

Breathing new life into

COPD management



The Global Initiative for Chronic Obstructive Lung Disease (GOLD) promotes awareness, education, and care for patients with chronic obstructive pulmonary disease (COPD). The committee annually reviews evidence-based guidelines for prevention, diagnosis, and treatment of COPD. We give you an overview of these guidelines.

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GOLD is a consortium of international experts whose objective is to improve worldwide awareness, education, and care for COPD patients. Formed in 1997 in collaboration with the World Health Organization (WHO) and the National Heart, Lung and Blood Institute, the consortium published its initial report in 2001, following a comprehensive review of existing guidelines for COPD.

After reviewing the world's literature each year, committee members continue to develop evidence-based guidelines for preventing, diagnosing, and treating COPD. Annual updates are available online at <http://www.goldcopd.org>. In this article, we give you an overview of these important guidelines to promote their use in the clinical setting.

Coming to terms

The term COPD doesn't describe one disease process; rather, it encompasses pathology from different disease states that ultimately produce chronic and irreversible limitations in airflow. The GOLD report

defines COPD as a preventable and treatable disease characterized by airflow limitation that also has some extrapulmonary (outside of the lung) effects that may contribute to other comorbidities in certain patients. The airflow limitation is progressive in nature and is associated with an abnormal lung inflammatory response.

Chronic bronchitis and emphysema have long been identified as the two categories beneath the umbrella term COPD. Interestingly, the 2010 GOLD report includes neither of these disease entities in its definition of COPD. Bronchitis isn't always associated with airflow obstruction, and the alveolar destruction that's indicative of emphysema is only one of multiple lung abnormalities present in COPD. However, the 2010 report does describe the characteristic airflow limitation of COPD developing from small airway disease (obstructive bronchiolitis) and lung tissue destruction (emphysema).

Chronic bronchitis is diagnosed by the presence of a cough with sputum production for 3 months a year for 2 consecutive

years. Although this is a disease of the small airways, its definition doesn't include reference to airflow limitation, and many patients who don't have a chronic cough and sputum production may develop airflow limitation. However, the presence of a cough and sputum production doesn't exclude a diagnosis of COPD.

Obstructive bronchiolitis involves narrowing of the small airways that may result from various pathologic states. This broader term better reflects the airflow limitation of COPD as defined by the GOLD report.

Emphysema is associated with alveolar destruction, which reduces the surface area available for gas exchange. This structural change decreases elastic recoil—the ease with which the lung

relaxes during expiration—and results in alveolar hyperinflation and air trapping. There are two commonly recognized types of emphysema: centriacinar or centrilobular, and panacinar (see *Lung changes in emphysema*).

Deep impact

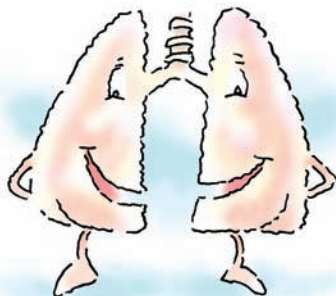
Although 16 million patients in the United States may already be diagnosed with COPD, another 14 million may be undiagnosed. In the United States, COPD accounted for 1.5 million emergency visits and more than 725,000 hospitalizations in 2000, and more than \$32 billion in costs in 2002. According to the WHO, the incidence of COPD is on the rise. Currently ranked as the fourth leading cause of morbidity and mortality in the United States, COPD is predicted to become number three by the year 2020.

Not only does this chronic disease present a significant health burden, it also has costly economic and social ramifications. The direct healthcare costs to treat, diagnose, and manage the disease are burdensome not only to patients but also to the U.S. healthcare system. This debilitating disease prevents many patients from earning a living, leading them to rely on family, other caregivers, and indirectly on society as a whole for financial support.

