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COPD Exacerbations
Evidence-based guidelines for identification, assessment, and management.

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Overview: Chronic obstructive pulmonary disease (COPD) is the third leading cause of death in the United States. It's estimated that more than 13 million U.S. adults have COPD, and as many as 24 million U.S. adults have evidence of impaired lung function, suggesting that COPD is underdiagnosed. **[Leah: We can't say "13 million people *are diagnosed* with COPD" because the 13 million is based on an analysis of raw *survey* data from the National Center for Health Statistics, not *diagnostic* data.]** Even when patients receive optimal COPD therapy, they periodically experience exacerbations, which reduce lung function and quality of life, increase risk of death from COPD, and account for the majority of costs related to COPD treatment. A previous *AJN* article focused on the management of stable COPD in the outpatient setting (see "An Evidence-Based Approach to COPD," in *AJN*'s March 2012 issue). This article outlines current guidelines and evidence-based recommendations for identifying, assessing, and managing COPD exacerbations.

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death in the United States.(Kochanek 2011) Based on an analysis of raw data from the Centers for Disease Control and Prevention, the American Lung Association estimates that, as of 2008, more than 13 million adults in the United States had COPD, though data from the National Health and Nutrition Examination Survey III indicate that as many as 24 million Americans, or roughly 5% to 10% of the U.S. population, have evidence of impaired lung function, suggesting that COPD is underdiagnosed.(ALA 2011; Mannino et al 2002) The National Heart, Lung, and Blood Institute (NHLBI) estimates the annual cost of COPD in the United States to be $38.8 billion in 2005 dollars, more than half of which can be directly attributed to inpatient, outpatient, and pharmaceutical expences.(Foster et al 2006) Even when patients receive optimal COPD therapy, they periodically experience exacerbations, which reduce lung function and quality of life, increase risk of death from COPD, and often require inpatient hospital treatment.(GOLD 2011; Kessler et al 2006; Soler-Cataluna et al 2005; Toy et al., 2010) Of the 53.7 billion dollars spent in the United States in 2008 on asthma and COPD, approximately 30% ($16.2 billion) was accounted for by inpatient hospital admissions and emergency room visits.(National Heart, Lung, and Blood Institute. *Morbidity and mortality: 2012 chart book on cardiovascular, lung, and blood diseases*. Bethesda, MD: National Institutes of Health; 2012 Feb. Chart book;

http://www.nhlbi.nih.gov/resources/docs/2012_ChartBook_508.pdf) Hospital admission is an independent risk factor for death from COPD—for patients admitted with hypercarbic exacerbations, inpatient mortality is about 10%, with mortality after two years roughly 50%.(Connors et al., 1996)

Following up on a previous *AJN* article that reviewed the essential features of COPD and its management in the outpatient setting (see "An Evidence-Based Approach to COPD," in *AJN*'s March 2012 issue), this article focuses on COPD exacerbations. After briefly reviewing COPD pathophysiology, we discuss current recommendations for identifying, assessing, and managing exacerbations, as put forward in evidence-based guidelines compiled by the Global Initiative for Chronic Obstructive Lung Disease (GOLD), an international committee comprised of thought leaders from such groups as the NHLBI; the U.S. Department of Health and Human Services' National Institutes of Health (NIH), and the World Health Organization (WHO).(GOLD 2011)

COPD: THE BASICS

COPD is a lung disease characterized by progressive airflow limitation, resulting from small airway disease and parenchymal destruction.(GOLD 2011) Major risk factors include exposure to smoke (including tobacco, cooking fires, and fuel), occupational dust, or fumes.(Kohansal et al 2009; Matheson et al 2005; Sezer, Akkurt, Guler, Marakoglu, & Berk 2006) Other risk factors include family history, female gender, α1-antitrypsin deficiency, recurrent respiratory infections, low socioeconomic status, poor nutrition, and asthma.(Eisner et al., 2010; GOLD, 2011)

