INTRODUCTION — The best strategy for management of acute exacerbations of asthma is early recognition and intervention, before attacks become severe and potentially life-threatening. Detailed investigations into the circumstances surrounding fatal asthma have frequently revealed failures on the part of both patients and clinicians to recognize the severity of the disease and to intensify treatment appropriately [1].

The management of acute asthma exacerbations will be presented here. An overview of asthma management, the identification of risk factors for fatal asthma, and the use of mechanical ventilation in severe exacerbations of asthma are discussed separately. (See "An overview of asthma management" and "Identifying patients at risk for fatal asthma" and "Invasive mechanical ventilation in adults with acute exacerbations of asthma").

ALGORITHMS FOR ASSESSMENT AND TREATMENT — The National Asthma Expert Panel has published useful algorithms on the management of acute exacerbations of asthma for both the home and acute care settings [algorithm 1 and algorithm 2] [2]. These algorithms may be used for asthma exacerbations of any severity. A table outlining the emergency management of severe asthma exacerbations in adults is also provided (table 1).

The basic principles of care are the following:

- Assess the severity of the attack
- Assess potential triggers (eg, dander, pollen, mold, respiratory infection, nonsteroidal anti-inflammatory drugs [NSAIDs], nonadherence)
- Use inhaled short-acting beta agonists early and frequently, and consider concomitant use of ipratropium for severe exacerbations
- Start systemic glucocorticoids if there is not an immediate and marked response to the inhaled short-acting beta agonists
- Make frequent (every one to two hours) objective assessments of the response to therapy until definite, sustained improvement is documented
- Admit patients who do not respond well after four to six hours to a setting of high surveillance and care
- Educate patients about the principles of self-management for early recognition and treatment of a recurrent attack and develop an "asthma action plan" for recurrent symptoms (see "What do patients need to know about their asthma?")

Ideally, patients assess the severity of an attack at home by following an individualized written “asthma action plan.” Asthma action plans are based upon symptoms and peak flow measurements and provide clear instructions on how to detect and respond to changes in these parameters [2,3]. An example is available through the National Heart Lung and Blood Institute (NHLBI Asthma action plan). Peak flow meters are available at low cost (eg, $30 or less). The asthma action plan should include clear instructions about how to reach the clinician during exacerbations. Instructions to a patient regarding self-administration of medications and self-monitoring differ depending upon the patient’s history (eg, medications currently used to treat asthma, prior history of severe exacerbations, and prior experience with oral glucocorticoids) and ability to understand and follow directions. (See "What do patients need to know about their asthma?", section on 'Asthma action plans' and "What do patients need to know about their asthma?", section on 'Attack prevention'.)
DETECTING AN EXACERBATION — Some patients are very sensitive to increased asthma symptoms, while others perceive reduced airflow only when it becomes marked. For the latter group, a decrease in peak expiratory flow may be the first sign that asthma control is deteriorating.

Symptoms — Symptoms that patients should recognize as suggesting an asthma exacerbation include breathlessness, wheezing, cough, and chest tightness. Some patients also report reduced exercise tolerance and fatigue as symptoms of an asthma exacerbation.

Peak flow — Measurement of expiratory airflow with a peak expiratory flow (PEF) meter (or spirometer) provides an assessment of the severity of airflow limitation [4]. Peak flow measurements take less than one minute to perform and are safe and inexpensive. However, careful instruction is needed to obtain reliable measurements. Peak flow monitoring is particularly useful in patients with poor perception of asthma symptoms. (See "Peak expiratory flow rate monitoring in asthma".)

Normal values for PEF differ with sex, height, and age (table 2A-C). Each patient should establish a baseline measure with which to compare future readings. A decrement in peak flow of greater than 20 percent from normal, or from the patient’s personal best value, signals the presence of an asthma exacerbation. The difference in peak flow from the patient’s baseline helps one gauge the severity of the change. A PEF less than 50 percent of baseline should be considered a severe attack.

Risk factors for fatal asthma — Some patients are at greater risk for fatal asthma attacks. It is helpful to identify such patients to educate them about identifying early warning signs of deterioration based on PEF monitoring, following a prednisone-based action plan, and seeking emergency care promptly. (See "Identifying patients at risk for fatal asthma", section on 'Identifying high-risk patients'.)

Risk factors for a fatal asthma attack include [3]:

- Previous severe exacerbation (eg, intubation or intensive care unit admission)
- Hospitalization or emergency department visit for asthma in the past year
- Three or more emergency department visits for asthma in the past year
- Not currently using inhaled glucocorticoids
- Recent or current course of oral glucocorticoids
- Use of more than one canister of short-acting beta agonist per month
- Difficulty perceiving asthma symptoms or severity of exacerbations
- History of poor adherence with asthma medications and/or written asthma action plan
- Illicit drug use and major psychosocial problems, including depression
- Comorbidities, such as cardiovascular or chronic lung disease

INITIAL HOME TREATMENT — The goals of initial home management are to relieve airflow limitation, prevent or reverse hypoxemia, and initiate or augment controller therapy to prevent a recurrence. Patients with serious exacerbations frequently require evaluation in the office and may need to proceed to the emergency department if improvement is not quickly apparent. Home management is not meant to replace supervised medical care in seriously ill patients.

Short-acting beta agonist — When the onset of an exacerbation is recognized, the patient should self-administer an inhaled short-acting beta agonist (SABA; eg, albuterol, levalbuterol) by one of the following methods (table 3) [2,3,5]:

- Two to six puffs from a metered dose inhaler (depending upon the dose that is typically effective and tolerated by that individual; typically two to four puffs are used for a mild to moderate exacerbation), preferably with a chamber or spacer device. This dose can be repeated every 20 minutes for the first hour.
- A nebulized treatment (eg, albuterol 2.5 mg), repeated every 20 minutes for first hour, if needed.

After the first hour, if the patient feels initial improvement with the SABA, he or she should repeat a peak flow measurement. Based upon the response to the inhaled beta agonist, the patient should either continue self-care or seek medical attention, as described below (algorithm 1). The subsequent dose of the SABA depends on the severity of the exacerbation. Mild exacerbations usually respond to two to four puffs every three to four hours, while more severe exacerbations may require six to eight puffs every one to two hours. Patients should contact their clinician, if they need high doses of inhaled beta agonists beyond the first hour of self-treatment. (See 'Disposition based on response' below.)

