

# Treatment of Acute Exacerbations of COPD

**Amy R. Blanchard, MD**

Assistant Professor of Medicine  
Director, Adult Cystic Fibrosis Center  
Medical College of Georgia  
Augusta, Georgia

*Chronic obstructive pulmonary disease (COPD), the fourth leading cause of death in the United States, is increasing worldwide and is projected to be the third leading cause of death in the United States by the year 2020 (1). It affects nearly 16 million Americans, and more than \$18 billion is spent annually on medications, physician visits, and hospitalizations. COPD is characterized by chronic airflow obstruction with episodic acute exacerbations, which result in increased morbidity and mortality. Patients hospitalized with exacerbations have an overall mortality rate of 3% to 4%, and up to 24% of patients requiring care in the intensive care unit die (2). Since forced expiratory volume in 1 second correlates closely with life expectancy and exacerbation rate, early diagnosis (through spirometric testing) and prevention may reduce acute exacerbations and health care costs.*

Despite the fact that exacerbations of chronic obstructive pulmonary disease (COPD) contribute significantly to increased morbidity and mortality and to rising health care costs, no standard definition or classification system exists. Clinical diagnosis is based on worsening dyspnea and increased sputum volume or purulence. An international group of respiratory physicians has proposed the following definition of COPD exacerbations (3):

- A sustained worsening of the patient's condition from the stable state and beyond normal day to day variations that is acute in onset and necessitates a change in regular medication in a patient with underlying COPD.

The group also proposed the following staging/classification system based on health care utilization:

- Mild: patient has an increased need for medication, which can be managed in patient's normal environment.
- Moderate: patient has an increased need for medication and feels the need to seek additional medical assistance.

- Severe: patient/caregiver recognizes deterioration in condition requiring hospitalization.

In 1987, Anthonisen and colleagues classified exacerbation severity based on presenting symptoms (**Table I**), ranging from severe (type III) to mild (type I) (4). Mild exacerbations may be treated on an outpatient basis, whereas moderate to severe flares may require hospitalization and/or intensive care unit (ICU) admission.

Most patients with COPD have 1 to 2 exacerbations per year; however, exacerbation frequency increases with COPD severity (5). Exacerbations may be precipitated by respiratory infection, environmental exposures, and comorbid conditions such as heart failure, systemic infection, and pulmonary thromboembolism. Predictors of treatment failure include a lower baseline forced expiratory volume in 1 second ( $FEV_1$ ), a history of previous relapses and need for continually more aggressive bronchodilator therapy, malnutrition, older age, and presence of comorbid conditions such as malignancy or congestive heart failure. Investigators for the Study to Understand

TABLE I.

## CLASSIFICATION OF ACUTE EXACERBATIONS OF COPD

Type III	All 3 cardinal symptoms*
Type II	2 of 3 cardinal symptoms
Type I	1 of 3 cardinal symptoms <i>and</i> 1 of the following: – Upper respiratory tract infection in the past 5 days – Fever without other apparent cause – Increased wheezing – Increased cough – Increase in respiratory rate or heart rate by 20% above baseline

\*Cardinal symptoms = (1) worsening dyspnea, (2) increase in sputum purulence, (3) increase in sputum volume. Adapted with permission from Anthonisen NR, Manfreda J, Warren CP, et al. Antibiotic therapy in exacerbations of chronic obstructive pulmonary disease. *Ann Intern Med.* 1987;106:196–204.

Prognoses and Preferences for Outcomes and Risks of Treatments (SUPPORT) reported 180-day mortality after acute exacerbations of COPD to be 33%, while 2-year mortality was 49% (6). Frequent exacerbations, especially in smokers, may result in acceleration of the progressive decline in lung function (7).

### KEY POINT

The most common etiologies of COPD are infections, ranging from viral respiratory tract infection to bacterial pneumonia.

## TREATMENT OF ACUTE EXACERBATIONS

### Antibiotics

Although a variety of nonspecific irritants can cause acute exacerbations, the most common etiologies are infections, ranging from viral respiratory tract infection to bacterial pneumonia. In a recent study, viruses (most commonly rhinovirus) were detected in 39.2% of COPD exacerbations (8). In contrast to healthy nonsmokers, the lower airways of COPD patients are often colonized with bacteria; thus, the presence of an organism on culture does not necessarily indicate bacterial infection. The 3 most common organisms cultured in

mild to moderate stages of the disease and implicated as etiologic agents are *Haemophilus influenzae*, *Moraxella catarrhalis*, and *Streptococcus pneumoniae*. Patients with severe disease or who have received long-term antibiotic therapy may be colonized with gram-negative enteric pathogens or with resistant organisms.

