Chapter 70: Chronic Obstructive Pulmonary Disease

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FIGURE 70-1.

INTRODUCTION AND EPIDEMIOLOGY

Chronic obstructive pulmonary disease (COPD) is characterized by persistent airflow limitation that is generally progressive and associated with an abnormal inflammatory response to noxious particles or gases.[1,2,3,4,5,6] COPD has two main forms: *chronic bronchitis*, defined in clinical terms, and *emphysema*, defined in terms of anatomic pathology. This traditional categorization is often indistinct, limiting the clinical utility of the definitions.[2,3,4,5,6] **Chronic bronchitis** is the presence of chronic productive cough for 3 months in each of 2 successive years, where other causes of chronic cough have been excluded.[2,3,4,5,6] **Emphysema** results from destruction of bronchioles and alveoli. The World Health Organization's **Global Initiative for Chronic Obstructive Lung Disease** definition of COPD encompasses chronic bronchitis, emphysema, bronchiectasis, and asthma, and acknowledges that most patients have a combination of the different diseases.

COPD accounted for 715,000 U.S. hospitalizations in 2010,[7] with $49.9 billion estimated as the cost for care.[7] The prevalence of COPD in women has doubled in the past few decades, and women now account for >50% of COPD-related deaths; the prevalence has remained stable in men.[8] The prevalence of COPD is highest in those countries that have the greatest cigarette use.

CHRONICALLY COMPENSATED CHRONIC OBSTRUCTIVE PULMONARY DISEASE

PATHOPHYSIOLOGY

Although tobacco smoke is the major risk factor for developing COPD, only 15% of smokers will develop COPD. Occupational dust, chemical exposure, and air pollution are other risk factors for COPD. α_1-Antitrypsin deficiency accounts for <1% of COPD patients.

Irritants, notably tobacco smoke and air pollutants, trigger an increase in inflammatory cells in the airways, lung interstitium, and alveoli. Proteases eventually break down lung parenchyma and stimulate mucus secretion. Mucus-secreting cells replace cells that normally secrete surfactant and protease inhibitors. These changes result in a loss of elastic recoil, narrowing, and collapse of the smaller airways. Mucous stasis and bacterial colonization develop in the bronchi. The earliest objective changes in the evolution of COPD are clinically imperceptible; these early changes are small increases in peripheral airway resistance or lung compliance. Because dyspnea and hypersecretion often progress insidiously, it may take decades before COPD becomes clinically evident. The Global Initiative for Chronic Obstructive Lung Disease guidelines are helpful for the early diagnosis and treatment of COPD (**Table 70-1**),[6] although there is only a weak correlation between forced expiratory volume in 1 second (FEV_1), symptoms, and health-related quality of life.[6]

TABLE 70-1

Classification of COPD Severity[2,3,4,5,6]

Stage	In Patients with FEV_1/FVC <0.7:
Mild COPD	FEV_1 ≥80% predicted
Moderate COPD	FEV_1 between 50% and 79% predicted
Severe COPD	FEV_1 between 30% and 49% predicted
Very severe COPD	FEV_1 <30% predicted

Abbreviations: COPD = chronic obstructive pulmonary disease; FEV_1 = forced expiratory volume in 1 second; FVC = forced vital capacity.

The central element of chronic lower airway obstruction is impedance to expiratory airflow due to increased resistance or decreased caliber of the small bronchi and bronchioles. Airflow obstruction results from a combination of airway secretions, mucosal edema, bronchospasm, and bronchoconstriction. Exaggerated airway resistance reduces total minute ventilation and increases respiratory work.

In emphysema, alveolar and capillary surfaces are distorted or destroyed, resulting in alveolar hypoventilation and ventilation–perfusion mismatch. The result is hypoxemia and hypercarbia. Sleep may blunt the ventilatory response to hypercarbia. The right ventricle hypertrophies and dilates, resulting in pulmonary hypertension and right ventricular failure. Right ventricular pressure overload is associated with atrial and ventricular arrhythmias. (See chapters 57, "Systemic Hypertension and chapter 58, "Pulmonary Hypertension.")

