Management Of Acute Asthma In The Emergency Department

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Abstract

Asthma is primarily a clinical diagnosis that is made from a combination of historical features and clinical examination findings. The mainstay of asthma treatment includes short-acting beta agonist therapy (albuterol) and steroids. Handheld inhalers are sufficient for most inhaled therapy; all patients on inhalers should be provided with a spacer. The severity of asthma exacerbations is determined by 3 features: (1) clinical presentation, (2) peak expiratory flow rates, and (3) vital signs. Additional testing, such as chest x-ray and blood gas measurements, is reserved for select patients. Spirometry aids in the diagnosis of asthma and measurement of severity, but it is not always required, nor should it be solely relied upon to make disposition decisions. Inhaled ipratropium decreases hospitalization rates, and it should be routinely used. Levalbuterol provides little to no advantage over less-expensive racemic albuterol. Noninvasive positive pressure ventilation may be utilized in patients with moderate to severe exacerbations. Ketamine may be considered in severe exacerbations, but it should not be used routinely. Magnesium sulfate may be beneficial in severe asthma exacerbations, but routine use for mild to moderate exacerbations is not indicated.

CME Objectives

Upon completion of this article, you should be able to:

1. Identify and distinguish key features of mild to severe asthma and triage patients based on initial history and physical examination.
2. Formulate a broad differential diagnosis of new-onset asthma based on a patient's age and comorbidities.
3. Describe the underlying pathophysiology of asthma and the basic treatment options that are critical in the management of asthma.

Prior to beginning this activity, see the back page for faculty disclosures and CME accreditation information.

Case Presentations

A 19-year-old college student presents with marked dyspnea and dysphagia. He reports a history of asthma, for which he takes albuterol as his only medication. Over the last 3 days, he has been coughing and wheezing with increasing severity. Even though he has been using his albuterol inhaler every 2 hours, there has been minimal to no response. EMS administered a 10-mg albuterol nebulizer treatment and magnesium sulfate intravenously en route to the ED. Upon arrival, the patient appears in extremis, and you wonder if there is something you can do to avoid intubation . . .

While establishing IV access and calling respiratory therapy for your first patient, a 24-year-old Hispanic female with a history of asthma who is 15 weeks pregnant presents with tachypnea and acute shortness of breath with audible wheezing. She has been taking albuterol and fluticasone at home with no relief of symptoms. She has a blood pressure of 110/78 mm Hg, heart rate of 110 beats/min, respiratory rate of 40 breaths/min, and pulse oximetry of 93% on room air. Physical exam demonstrates accessory respiratory muscle usage, decreased breath sounds, and expiratory wheezing. You recognize that your patient is at risk for deteriorating, and you wonder which interventions are safest to use in pregnancy . . .

Just as you think you are getting control of your first 2 patients, a 6-year-old girl is brought in by her mother with the chief complaint of "mild bronchitis." Her mother reports that the girl's symptoms began 3 days ago, with initial upper respiratory infection symptoms that progressed to nocturnal cough and mild wheezing. She is otherwise well. According to her mother, the girl has 2 to 3 bouts per year of this "bronchitis" that require emergency care. She has had 2 ED visits within the last year, with no prior hospitalization for her bronchitis. Her vital signs are: blood pressure of 95/55 mm Hg, heart rate of 98 beats/min, respiratory rate of 28 breaths/min, temperature of 37.2°C, and a pulse oximetry of 94% on room air. Her physical exam is only significant for end-expiratory wheezing with no use of accessory muscles and no stridor. The case seems straightforward, but you wonder if there is something you are missing . . .

Introduction

Asthma is the most common chronic respiratory disease, affecting up to 10% of adults and 30% of children in the Western world. Asthma is a worldwide health problem, affecting over 300 million individuals of all ages and ethnicities. It is estimated that, worldwide, 250,000 people die prematurely each year as a result of asthma.[1]

Asthma is a chronic inflammatory disorder of the lungs that is associated with airway hyper-responsiveness that leads to recurrent episodes of wheezing, shortness of breath, chest tightness, and coughing. The airflow obstruction caused by asthma is reversible either spontaneously or with medication.

Asthma is defined by its clinical, physiologic, and pathologic characteristics, with reversible wheezing as the most common finding. From a public health point of view, understanding the underlying causes of asthma and its exacerbants is key to preventive strategies. From an emergency medicine perspective, having clear strategies on how to best manage acute presentations is key to good outcomes. This issue of *Emergency Medicine Practice* provides an evidence-based review of asthma as it relates to emergency department (ED) care and establishes best-practice approaches to management.

Critical Appraisal Of The Literature

The Ovid MEDLINE®, CINAHL (Cumulative Index to Nursing and Allied Health Literature), and PubMed databases were searched using the subject heading *asthma*. Major terms included: *asthma, emergency department, epidemiology, score, treatment, steroid, inhaled, nebulizer,* and *guideline*. The literature search was initially limited to relevant titles from the past 10 years; however, upon finding literature suitable for this review, additional references were added. Additionally, searches were conducted using the minor headings listed throughout this review. Searches identified observational studies, case series, and randomized trials that were available in English. The Cochrane Database of Systematic Reviews was also searched. Reference listings from major textbooks and significant primary literature were reviewed for relevant articles. National Asthma Education and Prevention Program (NAEPP) Expert Panel Report 3 (EPR-3) guidelines were included, and their references were reviewed.

Existing literature on asthma is very broad and spans several decades. Surprisingly, there is a limited amount of new research on acute asthma management that impacts clinical decision-making, and many of the treatments used today have been vetted over several decades. In performing this review, we prioritized data from randomized controlled trials to form recommendations and opinions, but such high-quality evidence was not always available. Given the rarity of severe asthma, studies involving critically ill patients are extremely limited, compared to mild and moderate asthma. As such, the amount of high-quality prospective data are limited, and we were often forced to draw conclusions from literature that is subject to bias.

Currently, the literature on treatments for mild and moderate asthma is robust, and most modalities have been well evaluated. Future studies should focus on severe asthma. Reliable methods for triaging asthma exacerbations do not currently exist, and this is yet another area in need of future

studies. It is also worth noting that most ED-based literature on asthma uses a "working" definition of asthma rather than relying on strict criteria; thus, some of the studies likely enrolled patients with nonasthmatic wheezing.

Epidemiology

Prevalence Of Asthma And Acute Exacerbations

More than 17 million Americans have asthma. Despite the availability of effective therapy for controlling asthma, it continues to be underdiagnosed and undertreated, and its incidence is increasing.[2,3] In the United States, asthma disproportionately burdens socioeconomically disadvantaged urban communities. In developed countries, asthma is more common among individuals who are economically disadvantaged, while in developing countries it is more common among the affluent. Asthma is twice as common in boys as in girls, yet severe asthma occurs at equal rates in children. Women have a higher rate of asthma than men, and asthma is more common in the young than the old.[4-6]

Epidemiology Of Asthma Hospitalizations And Deaths

Hospitalizations for asthma exacerbations are common in the United States, and one-third of all deaths from asthma occur in hospitalized patients.[7] Seasonal trends in asthma-related hospitalizations are widely recognized, with the highest admission rates occurring in the fall and winter months; however, asthma-related intensive care unit (ICU) admissions and intubations remain constant as a percentage of total asthma-related hospitalizations (approximately 10%), and the rates do not decline during the summer months when overall asthma-related hospitalization rates are the lowest.[8-10] Older adult patients with acute asthma exacerbations requiring hospital admission have significant associated morbidity and mortality. Analysis of the 2006 to 2008 Nationwide Emergency Department sample revealed an annual number of inhospital asthma-related deaths of 1144 (0.06%); 101 died in the ED, and 1043 died as inpatients. By age group, there were 37 asthma-related deaths per year in children, 204 in younger adults, and 903 in older adults. After adjusting for comorbidities, older asthma patients had a 5-fold increased risk of overall mortality compared to younger adults.[11]

Although risk factors (see Table 1) are good historical features to inquire about, they should not be relied upon for triaging. One study reviewed 51 consecutive asthma deaths in 1 state in Australia, and it found that 33% of the patients who died had a history of trivial to mild asthma, 32% had never been hospitalized, only 22% had an ICU admission

in the past, and 11% were reported to be symptom-free prior to their terminal hospitalization. Only 1 patient met criteria for severe uncontrolled asthma.[12] Thus, like many diseases presenting to the ED, a lack of risk factors does not necessarily confer a lack of risk.

Etiology And Pathophysiology

Acute Exacerbations

Asthma is a chronic inflammatory disorder of the airways and involves mast cells, eosinophils, T-lymphocytes, neutrophils, and epithelial cells. Bronchial constriction and mucosal edema cause recurrent symptoms of breathlessness, wheezing, chest tightness, and cough. The inflammation appears to be linked to an increase in airway hyperresponsiveness to a variety of environmental stimuli. The diagnosis of asthma is derived from the patient's medical history and results of physical examination, and it is based on episodic symptoms of reversible airflow obstruction and the exclusion of alternative diagnoses. Certain cases in which the diagnosis is not clear may require further diagnostic evaluation, including spirometry, bronchial inhalation challenge tests, blood and sputum studies, chest x-ray examination, or a combination of these procedures.[14-16] However, these tests are generally not required in the ED evaluation of a patient with a known or suspected acute asthma exacerbation.

Viruses have been found in approximately 80% of wheezing episodes in school-aged children and in approximately 50% to 75% of acute wheezing episodes in adults; rhinovirus is the most common virus detected.[17-19] Identifying the trigger is challenging in the ED, and it may not change acute

Table 1. Risk Factors For Death From Asthma[13]

Asthma History
- Previous severe exacerbation (ie, intubation, ICU admission)
- Two or more hospitalizations for asthma in the past year
- Three or more ED visits for asthma in the past year
- Hospitalization or ED visit for asthma in the past month
- Using > 2 canisters of a short-acting beta agonist per month
- Difficulty perceiving asthma symptoms or severity of exacerbations

Social History
- Low socioeconomic status or inner-city residence
- Illicit drug use
- Major psychosocial problems

Comorbidities
- Cardiovascular disease
- Concomitant lung disease
- Chronic psychiatric disease

Abbreviations: ED, emergency department; ICU, intensive care unit.

management. Modifiable triggers that can be identified (eg, allergens, inhalants, and smoking) may help future management.