If after initial home treatment the patient has symptoms or signs suggestive of a severe exacerbation (eg, marked breathlessness, inability to speak more than short phrases, use of accessory muscles) or a peak...
flow less than 50 percent of baseline, he or she should seek urgent medical attention. Patients may be advised by their clinician to take a dose of oral glucocorticoids (eg, prednisone 40 mg) on route to the emergency department.

**Risks associated with inhaled epinephrine** — Racemic epinephrine liquid for inhalation (eg, Asmaneprin and S2) and epinephrine inhalers are available over-the-counter in some countries and marketed directly to consumers for temporary relief of asthma symptoms. These products are administered via hand-held nebulizer or by dry powder inhalation and have not been evaluated by the US Food and Drug Administration (FDA) for safety or efficacy. The FDA has issued a warning about multiple adverse events associated with these products, including symptoms such as chest pain, nausea and vomiting, increased blood pressure, tachycardia, and hemoptysis, and also defective atomizer devices [6]. Epinephrine is NOT beta-2 adrenergic receptor selective, so it carries a greater risk of beta-1 adrenergic type adverse effects, especially when used in excess doses.

It is important to ensure that patients have ready access to the more effective, inhaled short-acting beta-2 agonists (SABA) and to advise against use of the nonselective epinephrine-based products [7-9].

**Disposition based on response** — The assessment of response to initial home therapy is based on the degree of improvement in symptoms and return of peak flow to baseline.

**Good response** — If the patient’s symptoms (wheezing, dyspnea) resolve and the repeat peak flow measurement increases to above 80 percent of the patient’s personal best over the course of approximately one hour, then the patient may safely continue self-treatment (algorithm 1). Other important early interventions include removal of or from the offending stimulus (if known), continued administration of inhaled short-acting beta agonists, and consideration of a short course of oral glucocorticoids if beta agonists do not fully correct the decrement in peak flow or if symptoms recur.

The patient should contact their clinician for instructions about how to continue care. Depending on the clinical situation, the clinician may advise continuing the current therapy, increasing the dose of inhaled glucocorticoids, or initiating a short course of oral glucocorticoids. If the patient’s symptoms resolve, his or her peak expiratory flow (PEF) returns to >80 percent of baseline after the initial dose(s) of SABA, and he or she remains improved for three to four hours, oral glucocorticoids are usually not necessary.

- For these patients, the clinician may decide that no change in therapy is needed (eg, prompt improvement after a transient exposure to an allergen)
- Alternatively, the clinician may determine that the patient is experiencing a more general deterioration in asthma control that warrants an increase in the inhaled glucocorticoid dose (see "Treatment of moderate persistent asthma in adolescents and adults", section on ‘Assessing control’)

For patients with more severe initial symptoms or a more profound decrease in PEF, oral glucocorticoids are usually appropriate despite the good initial response to SABA treatment. (See ‘Oral versus intravenous dosing’ below.)

**Incomplete response** — An incomplete response to inhaled short-acting bronchodilator is manifest by continued symptoms and a PEF in the range of 50 to 80 percent of personal best. The patient should initiate oral glucocorticoids according to his or her prednisone-based action plan and contact his or her clinician for advice (algorithm 1). Timely administration of oral glucocorticoids for asthma exacerbations is probably the single most effective strategy for reducing emergency department visits and hospitalizations for acute asthmatic attacks.

Other early interventions include removal of or from the offending stimulus (if known), continued administration of inhaled short-acting beta agonists every three to four hours, and intermittent measurements of peak flow to assess response.

**Need for urgent medical attention** — Patients should seek immediate medical attention if they have a PEF less than 50 percent of baseline after one or two doses of inhaled beta-agonist by a metered dose inhaler or nebulizer, report symptoms or signs of severe exacerbation (eg, marked breathlessness), or are at high risk for a fatal attack. (See ‘Risk factors for fatal asthma’ above.)

Inhaled beta agonists should continue to be administered while help is arriving.

**Increasing the dose of inhaled glucocorticoid** — While not a substitute for the oral glucocorticoids that are needed for moderate to severe exacerbations, a large increase in the dose of inhaled glucocorticoids may be beneficial in some patients, when given early in an exacerbation. As an example, among patients with a mild increase in asthma symptoms and a small decrease in peak flow (eg, 15 percent for two days or
30 percent for one day), quadrupling the dose of inhaled glucocorticoids, rather than no change, resulted in a decrease in the likelihood of needing oral glucocorticoids (relative risk [RR] 0.43, 95% CI 0.24-0.78) [10]. However, further study of this approach is needed before widespread implementation.

In contrast, doubling the dose of inhaled glucocorticoids is not recommended in the setting of an acute exacerbation as an alternative to oral glucocorticoids [2,11]. In a trial of 390 patients with asthma, subjects were randomly assigned to treatment with twice their usual dose of inhaled glucocorticoid or placebo (delivered using an identical inhaler) when asthmatic symptoms worsened [12]. Over the course of the one-year study, over half of all patients experienced an episode of worsening of their asthma, and 12 percent eventually required systemic glucocorticoid therapy with prednisolone to control their symptoms. There was no significant difference in the need for systemic glucocorticoid therapy between groups, suggesting a lack of efficacy of the increased dose of inhaled glucocorticoids.

**URGENT CARE ASSESSMENT** — Once the patient presents for evaluation, a number of clinical findings and objective tests can assist the clinician in confirming that the symptoms are caused by an exacerbation of asthma, assessing the severity of an asthma attack, and excluding complicating factors (eg, pneumonia, atelectasis, pneumothorax, pneumomediastinum) (algorithm 2). The diagnosis of asthma and the differential diagnosis of nonasthmatic wheezing are discussed separately. (See "Diagnosis of asthma in adolescents and adults" and "Evaluation of wheezing illnesses other than asthma in adults").

**Clinical findings** — A focused history and physical are obtained concurrently with the initiation of therapy in order to confirm the diagnosis and assess the severity. A severe asthma exacerbation is potentially life-threatening and needs prompt and intensive care. The presence of certain clinical findings may help to identify patients experiencing severe asthma attacks. Tachypnea, tachycardia >120 beats/min, use of accessory muscles of inspiration (eg, sternocleidomastoid muscles), diaphoresis, inability to speak in full sentences or phrases, inability to lie supine due to breathlessness, and pulsus paradoxus (ie, a fall in systolic blood pressure by at least 12 mmHg during inspiration) all are indicative of severe airflow obstruction [3,13]. Unfortunately, these findings are not sensitive indicators of severe attacks; up to 50 percent of patients with severe airflow obstruction will not manifest any of these abnormalities [14]. (See "Examination of the arterial pulse", section on 'Pulsus paradoxus'.)

