Asthma affects 7% to 10% of the elderly. Epidemiologic studies suggest that asthma is underdiagnosed and undertreated in all age groups. Part of the problem is that the transient nature of asthma allows many patients to tolerate intermittent respiratory symptoms before seeking medical care. Another important factor resulting in underdiagnosis of asthma is the sometimes nonspecific nature of the symptoms.

About half of cases of asthma develop before 10 years of age and another third before 40 years. The 2:1 male-to-female preponderance of asthma in childhood equalizes by 30 years of age.

The average asthmatic patient has 15 days of restricted activity each year and spends 5.8 days in bed. Approximately 2 million emergency department (ED) visits, 484,000 hospitalizations, and more than 4000 deaths per year are attributed to asthma. In the United States alone, the estimated direct and indirect cost of asthma in all age groups was $56 billion in 2007.

**PATHOPHYSIOLOGY**

All asthmatic patients have hyperresponsive airways that narrow when exposed to various stimuli: allergic, infectious, pharmacologic, environmental, occupational, exercise related, and emotional.

Allergic asthma occurs when inhaled allergens bind to immunoglobulin E molecules bound to mast cells in the lining of the tracheobronchial tree. During the early response, various mediators are released and cause greater vascular permeability, mucosal edema, and contraction of bronchial smooth muscle. A second wave of reaction, the late response, is seen hours to days later; it involves accumulation of inflammatory cells in the bronchial mucosa, thus perpetuating the reaction. The release of mediators and regulation of the inflammatory process in asthma are complex, redundant, and self-perpetuating.

Although several theories attempt to explain the pathophysiologic changes that occur in nonallergic asthma, none adequately explain all clinically observed phenomena. Research suggests that even patients without atopy have pathophysiology similar to that in atopic patients. Respiratory infections, particularly viral infections, may precipitate bronchospasm. Viruses cause mucosal inflammation and lower the firing threshold of the subepithelial vagal receptors, which results in enhanced airway reactivity that may last up to 8 weeks, even in nonasthmatic persons. Pharmacologic agents, such as aspirin and nonsteroidal antiinflammatory
compounds, coloring agents, and beta-blockers, also induce acute asthma. In addition, sulfating agents, which are used widely as food preservatives and antioxidants in pharmaceutical products, can exacerbate asthma.

A large variety of occupational dust and fumes may provoke acute airway obstruction. Patients with occupational asthma classically report a cyclic history; they are symptom free during weekends, vacations, and on arrival at work. Exercise may also stimulate an asthma attack. Exercise-induced bronchospasm is usually noted within 5 to 20 minutes after the completion of exercise and is related to thermal changes in the respiratory tree. Exercising in a cold, dry environment causes a more marked response than does exercising in a warm, humid environment. Finally, endocrine factors, such as variations in progesterone and estradiol levels, also influence asthma exacerbations, probably through modification of vagal efferent activity.

Most patients with asthma seem to display an exaggerated bronchoconstrictive response to a variety of exogenous and endogenous stimuli, and inflammation plays a key role. The final common pathway is as follows:

- Airway narrowing
- Bronchial wall edema
- Bronchial smooth muscle contraction
- Mucosal plugging
- Enhanced airway reactivity and remodeling of the airway wall, which results in increased airway resistance
- Decreased forced expiratory volumes and flow rates
- Lung hyperinflation
- Increased work of breathing
- Ventilation-perfusion mismatch

### PRESENTING SIGNS AND SYMPTOMS

When evaluated in the ED, many patients relay a history of asthma, but some do not. Patients with a severe asthma attack may be in obvious respiratory distress, with rapid breathing and loud wheezing, but patients with mild exacerbation may exhibit coughing and end-expiratory wheezing. The classic symptoms of asthma consist of the triad of dyspnea, wheezing, and coughing, but physical findings during an asthma exacerbation can be variable. Early symptoms include a sensation of chest constriction and coughing. As the exacerbation progresses, wheezing becomes apparent, expiration becomes prolonged, and use of the accessory respiratory muscles may become evident. Patients may sit upright or lean forward in an attempt to decrease the work of breathing. Use of the accessory muscles of inspiration indicates diaphragmatic fatigue, whereas the appearance of paradoxic respirations reflects impending ventilatory failure. Alteration in mental status heralds respiratory arrest.

