Managing Stable COPD: An Evidence-Based Approach

Applying the current GOLD recommendations to nursing practice.

ABSTRACT: Chronic obstructive pulmonary disease (COPD) affects as many as 16 million Americans and is expected to be the third leading cause of death worldwide by 2020. To increase awareness of COPD, encourage related research, and improve care of patients with this chronic disease, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) was launched in 1998 and published an evidence-based report on COPD prevention and management strategies in 2001 that has been revised regularly. The fourth major revision, which was published in 2017 and revised in 2018, includes significant changes related to COPD classification, as well as to pharmacologic, nonpharmacologic, and comorbidity management. The authors discuss the changes to the GOLD recommendations and, using a patient scenario, explain their application to clinical practice.

Keywords: chronic obstructive pulmonary disease, COPD, Global Initiative for Chronic Obstructive Lung Disease, management

In the United States, chronic obstructive pulmonary disease (COPD) affects nearly 16 million people and was the third leading cause of death in 2014.[1] Globally, COPD is the fourth leading cause of death, though it is expected to be third by the year 2020.[2]

COPD, which is usually caused by significant exposure to noxious stimuli, is characterized by limited airflow, accompanied by dyspnea, cough, mucus production, inflammation, and periods of worsening symptoms, called exacerbations, which trap gas in the lungs.[2] The chronic inflammation of COPD destroys tissue in the lung parenchyma (resulting in emphysema), constricts the small airways (causing obstructive bronchiolitis), and reduces lung elastic recoil (see Figure 1).[2] Patients with COPD may have chronic bronchitis according to some definitions of the disease, but the majority do not have cough and sputum production that persists for at least three months in each of two consecutive years, as the term *chronic bronchitis* is often clinically defined.[2]

Although COPD, like asthma, is an obstructive lung disease, the pathophysiology underlying the two conditions differs significantly. In COPD, cytotoxic $CD8^+$ T cells and inflammatory cells, such as neutrophils, macrophages, and eosinophils, are predominant.[3] By contrast, in asthma, mast cells and their mediators play a primary role.

To increase awareness of COPD, encourage related research, and improve care by disseminating evidence-based COPD prevention and management strategies, the Global Initiative for Chronic Obstructive Lung Disease (GOLD) was launched in 1998 as a collaboration between the National Heart, Lung, and Blood

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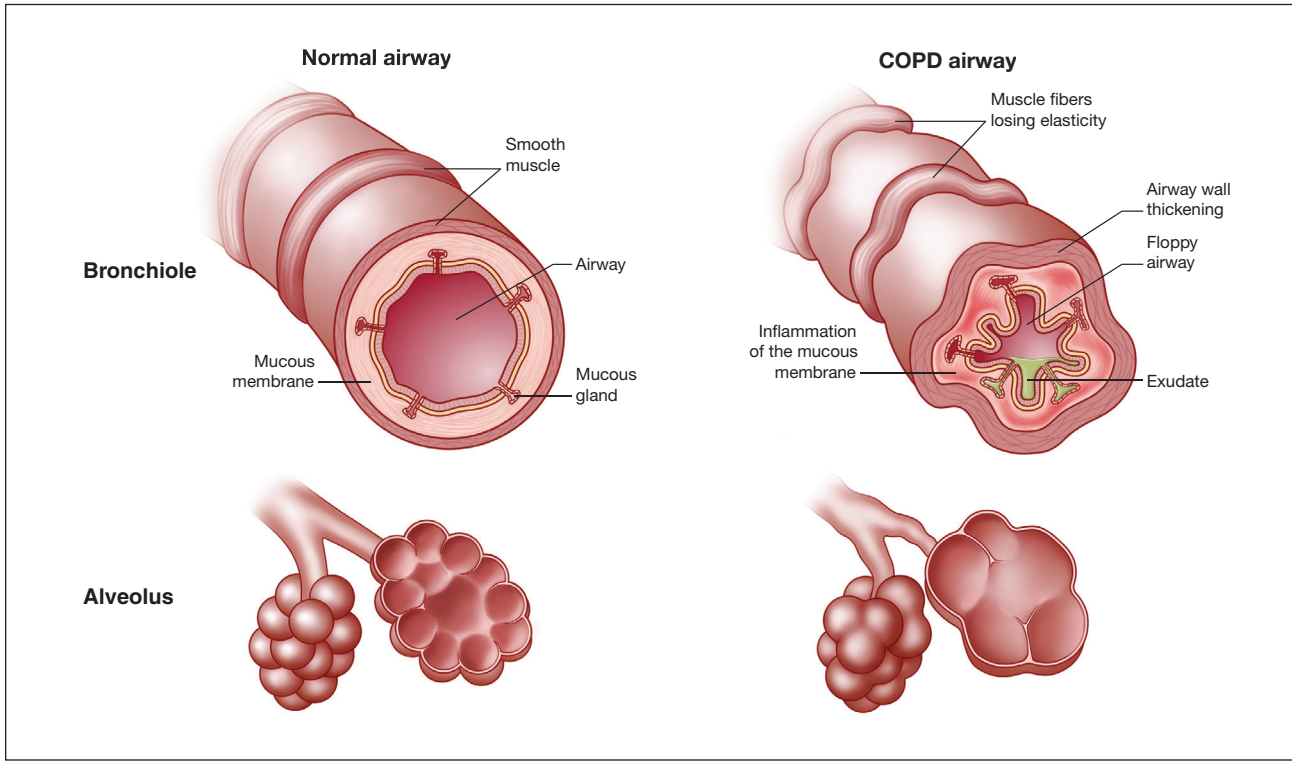
Figure 1. The Normal vs. the COPD Airway



In chronic obstructive pulmonary disease (COPD), the small airways become constricted. The bronchiole wall becomes thickened and inflamed, and exudate and mucus accumulate in the lumen. Alveolar membranes break down, further impairing ventilation. Illustration by Sara Jarret.