Spirometry has some limitations to its use in the ED, but it may be used in the proper context to confirm presence, variability, and reversibility of airflow obstruction as well as to measure change in airflow obstruction as changes are made in therapy. Spirometry may also be used to help exclude asthma mimics. In a prospective study of 56 patients, spirometry helped find a new diagnosis in 30% of patients.[20] Asthma is diagnosed when spirometry shows a clinically significant response to bronchodilator use (> 15% peak expiratory flow [PEF] rate), frequently with normalization of values. A methacholine challenge test may be useful in patients who have normal spirometry results despite symptoms suggesting asthma; however, this has no use in the ED.[21-23] Considerations for ordering additional testing must be individualized based on the clinical circumstances.

Comorbid conditions such as sinusitis, seasonal allergies, gastroesophageal reflux disease, and hypothyroidism may worsen asthma.[24,25] A smoking history of > 20 pack-years, even in a patient who has clearly had asthma in the past, should raise suspicion of chronic obstructive pulmonary disease (COPD). Dyspnea alone or exertional chest pain should suggest a diagnosis other than asthma; in particular, this suggests a diagnosis of cardiac or thromboembolic disease. For patients who comply with recommended therapy, poor response to treatment should also raise suspicion as to the correct diagnosis.

Differential Diagnosis

There is significant overlap of asthma clinical features with a variety of different disease processes. The differential diagnoses among adults and children varies, so only common mimics will be discussed here. **(See Table 2.)**

Chronic Obstructive Pulmonary Disease

Acute COPD shares the greatest symptom overlap with acute asthma. It has been shown that > 40% of patients with COPD will report a history of asthma.[26,27] One study linked the presence of asthma to more-frequent respiratory exacerbations in subjects with COPD.[28] Because asthma and COPD are both obstructive pathophysiological processes that are responsive to the same treatments, the clinical distinction between asthma and COPD is not as crucial as rapid recognition of the features of severe obstructive disease.

COPD-related complications add another level of complexity to the undifferentiated patient in respiratory distress. A known complication presenting

to the ED is pneumothorax. Secondary pneumothorax is widely known to occur in patients with significant smoking history, primary lung disease, and primary COPD. It is thought that increased pulmonary pressure due to coughing, with a bronchial plug of mucus or bronchial plug of phlegm, may play a role. The incidence of an underlying pulmonary disease process (such as COPD) is higher in patients with spontaneous pneumothoraces.[29]

Acute Decompensated Heart Failure

Acute decompensated heart failure (ADHF) should remain high on the differential diagnosis list for a patient presenting with dyspnea. ADHF presenting with wheezing is often known as "cardiac asthma," and it can greatly obscure the clinical picture in the elderly population.[30] Some studies have postulated that ADHF with cardiogenic pulmonary edema causes wheezing due to elevation of pulmonary or bronchial vascular pressure that results in reflex bronchoconstriction, decrease in airway size from reduced lung volume, obstruction from intraluminal edema fluid, and bronchial mucosal swelling.[31-36] One study comparing the presentation prevalence, ED identification, and management of ADHF in elderly patients presenting with cardiac asthma or classic pulmonary edema features showed that 48% of patients with cardiac asthma received bronchodilators, in contrast to 14% of patients with classic heart failure.[31] This significant overlap emphasizes the clinical dilemma often faced by emergency clinicians and underlines the importance of a broad initial differential diagnosis.

Table 2. Differential Diagnosis Of Wheezing In Adults And Children

Adults
- Chronic obstructive pulmonary disease
- Acute coronary syndromes
- Congestive heart failure
- Pulmonary embolism
- Pneumothorax
- Pneumonia
- Airway foreign body
- Gastroesophageal reflux disease
- Vocal cord dysfunction
- Cystic fibrosis
- Chronic bronchitis
- Sinus disease
- Upper respiratory tract infection

Children
- Croup
- Viral and bacterial pneumonia
- Airway foreign body
- Bronchiolitis
- Tracheomalacia
- Viral upper respiratory tract infection

Other Disease Processes In The Differential Diagnosis

Other important processes seen in the ED that should be high on the differential diagnosis list include acute coronary syndromes, pulmonary embolism, and pneumonia. One recent study noted

Table 3. Common Pediatric Complaints Misdiagnosed As Asthma

Complaint	Features
Vocal cord dysfunction	• Intermittent shortness of breath, wheezing, stridor, cough • Commonly presents in adolescent females[38] • Has little to no response to bronchodilators
Croup	• Generally involves the larynx and trachea • May present with wheezing, but typically has a characteristic "barking" cough with inspiratory stridor • Acute onset with fever
Bronchiolitis	• Commonly mistaken for asthma • High prevalence in children aged < 2 y, with a peak age of 3-6 mo • Low-grade fever • Tends to sound harsher and less melodious • Occurs with other upper respiratory infection-like symptoms, ie, congestion, coryza, etc[39]
Airway foreign body	• History may reveal symptomless period followed by paroxysms of respiratory distress • Sudden onset of asthma-like symptoms, including wheezing, coughing, and choking • Recurrent or nonresolving pneumonia • Failure to improve with standard asthma therapies[40]
Tracheomalacia	• Usually diagnosed during first 2 mo of life • Clinical features of respiratory distress, tachypnea, and accessory muscle usage rarely seen • Strong inspiratory component • Wheeze severity is usually activity-dependent: better with rest, worse with activity • Minimal to no improvement with bronchodilators; may even cause worsening[41]
Gastroesophageal reflux disease	• Cough, recurrent bronchitis, pneumonia, wheezing, and asthma are commonly associated • Incidence among patients with asthma ranges from 38%-48%[42]
Bacterial and viral pneumonia	• May cause transient wheezing in children aged < 3 y due to physiologically narrow airways • Less important to distinguish from asthma; more important to identify respiratory distress

a 3-fold increased risk of pulmonary embolism in patients with severe asthma exacerbations and asthma with chronic oral steroid use.[37] Whether the increased incidence may be due to testing bias is unclear. All of these can initially present similarly to asthma exacerbations, with dyspnea, hypoxia, tachycardia, and chest pain. In addition, given the airway hyperresponsiveness well known in the pathophysiology of asthma, all of these conditions can act as triggers and should be clinically excluded.

Effective management of a patient in respiratory distress requires a thorough understanding of initial clinical features and rapid development of a broad differential diagnosis list that is population specific. **(See Tables 2 and 3.)**

Prehospital Care

Development of emergency medical services (EMS) protocols for asthma and training of personnel are largely based on data from ED management. Given the paucity of testing available in the field, EMS providers must rely on their history and physical examination to guide therapy and management even more than emergency clinicians. Inhaled short-acting beta agonists are the mainstay of therapy. EMS providers can give this medication safely, and prehospital administration has been shown to improve symptoms prior to ED arrival.[43,44] All EMS providers should have a standing order to administer short-acting beta agonists for suspected asthma exacerbations via nebulizer or metered-dose inhaler with spacer. However, administration of short-acting beta agonists should not delay transport to an ED. If inhaled treatments are not available, subcutaneous or intramuscular epinephrine can be considered.[13] There is limited evidence on the use of intramuscular epinephrine in asthma compared to other standard treatments. In the past, subcutaneous epinephrine was considered the standard treatment modality for asthma; however, more recent evidence suggests that intramuscular epinephrine in the thigh has more rapid absorption.[45] Therefore, we recommend the intramuscular route.

There is no high-quality evidence for or against the use of magnesium (intravenous [IV] and inhaled) in the prehospital setting. The use of magnesium in the ED will be explored in later sections. The benefits of magnesium appear to be limited to severe asthma and only with respect to hospital admission rates; thus, it probably has little to no use for most EMS systems with shorter transport times. Unless extreme transport times are present, we recommend against the use of magnesium in the prehospital setting.

A fear among some EMS providers is giving albuterol to a dyspneic patient without a clear diagnosis of asthma, given some symptom overlap with

Clinical Pathway For Management Of Asthma In The Emergency Department

- Initial history (including detailed asthma history) and physical examination
- Vital signs, including oxygen saturation, heart rate, and respiratory rate
- Consider PEF or ETCO$_2$ monitoring

Mild
- Mild end-expiratory wheezing only
- Oxygenation > 90%
- Minimal to no use of accessory muscles
- Vital signs within normal limits
- Speaking in full sentences
- FEV1 or PEF > 70% predicted

Moderate
- Oxygenation > 90%
- Accessory muscle usage but still able to speak
- Elevated respiratory rate
- Elevated heart rate
- FEV1 or PEF 40%-69% predicted

Severe
- Oxygenation < 90%
- Significant accessory muscle usage
- Vital signs with significant stress
- Altered mental status
- FEV1 or PEF < 40% predicted

- Give inhaled SABA by nebulizer **or** MDI + spacer
- Administer first dose of oral steroids
(Class I)

- High-dose SABA + ipratropium by nebulizer **or** MDI + spacer every 20 min for first hour
- Administer first dose of oral steroids immediately
(Class I)

- High-dose SABA + ipratropium by nebulizer **or** MDI + spacer every 20 min for first hour
- Consider continuous nebulized albuterol therapy if no clinical improvement with intermittent therapy
- Administer first dose of oral steroids immediately
- Consider magnesium IV and adjunctive therapies
(Class I)

- Reassess
- Improved?

YES **NO**

- Reassess
- Improved after 1 h?

YES **NO**

- Reassess
- Improved?

YES **NO**

Continue to "moderate" path

- Admit to hospital ward
- If worsening, move to "severe" path

Admit to hospital ward

- Consider initiating ICS or adjusting current dose as indicated
- Continue treatment with inhaled SABA, 2-6 puffs every 3-4 h, as needed
- Discharge home with:
  - Continued oral steroid therapy for 5 days
  - Clear and simple return precautions
  - Reliable follow-up
  - Instruction on proper technique for using inhaled medication with spacer
(Class I)

- Continue current therapy
- Make admit vs discharge decision < 4 h from arrival
- If stable in < 4 h and ready for discharge, refer to "mild" path for discharge planning
- If worsening, move to "severe" path
(Class II)

Evidence of impending or actual respiratory arrest:
- Prepare for intubation without delay
- Continue inhaled SABA while preparing for intubation
- Recommended RSI medications: ketamine 2 mg/kg + rocuronium **or** succinylcholine
- If not requiring intubation at this time, consider starting NIPPV
- If not improved, consider admission to hospital ICU
(Class II)

See class of evidence descriptions on page 7.

