

Taming the Epidemic: A New Treatment Paradigm

We can reduce COPD related morbidity and mortality by focusing our attention and resources on proven interventions and by routinely performing annual spirometry to monitor changes.



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Chronic obstructive pulmonary disease (COPD) is currently the fourth leading cause of death in the United States and is projected to be the third leading cause of death—in both men and women—by 2020. A concerted effort is called for to slow this increase and reduce mortality. Effective treatment of COPD requires a thorough understanding of pharmacologic therapies, knowing when pulmonary rehabilitation is most beneficial, and judging when admission is warranted to manage an exacerbation.

This comprehensive course presents the current best practices for managing the many facets of COPD including the newly updated recommendations from the Global Initiative for Chronic Obstructive Lung Disease (GOLD). Throughout this course and within the pages of this journal you will find materials to help you build an extensive, practical toolkit that can be used to immediately improve provision of care to your patients with COPD.

Chronic obstructive pulmonary disease (COPD) is insidious because it is often not recognized until after age 50, when lung damage is already severe and lung function is diminished by as much as 50%. Unlike coronary artery disease and stroke where incremental advancements have been made to reduce the rate of death from disease, COPD deaths are increasing. COPD related mortality rates have risen by more than 160% over the last 3 decades. Much of the increase has occurred in women, whereas in recent

years, the mortality rate has been fairly stable in men.¹ COPD is currently the fourth leading cause of death and is predicted to become the third leading cause of death in the United States by 2020.²

Conventional wisdom suggests that we can do very little for our patients with COPD, but this is simply not true. We *can* help reduce COPD-related morbidity and mortality by focusing our attention and resources on proven interventions

Learning Objectives

Upon completion of this course, you should be able to:

- ◆ Treat a patient with COPD using the therapeutic options described
- ◆ Develop treatment plans for your patients with COPD based on evidence-based practice
- ◆ Adjust treatment for COPD as the patient's condition changes

For information about earning credit for this activity, see page 2. Completion of this self-study activity should take about 1 hour.

Course ID: AB0490

Abbreviations

AAT	α_1 -Antitrypsin
COPD	chronic obstructive pulmonary disease
DPI	dry-powder inhaler
FEV ₁	forced expiratory volume in 1 second
FVC	forced vital capacity
GOLD	Global Initiative for Chronic Obstructive Lung Disease
MDI	metered-dose inhaler
PaO ₂	partial pressure of oxygen
PaCO ₂	partial pressure of carbon dioxide
SaO ₂	arterial oxygen saturation

shown to slow or alter the progression of disease, by educating our patients and colleagues about the changing face of COPD, and by urging people to stop smoking. As many as 80% of all COPD diagnoses are related to smoking which is why smoking cessation alone stands to have an enormous impact on the burden of disease.¹

Defining COPD

COPD is a disease state characterized by airflow limitation that is not completely reversible. In most cases, the airflow limitation is progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases.³

COPD is primarily comprised of two related diseases: chronic bronchitis and emphysema. Chronic bronchitis is defined as cough and sputum production lasting at least 3 months for at least 2 consecutive

years, often in association with frequent bronchial infections. Mucus production alone, however, is not necessarily damaging. COPD occurs when inflammation of the airways cause permanent narrowing of the bronchioles. Chronic bronchitis without permanent airflow obstruction is *not* considered COPD.

Emphysema is a pathologic term that indicates abnormal and permanent enlargement of the distal airspaces, reduction in the number of alveoli, and destruction of the alveolar walls with diminished elastic recoil. Loss of gas-exchange surface area causes decreased oxygenation, which may lead to hypoxemia. Loss of elastic recoil leads to air trapping on expiration, increasing residual volume (barrel-chest appearance).³⁻⁵ [For detailed information on the pathophysiology and causes of COPD, see Course AB0491, *What Is COPD?*, on the companion CD.]

Exercise 1

COPD, emphysema, and chronic bronchitis are interchangeable terms for the same disease entity.

- True
- False

Answer on page 26.