CLINICAL FEATURES

The hallmark symptoms are chronic and progressive dyspnea, cough, and sputum production; these may vary from day to day.[2,3,4,5,6] Minor hemoptysis is frequent, especially in chronic bronchitis and bronchiectasis, although it may herald lung carcinoma. Physical findings may include tachypnea, accessory respiratory muscle use, or pursed-lip exhalation. Lower airway obstruction causes expiratory wheezing, especially during maximum forced exhalation, and prolongation of the expiratory time. Patients with chronic bronchitis exhibit coarse crackles as uncleared secretions move about the central airways. In patients with emphysematous disease, there is expansion of the thorax, impeded diaphragmatic motion, and global diminution of breath sounds. Poor dietary intake and excessive caloric expenditure for the work of breathing cause weight loss, notably in emphysema. In the early stages, arterial blood gas measurements reveal mild to moderate hypoxemia without hypercapnia.

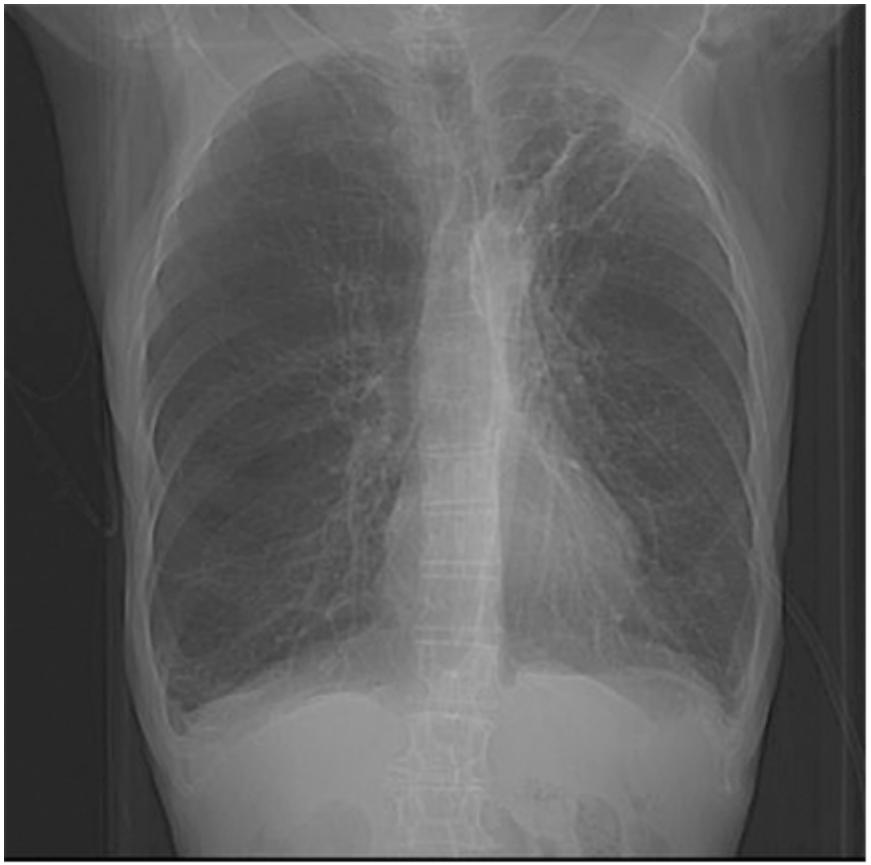
As COPD advances, especially when the FEV_1 falls below 1 L, hypoxemia becomes more severe and hypercapnia develops. Arterial oxygenation worsens during acute exacerbations, exercise, and sleep. Clinical signs of severe COPD include facial vascular engorgement from secondary polycythemia, and tremor, somnolence, and confusion from hypercarbia. Right heart failure may occur and be seen as edema or ascites, and the signs are often disguised or underestimated by the seemingly more overwhelming signs of respiratory disease. If concomitant left heart failure exists, the cardiac auscultatory findings may be overshadowed by the pulmonary inflation abnormalities of COPD.

