NEEDS ASSESSMENT
Chronic obstructive pulmonary disease (COPD), a progressive lung disease characterized by airflow limitation that is not fully reversible, is the fourth leading cause of death in the United States. Primarily caused by cigarette smoking, COPD affects nearly 10 million Americans, although it largely remains under-recognized and undiagnosed. Because the effects of COPD are more easily treated in the earlier stages of disease progression, primary care clinicians can play a crucial role in patient care by recognizing and diagnosing COPD, educating and counseling patients, and prescribing optimal pharmacologic and nonpharmacologic therapies.


LEARNING OBJECTIVES
After completing this activity, participants should be better able to:
• Cite the prevalence of COPD and burden of disease
• Identify the symptoms of COPD
• Describe available diagnostic tests
• Explain nonpharmacologic management and techniques for patient education about COPD
• Describe the available pharmacologic treatments for COPD

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Dennis E. Niewoehner, MD, serves as a consultant, receives grant support, and is on the speakers’ bureau for Boehringer Ingelheim; is on the speakers’ bureau for Pfizer Inc.; and serves as a consultant for Adams Respiratory Therapeutics, Forest Laboratories, and GlaxoSmithKline. Claire Murphy, RN, MSN, NP-C, has nothing to disclose with regard to commercial support. Mary Ettari, MPH, PA-C, has nothing to disclose with regard to commercial support. Roy C. Blank, MD, is on the speakers’ bureau for Merck, Pfizer Inc. and Takeda Pharmaceutical Company Ltd. Jason Worcester, MD, has nothing to disclose with regard to commercial support. The use of formoterol, salmeterol, or tiotropium for managing exacerbations of COPD is an off-label/unapproved use.

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COPD: Practical Strategies for Improved Diagnosis and Effective Management

Health care providers are under increasing pressure to improve the quality of care they provide to patients. With Pay for Performance, the Center of Medicare and Medicaid Services now pays providers based on their performance within specific measures. The American Board of Medical Specialties mandates that all specialty boards add performance improvement as a requirement for Maintenance of Certification by 2010. In 2004, the American Medical Association provided criteria for offering continuing medical education (CME) credits for performance improvement activities. Health care providers can now look to CME initiatives to guide them in efforts to make meaningful improvements in their practices.

BUSM Mentor QI® Activity: Performance Improvement Program

Boston University School of Medicine’s performance improvement initiative COPD: Strategies for Diagnosis and Effective Management was designed to help primary care clinicians improve their diagnosis and management of patients with chronic obstructive pulmonary disease (COPD). Through a Web-based interface, participants complete an initial practice assessment through a review of 10 patient charts and develop an action plan based on suggested interventions. Three months later, they conduct a follow-up assessment by completing another review of 10 patient charts. A resource library and list of suggested quality improvement interventions are hosted on the dedicated Web site, MENTORQI.com. Participants also have access to faculty mentors who can answer questions and offer advice.

This monograph aims to disseminate the lessons learned from the COPD performance improvement initiative and to provide the latest information about COPD and practical strategies to improve diagnosis and management.

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COPD: Practical Strategies for Improved Diagnosis and Effective Management

PERSPECTIVES ON COPD
Chronic obstructive pulmonary disease (COPD) is defined as a disease state that is both preventable and treatable and is characterized by the presence of airflow obstruction that is not fully reversible. Airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity, and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases. Emphysema and chronic bronchitis are the most common forms of COPD and often coexist.

A leading cause of morbidity and mortality in the United States and worldwide, COPD is nevertheless frequently underdiagnosed and misdiagnosed. The primary etiology is cigarette smoking. Other established causes of COPD include occupational exposure to noxious gases and particles, exposure to secondhand smoke, and hereditary alpha-1-antitrypsin deficiency.

COPD is characterized by a decline in lung function and an increase in symptoms and exacerbations that ultimately affects survival. Exacerbations involve a rapid worsening of symptoms that requires medical intervention. The goal of outpatient treatment and management is to prevent exacerbations and relieve symptoms, while decelerating the decline of lung function.

EPIDEMIOLOGY
Prevalence: An Underestimated Disease
COPD is the fourth leading cause of death in the United States; in 2005, chronic diseases of the lower respiratory tract were responsible for nearly 131,000 deaths. The prevalence and morbidity of COPD are believed to be significantly underestimated. In 2000, although an estimated 10 million American adults reported physician-diagnosed COPD, data from the National Health and Nutrition Examination Surveys (NHANES) III estimated that 24 million US adults showed evidence of impaired lung function, indicating that COPD is probably significantly underdiagnosed.

The World Health Organization predicts that by 2030, COPD will become the fourth leading cause of death worldwide. In the United States, COPD was responsible for 8 million physician office and hospital outpatient visits, 1.5 million emergency department visits due to disease exacerbation, and 726,000 hospitalizations in the year 2000. Approximately 2% of all hospitalizations in the United States are because of COPD.

Gender: Increasing Incidence in Women
Historically, COPD has been seen as a male disease, but the incidence of COPD has been increasing among women...
The death rate for women more than doubled between 1980 and 2000, from 20.1/100,000 in 1980 to 56.7/100,000 in 2000. Conversely, the mortality rate for men during the same time period increased only slightly, from 73.0/100,000 in 1980 to 82.6/100,000 in 2000. For the first time, the number of COPD-related deaths among women surpassed that for men in 2000 (Figure 1), and COPD hospitalizations of women outnumbered those of men (404,000 vs. 322,000). The growing prevalence of COPD among women in developed nations is believed to reflect changing patterns of tobacco use, and some data now suggest that women may be more susceptible to the effects of tobacco than men.

The Age Factor
COPD has traditionally been considered a disease that affects elderly individuals. Although some data have indicated that the typical COPD patient is older than age 65, other studies have found that COPD does affect younger persons, including working-age adults. Tinkelman and Corsello reported that the stereotype of a COPD patient as being unemployed and of advanced age might not apply to a large segment of patients who have already been diagnosed with the disease. They found that nearly half (49.7%) of these patients, who were drawn from a large dataset of individuals enrolled in a disease management program for COPD, were younger than age 65. A large portion (46.1%) was also employed and had missed an average of 4.6 days of work in the previous 6 months because of COPD.

The Significance of Race
In 2001, prevalence of COPD was higher for whites than for blacks across the 25- to 44-year age groups and the 65-year and older age groups. Among individuals aged 45-64 years, chronic bronchitis was more prevalent in women, with black females having the highest prevalence. For emphysema, the highest prevalence was seen in white men and the lowest in black women.

COPD death rates were also consistently higher among white than among black Americans between 1995 and 2000. In 2000, the death rate was 63% higher in whites than in blacks (70.1 vs. 42.9 per 100,000 population). Although black Americans tend to have an overall lower prevalence of COPD and related mortality, they also tend to have a higher rate of COPD-related hospitalizations and emergency room visits.

Direct and Indirect Costs of COPD
The economic burden of COPD in the United States is significant. In 2002, the total cost of COPD was estimated to be $32.1 billion, with direct medical interventions accounting for $18 billion and indirect expenses accounting for $14.4 billion. Nonpulmonary comorbidities are common in persons with COPD. Cardiovascular and chronic lung diseases are frequently concurrent. Studies show that only a small decrease in expiratory flow volumes raises the risk for stroke, ischemic heart diseases, and sudden cardiac deaths 2- to 3-fold, even in the absence of other risk factors. Other systemic effects of COPD include osteoporosis, weight loss and muscle wasting, and depression.

Risk Factors
The risk factors for COPD are multifaceted. The disease is probably caused by an interaction of factors that are intrinsic to the host and environmental exposures. Cigarette smoking is the most commonly encountered risk factor for COPD. However, among the COPD patient populations of participants in the Boston University School of Medicine (BUSM) current web-based MENTOR QI activity, 16% of patients were NOT queried in the last year about smoking, indicating a need for greater awareness about assessment techniques. (See page 11 for information about smoking cessation.)

Although not all people who smoke or use tobacco develop COPD, the absolute risk of developing COPD after 25 years of continuous smoking is at least 25%. Other inhalation risk factors include exposure to secondhand tobacco smoke, air pollution, occupational dusts and chemicals, and indoor air pollution, including smoke from home cooking and heating. Other probable risk factors include low birth weight, a history of childhood respiratory infections, low socioeconomic status, airway hyperreactivity, IgA deficiency, and asthma. The only established genetic risk factor thus far is a deficiency in a trypsin antagonist known as alpha 1-antitrypsin. A familial risk for COPD has been observed, suggesting that genetic factors may influence susceptibility.

