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TO: ALL LUCAS COUNTY PARAMEDICS

FROM: Brent Parquette, NREMT-P
Lucas County EMS Continuing Education Program Administrator

DATE: August 7, 2012

SUBJECT: **Continuing Education – September 2012**

I hope you all enjoyed the summer months! And yes, with the fall months approaching, that means a return to continuing education classes. In the month of September we will once again review our approach to ***“Respiratory Emergencies.”***

With ongoing advancements in medicine, EMS providers are required to maintain their knowledge about clinical trends, and of course, new equipment and technology. That being said, we will be introducing a new CPAP device and expanding the scope of our respiratory protocols to include this therapy in the setting of COPD, Asthma and Pneumonia. You will also be trained on nebulized medications used in conjunction with CPAP delivery.

Waveform capnography has proven to be an invaluable diagnostic tool in the setting of critical patients with an advanced airway in place. We will also be adding the ability to monitor end-tidal CO₂ in your moderate-to-severe respiratory patients that do not require advanced airway placement.

I have attached a clinical review article entitled ***“Fighting for Air: Recognize & Treat the Severely Asthmatic Patient.”*** Please take time to read the entire article and then test your comprehension with the post-article quiz provided. The reading assignment is intended to prepare you to participate in case discussion and simulation activities during class in September.

As always, if you have any questions or comments please feel free to contact me. I look forward to seeing all of you in the coming month.

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Fighting for Air

RECOGNIZE & TREAT THE SEVERELY ASTHMATIC PATIENT

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2012 JEMS GAMES

In March 2012, an acute asthma patient will be among the victims managed at the JEMS Games clinical competition at the EMS TODAY conference.

This comprehensive clinical article will therefore assist participating teams and attendees in preparing for the JEMS Games, understanding this complex and frequently encountered medical event and obtaining CE credit on JEMSCE.com.

In addition, JEMS Games founding sponsor, Laerdal Medical Corp., will provide a special Simulation Tool Kit to each person attending the JEMS Games finals on March 2, 2012. The tool kit will include access to the acute asthma simulation featured at the JEMS Games to allow services to replicate the simulation on their agency and training center simulator.

While out on the road, Rescue 4 is dispatched to a call for a patient complaining of “difficulty breathing” a few blocks away. The EMS crew arrives at a multi-story apartment complex and determines that the patient is located in an apartment on the third floor. Two crew members decide to take the ALS gear up the stairs to get to the patient faster. The third provider takes the stretcher to the elevator.

On arrival at the apartment, the door is found open, and the EMS crew can hear audible wheezing emanating from the living room. The patient is a 17-year-old black female weighing approximately 90 kg. She’s in an upright position with her hands on her knees, slumping forward with severely labored breathing. She’s in obvious distress with the following vital signs: BP 124/86, pulse 124 bpm and RR 42. When asked her name, the patient is only able to reply with a faint “Shauna” before taking another shallow breath.

The patient’s mother informs the crew that the patient suffers from severe persistent asthma and hay fever, for which the doctor has prescribed albuterol, Fluticasone, Singulair and prednisone. She was seen at the local hospital and admitted into the intensive care unit one month prior for a similar asthma attack. She has had three less severe attacks since then. It appears that the patient hasn’t been compliant in taking all her medications because the prednisone bottle appears full and the allergy medication hasn’t been taken at all.

The lead paramedic instantly recognizes the urgency of the patient’s condition due

The patient’s mother informs the crew that the patient suffers from severe persistent asthma and hay fever.

to her increased work of breathing. Oxygen is applied via a non-rebreather mask at 15 liters per minute. Auscultation of breath sounds reveals both inspiratory and expiratory wheezing with minimal air exchange and retraction. Another provider prepares to administer albuterol and ipratropium (Atrovent) to be administered by hand-held nebulizer, as dictated by local EMS protocol. The pulse oximeter reads 87%, and the capnometer shows a shark fin waveform that indicates incomplete or obstructed exhalation, typical during a severe asthma attack. As transport begins, her vitals are BP 130/88, pulse 130 bpm and RR 40.

A continuous positive airway pressure (CPAP) mask is applied with a setting of 5 cm positive end-expiratory pressure (PEEP). The lead medic administers 0.3 mg of 1:1,000 epinephrine subcutaneously. Once an IV is established, the patient is also given 125 mg of methylprednisolone IVP. On arrival at the hospital, she's markedly improved following the aggressive therapy.