Routine sputum Gram stain and culture are not cost-effective in the outpatient setting, but may be beneficial in patients refractory to standard therapy or in whom broad-spectrum antibiotics have been used recently. A study by Stockley and colleagues (9) demonstrated that sputum color might be helpful in deciding who may benefit most from antibiotics in this era of emerging antimicrobial resistance. In this study, green sputum present during an exacerbation was 94.4% sensitive and 77% specific for a high bacterial load on sputum culture.

Standard treatment of COPD exacerbations has included empiric antibiotic therapy aimed at common pathogens. Anthonisen et al (4) showed the benefit of antibiotics in moderate and severe exacerbations, while a recent meta-analysis demonstrated the benefit of antibiotics compared with placebo (10). Relapse rates of up to 32% are reported in patients not treated with antibiotics compared with 19% in those receiving antibiotics (11).

Most patients, especially those with mild to moderate COPD, respond to treatment with the less expensive, older “first-line” antibiotics—amoxicillin, trimethoprim-sulfamethoxazole, erythromycin, or doxycycline. Patients with severe

underlying lung disease have a higher exacerbation rate, with more than half the treatment failures resulting in hospitalization (12). Some studies suggest that compared with first-line therapeutic agents, the use of “second-line” agents—amoxicillin/clavulanate, ciprofloxacin, or azithromycin—significantly reduces treatment failure rates and increases time between exacerbations, thus lowering the total cost for management of acute exacerbations (13). This may be due to the presence of resistant pathogens (*S pneumoniae*) and/or organisms such as *Enterobacteriaceae* and *Pseudomonas* sp, which have been demonstrated in patients with more severe COPD (14). The macrolides may also exert their efficacy through anti-inflammatory/anti-secretory actions (15). Factors associated with treatment failure include FEV<sub>1</sub> <35% predicted, frequent exacerbations, history of sinusitis or previous pneumonia, and use of supplemental oxygen or maintenance oral corticosteroids.

The antibiotic choice for COPD exacerbations should depend on severity of the underlying disease and exacerbation, sputum character, and local susceptibility patterns. Other desirable pharmacologic features include ease of administration, infrequent dosing, low incidence of side effects, and cost-effectiveness. There is little evidence regarding the appropriate duration of antibiotic therapy, but a 7- to 10-day course should be sufficient in the absence of pneumonia. Some of the newer agents can be given for even shorter courses (eg, azithromycin).

### Corticosteroids

Two large randomized and controlled trials have shown the benefit of systemic corticosteroids in the treatment of acute exacerbations of COPD (16, 17). The Systemic Corticosteroids in Chronic Obstructive Pulmonary Disease Exacerbations (SCCOPE) Trial (16) demonstrated the utility of systemic corticosteroids in patients hospitalized with exacerbations of COPD. High-dose systemic corticosteroids resulted in a shortened hospital stay (1 day), improved FEV<sub>1</sub> during the first 3 days of hospitalization, and also lowered the rate of treatment failure at 30 and 90 days compared with placebo. Davies et al (17) also reported improve-

ment with a 2-week course of oral prednisolone 30 mg, administered once daily. Treatment with systemic corticosteroids for more than 2 weeks provides no added benefit and increases the risk of adverse effects. In contrast, inhaled corticosteroids are of no benefit in the treatment of acute exacerbations of COPD (18).

### KEY POINT

**Acute exacerbations of COPD are treated by reducing airway obstruction from mucus, inflammation, and bronchoconstriction. Reducing irritant exposure, smoking cessation, and the use of inhaled bronchodilators may reduce the exacerbation rate and improve quality of life.**

### Bronchodilator Therapy

Airflow obstruction due to mucosal edema, inflammation, and bronchoconstriction is one of the primary pathophysiologic abnormalities in acute COPD exacerbations. Inhaled short-acting beta-2 agonists and anticholinergics are equally effective bronchodilators, and are superior to methylxanthines and older sympathomimetics (19). The American College of Chest Physicians and the American College of Physicians – American Society of Internal Medicine (ACP-ASIM) recommend that consideration be given to using an inhaled anticholinergic bronchodilator (ipratropium) first-line with the addition of a short-acting beta-2 agonist, if needed, because of the lower incidence of side effects with anticholinergics (19). Mode of delivery should be chosen on an individual patient basis, as nebulization of bronchodilators shows no distinct advantage over the use of a metered-dose inhaler (MDI) at equivalent doses in patients able to correctly use MDIs.