Abbreviations: ETCO$_2$, end-tidal carbon dioxide; ICS, inhaled corticosteroids; IV, intravenous; MDI, metered-dose inhaler; NIPPV, noninvasive positive-pressure ventilation; PEF, peak expiratory flow; RSI, rapid sequence intubation; SABA, short-acting beta agonist; FEV1, forced expiratory volume in 1 second.

ADHF. In ADHF, albuterol appears to be safe and may even improve symptoms, despite the different pathophysiology.[44,46] Available evidence suggests that inhaled albuterol should not be withheld from a dyspneic patient if asthma is suspected, despite diagnostic uncertainty.

Corticosteroid administration should be decided by individual EMS systems based on transport times and access to care. The overwhelming majority of EMS systems do not appear to administer steroids prehospital.[47] We recommend against the use of corticosteroids by prehospital providers where transport times do not exceed 45 to 60 minutes because it is unlikely that any benefit would be seen during that transport period. However, most therapies will be limited based on scope-of-practice limitations to the EMS providers. There is no evidence to guide EMS "treat-and-street" protocols, and given the associated risks, we recommend against this.

Emergency Department Evaluation

Asthma typically presents with wheezing in combination with other symptoms such as cough, dyspnea, tachypnea, tachycardia, agitation, hypercapnia, hypoxia, accessory muscle use, nasal flaring, chest pain/tightness, decreased PEF, and decreased forced expiratory volume in 1 second (FEV1). According to the NAEPP EPR-3, combinations of these symptoms in addition to functional assessment using arterial oxygen pressure (PaO_2), arterial oxygen saturation (SaO_2), PEF rates, and partial pressure of carbon dioxide (PCO_2) can be used to categorize an acute asthma exacerbation as moderate, mild, or severe.[13] Features such as jugular venous distention, lower extremity edema, and dyspnea without wheezing may suggest another etiology.

Historical features, in addition to common clinical manifestations, are vital in the initial assessment of asthma in the ED. Pulsus paradoxus, while classically discussed, has no role in triaging severity. Patients should be asked about a known diagnosis of asthma, the duration of their symptoms, and the frequency of use of asthma control medications. In addition, questions about a patient's history of severe respiratory distress, history of intubation, comorbid conditions (eg, COPD, heart failure, pneumonia), history of atopic symptoms (eg, seasonal rhinorrhea, eczema in children, and conjunctival congestion) are crucial to guiding early diagnosis and treatment.

Mild Exacerbations

Clinical manifestations can be limited to end expiratory wheezing on auscultation, dyspnea on exertion, tachycardia, mild agitation, and normal or near-normal vital signs.[13] Subjective chest tightness and intermittent cough are also common findings. A wheeze is defined as a continuous high-pitched sound with a musical quality that emits from the chest during expiration.[48] There is no validated method for specifically quantifying/qualifying wheezing alone with regards to severity of an asthma exacerbation. Despite wheezing being a common finding in asthma, it is an unreliable indicator of asthma severity. Distal airway inflammation or obstruction may not produce wheezing, as is often seen in exercise-induced asthma.[48] Cough is typically nonproductive, and it is often increased with agitation and exertion and at night or in early morning.

Moderate Exacerbations

The distinction between mild and moderate asthma exacerbations can be measured by the presence of the aforementioned features plus the addition of mild accessory muscle use, tachycardia, and/or conversational dyspnea. With regard to the pediatric and infant population, SaO_2 is crucial, as the other features can be less predictive of severity on initial presentation.[49] Other signs of moderate to severe

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA*. 1992;268(16):2289-2295.

asthma in infants include paradoxical breathing, accessory muscle use, cyanosis, and respiratory rate > 60 breaths/min.

While functional measurements with PEF and FEV1 can be valuable in categorizing severity of asthma and can aid in identifying patients who require hospitalization, they are not routinely used in the ED. In patients experiencing moderate to severe respiratory distress, accurate results for functional tests are difficult for the patient and are an unrealistic goal.[13] In the pediatric population, PEF rates have been shown to be largely difficult to obtain. One study revealed that 65% of children between 5 and 18 years of age were unable to perform this test.[50] Another ED-based study found that over 70% of children aged 6 years and nearly 20% of children aged 17 years (with all ages in between) were unable to provide reliable measurements.[51] Additionally, an inpatient study compared FEV1/FEV25-50 (the gold standard diagnostic criteria used in this study) to PEF measurements. Normal PEF measurements yielded a negative predictive value of 88% in children with mild air trapping and a negative predictive value of only 53% in children with severe air trapping.[52] Thus, when compared to the gold standard, the value of PEF measurements in acute asthma is limited. Asthma remains, largely, a clinical diagnosis. Functional measurements add limited triaging value to the early management in the ED and must be used in the proper clinical context.

Severe Exacerbations

Cardinal features of severe asthma and/or impending respiratory failure include hypoxia, severe agitation, limited air movement on auscultation with absence of wheezing, nasal flaring, severe accessory muscle use with signs of exhaustion, hypercapnia ($PaCO_2$ of > 42 mm Hg), cyanosis, upright sitting position, and altered mental status.[13] Though patients with signs of severe asthma exacerbation are at risk for respiratory failure, they are at a greater risk for impending respiratory failure if they: (1) are known to be noncompliant with medications, (2) have been intubated in the past for asthma, or (3) report a long duration of symptoms prior to presentation to the ED. The emergency clinician should recognize manifestations of severe asthma and impending respiratory failure within the first few seconds of initial examination.

Diagnostic Studies

Laboratory Studies

Laboratory studies are unlikely to add to the management of acute asthma exacerbations except in cases with comorbid disease or when the diagnosis is unclear. A complete blood count may be helpful in patients with purulent sputum or fever. Theophylline levels are necessary in patients treated with theophylline due to the narrow therapeutic index. Consider electrolyte evaluation in patients taking diuretics or with renal disease, as beta agonists can decrease levels of potassium, magnesium, and phosphate.[53]

Other laboratory studies, such as brain-natriuretic peptide (BNP) and D-dimer, as well as electrocardiogram may be considered when the diagnosis is unclear or when other clinical indications exist. Without specific clinical indications, these tests should not be routinely ordered.

Arterial Blood Gas

Initial blood gas findings in severe status asthmaticus are hypoxia and hypocarbia. As symptoms progress, hypercarbia occurs. The finding of hypercarbia on blood gas analysis is a strong predictor of impending respiratory failure. Metabolic acidosis on blood gas is an indicator of impending arrest.[53] Nonetheless, the decision to intubate and mechanically ventilate the asthma patient should not be based on blood gas analysis; rather, it should be based on the clinical picture of mental status, respiratory effort, and air movement. After a patient is intubated, further monitoring of the clinical course in an obtunded patient is best performed with blood gas analysis.[54] Arterial blood gas sampling can be very painful for patients, and it provides little data above other noninvasive clinical markers in most patients.[55,56] Venous blood gas sampling is less painful and can be used as a reliable screening test to evaluate for acidosis and hypercarbia.[57]

End-Tidal Carbon Dioxide Monitoring

Acute asthma exacerbation typically results in hypocarbia due to hyperventilation. Consequently, end-tidal monitoring in an acute asthma exacerbation should demonstrate low carbon dioxide (CO_2) levels. Normal or increased CO_2 levels are concerning for impending respiratory failure. Langhan et al noted a correlation of lower end-tidal carbon dioxide ($ETCO_2$) levels with severity of asthma, and $ETCO_2$ levels < 30 mm Hg were associated with more-severe exacerbation and need for inpatient treatment.[58] Kunkov et al described a novel approach, utilizing the ratio of the length of the plateau portion of the exhalation divided by the respiratory rate. Ratios of < 0.15 were predictive of admission.[59] Limited-quality data also suggest that $ETCO_2$ monitoring may be a substitute for blood gas analysis when evaluating for hypercarbia.[60] Though promising, these methods lack large-scale validation; thus, we cannot recommend them for routine use at this time.

Chest Radiography

White et al reported that the admission chest radiograph for acute asthma patients demonstrated an ab-

normality 34% of the time. Abnormalities included increased interstitial markings, focal infiltrates, and pneumothorax.[61] However, of the 20 radiographs with abnormalities, changes in clinical management occurred in only 8 patients. This study was limited to patients admitted for asthma exacerbations and thus represented a group with more-severe disease; results should be interpreted accordingly. Gershel et al performed a prospective study of pediatric patients aged > 1 year with a first-time episode of wheezing. Out of 350 patients, 21 had abnormalities on chest x-ray; only 7 were found to have pneumonia, and the most common finding was atelectasis. No pneumothoraces or foreign bodies were found. Of the positive films, the authors noted that 95% of the patients had 1 or more of the following findings: respiratory rate > 60 breaths/min, pulse > 160 beats/min, localized rales, or decreased breath sounds.[62] Available data suggest that clinicians can rely on the history and clinical examination to determine whether a chest x-ray is indicated; chest x-rays should not be considered routine. A lower threshold for ordering a chest film is reasonable in patients requiring admission, but they should not be considered an absolute requirement.

Peak-Flow Measurements

Peak flow measurements are advocated by NAEPP guidelines[13] and are relatively easy to perform. The equipment is inexpensive and portable, allowing the patient to perform measurements at home and incorporate peak flow measurement into an asthma action plan. Several studies have documented the utility of peak flow measurements in aiding disposition decisions.[63-65] A recent study found that 83% of the total increase in peak flow in asthma patients occurs after the first inhalation treatment, suggesting limited value in serial measurements of peak flow after initial treatment.[66] In a study of 1825 asthma patients, the disposition was dictated by 4 variables: (1) history of asthma admission, (2) oxygen saturation rates, (3) initial treatment peak flow rates, and (4) posttreatment peak flow rates.[67] **(See Table 4 for the classification system.)** In cases of moderate to severe asthma exacerbations, both adult and pediatric patients may not be able to perform this functional test, which limits its applicability. Thus, PEF has limitations and may under-triage severe exacerbations. We suggest that PEF be used to provide additional data points, but it should not be relied upon in isolation to make disposition decisions. If PEF measurements are going to be used, serial measurements are likely to be of greater value than single, isolated measurements. The literature on PEF rate use in the ED is very mixed; thus, it is challenging to draw any definitive conclusions from it. In our opinion, routine PEF measurements should not be considered the standard of care at this time.