Institute, National Institutes of Health, and World Health Organization.[2] The first report was issued in 2001, with revisions published in 2006 and 2011; since 2011, the report has been revised annually. The fourth and most recent major revision was released in 2017, and included significant changes related to COPD classification, as well as to pharmacologic, nonpharmacologic, and comorbidity management (see Table 1[2,4,5]). Minor revisions appear in the 2018 report.

This article describes COPD risk factors and clinical manifestations, diagnostic testing, and the refined GOLD assessment tool used to classify COPD. It explains how COPD classification guides pharmacologic and nonpharmacologic management, and illustrates, using a patient scenario, how the revised GOLD recommendations can be put into practice. This article focuses on managing stable COPD, rather than COPD exacerbations.

COPD RISK FACTORS AND CLINICAL MANIFESTATIONS

A variety of risk factors are associated with COPD, though tobacco smoking is the most common. Additional risk factors include exposure to noxious particles or gases, including biomass fuels; occupational exposure to dust, chemicals, and fumes; female sex; and advanced age.[2,6-8] A genetic risk factor, alpha-1 antitrypsin deficiency (AATD), which is associated with the inability to adequately synthesize the protein alpha-1 antitrypsin, is more prevalent among white patients. Patients with AATD tend to have an atypical risk profile in that they are often nonsmokers.[9] Other important risk factors for COPD include airway hyperresponsiveness, family history of asthma, and frequent childhood respiratory infections.[2,10] Low socioeconomic status has been identified as a risk factor as well, but that may be related to pollutant exposure, poor nutrition, residential crowding, or other factors associated with poverty.[2]

Clinical manifestations of COPD include the following[2]:

- progressive dyspnea that becomes worse with exercise
- chronic cough, which may be intermittent or nonproductive

- recurrent wheeze
- chronic sputum production

DIAGNOSTIC TESTING AND COPD CLASSIFICATIONS

COPD may be suspected if a patient has any known COPD risk factors and clinical manifestations. Peak expiratory flow measures have weak specificity and cannot be used to diagnose COPD; rather, diagnosis requires a thorough medical history of risk factors and symptoms, as well as spirometric testing.[2]

Spirometric classification. Spirometry measures the forced expiratory volume in the first second (FEV_1) of exhalation and the forced vital capacity (FVC)—the total volume exhaled. Spirometry measures are obtained both before and after a bronchodilator is administered. If the postbronchodilator ratio of FEV_1 to FVC is less than 70%, it is consistent with irreversible obstructive lung disease and indicative of COPD, though patients whose FEV_1/FVC falls between 60% and 80% should be retested for confirmation on a separate occasion.[2] If a patient meets the diagnostic criteria for COPD, the World Health Organization recommends ordering an AATD screening, and the GOLD report supports that recommendation.[2] AATD screens below 20% of predicted normal value suggest that the COPD has a genetic component and that all family members should be screened.[2]

The GOLD report creates four classifications of COPD based on the percentage of FEV_1 of predicted normal value[2]:

- GOLD 1 is a FEV_1 at or above 80% of predicted normal value
- GOLD 2 is a FEV_1 of 50% to 79% of predicted normal value
- GOLD 3 is a FEV_1 of 30% to 49% of predicted normal value
- GOLD 4 is a FEV_1 below 30% of predicted normal value

Exacerbation risk and symptoms. A further classification based on exacerbation risk and symptoms is used to determine optimal management strategies after COPD diagnosis.[2] Patients who within the past year have had two or more exacerbations, or one exacerbation requiring hospital admission, are at high risk for further exacerbations.[2] Patients who within the past year have had no exacerbations, or only one that did not require hospital admission, are at low risk for exacerbation.[2]

Symptoms and symptom burden can be assessed with either the COPD Assessment Test (CAT)[11] or the modified British Medical Research Council Questionnaire (mMRC).[12] The GOLD report, however, supports the CAT as a more robust assessment.[2] The CAT measures symptom burden on a Likert-type scale, with scores ranging from 0 to 40. The higher the score, the more symptoms the patient has. For classification purposes, a CAT score of less than 10 is considered a low symptom burden and a score of 10 or higher a high symptom burden.[2] The mMRC measures the patient's level of dyspnea using grades 0 to 4, with higher grades indicating a greater level.[12] For classification purposes, an mMRC score of 0 to 1 is interpreted as a low symptom burden and an mMRC score of 2 or higher as a high symptom burden.[2] The GOLD report suggests that either test can be used along with the patient's exacerbation history to classify a patient into Group A, B, C, or D (see Figure 2) and that patients should be reclassified during annual examinations and with any change in lung function.[2]

PREVENTIVE THERAPY

Smoking cessation is the most influential factor in slowing COPD progression.[2] Providers can use the "5 A's" model as a framework to empower patients to quit smoking.[2, 13, 14] This model identifies the five major interventional steps as follows:

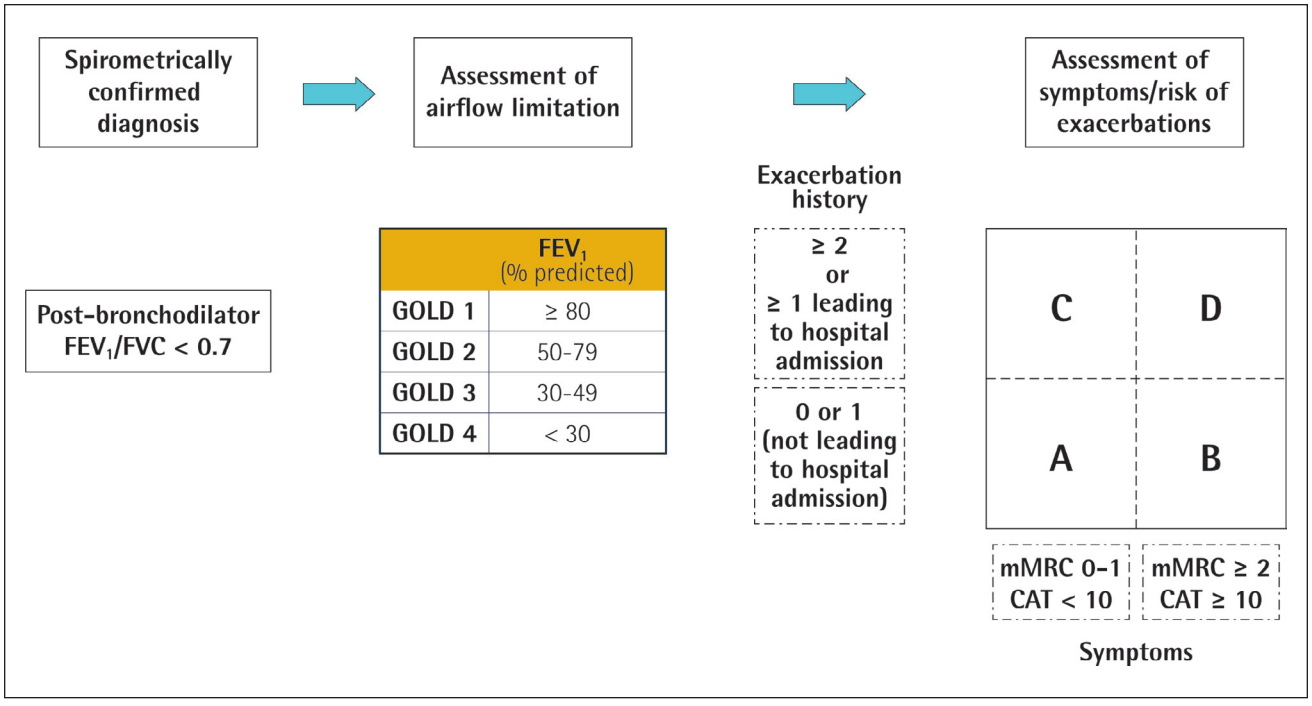
- ask—identify tobacco users and document status
- advise—urge the patient to quit
- assess—determine the patient's desire to quit
- assist—provide resources, such as cessation programs and information about pharmacologic aids
- arrange—schedule follow-up contact

Table 1. Significant Changes in the GOLD Report from 2011 to 2017[2, 4, 5]

Major Changes	2011 Report	2017 Report
Classification process	Group classification included spirometry, symptoms, and exacerbations	Stepwise classification separates spirometry from symptoms and exacerbation risk
Pharmacologic management	Outlined stepwise increase in medication	Discusses pharmacologic escalation and deescalation strategies
Nonpharmacologic management	Reviewed nonpharmacologic management	Offers recommendations for nonpharmacologic management
Comorbidity management	Reviewed evidence on comorbidities	Offers in-depth evidence-based recommendations for managing COPD comorbidities

COPD = chronic obstructive pulmonary disease; GOLD = Global Initiative for Chronic Obstructive Lung Disease.

Figure 2. The GOLD Refined ABCD Assessment Tool



The refined GOLD ABCD assessment tool guides clinicians in using spirometry, exacerbation history, and symptom scores from either the mMRC or the CAT to classify patients into Group A, B, C, or D. For example, at diagnosis, 53-year-old Beatrice Johnson, the subject of our patient scenario, has a postbronchodilator FEV_1/FVC below 70%, and her FEV_1 is 62% of predicted normal value. She has a CAT score of 18. Since she has had no exacerbations within the past year, she is categorized as COPD GOLD 2 Group B. If, however, within the past year she'd had an exacerbation that required hospitalization, she would be categorized as GOLD 2 Group D, even if all other variables remained the same. This is because that history puts her at high risk for further exacerbations. CAT = COPD Assessment Test; COPD = chronic obstructive pulmonary disease; FEV_1 = forced expiratory volume in the first second; FVC = forced vital capacity; GOLD = Global Initiative for Chronic Obstructive Lung Disease; mMRC = modified British Medical Research Council Questionnaire.

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A systematic review of 42 randomized trials that assessed the effect of smoking cessation advice from health care providers on a total of more than 31,000 smokers found that such advice increased a patient's likelihood of both quitting smoking and remaining a nonsmoker 12 months later, with intensive advice (involving longer initial counseling periods, adjunctive aids, and follow-up support) resulting in slightly higher rates of cessation.[15] Nicotine replacement products or medications to reduce tobacco dependence, or combination therapy with both, may further increase the likelihood of successful smoking cessation.[14]

Vaccinations. Influenza and pneumococcal vaccinations may help prevent exacerbations by reducing the likelihood of lower respiratory tract infections. In six trials that specifically included patients with COPD, inactivated influenza vaccine significantly reduced the total number of COPD exacerbations per vaccinated patient.[16] Pneumococcal vaccinations are recommended for anyone over age 65 and for those under age 65 who have chronic heart disease, lung disease, liver disease, diabetes, or alcoholism, as well as for cigarette smokers.[17] The GOLD report recommends that patients with COPD who are under age 65 should receive the 23-valent pneumococcal polysaccharide vaccine (PPSV23) and that patients with COPD who are over age 65 should receive the 13-valent pneumococcal conjugate vaccine (PCV13).[2]

MEDICATIONS USED IN STABLE COPD

Providers need to be familiar with both the medications used to treat COPD and the various means of delivery. COPD medications are commonly administered through inhalation directly to pulmonary tissue, which reduces the potential for adverse systemic effects. With inhaled medications, it's important to verify that the