DETECTION AND ACCURATE DIAGNOSIS OF COPD
COPD is both underdiagnosed and often misdiagnosed. The diagnosis of COPD is generally based on the presence of air-
flow obstruction that is not fully reversible and on a history of exposure to risk factors. The defining feature of COPD is irreversible airflow limitation that occurs during forced expiration, which may be the result of a loss of elastic recoil or of an increase in the resistance of the conducting airways.1

COPD should be suspected on the basis of the patient's medical history and physical examination; chest radiography can be helpful with the differential diagnosis.15 Confirmation of the diagnosis requires the use of spirometry to measure the forced expiratory volume in 1 second (FEV1) and its ratio to forced vital capacity (FVC), FEV1/FVC.1 In some patients, other tests may be needed to determine the phenotype and physiologic characteristics.16

COPD is often not diagnosed until clinical symptoms are present and the disease is moderately advanced. An early diagnosis of COPD, when the disease is most treatable, is desirable. Current therapies are more effective when used in the early stages of COPD,16 and further disease progression often may be prevented by the implementation of a successful smoking cessation program.

A clinical diagnosis of COPD should be considered in any patient who presents with dyspnea, a chronic cough, or chronic sputum production and/or has a history of exposure to known risk factors for the disease.16 The diagnosis is usually made when the patient is experiencing dyspnea with mild exertion that interferes with quality of life (Table 1).

Patients may not always communicate directly about their symptoms; this may play a role in the significant underdiagnosis of COPD.17 Some patients consider their symptoms a normal part of the aging process or of long-term tobacco use, making them less likely to report them to their practitioner.

**TABLE 1. Key Indicators for Considering a Diagnosis of COPD**

<table>
<thead>
<tr>
<th>Dyspnea that is:</th>
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<tbody>
<tr>
<td>• Progressive</td>
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<tr>
<td>• Usually worse with exercise</td>
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<tr>
<td>• Persistent (present every day)</td>
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<tr>
<td>• Described by the patient as an increased effort to breathe, heaviness, air hunger, or gasping</td>
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<tr>
<th>Chronic cough:</th>
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<tbody>
<tr>
<td>• May be intermittent and may be unproductive</td>
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<table>
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<tr>
<th>Chronic sputum production:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Any pattern</td>
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<table>
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<tr>
<th>History of exposure to risk factors, in particular:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Tobacco smoke</td>
</tr>
<tr>
<td>• Occupational dusts and chemicals</td>
</tr>
<tr>
<td>• Smoke from home cooking and heating</td>
</tr>
</tbody>
</table>

Adapted from GOLD 2007. Global Strategy for Diagnosis, Management, and Prevention of COPD.

For patients suspected of having COPD, clinicians should ask if he or she:
- Is a current/former smoker
- Has current exposure to secondhand smoke
- Has breathing difficulties during mild exercise or at night
- Is restricting physical activity
- Is complaining about exercise intolerance
- Has a productive morning cough and/or a cough lasting more than 2 weeks
- Has experienced a decline in activities of daily living
  - Are groceries now being delivered?
  - Is the patient no longer walking to work? Using a golf cart instead of walking?
  - Is there a decline in job performance?
- Clinical assessment is based on medical history and physical examination, use of specific tests as needed, and spirometry where appropriate. The diagnosis should be confirmed by spirometry. A postbronchodilator FEV1/FVC <0.70 generally will confirm the presence of airflow limitation that is not fully reversible.1 Comorbidities are also common in COPD patients and should be identified.

**Medical History**

The medical history should address current symptoms, past history of individual/family health conditions, and any history of risk exposure.18

The most common symptoms are cough, dyspnea, and sputum production. Cough, usually the first symptom to develop, may be intermittent at first and occur early in the morning. Gradually, however, it becomes more progressive and is present throughout the day.1 The cough is generally productive and is often considered an expected outcome of long-term tobacco use in smokers.

Similarly, sputum production is initially experienced in the morning but eventually is present throughout the day. The presence of sputum, which is often mucoid, for 3 or more months in 2 consecutive years is the standard epidemiologic definition of chronic bronchitis.19 Alterations in volume or color may be associated with the onset of an exacerbation. The presence of purulent sputum usually reflects an elevation of inflammatory mediators.

Dyspnea is the hallmark symptom of COPD and is often the reason patients seek medical care. It tends to be progressive and becomes more persistent over time. At first it may occur only on more intense physical exertion, such as when exercising or climbing stairs, but as COPD progresses, it may be present even at rest (Table 2).20

Wheezing and chest tightness may also be present, although they are nonspecific symptoms more often seen in patients with severe disease.2 Weight loss, malnutrition, and anorexia are frequently seen in patients with more advanced disease but
may also be associated with other pulmonary diseases, such as tuberculosis or bronchial tumors. Patients may also have rib fractures secondary to coughing bouts, or ankle swelling, which may indicate cor pulmonale. Patients with advanced disease should be assessed for psychiatric disorders such as depression and anxiety.

The Global Initiative for Chronic Lung Disease (GOLD) classifies COPD into 4 stages. For a given disease severity staged by spirometry, expected symptoms might include:

- **Stage 1, or Mild COPD:** The patient has a chronic cough and chronic sputum production. These symptoms can be present for many years before airflow limitations develop.
- **Stage 2, or Moderate COPD:** Airflow gradually becomes more limited, and patients often experience dyspnea. Typically, at this stage medical attention is sought and COPD is diagnosed; however, some patients may still be largely asymptomatic. A respiratory tract infection that significantly worsens lung function may convince the patient to seek medical care.
- **Stage 3, or Severe COPD:** Coughing and sputum production continue, with worsening of dyspnea. Additional pulmonary and extrapulmonary symptoms may develop, such as weight loss and respiratory failure.
- **Stage 4, or Very Severe COPD:** The patient has severe airflow limitation. Respiratory failure may also lead to effects on the heart such as cor pulmonale. Quality of life is very appreciably impaired, and exacerbations may be life threatening. The severity of COPD has also been classified according to spirometry (Table 3).

### Past Medical History and Review of Systems

Any of the following should be noted for a new patient known or believed to have COPD:

- Any history of childhood respiratory infections, including asthma, allergy, sinusitis, or nasal polyps, and other respiratory diseases such as tuberculosis
- Any family history of COPD or other chronic respiratory disease
- Any history of exposure to risk factors, such as occupational or environmental noxious agents and especially tobacco
- Any history of exacerbations or hospitalizations for respiratory disorders
- The presence of any comorbidities, such as cardiovascular disease, osteoporosis, or neurologic conditions
- The pattern of current symptom development
- Any history of unexplained weight loss
- The presence of nonspecific symptoms, such as wheezing and chest tightness or pain
- Prescription medications currently being taken, since some pharmaceutical agents, including beta-blockers, are usually contraindicated in COPD

Provider reminder systems can help standardize practice in the diagnosis and assessment of COPD. Consider creating checklists based on Table 1 or the bulleted list above. Checklists help ensure that patients who have or are suspected of having COPD receive consistent care. (See information on page 14 for additional ideas on how to implement reminder systems.)

### Physical Examination

A physical examination should be performed, although findings are frequently normal during the early stages of the disease. Although a useful and necessary component of patient care, physical examination is rarely diagnostic for COPD and is unreliable for detecting or excluding COPD as a diagnosis. The physical signs of airway impairment are often not

<table>
<thead>
<tr>
<th>TABLE 2. Modified Medical Research Council Questionnaire for Assessing the Severity of Breathlessness</th>
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<tbody>
<tr>
<td>• I get breathless only with strenuous exercise.</td>
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<tr>
<td>• I get short of breath when hurrying on a level surface or walking up a slight hill.</td>
</tr>
<tr>
<td>• I walk slower than people of the same age on a level surface because of breathlessness, or I have to stop for breath when walking at my own pace on a level surface.</td>
</tr>
<tr>
<td>• I stop for breath after walking about 100 yards or after a few minutes on a level surface.</td>
</tr>
<tr>
<td>• I am too breathless to leave the house, or I am breathless when dressing or undressing.</td>
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<th>TABLE 3. Spirometric General Classification of COPD Severity</th>
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<tbody>
<tr>
<td>Severity</td>
</tr>
<tr>
<td>Mild COPD</td>
</tr>
<tr>
<td>Moderate COPD</td>
</tr>
<tr>
<td>Severe COPD</td>
</tr>
<tr>
<td>Very severe COPD</td>
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FEV\textsubscript{1} = forced expiratory volume in 1 second; FVC = forced vital capacity (FVC). ATS/ERS. Standards for the Diagnosis and Management of Patients with COPD. 2005. http://www.thoracic.org/gps/copd/(Updated 2005 September 8.)
detectable until a substantial amount of lung damage has occurred. But any signs and symptoms identified during a physical examination, combined with a medical history, specific patient risk factors, and testing, may yield a differential diagnosis of COPD.