Treatment

Beta Agonists

Short-acting beta agonists (the most common of which is albuterol) are the cornerstone of asthma therapy in the ED.[68-71] **(See Table 5, pages 10-11, for suggested drug dosing.)** Despite the fact that most emergency clinicians are familiar with short-acting beta agonists, controversy still exists over optimal drug selection, routes, and methods of administration. Long-acting beta agonists (eg, salmeterol) have no role in the ED.[13,72] Several studies have found an association between long-acting beta agonist use and asthma-related deaths.[73] Whether this association is from the medication itself or whether the selection of patients had more significant chronic disease is unclear at this time. A postmarketing surveillance study on salmeterol by the manufacturer was halted early due to a strong association between its use and asthma-related deaths. Since that time, the manufacturer has voluntarily used a United States Food and Drug Administration (FDA) "black box" warning on salmeterol.[74]

Racemic Albuterol Versus Levalbuterol

Racemic albuterol has been the primary short-acting beta agonist in use for more than 40 years.[75] Standard racemic albuterol exists as 2 nonsuperimposable enantiomers. The racemic form contains equal parts of R- and S-albuterol.[76] In 1999, a formulation of isolated R-albuterol became commercially available as a nebulized solution.[76] Levalbuterol was marketed as having a better side-effect profile (specifically regarding tachycardia) from more beta selectivity and less of the deleterious effects of S-albuterol. This came with a significant increase in cost.[76] At the authors' hospital's pharmacy, levalbuterol costs > 8 times the price of racemic albuterol solution.

Bench studies have suggested that S-albuterol has detrimental effects, including increased airway smooth muscle contraction potential and internalization of receptors.[77] However, studies comparing the

Table 4. Suggested Peak Flow Rate When Height And Baseline Measurements Are Not Available

Asthma Severity	Peak Expiratory Flow (L/min)	
	Men	Women
Mild	≥ 400	≥ 300
Moderate	250 to 399	200 to 299
Severe	150 to 249	120 to 199
Very severe	< 150	< 120

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clinical effects of R-albuterol to racemic albuterol have lacked significant findings. One study found a difference when asthma patients were given a methacholine challenge, but it was only statistically significant at the 180-minute time mark, and the statistical methodology of this study has been debated.[78] The sum of current available evidence does not support this claim.

Hardasamalani et al randomized patients aged 5 to 21 years with acute exacerbations to receive up to 3 treatments of levalbuterol or albuterol plus standard therapy. No differences in oxygen saturations, respiratory rates, or peak flow rates were found.[79] Qureshi et al's randomized controlled study involved 129 children aged 2 to 14 years with moderate to severe exacerbations. No differences were

found on any of the measured outcomes, including clinical scores and FEV1.[80] Andrews et al randomized 81 children aged 6 to 18 years with severe asthma requiring continuous therapy to racemic albuterol or levalbuterol. There was no difference in time on continuous therapy or change in pulse rate, serum potassium, or serum glucose.[81]

Nowak et al randomized adults with acute exacerbations, comparing the 2 formulations plus standard therapy. No differences were found in time to ED discharge, hospitalization rate, or relapse rates. A clinically insignificant improvement in the percentage of predicted FEV1 in a subset of patients on chronic inhaled steroids was noted.[82] Thompson et al randomized adults admitted with acute asthma or COPD exacerbations, and

Table 5. Suggested Drug Dosing Guide For The Treatment Of Asthma[13] (Continued on page 11)

Medication	Pediatric Dose	Adult Dose	Pharmacokinetics	Comments
Albuterol nebulizer solution	• 0.15 mg/kg (minimum 2.5 mg) every 20 min for up to 3 doses, then 0.15-0.3 mg/kg every 1-4 h as needed • For continuous use, 0.5 mg/kg/h	• 2.5-5 mg every 20 minutes up to 3 doses, then 2.5-10 mg every 1-4 h as needed • For continuous use, 10-15 mg/h	• Onset of action: 5-15 min • Peak effect: 0.5-2 h • Duration of action: 3-4 h	• Set gas flow rate at 6-8 L/min • May mix ipratropium into same solution
Albuterol MDI (90 mcg/puff)	• 4-8 puffs every 20 min for 3 doses, then as needed every 1-4 h	• 4-8 puffs every 20 min up to 4 h, then every 1-4 h after	• Onset of action: 5-15 min • Peak effect: 25 min • Duration of action: 3-4 h	• Spacer should be used with all pediatric patients and most adult patients
Levalbuterol nebulizer solution	• 0.075 mg/kg (minimum 1.25 mg) every 20 min up to 3 doses, then 0.075-0.15 mg/kg up to 5 mg every 1-4 h	• 1.25-2.5 mg every 20 min for 3 doses, then 1.25-5 mg every 1-4 h	• Onset of action: 10-17 min • Peak effect: 1.5 h • Duration of action: 5-6 h	• Dosing is similar to albuterol; use half the mg dose
Levalbuterol MDI	• 4-8 puffs every 20 min for 3 doses, then as needed every 1-4 h	• 4-8 puffs every 20 min up to 4 h, then every 1-4 h after	• Onset of action: 5-10 min • Peak effect: 77 min • Duration of action: 3-4 h	• Spacer should be used with all pediatric patients and most adult patients
Epinephrine 1:1000 (1 mg/mL)	• 0.01 mg/kg up to 0.3-0.5 mg every 20 min for 3 doses, SQ	• 0.3-0.5 mg every 20 min for 3 doses, SQ	• Onset of action: 5-10 min • Peak effect: 1 h • Duration of action: 4 h	• No proven advantage over inhaled SABAs
Terbutaline (1 mg/mL)	• 0.01 mg/kg every 20 min for 3 doses, then every 2-6 h as needed, SQ	• 0.25 mg every 20 min for 3 doses, SQ	• Onset of action: 15 min • Peak effect: 30-60 min • Duration of action: 90 min to 4 h	• No proven advantage over inhaled SABAs
Ipratropium bromide (0.5 mg/2.5mL or 0.2 mg/mL nebulizer)	• Age < 12 y: 1.25 mL every 6 h • Age ≥ 12 y: dose as adult	• 2.5 mL every 6 h	• Onset of action: 15 min • Peak action: 1-3 h • Duration of action: 3-4	• May mix with albuterol solution in nebulizer

the intent-to-treat analysis found no difference in pulmonary function testing, symptom evaluation by multiple scoring systems, total hospital cost, hospital length of stay, unscheduled additional treatments, or adverse side effects. Additional pre-hospital data suggest no differences in efficacy by peak flow measurements.[83]

Tripp et al randomized adult patients with chronic asthma in a crossover study. After 16 doses by metered-dose inhaler, the mean increase in heart rate was 3.5 beats/min higher in the racemic group.[84] Lam et al compared the 2 formulations in ICU patients and found no difference in pulse changes.[85] Khorfan et al randomized adults to 1 of the 2 formulations (in addition to both receiving ipratropium) and found no significant differences in rates of tachycardia or tachyarrhythmias. Premature ventricular contractions were noted in both groups.[86]

The sum of available evidence suggests that there is little to no difference in clinical efficacy or safety between racemic albuterol and levalbuterol, and the use of levalbuterol is almost certainly not justified because of the increased cost.

Holding Chambers

A Cochrane review found that metered-dose inhalers with spacers are at least as effective, and likely more effective, than nebulized medications.[87] We recommend that spacers be routinely prescribed and used with inhalers, especially for young children. Bedside education is highly encouraged.

Table 5. Suggested Drug Dosing Guide For The Treatment Of Asthma[13] (Continued from page 10)

Medication	Pediatric Dose	Adult Dose	Pharmacokinetics	Comments
Ipratropium bromide MDI (18 mcg/puff)	• 4-8 puffs every 20 min as needed, for 3 h	• 8 puffs every 20 minutes, as needed, for 3 h	• Onset of action: 15 min • Peak effect: 1-2 h • Duration 2-5 h	• Use with spacer as indicated
Albuterol with ipratropium nebulizer solution (each 3 mL contains 0.5 mg ipratropium bromide and 2.5 mg albuterol)	• 1.5-3 mL every 20 min for 3 doses, then as needed	• 3 mL every 20 min for 3 doses, then as needed	• See individual drugs	• May be used for up to 3 h in initial management • No proven advantage beyond 3 h
Albuterol with ipratropium MDI (each puff contains 18 mcg ipratropium bromide and 90 mcg albuterol)	• 4-8 puffs every 20 min as needed, up to 3 h	• 8 puffs every 20 min as needed, up to 3 h	• See individual drugs	Not applicable
Ketamine	• 0.3 mg/kg to maximum dose of 25 mg IV bolus, then 0.5 mg/kg/h for 2 h	• 0.3 mg/kg to maximum dose of 25 mg IV bolus, then 0.5 mg/kg/h for 2 h	• Onset of action: 15 sec • Duration of action: 5-10 min	• This is low-dose ketamine NOT for intubation; may slow bolus to slow IV push over 5 min to reduce risk of dissociative effects • Induction dose: 1-2 mg/kg
Magnesium sulfate	• 25-75 mg/kg up to 2 g IV over 20 min	• 2g IV over 20 min	• Onset of action: immediate • Duration of action: 30 min	• No benefit in mild to moderate exacerbations; reserve for severe exacerbations
Prednisone	• 1-2 mg/kg divided into 2 doses PO, maximum dose 60 mg/day	• 40-80 mg/day divided into 1-2 doses	• Peak plasma: 1-3 h • Duration of action: biologic 8-36 h	• Outpatient "burst" 3-10 days, depending on severity
Methylprednisolone	• 1-2 mg/kg divided into 2 doses, maximum dose 60 mg/day	• 40-80 mg/day divided into 1-2 doses	• Peak effect: 1-2 h • Duration of action: biologic 30-36 h	• Outpatient "burst" 3-10 days, depending on severity
Prednisolone	• 1-2 mg/kg divided into 2 doses PO, maximum dose 60 mg/day	• 40-80 mg/day divided into 1-2 doses PO	• Peak plasma: 1 h • Duration of action: biologic 18-36 h	• Outpatient "burst" 3-10 days, depending on severity
Dexamethasone	• 0.6 mg/kg, to maximum dose of 16 mg, for 2 doses	• 16 mg for 2 doses	• Peak plasma: 1-2 h • Duration of action: biologic 36-54 h	• Give 1 dose in ED and 1 dose in the next 1-2 days

Abbreviations: IV, intravenous; PO, by mouth; SABA, short-acting beta agonist; SQ, subcutaneous.