DIAGNOSIS

The diagnosis of chronic, compensated COPD is confirmed by spirometry: a postbronchodilator FEV_1 of <80% predicted, and a ratio of FEV_1 to forced vital capacity of <0.7.[6] Once the disease progresses, the percentage of predicted FEV_1 is a better measure of disease severity.[2,3,4,5,6]

Chronic bronchitis is not radiographically apparent unless bronchiectasis is present. In emphysema, radiographs show hyperaeration, seen as increased anteroposterior chest diameter, flattened diaphragms, increased parenchymal lucency, and attenuation of pulmonary arterial vascular shadows (**Figure 70-1**).

FIGURE 70-1.
Posteroanterior chest radiograph in a patient with chronic obstructive pulmonary disease.



Source: J.E. Tintinalli, J.S. Stapczynski, O.J. Ma, D.M. Yealy, G.D. Meckler, D.M. Cline: Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8th Edition
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Distinguishing acute heart failure from COPD is difficult. A B-type natriuretic peptide level <100 picograms/mL supports a diagnosis of COPD; levels >500 picograms/mL have a sensitivity of 80% and positive predictive value of 47% for acute heart failure (see chapter 62, "Respiratory Distress").[9] The ECG detects dysrhythmias or ischemia but does not accurately assess the severity of pulmonary hypertension or right ventricular dysfunction.

TREATMENT

Treatment for chronic compensated COPD includes oxygen, pharmacotherapy, measures to decrease mucus secretion, smoking cessation, and pulmonary rehabilitation.

Oxygen

Long-term oxygen therapy reduces COPD mortality. The goal of long-term oxygen therapy is to increase the baseline partial pressure of arterial oxygen (PaO_2) to ≥60 mm Hg or the arterial oxygen saturation (SaO_2) to ≥90% at rest. Criteria for long-term oxygen therapy are a PaO_2 ≤55 mm Hg, a SaO_2 ≤88%, or a PaO_2 between 56 and 59 mm Hg when pulmonary hypertension, cor pulmonale (sustained right ventricular failure), or polycythemia is present.[6]

Pharmacotherapy

Pharmacotherapy does not alter disease progression but provides symptomatic relief, controls exacerbations, improves quality of life, and improves exercise performance.[10] Inhaled long-acting β_2-agonists are preferred over short-acting formulations, coupled with anticholinergics. Combining bronchodilators with different mechanisms and duration of action may increase bronchodilation without increasing side effects.[11] Combination inhalers of short-acting β_2-agonists with anticholinergic agents include fenoterol/ipratropium and salbutamol/ipratropium. Long-acting inhaled β_2-agonists, such as salmeterol, formoterol, olodaterol, and indacaterol, are used on a regular basis, adding short-acting inhaled β_2-agonists, usually albuterol, as needed. Anticholinergic agents cause bronchodilation by blocking the effect of acetylcholine on muscarinic-3 receptors. Long-acting anticholinergic agents, such as tiotropium, aclidinium, and glycopyrronium, are preferred over short-acting agents, such as ipratropium bromide or oxitropium bromide.[12,13] Bronchodilators often only chronically improve FEV_1 by 10%.

Experts do not recommend long-term systemic corticosteroid therapy for all COPD patients,[14] because only about 20% to 30% improve. Short-term steroid use (days) aids in treating exacerbations. Regular treatment with inhaled corticosteroids is indicated for patients with a documented spirometric response to inhaled corticosteroids, those with an FEV_1 of <50%, or those with predicted and recurrent exacerbations requiring antibiotic treatment or systemic corticosteroids.[6] Long-term treatment with inhaled corticosteroids added to long-acting bronchodilators is recommended for patients at high risk of exacerbation. Combination inhalers with long-acting β_2-agonists plus corticosteroids include formoterol/budesonide, formoterol/mometasone, salmeterol/fluticasone, and vilanterol/fluticasone.[6]