The Value of Spirometry

Spirometry is a relatively simple tool that can be used for patients with lung disorders. Advancements in technology have made spirometry testing more reliable and relatively easy to integrate into a routine office visit. Considered a key component in confirming a diagnosis of COPD, spirometry remains the best standardized, most reproducible, and most objective measurement of airflow limitation. All clinicians who care for COPD patients should have access to high-quality spirometry. A survey of participants of the BUSM MENTOR QI activity showed that 23% of COPD patients did not receive a spirometry evaluation to confirm diagnosis. For 45% of the participants, spirometry was not available through their office or in their community, and 47% had not been trained to use the spirometer. (See the resources on page 9 to help overcome some of the barriers to appropriate use.)

Spirometry measures the rate at which the lung changes volume during forced breathing exercises. It begins with the patient taking a full inhalation, followed by a forced expiration that rapidly empties the lungs, measured by the FVC. The FEV₁ is the amount of the FVC that can be expelled in 1 second, and the ratio of these 2 measurements (FEV₁/FVC) is then calculated. Spirometry measurements are interpreted as percentages by comparing them with established reference values that are based on age, height, weight, gender, and race/ethnicity.

The normal values are approximately:

- FEV₁: 80% to 120%
- FVC: 80% to 120%

Absolute FEV₁/FVC ratio is within 5% of the predicted ratio.

Patients with COPD will typically show a decline in both FEV₁ and FVC. The degree of spirometric abnormality is usually indicative of disease severity (Figure 2).

Spirometry testing requires understanding, coordination, and cooperation between the patient and the technician/provider. The accuracy of the results depends on a variety of factors, including equipment performance, quality control, patient-examiner maneuvers, repeatability, and clinical assessment. Proper training of the technician/provider is essential, and the manufacturer may frequently provide inservices. Spirometry testing can also be performed after the patient has received an adequate dose of a short-acting inhaled bronchodilator in order to minimize variability. Patient preparation for spirometry involves the following:

- Enlist patient cooperation and effort to ensure the success of the procedure
- Do the spirometry test at least 3 times to ensure reproducibility
- Have the patient sit or stand (according to patient preference, although sitting is recommended for safety reasons)
- Explain the spirometry test
- Measure patient height/weight without shoes
- Ask about smoking, recent illness, use of medications, etc.
- Use noseclip (optional although recommended)
- Instruct the patient and demonstrate the test
- Position the tongue and mouth correctly
- Instruct the patient to:
  - Initially deeply inhale and exhale several times
  - Then inhale maximally
  - Then exhale deeply as rapidly and completely as possible for at least 6 seconds

Performing the spirometry test involves the following:

- Check the spirometer calibration
- Avoid having the patient lean forward
- Have the patient take several deep, cleansing breaths and exhale
- Have the patient inhale completely and rapidly with a pause of <1 second at total lung capacity, then exhale deeply as rapidly and completely as possible for at least 6 seconds
- Coach the patient through the procedure
- Do 3 tests with acceptability and reproducibility; usually no more than 8 are required
- Do not allow the patient to cough or talk
- Use the largest value
- Check the test’s repeatability and perform more maneuvers as necessary

Spirometry as a Screening Tool

The role of screening spirometry in the general population is controversial. There are no data to support the hypothesis that screening spirometry is effective in directing management decisions or in improving COPD outcomes in patients prior to symptom onset.
Dales et al showed that the results of screening spirometry in the primary care setting influenced the diagnosis of airflow obstruction and subsequent management, especially in patients with moderate to severe obstruction. In 1,034 patients, adding spirometry resulted in a new physician diagnosis of unsuspected airflow obstruction in 9% of patients, and a previous diagnosis of airflow obstruction was withdrawn after spirometry in 11% of patients. After evaluating the spirometry results, physicians reported that they would alter treatment in 15% of patients.

Opinions on routine screening are mixed among professional and public health organizations, although the more recent guidelines tend to be opposed to routine screening. In 2007, the American College of Physicians guidelines stated that “evidence is insufficient to support widespread use of spirometry for testing adults with no respiratory symptoms, including those with current and past exposure to COPD risk factors.” The 2008 update from the US Preventive Services Task Force recommends against screening adults for COPD using spirometry, concluding that “there is at least moderate certainty that screening for COPD using spirometry has no net benefit.” Other tests may be considered to make a differential diagnosis, to further assess lung function, and/or to establish the presence of comorbidities.

Spirometry, when performed properly, is valuable in confirming a diagnosis of COPD. Although not recommended as a screening tool for COPD, it may be a helpful measurement in some patient situations.

**Bronchodilator Reversibility Testing**
Generally performed once at the time of diagnosis, bronchodilator reversibility testing may be useful for excluding asthma, but only if the reversible component is relatively large (20%-30% or more over baseline). Evidence indicates that in

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Suggestive Features</th>
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<tr>
<td><strong>COPD</strong></td>
<td>Midlife onset&lt;br&gt;Slowly progressing symptoms&lt;br&gt;Mostly in long-term smokers&lt;br&gt;Dyspnea during exertion&lt;br&gt;Airflow limitation largely nonreversible</td>
</tr>
<tr>
<td><strong>Asthma</strong></td>
<td>Early onset, usually in childhood&lt;br&gt;Varying symptoms&lt;br&gt;Symptoms during the night/early morning&lt;br&gt;Presence of allergy, rhinitis, and/or eczema&lt;br&gt;Airflow limitation largely reversible</td>
</tr>
<tr>
<td><strong>Congestive heart failure</strong></td>
<td>More common in older patients but may occur at any age&lt;br&gt;May have acute or suble onset&lt;br&gt;Fine basilar crackles on auscultation&lt;br&gt;Dilated heart on chest radiography&lt;br&gt;Pulmonary edema&lt;br&gt;Volume restriction, not airflow limitation, on pulmonary function tests</td>
</tr>
<tr>
<td><strong>Bronchiectasis</strong></td>
<td>More common in older patients but may occur at any age&lt;br&gt;Large volume of purulent sputum&lt;br&gt;Commonly caused by bacterial infection&lt;br&gt;Coarse crackles on auscultation&lt;br&gt;Digital clubbing&lt;br&gt;Bronchial dilation and bronchial wall thickening on chest radiography/computed tomography (CT)</td>
</tr>
<tr>
<td><strong>Tuberculosis</strong></td>
<td>May occur at any age&lt;br&gt;Lung infiltrates on chest radiography&lt;br&gt;Confirmation by microbiologic evaluation</td>
</tr>
<tr>
<td><strong>Obliterative bronchiolitis</strong></td>
<td>Younger onset&lt;br&gt;Occurrence in nonsmokers&lt;br&gt;History of relevant exposure, such as noxious fumes, or underlying disease process, such as rheumatoid arthritis, other autoimmune disease, organ or marrow transplant, or viral bronchiolitis&lt;br&gt;Compatible findings on chest CT</td>
</tr>
<tr>
<td><strong>Diffuse panbronchiolitis</strong></td>
<td>May occur at any age&lt;br&gt;Occurrence primarily in male nonsmokers&lt;br&gt;Chronic sinusitis usually present&lt;br&gt;Diffuse small centrilobular nodular opacities and hyperinflation on chest radiography and high-resolution CT</td>
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**TABLE 4. Differential Diagnosis of COPD**
most COPD patients, a clinical response to bronchodilators or inhaled corticosteroids cannot be reliably predicted by response to a reversibility test. All patients with COPD respond to some extent to a bronchodilator, and bronchodilators should be administered irrespective of the reversibility test.

Chest Radiography
A chest x-ray, although rarely diagnostic for COPD, is useful primarily to rule out other diagnoses and should be obtained in all patients. It can help exclude conditions such as pneumonia, cancer, or congestive heart failure, which can cause symptoms similar to those of COPD. Common signs of emphysema that may be seen on radiography are flattening of the diaphragm, irregular lung radiolucency, and reduction or absence of vascularity, although these are not specific to the disease.