COPD is often confused with asthma, a pulmonary disease in which airflow obstruction is typically reversible. With optimal treatment, patients with asthma can usually achieve normal airflow through the airways. Patients with COPD, on the other hand, have airflow

obstruction that does not fully reverse, even with maximum therapy. Confusion often arises since the major obstructive lung diseases commonly overlap in etiology and patient presentation. COPD can have features of chronic bronchitis, emphysema, and/or asthma, but only if the airflow obstruction is not fully reversible.^{3,6} For this reason, careful differential diagnosis is the fundamental first step in caring for any patient with COPD. [For practical tools about how to diagnose and differentiate COPD, see Course AB0492, *Is It COPD? Making the Diagnosis*, on the companion CD.]

Key Symptoms and Risk Factors

The hallmark symptoms of COPD are cough, sputum production, and dyspnea on exertion. Since the 1950s, cigarette smoking has been the major known risk factor for COPD; however, genetic and environmental factors clearly contribute to COPD as well.⁷ Although as many as 80–90% of patients with COPD have a past or current history of smoking,^{1,8,9} only 10–20% of smokers ever develop symptomatic COPD.¹⁰ Individuals clearly vary in terms of their susceptibility to COPD and the effects of tobacco exposure.

Emerging data from epidemiologic studies and genetic research in twins and animal models suggest that several mechanisms, including exposure to cigarette smoke and industrial or environment pollutants, host factors (susceptibility genes and lung infections), and genetic-smoking interactions contribute to the complex disease of COPD.¹⁰⁻¹² At present, only α_1 -antitrypsin (AAT) deficiency has a proven genetic link to COPD, though several specific genes have been implicated.^{10,11} [For more information about genetic, behavioral, and environmental risk factors, see Course AB0491, *What Is COPD?*, on the companion CD.]

Components of COPD Treatment

According to the newly updated GOLD guidelines, “A COPD management program includes four components: assess and monitor disease, reduce risk factors, manage stable COPD, and manage exacerbations.”²³ Desired clinical

Quantifying COPD^{1,2,9,15}

In the United States

- ◆ **120,000** — Deaths from COPD in 2002: 61,000 women and 59,000 men (Women have exceeded men in COPD-related deaths since 2000.)
- ◆ **700,000** — Hospitalizations in 1998, or about 13% of all hospitalizations
- ◆ **10.7 million** — Prevalence of COPD
- ◆ **13 million** — Estimated prevalence of undiagnosed COPD or impaired lung function
- ◆ **\$30-37 billion** — Cost to the economy in 2004

Worldwide

- ◆ **75%** — People with COPD who are untreated
- ◆ **80%** — Patients hospitalized for an exacerbation who report a quality of life “worse than death”

outcomes of therapy are controlling reversible elements of airflow limitation, mucosal edema and congestion, mucus hypersecretion, smooth-muscle bronchoconstriction and leukocyte-mediated inflammation, abnormal lung mechanics, hypoxemia, and physiologic deconditioning.¹³ Table 1 summarizes the goals of COPD care.³

Step 1: Assess and Monitor Disease

In the past, COPD was most often diagnosed on the basis of productive cough and shortness of breath. However, current guidelines emphasize objective measurement of irreversible airflow limitation, with or without evident symptoms.³ Therefore, all patients who have positive risk factors should be tested, even if they are asymptomatic since lung damage may remain subclinical for many years.¹⁴ COPD is seriously underdiagnosed, especially in women and in people who have never smoked. Current epidemiologic data suggest that as many as 13 million Americans have undiagnosed—and therefore untreated—COPD.^{9,15} [For a number of evidence-based tools to help you screen patients for lung dysfunction, see Course AB0492, *Is It COPD? Making the Diagnosis*, on the companion CD.]

Spirometry is the standard for confirming COPD, where an FEV₁/FVC ratio of less than 70% [ratio of forced expiratory volume in 1 second (FEV₁) to forced vital capacity (FVC)] combined with a postbronchodilator FEV₁ of less than 80% predicted value establishes

Table 1: Goals of COPD Management³

- ◆ Smoking cessation
- ◆ Relief of symptoms
- ◆ Improvement of
 - Health status
 - Exercise tolerance
- ◆ Reduction of
 - Risk factors
 - Mortality risk
- ◆ Prevention of
 - Exacerbations
 - Disease progression
 - Complications
 - Adverse effects of treatment