INTRODUCTION

Asthma affects 300 million people worldwide, with a quarter of a million people dying from the disease each year.¹ In the U.S., asthma is responsible for more than 200,000 emergency department (ED) visits and another 10.5 million medical office visits each year.² In 2000, asthma claimed the lives of 4,487 people.³ Deaths caused by asthma are infrequent in children (0.3 per 100,000) when compared with adults (2.1 per 100,000).

Morbidity and mortality statistics vary by ethnicity and region of the country. For example, the death rate for blacks is significantly higher than for other ethnicities. This is also true for the Hispanic population, but to a lesser degree. Data suggest that 14.5% of Puerto Rico's population is asthmatic; although the greatest rise in asthma rates

LEARNING Objectives

- >> Generalize the pathophysiology of asthma.
- >> Highlight the major clinical manifestations of asthma.
- >> Explain the key components of the Initial Visual Assessment Technique.
- >> Describe physical exam parameters to obtain in a patient with a severe asthma attack.
- >> Identify the indices that point to a severe asthma attack.
- >> Describe, sequence and justify the rationale for EMS interventions for an asthma attack.
- >> List warning signals of a potentially fatal asthma attack.

was among black children (almost a 50% increase) between 2001 and 2009.³

The importance of the prevalence of asthma is reflected in the economic cost with an estimated financial burden in the U.S. of \$12.7 billion in 1998 and almost 60% attributable to direct costs (e.g., hospital inpatient, outpatient, ED and physician services); it's estimated that less than 20% of asthma patients account for 80% of direct costs, a portion of whom are likely the most severe asthma cases.³

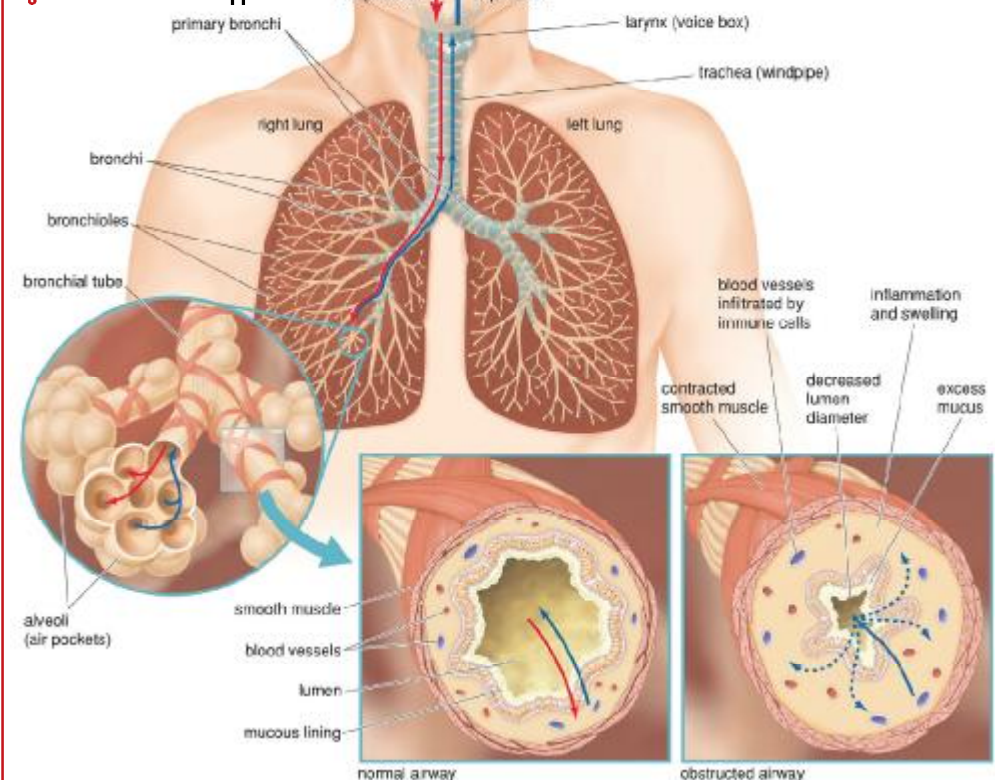
The prevalence of asthma demands EMS personnel understand the pathophysiology of the disease process and why some patients with asthma are at a significantly higher risk

than others. EMS personnel must be able to rapidly recognize and appropriately treat patients with moderate to severe asthma.