Alpha-1-antitrypsin Levels
Alpha-1-antitrypsin (AAT) should be measured in young patients, especially those of European descent, who present with symptoms of COPD and/or who have a strong family history of the disease. A serum value of alpha-1-antitrypsin less than 15% to 20% of the normal limits is highly suggestive of homozygous alpha-1-antitrypsin deficiency.

Making the Differential Diagnosis
In making the differential diagnosis, COPD and asthma must be differentiated. Although both disorders are characterized by airflow obstruction and inflammation, there are pronounced and subtle differences between them. A chronic asthmatic condition often cannot be distinguished from COPD using currently available imaging and physiologic testing techniques. In these cases, management should reflect the assumption that the 2 diseases coexist. Other disorders may also mimic some of the symptoms and pathology that are characteristic of COPD (Table 4).

MANAGEMENT OF COPD
The goal of managing COPD is to improve pulmonary function, provide symptom relief, decrease the incidence of exacerbations and hospitalizations, improve quality of life, slow the decline in lung function, increase exercise tolerance, prevent and treat complications, and increase life expectancy. Treatment is multifaceted, incorporates both nonpharmacologic and pharmacologic modalities, and evolves based on the patient’s general medical condition and the severity of the disease (Figure 3). The clinical course of COPD is progressive, characterized by chronic symptoms along with intermittent acute exacerbations. Therapeutic options for COPD reflect both the chronic and the acute nature of the disease.

Management of Stable Disease
The management of stable COPD primarily addresses symptoms and strives to improve quality of life. The basic components of managing stable disease are patient education about COPD, including its risk factors, particularly smoking; nonpharmacologic treatment; and pharmacologic therapies. Available pharmacologic agents are used to prevent and control symptoms as well as to reduce the frequency and severity of exacerbations.

The Role of Patient Education
Education plays an important role in helping the patient improve coping skills and overall health status as well as quitting smoking. Its role has not been well studied in the setting of COPD.

### TABLE 5. Topics for Patient Education

<table>
<thead>
<tr>
<th>All Patients:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Reducing risk factors</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stages 1-3: Mild (1), Moderate (2), and Severe (3) COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above topics, plus:</td>
</tr>
<tr>
<td>• The nature of COPD</td>
</tr>
<tr>
<td>• How to use inhalers and other treatments</td>
</tr>
<tr>
<td>• Recognition and treatment of exacerbations</td>
</tr>
<tr>
<td>• Strategies for minimizing dyspnea</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stage 4: Very Severe COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above topics, plus:</td>
</tr>
<tr>
<td>• Complications</td>
</tr>
<tr>
<td>• Oxygen treatment</td>
</tr>
<tr>
<td>• Use of advance directives and end-of-life decisions</td>
</tr>
</tbody>
</table>

However, a survey of participants in the BUSM MENTOR QF activity showed that for 30%, their office did not offer/encourage patient education for COPD patients.

Education can help patients understand the nature of the disease, risk factors for progression and exacerbations, and the role of health care workers in achieving optimal management and health outcomes. The educational strategies and tools used should be individualized to each patient, reflecting factors such as the patient’s stage of disease and environment (Table 5).

One study showed that hospitalizations were reduced in patients who followed a weekly comprehensive COPD patient education program over a 2-month period with monthly telephone follow-up.3 Hospital admissions for exacerbations were reduced by 39.8% compared with the usual-care group, and admissions for other health problems were reduced by 57.1%. There were also reductions in emergency room visits (41.0%) and unscheduled physician visits (58.9%).

**Smoking Cessation**

Smoking cessation is the single most effective and most important intervention available in the prevention and the management of COPD and is an effective measure to implement during any stage of the disease.1 Smoking cessation programs should be offered to and encouraged for all patients who are still smokers.

Smoking cessation interventions can be classified as behavioral, pharmacologic, and alternative methods (including hypnosis, acupuncture, aversive therapy, exercise, and opioid agonists).23 The US Department of Health and Human Services recommends a 5-step strategy that may be useful to health care providers in helping their patients stop smoking (Table 6).

Individual, group, and telephone counseling have been found to be helpful in stopping smoking.25 An analysis of six studies in the Cochrane Collaboration review showed that the absolute smoking cessation rate with group counseling was 10%, while a US Department of Health and Human Services analysis of 58 studies showed a lower rate, 3.1%.24

Pharmacotherapy is another option, especially for patients who have been unable to quit or sustain abstinence with behavioral interventions alone. Currently, 7 agents have been shown to increase long-term smoking abstinence rates: bupropion SR, nicotine gum, nicotine inhaler, nicotine lozenge, nicotine nasal spray, nicotine patch, and varenicline.25

In a head-to-head comparison trial, varenicline, a nicotinic receptor partial agonist, was significantly more effective than bupropion or placebo for smoking cessation at the end of 12 weeks of drug treatment and at 24 weeks. For weeks 9 through 12, the 4-week continuous abstinence rates were 44.0% for varenicline vs 17.7% for placebo (P < .001) and vs 29.5% for bupropion SR (P < .001).26,27 Two identically designed phase 3 randomized trials conducted at different centers had similar results. However, many of those who had been successful at quitting relapsed. By week 52, abstinence rates had declined to 21.9% for varenicline, 16.1% for bupropion, and 8.4% for placebo.26,27 Relapse rates were similar in the second study.27

Although counseling and medication are effective therapies
when used separately, their efficacy is greatly enhanced when they are combined. Combinations of certain medications also may be more effective.

**SMOKING CESSATION RESOURCES**

- **Centers for Disease Control**
  Patient quiz about smoking cessation: [http://www.cigarettefree.org/quit-smoking/nicotine_addiction.asp](http://www.cigarettefree.org/quit-smoking/nicotine_addiction.asp)
- **Office of the US Surgeon General**
  5 steps to tobacco use intervention: [http://www.surgeongeneral.gov/tobacco/5steps.htm](http://www.surgeongeneral.gov/tobacco/5steps.htm)
- **National Library of Medicine**
- **Quitnet**

**Pulmonary Rehabilitation**

Pulmonary rehabilitation encompasses a wide range of modalities, including exercise training, education, psychosocial/behavioral intervention, nutritional therapy, outcome assessment, and promotion of long-term adherence to the rehabilitation recommendations. However, a survey of participants in the BUSM MENTOR QRMTM activity showed that exercise training was not recommended for 66% of patients with dyspnea.

Clinical guidelines from the American College of Physicians (ACP) recommend the use of pulmonary rehabilitation for symptomatic individuals with COPD who have an FEV₁ <50% predicted. The principal goals of pulmonary rehabilitation are to reduce symptoms, improve quality of life, and increase physical and emotional participation in everyday activities. However, in patients with an FEV₁ >50% predicted, the evidence for its efficacy is not clear.

**Pharmacologic Management of Stable COPD**

ACP clinical guidelines recommend that treatment for stable COPD be reserved for patients who have respiratory symptoms and FEV₁ <60% predicted as documented by spirometry. Symptomatic patients with FEV₁ <60% predicted should receive one of the following maintenance therapies: long-acting inhaled beta-agonists, long-acting inhaled anticholinergics, or inhaled corticosteroids.

Combination inhaled therapies may also be considered for symptomatic patients with an FEV₁ <60% predicted, and oxygen therapy should be prescribed for individuals with resting hypoxemia (PaO₂ ≤55 mm Hg).

**Bronchodilators.** Bronchodilators form the foundation of symptomatic treatment of the reversible component of airway obstruction in COPD and are typically prescribed for the relief of bronchospasm. According to the GOLD guidelines, bronchodilators are administered on as-needed basis for relief of persistent or worsening symptoms or on a regular basis to prevent or reduce symptoms. However, a survey of participants of the BUSM MENTOR QRMTM activity showed that 8% of COPD patients were on no inhaled bronchodilator.

The principal bronchodilator treatments are beta-agonists, anticholinergics, and methylxanthines, which are used as monotherapy or in combination. Short-acting beta-agonists have historically been one of the classes of bronchodilators most widely used for brief symptom relief. While they have a rapid onset of bronchodilating effect, their duration of action is only up to 6 hours, necessitating multiple daily dosing.