Theophylline is relegated to an adjunct COPD therapy.[15] Theophylline inhibits phosphodiesterase and has an anti-inflammatory effect. It is not commonly used, but can be used in some patients not well controlled with inhaled corticosteroids or long-acting β_2-agonists. Although retrospective studies suggest that statins decrease the rate and severity of exacerbations, rate of hospitalization, and mortality, a large

prospective trial failed to demonstrate benefit of daily simvastatin over placebo.[16] Daily azithromycin may decrease acute exacerbations in older patients and those with milder Global Initiative for Chronic Obstructive Lung Disease staging.[17]

Secretion Mobilization

Respiratory secretions are kept mobilized by generous oral fluid intake and room humidification. Limit the use of antihistamines, antitussives, mucolytics, and decongestants. Expectorants are not of clear benefit.

Smoking Cessation and Pulmonary Rehabilitation

Smoking cessation is the only intervention that can reduce both the rate of decline in lung function[6] and mortality from respiratory causes.[2,3,4,5,6] The ED is a site to attempt smoking cessation interventions.[18] A combination of nicotine replacement therapy or medications and behavioral interventions can assist patients with smoking cessation, especially with referral to a program.[19]

Pulmonary rehabilitation can improve exercise capacity and quality of life and is recommended in patients with moderate to severe COPD. Pneumococcal vaccination and influenza vaccination are key to dampen acute infections.[6]

ACUTE EXACERBATIONS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Acute exacerbations of COPD are characterized by worsening of respiratory symptoms beyond normal day-to-day variations[20] and are usually triggered by an infection or respiratory irritant. More than 75% of patients with acute exacerbations have evidence of viral or bacterial infection, with up to half specifically due to bacteria.[21,22] Other important triggers for exacerbations are hypoxia, cold weather,[23] β-blockers, narcotics, or sedative-hypnotic agents. The final common pathway for an exacerbation is the release of inflammatory mediators that result in bronchoconstriction, pulmonary vasoconstriction, and mucus hypersecretion. The work of breathing increases due to higher airway resistance and lung hyperinflation. The oxygen demand of respiratory muscles increases, generating additional carbon dioxide and causing hypercapnia, resulting in further physiologic stress.[23] Acute exacerbations of COPD are primarily due to ventilation–perfusion mismatch rather than the expiratory airflow limitation seen with asthma exacerbations.[24] Supplemental oxygen increases blood oxygen concentrations and can help reverse pulmonary vasoconstriction.

CLINICAL FEATURES

The most life-threatening feature of an acute exacerbation is hypoxemia (arterial saturation <90%). Signs of hypoxemia include tachypnea, tachycardia, systemic hypertension, cyanosis, and a change in mental status. With increased work of breathing, carbon dioxide production increases; alveolar hypoventilation creates arterial carbon dioxide retention and respiratory acidosis.

The patient tries to overcome severe dyspnea and orthopnea by sitting in an up-and-forward position, using pursed-lip exhalation, and engaging accessory muscles to breathe. Pulsus paradoxus (a drop of >10 mm Hg in systolic blood pressure during respiratory cycles) may be noted during palpation of the pulse or during blood pressure recording. **Complications, such as pneumonia, pneumothorax, pulmonary embolism, or an acute abdomen, may exacerbate COPD. Other acute triggers include asthma, congestive heart failure, pneumonia, pulmonary embolism, tuberculosis, and metabolic disturbances.**

DIAGNOSIS

With the history, seek causes for exacerbation and triggers plus sputum changes; then assess oxygenation and acid-base status, and perform a physical examination.

Pulse oximetry may identify hypoxemia, and capnography may identify hypercarbia. Arterial blood gas analysis is the best tool in acute evaluation for assessing oxygenation, ventilation, and acid-base disturbances. Arterial blood gases clarify the severity of exacerbation and the probable clinical course. Respiratory failure is characterized by an arterial PaO_2 of <60 mm Hg or an arterial SaO_2 <90% in room air. Respiratory acidosis is present if the partial pressure of carbon dioxide (Pco_2) is >44 mm Hg. If the pH is <7.35, there is an acute and uncompensated component of respiratory or metabolic acidosis present.