Concomitant symptoms such as fever, purulent sputum production, or pleuritic chest pain should raise the possibility of an alternative diagnosis such as infection or pneumothorax.

**Peak flow measurement** — Measurement of expiratory airflow with a peak flow meter (or spirometer) is the best method for objective assessment of the severity of an asthma attack. Peak flow measurements can also be used to monitor a patient's response to treatment and as a predictive marker for the possibility of hypercapnia, as discussed below. Normal values differ with gender, height, and age (table 2A-C), but in general, a peak flow rate below 200 L/min indicates severe obstruction most adults except those who are very short or elderly [2]. A peak flow less than 50 percent of baseline for that individual signifies a serious exacerbation. (See "Peak expiratory flow rate monitoring in asthma").

**Oxygenation** — The ready availability of pulse oxygen saturation testing by transcutaneous oximetry allows noninvasive screening for hypoxemia among patients with severe asthma attacks. Current guidelines recommend the use of transcutaneous pulse oximetry monitoring particularly among patients who are in severe distress, have a forced expiratory volume in one second (FEV1) or peak expiratory flow less than 50 percent of baseline, or are unable to perform lung function measurements [2]. There is no contraindication to the use of continuous transcutaneous oximetry during all asthmatic attacks.

Marked hypoxemia (arterial partial pressure of oxygen [PaO2] <60 mmHg [8 kPa], oxygen saturation [SaO2] <90 percent) is infrequent during uncomplicated asthma attacks; its presence suggests life-threatening asthma and possible complicating conditions, such as pneumonia or atelectasis due to mucus plugging. Severe hypoxemia poses the risk for severe cardiovascular or neurologic complications and death.

**Hypercapnia** — Simple peak flow measurements provide a useful screening tool for hypercapnia (eg, PaCO2 >45 mmHg or >6 kPa), in contrast to hypoxemia, making routine assessment of arterial blood gases (ABG) unnecessary in the majority of patients. In the absence of respiratory depressant medications such as narcotics or sedatives, hypercapnia is rarely present when the peak expiratory flow (PEF) is ≥25 percent of normal or ≥200 L/min [15-17]. Thus, arterial blood gas measurements in acute asthma are indicated in the following settings:

- Patients with persistent dyspnea whose PEF is below 25 percent of normal or below 200 L/min despite initial bronchodilator therapy (meaning that if the PEF is this low initially, give bronchodilator and see if there is improvement rather than doing an immediate ABG)
●Selected patients with a PEF 25 to 50 percent of normal whose respiratory status is deteriorating despite intensive therapy
●Patients who are too ill to perform a peak flow measurement
●Patients who demonstrate signs or symptoms of hypercapnia, such as depressed consciousness, inappropriately slow respiratory rate, or myoclonus

Respiratory drive is almost invariably increased in acute asthma, resulting in hyperventilation and a correspondingly decreased PaCO₂. Thus, a normal PaCO₂ (eucapnia) during an asthma exacerbation indicates that airway narrowing is so severe that the respiratory system cannot respond adequately to the output of the respiratory center. Hypercapnia and respiratory failure can then develop rapidly with any further airway obstruction or with respiratory muscle fatigue. Progressive hypercapnia during an exacerbation of asthma is generally an indication for mechanical ventilation.

Measurement of the tension of carbon dioxide (PvCO₂) in peripheral venous blood is becoming a more common practice in emergency departments. A normal or low venous blood PvCO₂ reliably predicts a normal or low arterial tension of carbon dioxide (PaCO₂) and can be used to exclude hypercapnia, although the overall correlation between PvCO₂ and PaCO₂ is poor [18]. (See "Venous blood gases and other alternatives to arterial blood gases", section on 'Venous blood gases'.)

Chest radiograph — Chest radiographs are generally unrevealing in acute asthma attacks and are not routinely required in the emergency department [3,19]. However, a chest radiograph should be obtained when a complicating cardiopulmonary process is suspected (eg, temperature >38.3°C, unexplained chest pain, leukocytosis, or hypoxemia), when a patient requires hospitalization, and when the diagnosis is uncertain [3]. The most common abnormality is pulmonary hyperinflation. Other abnormal findings (eg, pneumothorax, pneumomediastinum, pneumonia, or atelectasis) are infrequent, occurring in only about 2 percent of chest radiographs obtained among patients presenting to emergency departments for the treatment of acute asthma [20,21].

Chest radiographs are also useful for patients at high risk for comorbidities (eg, based upon a history of intravenous drug abuse, immunosuppression, granulomatous disease, recent seizures, cancer, chest surgery, or heart failure) [19,22].

TREATMENT IN URGENT CARE SETTING — A table outlining the emergency management of severe asthma exacerbations in adults is provided (table 1).

Approach — The primary goals of therapy for acute severe asthma are the rapid reversal of airflow limitation and the correction, if necessary, of severe hypercapnia or hypoxemia. Airflow limitation is most rapidly alleviated by the combination of repeated administration of inhaled bronchodilators and early institution of systemic glucocorticoids (algorithm 1 and algorithm 2). Until their respiratory distress has abated, patients should receive close monitoring, including serial measurements of lung function (eg, peak expiratory flow), to assess the response to treatment.

Oxygen — Supplemental oxygen should be administered to virtually all patients with an asthma exacerbation that requires treatment in the emergency department or hospital. The flow of oxygen is titrated to maintain the pulse oxygen saturation (SpO₂) ≥90 percent (>95 percent in pregnancy) [3]. Usually, oxygenation is easily maintained with nasal cannula, but occasionally a face mask delivery is needed. There is some evidence that titrating oxygen supplementation to an oxygen saturation of 90 to 95 percent is preferable to high-flow 100 percent oxygen therapy [3]. In a trial that randomly assigned 106 patients with severe asthma exacerbations to mask oxygen at 8 L/min or titrated oxygen to achieve 93 to 95 percent saturation for 60 minutes, the transcutaneous carbon dioxide tension increased ≥4 mmHg in 44 percent of the high oxygen concentration group compared with 19 percent of the titrated oxygen group (relative risk [RR] 2.3, 95% CI 1.2-4.4) [23].