Table 2. Questions for Disease Monitoring

To monitor exposure to risk factors:
<ul style="list-style-type: none"> ◆ Have you been smoking cigarettes? <ul style="list-style-type: none"> • If so, how many cigarettes per day are you smoking? • Would you like to quit smoking? ◆ Has there been any change in your working environment?
To monitor disease progression and development of complications:
<ul style="list-style-type: none"> ◆ How much can you do before you get short of breath? (Use an everyday example, such as walking up flights of stairs, up a hill, or on flat ground.) ◆ Has your dyspnea worsened, improved, or stayed the same since your last visit? ◆ Have you had to reduce your activities because of dyspnea or other symptoms? ◆ Have any of your symptoms worsened since your last visit? ◆ Have you experienced any new symptoms since your last visit? ◆ Has your sleep been disrupted because of dyspnea or other symptoms? ◆ Since your last visit, have you missed any work because of your symptoms?
To monitor pharmacotherapy and other medical treatment:
<ul style="list-style-type: none"> ◆ What medications are you taking? <ul style="list-style-type: none"> • How often do you take each medication? • How much do you take each time? • Has your medication been effective in controlling your symptoms? • Has your medication caused you any problems? ◆ Have you missed or stopped taking any regular doses of your medications for any reason? ◆ Have you had trouble filling your prescriptions (eg, because of financial reasons or because the drug is not on the formulary)? ◆ Could you please show me how you use your inhaler? ◆ Have you tried any other medicines or remedies?
To monitor exacerbation history:
<ul style="list-style-type: none"> ◆ Since your last visit, have you had any episodes or times when your symptoms were a lot worse than usual? <ul style="list-style-type: none"> • If so, how long did the episode(s) last? • What do you think caused the symptoms to get worse? • What did you do to control the symptoms?

Adapted from: Global Initiative for Chronic Obstructive Lung Disease, 2004.

airflow limitation that is not fully reversible. Healthcare providers who care for patients with COPD should have access to spirometric testing for both initial diagnosis and follow-up monitoring. For patients with an FEV₁ of less than 40% predicted value, the addition of arterial blood gas analysis assists in evaluating possible respiratory or right-sided heart failure.¹⁴ [For a refresher course on how to perform spirometry and interpret the results, see Course AB0493, *Using Spirometry Effectively as a Diagnostic Tool*, on the companion CD.] On follow-up visits, patients should be asked about new or persistent exposure to risk factors, disease progression and complications, and COPD exacerbations (Table 2).¹⁶

Step 2: Modification of Risk Factors

Risk factor modification and education are essential for all patients. The main areas of focus are smoking cessation, the development of self-care and coping skills, and lifestyle changes to improve the patient's overall health status. In healthy adults, respiratory function naturally declines after age 25, with FEV₁ decreasing at a rate of 20–30 mL/year.¹⁷ Tobacco exposure accelerates this loss in an approximate dose-response fashion. Therefore, quitting smoking is by far the single most effective and cost-effective means of preventing COPD and slowing its progression. Other important measures are limiting the patient's exposure to occupational and environmental pollutants, such as industrial chemicals,

Table 3: Recommended Treatment for Each Stage of COPD³

Stage	0: At risk	I: Mild	II: Moderate	III: Severe	IV: Very Severe
Characteristics	<ul style="list-style-type: none"> ◆ Chronic symptoms ◆ Risk factors ◆ Normal spirometry 	<ul style="list-style-type: none"> ◆ FEV₁/FVC < 70% ◆ FEV₁ ≥ 80% ◆ With or without symptoms 	<ul style="list-style-type: none"> ◆ FEV₁/FVC < 70% ◆ 50% ≤ FEV₁ < 80% ◆ With or without symptoms 	<ul style="list-style-type: none"> ◆ FEV₁/FVC < 70% ◆ 30% ≤ FEV₁ < 50% ◆ With or without symptoms 	<ul style="list-style-type: none"> ◆ FEV₁/FVC < 70% ◆ FEV₁ < 30% or FEV₁ < 50% of predicted plus chronic respiratory failure
Treatment	Avoidance of risk factors; influenza vaccination				
	Add a short-acting bronchodilator when needed				
	Add regular treatment with ≥1 long-acting bronchodilators Add rehabilitation				
	Add inhaled glucocorticoids if repeated exacerbations				
	Add long-term oxygen if chronic respiratory failure Consider surgical treatments				

Source: The Global Initiative for Chronic Obstructive Lung Disease (GOLD). www.goldcopd.org/Guidelineitem.asp?l1=2&l2=1&intl=1116

noxious gases, household dust, and air pollution.^{14,17} [For useful quit-smoking resources, see Course AB0495, *A Patient-Centered Approach to Smoking Cessation*, on the companion CD.]