Within the parenchyma, inflammation diminishes the elastic recoil of lung tissue and destroys alveolar attachments to small airways. This parenchymal destruction, known as emphysema, reduces gas exchange and causes lungs to collapse during expiration, limiting airflow (as measured by the forced expiratory volume in the first second [FEV_1] of exhaling) to a degree that is not fully reversible.(Barnes, Shapiro, & Pauwels, 2003; GOLD, 2011) Combined with mucus plugging, these changes contribute to the development of dyspnea, cough, and sputum production, which characterize COPD. Although the progressive and variable nature of COPD is unique to each individual, a rise in exacerbation frequency generally indicates disease progression.(Donaldson, Seemungal, Bhowmik, & Wedzicha, 2002; Hurst et al., 2010)

ETIOLOGY AND PATHOPHYSIOLOGY OF EXACERBATI ONS

The GOLD guidelines define a COPD exacerbation as "an acute event characterized by a worsening of the patient's respiratory symptoms that is beyond normal day-to-day variations and leads to a change in medication."(GOLD, 2011) Exacerbations are characterized either symptomatically or through health care resource utilization, but because the latter depends on patient symptom recognition and resource availability, incidence of exacerbation is likely to be significantly underestimated.(GOLD, 2011; Trappenburg et al., 2011) Certain patients seem more prone to exacerbations, with those experiencing two or more exacerbations a year being termed "frequent exacerbators."(GOLD, 2011; Hurst et al., 2010) Exacerbations accelerate the deterioration of lung function, as evidenced in declining peak expiratory flow (PEF) and FEV_1 values.(Donaldson et al., 2002) While patients may recover lung function over the course of several weeks, in many cases it does not return to baseline.(GOLD, 2011; Kanner, Anthonisen, & Connett, 2001) Disease progression, exacerbation frequency, and diminishing lung function form a vicious cycle.

Causes and risk factors for COPD exacerbation include air pollution, temperature changes, and most commonly, bacterial and viral infections.(Peacock et al., 2011; Sethi & Murphy, 2008; GOLD, 2011) Treatment nonadherence and interruption of maintenance therapy also can precipitate exacerbations, though a cause can't be identified in about one third of all cases.(GOLD, 2011) Exacerbations are significantly more likely to occur during cold winter months than during any other time of year.

During exacerbation, precipitants worsen the bronchial inflammation characteristic of stable COPD, as evidenced by a rise in bronchial neutrophils and signs of oxidative stress.(Drost et al., 2005; Qiu et al., 2003) Likewise, levels of such systemic inflammatory markers as interleukin-6, C-reactive protein, and plasma fibrinogen rise during exacerbation.(Wedzicha et al., 2000; Wedzicha & Seemungal, 2007) Bronchial inflammation increases pulmonary secretion, smooth muscle contraction, airway edema, and parenchymal destruction. Resultant airway narrowing and airflow obstruction further alter lung mechanics.

As functional lung volumes change, pathophysiologic sequelae become clinically evident. Airway narrowing reduces expiratory flow, causing rapid, shallow breathing that leads to lung hyperinflation. Respiratory muscles become overworked, and dyspnea ensues. Cough and ventilation–perfusion mismatch worsen (see "Ventilation–Perfusion Ratio)**[BOX]**. Parenchymal destruction inhibits gas exchange, resulting in hypoxemia, with or without hypercapnia.(GOLD, 2011)

IDENTIFYING EXACERBATIONS

COPD exacerbations tend to be characterized by patient reports of increased symptom severity rather than by the onset of new symptoms.(Jones, Chen, Wilcox, Sethi, & Leidy, 2011) Symptoms that commonly worsen during exacerbation include the following:(GOLD, 2011; Jones et al., 2011)

- dyspnea and cough, with or without sputum production
- chest congestion
- chest discomfort
- sleep disturbance
- feelings of weakness, fatigue, fear, or worry

Almost half of patients experiencing a COPD exacerbation report a significant decline in physical activity.(Miravitlles, Anzueto, Legnani, Forstmeier, & Fargel, 2007)

Hallmark clinical features of exacerbation include inflamed, narrowed airways, leading to tachypnea and accessory muscle use. Pursed lip breathing may be more pronounced, as patients try to prevent airway collapse and increase oxygenation. Reduced distance between the cricoid cartilage and suprasternal notch, Hoover's sign (paradoxical retraction of the lower rib cage margin with inspiration), and resonant percussion over the heart reflect pulmonary hyperinflation. Tachycardia and cyanosis may occur in patients with hypoxia. In severe cases, patients may exhibit hypotension, flapping tremor (an involuntary trembling of the hand when the wrist is extended), and mental status changes.