In acute respiratory acidosis, the serum bicarbonate rises by 1 mEq/L for each 10-mm Hg increase in Pco_2, and the pH will change by 0.008 × (40 – Pco_2). In chronic respiratory acidosis, the bicarbonate rises by 3.5 mEq/L for each 10-mm Hg increase in Pco_2, and the pH will change by 0.03 × (40 – Pco_2) (Formulas 1 and 2). Changes outside of these ranges suggest an accompanying metabolic disorder (see chapters 15, "Acid-Base Disorders" and 62, "Respiratory Distress").

Frequently, patients with an acute COPD exacerbation are too dyspneic to perform bedside pulmonary function tests, and measurements are often inaccurate.[2,3,4,5,6] Similarly, physical examination and physician estimates of pulmonary function are inaccurate.[25]

Assessment of sputum includes questions about changes in volume and color, especially an increase in purulence. An increase in sputum volume and change in sputum color suggest a bacterial infection and the need for antibiotic therapy.[24,26] Sputum cultures usually contain mixed flora

and do not help guide ED antibiotic selection.[2,3,4,5,6]

Ancillary Studies

Radiographic abnormalities are common in COPD exacerbation and may identify the underlying cause of the exacerbation, such as pneumonia, or may identify an alternative diagnosis such as acute heart failure.[27]

The ECG can identify ischemia, acute myocardial infarction, cor pulmonale, and dysrhythmias. Measure levels in patients who take theophylline. Other tests, such as CBC, electrolytes, B-type natriuretic peptide, d-dimers, and CT angiography of the chest, are chosen based on clinical findings.

TREATMENT

The goals of treatment are to correct tissue oxygenation, alleviate reversible bronchospasm, and treat the underlying cause (Table 70-2). Factors that influence therapy in the ED include a patient's mental status changes; the degree of reversible bronchospasm; recent medication usage and assessment for drug toxicity; prior history of exacerbation courses, hospitalization, and intubation; presence of contraindications to any drug or drug class; and specific causes or complications from the exacerbation. Patients who do not respond as expected to standard therapy should prompt a reevaluation for other potentially life-threatening issues. See Table 70-3 for an overview of the differential diagnosis of COPD exacerbations.

TABLE 70-2

ED Management of COPD Exacerbations[2,3,4,5,6]

- Assess severity of symptoms
- Administer controlled oxygen
- Continuous cardiovascular status monitoring
- Perform arterial blood gas measurement after 20–30 min if arterial oxygen saturation remains <90% or if concerned about symptomatic hypercapnia
- Administer bronchodilators
  - β_2-Agonists and/or anticholinergic agents by nebulization or metered-dose inhaler with spacer
- Add oral or IV corticosteroids
- Consider antibiotics if increased sputum volume, change in sputum color, fever, or suspicion of infectious etiology of exacerbation
- Consider adding IV methylxanthine if above treatments do not improve symptoms
- Consider noninvasive mechanical ventilation
- Evaluation may include chest radiograph, CBC with differential, basic metabolic panel, ECG
- Address associated comorbidities

TABLE 70-3

Critical Differential Diagnosis of Chronic Obstructive Pulmonary Disease (COPD) Exacerbations[2,3,4,5,6]

Diagnosis	Clinical Features	Caveats
Asthma	Earlier onset Varying symptoms Family history Reversible airflow	Can coexist with COPD. Many patients diagnosed with asthma actually have COPD or mixed asthma-COPD
CHF	Presence of orthopnea (LR, 2.0) and dyspnea with exertion (LR, 1.3) slightly favors CHF Jugular venous distention, hepatojugular reflux, bibasilar rales Chest x-ray may show cardiomegaly or interstitial edema BNP <100 picograms/mL not likely to be CHF; BNP >500 picograms/mL more likely to be CHF	Can coexist with COPD. Shares some historical elements also found in COPD. Multiple conditions can falsely elevate or decrease the BNP level.
PE	Risk factors include older age, recent surgery or trauma, prior venous thromboembolic disease, hereditary thrombophilia, malignancy, smoking, and use of medications containing estrogen Patients with intermediate to high pretest probability may require further testing, such as CT angiography; d-dimer may be useful in ruling out PE in low-risk patients	20%–25% of patients with a severe COPD exacerbation with an unclear trigger have a PE. Triad of PE (pleuritic chest pain, dyspnea, tachycardia, and hypoxemia) unusual.
ACS	Obtain ECG or troponin in those with chest pain or dyspnea and risk factors for ACS	Dyspnea may be the primary complaint in patients with ACS.
Pneumothorax	Obtain chest x-ray, US, or CT	COPD is a risk factor for spontaneous pneumothorax.