Step 3: Manage Stable Disease

Drug treatment is aimed at preventing and improving the patient's symptoms, reducing the frequency and severity of acute exacerbations, and improving the patient's overall quality of life and exercise tolerance or ability to perform their activities of daily living.³

The current recommended approach to managing stable COPD is a stepwise increase in treatment based on the five stages of COPD (Table 3). The major drug classes used to treat stable disease are short- and long-acting bronchodilators, corticosteroids, and vaccines (Table 4).

Nonpharmacologic options for advanced disease include oxygen therapy and various surgical procedures. Although none of the current medical therapies halts or reverses the progression of COPD, they can effectively reduce

the severity and frequency of symptoms and complications.¹⁴

Bronchodilators

The first-line agents for treating COPD are the bronchodilators, mainly β_2 -agonists and anticholinergics. Table 4 shows available medications for COPD, including short-acting rescue medications and long-acting maintenance therapy. Selection of the specific agent or combination should be based on the availability of the drug and on the patient's therapeutic responses and adverse reactions. A combination of different classes of bronchodilators may be safer and more effective than escalating dosages of monotherapy if initial treatment is inadequate.¹⁴

Long-acting agents salmeterol or formoterol are taken twice daily to support bronchodilation through the day and to possibly help clear excessive mucus. An anticholinergic might be added to enhance bronchodilation because of its different mechanism of action.¹³ The newest anticholinergic, tiotropium, appears to be more effective than

ipratropium in achieving bronchodilation, and it offers the convenience of one dose per day compared with four doses per day for ipratropium. In clinical trials, adverse effects for the two drugs were similar, except for an increased incidence of dry mouth with tiotropium (15% vs 10%).¹⁸ Although theophylline has been used to manage COPD for nearly a century, it is currently a third-line agent and usually added to existing maintenance therapy. Theophylline is thought to inhibit inflammation and thus limit hyperinflation of the small airways. However, nausea and headaches are common adverse effects.¹⁹

Glucocorticosteroids

Inhaled glucocorticosteroids are indicated to reduce the frequency of recurrent exacerbations in patients with symptomatic severe (stage III) or very severe (stage IV) COPD and thus preserve their overall health status. However, long-term use of systemic (oral) glucocorticosteroids is not advised because adverse effects (eg, steroid myopathy) may outweigh therapeutic benefits, as the drugs do not halt the ongoing decline in

FEV₁.¹⁴ Several studies have shown that an inhaled glucocorticosteroid combined with a long-acting β -agonist is more effective in stabilizing lung function and delaying exacerbations than either drug alone.^{14,20,21} In a randomized, double-blind multicenter study, combination therapy was also more effective than an anticholinergic plus a short-acting β -agonist (fluticasone propionate–salmeterol vs ipratropium bromide–albuterol [salbutamol]) in controlling lung function and symptoms.²² Some guidelines recommend a 2-week trial of an oral glucocorticosteroid (eg, prednisolone) to identify patients who might benefit from long-term glucocorticosteroid therapy. However, current evidence suggests that current smoking significantly blunts the response to the trial ($P < 0.001$) and that the short-term response is a poor predictor of future benefit.^{14,23}

Exercise 2

A 50-year-old man owns a furniture refinishing business. He presents with a chronic (nonproductive) cough and dyspnea while walking up stairs or for long distances (eg, more than a couple blocks). He has a smoking history of 31 pack-years. What COPD-related interventions are recommended for this patient?

- Education about reducing risk factors
- Smoking cessation
- Annual influenza vaccination
- A, B, and C

Answer on page 26.

