COPD Exacerbations

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

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 have evidence of impaired lung function, suggesting that COPD is underdiagnosed. 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. This article, the second in a two-part series on COPD, outlines current guidelines and evidence-based recommendations for identifying, assessing, and managing COPD exacerbations (the first article in the series, "An Evidence-Based Approach to COPD," March 2012, focused on the management of stable COPD in the outpatient setting).

Keywords: chronic obstructive pulmonary disease, exacerbation, respiratory disease

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death in the United States.[1] 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 third National Health and Nutrition Examination Survey 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.[2,3] 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 expenses.[4] 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.[5-8] Of the $53.7 billion spent in the United States in 2008 on asthma and COPD, approximately 30% ($16.2 billion) was accounted for by inpatient hospital admissions and ED visits.[9] Hospital admission is an independent risk factor for death from COPD; in patients admitted with hypercarbic exacerbations, inpatient mortality is about 10% and mortality after two years is roughly 50%.[10]

In this article, after briefly reviewing COPD pathophysiology, we discuss current recommendations for identifying, assessing, and managing COPD exacerbations, as put forward in evidence-based guidelines compiled by the Global Initiative for Chronic Obstructive Lung Disease (GOLD), an international committee of thought leaders from such groups as the NHLBI; the National Institutes of Health, and the World Health Organization.[5]

COPD: THE BASICS

COPD is a lung disease characterized by progressive airflow limitation resulting from small-airway disease and parenchymal destruction.[5] Major risk factors include exposure to smoke (including tobacco, cooking fires, and fuel), occupational dust, or fumes.[11-13] Other risk factors include family history,

female sex, α_1-antitrypsin deficiency, recurrent respiratory infections, low socioeconomic status, poor nutrition, and asthma.[5, 14]

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.[5, 15] 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.[16, 17]

ETIOLOGY AND PATHOPHYSIOLOGY OF EXACERBATIONS

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."[5] 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