Diagnosis	Clinical Features	Caveats
Pneumonia	Obtain chest x-ray	Frequently coexists with a COPD exacerbation.

Abbreviations: ACS = acute coronary syndrome; BNP = B-type natriuretic peptide; CHF = congestive heart failure; LR, likelihood ratio; PE = pulmonary embolism.

Oxygen

Administer oxygen to raise the PaO_2 above 60 mm Hg or the SaO_2 above 90%. Use any of the following devices: standard dual-prong nasal cannula, simple facemask, Venturi mask, or nonrebreathing mask with reservoir and one-way valve. Because oxygen administration may produce hypercapnia, arterial blood gases and/or continuous end-tidal carbon dioxide and oxygen saturation monitoring with venous blood gases will allow optimal assessment of the P_{CO_2} and acid-base status. It may take 20 to 30 minutes from administration of supplemental oxygen for improvement to occur. If adequate oxygenation is not achieved or respiratory acidosis develops, assisted ventilation may be required.

β_2-Adrenergic Agonists

Short-acting β_2-agonists and anticholinergic agents are first-line therapies in the management of acute, severe COPD.[2,3,4,5,6] Both lead to improved clinical outcomes and shorter ED lengths of stay, especially when used together.[2,3,4,5,6] Aerosolized forms, using nebulizer or metered-dose inhalers, deliver drug to the target area optimally and minimize systemic toxicity. β_2-Agonists are best given every 30 to 60 minutes if tolerated.[2,3,4,5,6] Nebulized aerosols every 20 minutes may result in more rapid improvement of FEV_1, but more frequent side effects,[28] including tremor, anxiety, and palpitations. Continuous cardiac monitoring is helpful, especially for patients with heart disease.

Anticholinergics

Some guidelines favor β_2-agonists as a first-line therapy, whereas others favor anticholinergic agents. Ipratropium bromide given as a single dose by metered-dose inhaler with a spacer or as an inhalant solution by nebulization (0.5 milligram or 2.5 mL of the 0.02% inhalant solution) is the

usual agent of choice, although aerosolized glycopyrrolate (2 milligrams in 10 mL of saline) is also effective. Side effects are minimal and appear to be limited to dry mouth and an occasional metallic taste.

Evidence regarding the efficacy of the combination of a β_2-adrenergic agent and an anticholinergic agent compared with a single agent alone is conflicting, although many physicians favor using this combination initially and some favor using it if the response to maximal doses of a single bronchodilator is poor. Long-acting inhaled anticholinergics, such as tiotropium, aclidinium, and glycopyrronium, are not used for the acute management of COPD.[2,3,4,5,6]

Corticosteroids

The use of a short course (5 to 7 days) of systemic steroids improves lung function and hypoxemia and shortens recovery time in acute COPD exacerbations.[29] Use of corticosteroids in the ED does not affect the rate of hospitalization but does decrease the rate of return visits. The lack of effect on hospitalization rates is likely due to the approximately 6-hour delay before onset of action. There appears to be no clear benefit from a dose >40 to 60 milligrams of oral prednisone daily.[29] Hyperglycemia is the most common adverse effect.

Antibiotics

Prescribe antibiotics if there is evidence of infection, such as change in volume of sputum and increased purulence of sputum.[26] **Choose agents directed at the most common pathogens associated with COPD exacerbation: *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*.** There is no specific agent shown to be superior.[24,26] Initial antibiotics include macrolides (azithromycin), tetracyclines (doxycycline), or amoxicillin with or without clavulanic acid. There is little evidence regarding the duration of treatment, which ranges from 3 to 14 days.