Other Medications

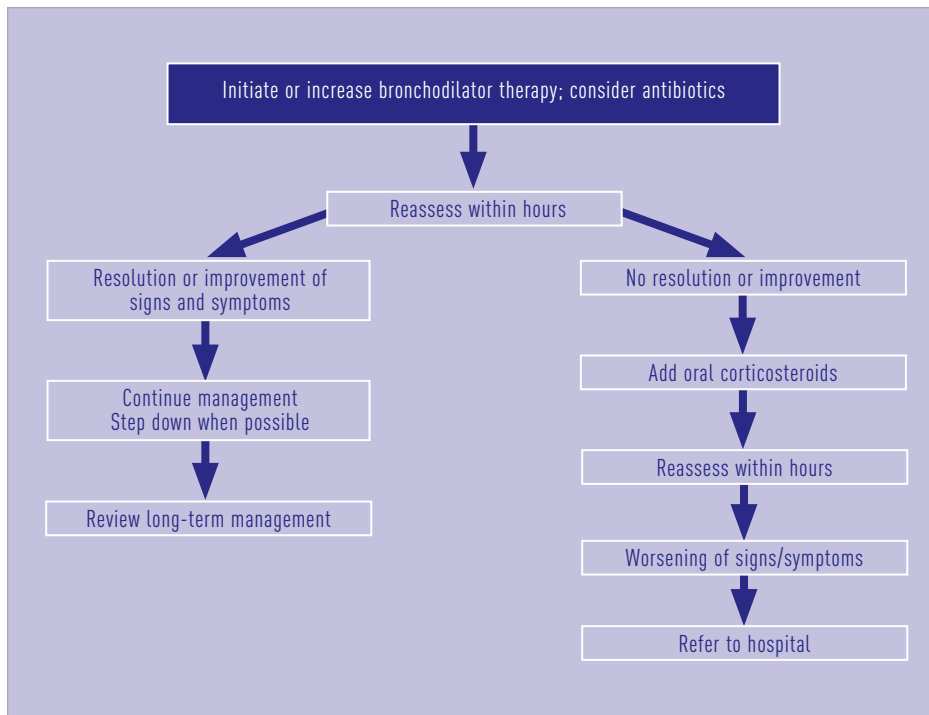
All patients with COPD or risk factors should receive annual vaccination with inactivated influenzae viruses in the fall and/or winter.¹⁴ Antibiotics should generally be reserved for confirmed bacterial infections, and routine use of mucolytic agents is not recommended because they seem to offer limited benefit in only some patients with viscous secretions. Likewise, antitussives should not be prescribed because they hinder the protective action of coughing in patients with COPD.¹⁴

Table 4: Drugs Commonly Used to Manage COPD¹⁴

Drug	Inhaler (μ g)	Solution for Nebulizer (mg/mL)	Oral	Vials for Injection (mg)	Duration of Action (hours)
β_2-agonists					
Short-acting					
Albuterol	90 (MDI)	2.5, 5	2, 4, 8 mg tablet; 2 mg/5 mL syrup		4–6
Levalbuterol		0.31 mg/3 mL, 0.63 mg/3 mL, 1.25 mg/3 mL			5–8
Pirbuterol	200 (MDI)				4–6
Terbutaline			2.5, 5 mg tablet	0.25, 0.5	4–6
Long-acting					
Formoterol	12 (DPI)				12+
Salmeterol	50 (DPI)				12+
Anticholinergics					
Short-acting					
Ipratropium bromide	18 (MDI)	0.25–0.5			6–8
Long-acting					
Tiotropium	18 (DPI)				24+
Combination short-acting β_2-agonist plus anticholinergic in one inhaler					
Albuterol/Ipratropium	90/18 (MDI)	2.5/0.5			6–8
Methylxanthines					
Aminophylline				240 mg	Variable, up to 24
Theophylline (SR)			100–600 mg tablet		Variable, up to 24
Inhaled glucocorticosteroids					
Beclomethasone	40, 80 (MDI)				
Budesonide	200 (DPI)	0.25/2 mL, 0.5/2 mL			
Fluticasone	44, 110, 220 (MDI)				
Triamcinolone	100 (MDI)				
Flunisolide	250 (MDI)				
Combination long-acting β_2-agonist plus glucocorticosteroid in one inhaler					
Fluticasone/Salmeterol	100/50, 250/50, 500/50 (DPI)				
Systemic glucocorticosteroids					
Prednisone			5–60 mg tablet; 5 mg/5 mL liquid		
Methylprednisolone			2, 4, 8, 16, 24, 32 mg tablet		
Prednisolone			5 mg tablet; 5 mg/5 mL, 15 mg/5 mL liquid		

MDI is metered-dose inhaler; DPI, dry-powder inhaler. Adapted from: Global Initiative for Chronic Obstructive Lung Disease, updated 2005.