Inhaled beta agonists — The mainstay of bronchodilator treatment is inhalation of short-acting beta-2-selective adrenergic agonists (SABA), such as albuterol, levalbuterol, or fenoterol (available outside the United States) [2,3,5,24]. (See "Beta agonists in asthma: Acute administration and prophylactic use").

Dosing — The standard regimen for initial care in the emergency department is inhaled albuterol (or an equivalent) (table 3). We make every effort to provide three treatments with inhaled SABA within the first hour in the emergency department. The delivery method varies with the setting and severity of the attack (table 3) [2]:

• Standard nebulization – Albuterol 2.5 to 5 mg by jet (also called "hand-held" or "updraft") nebulization every 20 minutes for three doses, then 2.5 to 10 mg every one to four hours as needed. The higher doses are associated with more frequent and severe sympathomimetic side effects and
are generally reserved for refractory attacks. (See "Delivery of inhaled medication in adults", section on 'Nebulizers'.)

- Metered dose inhaler (MDI) – **Albuterol** by MDI with a spacer or chamber device (eg, Aerochamber, Optichamber, Vortex, and others), four to eight puffs every 20 minutes, for the first hour. Most patients can then transition to dosing every one to four hours, and rarely require dosing at more frequent intervals.
- Continuous nebulization – For patients in the intensive care unit, some clinicians prefer continuous nebulization, administering 10 to 15 mg over one hour, but this requires a special apparatus (figure 1). The technique of continuous nebulization is discussed separately. (See "Delivery of inhaled medication in adults", section on 'Continuous nebulization'.)

### Nebulizer versus MDI

The relatively large particle size generated by jet nebulizers and the loss of medication from the expiratory port of the nebulizer system make this method of delivery relatively inefficient compared to a metered dose inhaler (MDI). Comparisons of MDI plus spacer with nebulizer delivery, using the same beta agonist but in much reduced doses when given by MDI, have demonstrated comparable improvements in lung function [25-30]. (See "Delivery of inhaled medication in adults".)

In a systematic review and meta-analysis that included 13 trials (5 open-label) with a total of 729 adults, the outcomes of delivery of beta-agonist via MDI-spacer or nebulizer were examined [29]. The risk ratio (RR) of admission after emergency department after administration of beta-agonist via spacer versus nebulizer was 0.94 (95% CI 0.61-1.43). Peak flow and FEV1 responses and the duration of emergency department stay were similar between groups. The baseline severity of asthma varied from moderate to severe.

Four to six carefully administered inhalations from an MDI with spacer have generally been found to equal one nebulizer treatment, although the equivalent dose has not been precisely defined. In comparative trials, administration of beta agonist by MDI with spacer was directly supervised to ensure the patient's proper coordination and inhalational technique.

Many emergency departments (including our own) continue to rely on nebulized administration of beta agonists for acutely ill asthmatic patients, taking advantage of the simplicity of delivery during the patient's tidal breathing. As patients recover in the hospital from acute, severe attacks, the transition can be made from nebulized beta agonists to beta agonists by MDI with a spacer or chamber device without loss of efficacy and with the opportunity for patient education in proper inhaler technique [31]. (See "The use of inhaler devices in adults".)

### Inhaled anticholinergics

In accordance with current guidelines for asthma management prepared by the Expert Panel of the National Asthma Education and Prevention Program (Expert Panel Report III), we suggest that inhaled *ipratropium* be used in addition to inhaled *albuterol* for patients with severe exacerbations who are in the emergency department [2,3].

The adult dosing of *ipratropium* for nebulization is 500 mcg every 20 minutes for three doses, then as needed [2]. Alternatively, *ipratropium* can be administered by MDI at a dose of four to eight inhalations every 20 minutes, as needed for up to three hours.

Some investigators have reported that the combination of a selective beta-2 agonist with *ipratropium* provides greater bronchodilation than beta-2 agonists alone, particularly among patients with the most severe airflow obstruction [32]. Meta-analyses also have supported the use of *ipratropium* in the setting of very severe airflow obstruction [33,34]. However, positive findings have not been universal. Other large trials were unable to demonstrate an advantage for combined *albuterol* and *ipratropium* over *albuterol* alone, either for the entire population of patients or for any identifiable subgroups [35,36]. Furthermore, in contrast to the meta-analysis that found an advantage in the subset of patients with more severe obstruction, a large trial in New Zealand found no advantage with combined *albuterol* and *ipratropium* over *albuterol* alone among those with the most severe obstruction (FEV1 <1 L), although greater bronchodilation was achieved with the combined use of *albuterol* and *ipratropium* in the group as a whole [37].

We typically stop inhaled short-acting anticholinergic therapy once the patient is admitted to the hospital, except in patients with refractory asthma who require treatment in the intensive care unit, patients on monoamine oxidase inhibitor therapy (who may have increased toxicity from sympathomimetic therapy due to impaired drug metabolism), patients with chronic obstructive pulmonary disease with an asthmatic component, and patients whose asthma has been triggered by beta-blocker therapy. This approach is based on studies in children that found no benefit from continuation of inhaled anticholinergic bronchodilators in hospitalized patients [38,39] and is supported by current guidelines [2,3].
Systemic glucocorticoids — Systemic glucocorticoid therapy is essential for the resolution of asthma exacerbations that are refractory to intensive bronchodilator therapy because the persistent airflow obstruction is likely due to airway inflammation and intraluminal mucus plugging. In concurrence with current guidelines, we recommend early administration of systemic glucocorticoids for patients with the following [2]:

- A severe exacerbation with a peak expiratory flow <40 percent of baseline warrants immediate administration. Of note, in the urgent care setting, the criterion for a severe asthmatic attack changes from the <50 percent used for decision-making at home to <40 percent.
- A moderate exacerbation with a peak expiratory flow >40 but <70 percent of baseline.
- Lack of full correction in the decrement in peak flow after several treatments with inhaled short-acting beta agonists.
- An asthma exacerbation despite ongoing daily or alternate day oral glucocorticoid therapy. Such patients require supplemental glucocorticoids above their baseline dose.