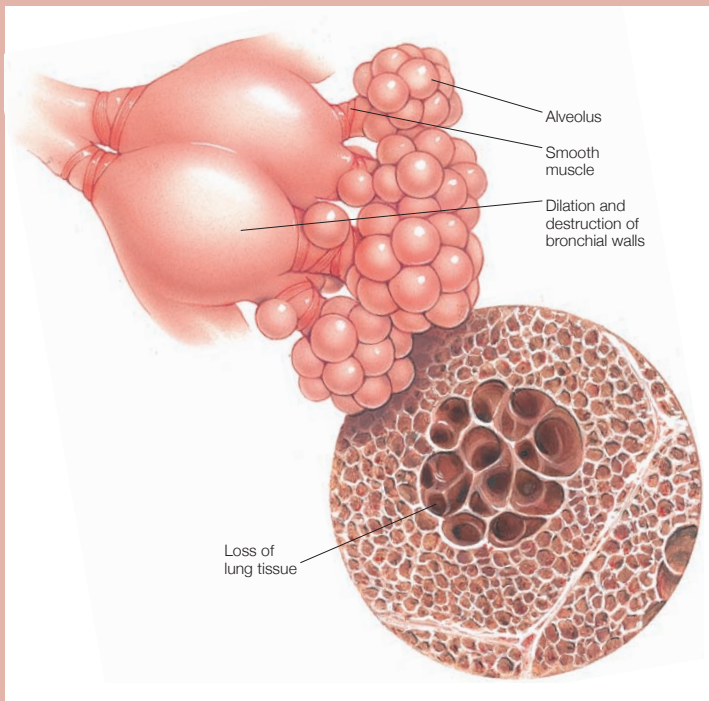
Structural changes

Exposure to irritating, inhaled substances such as cigarette smoke normally causes an inflammatory response in the airways and lungs. In COPD, an exaggerated response to noxious stimuli disrupts the body's normal defense mechanisms. This abnormal response results in chronic inflammation and structural changes in the trachea and bronchi, bronchioles, respiratory bronchioles and alveoli, and the pulmonary vessels.

The airways narrow due to the body's attempts to adapt to injurious stimuli and chronic inflammatory changes. The inflammatory response causes an increase in the number of macrophages and CD8+



Lung changes in emphysema



Source: *Pathophysiology Made Incredibly Visual!* Philadelphia, PA: Lippincott Williams & Wilkins; 2008:52.

lymphocytes, which combine to destroy tissue throughout the lungs. In the trachea and bronchi, an increase in goblet cells and enlarged submucosal glands increase mucus secretion and further narrow the airways. Bronchioles narrow from thickening of airway walls and from increased exudate.

Chronic airway inflammation may also lead to pulmonary vasoconstriction secondary to chronic hypoxemia. This leads to pulmonary hypertension, which in turn may cause right-sided heart failure (cor pulmonale).

Continued exposure to noxious irritants and inflammatory changes cause fibrotic changes in the small airways, obstructing airflow during expiration. As the alveoli become further damaged, hyperinflation results from loss of elasticity. As COPD progresses, alveolar hyperinflation makes inspiration more difficult, reduces gas exchange, and leads to reduced oxygen uptake and carbon dioxide retention.

Where there's smoke, there's fire

One of your important roles in the prevention and treatment of COPD is to identify patient risk factors for this disease. Risk factors include genetics and environmental exposures.

Of every two people with the same smoking history, one may develop COPD; of every six people diagnosed with COPD, one has never smoked. A genetic predisposition is thought to be the reason these nonsmokers develop COPD.

Genetic risk factors for COPD aren't clearly understood and require further research, but the most studied and documented genetic risk factor is alpha₁ antitrypsin deficiency. Alpha₁ antitrypsin is an enzyme produced mostly in the liver that primarily protects the lungs from injury. A deficiency of this enzyme predisposes adults to the early development of emphysema.

Risk factors for developing COPD

- Genetic factors
- Exposure to particles
- Tobacco smoke
- Occupational dusts (organic and inorganic)
- Indoor air pollution from heating and cooking with biomass fuels such as wood and dung in poorly vented dwellings
- Outdoor air pollution
- Lung growth and development problems
- Oxidative stress
- Respiratory infections
- Lower socioeconomic status
- Poor nutrition
- Comorbidities

Source: Bauldoff GS. When breathing is a burden: how to help patients with COPD. *Am Nurse Today*. 2009;4(9):17-23.

An estimated 100,000 people in the United States have this enzyme deficiency, but only about 10% of them have been diagnosed. Although both smokers and nonsmokers with the deficiency experience a decline in lung function, smokers have an increased risk of rapid disease progression.

Cigarette smoking is by far the most common cause for the development and progression of COPD. In addition, exposure to secondhand smoke (passive smoking) increases the risk of COPD in nonsmokers. Exposure to cigarette smoke initiates the inflammatory cascade in the airways and lungs, and continued exposure to noxious irritants may result in obstructive airflow limitation.

