

# Asthma: A true out-of-hospital emergency

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## Introduction

EMS, like all health care entities, has faced the need to justify the costs of medical interventions with the benefits. Some of the treatments provided by EMS are still being justified. However, certain patient populations benefit from immediate emergency care, even at the advanced life support level. One such patient population consists of those patients suffering from acute respiratory distress. In fact, the *New England Journal of Medicine* reported in 2007 that the addition of advanced life support protocols for respiratory distress decreased the rate of death for these patients by 1.9% (Stiell, et al., 2007).<sup>1</sup>

At all ages, asthma is a frequent cause of respiratory distress and one where early prehospital intervention may make a significant difference. In the United States alone, asthma accounts for over 200,000

visits to hospital emergency departments annually.<sup>2</sup> With many conditions, notably trauma, EMS has learned that transport is treatment. In the case of asthma, however, an EMS provider's proper assessment and aggressive management can literally be lifesaving.

## Assessment of asthmatic patients

The first step in determining what treatment pathways to pursue with the asthmatic patient is through a proper and complete assessment.

An "across the room" assessment can often be conducted by an experienced provider in 30 seconds or less. Such an assessment creates a logical framework that leads health care providers through a process that experienced medics and physicians often perform without conscious consideration. Such an

assessment should evaluate, at a minimum, appearance, work of breathing and circulation.

By using this framework, the severely asthmatic patient can often be identified prior to asking the first question. This is important because the more critical the patient, the more rapidly interventions should be initiated and the less time spent on the history and physical exam.

### ***Appearance***

An experienced provider can form an impression within seconds of seeing a patient. These experienced providers can often make an almost instantaneous determination of “sick” or “not sick.” This evaluation is typically based upon patient position and behavior. Signs of impending respiratory distress may include the tripod position, extension of the neck, flaring of nostrils, cyanosis (a late, ominous sign), accessory muscle use and a generally fatigued appearance. Any or all of these factors are indicative of severe respiratory distress.<sup>3</sup>

### ***Work of breathing***

Work of breathing is assessed by watching the accessory muscles of respiration. When the accessory muscles of inspiration and expiration are being used, the patient is in significant distress. Abdominal movement can also be observed and provides good information about the severity of the disease. Paradoxical abdominal movement is highly suggestive of inspiratory muscle fatigue. Paradoxical abdominal movement occurs when the diaphragm is so tired that when the chest wall moves outward, the subsequent change in pressure pulls the diaphragm upward, thereby pulling the abdominal contents inward.<sup>4</sup>

### ***Circulation***

The skin is an excellent indicator of circulatory status. When the circulatory system is under stress, it often reroutes circulation from non-vital organs, including the skin. Visible skin signs of severe respiratory distress often include mottling and cyanosis.

### ***Face to face***

Few asthmatics develop a hyperacute “sudden asphyxic asthma” that results in death within hours. For the majority of asthmatics, a severe asthma attack comes after a period of poor overall control of their asthma, whether from noncompliance with medications or an exacerbation of symptoms.

Patients with asthma may present with many symptoms, typically including dyspnea, wheezing and coughing. Coughing appears early in asthma attacks and may be the only complaint. Thus, asthmatic patients can be overlooked in the field as having “cold” or “flu-like” symptoms. Coughing is often seen in elderly patients.<sup>5</sup> The ability to perform end-tidal capnography on these patients can clue the EMS provider in to bronchospasms that may be hidden either by coughing or low volume.

### ***Medical history***

These patients will typically have a medical history of asthma or other respiratory disease. EMS providers should thoroughly discuss the patient’s prior asthma history and symptoms, which may provide clues about the severity of the asthmatic’s disease and risk factors for death from asthma. It’s also important to determine whether the patient has had any previous attacks within the past 12 hours. If the patient is experiencing the second phase of an asthma attack, the swelling

and inflammation of the lower airway may not respond to bronchodilators, which are often a first-line therapy for asthmatic patients.<sup>6</sup>

### ***Physical assessment***

The patient who has an altered mental status and is taking short, shallow breaths is quite obviously experiencing a severe attack, which will likely require aggressive, rapid treatment. In addition to the observations made during the initial assessment and the clues revealed by the history, several other findings may

aid providers in recognizing the severe asthmatic. In this instance, the inability to speak more than a few words suggests severe distress.