Table adapted from National Asthma Education and Prevention Program Expert Panel Report 3 guidelines[13] with revisions made based on literature discussed.

11

Metered-Dose Inhalers Versus Nebulizers

The debate often arises on use of metered-dose inhalers versus nebulizer treatments for patients in the ED and at home. Most patients do not have nebulizer equipment at home. A Cochrane review on the use of a metered-dose inhaler with spacer versus nebulizer found no difference in hospitalization rates and no difference in peak flow changes or FEV1 changes; length of stay was similar for adults. They did find that, for children, the ED length of stay was shorter by 0.53 hours in the metered-dose inhaler with spacer group.[87] Even in severe asthma exacerbations, they have equal efficacy.[88] One hospital switched from a protocol using nebulizers to metered-dose inhalers, plus spacers, over an 18-month period and found that the switch resulted in improvements in peak flow measurements, decreased ED length of stay, decreased number of treatments required, improved discharge oxygen saturation measurements, and decreased bounce-back rates at 14 and 21 days.[89]

Administration by metered-dose inhaler plus spacer requires less usage of hospital personnel,[90] and it can be used as an opportunity to review with patients and parents the proper administration of the medication.[13] Prospective audit studies have found that nebulizers cost an average of $0.89 more per treatment in personnel usage.[91] Moreover, < 10% of nebulizer machine owners ever receive the recommended servicing and cleaning of their machines.[92] Given the available evidence, we recommend the use of a metered-dose inhaler plus spacer over a nebulizer for both adults and children with asthma.

Intermittent Versus Continuous Nebulizer Treatments

A Cochrane review suggests that continuous nebulizer treatments in patients with severe exacerbations may produce a modest reduction in hospitalization rates.[93] We suggest that this type of therapy be reserved for severe asthma.

Inhaled Versus Intravenous Or Subcutaneous Beta Agonists

A Cochrane review of IV and subcutaneous formulations of beta agonists found them to be inferior to inhaled therapy when they were evaluated for improvements in peak flow measurements. In addition, there was no improvement in autonomic side effects by using the IV or subcutaneous route. Neither the available evidence nor NAEPP guidelines support the routine use of injectable beta agonists in asthma of all levels of severity.[13,94]

Recently, an FDA warning was released advising of the risks associated with the use of terbutaline for preterm labor, including increased heart rate, transient hyperglycemia, hypokalemia, cardiac arrhythmias, pulmonary edema, and myocardial ischemia. While the indication discussed here is not for obstetric use, the side effects of the medication are not unique to pregnant women, and clinicians must weigh the risks and benefits.[95]

Ipratropium Bromide

Ipratropium bromide works by blocking the cholinergic stimulation of airway smooth muscle. When used alone, its efficacy in comparison to albuterol is clearly inferior; however, combination therapy (both metered-dose inhaler and nebulized) with albuterol when compared to albuterol alone has been shown in a Cochrane review, multiple studies, and meta-analyses to have improvements in pulmonary function measurements as well as decreased hospitalization rates.[96-100] The safety profile of ipratropium is excellent, with very little effect on heart rate.[101] Most of the randomized controlled trials use 1 to 3 combination therapy treatments in the intervention arm, so benefit beyond 3 consecutive treatments is not clear. We recommend ipratropium bromide be added to short-acting beta agonist therapy. **(See Table 5, pages 10-11, for suggested dosing.)**

Corticosteroids

Corticosteroids are a mainstay of therapy in acute asthma exacerbations. They work via 2 pathways: the reduction in airway inflammation and upregulation of beta receptors. A Cochrane review clearly demonstrated that the use of corticosteroids reduced relapse rates, hospitalization rates, and the use of short-acting beta agonists.[102] There is no preferred route of steroid administration, as oral, intramuscular, and IV routes all appear to have equal efficacy. Additionally, there is no evidence suggesting that high-dose steroids confer any benefit over standard lower doses.[103-105] **(See Table 5, pages 10-11, for suggested dosing.)**

Early administration of steroids in acute asthma has a direct correlation on hospitalization rates. Delays in steroid therapy of > 1 hour after presentation place patients at higher risk for needing admission.[106,107] Preliminary evidence suggested that inhaled corticosteroid therapy may be effective in the treatment of acute asthma, but a Cochrane review found the evidence insufficient to recommend routine use.[108,109] If a patient is already on chronic inhaled corticosteroid therapy, NAEPP guidelines suggest giving 4-fold their usual dose.[13]

Single-dose dexamethasone at 0.6 mg/kg (up to 18 mg), by both oral and intramuscular routes, is as effective as 3-day or 5-day therapy with prednisone.[110-112] Nonadherence to medication regimens with prednisone therapy is a significant problem that this treatment method solves.[113] For return to normal activity, 2 days of dexamethasone at 16 mg/day is superior to 5 days of prednisone at 50 mg daily.[114] Given the available evidence, we recom-

mend dexamethasone over other regimens for outpatient treatment.

We suggest that any patient that comes to the ED for asthma exacerbation warrants steroid therapy. There is very minimal downside to short-term steroid use. Side effects (such as hyperglycemia and psychosis) are dose- and duration-dependent.[115] Two reviews of 46 patients and 390 patients premedicated with corticosteroids for computed tomography scanning with IV contrast demonstrated an increase in serum glucose of minimal clinical significance in both diabetics and nondiabetics that resolved within 24 to 48 hours.[116,117]

Magnesium Sulfate

Magnesium sulfate is thought to work by inhibiting smooth muscle action potential.[118] Based on 2 Cochrane reviews, both IV and inhaled magnesium are effective for the treatment of asthma. Nonetheless, this benefit is subject to significant spectrum bias, as its efficacy is only consistently seen in severe exacerbations.[119,120] At this time, there is insufficient evidence of head-to-head comparison of IV versus inhaled formulations to suggest that one is superior to the other. We suggest that magnesium be used in severe exacerbations via whichever route is most clinically feasible.

Epinephrine

Several studies have compared the use of nebulized epinephrine versus nebulized short-acting beta agonists, but they failed to find a significant difference in efficacy or safety.[121-125] IV and subcutaneous epinephrine have been shown to be efficacious for the treatment of asthma.[126] Epinephrine appears to be safe to use in the elderly, with very little risk of ventricular dysrhythmias or other significant adverse events.[127,128] This should not be considered a first-line therapy, but it may be considered an adjunctive therapy that may be beneficial to give when the severity of the exacerbation limits the amount of nebulized medication that can be delivered due to air-flow obstruction. However, contemporary evidence is limited, and most of the data are from older studies. Newer data demonstrate more rapid absorption via the intramuscular route than the subcutaneous route.[45] **(See Table 5, pages 10-11, for suggested dosing.)**

Ventilation

The decision to intubate a critically ill asthma patient is challenging, given the continued difficulty in ventilation along with a myriad of complications and mortality risk.[129] In life-threatening asthma, FEV1 and peak flow measurements are not indicated, since the clinical presentation should replace the need for objective data. The decision to intubate is based on clinical judgment, and no single data point should be used to substitute for clinical judgment. Relative indications for intubation include worsening hypercapnea, exhaustion, and changes in mental status. The only absolute indications for intubation include respiratory arrest or coma. If intubation appears to be imminent, intubating semielectively will help ensure optimal intubating conditions. The procedure should be done by the most experienced airway management clinician in order to improve the odds of first-pass success.[13]

Permissive hypercapnia or controlled hypoventilation is the preferred ventilator strategy. This method involves sacrificing minute ventilation to allow adequate time for full exhalation between breaths. With this strategy, hypercapnea is acceptable in order to decrease the risk of complications.[130,131] We suggest the use of a volume-controlled mode where rate and tidal volume can be adjusted. **(See Table 6.)** Prolonged time for expiration (ie, a lower inspiration to expiration ratio) is recommended. Data supporting any particular ventilator mode are weak, so no recommendations can substitute for clinical judgment.

Intubation alone is not the definitive care for a severe asthma attack. Therapies to reverse the disease need to continue. Metered-dose inhalers have equivalent deposition in the lower airways to nebulizers and equivalence of efficacy.[132] The use of nebulizers has been tied to aerosolization of bacteria, leading to epidemics of nosocomial pneumonia. With the proper adapters, metered-dose inhalers can be administered without disconnecting the ventilator circuit, thus reducing the risk of ventilator-associated pneumonia. Ventilator flow has to be adjusted to account for nebulizer flow.