Carbon monoxide, a byproduct of cigarette smoking, adds to the problem. In the body, carbon monoxide combines with hemoglobin to form carboxyhemoglobin. These hemoglobin molecules are inefficient carriers of oxygen to the body's tissues and only worsen hypoxemia.

Other environmental risk factors include exposure to occupational dusts and chemicals, and indoor and outdoor air pollution. An estimated 19% of those

with COPD develop the disease secondary to occupational exposure, and 31% of them are nonsmokers.

The GOLD report cites several other risk factors that have been identified through research, including respiratory injuries during gestation and childhood that interfere with normal lung growth and development, asthma, lower socioeconomic status, viral and bacterial infections, and poor nutrition (see *Risk factors for developing COPD*).

Whether gender is a risk factor is unclear. Historically, COPD has been more prevalent in men than in women, but recent studies reveal that women have almost achieved equality with men in terms of COPD. Causes for this rise are thought to be related to women having smaller airways and more women smoking cigarettes over the past few decades.

GOLD star assessment

If you suspect your patient has COPD, obtain a thorough health history and physical assessment. The health history should include risk factors, pattern of

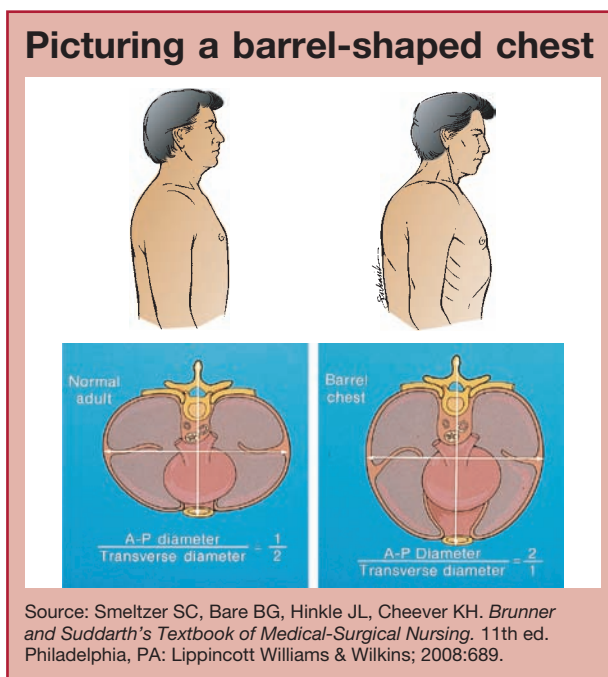
signs and symptoms (progressive, persistent dyspnea; chronic cough; and sputum production), and the presence of comorbidities. Possible physical assessment findings include decreased breath sounds, a barrel-shaped chest, and pursed-lip breathing (see *Picturing a barrel-shaped chest*).

The healthcare provider will confirm a diagnosis of COPD with spirometry. Considered the gold standard for diagnosing and monitoring disease progression, spirometry is the most accurate and objective means to measure airflow limitation. It also helps to differentiate COPD from other pulmonary diseases.

Spirometry includes measurement of **forced vital capacity (FVC)**, the maximal amount of air that can be rapidly and forcefully exhaled from the lungs after maximal inspiration, and **forced expired volume achieved in 1 second (FEV₁)**, the volume of air expired in the first second of FVC. The ratio of FEV₁/FVC is then calculated. A normal FEV₁/FVC ratio is greater than or equal to 70% of the predicted value based on height, age, and gender. A calculated ratio of less than 70% confirms airflow obstruction. COPD severity is classified as stages I through IV based on spirometry measurements (see *Using GOLD staging for COPD*).

Smoking out the dangers of COPD

Annually, smoking is responsible for more than 435,000 deaths in the United States, with tobacco-related disease accounting for \$96 billion in medical expenses. Approximately 20% of American adults currently smoke, and 4,000 children and adolescents will smoke their first cigarette today. Smoking cessation is the single most cost-effective strategy to reduce the risk of developing COPD or to slow its progression. More than 70% of current smokers have a desire to stop smoking. Take advantage of an opportunity to help them achieve this goal.