No single test can help NPs and other clinicians establish that a patient is experiencing an exacerbation. Rather, exacerbations are identified as such based on patient presentation. Early recognition is critical in initiating prompt treatment, thereby reducing risk of hospitalization, future exacerbations, and impaired quality of life.

ASSESSING EXACERBATION SEVERITY

Once you've established that a patient is experiencing an exacerbation, assess its severity based on the patient's medical history, signs, and symptoms. Consider prior FEV_1 values, duration of worsened symptoms, number of previous exacerbations, comorbidities, previous use of mechanical ventilation, and present treatment regimen.(GOLD, 2011) Use of accessory respiratory muscles, paradoxical chest wall movement, hemodynamic instability, worsening or new-onset central cyanosis, peripheral edema, or mental status decline are signs of severity.(GOLD, 2011)

After an initial assessment, a variety of tests can be used to quantify the severity of an exacerbation, establish a specific etiology, differentiate COPD exacerbation from other conditions with similar symptoms, and uncover potential comorbid conditions (Table 1).(GOLD 2011; Yawn & Thomashow 2011; Rizkallah, Man & Sin 2009; Soriano et al 2005) For example, lung hyperinflation, which is common in exacerbation, can mask coexisting cardiac and pulmonary signs. Investigate potential causes unrelated to COPD exacerbation based on patient history, clinical suspicion, and patient failure to respond to traditional exacerbation treatment.

Use of spirometry is not recommended during exacerbations. Spirometry is difficult for sick patients to perform properly and can be inaccurate during exacerbations.(GOLD, 2011)

MANAGEMENT GOALS AND TREATMENT SETTING

The goal of COPD exacerbation management is to reduce the impact of the current exacerbation and the likelihood of future exacerbations. Four general objectives guide management:

- address precipitating factors
- reduce air trapping to boost expiratory flow
- decrease pulmonary inflammation
- improve gas exchange

Some patients with COPD exacerbations can be safely and effectively treated at home; others require hospitalization (Table 2). (GOLD 2011; Asiimwe 2011) Closely monitor patients being treated at home for indications that hospital admission is required, such as significant increase in symptom intensity, onset of cyanosis or peripheral edema, need for ventilatory support, or insufficient improvement with home treatment.(GOLD, 2011) Depending on an institution's policy, patients requiring noninvasive ventilatory support may or may not require ICU admission. If treated outside of the ICU, such patients require extremely close monitoring, as their condition may deteriorate quickly. If noninvasive ventilation fails, clinicians need to be able to initiate invasive mechanical ventilation rapidly. All patients requiring mechanical ventilation must be admitted to the ICU. Changes in mental status or hemodynamic instability are indications for direct ICU admission.(GOLD, 2011) Likewise, severe dyspnea that is unresponsive to initial treatment, persistent or worsening hypoxemia ($PaO_2 < 40$ mmHg), or persistent or worsening acidosis ($pH < 7.25$) despite supplemental oxygen may indicate the need for invasive ventilatory support in the ICU.(GOLD, 2011)

Before admitting a patient to a hospital or ICU, verify the level of care the patient desires and confirm that any advance directive, living will, or health care proxy documentation is on file with the admitting institution, outlining patient wishes regarding the level of medical intervention he or she is to receive. Ideally, patients should be encouraged to establish these directives when clinically stable and capable of making rational judgments, not during exacerbation.