Among patients with significant airflow obstruction despite intensive treatment with bronchodilators, systemic glucocorticoids speed the rate of improvement [40]. However, the onset of action of systemic glucocorticoids is not clinically apparent until as long as six hours after administration. Thus, the beneficial effect is not likely to be observed during the few hours that the patient spends in the medical office or emergency department [41]. Early administration helps to minimize the delay in improvement anticipated with systemic glucocorticoids [42].

Oral versus intravenous dosing — The optimal dose for systemic glucocorticoids in asthmatic exacerbations remains unknown. The equivalent of prednisone 40 to 60 mg (or prednisolone 0.5 to 1 mg/kg) per day in a single or divided dose is typical for most asthma exacerbations (table 4), although a higher dose is often used for life-threatening asthma exacerbations [2]. When comparable doses are administered, the effect of glucocorticoids by oral and intravenous routes of administration is identical. Thus, in the absence of vomiting or respiratory failure, oral administration can be used instead of intravenous administration. Oral prednisone and methylprednisolone are rapidly absorbed (peak serum levels achieved at one hour after ingestion) with virtually complete bioavailability, and their efficacy is comparable to intravenous methylprednisolone. Thus, 40 to 60 mg of prednisone administered in a medical office may provide the maximal benefit to be derived from systemic glucocorticoids in acute asthma.

Intravenous glucocorticoids should be given to patients who present with impending or actual respiratory arrest, or are intolerant of oral glucocorticoids [2]. The exact dose of glucocorticoids to use for patients with life-threatening asthma is largely based on expert opinion [43]. A higher initial dose of methylprednisolone 60 to 80 mg every 6 to 12 hours is often chosen for patients who are admitted to the intensive care unit. A lower initial dose of 40 to 60 mg every 12 to 24 hours is likely adequate for patients who are admitted to the hospital, but do not require intensive care. A massive initial dose (eg, methylprednisolone 500 mg intravenous bolus) is no more effective than a large initial dose (125 mg) [44]. Glucocorticoids can be given intramuscularly if intravenous and oral access is not available.

Transition from parenteral to oral administration of glucocorticoids can occur when the patient can tolerate and absorb oral medication.

Duration — The duration of a systemic therapy necessary to effect complete resolution of symptoms and return of lung function to baseline varies from patient to patient and attack to attack. As a rough guide, most severe attacks that require hospitalization will resolve (with return of lung function to baseline) in 10 to 14 days. Alternatively, patients can stop their oral glucocorticoids sooner based on resolution of their symptoms and self-monitored peak flow values (eg, when peak expiratory flow is greater than 70 percent of baseline).

Tapering oral glucocorticoids is not necessary if the duration of glucocorticoid treatment is less than three weeks (a duration too brief to cause adrenal atrophy) and if inhaled glucocorticoids are concomitantly prescribed for ongoing therapy (to prevent relapse).

Magnesium sulfate — Intravenous administration of a single dose of magnesium sulfate (2 g infused over 20 min) is suggested for patients who have a life-threatening exacerbation or whose exacerbation remains severe (peak expiratory flow <40 percent of baseline) after one hour of intensive conventional therapy [2]. Intravenous magnesium sulfate has bronchodilator activity in acute asthma, possibly due to inhibition of calcium influx into airway smooth muscle cells [45]. Although the routine use of this agent does not seem to confer significant benefit beyond that achieved with the conventional use of beta agonists and systemic glucocorticoids, systematic reviews have concluded that it is helpful in the subgroup of patients with severe attacks [46-49].
Intravenous magnesium has an excellent safety profile; however, it is contraindicated in the presence of renal insufficiency, and hypermagnesemia can result in muscle weakness. (See “Symptoms of hypermagnesemia.”)

Nonstandard therapies — Several nonstandard therapies for acute exacerbations of asthma may be helpful, but cannot be recommended for routine use, due to insufficient evidence of efficacy. These include anesthetic agents (eg, ketamine), enoximone, parenteral beta-agonists, high-dose inhaled glucocorticoids, helium-oxygen gas mixtures, and leukotriene receptor antagonists.

Anesthetic agents — Some anesthetic agents (eg, intravenous ketamine, inhaled halothane, isoflurane, sevoflurane) have bronchodilating effects, and case reports have described responses in patients with refractory status asthmatics. The mechanism by which bronchodilation is produced remains unclear; a direct relaxant effect on airway smooth muscle, and attenuation of cholinergic tone, have both been proposed [50.51]. None of these agents has been evaluated in a randomized trial and some adverse outcomes have been reported [52].

- **Inhalational agents** — Experience is greatest with halothane for status asthmatics, but isoflurane and sevoflurane have also shown effectiveness in case reports [53-58]. Idiosyncratic reactions to these anesthetics have been described, and a given patient may respond better to one than to another. The dose of inhalational anesthetic is titrated to the clinical response (eg, improvement in airway resistance) and avoidance of intolerable side effects. Hypotension is often the limiting factor in the administration of these agents, and myocardial depression and increased ventricular irritability have been observed with halothane, particularly when used in the presence of acidosis, beta-agonists, and theophylline. The use of inhalational anesthetics for treatment of status asthmaticus is limited by its expense, the need for a full-time anesthesiologist at the bedside, adaptation of equipment for long-term provision of anesthetics, and the abrupt return of bronchoconstriction upon discontinuation. There is also the issue of scavenging anesthetic gases released into the immediate environment in order to avoid second-hand inhalation of aerosolized anesthetic gases by health care personnel or other patients.

- **Intravenous ketamine** — Several case reports have described successful treatment of children and adults with status asthmatics with intravenous ketamine [59-63]. Although experience with this therapy is limited, an intravenous bolus of 0.5 to 1 mg/kg is usually infused over two to four minutes, followed by a continuous infusion of 0.5 to 2 mg/kg per hour [62-63]. In one report, improvement followed increasing the infusion rate to 3 mg/kg per hour [62]. Beneficial results are seen within 30 minutes to several hours. Depending on the setting, ketamine infusions in this dose range may fall under the category of conscious sedation or of anesthesia and require appropriate monitoring. In general, though, ketamine can be administered in the intensive care unit, rather than the operating room, making it preferable to general anesthesia. (See "Induction agents for rapid sequence intubation in adults", section on ‘Ketamine’.)