Dependence on tobacco is both a chronic disease and an addiction. Stopping tobacco use is difficult and relapse is common, mainly because nicotine is highly addictive. To prevent or minimize the development of chronic disease, be proactive in assessing, educating, and intervening to help stop your patient's tobacco use. Be sure to consistently identify and document the smoking habits of each patient. Determine the age at which the patient began smoking, current smoking status, and the desire to stop smoking. Inform the patient about effective treatment options for smoking prevention and cessation.

The U.S. Public Health Service has compiled guidelines for smoking cessation entitled *Treating Tobacco Use and Dependence: 2008 Update. Quick Reference Guide for Clinicians*. These guidelines outline a five-step approach known as the 5 A's for clinicians to help their patients stop smoking (see *When smokers want to quit, use the 5 A's*). Use the guidelines with every patient encounter to identify tobacco users and encourage them to quit. Once a patient has expressed the desire to quit smoking, discuss the preparatory STAR quit plan with the patient (see *The STAR quit plan for smoking cessation*).

Also included in the guidelines are specific interventions, known as the 5 R's, which the clinician can use to educate and enhance motivation to stop smoking for smokers who don't want to quit (see *When smokers don't want to quit, use the 5 R's*).

Pharmacologic treatment

The goals of effective COPD management are to relieve signs and symptoms, prevent disease progression, improve exercise tolerance and health status, prevent and treat complications and exacerbations, and reduce mortality. Reaching the goals requires an individualized approach for every patient. To maximize your patient's quality of life, weigh the benefits against the risks of

Using GOLD staging for COPD

	Stage I: mild COPD	Stage II: moderate COPD	Stage III: severe COPD	Stage IV: very severe COPD
FEV ₁ /FVC	<70%	<70%	<70%	<70%
FEV ₁	≥80% of predicted	50%–79% of predicted	30%–49% of predicted	<30% of predicted or <50% of predicted AND chronic respiratory failure

Source: GOLD. Pocket guide to COPD diagnosis, management, and prevention. <http://www.goldcopd.com/Guidelineitem.asp?11=2&l2=1&intId=1116>.

pharmacologic and nonpharmacologic therapies for each patient.

Pharmacologic therapy for COPD focuses on controlling signs and symptoms and decreasing complications. Bronchodilators, a mainstay for symptom management, include beta₂-agonists, anticholinergics, and methylxanthines. The medications are typically prescribed for patients in this order, escalating the number of medications used for treatment as needed, based on signs and symptoms, presence of comorbidities, and the patient's general health.

Short- and long-acting bronchodilators manage signs and symptoms and prevent exacerbations. Short-acting or rescue inhalers are used as needed for the immediate relief of worsening symptoms. Salbutamol is an example of a short-acting **beta₂-agonist** with a 4- to 6-hour duration of action.

Long-acting bronchodilators are prescribed for patients who must use medications on a regular schedule to control their signs and symptoms. These are more effective and convenient than short-acting bronchodilators used frequently. Salmeterol, a long-acting bronchodilator, has a duration of action of 12 hours or longer. The preferred route of administration is via inhalation to maximize medication effectiveness and minimize systemic adverse reactions. To promote optimal therapy, teach your patient to correctly use inhalers, then reinforce your

teaching. Be sure to present education consistently as appropriate for the individual.

Regardless of a beta₂-agonist's duration of action, the mechanism of action is to relax smooth muscles in the airway. The resulting bronchodilation promotes expiratory flow and improves exhalation. This subsequently decreases hyperinflation and improves symptoms both at rest and with exercise.

The predominant adverse reactions to beta₂-agonists are sinus tachycardia, which may cause more serious cardiac dysrhythmias in some patients, and exaggerated tremors, a particular risk in older adults taking high doses. Several of the short-acting beta₂-agonists also come in oral preparations, but their onset of action is much slower and they're associated with more systemic adverse reactions than the inhaled formulations.

Inhaled **anticholinergics** facilitate bronchodilation by blocking acetylcholine receptor sites and preventing bronchoconstriction.

Ipratropium is a short-acting anticholinergic, with effects lasting up to 8 hours. Long-acting tiotropium has a duration of action greater than 24 hours, making it an appropriate choice for once-daily dosing. Treatment with a long-acting anticholinergic such as tiotropium helps to produce clinically significant improvement in lung

function, reduce COPD exacerbations, and improve the effectiveness of pulmonary rehabilitation.

The main adverse reaction to anticholinergics is dry mouth. Some patients taking ipratropium report a bitter metallic taste.