PATHOPHYSIOLOGY

The National Heart, Lung and Blood Institute defines asthma as "a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role."³ Inflammation causes many of the symptoms seen in an asthma attack, such as wheezing, breathlessness, chest tightness and coughing.³ These episodes don't create uniform airflow obstruction; instead, they create variable, widespread airflow obstruction that's often reversible through pharmacological

Figure 1: Asthma Attack Trapped Air



During the first phase of an asthma attack, the bronchioles constrict because of spasm and swelling.

intervention. The hallmark of the disease is increased bronchial hyperreactivity. This results in episodic bronchoconstriction as a consequence of smooth muscle contraction, inflammation of the bronchial walls and increased mucus production.⁴ Remember, swelling, spasm and secretions are the major causes of distress.

Compared with healthy individuals, asthmatics have airways that are hyperresponsive when exposed to bronchoconstricting stimuli. The degree to which an individual's airways are hyperresponsive determines the severity of the disease.³

Asthma isn't a single entity; it's categorized as either **atopic** or non-atopic. Atopic asthma is a hypersensitivity reaction, triggered by an environmental allergen or pollutant (e.g., chemical fumes, dust, pollen, animal dander or food). The usual triggers in non-atopic asthma are exposure to viruses that lead to respiratory infections, including rhinovirus and the parainfluenzae virus or such intrinsic factors as stress.⁴ This type of asthma occurs sporadically during the year.

It's also important to note that an asthma attack can occur in two phases. During phase one, the bronchioles constrict due to spasm and swelling often caused by the release of **histamine** and other pro-inflammatory mediators. A second phase may occur four to eight hours later, which leads to additional inflammation, edema and obstruction of airflow with plugging of the lower airways because of abundant mucous production. The first phase tends to respond to standard therapies, including inhaled beta agonists, while the second phase often requires additional medications, such as the administration of a corticosteroid, to reduce the inflammation and edema. Steroids are anti-inflammatory agents that treat the disease; beta agonists treat only the symptoms.

The importance of allergic airway inflammation and the role it plays in cases of severe asthma cannot be overstated. At the cellular level, a loss of airway epithelium, thickening of the basement membrane and hyperplasia of bronchial smooth muscle is observed—even in mild cases.³ In the severely asthmatic patient, these changes become more remarkable and are accompanied by thickening of the airway walls, subepithelial fibrosis, epithelial hypertrophy and growth of the mucus glands. This "airway remodeling" is likely the result of repetitive inflammation.³



The initial visual assessment technique (IVAT) assesses areas of appearance, work of breathing and circulation.

PHOTO: COURTNEY MCCAIN

Inflammation plays a major role in asthma. Inflammation is a process by which the immune system detects an allergen as foreign and, in turn, tries to use certain chemical mediators to quell or eliminate the foreign substance. In asthma, this causes bronchoconstriction, increased mucus production and **bronchospasm**.

Cells called **eosinophils** are highly implicated in asthma. Their presence suggests the allergic nature of asthma. Eosinophils are plentiful in immune reactions that are mediated by immunoglobulin E (IgE). Eosinophils produce major basic protein (MBP), which can cause the rupture of epithelial cell walls and significant damage to tissues in immune reactions.⁴ MBP also can increase airway mucus production and can cause histamine release.³ **Leukotrienes**—primarily LTC₄, LTD₄ and LTE₄—are potent bronchoconstrictors that mediate vasoconstriction and bronchospasm; they can cause increased vascular permeability, thus leading to the patient experiencing the symptoms of asthma.

ASSESSMENT

Who says the pediatric assessment triangle (PAT) is only for children? As every experienced clinician knows, patient assessment begins the moment the patient is seen.

Across the room: The PAT could be generalized and adapted for use with all patients. To distinguish from the PAT that's advocated by the American Academy of Pediatrics in their course, "Pediatric Education for Prehospital Personnel," this article will refer to that assessment as the initial visual assessment technique (IVAT), to reinforce that it should be applied to all patients.⁵ Like the PAT, the IVAT is an "across the room" assessment that can be conducted in 30 seconds or less.⁵ This assessment creates a system to guide healthcare providers through a process that experienced medics and physicians often perform without conscious consideration. The IVAT assesses areas of appearance, work of breathing and circulation.⁵

Using this assessment, 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. Also note that the IVAT can be accomplished in any order.