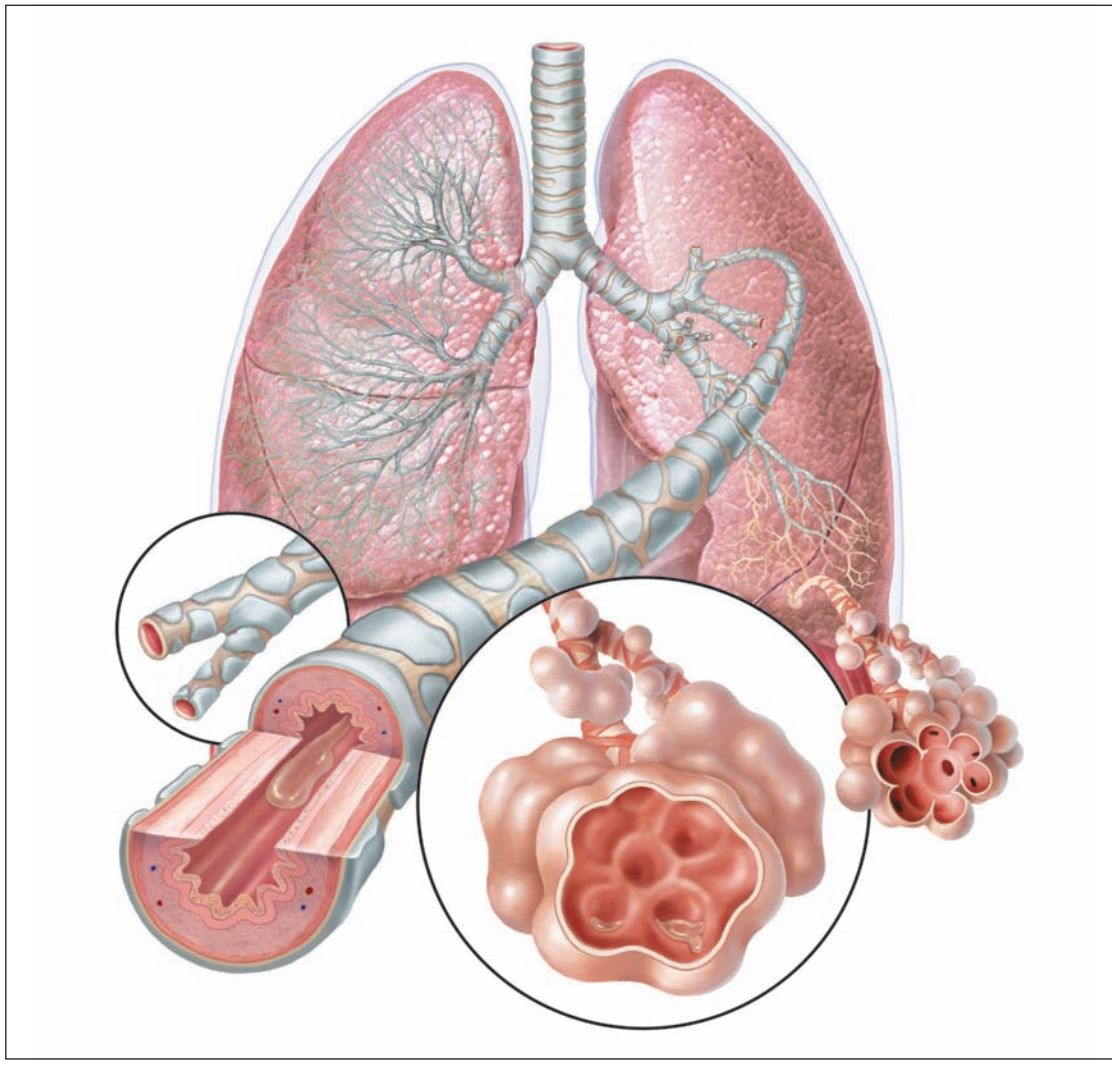

Figure 1. COPD is characterized by inflammation and destruction of alveoli that cause decreased gas exchange, collapse of lungs during expiration, and air trapping. During an exacerbation the inflammatory process worsens, resulting in increased mucus production, airway narrowing, and further destruction of alveoli. Illustration by Anne Rains.

Ventilation–Perfusion Ratio

After air enters the lungs, gases 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 called the "V/Q ratio." In normal, healthy adults, this ratio is approximately 1:1, representing 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 COPD, 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.

underestimated.[5, 18] Certain patients seem more prone to exacerbations, with those experiencing two or more a year termed "frequent exacerbators."[5, 17] Exacerbations accelerate the deterioration of lung function, as evidenced in declining peak expiratory flow and FEV_1 values.[16] While patients may recover lung function over the course of several weeks, in many cases it does not return to baseline.[5, 19] Disease progression, exacerbation frequency, and diminishing lung function form a vicious cycle.

No single test can help NPs and other clinicians establish that a patient is experiencing an exacerbation.

Causes and risk factors for COPD exacerbation include air pollution, temperature changes, and most commonly, bacterial and viral infections.[5, 20, 21] Treatment nonadherence and interruption of maintenance therapy can also precipitate exacerbations, though a cause can't be identified in about one-third of cases.[5] Exacerbations are significantly more likely to occur during cold winter months than during any other time of year.

During exacerbations, precipitants worsen the bronchial inflammation characteristic of stable COPD, as evidenced by a rise in bronchial neutrophils and signs of oxidative stress.[22, 23] Likewise, levels of such systemic inflammatory markers as interleukin-6, C-reactive protein, and plasma fibrinogen also rise.[24, 25] 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*). Parenchymal destruction inhibits gas exchange, resulting in hypoxemia, with or without hypercapnia.[5]

IDENTIFYING EXACERBATIONS

COPD exacerbations tend to be characterized by patient reports of increased symptom severity rather than by the onset of new symptoms.[26] Symptoms that commonly worsen during exacerbation include[5, 26]

