributions of these companies assured that the workshop participants could meet together and publications could be printed for wide distribution. The workshop participants, however, are solely responsible for the statements and conclusions in the publications.

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## KEY POINTS

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- **Chronic Obstructive Pulmonary Disease (COPD)** is a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.
- The most important **risk factor** for COPD is **cigarette smoking**. Pipe, cigar, and other types of tobacco smoking popular in many countries are also risk factors for COPD. **At every possible opportunity individuals who smoke should be encouraged to quit.**
- A **diagnosis** of COPD should be considered in any individual with symptoms and a history of exposure to risk factors. 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 improve and prevent 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.

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# WHAT IS CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)?

**Chronic Obstructive Pulmonary Disease (COPD)** is a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs 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.**

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\**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.

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# RISK FACTORS: WHAT CAUSES COPD?

**Tobacco Smoke:** The most important risk factor for COPD is cigarette smoking. Pipe, cigar, and other types of tobacco smoking popular in many countries are also risk factors for COPD.

**Other documented causes of COPD include:**

- 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.
- Outdoor air pollution, adds to the lungs' total burden of inhaled particles, although its specific role in causing COPD is not well understood.

**Passive exposure to cigarette smoke also contributes to respiratory symptoms and COPD.**

Respiratory infections in early childhood are associated with reduced lung function and increased respiratory symptoms in adulthood.

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# DIAGNOSING COPD

A diagnosis of COPD should be considered in any individual who presents characteristic symptoms and a history of exposure to risk factors for the disease, especially cigarette smoking (**Figure 1**).

**Figure 1: Key Indicators for Considering a COPD Diagnosis**

- **Chronic cough:** Present intermittently or every day.  
Often present throughout the day; seldom only nocturnal.
- **Chronic sputum production:**  
Any pattern of chronic sputum production may indicate COPD.
- **Acute bronchitis:** Repeated episodes.
- **Dyspnea that is:** Progressive (worsens over time).  
Persistent (present every day).  
Worse on exercise.  
Worse during respiratory infections.
- **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**).

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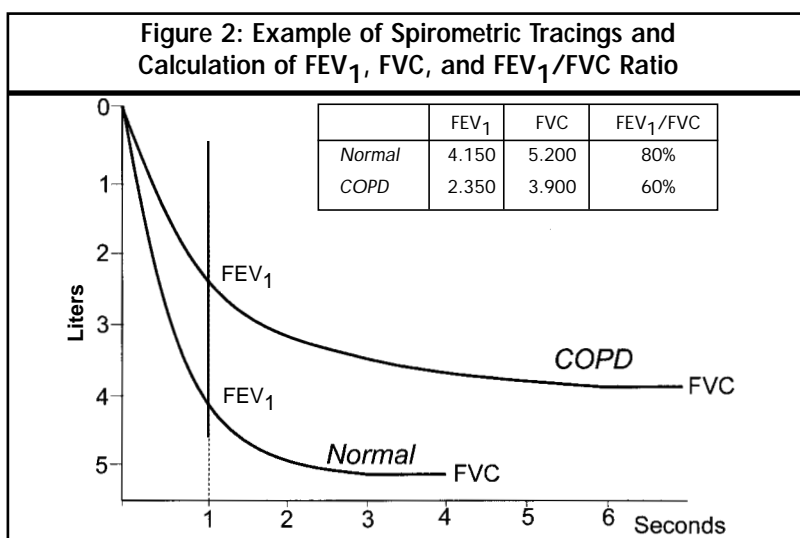
\*Where spirometry is unavailable, the diagnosis of COPD should be made using all available tools. Clinical symptoms and signs (abnormal shortness of breath and increased forced expiratory time) can be used to help with the diagnosis. A low peak flow is consistent with COPD but has poor specificity since 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 **V**ital **C**apacity (**FVC**) and
- Forced **E**xpiratory **V**olume in one second (**FEV<sub>1</sub>**).

Calculate the FEV<sub>1</sub>/FVC ratio.