Figure 1: How to Manage COPD Exacerbations in Home-Care and Outpatient Settings¹⁴



Adapted from: Global Initiative for Chronic Obstructive Lung Disease, updated 2005.

Finally, severe autosomal-codominant AAT deficiency is responsible for 5% or less of all cases of emphysema in the United States.⁹ Therefore, young patients (ie, those in their 30s or 40s) with AAT-related COPD may benefit from augmentation therapy,⁹ though this treatment is expensive (total healthcare costs about \$40,000 per year).²⁴ Still, the World Health Organization (WHO), American Thoracic Society (ATS) and the European Respiratory Society (ERS) recommend that all individuals with COPD be tested for AAT deficiency with a simple blood or mouth-swab test.²⁵

Drug-Delivery Devices

Bronchodilators and corticosteroids are delivered by means of several devices, including metered-dose inhalers (MDIs), nebulizers, several types of dry-powder inhaler (DPI), and nebulizers. The wide variety of devices and differences in the instructions for each type are common causes of confusion and error by patients, especially elderly patients, as well as healthcare professionals. With MDIs, frequent problems are a lack of coordination to accomplish breath actuation (27% of patients), the patient's inability to hold his or her breath long

enough after inhalation (26%), inspiratory flow that is too fast (19%), inadequate shaking or mixing of the drug before use (13%), and abruptly stopping inspiration when the cold aerosol spray hits the patient's throat (6%).

Although spacers and holding chambers are intended to simplify inhalation with MDIs and to limit deposition of the drug in the oropharyngeal space, these add-on devices can result in problems such as added cost, incorrect assembly, and bulkiness and lack of portability. With DPIs, errors in technique are most common. Examples are failure to hold the device upright (35%), to fully exhale before inhaling (24%), to hold one's breath (23%), to exhale through the mouthpiece (19–20%), and to inhale forcefully (17%).²⁶

Step 4: Manage Exacerbations

Because COPD is often associated with exacerbations of symptoms, patients with COPD should be given a short-acting β -agonist, such as albuterol, with or without a short-acting anticholinergic agent to manage acute episodes. Many studies show that a combination of these drugs is more effective than either alone because their different mechanisms of action are additive or possibly synergistic.¹³

Table 5 summarizes the symptoms and spirometric, laboratory, and imaging results that are the most clinically relevant markers of COPD exacerbations.¹⁴

Many exacerbations—and increasingly those related to end-stage COPD—are managed at home or in outpatient settings.¹⁴ Figure 1 and Table 6 show the how to address most cases and when to refer patients for admission. The mainstays of home care of exacerbations are increasing doses and/or frequencies of established bronchodilator treatment and possibly the addition of an anticholinergic drug. For severe episodes, short-term, high-dose nebulizer therapy might be helpful. In patients with a baseline FEV₁ of <50% of predicted, a systemic corticosteroid (eg, prednisolone 40 mg/day for 10 days) may hasten the resolution of symptoms and the recovery of lung function.¹⁴

Table 5: Markers of COPD Exacerbations¹⁴

Marker	Diagnostic	Indicative of Severity	Indicative of Treatment Response	Predictive
FEV ₁ [% of predicted]	No	Possibly	Possibly	Yes
PaO ₂ , PaCO ₂ , SaO ₂	No	Possibly	Yes	Uncertain*
Sputum volume and color	Yes	Yes	Yes	Uncertain*
Imaging	Possibly	No	No	No
Inflammatory markers	Uncertain*	Uncertain*	Uncertain*	Possibly

PaO₂ = partial pressure of oxygen; PaCO₂ = partial pressure of carbon dioxide; SaO₂ = arterial oxygen saturation
*Insufficient evidence

Table 6: When to Admit a Patient for Inpatient Management of an Exacerbation^{3,14}