When employing controlled hypoventilation, the use of proper pain control and sedative agents is critical. With deep sedation agents (such as propofol) along with an opioid for analgesia, neuromuscular blocking agents may be avoided. Retrospective data suggest that asthma patients who are given neuromuscular blocking agents are at higher risk for postextubation myopathy, ventilator-associated pneumonia, and increased length of ICU stay.[133]

Table 6. Recommended Initial Ventilator Settings[137]

- Volume-controlled ventilation
- Tidal volume: 8 mL/kg ideal body weight
- Rate: 10 breaths/min
- Inspiratory flow rate: 80-100 L/min
- Expiratory time: 4-5 sec
- PEEP: 0 cm H_2O
- FiO_2 kept to the minimum required to keep SaO_2 > 90%

Abbreviations: FiO_2, fraction of inspired oxygen; PEEP, positive end-expiratory pressure; SaO_2, arterial oxygen saturation.
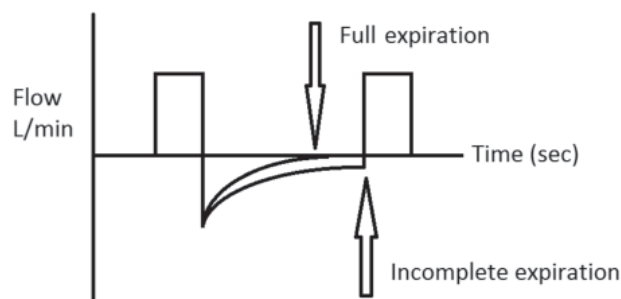
Hyperinflation is the most common cause of postintubation hypotension.[134] Hyperinflation is usually the result of breath-stacking (or auto-PEEP [positive end-expiratory pressure]) that is caused by inadequate exhalation between delivered breaths, leading to subsequent increases in pulmonary pressure and increases in total lung volume. **(See Figure 1.)** Breath-stacking can be assessed in 2 ways: (1) plateau pressures can be checked by performing an end-inspiratory hold, or (2) an end-inspiratory apnea test can be performed using the ventilator to measure the exhaled breath volume. Plateau pressures should used to assess hyperinflation rather than peak pressures, as peak pressures are affected more by bronchospasm than by hyperinflation. Generally, plateau pressures of < 30 cm H_2O are a sign of minimal or no breath-stacking. The ventilator's graphical representations of flow/time can be used to confirm that expiratory flow ceases before the next breath is delivered. **(See Figures 2 and 3.)**

Decreasing the respiratory rate has a direct correlation to decreasing plateau pressures. If the plateau pressures are elevated or the flow/time graph indicates breath-stacking, then the respiratory rate should be decreased.[135] The use of PEEP has been found to have a direct impact on end-inspiratory volume, functional residual volume, plateau pressures, esophageal pressures, and central venous pressures. One study found that cardiac output and blood pressure decreased proportionally with increases in PEEP.[136] Thus, we recommend initial ventilator settings with zero PEEP during the patient's ED stay. As the patient is liberated from the ventilator in the ICU, PEEP may be useful.

Clinical Pearls For Ventilator Management
- Maintain low to zero PEEP; do not go any higher than 5 cm H_2O unless absolutely indicated.
- If the initial respiratory rate does not allow ad-

equate exhalation, reduce the respiratory rate as needed until breath-stacking stops.
- Use the largest-size tube when intubating; ≥ 8.0 is ideal; this will make pulmonary toilet management much easier.
- Use plateau pressure (not peak pressure) to guide your management of the ventilator. Dial down the respiratory rate as needed to maintain acceptable plateau pressures.
- Use the ventilator breath tracing to ensure that subclinical breath-stacking is not occurring. These subclinical findings on the tracking will help prevent them from becoming clinically significant. **(See Figure 1.)**

Figure 2. Normal Ventilator Flow Tracing
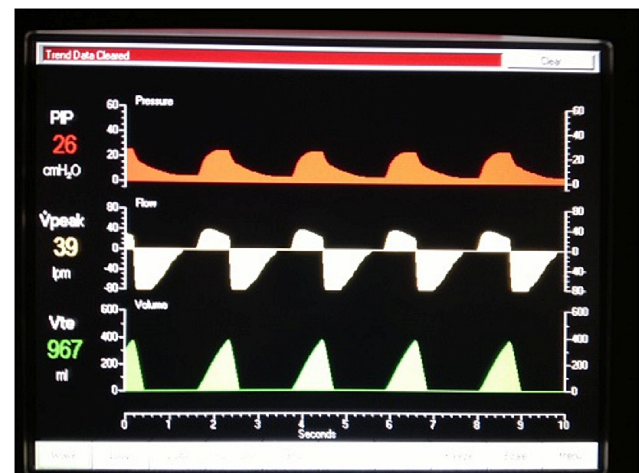
Image courtesy of Philip Mason, MD.

Figure 3. Breath-Stacking On The Ventilator Tracing
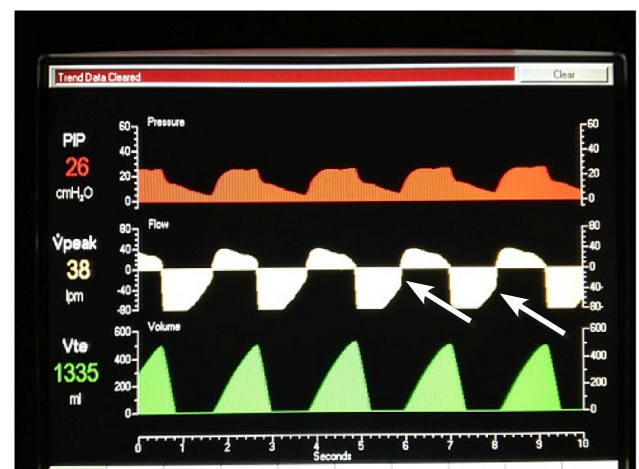
Arrows point to breath-stacking on a dynamic ventilator breath tracing. Image courtesy of Philip Mason, MD.

Figure 1. Illustration Of Breath-Stacking On The Ventilator Tracing

The arrow on the top demonstrates what a full expiration looks like on the ventilator tracing. This is what is ideal for the patient. The arrow on the bottom demonstrates an incomplete expiration prior to the initiation of the next breath. Breath-stacking will lead to an auto-PEEP phenomenon with subsequent clinical effects.

Controversies And Cutting Edge

Theophylline

Theophylline is a methylxanthine and thus bears structural and pharmacological similarities to caffeine. The primary action involves relaxation of bronchial smooth muscle, anti-inflammatory effects, and stimulation of the medullary respiratory center. Other effects of theophylline include: (1) increasing heart muscle contractility and efficiency, (2) increasing heart rate, (3) increasing blood pressure, and (4) increasing renal blood flow. This drug has a narrow therapeutic index and must be monitored to avoid toxicity.[138] A meta-analysis of randomized controlled trials demonstrated that the addition of theophylline to inhaled corticosteroids has the same therapeutic effects on improving lung function as simply increasing the dose of the steroid.[139] While there may be a limited role in long-term management, there is virtually no role in the acute setting.

Aminophylline

Aminophylline is a combination of theophylline and ethylenediamine. Its main pharmacological action is relaxation of bronchial smooth muscle. Multiple Cochrane reviews in both children and adults have found no benefit to standard care with the addition of aminophylline.[140-142] Given the lack of supporting evidence, prolonged time to onset, and toxic risks of theophylline, we recommend against this therapy.

Heliox

Heliox has gained interest for the treatment of both upper and lower airway obstruction. It is less dense than air or oxygen and thus provides more laminar flow in obstructed airways.[143] Evidence regarding the efficacy of heliox in the literature is sparse. The most recent randomized controlled trials have found that heliox-driven albuterol may be a useful adjunct therapy for adult asthma patients with severe asthma exacerbation after other therapies have been attempted. Current studies have shown that albuterol nebulized with heliox offers no clinical benefit over standard therapy in severe pediatric asthma.[144,145] If available, it may be considered as an adjunct therapy in severe asthma exacerbations when other therapies have failed. The mixture (typically 70% helium, 30% oxygen) limits its use in hypoxic patients.

Long-Acting Beta Agonists

Long-acting beta-adrenoceptor agonists (more specifically beta agonists, with duration > 8-12 h) are usually prescribed for moderate-to-severe persistent asthma in the chronic management setting. They are designed to reduce the need for short-acting beta agonists, as they have a duration of action of approximately 12 hours, making them candidates for sparing high doses of corticosteroids. However, long-acting beta agonists are not recommended for the treatment of acute asthma exacerbations because of their slower onset of action.[146,147]

Ketamine

The use of ketamine has been advocated for the treatment of acute asthma due to its bronchodilatory effects.[149] Case reports of the use of ketamine in severe asthma date back to the 1970s.[149] Ketamine has a good safety profile and a very low risk of major adverse events;[150] however, there is a paucity of high-quality evidence, which prevents its routine use in asthma. A recent Cochrane review notes that there is insufficient evidence to make specific recommendations.[151] Three randomized controlled trials have attempted to address this question. Allen and Macias randomized children with moderate-to-severe asthma to a 0.2 mg/kg ketamine bolus followed by 0.5 mg/kg/h for 2 hours versus placebo. Pulmonary index scores were measured throughout the 2

Time- And Cost-Effective Strategies

1. Ensuring expedited follow-up care and communicating with the patient's primary care provider are important for good patient care as well as cost savings. Using cost-of-illness modeling, adequate and sufficient treatment are key to earning annual savings.[176]

2. Impoverished urban children suffer disproportionately from asthma, and they under use preventive asthma medications. Administration of preventive asthma medication at school by the school nurse each school day has been shown to reduce symptoms and decrease healthcare utilization in urban children with asthma.[177] Discharge instructions from the ED specifically instructing the patient to have the school nurse administer outpatient medication can lead to overall healthcare cost savings.

3. Use of metered-dose inhalers with spacers in place of wet nebulizers to deliver albuterol to treat children with mild to moderate asthma exacerbations in the ED can yield significant cost savings for hospitals and, by extension, to both the healthcare system and families of children with asthma.[178,179]

4. A multidisciplinary approach to patient care incorporating clinical pharmacy services in the ED may improve disease state management and medication cost savings through generic prescription of asthma medications.[180]

5. Use of racemic albuterol instead of levalbuterol is most cost-effective, as the differences are clinically insignificant and racemic albuterol is much cheaper.

hours. They found no significant differences in scoring or admission rates.[152] Howton et al randomized 53 patients aged 18 to 65 years with acute asthma exacerbation with FEV1 < 40% of predicted value to a bolus of 0.2 mg/kg followed by 0.5 mg/kg/h for 3 hours versus placebo. No statistically significant differences were found for any measured parameter.[153] However, Hemmingsen et al conducted a randomized controlled trial in 14 patients on mechanical ventilation. Patients were randomized to a 1-mg/kg bolus or placebo. Statistically significant improvements in partial pressure of oxygen and stethoscopic evaluations were noted.[154]

Given the current available evidence, the routine use of ketamine in asthma patients is probably of little clinical value. Further studies to assess whether it may be useful in the critically ill are needed before conclusions can be drawn.