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



**Patients with COPD typically show a decrease in both FEV<sub>1</sub> and FEV<sub>1</sub>/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.**

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## Classification of COPD by Severity

**Stage 0: At Risk** - Chronic cough and sputum production; lung function is still normal.

**Stage I: Mild COPD** - Mild airflow limitation ( $FEV_1/FVC < 70\%$  but  $FEV_1 \geq 80\%$  predicted) and usually, 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 ( $50\% \leq FEV_1 < 80\%$  predicted), and usually the progression of symptoms, with shortness of breath typically developing on exertion.

**Stage III: Severe COPD** - Further worsening of airflow limitation ( $30\% \leq FEV_1 < 50\%$  predicted), increased shortness of breath, and repeated exacerbations which have an impact on patients' quality of life.

- Exacerbations of symptoms, which have an impact on a patient's quality of life and prognosis, are especially seen in patients with  $FEV_1 < 50\%$  predicted.

**Stage IV: Very Severe COPD** - Severe airflow limitation ( $FEV_1 < 30\%$  predicted) or the presence of chronic respiratory failure. Patients may have very severe (Stage IV) COPD even if the  $FEV_1$  is  $> 30\%$  predicted, whenever these complications are present.

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

**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**).

<b>Figure 3: Differential Diagnosis of COPD</b>	
<b>Diagnosis</b>	<b>Suggestive Features*</b>
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.

\*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.

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# COMPONENTS OF CARE: A COPD MANAGEMENT PROGRAM

The goals of COPD management include:

- Prevent disease progression
- Relieve symptoms
- 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**

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## 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, 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 and rheumatic disease, that 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.

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In addition to **spirometry**, the following **other tests** should be undertaken 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 and guide initial treatment decisions.
- **Inhaled glucocorticosteroid trial (6 weeks to 3 months):**  
To identify patients with airflow limitation that is responsive to inhaled glucocorticosteroid treatment. If objective benefit is not demonstrated, inhaled glucocorticosteroid should be discontinued.
- **Chest X-ray:** Seldom diagnostic in COPD but valuable to exclude alternative diagnoses, e.g., pulmonary tuberculosis.
- **Arterial blood gas measurement:** Perform in patients with  $FEV_1 < 40\%$  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 under 45 years, or in patients 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.**

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## 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 visit. More intensive strategies increase the likelihood of sustained quitting (**Figure 4**).
- Pharmacotherapy (nicotine replacement and/or bupropion) 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.*

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**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.

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## 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 improve 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.

- Give "as-needed" to relieve intermittent or worsening symptoms, and on a regular basis to prevent or reduce persistent symptoms.
- The choice between  $\beta_2$ -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 is more effective and convenient than treatment with short-acting bronchodilators, but more expensive.
- Combining drugs with different mechanisms and durations of action may increase the degree of bronchodilation for equivalent or lesser side effects.
- Theophylline is effective in COPD, but due to its potential toxicity inhaled bronchodilators are preferred when available.

<b>Figure 5: Commonly Used Formulations of Drugs for COPD</b>					
<b>Drug</b>	<b>Inhaler (<math>\mu</math>g)</b>	<b>Solution for Nebulizer (mg/ml)</b>	<b>Oral</b>	<b>Vials for Injection (mg)</b>	<b>Duration of Action (hours)</b>
<b><i><math>\beta_2</math>-agonists</i></b>					
<b>Short-acting</b>					
Fenoterol	100-200 (MDI)	1	0.05% (Syrup)		4-6
Salbutamol (albuterol)	100, 200 (MDI & DPI)	5	5mg (Pill) Syrup 0.024%	0.1, 0.5	4-6
Terbutaline	400, 500 (DPI)	—	2.5, 5 (Pill)	0.2, 0.25	4-6
<b>Long-acting</b>					
Formoterol	4.5—12 (MDI & DPI)				12+
Salmeterol	25-50 (MDI & DPI)				12+
<b><i>Anticholinergics</i></b>					
<b>Short-acting</b>					
Ipratropium bromide	20, 40 (MDI)	0.25-0.5			6-8
Oxitropium bromide	100 (MDI)	1.5			7-9
<b>Long-acting</b>					
Tiotropium	18 (DPI)				24+
<b><i>Combination short-acting <math>\beta_2</math>-agonists plus anticholinergic in one inhaler</i></b>					
Fenoterol/ Ipratropium	200/80 (MDI)	1.25/0.5			6-8
Salbutamol/ Ipratropium	75/15 (MDI)	0.75/4.5			6-8
<b><i>Methylxanthines</i></b>					
Aminophylline			200-600 mg (Pill)	240 mg	Variable, up to 24
Theophylline (SR)			100-600 mg (Pill)		Variable, up to 24
<b><i>Inhaled glucocorticosteroids</i></b>					
Beclomethasone	100, 250, 400 (MDI & DPI)	0.2-0.4			
Budesonide	100, 200, 400 (DPI)	0.20, 0.25, 0.5			
Fluticasone	50-500 (MDI & DPI)				
Triamcinolone	100 (MDI)	40		40	
<b><i>Combination long-acting <math>\beta_2</math>-agonists plus glucocorticosteroids in one inhaler</i></b>					
Formoterol/ Budesonide	4.5/80, 160 (DPI) (9/320) (DPI)				
Salmeterol/ Fluticasone	50/100, 250, 500 (DPI) 25/50, 125, 250 (MDI)				
<b><i>Systemic glucocorticosteroids</i></b>					
Prednisone			5-60 mg (Pill)		
Methyl- prednisolone	10-2000 mg		4, 8, 16 mg (Pill)		