Table 2. Medications Commonly Used for Maintenance in COPD

Drug	Duration of action (hours)
β-$_2$ agonists	
Short acting	
Fenoterol	4-6
Levalbuterol	6-8
Salbutamol (albuterol)	4-6, 12 ER
Terbutaline	4-6
Long acting	
Arformoterol	12
Formoterol	12
Indacaterol	24
Olodaterol	24
Salmeterol	12
Antimuscarinics	
Short acting	
Ipratropium bromide	6-8
Oxitropium bromide	7-9
Long acting	
Aclidinium bromide	12
Glycopyrronium bromide	12-24
Tiotropium	24
Umeclidinium	24
Combination of short-acting β-$_2$ agonist and antimuscarinic	
Fenoterol/ipratropium	6-8
Salbutamol/ipratropium	6-8
Combination of long-acting β-$_2$ agonist and antimuscarinic	
Formoterol/aclidinium	12
Formoterol/glycopyrronium	12
Indacaterol/glycopyrronium	12-24
Vilanterol/umeclidinium	24
Glycopyrrolate/formoterol	12
Olodaterol/tiotropium	24
Methylxanthines	
Aminophylline	Variable, up to 24
Theophylline (ER)	Variable, up to 24
Combination of long-acting β-$_2$ agonist and corticosteroid	
Formoterol/beclomethasone	
Formoterol/budesonide	
Formoterol/mometasone	
Salmeterol/fluticasone	
Vilanterol/fluticasone furoate	
Phosphodisterase-4 inhibitor	
Roflumilast	

COPD = chronic obstructive pulmonary disease; ER = extended release.
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patient understands and can demonstrate proper delivery technique.

All types of inhalers are frequently misused.[18] A systematic review of studies evaluating the use of dry powder inhalers by patients with asthma or COPD found that most patients did not use their inhalers appropriately, frequently failing to exhale before inhalation, position or load the inhaler correctly, inhale deeply, or hold their breath after inhalation; however, proper training in technique seemed to improve efficient use, especially when training sessions were repeated and technique was evaluated at regular intervals.[19] (The website of Use-inhalers, an independent health care organization unaffiliated with any pharmaceutical company, provides free training videos and patient instruction handouts in multiple languages: https://use-inhalers.com/professional-home.)

A prospective study of 14 healthy volunteers who did not use an inhaler regularly sought to identify the effects of the most common inhalation delivery errors during use of a dry powder salbutamol (also called albuterol) inhaler.[20] Investigators found that exhalation into the device before inhalation, insufficient inspiratory flow, and missed doses caused the greatest reduction in drug delivery.

COPD management goals. COPD medications are not curative, but rather pursue the two broad management goals of (1) reducing symptoms, thereby increasing exercise tolerance and improving health status, and (2) reducing risk of exacerbations, disease progression, and death.[2] Medications that reduce COPD symptoms and risk of exacerbation include bronchodilators (β-$_2$ agonists, antimuscarinics), anti-inflammatories (inhaled glucocorticoids, oral glucocorticoids, phosphodiesterase-4 inhibitors), antibiotics, mucolytics, and alpha-1 antitrypsin augmentation therapy. (See Table 2 for a selected list of medications.)

β-$_2$ agonists relax smooth muscle airways by stimulating β-$_2$ adrenergic receptors, thereby increasing cyclic adenosine monophosphate and inhibiting bronchoconstriction. There are short-acting and long-acting β-$_2$ agonists. Short-acting β-$_2$ agonists (SABAs) have a typical duration of four to six hours, and in inhaled form may be used as a rescue medication. Long-acting β-$_2$ agonists (LABAs), which typically have a duration of 12 hours, increase lung function, reduce exacerbations, and improve quality of life, though they do not significantly reduce mortality or adverse events.[21] A systematic review of 13 randomized controlled trials that compared the once-daily LABA indacaterol with placebo or twice-daily LABAs and included a total of 9,961 participants with stable COPD found that once-daily indacaterol was as effective as twice-daily LABAs in reducing the perception of dyspnea and improving lung function.[22] With respect to quality of life, as well, no measurable differences between indacaterol and twice-daily LABAs were noted. Adverse effects associated with β-$_2$ agonists include resting sinus tachycardia, cardiac rhythm disruptions, and exaggerated somatic tremor, which occurs primarily in older patients taking higher doses and is more pronounced when the drugs are taken orally rather than by inhalation.

Antimuscarinic drugs block bronchoconstriction by inhibiting acetylcholine on muscarinic receptors, thereby relaxing smooth muscle airways. Short-acting muscarinic antagonists (SAMAs), such as ipratropium (Atrovent), have a duration of four to six hours. SAMAs block the inhibitory muscarinic 2 (M2) neuronal receptor, which causes vagally induced bronchoconstriction. A systematic review of 11 randomized controlled trials that compared the antimuscarinic ipratropium, alone or in combination with a SABA, with a SABA alone (delivered by inhaler or nebulizer) found that ipratropium provided small benefits over SABAs in nonasthmatic adults with COPD in terms of lung function and the need for oral steroids.[23] Long-acting muscarinic antagonists (LAMAs), such as tiotropium (Spiriva, Spiriva Respimat), bind to M1, M2, and M3 receptors, binding for far longer periods to M3 receptors, and producing prolonged bronchodilation compared with SAMAs. While LAMAs may do little to improve FEV_1, they can improve lung function and health-related quality of life.[24] They have also demonstrated superiority in reducing exacerbation rates when compared with LABA treatment.[25, 26]

Antimuscarinics are associated with fewer systemic adverse effects; dry mouth (xerostomia) is the most common adverse effect. The following strategies can help patients manage xerostomia:

- Take frequent, small sips of water to promote hydration.
- Use sugar-free lozenges, gum, or mints to increase saliva production.
- Avoid foods high in salt, such as nuts and crackers.
- Do not use mouthwashes that contain alcohol.
- Limit the intake of caffeinated drinks because caffeine is dehydrating.
- Use a lip moisturizer.
- Stop smoking.