KEY Terms

Atopic: A congenital tendency to develop immediate allergic reactions.

Bronchospasm: A spasm of smooth muscle of the bronchi due to allergy, aspiration, exertion, infection or other irritation.

Eosinophil: A granular leukocyte with a nucleus that usually has two lobes connected by a slender thread of chromatin and cytoplasm containing coarse, round granules that are uniform in size.

Histamine: A compound found in all cells, produced by the breakdown of histidine.

Leukotriene: One of a group of biologically active compounds consisting of a straight-chain, 20-carbon carboxylic acid with one or two oxygen substituents and three or more conjugated double bonds.

Appearance: This is usually the first thing EMS providers note, saying to themselves, "this patient looks very ill" or "this patient looks like he might be OK" within seconds of seeing them. This is often based on patient position and behaviors. Many signs of severe respiratory distress relate to posture, including the tripod position, extension of the neck, flaring of nostrils, cyanosis (a late sign), accessory muscle use and the appearance of fatigue. All those factors suggest severe distress.

Work of breathing: This 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. In fact, when paradoxical abdominal movement is observed, it's a good sign of *inspiratory* muscle fatigue.⁶ Paradoxical abdominal movement occurs when the diaphragm is so tired that when the chest wall moves outward, the change in pressure pulls the diaphragm upward, thereby pulling the abdominal contents inward.

Circulation: Overall circulatory status is reflected in the skin. When the circulatory system experiences problems, it shunts circulation from non-vital organs, which includes the skin. Obvious signs of severe distress include mottling and cyanosis.

Face to face: It should be first noted that some, albeit few, asthmatics develop a hyperacute "sudden asphyxial asthma" that results in death within hours.³ For the majority of asthmatics, however, a severe asthma attack comes after a period of poor overall control of their asthma.

Patients with asthma present with many symptoms, including dyspnea, wheezing and coughing. Coughing appears early in asthma attacks and may be the only complaint.³ Thus, asthmatic patients can be overlooked in the field as having "cold" or

"flu-like" symptoms. Coughing is often seen in elderly patients, probably as a result of subepithelial vagal stimulation.³

Medical history: Most likely, patients will have a medical history of asthma or other respiratory disease. A list of history elements should be elicited because they'll provide clues about the severity of the asthmatic's disease and risk factors for death from asthma (see Table 1, p. 60). 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. Other important history elements include the presence of diseases, such as diabetes, peptic ulcers, hypertension and psychosis, which may worsen with the use of corticosteroids.³

Physical assessment: The patient who has an altered mental status and is taking short, shallow breaths is obviously having a severe attack. Aside from the observations made using the IVAT, and the clues revealed by the history, several other clinical indicators aid providers in recognizing the severe asthmatic. For example, 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 doesn't correlate with severity of the attack, except when the rate is greater than 40 breaths per minute.³ That rate can drop as the patient becomes fatigued and begins to go into respiratory failure. The presence of wheezing depends on airflow and the velocity of that airflow. It isn't usually a good indicator of the severity of an asthma attack; however, it's safe to say that the lack of wheezing and the lack of airflow despite maximal effort indicates a severe event.³ Note the inspiration-to-expiration ratio. Asthma usually results in a prolonged expiratory phase, which can be detected

using digital waveform capnography.

Tachypnea with tachycardia greater than 120 may indicate a severe attack.³ Blood pressure may reveal pulsus paradoxus. When present, a fall in systolic greater than 10 mmHg during inspiration signifies a severe attack.³

Skin can provide clues as to the severity of the attack. Diaphoresis can occur secondary to the increased work of breathing seen with an asthma attack. Profound diaphoresis accompanied by a decreasing level of agitation indicates severe asthma and may herald impending death.³ Cyanosis is uncommon because of the respiratory alkalosis caused by the attack.

MANAGEMENT

As illustrated by the opening case, rapid clinical intervention in the setting of a severe asthma attack is critical. Caregivers must recognize the need to treat the current condition and prepare for the next stages of progression. Management, although primarily pharmacological, may demand all your skills. For example, a patient receiving medications through a nebulizer may fatigue, requiring the provider to attach CPAP or use a bag-valve mask to assist respirations while using an adapter to continue nebulizing medications.