### VARIATIONS IN PRESENTING SIGNS AND SYMPTOMS

Patients with asthma exacerbations may simply exhibit coughing or have a feeling of chest tightness. At the other end of the spectrum are patients with a “silent chest,” which reflects very severe airflow obstruction and air movement insufficient to promote a wheeze.

A subset of asthmatic patients experience a sudden onset of severe symptoms. These individuals tend to respond rapidly to treatment but appear to be at significant risk for a fatal outcome.

### DIFFERENTIAL DIAGNOSIS

Wheezing, coughing, and dyspnea may be caused by many common conditions, including pneumonia, bronchitis, croup, broncholithiasis, chronic obstructive pulmonary disease, congestive heart failure, pulmonary embolism, allergic reactions, and upper airway obstruction from edema or a foreign body. Less common conditions with similar symptoms are cystic fibrosis, hypersensitivity pneumonitis, carcinoid syndrome, and exposure to odors, dust, and gas. A careful history and physical examination should help differentiate asthma from these other conditions.

### DIAGNOSTIC TESTING

As for all patients who come to the ED for care, a directed history and physical examination should be performed. Key historical points should be elicited, such as the duration and onset of the current attack, identification of precipitating causes, type and amount of medications used before arrival at the ED, response to previous therapy, including current or previous use of corticosteroids, frequency of ED visits and hospitalizations, previous need for intubation or ventilation, history of concurrent medications and allergies, and history of concurrent medical problems. At some point during the patient’s ED stay, effort should be made to evaluate both the severity of the obstruction and the adequacy of ongoing asthma control (Table 48.1).

The physical examination should focus on observing respiratory effort and use of accessory muscles and listening for wheezing or other abnormal breath sounds and prolongation of the expiratory phase. Although wheezing results from movement of air through narrowed airways, the intensity of the wheeze may not correlate with the severity of airflow obstruction. Tachycardia and tachypnea are usually present in patients with acute asthma, but vital signs normalize very quickly as the airflow obstruction is relieved. Therefore, a normal heart rate and respiratory rate are not reliable indicators of the degree of relief from obstruction.

Bedside spirometry provides a rapid, objective assessment of patients and helps both indicate the effectiveness of and guide therapy. Sequential measurements assist emergency physicians in assessing the severity of the problem and determining the response to therapy. Although forced expiratory volume in 1 second (FEV1) and the peak expiratory flow (PEF) rate measure the extent of large airway obstruction, patient cooperation is essential for these tests to be reliable. When possible, management decisions should be guided by a patient’s personal best PEF rate or FEV1 value or, if unknown, a percentage of the predicted value in addition to other physiologic and historical factors.

Pulse oximetry is a useful and convenient method for accessing oxygenation and monitoring oxygen saturation during treatment. Analysis of arterial blood gases is not indicated in the majority of patients with mild to moderate asthma.
exacerbation, but it is helpful if there is concern for hypoventilation with carbon dioxide retention and respiratory acidosis. Patients with the latter problems almost always have clinical evidence of severe attacks or spirometry demonstrating PEF or FEV1 less than 25% of the predicted value. Practitioners should be aware that a normal or slightly elevated PaCO2 (e.g., 42 mm Hg or higher) indicates extreme airway obstruction and fatigue and may herald the onset of acute ventilatory failure.

Routine radiography is unnecessary but is indicated if the possibility of pneumothorax, pneumomediastinum, pneumonia, or other medical conditions is a concern. In up to one third of asthmatic patients requiring admission, an abnormality is demonstrated on chest radiographs.

A routine complete blood cell count is not indicated and would probably show modest leukocytosis secondary to the administration of β2-agonist therapy or corticosteroid treatment. In patients taking theophylline before ED evaluation, a serum theophylline level should be determined. A routine electrocardiogram is also unnecessary; electrocardiographic abnormalities noted include right ventricular strain, abnormal P waves, and nonspecific ST-T wave abnormalities, which resolve with treatment. Older patients, especially those with coexisting heart disease, should undergo cardiac monitoring during treatment. Asthma severity index scores have failed to predict outcome better than clinical judgment does.