Admit a Patient to the <i>Hospital</i> When He or She Has:	Admit a Patient to the <i>Intensive Care Unit</i> When He or She Has:
<ol style="list-style-type: none"> 1. A marked increase in intensity of symptoms, such as the sudden development of resting dyspnea 2. Severe background COPD 3. New physical signs (eg, cyanosis, peripheral edema) 4. An exacerbation that does not respond to initial medical management 5. Clinically significant comorbidities 6. New arrhythmias 7. An uncertain diagnostic 8. An older age 9. Insufficient home support 	<ol style="list-style-type: none"> 1. Severe dyspnea that inadequately responds to initial emergency therapy 2. Confusion, lethargy, or coma 3. Any of the following, despite supplemental oxygen and noninvasive intermittent positive pressure ventilation: <ul style="list-style-type: none"> ◆ Persistent or worsening hypoxemia (PaO₂ < 5.3 kPa, 40 mm Hg) ◆ Severe or worsening hypercapnia (PaCO₂ > 8.0 kPa, 60 mm Hg) ◆ Severe or worsening respiratory acidosis (pH < 7.25)

Patients with severe COPD are at highest risk for respiratory acidosis, clinically significant comorbidities, and a need for controlled oxygen therapy or mechanical ventilation. Inpatient care may offer such patients the best likelihood of recovery. Patients hospitalized for COPD exacerbations generally receive supplemental oxygen to achieve 80 mm Hg (which will vary with supplemental oxygen and altitude) or SaO₂ of greater than 90%. Arterial blood gases should be monitored every 30 minutes. Methods of delivery range from nasal cannulas and Venturi masks to noninvasive negative- or positive-pressure ventilation to invasive ventilation by means of an orotracheal or nasotracheal tubes or tracheostomy.¹⁴

Strategies for Managing Advanced COPD

For some patients with advanced COPD, additional strategies of pulmonary rehabilitation, long-term home oxygen therapy (LTOT), and/or surgery may be needed.

Pulmonary Rehabilitation

Pulmonary rehabilitation is indicated beginning at stage II, or moderate COPD, as part of proactive and preventive care for patients with COPD. The updated 2005 GOLD guidelines highlight the early implementation of this strategy to reduce patients' symptoms, improve their quality of life, and support their daily physical and emotional function. Forward-thinking

pulmonary rehabilitation should be designed to address exercise deconditioning, social isolation, mood (especially depression), nutrition, muscle wasting, and weight loss.

Exercise 3

A 52-year-old woman has symptomatic stage I COPD. She is currently taking albuterol by MDI as needed. She calls you today because she has worsening symptoms that have not been fully relieved with the usual dose of her medication. What next treatment is appropriate for this patient?

- a. Increasing the dose of her short-acting β-agonist
- b. Adding an inhaled corticosteroid to her regimen
- c. Replacing her current medication with a long-acting β-agonist
- d. A or B

Answer on page 26.

Patients with any stage of COPD can benefit from exercise training. Data from one randomized controlled study illustrate the potential benefits. In this study, 42 patients hospitalized for acute exacerbations received standard treatment, including bronchodilators (by nebulizer), oxygen, oral or intravenous antibiotics, noninvasive ventilation as needed, and oral prednisolone (30–40 mg/day for 1–2 weeks).²⁶ Within 10 days of discharge,

patients in the study group (mean age ± standard deviation, 70 ± 9 years; FEV₁, 41.7% ± 18.9 of predicted) were enrolled in community-based pulmonary rehabilitation. A multidisciplinary team (comprising a physiotherapist, occupational therapist, dietician, pulmonologist, smoking-cessation advisor, social worker, pharmacist, and lay person) conducted two classes per week for 8 weeks. Each class consisted of aerobic and/or strength training for 1 hour and patient education in self-care, nutrition, and lifestyle modification for 1 hour. The team also customized exercises for each patient to perform at home for at least 20 minutes/day.