Long-acting bronchodilators, introduced over the past few years, have a duration of 12 hours or more and do not lose their effectiveness overnight or with regular use, thus reducing the need for multiple dosing. According to the GOLD guidelines, long-acting inhaled bronchodilators are more convenient for the patient and appear to have greater efficacy than short-acting bronchodilators.

Beta-agonists (Table 7) are available in metered-dose inhalers, nebulized formulations, and oral preparations; the inhaled formulations are preferred. Adverse events tend to be dose-related and occur more frequently with the oral formulations than with the inhaled forms.

Anticholinergics are available only via the inhaled route and, in COPD patients, cause bronchodilation by blocking muscarinic receptor subtype M₁. This action prevents acetylcholine from activating the receptor, thereby preventing or reversing bronchoconstriction. Some short-acting drugs also block M₂ receptors and can modify transmission at the preganglionic junction, but this action is less important in COPD. Tiotropium, a long-acting anticholinergic agent, has pharmacokinetic selectivity for both the M₁ and M₂ receptors.

Tiotropium can provide sustained bronchodilation for up to 36 hours, and short-acting inhaled anticholinergics have a longer duration than the short-acting beta-agonists (Table 8).

The onset of bronchodilation after administration of an anticholinergic is within 30 minutes, with a modest dose-response relationship as evaluated by FEV₁.
Systemic side effects are limited with anticholinergic drugs, because of their poor absorption. Common adverse events include dry mouth, which is most marked with tiotropium, and a metallic taste after inhalation, most commonly seen with ipratropium. In rare cases, closed-angle glaucoma has been associated with the use of wet nebulizer solutions delivered by face mask.

Inhaled beta-agonists and anticholinergics are considered first-line therapies and central to COPD management. Mahler et al compared inhaled salmeterol xinafoate, a long-acting inhaled beta-adrenergic agonist (42 mg twice daily), inhaled ipratropium bromide (36 mg 4 times a day), and inhaled placebo (2 puffs 4 times daily) over a 12-week period. Salmeterol xinafoate was superior to both placebo and ipratropium in improving lung function when administered at the recommended doses. Both salmeterol and ipratropium reduced dyspnea related to activities of daily living compared with placebo, but salmeterol also significantly lowered the rate of COPD exacerbations, which led to significantly greater improvements in health-related quality of life.

Preventing exacerbations is a key element in COPD management. In a large trial of 1,829 patients with moderate to severe COPD (mean baseline FEV1, 36% predicted), Niewoehner et al found that tiotropium significantly reduced the percentage of patients who experienced one or more exacerbations during a 6-month follow-up period compared with placebo.

A Cochrane review of 9 randomized controlled trials (6584 patients) showed that compared to placebo and ipratropium, tiotropium was more effective in reducing COPD exacerbations and related hospitalizations. The increases in FEV1 and FVC from baseline were significantly larger with tiotropium than with placebo, ipratropium, or long-acting beta-agonists over 6 to 12 months, and its use also improved health-related quality of life and symptom scores among patients with moderate and severe disease.

Methylxanthines, such as aminophylline, are the third class of bronchodilators. These agents are nonspecific phosphodiesterase inhibitors that increase levels of intracellular cAMP within airway smooth muscle. They can be administered orally or intravenously, and their bronchodilator effects are most pronounced at high doses. However, the need for high doses also increases the risk of toxicity.

The selection of a beta-agonist, an anticholinergic agent, or a methylxanthine is based on numerous factors, including the availability of medications, the cost, the patient’s improvement, and treatment-related adverse events. Trial data have shown that the combination of an anticholinergic and a beta-agonist is more effective than either used alone and may decrease the risk of side effects. In addition, combining a beta-agonist and an anticholinergic and/or theophylline may further improve lung function.

**TABLE 7. Commonly Used Beta-^-Agnists in the Treatment of COPD**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Duration of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenoterol</td>
<td>4-6 hours</td>
</tr>
<tr>
<td>Levalbuterol</td>
<td>4-6 hours</td>
</tr>
<tr>
<td>Salbutamol (albuterol)</td>
<td>4-6 hours</td>
</tr>
<tr>
<td>Terbutaline</td>
<td>4-6 hours</td>
</tr>
<tr>
<td><strong>Long-Acting</strong></td>
<td></td>
</tr>
<tr>
<td>Formoterol</td>
<td>12+ hours</td>
</tr>
<tr>
<td>Salmeterol</td>
<td>12+ hours</td>
</tr>
</tbody>
</table>


**TABLE 8. Commonly Used Anticholinergics and Combination Formulations in the Treatment of COPD**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Duration of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Short-Acting</strong></td>
<td></td>
</tr>
<tr>
<td>Ipratropium bromide</td>
<td>6-8 hours</td>
</tr>
<tr>
<td>Oxitropium bromide*</td>
<td>7-9 hours</td>
</tr>
<tr>
<td><strong>Long-Acting</strong></td>
<td></td>
</tr>
<tr>
<td>Tiotropium</td>
<td>24+ hours</td>
</tr>
<tr>
<td>Combination short-acting beta-agonists plus anticholinergic in 1 inhaler</td>
<td></td>
</tr>
<tr>
<td>Fenoterol/Ipratropium*</td>
<td>6-8 hours</td>
</tr>
<tr>
<td>Albuterol/Ipratropium</td>
<td>6-8 hours</td>
</tr>
</tbody>
</table>

*Not marketed in the United States.


**GLUCOCORTICOSTEROIDS.** Glucocorticosteroids act at multiple points within the inflammatory cascade, but improvements in symptoms and lung function gained with both oral and inhaled agents are less dramatic in patients with COPD than in those with asthma. Generally, glucocorticosteroids are reserved for patients with poor lung function. Because of their multiple side effects, oral steroids are usually not indicated for patients with stable disease, although they can be useful during exacerbations.

Inhaled glucocorticosteroids should be considered in patients who have severe COPD with frequent exacerbations, as they may reduce the rate of acute episodes. This includes symptomatic COPD patients with stage 3 or 4 disease, an FEV1 <50% predicted, and repeated exacerbations. Withdrawing from glucocorticosteroids may cause exacerbations in some patients, and their use increases the likelihood of pneumonia. Inhaled corticosteroid use has not been shown to reduce overall mortality.
Earlier trial data did not clearly demonstrate clinical benefit from combining therapy with a long-acting bronchodilator and an inhaled steroid compared with monotherapy. However, in the recently completed and very large TORCH trial, combined therapy with salmeterol and fluticasone was more effective in reducing exacerbations and improving health status than either drug given alone (Table 9). A post-hoc analysis of the TORCH trial indicated that pharmacotherapy with salmeterol plus fluticasone propionate, or either component alone, may reduce by a modest amount the rate of decline of FEV1 in patients with moderate-to-severe COPD, thus slowing disease progression.\(^2\)

**Immunization**

Clinicians treating patients with COPD are urged to administer immunizations to reduce the risk of serious respiratory illness. Vaccination of COPD patients against influenza can reduce the risk of mortality by up to 50\%.\(^3\)\(^,\)\(^4\) Pneumococcal vaccinations have been shown to reduce the incidence of community-acquired pneumonia and should be given to patients aged \(\geq 65\) years or those with severely impaired lung function (FEV\(_1\) \(<40\%\) predicted).\(^3\)\(^,\)\(^4\)\(^,\)\(^6\)\(^,\)\(^8\) However, a survey of participants of the BUSM MENTOR QI\(^6\) activity showed that in the last year, 39\% and 31\% of COPD patients did not receive an influenza vaccine and pneumococcal vaccine, respectively. Use of a reminder system may be helpful to clinicians in managing patients with COPD. A simple reminder system could consist of memo slips inserted into the front of the medical charts of all patients with COPD, briefly outlining the areas of treatment that should be addressed during all COPD visits. Using patient reminder and recall systems (postcards and letters, telephone or autodialer calls) also can help improve immunization rates.

**OTHER THERAPIES**

**Oxygen Therapy**

Long-term oxygen therapy improves survival, exercise, sleep, and cognitive performance and should be prescribed for patients with COPD and resting hypoxemia (PaO\(_2\) \(\geq 55\) mm Hg).\(^2\) Use of supplemental oxygen for 15 or more hours each day can help improve survival in patients who have severe airflow obstruction. The primary goal of oxygen therapy is to increase the baseline PaO\(_2\) to at least 8.0 kPa (60 mm Hg) at sea level and while at rest and/or to produce an SaO\(_2\) of at least 90\%.\(^1\)

Although oxygen therapy is generally safe and effective, potential physical hazards may occur during the storage and handling of oxygen—primarily fire and explosions. Patients lighting cigarettes while using oxygen therapy cause the majority of fires. The patient, family members, and other caregivers must be warned not to smoke near oxygen.