Enoximone — Enoximone, a selective phosphodiesterase III inhibitor available in Europe, was administered intravenously to eight patients with severe asthma exacerbations, six of whom had a respiratory arrest or hypercapnia [64]. The dose of enoximone varied, ranging from two doses of 25 mg to two doses of 100 mg. Three patients were given enoximone by continuous infusion after the initial boluses. Response to enoximone is described as rapid, within one to twenty minutes. The amount of standard medications (eg, inhaled beta-adrenergic agonist, inhaled ipratropium, systemic glucocorticoid) given prior to enoximone was unclear from the report.

Phosphodiesterase inhibitors are associated with ventricular and atrial arrhythmias, hypotension, and hepatotoxicity. Further study is needed to evaluate the safety and efficacy of intravenous enoximone in patients with acute exacerbations of asthma that are refractory to standard measures.

Parenteral beta-agonists — In general, inhaled short-acting selective beta agonists have a greater efficacy and lower incidence of adverse effects (eg, tachycardia, arrhythmias, myocardial injury) compared with parenteral nonselective beta-agonists. Thus, parenteral beta-agonists are generally avoided when treating asthma exacerbations in adults. However, a few exceptions exist:

- For patients suspected of having an anaphylactic reaction or unable to use inhaled bronchodilators for a severe asthma exacerbation, epinephrine 0.3 to 0.5 mg may be given intramuscularly (eg, 0.3 to 0.5 mL of 1 mg/mL [also labeled 1:1000] solution).
- For patients with severe asthma and impending or actual respiratory failure not responding to standard therapy, but without evidence of anaphylaxis, there are rare reports of response to parenteral epinephrine 0.3 to 0.5 mg subcutaneously (eg, 0.3 to 0.5 mL of 1 mg/mL [also labeled 1:1000] solution).
• Similarly, there are rare reports of response to terbutaline in refractory asthma. The dose of terbutaline is 0.25 mg by subcutaneous injection every 20 minutes up to three doses. Terbutaline OR epinephrine may be used, but NOT both.

High-dose inhaled glucocorticoids — While increasing the dose of inhaled glucocorticoids is recommended for patients with gradually deteriorating control of asthma, high-dose inhaled glucocorticoids are not recommended as an alternative to oral glucocorticoids for patients with a discrete asthma exacerbation. Despite a few reports suggesting that high-dose inhaled glucocorticoids have an effect comparable to oral or intravenous glucocorticoids following initial stabilization in the emergency department [65-68], several larger controlled trials and meta-analyses have drawn the opposite conclusion [11,12,69-74].

Helium-oxygen — Helium-oxygen (heliox) mixtures have been administered to patients with asthma in an attempt to decrease the work of breathing and improve ventilation. The rationale of this intervention is based on the low density of helium, which reduces airflow resistance. However, routine use of helium-oxygen mixture cannot be recommended due to conflicting data about its efficacy. Rarely, heliox is used in patients with severe exacerbations that are unresponsive to standard therapy or when a component of upper airway obstruction is thought to contribute to airflow obstruction. The physiology and clinical application of heliox are reviewed separately. (See “Physiology and clinical use of heliox” and “Clinical presentation, diagnostic evaluation, and management of central airway obstruction in adults.”)

Despite the theoretical benefits of heliox, studies have reported conflicting results concerning its efficacy in asthma exacerbations. Two systematic reviews of the published literature found insufficient evidence, due to the small size and nonrandomized design of available studies, to recommend the use of helium-oxygen gas mixtures in the treatment of asthmatic attacks. (See “Physiology and clinical use of heliox”, section on ‘Use in adults’.)

Nebulization of albuterol using a heliox-oxygen gas mixture as the driving gas, instead of the usual compressed air or oxygen, results in a smaller particle size, which may improve drug penetration to the small airways. However, clinical trials have failed to provide consistent evidence of benefit. The use of heliox for aerosol delivery is discussed separately. (See “Physiology and clinical use of heliox”, section on ‘Aerosol delivery’.)

Leukotriene receptor antagonists — Leukotriene receptor antagonists are an established therapy for chronic asthma, but the role of these medications in the management of acute exacerbations is unclear [75-79]. We do not administer leukotriene receptor antagonists as part of routine treatment of acute exacerbations, except in patients whose exacerbation was triggered by ingestion of aspirin or a nonsteroidal anti-inflammatory drug (NSAID).

The value of acute administration of an intravenous leukotriene receptor antagonist was assessed in a randomized trial of 201 adults with acute asthma [75]. Patients who had a one-second forced expiratory volume (FEV₁) <70 percent predicted following inhalation of nebulized albuterol were randomly assigned to treatment with montelukast (7 mg or 14 mg IV) or placebo. Montelukast therapy was associated with a 15 percent increase in FEV₁ at both dosages, compared with a 4 percent increase in patients treated with placebo (figure 2). However, no intravenous preparation of a leukotriene receptor antagonist is currently available in the United States.

A larger randomized trial assessed the effect of zafirlukast in 641 adults with acute asthma [76]. Patients treated in the emergency department with a single oral dose of zafirlukast 160 mg (four times the usual total daily dose) were less likely than those receiving placebo to require prolonged observation or hospital admission (10 versus 15 percent, respectively). In addition, patients who received oral zafirlukast 20 mg per day for six days after leaving the emergency department were less likely than patients given placebo to require medical care for relapse during the 28-day follow up period (24 versus 29 percent, respectively).

These significant findings are of small magnitude and need to be confirmed in larger trials.

Ineffective therapies — Intravenous methylxanthines and empiric antibiotic therapy are not recommended as treatments for acute asthma exacerbations [2].

Methylxanthines — The use of intravenous methylxanthines, once the standard of care for severe asthmatic attacks, has been shown to be relatively ineffective and is no longer recommended in this setting [2,3,80]. These agents are not as potent as the beta agonists when used alone for the treatment of asthma and provide no further bronchodilation beyond that achieved with inhaled beta agonists alone when used in combination (figure 3) [81,82]. In addition, methylxanthines appear to increase the incidence of adverse effects when combined with beta-agonist bronchodilators [82].
Studies extending over several hours of emergency department care have also failed to show a benefit of theophylline therapy in terms of need for or duration of subsequent hospitalization [figure 4] [83]. For patients who are taking oral theophylline at presentation, we typically continue maintenance oral therapy (and check a theophylline blood level) during hospitalization; but if continued oral intake is not possible, we would very rarely use intravenous therapy with aminophylline or theophylline.