- 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.[27]

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 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.[5] Signs of severity include use of accessory respiratory muscles, paradoxical chest wall movement, hemodynamic instability, worsening or new-onset central cyanosis, peripheral edema, or mental status decline.[5]

After an initial assessment, a variety of tests can be used to quantify the severity of an exacerbation, establish a specific etiology, differentiate a COPD exacerbation from other conditions with similar symptoms, and uncover potential comorbid conditions. For example, lung hyperinflation, which is common in COPD exacerbations, can mask coexisting cardiac and pulmonary signs. Based on patient history, clinical suspicion, or patient failure to respond to traditional exacerbation treatment, investigate the causes of symptoms that may be unrelated to COPD exacerbation. For a list of tests and the indications for their use, see Table 1.[5, 28-30]

Use of spirometry is not recommended during exacerbations. Spirometry is difficult for sick patients to perform properly and can be inaccurate if performed during an exacerbation.[5]

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 guiding management are to:

- 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 (see Table 2 for a list of determining factors[5, 31]). Closely monitor patients 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.[5] Patients needing noninvasive ventilatory support may or may not require ICU admission, depending on an institution's policy. If treated outside 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.[5] Likewise, severe dyspnea that is unresponsive to initial treatment, persistent or worsening hypoxemia (when partial pressure of oxygen in arterial blood [PaO_2] is less than 40 mmHg), or persistent or worsening acidosis (when pH is less than 7.25) despite supplemental oxygen may indicate the need for invasive ventilatory support in the ICU.[5]

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. Ideally, patients should be encouraged to establish these directives when clinically stable and capable of making rational judgments, not during an exacerbation.

For an overview of the COPD interventions discussed below and the reasons for their use, see *Interventions Used in COPD Management*.[5]

PHARMACOTHERAPY

Bronchodilators. In both outpatient and inpatient settings, the first intervention usually involves increasing

Interventions Used in COPD Management[5]

- **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 (partial pressure of carbon dioxide in arterial blood, > 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

Table 1. Tests for Assessing the Severity and Etiology of COPD Exacerbations[5, 28-30]

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 measurement	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 with 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 because of common cardinal symptom of breathlessness
Quantitative D-dimer CT pulmonary angiography	Severe dyspnea Concurrent symptoms such as syncope, tachypnea, tachycardia, pleuritic chest pain, hemoptysis, and anxiety	Symptoms of chronic lung 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 in patients with COPD Important in differentiating ischemic heart disease and cardiac rhythm disturbances from COPD exacerbation

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.

the dose or frequency of a currently prescribed, short-acting inhaled bronchodilator, such as the β_2-agonist albuterol (Ventolin HFA and others) (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 agent, such as ipratropium (Atrovent) (prescribed at 500 mcg nebulized, given six to eight hours apart, or 34 mcg by metered dose inhaler, dosed every two to four hours). Even if the patient was not previously using a short-acting anticholinergic, one can be initiated during an exacerbation. With rapid onset of action, bronchodilators 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_2-agonists, and dry mouth, urinary retention, and constipation, associated with anticholinergics. Once the patient improves, medications that were initially increased with acute symptoms should be decreased when clinically possible.

Considered second-line therapy, methylxanthines, such as theophylline (Bronkodyl and others), are less effective and have a greater number of adverse effects than the inhaled bronchodilators.[5] Methylxanthines relax bronchial smooth muscle by inhibiting phosphodiesterase and antagonizing adenosine receptors. Although these agents 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.[32]

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 (Prelone and others) 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 (Pulmicort and others) may have fewer adverse effects, though it's more costly.[5] 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.[5] This recommendation reflects the fact that purulent sputum is associated with bacteria in the lower respiratory tract.[5]

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During COPD exacerbations, the most common causative bacteria are *Haemophilus influenzae*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis*,[21] though atypical and resistant organisms may also be present. Specific antibiotic choice should be determined based on the GOLD guidelines and designed to minimize antibiotic resistance.[5] 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.[5] 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.[5]