PHARMACOTHERAPY

Bronchodilators. In both outpatient and inpatient settings, the first intervention usually involves increasing the dose or frequency of a currently prescribed, short-acting inhaled bronchodilator, such as the β**[Greek beta symbol]**$_2$**[subscript]**-agonist albuterol (prescribed at 2.5 mg nebulized or 180 mcg by metered dose inhaler, dosed every 20 minutes for up to two hours or until the patient improves clinically or develops adverse effects), with or without a concurrent short-acting anticholinergic, such as ipratropium (prescribed at 500 mcg nebulized, given six to eight hours apart, or 34 mcg by metered dose inhaler, dosed every two to four hours). If the patient was not previoulsy using a short-acting anticholinergic, it can be initiated during an exacerbation (see "Interventions Used in COPD Management")**[BOX]**.(GOLD, 2011) With rapid onset of action, these medications quickly reverse bronchoconstriction, reducing lung volume, increasing expiratory flow, and preventing hyperinflation. Monitor patients taking these medications for such adverse effects as hypokalemia and anxiety, associated with β**[Greek beta symbol]**$_2$**[subscript]**-agonists, and dry mouth, urinary retention, and constipation, associated with anticholinergics. If the patient improves with outpatient treatment, medications that are initially increased with acute symptoms should be decreased when clinically possible.

Considered second-line therapy, methylxanthines, such as theophylline, are less effective and have a greater number of adverse effects than the inhaled bronchodilators.(GOLD, 2011) Methylxanthines relax bronchial smooth muscle by inhibiting phosphodiesterase and antagonizing adenosine receptors. Although they may be prescribed for the purpose of expanding airway diameter and preventing hyperinflation, patient benefits are not impressive, and adverse effects—including headache, nausea, vomiting, abdominal discomfort, and restlessness—are significant.(Barr, Rowe, & Camargo, 2003)

Systemic glucocorticosteroids administered during COPD exacerbation may shorten recovery time, improve lung function, and decrease hypoxemia. Likewise, they may reduce risk of early relapse, treatment failure, and length of hospital stay. By lessening pulmonary inflammation, steroids improve airflow.

In both inpatient and outpatient settings, the steroid prescribed most often for COPD exacerbation is oral prednisolone at a dosage of 30 to 40 mg daily for 10 to 14 days. Higher dosages and longer treatment periods provide no added benefit and increase the likelihood and severity of such adverse effects as hyperglycemia, nervousness, insomnia, and muscle atrophy. Alternatively, nebulized budesonide may be used with fewer adverse effects, though it's more costly.(GOLD, 2011) In patients requiring invasive mechanical ventilation, muscle atrophy secondary to steroid use may be potentiated by the simultaneous use of nondepolarizing neuromuscular-blocking agents, thereby delaying weaning and extubation.

Steroids should be used in conjunction with, not in place of, other exacerbation therapies, such as inhaled bronchodilators. Bear in mind that patients treated frequently with corticosteroids are at elevated risk for osteoporosis.

Antibiotics are used in the outpatient setting to treat exacerbations when patients have clinical signs of bacterial infection, as evidenced by increased sputum purulence and volume, with or without dyspnea, or increased sputum purulence and dyspnea, with or without increased sputum volume.(GOLD, 2011) This recommendation reflects the fact that purulent sputum is associated with lower respiratory tract bacteria.(GOLD, 2011)

During COPD exacerbations, the most common causative bacteria are *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis*,(Sethi & Murphy, 2008) though atypical and resistant organisms may also be present. Specific antibiotic choice should be determined based on GOLD and local guidelines designed to minimize antibiotic resistance.(GOLD, 2011) In the inpatient and outpatient settings, selection and use differ in only one way: for patients requiring either invasive or noninvasive mechanical ventilation, antibiotic treatment should not be empirical, but based on sputum cultures because gram-negative and treatment resistant organisms are more common in these patients.(GOLD, 2011) Regardless of inpatient or ICU status, oral antibiotics are preferred to IV antibiotics if the patient is able to take medications by mouth and the antibiotic is available in an oral form. Typically, duration of treatment is five to 10 days.(GOLD, 2011)