Methylxanthines

Methylxanthines, such as theophylline (oral) and aminophylline (parenteral), inhibit phosphodiesterases and may enhance respiration in two ways: by improving the mechanics of breathing (at the smooth muscle and diaphragm) and through an anti-inflammatory effect that happens at lower doses than used previously for bronchodilation and potentiating exogenous steroid effects. Data are conflicting on the value in acute COPD care, and these agents may induce nausea and vomiting.[30,31] The therapeutic index is narrow, so drug levels must be monitored. Methylxanthines (aminophylline 3 to 5 milligrams/kg IV over 20 minutes) are third-line options after inhaled therapies and steroids and when first-line therapies fail.

Noninvasive Ventilation

Indications and relative contraindications of noninvasive ventilation are listed in **Table 70-4** Noninvasive ventilation can be delivered by nasal mask, full facemask, or mouthpiece. Patients with respiratory failure who receive noninvasive ventilation have better outcomes in terms of intubation rates, short-term mortality rates, symptomatic improvement, and length of hospitalization.[32] Disadvantages of noninvasive positive-pressure ventilation include slower correction of gas exchange abnormalities, risk of aspiration, inability to control airway secretions directly, and possible complications of gastric distention and skin necrosis. **Contraindications to noninvasive ventilation include an uncooperative or obtunded patient, inability of the patient to clear airway secretions, hemodynamic instability, respiratory arrest, recent facial or gastroesophageal surgery, burns, poor mask fit, or extreme obesity**. Noninvasive ventilation methods are discussed in detail elsewhere (see chapter 28, Noninvasive Airway Management).

TABLE 70-4

Indications and Relative Contraindications for Noninvasive Ventilation[2,3,4,5,6]

Selection criteria	Acidosis (pH <7.36)/hypercapnia ($Paco_2$ >50 mm Hg)/oxygenation deficit (Pao_2 <60 mm Hg or Sao_2 <90%) Severe dyspnea with clinical signs like respiratory muscle fatigue or increased work of breathing
Exclusion criteria (any)	Respiratory arrest Cardiovascular instability (hypotension, arrhythmias, myocardial infarction) Change in mental status; uncooperative patient High aspiration risk Viscous or copious secretions Recent facial or gastroesophageal surgery Craniofacial trauma Fixed nasopharyngeal abnormalities Burns Extreme obesity

Abbreviations: $Paco_2$ = partial pressure of arterial carbon dioxide; Pao_2 = partial pressure of arterial oxygen; SaO_2 = arterial oxygen saturation.

All patients receiving noninvasive positive-pressure ventilation require continuous cardiorespiratory monitoring and frequent reassessment for setting changes and for tolerance of therapy.

Assisted Ventilation

Mechanical ventilation is indicated if there is evidence of respiratory muscle fatigue, worsening respiratory acidosis, deteriorating mental status, or refractory hypoxemia (Table 70-5). The goals of assisted ventilation are to rest ventilatory muscles and to restore adequate gas exchange. After endotracheal intubation, the methods most commonly used are assist control ventilation, pressure support ventilation, or pressure support ventilation in combination with intermittent mandatory ventilation. Adverse events associated with invasive ventilation include pneumonia, barotrauma, and inability to wean the COPD patient from the ventilator.

TABLE 70-5

Indications for Intubation with Mechanical Ventilation[2,3,4,5,6]

Unable to tolerate noninvasive ventilation (NIV) or NIV failure
Respiratory or cardiac arrest
Respiratory failure
Decreased consciousness or increased agitation
Massive aspiration
Persistent inability to remove respiratory secretions
Hypotension
Persistent hypoxemia despite optimal respiratory treatment
Hemodynamic instability

Current evidence does not support the use of a mixture of helium and oxygen or magnesium in the treatment of an acute COPD exacerbation.