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**TABLE 9. Commonly Used Glucocorticosteroids and Combination Formulations in the Treatment of COPD**

<table>
<thead>
<tr>
<th>Inhaled</th>
<th>Inhaled Combinations</th>
<th>Systemic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beclomethasone</td>
<td>Formoterol/</td>
<td>Prednisone</td>
</tr>
<tr>
<td></td>
<td>Budesonide</td>
<td></td>
</tr>
<tr>
<td>Budesonide</td>
<td>Salmeterol/</td>
<td>Methylprednisolone</td>
</tr>
<tr>
<td></td>
<td>Fluticasone</td>
<td></td>
</tr>
<tr>
<td>Fluticasone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triamcinolone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


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**IMMUNIZATION RESOURCES**

- **Centers for Disease Control**
  Strategies for increasing vaccination rates:
  http://www.cdc.gov/vaccines/recs/rate-strategies/flustrat.htm

- **Immunization Action Coalition**
  Information on influenza vaccinations:
  http://www.immunize.org/vis/vis_fluinactive.asp

**OXYGEN THERAPY RESOURCES**

- **The National Lung Health Education Program**
  Considerations for prescribing long-term oxygen therapy:
  http://www.nhlbi.nih.gov/guidelines/cpap/cpap.htm

  Step-by-step guide to prescribing home oxygen:
  http://www.nhlbi.nih.gov/guidelines/cpap/cpap.htm
Surgery
Surgical interventions in COPD include lung transplantation, lung volume reduction procedures, and bullectomy, an older surgical procedure for bullous emphysema.1

With advances in immune suppression and improved understanding of the timing of interventions and the selection of appropriate recipients, lung transplantation has become a realistic option. Although transplantation has been shown to improve both quality of life and lung function, the procedure is limited by a lack of suitable donors and by its high cost. Aside from mortality related to the surgery, complications include acute rejection, bronchiolitis obliterans, bacterial infections, and opportunistic fungal infections with organisms such as Candida and Aspergillus. Criteria for referral for lung transplantation include FEV1 <35% predicted, PaO2 <7.3–8.0 kPa (55–60 mm Hg), PaCO2 >6.7 kPa (50 mm Hg), and secondary pulmonary hypertension.

In lung volume reduction surgery, the goal is to reduce hyperinflation of one or both lungs by surgical and/or laser resection. This method is primarily for patients with upper-lobe emphysema and low exercise capacity who are more likely to have greater survival benefit and improved lung function with lung volume reduction surgery than with medical treatment. Another procedure, bullectomy, involves the removal of one or more bullae, defined as airspaces greater than 1 cm in diameter that form as a result of pulmonary tissue destruction. The most common indications for bullectomy are severe dyspnea in the setting of a large bulla occupying at least 30% of the hemithorax, pain, and spontaneous pneumothorax.

MANAGEMENT OF EXACERBATIONS
COPD exacerbations are a common cause of morbidity and mortality and affect the quality of life and prognosis. Although exacerbations are more common in patients with severe disease, they may occur at every stage of COPD.

Mortality rates for hospitalized patients with COPD exacerbations are approximately 10%, and the long-term outcome is poor.1 For patients needing mechanical ventilation, mortality reaches 40% at 1 year, while all-cause mortality may reach 49% at 3 years following hospitalization. Preventing exacerbations and reducing their frequency can have a significant effect on patient health and on the economic burden of COPD.

The most common causes of exacerbations are infections of the tracheobronchial tree and air pollution. A significant number of patients, however, may also have bacterial colonization during stable periods.

Inhaled bronchodilators are effective treatments for acute exacerbations,20 and systemic glucocorticosteroids have been shown to reduce their severity and shorten recovery times. Patients with signs of infection, such as fever and/or an increased volume of sputum and a change in its color, may benefit from antibiotic therapy. A Cochrane Review of 11 trials with 917 patients showed that use of antibiotics in COPD exacerbations with increased cough and sputum volume lowered the risk of short-term mortality by 77%.20

The American Thoracic Society and the European Respiratory Society in their current guidelines devised an operational classification of severity of exacerbations to help rank their clinical relevance, outcome, and treatment:20

- Level 1 episodes: treated at home
- Level 2 episodes: require hospitalization
- Level 3 episodes: lead to respiratory failure

Treatment for Level 1 episodes generally consists of the following:20

- Patient education—check inhalation technique and consider use of spacer devices
- Bronchodilators—short-acting beta-agonist and/or ipratropium metered-dose inhaler (MDI) with spacer or hand-held nebulizer as needed
  - A long-acting bronchodilator, such as tiotropium, salmeterol, or formoterol, may have to be added to the regimen
- Corticosteroids—systemic oral agents
  - Prednisone 30–40 mg daily may be given for 10 days (dose may vary)
  - Inhaled corticosteroids may be considered
- Antibiotics, in patients with altered sputum characteristics
  - The choice of agent should be based on local bacterial resistance patterns
  - Commonly used antibiotics include amoxicillin/ampicillin, cephalosporins, doxycycline, and macrolides
  - If patient has failed prior antibiotic therapy, consider amoxicillin/clavulanate, respiratory fluoroquinolones (gatifloxacin, levofloxacin, moxifloxacin), cefdinir, cefprozil, or cefuroxime

Treatment for Level 2 hospitalization episodes generally consists of the following:20

- Bronchodilators—short-acting beta-agonist (albuterol [salbutamol]) and/or ipratropium MDI with spacer or hand-held nebulizer as needed
- Supplemental oxygen (if saturation <90%)
- Corticosteroids
  - If tolerated, oral prednisone 30–40 mg daily may be given for 10 days. If patient is unable to tolerate an oral
TABLE 10. Referral to Pulmonologist

<table>
<thead>
<tr>
<th>Circumstances Possibly Requiring Specialist Review</th>
<th>Role of Respiratory Specialist</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate or severe COPD</td>
<td>Confirm diagnosis and optimize therapy. Cease inappropriate or ineffective therapies. Assess side effects. Determine need for nebulized therapy. Assess</td>
</tr>
<tr>
<td>Uncertain diagnosis (&lt;10 pack-year smoking history, &lt;40 years of age, or rapid decline in FEV₁)</td>
<td>Confirm diagnosis and exclude other diagnoses (eg, asthma, bronchiolitis obliterans, pulmonary embolism, cancer, heart failure, pneumothorax, anemia). Determine other etiologic factors. Determine if the patient is predisposed (eg, alpha 1-antitrypsin deficiency).</td>
</tr>
<tr>
<td>Recurrent infections, exacerbations</td>
<td>Exclude other conditions (eg, bronchiectasis, cystic fibrosis, immunologic abnormality, aspiration).</td>
</tr>
<tr>
<td>Symptoms disproportionate to lung function impairment</td>
<td>Exclude complications of COPD or comorbidities (eg, pulmonary hypertension, cardiac disease). Consider sleep study.</td>
</tr>
<tr>
<td>Cor pulmonale</td>
<td>Confirm diagnosis and optimize treatment. Assess need for oxygen or other ventilatory support.</td>
</tr>
<tr>
<td>Suspected chronic hypoxemia</td>
<td>Confirm chronic or nocturnal hypoxemia. Assess for ambulatory oxygen therapy.</td>
</tr>
<tr>
<td>Bullous lung disease or severe emphysema</td>
<td>Determine suitability for bullectomy or lung volume reduction surgery.</td>
</tr>
<tr>
<td>Severe disability or respiratory failure</td>
<td>Determine suitability for lung volume reduction surgery or lung transplantation or home ventilation.</td>
</tr>
</tbody>
</table>

Agent, then intravenous prednisone at an equivalent dose may be given for 14 days

- Consider inhaled corticosteroids by MDI or hand-held nebulizer
- Antibiotics based on local bacterial resistance patterns, initiated in patients whose sputum characteristics have changed
  - Antibiotics include amoxicillin/clavulanate, respiratory fluoroquinolones (gatifloxacin, levofloxacin, moxifloxacin)
- If *Pseudomonas* spp. and/or *Enterobacteriaceae* spp. is suspected, consider combination therapy

WHEN TO REFER
The confirmation of a COPD diagnosis, especially its differentiation from asthma, may require specialized knowledge and testing. A consultation with a specialist and referral for pulmonary function tests (if not available in the primary care office) should be considered in certain circumstances (Table 10).