OXYGEN THERAPY

During exacerbation, oxygen requirements typically increase, particularly in such low cardiac output states as hypotension, or such high metabolic states as pneumonia. Consider oxygen use in patients with hypoxemia, worsening hypercapnia, or acidosis.(GOLD, 2011) Oxygen should be titrated to improve hypoxemia, with an arterial oxygen saturation (SaO_2) goal of 88% to 92% in patients without complications.(Austin, Wills, Blizzard, Walters, & Wood-Baker, 2010; GOLD, 2011) This modest SaO_2 goal balances the far-reaching negative effects of prolonged hypoxemia against the potential for impaired respiratory muscle function. While impaired respiratory muscle function was once thought to arise exclusively from reduced respiratory drive, it is now understood to result from a combination of this and a worsening ventilation–perfusion mismatch. Usually, only small increases in inspired oxygen are needed to oxygenate tissues sufficiently. In patients whose exacerbations are complicated by low cardiac output or a high metabolic state, a higher SaO_2 goal may be set. In such cases, noninvasive (by nasal cannula or facial mask) or invasive (by endotracheal tube or tracheostomy) mechanical ventilation may be required.

Because excess oxygenation can lead to hypercapnia and acidosis in patients with COPD, arterial blood gasses (ABGs) should be checked 30 to 60 minutes after oxygen therapy is initiated. Venturi masks deliver high-flow oxygen at relatively accurate rates, but the less precise nasal cannula, which delivers lower concentrations of oxygen, is usually better tolerated because it allows patients the freedom to eat, drink, and speak. Because oxygen delivery is more variable with the nasal cannula, measure ABGs in patients using this oxygen delivery method more frequently, based on the stability of the their condition, the frequency of oxygen titration, and clinical judgment.

Noninvasive ventilation, which is associated with lower rates of nosocomial pneumonia than mechanical ventilation, is a first-line intervention in patients with moderate to severe acidosis or hypercapnia who are also tachypneic and dyspneic despite less invasive medical treatment.(GOLD, 2011; Anonymous, 1999) Consider noninvasive ventilation when pH is between 7.25 and 7.35, when partial pressure of carbon dioxide in arterial blood ($PaCO_2$) is greater than 45 mmHg(GOLD, 2011; Anonymous, 1999), or when severe dyspnea is accompanied by respiratory muscle fatigue or increased work of breathing.(GOLD, 2011; Anonymous, 1999) In patients who do not wish to be intubated, noninvasive ventilation offers an alternative means of ventilatory support. Noninvasive ventilation should be used only in a closely monitored setting in case the need for invasive mechanical ventilation arises.

After determining that a patient would benefit from noninvasive ventilation, rule out any contraindications related to mask placement or the need for greater effect. The tight-fitting, restrictive mask is generally not well tolerated by patients who are agitated, uncooperative, or whose mental state is altered. Likewise, patients who are at a high risk for vomiting or aspiration; patients with significant secretions; and those with recent gastroesophageal or facial surgery or trauma—whose injuries may be compounded by the mask—are poor candidates for noninvasive ventilation. Patients with profound hypoxemia, cardiovascular instability, or respiratory arrest, are generally better served by invasive mechanical ventilation.

Monitor patients using noninvasive ventilation closely. The first signs of treatment success are reductions in the respiratory rate and the use of accessory muscles during respiration. Consider measuring ABGs 20 to 30 minutes after initiating noninvasive mechanical ventilation; if acidosis or hypercapnia has not responded by then, consider invasive mechanical ventilation.

Invasive mechanical ventilation can be used during severe COPD exacerbations. Significant risks of mechanical ventilation include hypotension, ventilator-acquired pneumonia, failure to wean from the ventilator, and barotrauma.

Consider initiating mechanical ventilation when a trial of noninvasive ventilation is either contraindicated or has failed.(GOLD 2011) Patients who are in respiratory arrest, have respiratory pauses, or are gasping for air should be considered for mechanical ventilation, as should hemodynamically unstable patients (including those with severe ventricular arrhythmias or symptomatic bradycardia) and patients with impaired mental status, history of recent massive aspiration, or the inability to remove respiratory secretions. Life-threatening hypoxemia in patients who are unable to tolerate noninvasive ventilation is a laboratory indication for mechanical ventilation.(GOLD, 2011; Conti, 2002)

Mechanical ventilation during COPD exacerbation improves gas exchange and reduces respiratory muscle work. Controlled ventilator settings that address specific COPD respiratory deficits, such as hyperinflation and elevated intrinsic positive end-expiratory pressure, help achieve therapeutic effects. Decreasing respiratory rate while limiting tidal volume increases expiratory time, improving patient–ventilator synchrony and reducing hyperinflation. As optimized lung mechanics normalize blood gases, the ventilation–perfusion ratio improves.