Leukotriene Inhibitor Agonists

Leukotriene inhibitor agonists have effects on multiple pathways including bronchoconstriction, inflammatory cell recruitment, vascular leakage, mucus production, and airway remodeling.[155] Montelukast appears to be the most commonly studied in this class of drugs.

A recent Cochrane review stated that the current evidence does not support the routine use of leukotriene inhibitor agonists in acute asthma. The review cited the lack of high-quality trials involving children and the variable end-point measurements as the reasons for not supporting routine use at this time; however, they did note that further studies need to be done before conclusions can be drawn.[156] Carmago et al randomized 201 adults with moderate to severe asthma exacerbations to montelukast 7 mg, 14 mg, or placebo in addition to other standard

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Risk Management Pitfalls For Asthma Management In The Emergency Department

1. **"The treatment seemed straightforward; I didn't think their home situation was any of my business."**
Psychosocial problems need to be identified and addressed as part of asthma management, because, even with best practice, these problems place patients at an increased risk of dying. Family psychosocial problems and financial problems are associated with increased risk of mortality for patients aged > 31 years but not for younger patients. Males were at increased risk of mortality from asthma exacerbation overall, but females with family problems are at greater risk than males with family problems. Alcohol use increased the risk of mortality for individuals who received only verbal instructions without a written action plan.

2. **"I thought the longer-acting medication would help reduce the need for repeat treatments."**
Clinical studies of long-acting beta agonists compared to placebo in asthma patients using variable doses of inhaled corticosteroids have raised the issue of mortality risk in patients with asthma who are taking regular long-acting beta agonists. Long-acting beta agonists added to inhaled corticosteroids reduces asthma-related hospitalizations compared to inhaled corticosteroids alone, and there is no statistical increase in mortality. However, long-acting beta agonist treatment without inhaled corticosteroids does increase mortality risk in asthma. Healthcare providers must understand the essential need for adequate dosing of inhaled corticosteroids to control airway inflammation.

3. **"The patient didn't have any questions, so I didn't think she really wanted to hear all the intricate details. "**
Prescription of steroids in the treatment of acute asthma can lead to the following complications: avascular necrosis, mood changes, visual complaints, and infection. A provider treating patients with steroids must be diligent in explaining the potential side effects of steroids. The informed consent process, documentation, and close monitoring of patients are critical to avoid potential litigation.

4. **"I was concerned about the fetal side effects and figured that short-acting beta agonist therapy was sufficient."**
Maternal asthma is associated with an increased risk of spontaneous abortion. Standard medical treatment of acute asthma does not increase the risk of congenital anomalies in the offspring when taken during the first trimester of pregnancy.

5. **"I thought I would see how the patient responded to standard therapies before starting noninvasive positive-pressure ventilation."**
Noninvasive ventilation (NIV) has been shown to be effective in a wide variety of clinical settings; however, reports of NIV in asthma patients are scarce. There are a few prospective clinical trials reporting promising results in favor of the use of NIV in a severe asthma attack. A trial of NIV prior to invasive mechanical ventilation seems acceptable and

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therapies. They found improvement in FEV1 (15% vs 4%) at 20 minutes that was sustained through 2 hours with no difference between the 2 doses in the intervention arm. The intervention groups also required less beta agonist treatments, with no adverse events noted.[157] Dockhorn et al conducted a randomized controlled crossover trial in patients with mild to moderate asthma studied over a 24-hour period. They noted that the IV formulation of montelukast (which has limited availability) had an onset of action within 15 minutes and peak effects seen shortly after that, with a mean improvement in FEV1 of 21%. The oral formulation had similar effects, with a mean improvement of 16% and peak effects seen within 2 hours (compared to a mean 8% improvement in the placebo arm).[158] Conclusive evidence is lacking, but this therapy may be useful in severe asthma. Further research is needed.

Noninvasive Positive Pressure Ventilation

Noninvasive positive pressure ventilation (NIPPV) has demonstrated efficacy in acute exacerbations of COPD and congestive heart failure.[159] The use of NIPPV has been slow to integrate within the arsenal of asthma treatments.

Soroksky et al randomized 30 patients with severe asthma to NIPPV or a sham device in addition to a protocolized treatment. NIPPV was started at 8/3 (cm H_2O inspiratory positive airway pressure/continuous positive airway pressure) and titrated up every 15 minutes to a maximum of 15/5, as tolerated. The primary outcome of FEV1 improvement at 4 hours was 54% versus 28%. The intent-to-treat analysis for hospital admission had an 18% admission rate in the intervention group versus 63% in the control group.[160]

Risk Management Pitfalls For Asthma Management In The Emergency Department

may benefit patients by decreasing the need for intubation and by supporting pharmaceutical treatments. Although selecting the appropriate patients for NIV use is a key factor in successful NIV application, how to distinguish such patients is still quite controversial. If this technology is going to be employed, reaching for it early will likely yield more benefit.

6. **"I knew the patient was sick, but $ETCO_2$ seemed sufficient."**
In adult asthma patients with acute exacerbations, concordance between $ETCO_2$ measured by capnography and $PaCO_2$ measured by blood gas is high. However, capnography is not a replacement of blood gas as an accurate means of assessing alveolar ventilation in acute asthma.

7. **"We had trouble getting IV access, so I thought the nebulized therapy would suffice."**
The use of IV magnesium sulfate (in addition to beta agonists and systemic steroids) in the treatment of acute asthma improves pulmonary function and reduces the number of hospital admissions for children; it only improves pulmonary function for adults. Though the use of nebulized magnesium sulfate appears to produce benefits for adults, the routine use of this form of magnesium sulfate should not be considered standard of care at this point.

8. **"Steroids from the discharge pharmacy seemed much easier."**
Early administration of steroid therapy is essential. Current literature suggests that early administration decreases hospitalization rates and bounce-back rates. When treating for acute exacerbations, steroid therapy should be administered early.

9. **"PEF rate values were improved, so discharge seemed appropriate."**
Proper triage of acute exacerbations must be based on complete clinical and psychosocial factors as a package. There is no single clinical factor that can be relied upon for triaging. Additionally, lack of historical risk factors does not equal lack of morbidity and mortality risk.

10. **"It seemed that if we could have held off a little bit longer, the patient's course would turn around."**
When intubation is clinically indicated, the emergency clinician should proceed without delay. Waiting to intubate when intubation is clinically indicated will lead to increased likelihood of procedural complications and respiratory arrest. We recommend that only the most experienced provider perform the procedure, given the increased need for first-pass success.

Pediatric literature on NIPPV specifically for asthma is limited. Several studies have demonstrated feasibility and safety in the pediatric population. Yanez et al randomized 50 patients admitted to the pediatric ICU for acute respiratory failure, including asthma, to NIPPV or conventional therapy. NIPPV was started at 12/6 and titrated up to 18/12, as tolerated. Statistically significant differences in heart rate and respiratory rates at 1 hour and 6 hours were noted. The intubation rate was 28% in the intervention group compared to 60% in the control group.[161] A mixed retrospective and prospective descriptive analysis of 165 children admitted to the pediatric ICU also demonstrated both safety and efficacy in children as young as 7 months of age.[162]

The benefits of NIPPV appear to outweigh the risks. Despite the lack of large, robust trials, given the likely benefit of NIPPV, we recommend the liberal use of this technology, when indicated.

Special Circumstances

Pediatric Patients

Asthma is a leading cause of ED visits and hospitalizations, and it is frequently unrecognized and undertreated.[163,164] Many asthma deaths and near-fatal exacerbations occur in children with no prior history of hospitalizations for asthma.[165] While recurrent wheezing, chest tightness, and dyspnea are common hallmarks of asthma, children may present with a history of chronic cough or chest pain as the sole manifestation of asthma.[166] Patients with sickle cell disease have an increased risk of acute chest syndrome during asthma exacerbations due to hypoxia inducing vaso-occlusive disease. Early, aggressive treatment with bronchodilators and systemic corticosteroids decreases the duration of asthma and relapse rates.[107] Children aged < 8 years typically have difficulty coordinating peak expiratory flow rate testing; thus, providers must rely on other clinical observations to gauge severity and improvement. This topic is explored in depth in the May 2013 issue of *Pediatric Emergency Medicine Practice*, "Management Of Acute Asthma In The Pediatric Patient: An Evidence-Based Review."

Pregnancy

Uncontrolled asthma in pregnancy is associated with low birth weights, pre-eclampsia, and cesarean section.[167,168] Short-acting beta agonists, ipratropium bromide, and inhaled corticosteroids appear to be safe in pregnancy, and their benefits outweigh the maternal and fetal risks of uncontrolled asthma.[169] However, multiple studies demonstrate that pregnant women are less likely to receive systemic steroids following an ED visit for asthma exacerbation.[170,171] In a large trial of pregnant women exposed to systemic corticosteroids, there was no major increase in terato-genic risk, although there was a small increase in oral cleft incidence.[172] In a large trial of over 1000 babies, preterm neonates exposed to repeated doses of glucocorticoids to increase lung maturity had no increase in neurologic sequelae compared to those exposed to a single dose.[173]

Disposition

Admission Versus Discharge

The decision to admit is largely based on clinical course, response to treatment, and availability of follow-up care. Persistent hypoxia despite aggressive bronchodilators and early corticosteroids is an indication for admission. Discharge plans should include follow-up care and an asthma action plan. NAEPP guidelines suggest the utilization of peak expiratory flow rate as a marker for asthma severity and improvement, with a discharge goal of 70% of predicted.[13] As previously discussed, there are inherent challenges associated with the use of peak expiratory flow that must be considered in the appropriate clinical context. The decision to admit versus discharge still largely remains a clinical decision until better decision-aid tools are developed.

Asthma Scoring Systems

Multiple asthma scoring systems are described in the literature to assist the emergency clinician with disposition. At least 18 such scoring systems have been published, many of which have not been validated.[174,175] One largely validated scoring system in pediatrics, the Pediatric Asthma Severity Score (PASS), which was found to predict the need for admission, is based on clinical findings of wheezing, air entry, work of breathing, prolonged expiration, and mental status.[50] Interestingly, the gold standard the PASS used was the clinician's decision to admit. In our opinion, it unfairly weighs different aspects of the clinical examination. Specifically, alterations in mental status are weighed the same as other examination findings, but altered mental status is a hard indication for admission regardless of other findings. Thus, it is our opinion that this scoring system should be used with caution, and it may be better used as a research tool than a clinical decision tool.