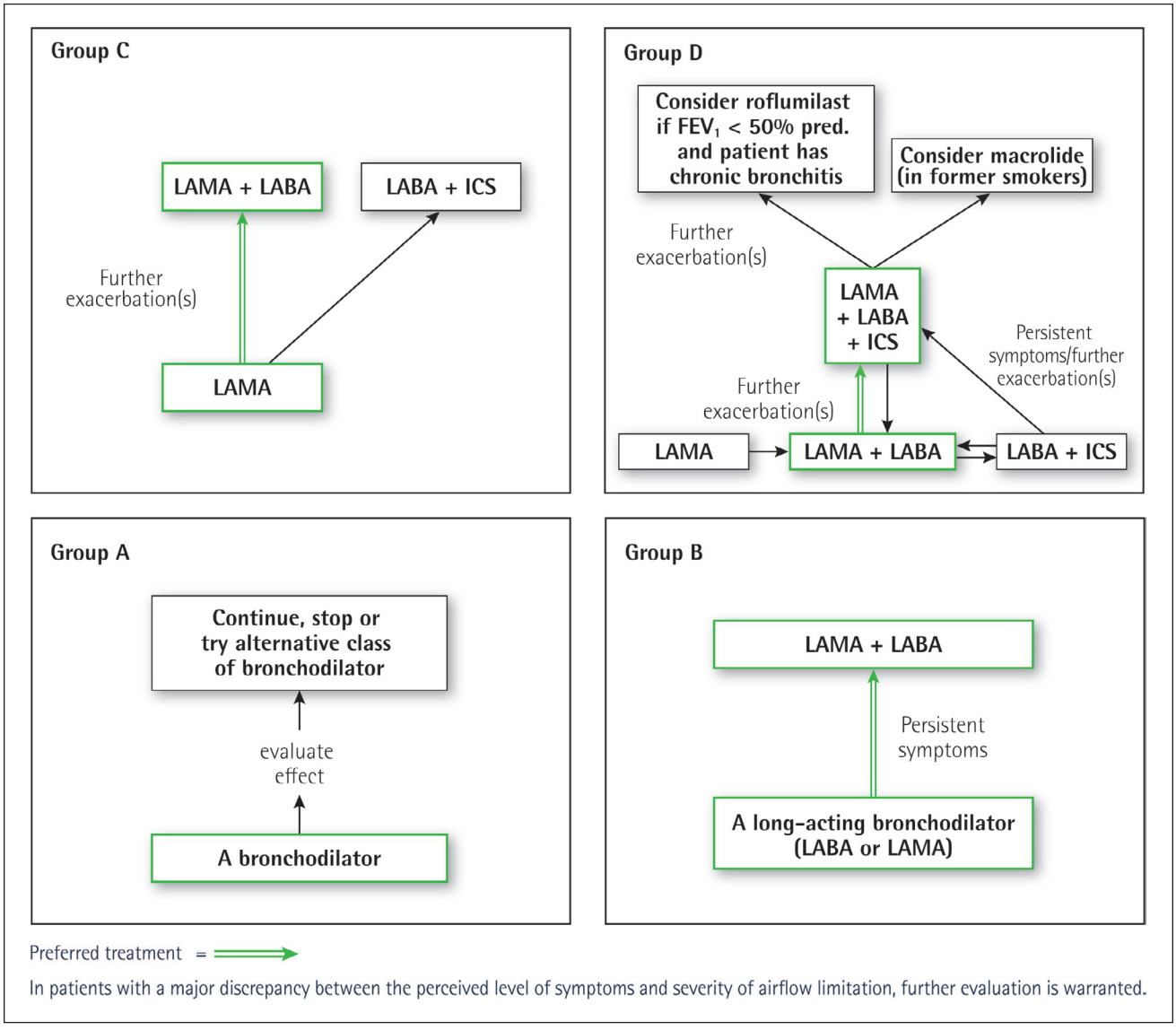
Methylxanthines, such as theophylline (Elixophyllin, Theo-24), have a modest bronchodilatory effect and may improve lung function when added to the LABA salmeterol.[27, 28] This medication, however, has a narrow therapeutic window and requires close monitoring of the patient's serum levels.

Combination SABA–SAMA inhalation therapy may increase bronchodilation by acting on different mechanisms while limiting dose-related adverse effects. Together, these therapies have been shown to improve FEV_1 response compared with either medication alone.[29]

Combination LABA–ICS vs. LABA–LAMA inhalation therapy. Inhaled corticosteroids (ICSs) should not be used as stand-alone therapy in patients with

stable COPD, and may have such adverse effects as oral candidiasis, hoarse voice, skin bruising, and pneumonia. An ICS combined with a LABA, however, is more effective at lower doses than either therapy is alone.[30] A systematic review of 15 randomized, double-blind studies involving 7,814 participants that compared LABA–ICS combination therapy with ICS therapy alone found that the number of exacerbations per study participant and the probability of death were reduced with combination treatment, and lung function and quality of life also improved.[30] However, another systematic review of 11 studies

Figure 3. The GOLD Pharmacologic Treatment Algorithm



The GOLD pharmacologic treatment algorithm aids clinicians in escalating and deescalating COPD medication based on patient classification. As indicated in Figure 2, patients are classified as either Group A, B, C, or D according to the number of exacerbations they have had in the past year and the level of their symptoms as determined by either the mMRC or the CAT score. Starting from the bottom left of the patient's group box, move along the arrow when escalating medication. Move back to previous medications when deescalating. CAT = COPD Assessment Test; COPD = chronic obstructive pulmonary disease; GOLD = Global Initiative for Chronic Obstructive Lung Disease; mMRC = modified British Medical Research Council Questionnaire.