Generally, larger endotracheal tubes (7.5 to 8 mm internal diameter) are used to minimize airflow resistance. Bronchodilators and other medications that improve airflow should be maximized while the patient is intubated. In order to minimize inhaled drug deposition within the endotracheal tube, spacer devices should be used with inhalers, and nebulizers should be placed in the inspiratory line at least 30 cm from the endotracheal tube. When titrating ventilator settings, bear in mind the patient's preadmission baseline $PaCO_2$ so as not to reduce the ventilator-assisted $PaCO_2$ to a level that cannot be sustained.

As acidosis and hypoxia improve with mechanical ventilation, secretions lessen and respiratory muscles strengthen. Ventilator weaning can be difficult and hazardous, with the biggest challenge being to maintain the balance between respiratory workload and muscle strength.(Purro et al., 2000) Patients with COPD meet standard ventilator-weaning criteria less often than patients without COPD, but when they meet the criteria, they are more likely to wean successfully than their counterparts without COPD.(Calverley, 2011) Currently, there is little consensus on the best method by which

to wean patients with COPD. Available options include pressure support, T-piece, and bridging with noninvasive ventilation. In an improving patient, the general aproach to weaning and extubation is to withhold sedatives and analgesics prior to a spontaneous breathing trial, while considering immediate transfer from invasive to noninvasive ventilation.

OTHER HOSPITAL-BASED THERAPIES

Secretion clearance is important in preventing complications and maximizing comfort for all patients experiencing COPD exacerbation. In patients not receiving inhaled steroids, the mucolytic medication N-acetylcysteine may reduce exacerbations through its antioxidant properties.(Decramer et al., 2005)**]** Although it is not formally recommended for secretion clearance, the 2011 GOLD guidelines weakly recommend its use, noting that further research is needed to define its role in COPD treatment.(GOLD, 2011)

Regardless of thromboembolic history, hospitalized patients with COPD should receive prophylactic therapy for deep vein thrombosis. Because these patients often have right ventricular hypertrophy and large pulmonary arteries, they're at elevated risk for blood clots, especially if immobilized, polycythemic, or dehydrated—all of which can occur during exacerbation.

HOSPITAL DISCHARGE AND FOLLOW-UP

Clinical stability can be gauged by satisfactory ABG values, use of rescue inhaler therapy no more frequently than every four hours, and the ability to eat and sleep without frequent interruptions from dyspnea. After a patient has demonstrated clinical stability for 12 to 24 hours, hospital discharge can be considered. For patients who were hypoxemic during the admission, ABGs or pulse oximetry should be measured prior to discharge. (GOLD, 2011) Patients who were mobile before hospital admission should be able to walk at least across the room.

Patients and caregivers need to feel comfortable with the treatment plan. Social workers or other team members should arrange for any necessary home equipment or supplies. The medical team should address any social problems that might interfere with care or adherence.

After discharge, follow up with outpatient assessment in four to six weeks. If the patient had any degree of hypoxemia during admission, a follow-up ABG or pulse oximetry reading should be performed within three months of discharge. Initiate measures to prevent future exacerbations before discharge and reinforce at all subsequent follow-up visits.(GOLD, 2011)

PREVENTION

Because high rates of relapse are typical among patients who have experienced COPD exacerbations, prevention is key.