SUMMARY
COPD continues to be a major medical problem in the United States and globally, causing significant morbidity and mortality as well as placing a significant economic burden on health care systems. However, COPD continues to be underdiagnosed and misdiagnosed. Undiagnosed airflow obstruction is common in the general population and may be associated with impaired health and functional status. Smoking cessation remains the most important factor in preventing or treating COPD, although for many patients this is difficult to achieve.

Current therapies can improve health status and reduce exacerbations, with the goal of slowing the decline in lung function. However, the ability to reverse the course of the disease has not yet been achieved. COPD is usually progressive, and lung function can be expected to decline even when the best available care is administered. Practitioners must be aware that COPD exerts a debilitating effect on...
both the pulmonary system and other sites in the body and that optimal management must focus not only on airflow obstruction but also on the inflammatory component and systemic nature of the disease.

For standard management practices, such as confirming diagnosis of COPD with spirometry or assessing patients’ smoking status, office systems can help improve the quality of care delivered to patients with COPD. Strategies such as reminder systems, organizational changes, and patient education materials that are described throughout this monograph are integral to managing patients who have the chronic illness of COPD.

REFERENCES


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**COMBATING THE PSYCHOSOCIAL IMPACT OF COPD**

By Claire Murphy, RN, MSN, NP-C

COPD, along with its physical impact on daily activities, frequently includes an emotional and social component. The physical impairments of COPD can initiate concomitant feelings of fear, anxiety, depression, and despair. Because many patients maintain some degree of functional daily activity, these feelings often may be obscured, and the patient may not communicate them to the clinician unless questioned.

The innate capacity to breathe freely is a significant but unconscious task—unless you are a person with COPD. The sense of breathlessness and dyspnea can be overwhelming and produce intense feelings of panic and anxiety in the patient with COPD, especially with progression of the disease. Prolonged and chronic exacerbations can predispose the patient to fear and the avoidance of social and physical activities that might intensify symptoms. The familial impact of COPD may further elicit feelings of worthlessness and being a burden. A sedentary lifestyle involving limited physical endurance, decreased activity, and social isolation can affect sleeping patterns and nutritional status. Real and perceived alterations in socio-economic status as a result of the chronicity of the disease can contribute to feelings of despair and hopelessness, affecting the patient’s self-image.

In managing patients with COPD, clinicians must investigate and integrate optimal solutions that address the psychological aspects of care, using the same diligence and persistence that they use for medical management. During office visits, clinicians should routinely make targeted inquiries regarding reflective thoughts about the psychosocial impact of COPD. Consideration of referral to pulmonary rehabilitation programs and COPD support groups is essential for preserving optimal lung function and promoting participation in self-care. When necessary, referral to mental health specialists for further assessment, evaluation, and treatment of anxiety and depression also can be very beneficial to patients.

Often clinicians experience frustration in managing COPD in patients who are not exhibiting a positive response to treatment, due to noncompliance with the suggested course of therapy or to the evolving chronicity of the disease. A paucity of expertise in this specialty can further compound one’s feeling of frustration. Collaboration with pulmonary providers specializing in COPD management can offer valuable insight into other potential directions and modalities of treatment as well as provide support from peers and colleagues who deal with these issues.
CASE STUDY 1:
A 55-Year-Old Smoker With Chronic Cough and Shortness of Breath

By Dennis E. Niewoehner, MD

Mr. G is a 55-year-old white construction worker who had smoked regularly since age 17. He had generally good health and rarely sought medical attention. In recent years, he had difficulty “keeping up with the younger guys.” He also had a chronic productive cough with occasional persistent “chest colds.” He had not received an influenza vaccination for several years.

PATIENT PRESENTATION
A few days after Christmas, he developed a sore throat, a cough, a fever, and increasing breathing difficulties. Over the next 5 days, he became so short of breath that he went to the emergency department. He was febrile, had labored breathing with expiratory wheezing, and had oxygen saturation of 82% on room air.

DIAGNOSIS
Chest x-ray was unremarkable, but a rapid diagnostic test of a nasal swab was positive for influenza. Mr. G was diagnosed with severe COPD exacerbation due to influenza, a common cause of severe exacerbations in nonimmunized COPD patients, and was admitted to the hospital. Earlier diagnosis of COPD and appropriate management would likely have prevented this serious illness.

TREATMENT
Upon admission, Mr. G received regularly scheduled bronchodilators, oxygen, and oral prednisone (60 mg daily). His condition improved, and he was discharged on day 5 and given sufficient prednisone to complete a 10-day course, a short-acting bronchodilator for rescue use, and a long-acting bronchodilator for maintenance. Because his room air saturation had improved to 90%, home oxygen was not prescribed. Mr. G declined smoking cessation therapy. He was scheduled for a return clinic appointment and pulmonary function tests in 6 weeks.

Influenza antiviral therapy was not given because it is effective only if administered within 2 days of the onset of symptoms. As a specific viral etiology was identified, no antibiotic was required unless a secondary bacterial infection was suspected.

PATIENT FOLLOW-UP
On his return visit, Mr. G felt much better. Pulmonary function tests confirmed severe COPD: FEV₁ was 45% predicted and the FEV₁/FVC was 0.51. As Mr. G now agreed to quit smoking, nicotine replacement therapy was prescribed and he was referred to a support group. He received a pneumococcal vaccination and was encouraged to get an annual influenza vaccination and return to his job. He was educated about the symptoms of exacerbations and urged to seek medical attention as soon as they occurred. If Mr. G’s condition remains stable, follow-up visits should be scheduled at 6- to 12-month intervals.
CASE STUDY 2:
A 55-Year-Old White Woman With Shortness of Breath and Diminished Exercise Capacity
By Roy C. Blank, MD

Mrs. H is a 58-year-old white female homemaker who presents to the office complaining of shortness of breath. She reports a gradual but progressive decline in her exercise capacity. Dyspnea on exertion occurs when climbing 1-2 flights of stairs and at 1 city block if she is walking up an incline. She reports some difficulty completing more strenuous household chores. Mrs. H experiences fatigue every afternoon and a chronic cough most of the time. Her symptoms developed over several years, and she has difficulty providing an exact time of onset. She denies having orthopnea, paroxysmal nocturnal dyspnea, exertional chest pain or any symptoms of chest tightness during exercise.

PATIENT HISTORY
Mrs. H is being treated for dyslipidemia and hypertension. She has no previous history of malignancy, diabetes mellitus, or heart disease. Her medications include pravastatin 40 mg daily, lisinopril/hydrochlorothiazide 20/12.5 mg daily, and an over-the-counter inhaled bronchodilator that has provided minimal benefit.

She has smoked 1 pack of cigarettes daily for the past 30 years. Mrs. H has minimal alcohol intake and no previous occupational exposure to respiratory irritants. Her family history shows no indication of increased incidence of pulmonary disease.

PHYSICAL EXAMINATION
On physical examination, blood pressure is 126/78, pulse is 74 beats/minute, and respirations are 14/minute. Height is 72 in, weight is 192 lb, and body mass index is 25.6. Head, ear, eye, nose, and throat examination is negative; thyroid is normal. Cardiovascular examination is negative, with no findings for congestive heart failure; there are normal peripheral pulses, no bruits, and no edema. Pulmonary examination shows a mild reduction in breath sounds with a few scattered expiratory wheezes. Abdominal and neurologic examinations are negative. Examination of extremities is negative, with no cyanosis or nail clubbing.

DIAGNOSTIC TESTING
In-office testing shows that EKG is normal. Chest x-ray shows no suggestive findings for COPD and no other abnormalities. Echocardiogram reveals normal left ventricular function, no valvular abnormalities, and no increase in pulmonary pressure. Spirometry results show that FEV1/FVC is 60% and FEV1 is 55% predicted. Pulse oximetry is 95%. Hemoglobin and brain natriuretic peptide are normal.

DIAGNOSIS
Mrs. H is advised that she has moderately severe COPD that explains her shortness of breath. The physician discusses with her the chronic nature of COPD and makes specific recommendations regarding disease management. An appointment is made to see the office nurse practitioner for chronic pulmonary disease instruction, initiation of medications, and smoking cessation therapy. The office nurse practitioner provides educational materials about smoking cessation and COPD.