Summary

Asthma is diagnosed clinically, and therapy should be based around inhaled albuterol and systemic steroids. Magnesium and NIPPV should be strongly considered in severe exacerbations. Other therapies should be considered for specific clinical indications and individual patient needs. Emergency clinicians should use their clinical judgment to guide therapies and disposition.

Case Conclusions

The 19-year-old male had an extensive asthma history, including ICU admissions, but he was never intubated. His vital signs on presentation were: blood pressure, 117/86 mm Hg; heart rate, 124 beats/min; respiratory rate, 28 breaths/min, temperature, 37.3°C, and pulse oximetry, 87% on room air. The physical examination was significant for diffuse end expiratory wheezing throughout all lung fields, and crepitus was palpable along the patient's anterior neck. You immediately placed him on a monitor, gave supplemental oxygen with nebulized therapy, and administered corticosteroids. A chest radiograph was obtained, and it demonstrated the presence of subcutaneous emphysema and pneumomediastinum. The patient's work of breathing improved only slightly with the first nebulizer treatment, and his ETCO$_2$ read 32. You decided to place him on NIPPV with continuous nebulizer treatments. You also administered intramuscular epinephrine. After 10 minutes of this therapy, the patient's work of breathing began to improve. His ETCO$_2$ and oxygen saturation started to normalize on NIPPV. A diagnosis of acute severe asthma complicated with subcutaneous emphysema and pneumomediastinum was made, and the patient was admitted to the intermediate care unit.

For the 24-year-old pregnant female with a mild asthma exacerbation, you began 3 consecutive metered-dose inhaler treatments with albuterol. You also administered 16 mg of dexamethasone orally. Peak flows performed before and after the first treatment were 125 L/min (predicted 235), and auscultation revealed loud expiratory wheezing and better airflow. Peak expiratory flow rate continued to improve, and there was clearing of breath sounds and much-improved airflow. Her respiratory rate was 24 breaths/min at that time, and her heart rate was 108 beats/min. After 2 hours, her symptoms were nearly resolved; you gave her a prescription for repeat dexamethasone with a metered-dose inhaler refill and sent her home.

You treated the 6-year-old girl with the first-time episode of fever and wheezing with 1 hour of serial albuterol/ipratropium treatments, and she demonstrated marked improvement in air movement with decreased wheeze and pulse oximetry of 98% on room air. You discussed the treatment plan with her parents and discharged her with appropriate medications and a referral to her pediatrician.

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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1. **What is the most common cause of asthma exacerbations?**
   a. *Staphylococcal aureus*
   b. *Streptococcal pneumonia*
   c. *Pseudomonas aeruginosa*
   d. Viruses

2. **Which of the following statements about chest radiographs and asthma is TRUE?**
   a. Elevated heart rate and respiratory rate and abnormal breath sounds correlate with increased probability of radiographic abnormality in acute asthma.
   b. History and clinical examination are not enough to determine whether a radiograph is needed in acute asthma.
   c. An elevated white blood cell count is required for ordering a chest radiograph in acute asthma.
   d. Over half of patients admitted for acute asthma have a chest radiograph abnormality.

3. **Which of the following is FALSE?**
   a. Short-acting beta agonists are the cornerstone of asthma therapy in the ED.
   b. There is no difference in clinical efficacy of the racemic albuterol and levalbuterol, when properly dosed.
   c. Metered-dose inhalers with a holding chamber (spacer) versus nebulizers in the ED lead to increased hospitalization rates for adults.
   d. When compared to albuterol alone, combination therapy with ipratropium bromide and albuterol has been shown to decrease hospitalization rates.

4. **In mild to moderate asthma exacerbations, nebulized albuterol is superior to metered-dose inhalers.**
   a. True
   b. False

5. **Regarding the use of steroids in acute asthma, which of the following is FALSE?**
   a. Single-dose dexamethasone, by both oral and intramuscular routes, are as effective at 5-day therapy with prednisone.
   b. Inhaled corticosteroid therapy may be effective in the treatment of asthma.
   c. Delays in steroid therapy > 1 hour after presentation place patients at higher risk for admission.
   d. High-dose steroids provider greater benefit over lower standard doses in acute asthma.

6. **In addition to albuterol and corticosteroids, which of the following medications has been shown to decrease hospitalization rates in pediatric patients?**
   a. Epinephrine
   b. Montelukast
   c. Ipratropium bromide
   d. Levalbuterol

7. **A patient presents with a severe asthma exacerbation requiring admission. In addition to albuterol and corticosteroids, which of the following has been shown to improve symptoms in severe exacerbations?**
   a. Ketamine
   b. Magnesium
   c. Epinephrine
   d. Levalbuterol

8. **Which of the following is considered a hard indication for intubation?**
   a. Peak flow rate < 200
   b. Hypocarbia on venous blood gas testing
   c. Respiratory rate > 40 breaths/min
   d. Altered mental status

9. **Which of the following statements is FALSE?**
   a. Main actions of theophylline involve relaxation of bronchial smooth muscle, anti-inflammatory effects, and stimulation of the medullary respiratory center.
   b. Aminophylline has a delayed onset time and dangerous toxicity profile.
   c. Heliox-driven albuterol may be a useful adjunct therapy for adult asthmatic patients with severe asthma exacerbation.
   d. Initiating theophylline therapy should be considered in the ED care of asthmatic patients.

10. **Short-acting beta agonists, ipratropium, and inhaled corticosteroids appear to be safe in pregnancy and outweigh the maternal and fetal risks of uncontrolled asthma.**
    a. True
    b. False

Coming Soon In
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An Evidence-Based Approach To The Evaluation And Treatment Of Low Back Pain In The Emergency Department

AUTHOR:

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Low back pain is the most common musculoskeletal complaint seen in the emergency department. Estimates of the annual healthcare expenditures for low back pain in the United States exceed $90 billion annually, which does not even take into account lost productivity and business costs. Although medicine has developed extraordinary tools to visualize spinal anatomy noninvasively and has elucidated the molecular mechanism of neurotransmission, we are still challenged by the many elements that constitute how humans feel and interpret pain.

In the emergency department, interspersed between patients with musculoskeletal back pain are patients with back pain who are harboring lesions that may put them at risk for permanent neurologic or even life-threatening sequela. By utilizing a focused approach, the emergency clinician will be able to identify these "red flag" patients and initiate a workup in a timely way. This issue of *Emergency Medicine Practice* reviews the progress to date and guides the emergency clinician to rationally and cost-effectively evaluate the patient presenting with low back pain symptoms. This review explores the evidence-based evaluation of the patient with low back pain and provides guidance on risk stratification pertaining to laboratory assessment and radiologic imaging in the ED. Effective and proven strategies to avoid medical errors, provide better care for patients, and help manage costs and precious healthcare resources are included.

Emergency Department Management Of Vaginal Bleeding In The Nonpregnant Patient

AUTHOR:

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Abnormal uterine bleeding is the most common reason women seek gynecologic care, and many of these women present to the emergency department for evaluation. It is essential that emergency clinicians have a thorough understanding of the underlying physiology of the menstrual cycle to appropriately manage a nonpregnant woman with abnormal bleeding.

Abnormal vaginal bleeding can be very distressing for a woman. At a minimum, it is an inconvenience that disrupts daily life; in severe cases, bleeding can result in life-threatening hemorrhage requiring emergency intervention. Abnormal bleeding may also herald serious underlying pathology, such as cancer. Many women presenting to the ED for evaluation of vaginal bleeding fear that they are in the serious category, and the provider must be sensitive to her concerns. The role of the emergency clinician is to rule out life-threatening complications associated with abnormal uterine bleeding and to obtain gynecologic consultation as needed. Emergency clinicians are often hesitant to initiate therapies to temporize a stable patient's bleeding and will refer the patient to a gynecologist to manage on an outpatient basis, but emergency clinicians can be encouraged to initiate treatment to temporize an acute bleeding episode until timely follow-up with a gynecologist can be obtained. This issue of *Emergency Medicine Practice* reviews common causes of abnormal bleeding including anovulatory, ovulatory, and structural causes in both stable and unstable patients.

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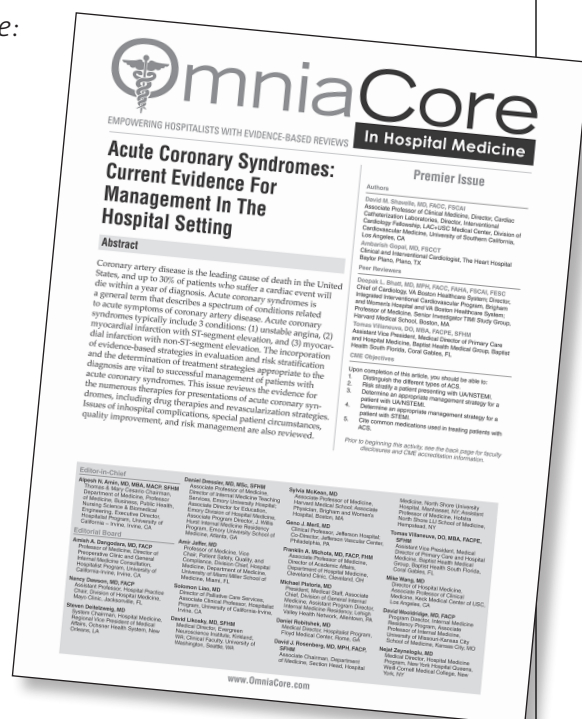
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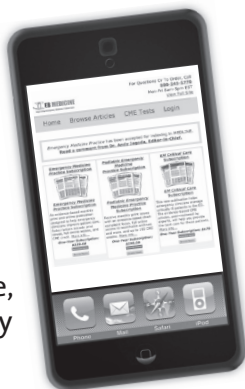
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Date of Original Release: June 1, 2013. Date of most recent review: May 10, 2013. Termination date: June 1, 2016.

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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