Endotracheal intubation (ETI), while discouraged, may also be required. ETI should be considered if local protocols allow, particularly for those patients who present with a RR of 40 or more, fatigue (i.e., a patient telling you they're tired of breathing), failure to improve with maximal initial therapy, altered mental status; worsening pulses (bradycardia) and/or peak flow in red zone and severe hypercapnia and pH less than 7.3.

Coaching the patient through their breathing treatment is an essential skill that's often neglected. The goal is twofold. First, providers should coach the patient to breathe deeply through the mouth to ensure the medicine and oxygen reach as deeply into the lungs as possible. Second, coaching helps reduce anxiety. To decrease the patient's anxiety level, it might be advantageous to limit the patient care area to personnel who are necessary to provide care. Scene control during a critical call is required to provide the highest quality emergency medical care.

A core group of pharmacological agents

can be used in the prehospital setting. In selecting the appropriate pharmacological agent, EMS providers usually give preference to agents that are fast acting, have few side effects, are safe to administer and provide maximal benefit. The current school of thought for treatment of an acute asthma attack includes the use of rapid-acting, beta-2 sympathetic agonists, inhaled anticholinergics, IV corticosteroids and IV magnesium sulfate, a medication that many systems use for a severe asthma patient who isn't responding to the first-line drugs. However, crews must watch for contraindications of magnesium, including hypocalcemia, persistent hypertension, heart block, myocardial damage, shock and myasthe-

nia gravis. beta-2 receptor activation, resulting in adenylyl cyclase being stimulated and thus, the increasing translation of adenosine triphosphate (ATP) to cyclic AMP (cAMP).⁷ An increase in cAMP within vascular smooth muscle causes generalized smooth muscle relaxation.

The biochemical explanation is this: Smooth muscle relaxation occurs because cAMP inhibits myosin light chain kinase, which is responsible for phosphorylating smooth muscle myosin.⁸ In other words, it inhibits what the smooth muscle needs to contract, thus increasing intracellular cAMP caused by beta-2 agonists inhibits myosin light chain kinase and producing less contractile force.

Simply put, such medications as albuterol

One clinical indicator that a patient is improving after bronchodilator therapy includes diminished breath sounds that are transition to wheezes that can now be heard. However, maximal effectiveness may not be reached for up to 30 minutes. The bronchodilatory effects typically last for three or four hours.



nia gravis.

Epinephrine is a nonselective alpha and beta agonist that stimulates the release of norepinephrine and causes bronchodilation. The acceptable dose to administer to a patient with a pulse who is experiencing an asthma attack is 0.3 mg of 1:1,000 subcutaneously or via IM injection. Epinephrine must be given with caution because it may cause or increase the severity of hypertension, tachycardia, and/or ventricular ectopic activity. This is of special concern if the patient is taking digitalis, because it causes the heart to become sensitive to the effects of epinephrine.

Beta-2 sympathetic agonists, such as albuterol (proventyl and ventolin) have been used in the prehospital and emergency care fields for many years as a rescue medication for asthma. Albuterol is the one of the most popular rapid-acting beta-2 sympathomimetic drugs used for treatment of emergent bronchoconstriction and bronchospasm on the market. Bronchodilation occurs due to

(a selective beta-2 stimulant), increase cAMP, which makes smooth muscle weaker. Therefore, activation of beta-2 adrenoceptors in the lungs causes bronchodilation. It's important to understand that this bronchodilation is a relaxation of the muscle, but isn't complete.⁹ When albuterol is administered, bronchodilation can occur within minutes. The acceptable initial dose of albuterol is 2.5 mg.

One clinical indicator that a patient is improving after bronchodilator therapy includes diminished breath sounds that are transition to wheezes that can now be heard. However, maximal effectiveness may not be reached for up to 30 minutes. The bronchodilatory effects typically last for three or four hours.