Use of exhaled nitric oxide measurements and other serum and urine markers for detection of the severity of the asthma exacerbation is currently under investigation.

**TREATMENT**

The goal of treatment of acute asthma in the ED is to rapidly reverse the airflow obstruction with repetitive or continuous administration of inhaled β2-agonists, ensure adequate oxygenation, and relieve inflammation. The National Asthma Education and Prevention Program Expert Panel has developed guidelines for emergency treatment of asthma (Fig. 48.1), as have other organizations around the world. Prehospital treatment with oxygen and β2-agonists is usually initiated. The following types of medications have been shown to be effective for the treatment of acute asthma: β2-agonists, anticholinergics, and glucocorticoids (Table 48.2). Magnesium should be considered in patients with severe obstruction. Current evidence does not support the use of heliox (helium-oxygen mixture) or ketamine, even when the aforementioned medications fail to relieve bronchospasm. Mast cell–stabilizing agents, methylxanthines, and leukotriene modifiers are currently reserved for maintenance therapy only.

**PHARMACEUTICALS**

**β2-Agonist Agents**

β2-Agonists are the preferred initial rescue medications for acute bronchospasm. In addition to bronchodilation, these drugs inhibit the release of mediators and promote mucociliary clearance.

The most common side effect of β2-agonist drugs is skeletal muscle tremor. Patients may also experience nervousness, anxiety, insomnia, headache, hyperglycemia, palpitations, tachycardia, and hypertension. Despite earlier concerns about the potential cardiotoxicity of these agents, clinical experience has not revealed significant problems.
Fig. 48.1 Management of asthma exacerbations: emergency department- and hospital-based care. FEV₁, Forced expiratory volume in 1 second; PEF, peak expiratory flow; PEFR, peak expiratory flow rate. (Adapted from National Institutes of Health, National Heart, Lung and Blood Institute, National Asthma Education and Prevention Program. Expert Panel Report 3: guidelines for the diagnosis and management of asthma. NIH Publication No. 08-4051. Bethesda, MD: U.S. Department of Health and Human Services; August 2007.)
Table 48.2  Medications Used to Treat Asthma Exacerbations