Vital signs can provide some clues as to the severity of the attack. The respiratory rate alone typically does not correlate with severity of the attack, except in cases when the rate is greater than 40 breaths per minute. Respiratory rates may drop as the patient becomes fatigued and begins to go into respiratory failure. The presence of wheezing is dependent on airflow and the velocity of that airflow. As

## **Risk factors for death from asthma<sup>9</sup>**

- ❖ Past history of sudden severe exacerbation.
- ❖ Prior intubation for asthma.
- ❖ Prior asthma admission to an intensive care unit.
- ❖ Two or more hospitalizations for asthma in the past year.
- ❖ Three or more emergency department care visits for asthma in the past year.
- ❖ Hospitalization or an emergency department care visit for asthma within the past month.
- ❖ Use of >2 MDI short-acting beta-2 agonist canisters per month.
- ❖ Current use of or recent withdrawal from systemic corticosteroids.
- ❖ Difficulty perceiving severity of airflow obstruction.
- ❖ Comorbidities, such as cardiovascular diseases or other systemic problems.
- ❖ Serious psychiatric disease or psychosocial problems.
- ❖ Illicit drug use, especially inhaled cocaine and heroin.

such, wheezing is rarely a good indicator of the severity of an asthma attack. However, a lack of wheezing and the lack of airflow despite maximal effort indicates a severe episode. Asthma usually results in a prolonged expiratory phase, which can be detected using digital waveform capnography.<sup>7</sup>

Tachypnea presenting simultaneously with tachycardia at a rate greater than 120 may indicate a severe attack. In a severe attack, blood pressure may reveal pulsus paradoxus, *i.e.*, a fall in systolic greater than 10 mmHg during inspiration.

In some instances, skin can provide clues as to the severity of the attack. Diaphoresis can arise subsequent to the increased work of breathing seen with an asthma attack. Profound diaphoresis accompanied by a decreasing level of agitation are ominous signs of an impending crashing patient. Cyanosis is uncommon because of the respiratory alkalosis caused by the attack.<sup>8</sup>

While wheezing is considered a classic symptom of asthma, other diseases and conditions may produce wheezing. These conditions may include pneumonia, bronchitis, emphysema, foreign body aspiration, heart failure, pneumothorax, pulmonary embolism and toxic inhalation.<sup>10</sup>

## Pathophysiology

For most patients, asthma, also known as reactive airway disease, generally occurs intermittently in acute episodes of varying durations. In between these attacks, the patient is typically asymptomatic.

During an asthma attack, reversible airflow obstruction occurs, caused by bronchial smooth muscle retraction and secretions of mucus that result in bronchial plugging. Additionally, inflammatory changes occur to the bronchial walls. This

airflow resistance may lead to alveolar hypoventilation, marked ventilation-perfusion mismatch (potentially leading to hypoxemia) and carbon dioxide retention, which in itself stimulates hyperventilation. The carbon dioxide retention exhibits itself in waveform capnography with the “shark-fin” pattern consistent with reactive airway diseases. The obstruction of both inspiration and expiration causes pressure to remain high in the lungs as a result of the air trapping.<sup>11</sup>

In severe asthma attacks, greater use of accessory muscles occurs and increases the chances of respiratory fatigue. This labored breathing may create high thoracic pressures, reducing the amount of blood returning to the left ventricle (left ventricular preload). Near-fatal asthma is characterized by this resulting drop in cardiac output and systolic blood pressure.<sup>12</sup>

*Status asthmaticus* is a severe, prolonged episode of asthma that has not been stopped despite repeated dosages of bronchodilators. These episodes may be triggered either by a sudden airway spasm or in a more subtle instance, such as a respiratory tract infection or repeated exposure to allergens. Additionally, these patients are prone to dehydration and may potentially require intravenous fluid administration.<sup>13</sup>