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involving 9,839 participants found that a twice-daily regimen of a LABA–LAMA combination produced a greater improvement in FEV_1 and reduced exacerbations and risk of pneumonia, while increasing quality of life measures, when compared with a LABA–ICS.[31] LABA–LAMA therapy is, therefore, preferred over LABA–ICS.

Inhaled triple therapy consisting of a LABA, a LAMA, and an ICS may improve lung function and patient outcomes.[32] Adding a LAMA to existing LABA–ICS therapy has been shown to improve lung function in patients at high risk for exacerbation.[33] Providers should consult the refined GOLD ABCD assessment tool and pharmacologic treatment algorithm before initiating triple therapy (see Figure 3).[2] An analysis of prescribing practices in the United Kingdom suggests there may be a tendency toward premature use of triple therapy in patients assigned to GOLD Groups A, B, or C.[34]

Short-term oral glucocorticoids play a role in managing COPD exacerbations though they have numerous adverse effects, including bone fracture, epigastric disturbance, psychiatric symptoms, skin conditions, and hyperglycemia.[35]

Phosphodiesterase-4 inhibitors should be used only in patients determined to be in GOLD Group D with a FEV_1 below 50% of predicted normal value and chronic bronchitis.[2] Common adverse effects associated with these medications include nausea, vomiting, diarrhea, sleep disturbance, and headache.[36]

Continuous macrolide antibiotic therapy has been shown in some studies to reduce exacerbation rates in patients with COPD of moderate severity.[37] Studies of pulsed macrolide antibiotic therapy, on the other hand, have produced mixed results.[37, 38] Continuous antibiotic therapy may lead to bacterial resistance and should be used with caution.

Mucolytic therapy may reduce COPD exacerbation risk and modestly improve health status.[39]

Alpha-1 antitrypsin augmentation therapy, which is administered only by IV infusion, may help with the subset of COPD patients who have AATD.[9]

USING COPD CLASSIFICATION TO GUIDE TREATMENT

The refined GOLD ABCD assessment tool and pharmacologic treatment algorithm assist clinicians in adhering to evidence-based pharmacologic management strategies.

In Group A, patients may use a short-acting bronchodilator, such as a SABA or SAMA. However, regular use of short-acting bronchodilators is not recommended. The GOLD report advocates monitoring outcomes in patients using a short-acting bronchodilator, and if symptoms are not under control or patients experience adverse effects, bronchodilator treatment may need to stop or another class of bronchodilators may need to be prescribed.[2] For example, if a patient is using a short-acting bronchodilator too frequently or with poor symptom control, consider prescribing a LAMA.

In Group B, patients can start therapy with a long-acting bronchodilator, either a LAMA or a LABA. If symptoms persist, the provider may add another long-acting bronchodilator to the regimen, prescribing a LABA–LAMA combination. If symptoms decrease with LABA–LAMA combination therapy, the provider may consider returning the patient to a single bronchodilator.[2]

In Group C, patients may start with a LAMA, but if symptoms persist, a second long-acting bronchodilator, such as a LABA, may need to be added, or a LABA–ICS combination substituted. (Since ICSs elevate the risk of pneumonia, LABA–LAMA combination therapy is preferred.[2])

In Group D, the GOLD report recommends starting with LABA–LAMA combination therapy, because this group of patients has had better results with that combination than with single bronchodilator therapy, and LABA–LAMA combinations have proven superior to LABA–ICS combinations in preventing exacerbations. In addition, Group D patients are at elevated risk for pneumonia when treated with an ICS. However, in patients with a history of asthma–COPD overlap, the provider should ensure that combination therapy includes an ICS and then adjust the medication regimen to that which produces the fewest adverse effects with the greatest reduction in dyspnea. If the patient's symptoms persist without relief or the patient has more exacerbations, triple therapy with a LAMA, a LABA, and an ICS may be prescribed to control symptoms. For patients who continue to have exacerbations and a FEV_1 less than 50% of predicted normal value with chronic bronchitis, recommendations include adding the phosphodiesterase-4 inhibitor roflumilast (Daliresp) or a macrolide antibiotic to the regimen.[2]

Providers may consider escalating or deescalating a patient's COPD medication regimen as patient symptoms and exacerbation risk intensifies or abates.[2] See *How to Put the Global Initiative for Chronic Obstructive Lung Disease (GOLD) Recommendations into Practice: A Patient Scenario*.[2, 40]

NONPHARMACOLOGIC MANAGEMENT

A growing body of evidence supports nonpharmacologic management strategies to improve quality of life in patients with COPD.

Pulmonary rehabilitation and exercise. Pulmonary rehabilitation, defined as "exercise training for at least four weeks" that may or may not include related education or psychological support, has been shown to have multiple benefits, such as improved health-related quality of life, decreased dyspnea, and increased exercise capacity.[2, 41] The American Thoracic Society and the European Respiratory Society issued a policy statement that advocated pulmonary rehabilitation

How to Put the Global Initiative for Chronic Obstructive Lung Disease (GOLD) Recommendations into Practice: A Patient Scenario

Beatrice Johnson, a 53-year-old woman who works as a sales representative, arrived at her primary care appointment for evaluation of a chronic cough and worsening shortness of breath. (This case is a composite based on our experience.) Ms. Johnson's cough and breathlessness started gradually about two years ago, but both have progressed since that time. She says the cough is worse in the morning, and that she expectorates two to three tablespoons of clear phlegm upon awakening. She can walk about one block on flat ground or up a half flight of stairs before she needs to stop for a rest. She has occasional wheezing. She uses an albuterol inhaler several times a week, which slightly improves her symptoms. She has been smoking a pack of cigarettes per day since she was 20 years old. She has never seriously attempted smoking cessation but is considering it now. She reports no radiating chest pain but says her chest feels tight at times. She says she has never been hospitalized or treated in the ED for respiratory distress. Her most recent immunization was the tetanus–diphtheria–pertussis vaccine six years ago. Her family respiratory history is unremarkable.