OXYGEN THERAPY

During exacerbations, oxygen requirements typically increase, particularly in low cardiac output states such as hypotension or high metabolic states such as pneumonia. Consider oxygen use in patients with

hypoxemia, worsening hypercapnia, or acidosis.[5] Oxygen should be titrated to improve hypoxemia, with an arterial oxygen saturation (SaO_2) goal of 88% to 92% in patients without complications.[5, 33] This modest 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 sufficiently oxygenate tissues. However, a higher SaO_2 goal may be set for patients whose exacerbations are complicated by low cardiac output or a high metabolic state. 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 gases (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 speak, eat, and drink. Because oxygen delivery is more variable with the nasal cannula, measure ABGs more frequently in patients using this delivery method, based on the stability of 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.[5, 34] 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,[5, 34] or when severe dyspnea is accompanied by respiratory muscle fatigue or increased work of breathing.[5, 34] 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 or uncooperative, or whose mental state is altered. Likewise, patients who are at 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.

Research supports that patients commonly rely on self-taught self-management during exacerbations, but a large number of COPD exacerbations are unreported, suggesting that clinicians need to provide more comprehensive patient education.

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.[5] 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, a 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.[5, 35]

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 in internal diameter) are used to minimize airflow resistance. Use of 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, keep 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 maintaining the balance between respiratory workload and muscle strength.[36] Patients with COPD meet standard ventilator-weaning criteria less often than patients without COPD, but when they do, they are more likely to successfully wean than their counterparts without COPD.[37] Currently, there is little consensus on the best method by which to wean patients with COPD. Available options include pressure support, a T-piece, and bridging with noninvasive ventilation. In an improving patient, the general approach 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 in all patients experiencing COPD exacerbations. In patients not receiving inhaled steroids, the mucolytic medication N-acetylcysteine may reduce exacerbations through its antioxidant properties.[38] Although N-acetylcysteine is not formally recommended for secretion clearance, the GOLD guidelines weakly recommend its use, noting that further research is needed to define its role in COPD treatment.[5]

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 an exacerbation.

Table 2. Treatment Setting Considerations[5, 31]

Initial 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 exacerbations • 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, 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

COPD = chronic obstructive pulmonary disease; PaO_2 = partial pressure of oxygen in arterial blood.

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 on admission, ABGs or pulse oximetry should be measured prior to discharge.[5] Initiate measures to prevent future exacerbations before discharge.[5] Patients who were mobile before 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 on admission, a follow-up ABG or pulse oximetry reading should be performed within three months of discharge. Reinforce the measures to prevent future exacerbations that were initiated before discharge at all subsequent follow-up visits.[5]

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 degree 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.[5] For a comprehensive approach to help patients quit smoking, see the 2008 U.S. Department of Health and Human Services guidelines, *Treating Tobacco Use and Dependence*, found at www.ncbi.nlm.nih.gov/books/NBK12193.

Vaccinations. Like cigarette smoke, bacteria and viruses worsen airway inflammation, exacerbating disease. Appropriate vaccination use among patients with COPD is considered standard of care.[5] Annual influenza vaccinations reduce both disease exacerbations and death among patients with COPD.[39-41] 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.[39,42] 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.[39]

Pulmonary rehabilitation following a 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.[43]

Research supports that patients commonly rely on self-taught self-management during exacerbations,[18] but a large number of COPD exacerbations are unreported, suggesting that clinicians need to provide more comprehensive patient education.[44] A recent trial examining the effectiveness of a relatively simple COPD management plan found it reduced both hospitalizations and ED visits related to COPD exacerbations.[45] Teaching patients to recognize exacerbations and seek immediate intervention leads to prompt medical treatment and improved exacerbation outcomes.[46] For a comprehensive, easy-to-use, educational patient handout on COPD, see part one of this series, "An Evidence-Based Approach to COPD" (March 2012). ▼

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