Some EMS systems opt to administer lev-albuterol, which is a different formulation of albuterol, alone and in combination with ipratropium when giving nebulized breathing treatments. Ipratropium bromide is a popular inhaled anticholinergic (parasympatholytic)

agent that causes bronchodilation and blocks secretions. Ipratropium causes a dramatic decrease in bronchoconstriction and can help reverse the airway obstruction seen in asthma, especially when used in conjunction with a fast-acting beta-2 sympathomimetic agent, such as albuterol or levalbuterol. Ipratropium exerts its maximum effects in approximately 30–120 minutes after administration; its effects can last up to six hours.³

EMS providers use ipratropium as a secondary agent and not a primary treatment because it's not as effective a bronchodilator as albuterol and has a slower onset. For this reason, ipratropium shouldn't be used unaccompanied in patients suffering from a severe asthma attack. Using it in conjunction with albuterol/levalbuterol allows the patient to benefit from the rapid onset of action of the beta-2 agonist, as well as the slower but extremely effective anticholinergic treatment.³

Magnesium sulfate is another safe, fast-acting, effective treatment option for the patient experiencing a severe asthma attack. Studies have demonstrated that it causes bronchial smooth muscle relaxation.¹⁰ Data suggest that the mechanism by which magnesium sulfate causes smooth muscle relaxation is by blocking of calcium channels, as well as blocking of parasympathetic neuromuscular transmission. A combination of these effects might lead to the overall bronchodilatory effects of magnesium sulfate.

A reasonable IV dose of magnesium sulfate is 2–3 g administered over 20 minutes or at rates of up to 1 g/min while continuing aggressive inhalation therapy.³ Side effects associated with the administration of magnesium sulfate can include nausea, vomiting, flushing, a systemic feeling of warmth, hypotension, muscle weakness, a loss of deep tendon reflexes and respiratory depression.³ These side effects are usually manageable, as well as infusion-rate dependent. Be observant and continually reassess your patient for signs of impending respirator depression or failure during all treatment regimens.

A multitude of prehospital systems in the U.S.—such as South Carolina EMS and the New Hampshire Department of Safety and Division of Fire Standards and Training and EMS—administer IV methylprednisolone “Solu-medrol” for use in moderate to severe asthma, or those with clinical symptomology indicative of impending respiratory failure.

Table 1: Risk Factors for Death from Asthma

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

Source: Marx J, Hockberger R, Walls R. *Rosen's Emergency Medicine*. Elsevier: Philadelphia, 1078–1096, 2006.

The standard IV adult dose in the emergency setting ranges from 60–125 mg for adults every six to eight hours until patient improvement is seen. The onset of action of Solu-medrol occurs in hours, not minutes, so this agent isn't suitable a primary rescue drug to reverse an acute asthma attack.³ Methylprednisolone's maximal effectiveness usually peaks around the 24-hour mark.³

CONCLUSION

The rapid and proper assessment and management of the severe asthmatic can contribute to an improvement in morbidity and mortality. EMS personnel must have the ability to recognize the clues that suggest a patient is having a “severe” attack.

They must understand the pathophysiology that underlies the disease to appreciate that the “airway remodeling” and persistent inflammation are constantly lurking in the background, explaining why EMS interventions are primarily pharmacological but may require the use of all of the provider's skills. *JEMS*

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For more about asthma, visit jems.com/journal

REVIEW QUESTIONS

Test your comprehension with this post-article quiz. Answers are provided at the end. Photocopying is permitted for nonprofit training purposes only. For readers in need of continuing education credits, please visit JEMSCe.com to choose from courses that are CECBEMS approved and meet NREMT refresher requirements.