Early rehabilitation was safe, causing no adverse events. After 3 months, patients in the rehabilitation experienced significant improvements in both exercise capacity and health status (both *P* ≤ 0.02). In addition, patients receiving early pulmonary rehabilitation tended to have fewer emergency visits and fewer and

Table 7: Medicare Criteria for LTOT^{28,29}

Group	Criteria
I	<ul style="list-style-type: none"> ◆ PaO₂ < 55 mm Hg <i>and</i> ◆ SaO₂ < 88%
II	<ul style="list-style-type: none"> ◆ PaO₂ = 56–59 mm Hg <i>or</i> ◆ SaO₂ of 89% <i>plus</i> <ul style="list-style-type: none"> • Hematocrit ≥ 55% • Pulmonary hypertension <i>or</i> • Edema or congestive heart failure

Table 8: Topics for Patient Education¹⁶

Stage	Criteria
0	◆ Information and advice about reducing risk factors
I-III	Above, plus ◆ Information about the nature of COPD ◆ How to use inhalers and other treatments ◆ Recognition and treatment of exacerbations ◆ Strategies for minimizing dyspnea
IV	All of the above, plus ◆ Information about complications ◆ Information about oxygen treatment ◆ Advance directives and end-of-life decisions

short hospital admissions.²⁷

Oxygen Therapy

Because COPD inevitably progresses, many patients will eventually develop severe hypoxemia and require supplemental oxygen during periods of exertion, at night, or on a continuous basis. Randomized controlled trials have shown that, for patients with chronic respiratory failure, oxygenation for at least 15 hours/day confers a survival benefit.¹⁴ Evidence suggests long-term continuous oxygen therapy prolongs survival in patients with severe hypoxemia (<60 mm Hg or SaO_2 of $<90\%$) but not in patients with moderate hypoxemia or in those with only nocturnal desaturation.²⁸

The Centers for Medicare and Medicaid Services reimburse home oxygen therapy for adults with a documented PaO_2 of ≤ 55 mm Hg or an SaO_2 of $\leq 88\%$ while breathing room air at rest.²⁸ Patients with a PaO_2 of 56–59 mm Hg or an SaO_2 of 89% may also qualify for coverage if they have comorbidities such as P-pulmonale (P waves of ≥ 3 mm in lead II, III, or aVF of the electrocardiogram), right-sided heart failure (dependent edema), or erythrocytosis (hematocrit $> 55\%$).²⁹ Table 7 summarizes the criteria.

The main modalities for oxygen delivery are compressed gas stored in cylinders, liquid oxygen held in small reservoirs,

and concentrators that extract oxygen from ambient air.³⁰

Surgery

Surgical options, including bullectomy, lung-volume-reduction surgery (LVRS), and lung transplantation, are options for select patients with advanced COPD. Bullectomy may improve lung function and reduce shortness of breath, LVRS may increase exercise tolerance in upper-lobe emphysema and overall health status in heterogenous emphysema and transplantation may enhance quality of life and function in stage IV COPD.¹⁴ However, rejection with bronchiolitis obliterans is a potential complication of lung transplantation, and survival benefits are not clearly better than those achieved with pulmonary rehabilitation.²⁹ None of these procedures are recommended for widespread use.¹⁴

Exercise 4

A 63-year-old woman has had COPD since 1984. On her most recent office visit, her oxygen saturation was 86% at rest while breathing room air. Would this patient benefit from home oxygen therapy?

- a. Yes
- b. No

Answer on page 26.

Other Treatment Considerations

Managing Comorbidities

Individuals with COPD often have comorbidities such as sleep apnea, nutritional alterations leading to muscle wasting or even obesity, depression, gastroesophageal reflux disease, and osteoporosis, among other conditions.¹³ [For additional discussion on comorbidities, see Course AB0494, *COPD: Treating the Whole Patient*, on the companion CD.]

Patient Education

The 2005 GOLD guidelines state, “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.”³ Table 8 shows topics to address with patients at every stage of COPD.¹⁶

Exercise 5

A 45-year-old man presents with a new onset of thick sputum production and worsening episodes of shortness of breath. He has no history of smoking or diagnosed pulmonary disease. Spirometry is not indicated because of the patient’s young age and noncontributory history.

- a. True
- b. False

Answer on page 26.

Conclusion

A variety of therapeutic tools are available to optimize and individualize the care of patients with symptomatic stable COPD.¹³ Different strategies should be implemented depending on the stage of COPD and on changes in the patients’ condition over time. An understanding of the progressive nature of COPD, early recognition of disease onset and acute exacerbations and thorough knowledge of the current pharmacologic and nonpharmacologic options will offer patients the best chance for prolonged survival with preserved quality of life and functional capacity. ◆