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>ADULT DOSE</th>
<th>CHILD DOSE</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Short-Acting Inhaled β2-Agonists</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Albuterol</td>
<td></td>
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</tr>
<tr>
<td>Nebulizer solution (5.0 mg/mL, 2.5 mg/3 mL, 1.25 mg/3 mL, 0.63 mg/3 mL)</td>
<td>2.5-5 mg every 20 min for 3 doses, then 2.5-10 mg every 1-4 hr as needed, or 10-15 mg/hr continuously</td>
<td>0.15 mg/kg (minimum dose, 2.5 mg) every 20 min for 3 doses, then 0.15-0.3 mg/kg up to 10 mg every 1-4 hr as needed, or 0.5 mg/kg/hr by continuous nebulization</td>
<td>Only selective β2-agonists are recommended. For optimal delivery, dilute aerosols to a minimum of 3 mL with gas flow of 6-8 L/min.</td>
</tr>
<tr>
<td>MDI (90 mcg per puff)</td>
<td>4-8 puffs every 20 min up to 4 hr, then every 1-4 hr as needed</td>
<td>4-8 puffs every 20 min for 3 doses, then 1-4 hr inhalation maneuver. Use spacer/holding chamber.</td>
<td>As effective as nebulized therapy if patient is able to coordinate.</td>
</tr>
<tr>
<td>Bitolterol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nebulizer solution (2 mg/mL)</td>
<td>See albuterol dose</td>
<td>See albuterol dose</td>
<td>Use has not been studied for severe asthma exacerbations. Do not mix with other drugs</td>
</tr>
<tr>
<td>MDI (370 mcg per puff)</td>
<td>See albuterol dose</td>
<td>See albuterol dose</td>
<td>Use has not been studied for severe asthma exacerbation.</td>
</tr>
<tr>
<td>Levalbuterol (R-albuterol) nebulizer solution (0.63 mg/3 mL, 1.25 mg/3 mL)</td>
<td>1.25-2.5 mg every 20 min for 3 doses, then 1.25-5 mg every 1-4 hr as needed, or 5-7.5 mg/hr continuously</td>
<td>0.075 mg/kg (minimum dose, 1.25 mg) every 20 min for 3 doses, then 0.075-0.15 mg/kg up to 5 mg every 1-4 hr as needed, or 0.25 mg/kg/hr by continuous nebulization</td>
<td>0.63 mg of levalbuterol is equivalent to 1.25 mg of racemic albuterol in both efficacy and side effects.</td>
</tr>
<tr>
<td>Pirbuterol MDI (200 mcg per puff)</td>
<td>See albuterol dose</td>
<td>See albuterol dose</td>
<td>Use has not been studied for severe asthma exacerbations.</td>
</tr>
<tr>
<td><strong>Systemic (Injected) β2-Agonists</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epinephrine 1:1000 (1 mg/mL)</td>
<td>0.3-0.5 mg every 20 min for 3 doses SC</td>
<td>0.01 mg/kg up to 0.3-0.5 mg every 20 min for 3 doses SC</td>
<td>No proven advantage of systemic therapy over aerosol</td>
</tr>
<tr>
<td>Terbutaline (1 mg/mL)</td>
<td>0.25 mg every 20 min for 3 doses SC</td>
<td>0.01 mg/kg every 20 min for 3 doses, then every 2-6 hr as needed SC</td>
<td>No proven advantage of systemic therapy over aerosol</td>
</tr>
<tr>
<td><strong>Anticholinergics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ipratropium bromide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nebulizer solution (0.25 mg/mL)</td>
<td>0.5 mg every 30 min for 3 doses, then every 2-4 hr as needed</td>
<td>0.25 mg every 20 min for 3 doses, then every 2 to 4 hr</td>
<td>May mix in same nebulizer with albuterol. Should not be used as first-line therapy; should be added to β2-agonist therapy.</td>
</tr>
<tr>
<td>MDI (18 mcg per puff)</td>
<td>4-8 puffs as needed</td>
<td>4-8 puffs as needed</td>
<td>Dose delivered from MDI is low, and its use has not been studied for asthma exacerbations.</td>
</tr>
<tr>
<td>Ipratropium with albuterol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nebulizer solution (each 3-mL vial contains 0.5 mg ipratropium bromide and 90 mcg albuterol)</td>
<td>3 mL every 30 min for 3 doses, then every 2-4 hr as needed</td>
<td>1.5 mL every 20 min for 3 doses, then every 2-4 hr</td>
<td>Contains EDTA to prevent discoloration. This additive does not induce bronchospasm.</td>
</tr>
<tr>
<td>MDI (each puff contains 18 mcg ipratropium bromide and 90 mcg albuterol)</td>
<td>4-8 puffs as needed</td>
<td>4-8 puffs as needed</td>
<td></td>
</tr>
</tbody>
</table>

Continued
evidence of myocardial ischemia are rare, especially in patients without a previous history of coronary artery disease. Aerosol therapy with β₂-agonist drugs produces excellent bronchodilation with minimal systemic absorption and few side effects. Aerosol delivery may be achieved with a metered dose inhaler (MDI) with a spacing device or a compressor-driven nebulizer. A spacing device attached to the inhaler can improve drug deposition when patient technique is inadequate. Even with optimal technique only a maximum of 15% of the dose of the drug is retained in the lungs, regardless of the aerosol method used. Since 2008, dry-powder delivery devices and MDIs using hydrofluoroalkane as propellant have been approved, although the nebulizer method is still used, especially for patients without functional lungs. Aerosolized ipratropium bromide, 0.5 mg, should be administered every 4 to 6 hours until a maximum of 20 minutes or on a continuous basis. Subcutaneous administration of terbutaline or epinephrine may be used in patients unable to coordinate aerosolized or MDI treatments or to tolerate aerosolized medications. Intravenous β₂-agonist infusions offer no advantage over aerosolized or MDI-delivered agents and carry potential risk. Salbutamol inhaler is indicated only as maintenance therapy, should never be used more frequently than twice per day, and is to be avoided for the treatment of acute exacerbations.