The role of aminophylline in the treatment of COPD exacerbation has remained controversial, given its narrow therapeutic range and high incidence of side effects. In more severe exacerbations, the use of oral or intravenous methylxanthines

(theophylline, aminophylline) may be considered for an additive bronchodilator effect or as an anti-inflammatory agent. Additionally, the methylxanthines may have a beneficial effect on diaphragmatic function. Theophylline levels should be monitored closely to avoid serious side effects.

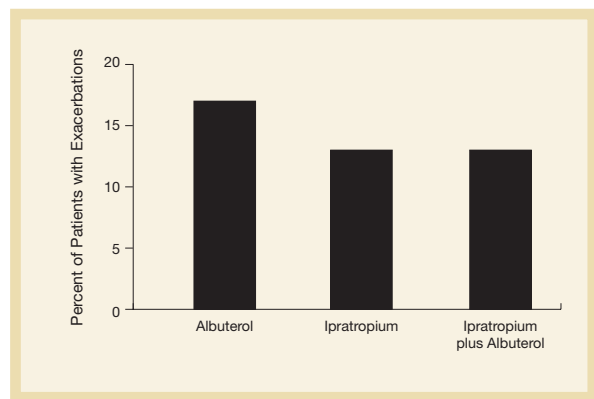
### Mucus Clearance Strategies

Adequate hydration may help to thin secretions. The use of expectorants such as guaifenesin or iodides to promote mucus clearance has not been shown to shorten the disease course but may improve symptoms. Chest physiotherapy may be ineffective and perhaps even detrimental (19). However, chest physiotherapy can benefit patients with thick tenacious sputum who produce >25 mL of mucus daily or who have atelectasis. Caution should be exercised, however, as patients may develop worsening ventilation/perfusion mismatch and gas exchange. A modified technique using chest percussion and encouragement to cough may be the most beneficial approach (20).

### PREVENTION

Some long-term maintenance bronchodilator regimens have been shown to reduce the exacerbation rate in patients with COPD. The combination of ipratropium and albuterol provides greater bronchodilation than either individual component without additional side effects (21), and may decrease exacerbation rate, hospitalization rate, and health care costs (22). Similarly, the use of a long-acting beta-2 agonist (salmeterol or formoterol), either alone or in combination with an anticholinergic bronchodilator, has been shown to reduce the exacerbation rate (**Figure**). Donohue et al (23) recently demonstrated that the once-daily anticholinergic tiotropium\* (not yet available in the United States) compared with twice-daily salmeterol produced superior bronchodilation as well as improvement in dyspnea and quality of life scores. Exacerbation rate was not studied but might be expected to be comparable to that of ipratropium.

Inhaled corticosteroids may benefit a subset of patients with reactive airways, and in moderate to



**Figure.** Anticholinergic bronchodilator therapy resulted in 30% fewer exacerbations over a 3-month study period in patients with COPD. Significant reductions were seen in both the ipratropium group and the ipratropium plus albuterol group. Reprinted with permission from Friedman M, Serby CW, Menjoge SS, et al. *Pharmacoeconomic evaluation of a combination of ipratropium plus albuterol compared with ipratropium alone and albuterol alone in COPD.* *Chest.* 1999;115:635–641.

severe disease may reduce exacerbation rates and/or severity. Current guidelines recommend inhaled corticosteroids in the following 2 classes of patients: patients demonstrating a symptomatic improvement and a documented spirometric response to inhaled glucocorticoids; and those with an FEV<sub>1</sub> <50% and repeated exacerbations requiring treatment with antibiotics or oral glucocorticoids (24).

### Modifiable Risk Factors

Tobacco smoking is the primary cause of COPD, and smoking cessation can clearly alter the natural history of the disease. In addition, education on proper bronchodilator technique, influenza vaccination, and pulmonary rehabilitation are preventive measures that may reduce exacerbation rates (25). The pneumococcal vaccine is recommended, although data supporting its use are not as strong as with the influenza vaccine.

A study examining the prevalence of potentially modifiable risk factors in hospitalized patients found that 28% had not received the influenza vaccine; 86% were not enrolled in pulmonary rehabilitation programs; 28% did not adhere to long-term oxygen therapy; 43% demonstrated poor inhaler technique; and 47% were either

\*Not FDA approved. Application in process.

TABLE II.