MDI=metered dose inhaler; DPI=dry powder inhaler

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Regular nebulized bronchodilator therapy for a stable patient is not appropriate unless it has been shown to be better than conventional doses by metered dose inhaler.

**Glucocorticosteroids:** Regular treatment with inhaled glucocorticosteroids is only appropriate for patients with:

- symptomatic improvement and a documented spirometric response to inhaled glucocorticosteroids or
- an FEV<sub>1</sub> < 50% predicted and repeated exacerbations (for example, 3 in the last three years).

Prolonged treatment with inhaled glucocorticosteroids may relieve symptoms in this carefully selected group of patients but does not modify the long-term decline in FEV<sub>1</sub>. The dose-response relationships and long-term safety of inhaled glucocorticosteroids in COPD are not known. Long-term treatment with oral glucocorticosteroids is not recommended.

**Vaccines:** *Influenza* vaccines reduce serious illness and death in COPD patients by 50%. Give once (in Autumn) or twice (in Autumn and Winter) each year. There is no evidence for recommending the general use of pneumococcal vaccine for COPD.

**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.

**Respiratory Stimulants:** Not recommended for regular use.

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

**Rehabilitation** programs should include, at a minimum:

- Exercise training
- Nutrition counseling
- Education.

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

Patients at all stages of disease benefit from exercise training programs, with improvements in exercise tolerance and symptoms of dyspnea and fatigue. Benefits can be sustained even after a single pulmonary rehabilitation program, whether it is conducted in an inpatient, outpatient, or home setting. The minimum length of an effective rehabilitation program is two months; the longer the program continues, the more effective the results

**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 arterial pressure, polycythemia (hematocrit > 55%), exercise capacity, lung mechanics, and mental state.

**The goal of long-term oxygen therapy is to increase the baseline PaO<sub>2</sub> at rest to at least 8.0 kPa (60 mm Hg) at sea level, and/or produce SaO<sub>2</sub> at least 90%, which will preserve vital organ function by ensuring an adequate delivery of oxygen.**

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

- PaO<sub>2</sub> is at or below 7.3 kPa (55 mm Hg) or SaO<sub>2</sub> is at or below 88%, with or without hypercapnia; or
- PO<sub>2</sub> is between 7.3 kPa (55 mm Hg) and 8.0 kPa (60 mm Hg) or SaO<sub>2</sub> is 89%, if there is evidence of pulmonary hypertension, peripheral edema suggesting congestive heart failure, or polycythemia.