Methylxanthines inhibit phosphodiesterase, which increases cyclic adenosine monophosphate, relaxes bronchial smooth muscle, and promotes bronchodilation. Theophylline, the best-known drug in this class, was once a mainstay of COPD management. Today it's prescribed much less frequently because of its narrow therapeutic window, which outweighs its potential benefits for many patients. However, theophylline is still sometimes prescribed for additional symptom control.

Methylxanthines are given via the oral and I.V. route, and the most common adverse reactions are headache, insomnia, nausea, and heartburn. Atrial and ventricular dysrhythmias and seizures are possible signs of toxicity.

Combination therapy using bronchodilators with varied mechanisms and durations of action may improve symptom control. But combination therapy also increases costs, requires a higher level of adherence to the prescribed therapy, and may be no better than using a higher dose of a single medication—as long as adverse reactions aren't an issue. Treatment is individualized to each patient's condition and response.

Glucocorticosteroids are commonly used for their anti-inflammatory effects, but according to the GOLD report, their role in managing COPD is limited. Patients benefiting the most are those with advanced stages of COPD—stages III and IV—because they tend to have more severe signs and symptoms and more frequent exacerbations. The regular use of inhaled glucocorticosteroids reduces the frequency of exacerbations.

Using an inhaled glucocorticosteroid in combination with an inhaled long-acting beta₂-agonist is more effective than using



The STAR quit plan for smoking cessation

Set a quit date within 2 weeks of the decision to stop smoking.

Tell family, friends, and coworkers about the decision to quit and enlist their support.

Anticipate challenges to quitting, especially during the first few weeks.

Remove all tobacco products from your environment (work, home, and car).

Source: Adapted from Fiore MC, Jaén CR, Baker TB, et al. *Treating Tobacco Use and Dependence: 2008 Update. Quick Reference Guide for Clinicians*. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service; 2009.

either of them alone to reduce exacerbations and slow the decline in lung function.

Inhaled glucocorticosteroids may increase the risk of pneumonia, particularly when combined with a long-acting beta₂-agonist. The use of oral glucocorticosteroids hasn't proved to be effective as either a short- or long-term treatment for patients with COPD. Furthermore, chronic treatment with oral systemic glucocorticosteroids may induce a steroid myopathy. This increase in muscle weakness may contribute to respiratory failure in patients with end-stage COPD, who are already suffering from a decline in overall physical functioning. For these reasons, the chronic use of systemic steroids should be avoided.

Minimizing infection risk

Patients diagnosed with COPD should receive a yearly seasonal influenza vaccine. This has proved to reduce illness and death by 50%. Patients age 65 or older should also receive the pneumococcal vaccine to reduce the risk of community-acquired pneumonia.

Routine prophylaxis with other medications sometimes prescribed for patients with COPD isn't indicated. The GOLD report recommends that antibiotic use be reserved for COPD exacerbations in the presence of a bacterial infection, and doesn't promote the use of mucolytics and cough suppressants in the management of stable COPD.

Nonpharmacologic treatment

Pulmonary rehabilitation involves a multidisciplinary team approach that includes assessment, exercise training, nutrition counseling, smoking cessation, and education for the patient. Many clinical trials have shown that participating in a pulmonary rehabilitation program reduces dyspnea and fatigue and improves exercise tolerance and quality of life for patients with COPD. Patients with COPD at all disease stages benefit from pulmonary rehabilitation programs lasting at

When smokers want to quit, use the 5 A's

1. **ASK:** Ask about tobacco use at every new encounter.
2. **ADVISE:** Urge every tobacco user to quit.
3. **ASSESS:** Determine willingness to quit.
4. **ASSIST:** Develop a quit plan.
5. **ARRANGE:** Follow up.

Source: Adapted from Fiore MC, Jaén CR, Baker TB, et al. *Treating Tobacco Use and Dependence: 2008 Update. Quick Reference Guide for Clinicians*. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service; 2009.

When smokers don't want to quit, use the 5 R's

1. **RELEVANCE:** Ask your patient why quitting is personally relevant (such as family or health concerns).
2. **RISKS:** Ask your patient to identify risks of tobacco use (such as shortness of breath or lung cancer).
3. **REWARDS:** Ask your patient to identify rewards or benefits of quitting smoking (such as improving health and saving money).
4. **ROADBLOCKS:** Ask your patient to identify barriers or roadblocks to quitting smoking (such as weight gain or losing the enjoyment of smoking).
5. **REPETITION:** Repeat this process with every new encounter with your patient.