**Empiric antibiotics** — Clinical practice guidelines recommend against empiric antibiotic therapy for the treatment of an asthma exacerbation, because most respiratory infections that trigger an exacerbation of asthma are viral rather than bacterial [84]. However, the trials on which this recommendation is made did not use antibiotics that cover atypical organisms, such as mycoplasma or chlamydia [85]. In general, we reserve antibiotics for treatment of suspected bacterial sinusitis or pneumonia complicating an asthmatic attack. The use of antibiotics in acute asthma is discussed in more detail elsewhere.

**Nebulized furosemide** — Nebulized furosemide has been studied both as prophylactic treatment for patients with exercise-induced bronchoconstriction, and as therapy for acute asthma. Small early studies reported that nebulized furosemide in combination with albuterol was safe, well tolerated, and resulted in significantly improved peak expiratory flow rates compared with albuterol alone [86]. However, subsequent larger trials have not noted a significant clinical benefit when nebulized furosemide was used in conjunction with albuterol or compared to placebo [87-89]. Thus, the therapeutic value of this intervention, if any, appears to be small.

**MECHANICAL VENTILATION AND NONINVASIVE POSITIVE PRESSURE VENTILATION** — The decision to intubate and initiate mechanical ventilation during a severe asthma attack is clinical. Slowing of the respiratory rate, depressed mental status, inability to maintain respiratory effort, worsening hypercapnia and associated respiratory acidosis, or inability to maintain an oxygen saturation >95 percent despite high-flow supplemental oxygen suggest that the patient requires intubation. In the absence of anticipated intubation difficulty, rapid sequence intubation is preferred. Nasal intubation is not recommended.

The goal of mechanical ventilation is to maintain adequate oxygenation and ventilation while minimizing elevated airway pressures. This is accomplished by using low tidal volumes (6 to 8 mL/kg), and low respiratory rates (10 to 12 breaths/minute). The initial inspiratory flow is usually 60 to 80 L/min, but may need to be increased to prolong time for exhalation in patients with more severe obstruction. In some patients, elevations in arterial carbon dioxide tension (PaCO₂) must be tolerated to avoid barotrauma (ie, using the strategy of permissive hypercapnia). (See "Invasive mechanical ventilation in adults with acute exacerbations of asthma", section on ‘Initial ventilator settings’ and "Permissive hypercapnia".)

The role of noninvasive positive pressure ventilation is not as well studied in asthma as in chronic obstructive pulmonary disease and heart failure. (See "Noninvasive positive pressure ventilation in acute respiratory failure in adults", section on ‘Asthma’.)

**DISPOSITION** — Patients with acute, severe asthmatic exacerbations are at risk for further deterioration in lung function, respiratory failure, and asphyxic death. In many cases, airway obstruction remains labile for days following an acute exacerbation, with wide swings in expiratory flow over minutes or hours. Nocturnal deteriorations are common.

**Indications for hospitalization or discharge** — The purpose of hospitalization during an acute exacerbation of asthma is close observation and availability of aggressive interventions in the event of worsening asthma. Hospitalization also serves to remove the patient from stimuli in the home environment that potentially aggravate asthma, ensure medication compliance, and permit inactivity during recovery from the illness.

Identifying patients who should be kept for observation or hospital admission remains largely a matter of clinical judgment guided by the patient’s response to therapy, severity of respiratory symptoms, and degree of airflow limitation, as assessed by peak expiratory flow (PEF). Guidelines from the Expert Panel 3 of the National Asthma Education and Prevention Program (algorithm 2) and from the Global Initiative on Asthma are available to help guide decision making [2,3].

- Patients who have not experienced substantial improvement after four to six hours of urgent care management including frequent inhaled beta-agonist bronchodilator treatments and oral glucocorticoids should be hospitalized.
- Severe symptoms of coughing, wheezing, and shortness of breath that preclude self-care are indications for continued in-hospital care.
● Peak flow measurements provide objective data and are useful for determining which patients are “at risk” for poor outcomes if discharged home and which are safe for transition to home management. The following peak flow parameters can help guide disposition decisions:

- Measured at the time of disposition, a peak expiratory flow less than 40 percent of predicted or of the patient's personal best value is a reason for continued supervised medical care, and consideration of admission to an intensive care setting [2].
- Patients with a peak expiratory flow 40 to 70 percent of predicted, who also demonstrate improving lung function, good asthma self-care skills, and a supportive home environment, can often be discharged home [2]. Other patients with lung function in the same range may be considered unsafe for discharge. Among the factors favoring continued observation in this group are: new onset asthma, multiple prior hospitalizations or emergency department visits for asthma, use of oral glucocorticoids at the time of presentation with the acute deterioration, and complicating psychosocial difficulties.
- Most patients with a peak expiratory flow above 70 percent of normal or their personal best can safely continue their care at home [2].

Medications upon discharge — An asthmatic attack has not fully resolved even when symptoms have abated. Residual airflow obstruction due to airway inflammation may last for several days. Thus, in addition to short-acting beta agonists to be used as needed, the patient will need glucocorticoids to treat the inflammation and prevent recurrent symptoms.

Oral glucocorticoids — Nearly all patients with a significant asthma exacerbation requiring emergency department evaluation should receive a course of oral glucocorticoids for 5 to 10 days [2,3]. A short course of oral glucocorticoids significantly reduces the likelihood of a repeat severe exacerbation with emergency department bounce back (“relapse”) within the succeeding two weeks, and Lessens the frequency of persistent severe symptoms evaluated at a two-week telephone follow-up [90,91]. As an example, one randomized controlled trial demonstrating efficacy used an eight day tapering schedule beginning at methylprednisolone 64mg/day [90].

For glucocorticoid courses lasting three weeks or less, there is no need to taper the dose if patients are also taking inhaled glucocorticoids.

Intramuscular glucocorticoids — Intramuscular injection of a long-acting glucocorticoid formulation at the time of discharge from the emergency department is occasionally used for patients without access to oral medication or at high risk of medical noncompliance. Intramuscular long-acting glucocorticoid formulations appear to be as effective as oral therapy in this setting [92-97]. In a randomized trial of 190 adult patients with acute asthma, intramuscular injection of long-acting methylprednisolone (160 mg) resulted in a similarly low rate of relapse as oral methylprednisolone given in a tapering schedule over eight days (total dose = 160 mg) [92].