**Corticosteroids**

Corticosteroids, highly effective drugs for asthma exacerbation, are a cornerstone of treatment. They are thought to produce beneficial effects by restoring β₂-agonist responsiveness and reducing inflammation. Onset of the antiinflammatory effects of corticosteroids is delayed at least 4 to 8 hours after intravenous or oral administration.

Data indicate that corticosteroids, administered within 1 hour of arrival in the ED, reduce the need for hospitalization of a patient with an asthma exacerbation. Although evidence for what constitutes the optimal dose for acute asthma is lacking, experts agree that an initial 40- to 60-mg dose of prednisone or an intravenous 60- to 125-mg bolus of methylprednisolone in patients unable to tolerate oral medications is usually adequate. No advantage has been demonstrated for higher doses. Additional doses should be given every 4 to 6 hours until significant subjective and objective improvement is achieved. Patients who are being discharged home after ED treatment should be prescribed a 3- to 10-day nontapering “burst” of oral steroids, such as prednisone, 40 to 60 mg/day, or its equivalent.

Current recommendations favor inhaled corticosteroids for maintenance of all patients with mild persistent asthma or more severe asthma. Therefore, consideration should be given to discharging any patient with mild persistent or more severe asthma with maintenance inhaled corticosteroid therapy in addition to the burst of oral steroids.

**Anticholinergics**

Aerosolized ipratropium bromide, 0.5 mg, should be administered to patients with severe exacerbation of asthma. Ipratropium is a synthetic quaternary derivative that is available as both a nebulized solution and in an MDI (18 mg per puff) and is well tolerated (see Table 48.2). Clinical trials indicate that adding ipratropium to β₂-agonist agents offers mild additional improvement in bronchodilation and significantly decreases the need for hospitalization. Side effects include dry mouth, thirst, and difficulty swallowing. Less commonly, tachycardia, restlessness, irritability, confusion, difficulty in micturition, ileus, blurring of vision, and an increase in intraocular pressure are noted.

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**Table 48.2 Medications Used to Treat Asthma Exacerbations—cont’d**

<table>
<thead>
<tr>
<th>MEDICATION</th>
<th>ADULT DOSE</th>
<th>CHILD DOSE*</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic Corticosteroids†</td>
<td></td>
<td></td>
<td>For outpatient “burst,” use 40-60 mg in a single dose or 2 divided doses for adults (children: 1-2 mg/kg/day; maximum, 60 mg/day) for 3-10 days</td>
</tr>
<tr>
<td>Prednisone, methylprednisolone, prednisolone</td>
<td>40-80 mg/day in 1 or 2 divided doses until PEF reaches 70% of predicted value or patient’s personal best</td>
<td>1 mg/kg (maximum, 60 mg/day) in 2 divided doses until PEF reaches 70% of predicted value or patient’s personal best</td>
<td>For outpatient “burst,” use 40-60 mg in a single dose or 2 divided doses for adults (children: 1-2 mg/kg/day; maximum, 60 mg/day) for 3-10 days</td>
</tr>
</tbody>
</table>

*Children younger than 12 years.
†Note: No advantage has been found for higher-dose corticosteroids for severe asthma exacerbations, nor does intravenous administration have any advantage over oral therapy, provided that gastrointestinal transit time or absorption is not impaired. The usual regimen is to continue the frequent multiple daily doses until the patient achieves a forced expiratory volume in 1 second or PEF of 50% of the predicted value or the patient’s personal best—which usually occurs within 48 hours—and then to lower the dosage to twice daily. Therapy after a hospitalization or ED visit may last 3 to 10 days. For corticosteroid courses lasting 1 week or less, there is probably no need to taper, especially if patients are concurrently taking inhaled corticosteroids. If the follow-up systemic corticosteroid therapy is to be given once daily, one study indicates that it may be more clinically effective to give the dose in the afternoon at 3 PM, with no increase in adrenal suppression.”

**EDTA**, Ethylenediaminetetraacetic acid; **MDI**, metered dose inhaler; **PEF**, peak expiratory flow; **SC**, subcutaneously.