Source: Adapted from Fiore MC, Jaén CR, Baker TB, et al. *Treating Tobacco Use and Dependence: 2008 Update. Quick Reference Guide for Clinicians*. Rockville, MD: U.S. Department of Health and Human Services, Public Health Service; 2009.

least 6 weeks; the longer the program, the better the results.

Oxygen therapy is usually reserved for patients with end-stage (stage IV) COPD and can be used as long-term continuous therapy, during activity, and to relieve shortness of breath. The goal of oxygen therapy is to achieve an SaO₂ of 90% or greater. The prescription for oxygen must specify liters/minute, method of delivery, and duration at rest, during activity, and while sleeping. Long-term administration of oxygen in patients with end-stage COPD and respiratory failure has demonstrated prolonged survival. However, like other COPD treatments, it doesn't reverse the disease process.

Surgical treatment may be an option for select patients who have COPD. Bullae (the enlarged alveolar air spaces that don't

COPD can't be cured, but with your help your patient can have a better quality of life.



contribute to gas exchange and compress functional lung tissue) may be excised with a **bullectomy**. This surgery reduces dyspnea and improves lung function by allowing reexpansion of the compressed lung region.

Lung volume reduction surgery removes part of the lung that's nonfunctional due to hyperinflation, reducing overcrowding of the chest cavity and improving elastic recoil of functional lung tissue. This surgery has improved patient survival by more than 50 months compared with medical treatment.

Lung transplantation is another surgical option for some patients with advanced COPD, but this therapy is limited by a shortage of donor organs. Maintaining immunosuppression is also very costly and associated with serious risks, including infections and malignancies. Although data show that lung transplantation doesn't improve longevity, it may improve the quality of life for select groups of patients with end-stage COPD.

The potential benefits of any surgery must be weighed against the risks, based on the patient's existing pulmonary status, comorbidities such as obesity or diabetes, and overall health.

Managing exacerbations

The GOLD report defines an exacerbation of COPD as "an event in the natural course of the disease characterized by a change in the patient's baseline dyspnea, cough, or sputum that's beyond normal day-to-day variations, is acute in onset, and may warrant a change in regular medication in a patient with underlying COPD." All patients with COPD, regardless of the stage, are at risk for an exacerbation. Triggers for an exacerbation include respiratory infection and air pollution. Approximately one-third of exacerbations are from an unknown cause.

When a patient is admitted with a COPD exacerbation, the first-line treatment

includes providing oxygen and determining if the patient requires intensive care. If admission to an ICU is warranted, the need for invasive versus noninvasive mechanical ventilation and hemodynamic stability must be quickly evaluated, followed by appropriate treatment.

To determine adequate oxygenation and ventilation, obtain arterial blood gases 30 minutes after initiating oxygen therapy. Start bronchodilator therapy with a short-acting inhaled beta₂-agonist as prescribed. In addition, glucocorticosteroids are recommended for 7 to 10 days. As prescribed, administer antibiotics to patients who present with one of the following:

- the three cardinal signs of COPD: increased dyspnea, increased sputum production, and increased sputum purulence
- two of the cardinal signs, with one of them being increased sputum purulence
- the need for invasive or noninvasive mechanical ventilation.

Evaluate your patient for pulmonary embolism, which must be ruled out with every COPD exacerbation that requires hospitalization.

Following the acute stage of exacerbation, educate your patient about preventing future exacerbations as part of discharge planning and teaching. Review smoking cessation, medication administration, inhaler technique, and recognition and management of worsening COPD symptoms. Initiating or continuing a pulmonary rehabilitation program is also highly recommended.

Because pharmacotherapy reduces the frequency of exacerbations and hospitalizations, pay special attention to instructing your patient about taking medications as prescribed to control COPD symptoms. Explain the importance of regular follow-up with the healthcare provider.

Quality of life is our specialty

The prevalence of COPD is increasing, but thanks to improved management strategies, many patients are surviving longer with a

better quality of life. Keep your knowledge and skills up to date by reviewing ever-evolving evidence-based best practices. By investing your time and educating yourself, you can have a greater impact on the quality of life for patients who must cope with COPD for the rest of their lives. ■

Learn more about it

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