Ms. Johnson's vital signs are as follows:

- temperature, 97.6°F (36.4°C)
- heart rate, 85 beats per minute
- respiratory rate, 20 breaths per minute
- blood pressure, 135/68 mmHg

Her body mass index is 22 kg/m^2 and oxygen saturation (SpO_2) is 95% on room air. She is alert, oriented, and in no acute distress while at rest. Her cardiac examination is unremarkable. Chest auscultation reveals slightly diminished breath sounds, with a prolonged expiratory phase and some scattered end-expiratory wheezes. She has no peripheral edema, cyanosis, or digital clubbing.

Because of her long smoking history, cough, dyspnea on exertion, and expiratory wheezes on physical examination, chronic obstructive pulmonary disease (COPD) is a potential diagnosis. Other considerations include asthma, congestive heart failure, bronchiectasis, tuberculosis, and lung cancer.[2] The health care provider orders spirometry.

The ratio of Ms. Johnson's postbronchodilator forced expiratory volume in the first second (FEV_1) to her forced vital capacity (FVC) is 58%; her FEV_1 is 62% of predicted normal value. On a six-minute walk test, her pulse oximetry reading shows no significant oxygen desaturation. A chest X-ray to rule out bronchiectasis, tuberculosis, and lung cancer is unremarkable. Based on these findings, her primary care provider diagnoses her with COPD and orders an alpha-1 antitrypsin deficiency (AATD) screening to determine whether there is a genetic component to Ms. Johnson's COPD. Her AATD screening is normal. The health care provider then assesses the severity of Ms. Johnson's COPD symptoms with the COPD Assessment Test (CAT), on which a score of less than 10 is considered a low symptom burden and a score of 10 or higher a high symptom burden.[2] Ms. Johnson's score is 18.

Classifying Ms. Johnson's COPD. Given Ms. Johnson's spirometry results—a postbronchodilator FEV_1/FVC below 70%, and a FEV_1 between 50% and 79% of predicted normal value—she is initially classified as COPD GOLD 2. Since she has had no exacerbations within the past year and has a CAT score of 18, she is further designated as being in GOLD Group B. At diagnosis, therefore, she is classified as COPD GOLD 2 Group B.

Prevention plus management. After diagnosis and classification, the health care provider uses the "5 A's" model (ask, advise, assess, assist, and arrange) to address Ms. Johnson's feelings about smoking cessation. Ms. Johnson says she wants to quit smoking. The provider assists her by enrolling her in a smoking cessation program and prescribing nicotine replacement therapy.

Additionally, the health care provider orders the 23-valent pneumococcal polysaccharide vaccine and the influenza vaccine, prescribes a long-acting muscarinic antagonist (LAMA) to be used daily, and counsels Ms. Johnson to use her albuterol inhaler for rescue. Ms. Johnson is provided information on the benefits of a pulmonary rehabilitation program and agrees to participate. An order is placed for pulmonary rehabilitation, which includes COPD education and stresses the importance of smoking cessation, medication adherence, proper inhalation techniques, optimal nutrition, exercise, and symptom monitoring. The patient and health care provider collaborate to create a personalized self-management plan and schedule a one-month follow-up appointment at the conclusion of the visit.

Follow-up. When Ms. Johnson returns for her one-month follow-up, she reports that she's participating in a smoking cessation program and has not smoked a cigarette since she started using nicotine replacement. She is, however, still having dyspnea and is unable to walk up a flight of stairs without resting.

In accordance with the GOLD report, the provider adds a long-acting β_2-agonist (LABA) to her medication regimen. A LAMA and LABA may be prescribed together in a single metered dose inhaler. (When prescribing COPD medications, providers should try to ensure that medications are covered by the patient's health insurance program and ask patients to verify current insurance coverage at each visit. The burden of prescription medication costs and out-of-pocket expenses is a significant barrier to patient medication adherence.[40])

At her follow-up appointment a month later, Ms. Johnson describes her symptoms as well managed. The health care provider continues Ms. Johnson's LABA–LAMA prescription and schedules a follow-up six months later. At this

follow-up, Ms. Johnson says she has not had any worsening of her COPD symptoms. She continues to adhere to the prescribed medication and smoking cessation regimen, and is participating in pulmonary rehabilitation. She is now able to walk up a full flight of stairs without resting. The provider suggests that Ms. Johnson maintain her plan of care and schedules a one-year follow-up appointment.

At her one-year follow-up, Ms. Johnson reports continued smoking abstinence, no exacerbations, and decreased breathlessness and cough. She is able to walk several blocks and one flight of stairs without resting. Her postbronchodilator FEV_1 is 64% of predicted normal value, and her CAT score is 13. Although her symptoms have improved, Ms. Johnson is still classified as GOLD 2 Group B.

Considering medication deescalation. Since Ms. Johnson's symptom management has improved, the health care provider may consider deescalating her medication by removing the LABA and continuing her LAMA therapy. The provider encourages continued smoking cessation and verifies that Ms. Johnson's vaccinations are current and that she is participating in an exercise program and following her self-management plan.

Changes in patient classification. Patients do not necessarily move in a linear fashion from GOLD Group A to D. If Ms. Johnson were to have an exacerbation that required hospital admission, the GOLD report advises health care providers to follow up with the patient after discharge, repeating spirometry and symptom assessment with either the CAT or the modified British Medical Research Council Questionnaire.[2] Even if Ms. Johnson's spirometry results and CAT score remained the same, Ms. Johnson would be reclassified as GOLD 2 Group D, because she would be considered at high risk for exacerbations.