DISPOSITION AND FOLLOW-UP

Patients who fail to improve, those who deteriorate despite medical therapy, those with significant comorbidity, or those without an intact social support system are admitted. Objective criteria regarding hospital admission, observation unit stay, and ED discharge are lacking. The Global

Initiative for Chronic Obstructive Lung Disease guidelines help guide the ED disposition decision-making process (Tables 70-6 and 70-7). Select patients without respiratory failure may avoid hospitalization with nurse-administered home care ("hospital at home care").[33] After ED discharge, 25% to 43% of patients with COPD exacerbation show ongoing or relapse of symptoms.[34,35,36]

TABLE 70-6

Indications for Hospital Admission[2,3,4,5,6]

Marked increase in intensity of symptoms, such as sudden development of resting dyspnea or inability to walk from room to room
Failure of exacerbation to respond to initial medical management
Significant comorbidities
Newly occurring dysrhythmias, heart failure
Frequent exacerbations and/or frequent relapse after ED treatment
Older age
Insufficient home support

TABLE 70-7

Indications for Intensive Care Admission[2,3,4,5,6]

Severe dyspnea that responds inadequately to initial emergency therapy
Respiratory or ventilatory failure (current or impending) despite supplemental oxygen and noninvasive positive-pressure ventilation
Decreasing level of consciousness or increasing confusion or agitation
Hemodynamic instability
Presence of comorbidities leading to end-organ failure

The following are associated with a higher risk for relapse within 2 weeks after an ED visit: five or more ED or clinic visits in the past year, the amount of activity limitation (based on a 4-point scale), the initial respiratory rate (for each 5 breaths/min over 16 breaths/min), and use of oral corticosteroids before arrival in the ED.[34,35,36]

If discharging from the ED or observation unit, arrange the following: (1) a supply of home oxygen, if needed; (2) adequate and appropriate bronchodilator treatment (usually a metered-dose inhaler with a spacer *and* teaching; nebulized therapies are reserved for those who cannot use the metered-dose inhaler); (3) short course of oral corticosteroids[2,3,4,5,6]; and (4) a follow-up appointment with the primary care physician or pulmonologist, preferably within a week. Reassess inhaler technique, reinforce importance of completion of steroid therapy and antibiotics, if prescribed, and review management plan.

PRACTICE GUIDELINES

American Thoracic Society/European Respiratory Society guidelines—http://www.thoracic.org/clinical/copd-guidelines/resources/copddoc.pdf

Australian Lung Association Chronic Obstructive Pulmonary Disease checklist—http://www.copdx.org.au

Canadian Thoracic Society guidelines—http://www.respiratoryguidelines.ca/guideline/chronic-obstructive-pulmonary-disease#guidelines-and-standards

Global Initiative for Chronic Obstructive Lung Disease guidelines—http://www.goldcopd.org/guidelines-global-strategy-for-diagnosis-management.html

National Institute for Health and Care guidelines—http://www.nice.org.uk/guidance/CG101

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USEFUL WEB RESOURCES

American Lung Association—http://www.lung.org

American Thoracic Society/European Respiratory Society treatment guidelines—http://www.thoracic.org/statements/

Australian Lung Association Chronic Obstructive Pulmonary Disease checklist—http://www.copdx.org.au

Canadian Thoracic Society treatment guidelines—http://www.respiratoryguidelines.ca/guideline/chronic-obstructive-pulmonary-disease

Global Initiative for Chronic Obstructive Lung Disease—http://www.goldcopd.com

USEFUL WEB AND TELEPHONE RESOURCES FOR SMOKING CESSATION

American Lung Association Lung Helpline—1-800-LUNGUSA

European Network of Quit Lines—http://www.enqonline.org/public/aboutus.php

Foundation for a Smokefree America—http://www.TobaccoFree.org

Health Canada, Canadian Quit Lines—http://www.hc-sc.gc.ca/index-eng.php

U.S. Quit Lines—Smoking Quit Line English or Spanish—call within the U.S., Monday through Friday 9 a.m. to 4:30 p.m. local time: 1-877-44U-QUIT (1-877-448-7848); TTY: 1-800-332-8615; http://www.cancer.gov/cancertopics/tobacco/quittingtips

Silverchair