A disadvantage of intramuscular glucocorticoids is that the duration of effect varies from one individual to another, and the time that the effect ceases is unknown in a given patient. Cutaneous atrophy at the injection site is also possible.

Inhaled glucocorticoids — Treatment with regular inhaled glucocorticoids constitutes an important method to prevent recurrent asthma attacks after discontinuation of oral glucocorticoids and to prevent the potential decline in lung function associated with any future severe asthma exacerbation [2,42]. Virtually every patient who has suffered an asthma attack severe enough to require urgent care should receive an inhaled glucocorticoid as part of his or her discharge medication plan (table 5). (See "An overview of asthma management".)

Among hospitalized patients, we typically begin (or resume, if previously taking) inhaled glucocorticoids as soon as patients are able to tolerate medication delivery from dry-powder inhalers or metered-dose inhalers (with spacers). The use of combination inhaled glucocorticoids plus long-acting beta-agonists during hospitalization has not been well studied. It seems reasonable to delay their use until administration of short-acting beta agonists has decreased in frequency to fewer than four times per day.

Upon discharge from the hospital or emergency department, some clinicians delay initiation (or reinitiation) of inhaled glucocorticoids until the oral glucocorticoid dose has been reduced to approximately 20 mg of prednisone or the equivalent. However, in our experience this approach more often leads to confusion and medication noncompliance than use of these inhaled medicines in parallel with oral glucocorticoids.

Patient education — Patients should be provided with information about asthma, and if they do not already have one, a personalized action plan (form 1). Follow-up care with a primary provider should be facilitated whenever possible. (See "What do patients need to know about their asthma?".)
INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

• Beyond the Basics topics (see “Patient information: Asthma treatment in adolescents and adults (Beyond the Basics)”, and “Patient information: Trigger avoidance in asthma (Beyond the Basics)”, and “Patient information: How to use a peak flow meter (Beyond the Basics)”, and “Patient information: Asthma inhaler techniques in adults (Beyond the Basics)”)

SUMMARY AND RECOMMENDATIONS — A table outlining the emergency management of severe asthma exacerbations in adults is provided (table 1).

• Early recognition and intervention are critical for successful management of asthma exacerbations. The basic principles of care are assessment of attack severity, repeated use of inhaled short-acting beta-agonists, early administration of oral or intravenous glucocorticoids, and frequent reassessment. (See ‘Introduction’ above.)

• Patients should be taught how to identify symptoms of an asthma exacerbation, and those with poor symptom perception should be taught how to measure peak expiratory flow (PEF). Patients should also learn what steps to take upon recognition of increased asthma symptoms and/or peak flow decline (form 1). These include immediate treatment with short-acting inhaled beta agonists, monitoring of medication response, and early self-administration of oral glucocorticoids, when needed (algorithm 1). (See ‘Initial home treatment’ above.)

• In the urgent care setting, the severity of an asthma exacerbation is assessed based upon symptoms, physical findings, peak expiratory flow (or forced expiratory volume in one second [FEV₁]) measurements, pulse oxygen saturation (SpO₂), and in certain circumstances, arterial blood gas measurement. (See ‘Urgent care assessment’ above.)

• The standard regimen for initial care in the emergency department is supplemental oxygen to maintain the SpO₂ ≥90 percent (>95 percent in pregnancy) and albuterol (or an equivalent) 2.5 to 5 mg by jet nebulization every 20 minutes for three doses, then 2.5 to 10 mg every one to four hours as needed. Alternatively, albuterol can be given by metered dose inhaler (MDI) with a spacer, four to eight puffs every 20 minutes for three doses, then four to eight puffs every one to four hours as needed. For critically ill patients, some clinicians prefer continuous nebulization, administering 10 to 15 mg over one hour. (See ‘Oxygen’ above and ‘Inhaled beta agonists’ above and “Delivery of inhaled medication in adults”, section on ‘Continuous nebulization’.)

• Early systemic glucocorticoids should be given to all patients who have a moderate or severe exacerbation, or in whom inhaled short-acting beta agonists do not fully correct the decrement in peak flow. The optimal dose is unknown; however, the equivalent of a prednisone dose of 40 to 60 mg per day in a single dose or two divided doses is typical for outpatient management. (See ‘Systemic glucocorticoids’ above.)

• The initial dose of systemic glucocorticoids used for the inpatient management of acute asthma exacerbations ranges from methylprednisolone 60 to 80 mg every 6 to 12 hours for patients in the intensive care unit to 40 to 60 mg every 12 to 24 hours for patients not requiring intensive care, depending on the severity of the asthma exacerbation and the patient’s pattern of response to systemic glucocorticoids during previous exacerbations. (See ‘Systemic glucocorticoids’ above.)

• Inhaled ipratropium may be helpful to patients with severe exacerbations who are in the emergency department; ipratropium does not appear to provide additional benefit to inhaled beta agonists during hospitalization. Adult dosing of ipratropium for nebulization is 500 mcg every 20 minutes for three doses, then as needed. Alternatively, ipratropium can be administered by MDI at a dose of four to eight inhalations every 20 minutes, then as needed for up to three hours. (See ‘Inhaled anticholinergics’ above.)

• A one-time infusion of magnesium sulfate, 2 grams administered intravenously over 20 minutes, is suggested for patients who have life-threatening exacerbations (ie, impending intubation for respiratory failure) or those whose exacerbation remains severe after one hour of intensive conventional therapy. (See ‘Magnesium sulfate’ above.)
● We recommend admitting patients to the hospital in a setting with a high level of patient monitoring and care, if they do not respond well after four to six hours of intensive therapy, if they have symptoms of coughing, wheezing, and shortness of breath that preclude self-care, and/or have a PEF <40 percent of predicted or their personal best value. Patients with a PEF ≥40 but <70 percent may need hospitalization, especially if they have new onset asthma, are at high risk for fatal asthma, or presented during a course of oral glucocorticoids. (See 'Indications for hospitalization or discharge' above.)

● Patients who are well enough to go home should be given a brief course of oral glucocorticoids (or an injection of intramuscular glucocorticoids), a prescription for inhaled glucocorticoids, a personalized asthma action plan (NHLBI Asthma Action Plan), and instructions to seek follow-up care (form 1). (See 'Disposition' above.)

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REFERENCES