If Ms. Johnson's COPD worsens, the health care provider may incorporate additional nonpharmacologic management strategies, such as additional education, exercise training, adjustments to her self-management plan, and palliative care as appropriate.

programs that include exercise training, education designed to promote physical and psychological health, smoking cessation, and self-management.[2] The GOLD report recommends that patients in Groups B through D be enrolled in a pulmonary rehabilitation program.[2]

Education and self-management. The GOLD investigators found that the benefits of stand-alone education were unclear.[2] However, they emphasize that, while education by itself may neither change behavior nor motivate patients, it can improve self-care skills and impart information about COPD, specific therapies, strategies for minimizing dyspnea, and when to seek help.[2] The GOLD report suggests that personalized self-management design plans may be created based on COPD group classification (see Table 3[2]).

Nutritional support. Some patients with COPD are prone to pulmonary cachexia, a loss of lean muscle mass associated with adverse outcomes. Patients with a low body mass index tend to have worse outcomes. Nutritional supplementation has been shown to increase lean body mass and skinfold thickness, improve respiratory muscle strength, and increase health-related quality of life.[42]

Oxygen therapy delivered for at least 15 hours per day has been shown to improve survival rates among patients with chronic respiratory failure and severe resting hypoxemia.[40] It has not, however, been shown to increase survival or time to first hospitalization among patients with stable COPD who have mild to moderate resting, nocturnal, or exercise-induced arterial desaturation.[43, 44]

Noninvasive positive-pressure ventilation (NPPV) is a self-administered respiratory support system that uses positive pressure to support spontaneous breathing. Among patients with COPD, NPPV has been shown to lower risks of in-hospital mortality and hospital-acquired pneumonia, reduce lengths of stay, and lower costs of hospital treatment, though patients with multiple comorbidities and those who are admitted with pneumonia may require invasive mechanical ventilation.[45] NPPV may also be beneficial at discharge. A randomized controlled trial of 166 patients who used bilevel NPPV during hospitalization

Table 3. The GOLD Personalized Self-Management Plans Based on Group[2]

Groups	Management Focus
Groups A, B, C, and D	Medication Behavioral risk factors Smoking cessation Physical activity Adequate sleep Healthy diet
Groups B and D	Breathlessness Energy conservation techniques Stress reduction strategies
Groups C and D	Symptom trigger avoidance Symptom monitoring Written action plan Communication with health care professional
Group D	Palliative strategies Advance care directive

GOLD = Global Initiative for Chronic Obstructive Lung Disease.

showed a statistically and clinically significant reduction in hospitalization readmission rates among those who were discharged with NPPV compared with those who were not (39.7% versus 75% at 180 days).[46] Furthermore, patients who were discharged with NPPV and readmitted were less likely to be admitted to the ICU (8% versus 32% at 180 days) and less likely to be intubated (6% versus 18% at 180 days).

Continuous positive airway pressure (CPAP). Patients who have both COPD and obstructive sleep apnea, known as "overlap syndrome," benefit from using CPAP during sleep. An analysis of outcomes among 228 patients with overlap syndrome who were treated with CPAP, 213 with overlap syndrome who were not treated with CPAP, and 210 who had COPD only, found that patients with overlap syndrome who were not treated with CPAP had a higher rate of exacerbation-related hospitalization and death from any cause over a median follow-up period of 9.4 years than patients who had COPD only. Patients with overlap syndrome who were treated with CPAP had no increased risk of either outcome.[47]

Interventional therapy. For selected patients with severe COPD, extensive parenchymal damage, hyperinflation, and no contraindications, there are a variety of interventional therapies—including bullectomy, lung volume reduction surgery, bronchoscopic lung volume reduction, endobronchial valve therapy, and lung volume reduction coil—that can decrease lung volume, increase recoil, and reduce hyperinflation.[2] The choice of intervention depends on the presence and size of bullae, the extent and pattern of emphysema, and the presence or absence of interlobar collateral ventilation. In patients with severe COPD and no contraindications, a lung transplant may be an option.[2] A Cochrane review found that patients who had lung volume reduction surgery were at increased risk for death three months after the procedure.[48]

MANAGING COMORBID DISEASES

Patients with COPD often have multiple comorbidities and chronic, systemic inflammation. Divo and colleagues found that the 12 comorbidities most strongly associated with increased risk of death in COPD were lung cancer, pulmonary fibrosis, congestive heart failure, coronary artery disease, atrial fibrillation or flutter, esophageal cancer, liver cirrhosis, gastric or duodenal ulcer, diabetes with neuropathy, pancreatic cancer, breast cancer, and anxiety.[49] Comorbid conditions do not alter the treatment of COPD, and a COPD diagnosis does not alter the treatment of other comorbid conditions, though the GOLD report does recommend limiting polypharmacy whenever possible.[2]

PALLIATIVE TREATMENT

Patients may benefit from palliative care even when receiving optimal pharmacologic therapy, as it may reduce dyspnea, fatigue, depression, and anxiety.[2] Research supports the use of opioids, neuromuscular electrical stimulation, oxygen therapy, and a fan blowing in the patient's face to decrease the sensation of breathlessness.[2] Benzodiazepines have not been shown to have any beneficial effect, and there is insufficient evidence to recommend distractive auditory stimulation (music), relaxation, counseling, breathing relaxation training, or psychotherapy at this point.[2]

Palliative treatment is not restricted to end-of-life care, but hospice provides a framework for delivering increasing palliative care, and providers may consider discussing hospice with patients to determine interest in this service. Family members of patients with COPD also need support and information in order to make decisions consistent with the patient's desires. ▼

For seven additional continuing nursing education activities on the topic of COPD, go to www.nursingcenter.com/ce.

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