Smoking cessation is associated with a reduced risk of COPD exacerbation, with the level of exacerbation reduction related to the duration of cessation. Guidelines support smoking cessation as the single most effective means of reducing COPD progression, regardless of disease status.(GOLD, 2011) For a comprehensive approach to help patients quit smoking, see the 2008 U.S. Department of Health and Human Services guidelines on Treating Tobacco Use and Dependence, found at: http://www.ncbi.nlm.nih.gov/books/NBK12193/.**[blue]**

Vaccinations. Like cigarette smoke, bacteria and viruses worsen airway inflammation, exacerbating disease. Appropriate vaccination use among patients with COPD is considered standard of care.(GOLD, 2011) Annual influenza vaccinations reduce both disease exacerbations and death among patients with COPD. (CDC, 2012; Poole, Chacko, Wood-Baker, & Cates, 2006; Schembri, Morant, Winter, & MacDonald, 2009) A one-time dose of pneumococcal vaccine should be administered to all patients with COPD, regardless of age. For those ages 65 and older, a one-time pneumococcal revaccination should be given if five or more years have passed since the original vaccination and if the patient was under age 65 at that time.(Alfageme et al., 2006; CDC, 2012) In order to prevent pertussis infection, all patients with COPD should receive a tetanus-diphtheria-pertussis vaccination (Tdap) with subsequent tetanus-diphtheria (Td) boosters every 10 years.(CDC, 2012)

Pulmonary rehabilitation following COPD exacerbation can prevent subsequent exacerbations and hospitalization. Comprehensive pulmonary rehabilitation programs include exercise training, nutrition counseling, strategies for improving breathing and conserving energy, and education. Remind patients that pulmonary rehabilitation reduces symptoms, improves quality of life, and increases physical and emotional participation in everyday activities in ways that cannot be addressed by medical therapy alone.

PATIENT TEACHING

It's vital to reinforce with patients the importance of adhering to treatment when COPD is stable. By simplifying treatment regimens, increasing patient self-management knowledge, and refining patient teaching skills, providers help optimize adherence and prevent exacerbations.(Lareau & Yawn, 2010)

Research supports that patients commonly employ self-taught self-management during exacerbations,(Trappenburg et al., 2011) but a large number of COPD disease exacerbations are unreported, suggesting that clinicians need to provide more comprehensive patient teaching.(Langsetmo, Platt, Ernst, & Bourbeau, 2008) A recent trial examining the effectiveness of a relatively simple COPD management plan found it reduced both hospitalizations and emergency room visits related to COPD exacerbations.(Rice et al., 2010) Teaching patients to recognize exacerbations and seek immediate intervention leads to prompt medical treatment and improved exacerbation outcomes.(Wilkinson et al., 2004) For a comprehensive, easy-to-use, educational patient handout on COPD, see "An Evidence-Based Approach to COPD," in *AJN*'s March 2012 issue.

Table 1. Tests for Assessing COPD Exacerbation Severity and Etiology(GOLD 2011; Rizkallah, Man & Sin 2009; Soriano et al 2005; Yawn & Thomashow 2011)

Test	Indication	Comments
• Pulse oximetry	• Worsened pulmonary symptoms	• Helps in assessing need for supplemental oxygen therapy
• Sputum culture and gram stain • Antibiotic sensitivity	• Change in sputum volume or appearance	• Perform if patient fails a round of empiric antibiotic treatment or has frequent exacerbations, severe airflow limitation, or requires mechanical ventilation
• ABG measurements	• Signs of respiratory failure • Need for mechanical ventilation	• Measure in all patients requiring hospitalization; vital if acute or acute-on-chronic respiratory failure suspected • Compare to previous ABG measurements • Diagnose respiratory failure if $PaO_2 < 60$ mmHg, with or without $PaCO_2 > 50$ mmHg, when breathing room air • Use to assess acid–base balance before initiating mechanical ventilation
• Electrolyte panel • Glucose level • Acid-base balance • Whole blood count	• Suspected metabolic disorder • Suspected leukocytosis, anemia, polycythemia	• Electrolyte abnormalities, poor glucose control, and acid-base imbalances are all associated with COPD exacerbations
• Brain natriuretic peptide • Radiography	• Elevated jugular venous pressure • Pitting ankle edema • Dyspnea unresolved with COPD exacerbation treatment	• Assists in differentiating congestive heart failure from COPD • Heart failure and COPD are easily confused due to common cardinal symptom of breathlessness
• Quantitative D-	• Severe dyspnea	• Symptoms of chronic lung

dimer • CT pulmonary angiography	• Such concurrent symptoms as syncope, tachypnea, tachycardia, pleuritic chest pain, hemoptysis, and anxiety	disease can mimic those of pulmonary embolism • Patients with COPD may be at elevated risk for pulmonary embolism
• Chest radiograph • ECG • Cardiac enzymes	• Chest discomfort not responsive to traditional exacerbation treatment • Symptoms of pulmonary edema such as progressive respiratory distress, dyspnea, or severe hypoxemia	• CVD is a common comorbidity among patients with COPD • Important in differentiating ischemic heart disease and cardiac rhythm disturbances from COPD exacerbation