- What best describes the pathophysiology of asthma?
 - Loss of surfactant leads to atelectasis, stiff lung and pulmonary edema
 - Breakdown of alveolar walls, leading to shunting, air trapping and hypoxia
 - Inflammation of lower airways, resulting in bronchial swelling, spasm and secretions
 - Swelling of the trachea and spasm of the vocal cords, leading to upper airway obstruction
- Which of these is part of the Initial Visual Assessment Technique (IVAT)?
 - Work of breathing
 - First set of vital signs
 - Glasgow Coma Score
 - Pulse oximetry reading
- Hypoxemia with cyanosis is seen in the _____ stage of asthma.
 - Early
 - Late
- What capnography waveform change indicates incomplete or obstructed exhalation requiring additional bronchodilators?
 - Loss of waveform entirely
 - Square waveform half its normal height
 - Increasingly narrower, rectangular waveforms
 - Shark fin pattern with loss of rectangular plateau
- A conscious and alert adult with a history of asthma became short of breath today while running. The patient can speak in full sentences, has diffuse bilateral wheezing and no exercise intolerance. Vital signs are BP 120/76, P 90, R 24, SpO₂ 95% and EtCO₂ 45. Which of these should be given first?
 - Magnesium sulfate IVP
 - Epinephrine 1:10,000 IVP
 - Albuterol and ipratropium (Atrovent)
 - Epinephrine 1:1,000 IM or subcutaneously
- An adult with a history of asthma is complaining of severe respiratory distress. They've taken eight puffs of their MDI in the past hour with no relief. Their skin is pale, warm and dry; breath sounds are absent in all lung fields, and the patient is speaking in one-syllable words. Vital signs are BP 130/80, P 64, R 40, RA SpO₂ 90% and capnography has a shark fin waveform with a numeric reading of 50. Besides CPAP and an inhaled beta agonist, which of these is indicated first as a rescue drug?
 - Magnesium IVP
 - Methylprednisolone IVP
 - Epinephrine 1:10,000 IVP
 - Epinephrine 1:1000 Sub-q or IM
- Which of these may be indicated (based on local protocols) for a patient with a severe asthma attack who isn't responding to the first-line drugs?
 - Magnesium sulfate IVP
 - Diphenhydramine IM
 - Azmacort by MDI
 - Epinephrine IVP
- What's the desired action of albuterol when treating an asthma attack?
 - Vagus nerve blocker
 - Coronary artery dilator
 - Smooth muscle inotrope
 - Selective beta-2 stimulant
- Which of these is an acceptable initial dose of albuterol for a patient with asthma?
 - 0.5 mg
 - 2.5 mg
 - 10 mg
 - 25 mg
- Which of these is a common side effect of albuterol?
 - Syncope
 - Bradycardia
 - Nervousness
 - Constricted pupils
- What's the action of ipratropium?
 - Alpha stimulant
 - Selective beta-2 agonist
 - Calcium channel blocker
 - Parasympatholytic (anticholinergic)
- Which of these indicates that a patient with a severe asthma attack is improving after bronchodilator therapy?
 - Patient develops pulsus paradoxus
 - Tachycardia transitions to bradycardia
 - Diminished breath sounds transition to wheezing
 - Peak flow readings go from the yellow to the red zone
- What's the desired action of epinephrine when given to a patient with an asthma attack?
 - Anticholinergic agent to dry secretions
 - Alpha stimulant resulting in vasoconstriction
 - Anti-inflammatory agent to decrease hyper-reactivity
 - Nonselective alpha and beta agonist to cause bronchodilation
- Which of these would be a precaution relative to giving epinephrine to a patient with an asthma attack?
 - Hypertension
 - Peanut allergy
 - Pulse rate that's borderline bradycardic
 - Diminished breath sounds in all lung fields
- Which of these is an acceptable dose of epinephrine for a patient with a pulse who's experiencing an asthma attack?
 - (1:1,000) 1 mg IM
 - (1:10,000) 1 mg/kg IVP
 - (1:1,000) 0.3 mg IM or subcutaneously
 - (1:10,000) 0.1 mg increments up to 1 mg IVP
- Which of these are common side effects of epinephrine when given to a patient with asthma?
 - Drowsiness and dry mouth
 - Tachycardia and dysrhythmias
 - Respiratory depression and irritability
 - Hypotension and increased respiratory rate
- What's the desired action of magnesium when given for an acute asthma attack?
 - Potent antihistamine
 - Suppresses nerve impulse transmission
 - Reverse the adverse side effects of beta agonists
 - Bronchial smooth muscle relaxant due to Ca blocking properties
- Which of these is a contraindication to giving magnesium to a patient with a severe asthma attack?
 - Tachycardia
 - Myasthenia gravis
 - Patient taking Paxil or Elavil
 - Patient with glucose intolerance
- What's the desired action of steroids when given to patients with asthma?
 - Rescue bronchodilator to stop an attack
 - Anti-inflammatory agent that treats the disease
 - Beta-2 stimulant to open narrowed bronchioles
 - Anticholinergic bronchodilator to supplement albuterol
- Which of these is at risk for a potentially fatal asthma attack?
 - History of intubation or syncope due to asthma
 - Needs to use their rescue inhalers at least twice a week
 - Needs to use inhaled steroids along with long-acting beta agonists
 - Wakes up at least once per week short of breath due to their asthma

1. C 2. A 3. B 4. D 5. D 6. D 7. A 8. D 9. B 10. C 11. D 12. C 13. D 14. A 15. C 16. B 17. D 18. B 19. B 20. A