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**SECTION V THORACIC AND RESPIRATORY DISORDERS**

**Table 48.2 Medications Used to Treat Asthma Exacerbations—cont’d**

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<td>1 mg/kg (maximum, 60 mg/day) in 2 divided doses until PEF reaches 70% of predicted value or patient’s personal best</td>
<td>For outpatient “burst,” use 40-60 mg in a single dose or 2 divided doses for adults (children: 1-2 mg/kg/day; maximum, 60 mg/day) for 3-10 days</td>
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*Children younger than 12 years.
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†Note: No advantage has been found for higher-dose corticosteroids for severe asthma exacerbations, nor does intravenous administration have any advantage over oral therapy, provided that gastrointestinal transit time or absorption is not impaired. The usual regimen is to continue the frequent multiple daily doses until the patient achieves a forced expiratory volume in 1 second or PEF of 50% of the predicted value or the patient’s personal best—which usually occurs within 48 hours—and then to lower the dosage to twice daily. Therapy after a hospitalization or ED visit may last 3 to 10 days. For corticosteroid courses lasting 1 week or less, there is probably no need to taper, especially if patients are concurrently taking inhaled corticosteroids. If the follow-up systemic corticosteroid therapy is to be given once daily, one study indicates that it may be more clinically effective to give the dose in the afternoon at 3 PM, with no increase in adrenal suppression.”

**EDTA**, Ethylenediaminetetraacetic acid; **MDI**, metered dose inhaler; **PEF**, peak expiratory flow; **SC**, subcutaneously.
At discharge, addition of tiotropium to the patient’s current inhaled corticosteroid dose is comparable to the addition of salmeterol—both are more effective in achieving disease control than is doubling the inhaled corticosteroid dose.27

**Magnesium**

Intravenous magnesium sulfate is indicated for the management of acute, very severe asthma, such as a patient with an FEV₁ less than 25% of predicted, but not in those with mild or moderate asthma exacerbation.28-30 The dose is 1 to 2 g intravenously delivered over a 30-minute period. Inhaled magnesium may also be a helpful adjunct in the treatment of a severe exacerbation.31,32 Magnesium is not a substitute for standard therapy regimens.

**Heliox, Ketamine, and Halothane**

Helium is not indicated for use in patients with mild or moderate asthma exacerbation, although several studies have demonstrated its effectiveness for very severe asthma.33 Several investigators have reported success with ketamine34,35 and halothane in patients in whom all other treatment modalities have failed. Controlled trials substantiating these claims are lacking.

**Mast Cell Modifiers**

Neither cromolyn nor nedocromil, both modulators of mast cell mediator release and eosinophil recruitment, is indicated for the treatment of acute bronchospasm.

**Leukotriene Modifiers**

Leukotriene modifiers improve lung function, diminish symptoms, and reduce the need for short-acting β₂-agonists.1 They are recommended as an alternative to low-dose inhaled corticosteroid therapy in patients with mild persistent asthma and as steroid-sparing agents with inhaled corticosteroids in those with moderate persistent asthma. Several leukotriene modifiers—montelukast, zafirlukast, and zileuton—are currently available as oral tablets for the treatment of asthma. Although intravenous montelukast has been demonstrated to cause rapid bronchodilation when used as adjuvant therapy for acute asthma in a single trial, recommending its use for the treatment of acute bronchospasm in the ED would be premature.36 Montelukast, zafirlukast, and zileuton have been associated with neuropsychiatric side effects.

**Theophylline**

Although theophylline is no longer considered a first-line treatment of acute asthma,37 some patients who come to the ED for treatment may be using it at home. Some data suggest that this agent has an antiinflammatory mechanism of action. When used in combination with inhaled β₂-agonists, theophylline appears to increase the toxicity—but not the efficacy—of treatment. The most common side effects of theophylline are nervousness, nausea, vomiting, anorexia, and headache. At plasma theophylline levels greater than 30 mcg/mL, there is a risk for seizures and cardiac arrhythmias.