## INDICATIONS FOR HOSPITAL ADMISSION FOR ACUTE EXACERBATIONS OF COPD

- Marked increase in intensity of symptoms
- Severe underlying COPD
- Onset of new physical signs (eg, cyanosis, edema)
- Failure to respond to initial medical management
- Significant comorbidities
- Newly occurring arrhythmias
- Older age
- Insufficient home support

Adapted with permission from Pauwels RA, Buist AS, Calverley PM, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. NHLBI/WHO Global Initiative for Chronic Obstructive Lung Disease (GOLD) Workshop summary. *Am J Respir Crit Care Med.* 2001;163:1256–1276.

current smokers or exposed to passive smoke (26). These areas represent targets for intervention, education, and prevention.

### Acute Respiratory Failure in COPD Exacerbation

In patients with COPD exacerbation, hypercapnic (ventilatory) failure is more common than hypoxemic respiratory failure; however, both may occur. Treatment is aimed at correcting physiologic abnormalities, relieving airflow obstruction, reversing precipitating factors, and preventing or reversing complications. Indications for hospitalization are listed in **Table II**.

Acute respiratory failure is manifested by a  $\text{PaO}_2 < 50$  on room air and/or a  $\text{PCO}_2 > 50$  with  $\text{pH} < 7.35$ . Oxygen is the cornerstone of therapy in hypoxemic patients. Supplemental oxygen, especially if excessive, may result in hypercapnia due to a number of factors. Fortunately only a minority of COPD patients retain  $\text{CO}_2$  in response to oxygen. Arterial blood gases should be monitored in patients given supplemental oxygen for acute exacerbation. Goals are to relieve severe hypoxemia and avoid significant worsening of respiratory acidosis.

Indications for mechanical ventilation (MV) in patients with exacerbations of COPD include labored breathing with respiratory rates  $> 30$  breaths/minute, moderate to severe respiratory aci-

dosis, decreased level of consciousness, respiratory arrest, and complicating comorbid conditions (eg, shock, sepsis, metabolic abnormalities). Ventilatory support can be provided with noninvasive positive-pressure ventilation (NIPPV), administered via a tight-fitting mask; or invasively via endotracheal tube or tracheostomy. NIPPV may reduce the need for intubation, mortality, and length of hospital stay (27,28). NIPPV should not be used in patients with respiratory arrest, impaired mental status, copious secretions or in those at high risk for aspiration.

Invasive MV is indicated in patients who fail NIPPV and in those with obtundation, inability to clear copious secretions, life-threatening acidosis, or cardiovascular instability. Greater degrees of respiratory acidosis ( $\text{pH} < 7.25$ ) at the time of presentation represent increased likelihood of intubation. The decision to initiate MV should depend on the patient's wishes, baseline functional status, and reversibility of the precipitating event.

Predictors of poor outcome in mechanically ventilated COPD patients include the presence of malignancy, multilobar pneumonia, increased non-respiratory APACHE II (Acute Physiology and Chronic Health Evaluation) score, and the need for MV for  $> 72$  hours. Previous episodes of MV seem to offer only a short-term survival benefit (29). The negative impact of prolonged ( $> 72$  hours) MV may help explain the benefits seen with the use of

NIPPV to allow for early extubation in COPD. If NIPPV can be used comfortably by patients, it has been shown to shorten weaning time, decrease ICU and hospital length of stay, and improve 60-day survival (30).

## SUMMARY

Acute exacerbations of COPD represent a major source of morbidity and mortality, with greatly increased health care costs and reduced quality of life. Treatment is aimed at reducing airway obstruction caused by mucus, inflammation, and bronchoconstriction. Reducing irritant exposure, smoking cessation, and the use of inhaled bronchodilators are interventions that may reduce the exacerbation rate and improve quality of life.

The combination of an intensified bronchodilator regimen and antibiotics (especially in patients with purulent sputum), and/or addition of oral corticosteroids, may shorten the duration and severity of exacerbations and reduce costs associated with hospitalization. The use of NIPPV in appropriate candidates with acute respiratory failure may avoid the need for invasive MV and improve outcomes. Spirometric testing of at-risk individuals will help identify patients early in the disease course and allow timely intervention to slow disease progression.

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## Dialogue Box

### ADVISORY BOARD

**What sputum characteristic do you find most valuable in treating a COPD exacerbation?**

#### BLANCHARD

The color of a patient's sputum is a useful piece of information. In fact, in one study, the presence of green sputum was found to be 94% sensitive and > 70% specific for a high bacterial load.