TREATMENT
Varenicline is prescribed and the nurse practitioner reviews potential side effects with the patient. Tiotropium 18 mcg daily via inhalation is initiated. A short-acting beta-agonist inhaler is provided for as needed PRN use, and the over-the-counter bronchodilator is discontinued. The nurse practitioner administers pneumococcal vaccination and schedules a reminder for influenza vaccination for the appropriate time of year. Referral is made to home respiratory service for nocturnal pulse oximetry.

PATIENT FOLLOW-UP
At 1-month follow-up with the physician, Mrs. H reports success with smoking cessation. Her normal nocturnal pulse oximetry result is reviewed. Mrs. H is compliant with her medications; she reports some improvement in her symptom of shortness of breath and has not needed the PRN bronchodilator. A follow-up physician visit in 3 months is scheduled, and Mrs. H is advised to call the office if she relapses with smoking or has any worsening of her symptoms.
Continued from Page 20

1. Approximately 10 million US adults reported physician-diagnosed COPD in 2000; however, NHANES III data indicate that impaired lung function actually affects about:
   A. 5 million US adults
   B. 16 million US adults
   C. 24 million US adults
   D. 45 million US adults

2. Analysis of CDC surveillance data for 1980-2000 showed that COPD-related mortality rates:
   A. Increased substantially in men and decreased substantially in women
   B. Increased substantially in women and increased slightly in men
   C. Increased substantially in women and decreased substantially in men
   D. Decreased substantially in both women and men

3. Clinicians should consider COPD in a patient with a history of exposure to which of the following risk factors?
   A. Tobacco smoke
   B. Occupational dusts and chemicals
   C. Smoke from home cooking and heating
   D. All of the above

4. The hallmark symptom of COPD and frequently the reason that patients seek medical attention is:
   A. Anorexia
   B. Bronchospasm
   C. Congestion
   D. Dyspnea

5. The most objective measure of airflow limitation and a key component in confirming a diagnosis of COPD is:
   A. Physical examination
   B. Chest x-ray
   C. Spirometry testing
   D. Measurement of alpha 1-antitrypsin levels

6. Which statement is true about smoking cessation for patients with COPD?
   A. Once a diagnosis of COPD has been made, smoking cessation will have no benefit.
   B. Group counseling for smoking cessation has been shown to be effective in about 0.5% of patients.
   C. Pharmacotherapy for smoking cessation is not an option for patients with COPD.
   D. Smoking cessation is the single most effective intervention available in the management of COPD.

7. A characteristic of long-acting bronchodilators is:
   A. Their extended period of action reduces the need for multiple dosing
   B. They lose their effectiveness with regular use
   C. They are available only in oral formulations
   D. They may be less effective than short-acting bronchodilators

8. Agents considered first-line treatments in the management of stable COPD include:
   A. Inhaled corticosteroids
   B. Methylxanthines
   C. Inhaled beta-agonist and anticholinergic bronchodilators
   D. Chronic oxygen therapy

9. Exacerbations of COPD:
   A. May occur at any stage of the disease
   B. Are more common in patients with severe disease
   C. Are commonly caused by infections
   D. All of the above

10. Which of the following is true about therapies for COPD?
    A. Pneumococcal immunization is recommended for patients aged ≥65 years and for those with severely impaired lung function
    B. Supplemental oxygen is recommended for all COPD patients who smoke
    C. The most common surgical intervention for COPD is bullectomy
    D. Combined therapy with salmeterol and fluticasone was less effective in reducing exacerbations than either drug given alone
COPD: Practical Strategies for Improved Diagnosis and Effective Management

Darken the circle with the correct answer(s) to each question in this activity.

3. A B C D 6. A B C D

In order to obtain credit, you must 1) Complete the post-test (a score of 70% or better must be achieved); 2) Complete the program evaluation form; 3) Mail or fax your completed post-test answers and Evaluation Form to: Code E.COPDHAYM08, Boston University School of Medicine Continuing Medical Education, 715 Albany Street, A305, Boston, MA 02118; Fax: 617-638-4905. Credit is available through February 14, 2009. For questions, please contact BUSM CME at 617-638-4605. Please allow 4-6 weeks after receipt of post-test and evaluation to receive certificate.

Name

Specialty

Institution

Street

City State ZIP

Telephone Fax

E-mail

(All information is confidential)

The amount of time I spent on this activity was _______________________ (max. 1 hour).

CME/CE EVALUATION FORM

1. How would you rate this activity overall? (5 = excellent, 1 = poor; please circle one) 5 4 3 2 1

2. In your opinion, did you perceive any commercial bias?  ❏ Yes  ❏ No  If yes, please explain:

3. Do you plan on making any changes in your practice as a result of this activity?  ❏ Yes  ❏ No  If yes, please explain:

4. What barriers, if any, do you anticipate encountering as you make changes in your practice?

5. Do you feel each of the following objectives was met?

<table>
<thead>
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<th>Objective</th>
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<th>No</th>
<th>Partially</th>
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<td>Cite the prevalence of COPD and burden of disease</td>
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<td>Identify the symptoms of COPD</td>
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<td>Describe available diagnostic tests</td>
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<td>Explain nonpharmacologic management and techniques for patient education</td>
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<td>Describe the available pharmacologic treatments for COPD</td>
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6. Do you feel that the information in this activity was based on the best evidence available?  ❏ Yes  ❏ No  If yes, please explain:

7. Which of the following competency areas do you feel have been improved as a result of this activity? (Mark all that apply)

<table>
<thead>
<tr>
<th>Competency Area</th>
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<td>Communication skills</td>
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8. Do you feel you need further education on this topic?  ❏ Yes  ❏ No  If yes, please specify:

9. Do you have any suggestions

10. Please rate the content of this activity (5 = excellent, 1 = poor; please circle one)

<table>
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<th>9a. Timely, up to date?</th>
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<td>9b. Relevant to your practice?</td>
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11. General Comments:

______________________________________________________________________________

______________________________________________________________________________

______________________________________________________________________________
Improve your Practice, earn up to 26 AMA PRA Category 1 Credits™ by Joining a COPD Virtual Community of Practice

Boston University School of Medicine has launched a COPD Performance Improvement initiative and as a participant of the Core Curriculum program, we would like to invite you to join. This program is targeted toward physicians, nurse practitioners, and physician assistants who are committed to making practice changes in the diagnosis and management of their patients with chronic obstructive pulmonary disease (COPD). In addition to a teleconference series, participants will be guided by experts through a performance improvement initiative that will allow them to make improvements in the care of their COPD patients.

The data you collect and the improvements you make can be used for re-certification and incentive initiatives with your managed care organizations and Centers for Medicare & Medicaid Services (CMS).

How to Participate
Participation is simple and self-directed and should take only a few hours for the entire program.
Stage A: Review 10 charts using our data collection tool to assess your current practice and identify areas for improvement. (Estimated time to complete each chart review is 7-8 minutes.) 5 AMA PRA Category 1 Credits™ for completion of Stage A
Stage B: Create and Implement Your Individualized Action Plan designed to outline the changes you plan to make in your practice. (Estimated time to complete is 1 hour.) 5 AMA PRA Category 1 Credit™ for completion of Stage B.
Stage C: 3 months after you have implemented your Individualized Action Plan, complete Review 10 additional charts using our data collection tool to see if you have improved your practice. (Estimated time to complete each chart review is 7-8 minutes.) 10 AMA PRA Category 1 Credits™ for completion of Stage C.

The teleconferences will allow you to discuss your findings from the chart review and your ideas for improving your practice with your colleagues and with experts in the field.

Learning Objectives
As a result of this activity, participants will:
• Assess their state of practice regarding the screening and treatment of patients with potential COPD
• Identify their knowledge gaps regarding the screening and treatment of patients with COPD
• Discover barriers in their practice regarding the treatment of these patients
• Develop and implement an action plan designed to improve their practice in the screening and treatment of patients with COPD
• Improve their practice of screening and treating patients with COPD
• Evaluate the results of their action plans and reflect on future opportunities for change

Target Audience
Primary care physicians, nurse practitioners, and physician assistants

Accreditation
Boston University School of Medicine is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.
Boston University School of Medicine designates this educational activity for a maximum of 26 AMA PRA Category 1 Credit(s)™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Grant Support
This program is supported by an unrestricted educational grant from Boehringer Ingelheim and Pfizer Inc.

Program is available through September 2011.

Go to http://www.bu.edu/cme/seminars/COPD/index.html for more information and to register.