**MECHANICAL VENTILATION**

When it appears that a patient needs more than the aforementioned treatments, noninvasive positive pressure ventilation may be attempted. Data showing that bilevel positive airway pressure reduces the need for intubation and mechanical ventilation are lacking.38-40

If the patient begins to exhibit signs of acute ventilatory failure with progressive hypercapnia and acidosis or becomes exhausted or confused, intubation and mechanical ventilation are needed to prevent respiratory arrest. Mechanical ventilation can eliminate the work of breathing and enable the patient to rest. It does not relieve the airflow obstruction. Direct, controlled oral intubation by an experienced physician is preferred.

The potential complications of mechanical ventilation in asthmatic patients are numerous: barotrauma, hemodynamic impairment, mucous plugging leading to increased airway resistance, atelectasis, and pulmonary infection. Air trapping and increased residual volume (intrinsic positive expiratory pressure) may be partially avoided with controlled mechanical hypventilation or permissive hypoventilation.39,40 This form of mechanical ventilation is achieved by using a reduced respiratory rate and low inspiratory volume and pressure and allowing adequate time for the expiratory phase. One can achieve the goal of ventilatory support—maintenance of adequate arterial oxygen saturation (90% or greater)—without concern about “normalizing” the hypercapnic acidosis. All patients requiring mechanical ventilation should be admitted to an intensive care unit.

**DISPOSITION**

Disposition decisions for patients after treatment of asthma exacerbation are rarely straightforward. A number of subjective and objective factors should be considered, as follows:

- Does the patient feel that the wheezing and air exchange have improved?
- Does auscultation confirm improvement or lack thereof?
- Has a significant improvement in FEV₁ or PEF been noted?
- What is the patient’s health care history?
- Is the patient usually compliant with care plans and medication regimens?
- Does the patient have access to prompt follow-up?
- Does the patient usually require hospitalization after an exacerbation?

Unfortunately, a formula for successful discharge without risk for early relapse does not yet exist, and up to 25% of patients treated in the ED for asthma return within 3 weeks.41-44

Because some degree of residual airflow obstruction, airway lability, and inflammation persists after treatment and discharge from the ED, a postdischarge treatment plan must be formulated.
Addition of a short, nontapering course of oral steroids to the scheduled use of a \( \beta_2 \)-agonist bronchodilator reduces relapse rates in discharged patients. Patients with chronic asthma who are not using controller medications at home should be prescribed and educated about the daily use of either inhaled corticosteroids or leukotriene modifiers, in addition to their rescue medications. Data indicate that relying on the primary care physician to prescribe these controllers at follow-up is inadequate. Current guidelines suggest that patients with a good response to treatment, as demonstrated by complete resolution of symptoms and a PEF or FEV\(_1\) value greater than 70% of predicted, can be safely discharged home. Patients with a poor response to treatment, as defined by persistent symptoms, a PEF or FEV\(_1\) value less than 50% of predicted, and persistent wheezing and dyspnea at rest, should be admitted. Many patients with an incomplete response to treatment, as defined by some persistence of symptoms and a PEF or FEV\(_1\) value between 50% and 70% of predicted, may be discharged home safely, provided that they have no risk factors for death from asthma. Patients who do not show adequate improvement over several hours because they are in the late phase of the exacerbation and those with significant risk factors for death from asthma should be admitted to either an observation unit or the hospital. Most patients have an incomplete response to treatment and fall into this “gray zone” of disposition decisions.

Studies indicate that the majority of asthmatic patients admitted to an observation unit where strict care protocols are followed can be successfully treated and discharged. Early follow-up care is indicated to monitor resolution of the exacerbation and to review the long-term medication and care plans for the ongoing management of asthma. High relapse rates despite the routine use of steroids strongly suggest the need for follow-up within days of the ED visit. Ideally, education of the patient begins in the ED, and a written plan of action that addresses both routine treatment and care of worsening symptoms is developed either in the ED or at follow-up. ED personnel should provide basic education about asthma and help connect the patient with a primary care provider or asthma specialist while providing discharge instructions. Review of the patient’s discharge medication, evaluation of inhaler technique, and instruction on the use of peak flow monitoring are just some of the issues that emergency physicians can teach and emphasize.

### SUGGESTED READINGS


### REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.