Thus, if a patient reports green sputum, I'm inclined to treat with an antibiotic. Conversely, in a patient who reports mucoid sputum and is not producing copious amounts of it, an antibiotic would likely be of less value.

### ADVISORY BOARD

**Which antibiotic do you favor in patients who report their sputum turning green?**

#### BLANCHARD

In most patients, I'd prescribe a first-line antibiotic such as doxycycline. For the patient who has taken multiple courses of antibiotics in the past or who is receiving chronic steroids, I would likely choose a second-line antibiotic, such as a cephalosporin. For patients with whom compliance is a concern, I'd probably select one that entails a brief course of therapy, such as azithromycin, or one of the fluoroquinolones, such as levofloxacin.

### ADVISORY BOARD

**Should COPD patients with moderate to severe exacerbations be initially managed with the same emergent treatment regimen typically employed in status asthmaticus?**



## Dialogue Box

### BLANCHARD

These patients should be treated very much like asthmatics when they enter the emergency room and should be immediately started on aggressive bronchodilator treatment as well as intravenous steroids to prevent worsening of their symptoms.

### ADVISORY BOARD

**What dose of steroids do you use in the hospitalized patient with an exacerbation?**

### BLANCHARD

I typically begin with intravenous methylprednisolone. I start at an initial dosage of 125 mg followed by 60 mg every 6 hours.

### ADVISORY BOARD

**What is your dosing regimen for COPD patients managed on an ambulatory basis?**

### BLANCHARD

I would treat outpatients with prednisone at a dosage of 30 to 40 mg a day for 7 to 10 days, at which point I would stop the steroid. Although many clinicians choose to do so, tapering the dose after a 10-day course is really not necessary.

### ADVISORY BOARD

**Is there any advantage to splitting the dosage?**

### BLANCHARD

No, I give it all in one dose.

### ADVISORY BOARD

**Are inhaled corticosteroids of any value in patients experiencing an exacerbation?**

### BLANCHARD

Inhaled steroids have no role in the acute management of exacerbations. They are of value in

patients with severe COPD and frequent exacerbations where inhaled steroids have been shown to reduce symptoms and exacerbations.

### ADVISORY BOARD

**What role do mucolytic agents play in the treatment of COPD exacerbations?**

### BLANCHARD

Although the GOLD guidelines found little evidence that mucolytic agents shortened the course of exacerbations or provided any benefit, they do seem to improve patient symptoms; thus, I use them in selected patients. Among the available mucolytics, N-acetyl cysteine has been shown to reduce mucus production, but enthusiasm for its use has been tempered by the finding that it can cause bronchospasm.

### ADVISORY BOARD

**What role does hydration play?**

### BLANCHARD

Patients with exacerbations tend to be tachypneic and, as a result, have drying of their secretions. Such patients should be advised to drink plenty of fluids. For those who are hospitalized, intravenous hydration should be provided to enable them to more effectively clear their secretions by thinning them out.

### ADVISORY BOARD

**Is aerosolized hydration of value?**

### BLANCHARD

Not particularly. Although hypertonic saline can improve expectoration, it can also cause bronchospasm, and in patients who are already sick, you don't want to administer anything that might cause or worsen bronchospasm.



## Dialogue Box

### ADVISORY BOARD

#### What about physical therapy?

#### BLANCHARD

Physical therapy is important. We try to get our patients involved in at least bedside physical therapy within 48 hours of admission. Many of these patients have been homebound for weeks with worsening dyspnea and reduced activity, so by the time they are hospitalized they tend to be chronically debilitated with muscle wasting. It is important to get these patients mobilized as soon as possible. Not to be overlooked is that physical activity will help them clear their secretions as well. For patients with a lot of sputum production, chest percussion coupled with encouragement to cough and in some patients, the use of high-frequency percussion vests to facilitate cough can also play an important role. We generally don't order postural drainage because of its potential for worsening gas exchange.

### ADVISORY BOARD

#### What has been your experience with noninvasive positive pressure ventilation?

#### BLANCHARD

Although noninvasive ventilation with a full-face mask has not been shown to be beneficial in patients with chronic, stable COPD, it may be of value in reducing the need for intubation and mechanical ventilation as well as for facilitating extubation. Drawbacks of noninvasive ventilation include the amount of time required to get the proper mask fit and the fact that a patient can become claustrophobic and uncomfortable with a large face mask. Another drawback is that it is a physician-intensive and respiratory therapist-intensive procedure. It is absolutely contraindicated in patients who are obtunded or unable to clear their secretions.