Abbreviations: ABG = arterial blood gas. COPD = chronic obstructive pulmonary disease. CT = computed tomography. CVD = cardiovascular disease. ECG = electrocardiography. $PaCO_2$ = partial pressure of carbon dioxide in arterial blood. PaO_2= partial pressure of oxygen in arterial blood.

Table 2. Treatment Setting Considerations (GOLD 2011; Asiimwe 2011)

Initial Treatment Setting	Patient Eligibility Factors
• Home	• Sufficient home support • Condition stabilizes with increased dose or frequency of currently prescribed medication, with or without antibiotic treatment
• Hospital (for assessment and possible admission)	• Insufficient home support • No improvement with initial home treatment • Severe underlying COPD • History of frequent exacerbation • Significant increase in symptom intensity • Advanced age • Onset of new physical signs (such as cyanosis or peripheral edema) • Need for ventilatory support • Significant preexacerbation comorbidities
• ICU	• Need for invasive ventilatory support and, depending on institutional protocol, need for noninvasive ventilatory support • Severe dyspnea not responsive to initial treatment • Persistent or worsening hypoxemia (PaO_2 < 40 mmHg) or acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation • Changes in mental status (from confusion or lethargy to coma) • Hemodynamic instability

[BOX]
Interventions Used in COPD Management(GOLD 2011)

- **Inhaled bronchodilators—**used in most exacerbations to improve airflow obstruction and reduce lung volume
- **Short-course oral steroids—**used in most exacerbations to reduce pulmonary inflammation; shorten recovery time; improve hypoxia and lung function; and reduce risk of relapse, treatment failure, and length of hospital stay
- **Antibiotics—**used in the presence of clinical signs of bacterial infection, as evidenced by increased sputum purulence and volume with or without dyspnea or increased sputum purulence and dyspnea with or without increased sputum volume, and with invasive or noninvasive mechanical ventilation to treat or prevent infection and, thereby, reduce mortality, incidence of intubation, and length of hospital stay
- **Noninvasive ventilation—**used in moderate to severe acidosis (pH 7.25 to 7.35) or hypercapnia ($PaCO_2$ > 45 mmHg), accompanied by tachypnea and dyspnea, and in patients who do not wish to be intubated to improve gas exchange; rest respiratory muscles; and reduce respiratory acidosis, respiratory rate, invasive mechanical ventilation rates and associated complications, and mortality
- **Mechanical ventilation**—used when noninvasive ventilation is inappropriate or has failed; with respiratory arrest, pauses, or gasps; and in patients with unstable hemodynamics (severe ventricular arrhythmias or symptomatic bradycardia), impaired mental status, recent history of massive aspiration, inability to remove respiratory secretions, or severe hypoxemia with inability to tolerate noninvasive ventilation to improve oxygenation and reduce acidosis

Ventilation–Perfusion Ratio [Text Box]
After air enters the lungs, gasses passively diffuse through lung tissues into the pulmonary circulatory system before being delivered to the rest of the body. The relationship between alveolar ventilation (referred to as V) and pulmonary capillary perfusion (referred to as Q) is referred to as the "V/Q ratio." In normal, healthy adults, this ratio approximates 1/1, symbolizing adequate pulmonary gas exchange, or well-matched alveolar–capillary ventilation and perfusion. Factors that negatively affect either ventilation or perfusion can disturb this homeostasis, causing what is referred to as a "V/Q mismatch." In patients with chronic obstructive pulmonary disease, numerous factors, including mucus, chronic airway obstruction, and bronchospasm, can impair ventilation, leading to a mismatch and, ultimately, contributing to hypoxemia and acid-base imbalances.

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