**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**.

<b>Figure 6 - Therapy at Each Stage of COPD</b>					
<b>Old</b>	<b>0: At Risk</b>	<b>I: Mild</b>	<b>II: Moderate</b>		<b>III: Severe</b>
<b>New</b>	<b>0: At Risk</b>	<b>I: Mild</b>	<b>II: Moderate</b>	<b>III: Severe</b>	<b>IV: Very Severe</b>
Characteristics	<ul style="list-style-type: none"> <li>• Chronic symptoms</li> <li>• Exposure to risk factors</li> <li>• Normal spirometry</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub>/FVC &lt; 70%</li> <li>• FEV<sub>1</sub> ≥ 80%</li> <li>• With or without symptoms</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub>/FVC &lt; 70%</li> <li>• 50% ≤ FEV<sub>1</sub> &lt; 80%</li> <li>• With or without symptoms</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub>/FVC &lt; 70%</li> <li>• 30% ≤ FEV<sub>1</sub> &lt; 50%</li> <li>• With or without symptoms</li> </ul>	<ul style="list-style-type: none"> <li>• FEV<sub>1</sub>/FVC &lt; 70%</li> <li>• FEV<sub>1</sub> &lt; 30% or presence of chronic respiratory failure or right heart failure</li> </ul>
	Avoidance of risk factor(s); influenza vaccination				
		Add short-acting bronchodilator when needed			
			Add regular treatment with one or more long-acting bronchodilators Add rehabilitation		
				Add inhaled glucocorticosteroids if repeated exacerbations	
					Add long-term oxygen if chronic respiratory failure Consider surgical treatments

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## Component 4: Manage Exacerbations

***COPD is often associated with exacerbations of symptoms. Many exacerbations are caused by infection of the tracheobronchial tree or an increase in air pollution, but the cause of about one-third of severe exacerbations cannot be identified.***

### How to Assess the Severity of an Exacerbation

*Lung function tests (may be difficult for sick patients to perform):*

- PEF < 100 L/min or FEV<sub>1</sub> < 1 L indicates a severe exacerbation.

*Arterial blood gas measurements (in hospital):*

- PaO<sub>2</sub> < 8.0 kPa (60 mm Hg) and/or SaO<sub>2</sub> < 90% with or without PaCO<sub>2</sub> > 6.7kPa, (50 mmHg) when breathing room air indicates respiratory failure.
- PaO<sub>2</sub> < 6.7 kPa (50 mm Hg), PaCO<sub>2</sub> > 9.3 kPa (70 mm Hg), and pH < 7.30 suggest a life-threatening episode that needs close monitoring or critical management.

*Chest X-ray:* Chest radiographs (posterior/anterior plus lateral) identify complications such as pneumonia and 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.

## 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 bronchodilator therapy. If not already used, add anticholinergics until symptoms improve.

**Glucocorticosteroids:** If baseline FEV<sub>1</sub> < 50% predicted, add 40 mg oral prednisolone per day for 10 days to the bronchodilator regimen. Nebulized abudesonide may be an alternative to oral glucocorticosteroids in the treatment of nonacidotic exacerbations.

**Antibiotics:** When symptoms of breathlessness and cough are increased and sputum is purulent and increased in volume, provide antibiotic coverage of the major bacterial pathogens involved in exacerbations, taking into account local patterns of antibiotic sensitivity.

### 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"> <li>• Marked increase in intensity of symptoms, such as sudden development of resting dyspnea</li> <li>• Severe background COPD</li> <li>• Onset of new physical signs (e.g., cyanosis, peripheral edema)</li> </ul> | <ul style="list-style-type: none"> <li>• Failure of exacerbation to respond to initial medical management</li> <li>• Significant comorbidities</li> <li>• Newly occurring arrhythmias</li> <li>• Diagnostic uncertainty</li> <li>• Older age</li> <li>• Insufficient home support</li> </ul> |
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# 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.
- **FEV<sub>1</sub>** (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.
- **FEV<sub>1</sub>/FVC**: FEV<sub>1</sub> expressed as a percentage of the FVC, gives a clinically useful index of airflow limitation.

The ratio FEV<sub>1</sub>/FVC is between 70% and 80% in normal adults; a value less than 70% indicates airflow limitation and the possibility of COPD.

FEV<sub>1</sub> 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<sup>1,2,3</sup>.

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## 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 FEV<sub>1</sub>, the worse the subsequent prognosis.
- FEV<sub>1</sub> 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.

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***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 FEV<sub>1</sub> should be the largest value obtained from any of 3 technically satisfactory curves and the FVC and FEV<sub>1</sub> values in these three curves should vary by no more than 5% or 100 ml, whichever is greater. The FEV<sub>1</sub>/FVC is calculated using the maximum FEV<sub>1</sub> 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.

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## 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>

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# NOTES