## Treatment options

Several treatment options exist for the asthmatic patient, particularly through pharmaceutical interventions. Aside from oxygen, the primary treatment has traditionally been considered an inhaled or nebulized beta-agonist. Additional therapies have also included steroids and anti-cholinergic agents. Severe patients often receive epinephrine and/or magnesium sulfate.<sup>14</sup>



The typical first-line therapy for asthmatic patients in the prehospital setting, aside from oxygen administration, is usually an inhaled beta-agonist medication. Beta-agonist medications bind to and stimulate beta-2 receptors, causing relaxation of bronchial smooth muscle and antagonize the acetylcholine receptors, producing bronchodilation. Common side effects of beta-agonist medications may include tachycardia and anxiety. Common inhaled/nebulized beta-agonist medications are Albuterol and levalbuterol (Xoponex). While individual EMS systems have differing protocols for the administration of these medications, typical dosing is 2.5 to 5 milligrams of Albuterol administered via nebulizer for three doses over 20 minutes or a continuous nebulizer treatment. These nebulized medications may be delivered by hand-held nebulizer, small volume nebulizer or even in conjunction with a continuous positive airway pressure device or with a bag valve mask.<sup>15</sup>

For patients who are refractory to inhaled/nebulized beta-agonist therapy and/or those patients who are in extremis, parenteral beta-agonist therapy may be recommended. The most common of these therapies is epinephrine 1:1,000, either administered subcutaneously or intramuscularly, depending on local protocol. The dosage usually varies from 0.3 to 0.5 milligrams, again dependent on local protocol. It should be noted that epinephrine effects both the alpha and beta receptors. An additional parenteral beta-agonist is terbutaline (Brethine). Unlike epinephrine, terbutaline is exclusively a beta-agonist. It is normally administered intramuscularly or subcutaneously at a dosage of 0.25 milligrams.<sup>16</sup>

The anticholinergic medication ipatropium (Atrovent) may also be of some value in asthmatic patients, particularly those who are smokers or who have

coexisting chronic obstructive pulmonary disease (COPD). Ipratropium is normally administered via nebulizer either by itself or simultaneously with a nebulized beta-agonist. The dosage is typically 0.5 milligrams. Ipratropium's anticholinergic properties assist asthmatic patients in controlling the bronchial secretions common with their condition.<sup>17</sup>

Intravenous corticosteroids help control the inflammatory responses that cause swelling (edema) restricting the bronchial passages. The typical intravenous corticosteroid in the prehospital setting is methylprednisone (Solu-Medrol) administered at a dosage of 40 to 125 milligrams. As corticosteroids may take hours to work, it is important to consider their administration early for them to take effect as quickly as possible.<sup>18</sup>

Magnesium sulfate is occasionally administered to patients for its effects as a smooth muscle relaxant in causing the relaxation of constricted bronchial muscles. Its dosage is typically two grams intravenously over 30 to 60 minutes.<sup>19</sup>

Several less common therapies exist as well. Heliox, a mixture of helium and oxygen, can help distribute oxygen as well as nebulized medications. It additionally decreases the work of breathing. It should be noted that the dosage of Albuterol will be doubled with Heliox. Ketamine is currently coming into more use in the prehospital setting as an induction agent for rapid sequence intubation due to the shortages of Etomidate as well as being used for sedation.<sup>20</sup> However, Ketamine is also noted to have some bronchodilatory effects, leading to its consideration in some settings for management of asthmatic patients refractory to all other therapies.

Additionally, many EMS systems are encouraging the use of continuous positive airway pressure (CPAP) systems for asthmatic patients. CPAP's delivery of

pressurized oxygen reduces the work of breathing, holds airway structures open, and improves oxygenation and alveolar recruitment.<sup>21</sup>

## Conclusion

Asthma patients are among the patients who can benefit from aggressive prehospital treatment. This treatment, though, must be based on both a thorough assessment of the patient as well as a complete understanding of the pathophysiology of the underlying disease process in conjunction with the appropriate therapies.

**This article is provided for education only. Always consult with your medical director and follow your local protocols in making treatment decisions.**

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