

Asthma exacerbations

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Objectives

At the end of the CE module, the EMS provider will be able to:

1. Describe different causes/stimuli and the pathophysiology behind an asthma exacerbation.
2. Recognize the different severity levels of an asthma exacerbation.
3. Recognize the risk factors that increase the chance of death.
4. List the drugs commonly used to treat this disease in the prehospital emergency field.

The call

It is one o'clock in the afternoon on a weekday; you and your paramedic partner are responding to a shortness of breath call. You just pull up at the two-story house on a cul-de-sac when a thin, middle-aged male runs out the front door toward your ambulance, flailing his arms wildly. He appears pale and terrified. When he gets very close to you, you can hear a short, high-pitched inspiratory and expiratory wheeze with every breath. His mouth is wide open as he gasps for air and every accessory respiratory muscle appears to be in use.

Introduction

Asthma is a chronic inflammatory pulmonary disease affecting approximately 25 million Americans—8.2 percent of adults and 9.4 percent of children nationwide.¹ The acute asthma exacerbation, also called an episode or attack, accounts for around 1.7 million emergency room visits and more than 3000 deaths per year.^{1,2} Considering these statistics, it is no surprise the “shortness of breath” call is commonplace in the prehospital setting, and most EMS systems carry numerous drugs to treat asthma-related symptoms.

The basic anatomy review

The respiratory system is divided into two basic units—the upper and lower respiratory tracts. The separation occurs at the level of the larynx, with the larynx being in the upper tract and the trachea belonging to the lower. Asthma is a lower respiratory disease; therefore, the anatomy and physiology of this tract will be the focus of this review. The trachea is a tube between 12 to 15 centimeters in length consisting of smooth muscle and cartilage. Due to the ability of smooth muscle to contract, the trachea has nearly 20 C-shaped cartilage pieces spaced out along its entire length that provide a framework to hold the trachea open. The esophagus is posterior to the trachea along the opening of the “C”, this arrangement allows for expansion of the esophagus when food is swallowed.

The inferior end of the trachea divides into the left and right primary bronchi. This bifurcation occurs at a cartilage section called the carina. The primary bronchi then divide into secondary bronchi. Each secondary bronchus supports a lobe of the lungs, the right lung has three lobes and the left lung is divided into two. This discrepancy is because the left lung has a section missing on its

medial side to accommodate the heart, called the cardiac notch, making it smaller than the right lung. Every bronchus continues branching and getting smaller; from the secondary bronchi to tertiary bronchi, then bronchioles, and then the smallest tubes called terminal bronchioles. This entire system resembles an inverted tree and is often called the bronchial tree. There are two structural aspects of the bronchial tree that play a significant role in the symptoms associated with an asthma exacerbation. As the diameter of the bronchial tube decreases the amount of cartilage decreases, and there is an increase in smooth muscle. The number of goblet cells also decreases.

The majority of the thoracic cavity is occupied by the right and left lungs, which are separated by the mediastinum. Each lung is cone-shaped and rests on the diaphragm with its apex near the level of the clavicle. The lungs are soft and spongy because they are composed of millions of tiny air sacs called alveoli. The bronchial tree's terminal bronchioles continue dividing into alveolar ducts; the ducts lead into clusters of alveoli. If the bronchial system can be described as a tree, the alveoli clusters resemble bunches of grapes at the ends of the tree branches. Each alveolar sac is surrounded by pulmonary capillaries where gas exchange takes place. The thin respiratory membrane between the alveoli and the pulmonary capillaries allows for quick and efficient gas exchange, and this is the only area in the pulmonary system where gas exchange occurs. That means all other space in the respiratory tree is dead space. Oxygenated blood leaves the pulmonary capillaries and travels throughout the body dispensing oxygen and picking up carbon dioxide. This deoxygenated blood makes its way back to the pulmonary capillaries where it diffuses into the alveoli and is exhaled. This continuous cycle of internal and external respiration is dependent on three variables: effective ventilation, adequate circulation (perfusion) and no impedance to diffusion or gas exchange. Asthma is considered a defect in ventilation.

The stimulus

Asthma is a chronic condition that can become a life-threatening emergency due to an acute exacerbation, or attack. These episodes are inflammatory responses to various stimuli, sometimes referred to as triggers. The stimulus causing an asthma exacerbation can be classified

as either intrinsic or extrinsic. Extrinsic stimuli are antigens that lead to an allergic response and cause an asthma exacerbation. These patients would show a positive skin test for the stimuli. Examples of extrinsic stimuli include cats, insects, peanuts and mold. Intrinsic stimuli are those not associated with a known antigen; these include a viral infection, exercise, cold weather and stress. Viral infection is a common cause of asthma exacerbation and may also be related to the development of asthma.³ Aspirin is a common intrinsic trigger; one in five asthmatic adults can suffer an exacerbation from aspirin.² There is a positive correlation between asthma, aspirin sensitivity and nasal polyps, called Samter's Triad, and patients with all three conditions are more likely to trigger an asthma attack with aspirin administration. This condition is found in females more often than in males.⁴ Previously asthma, and not just the stimulus, has been classified using the immune response associated with different triggers; extrinsic asthma and intrinsic asthma. Extrinsic asthma was related to an immune response while intrinsic asthma was not. However, research has shown similar immune-related responses associated with both extrinsic and intrinsic triggers; supporting the argument that the allergy / non-allergy classification may be too simplistic.⁴ Triggers are individual and vary between each asthmatic; while extensive, this list is not all-inclusive.

The call

When you open the doors to the back of the ambulance the patient is right behind you and climbs in. While you are preparing your equipment for your continued assessment and treatment, a woman claiming to be the patient's girlfriend comes to the back of the truck. She tells you he has been feeling sick for a few days with a productive cough and a fever. He is a pack a day smoker but hasn't been smoking for a couple days. He has a long history of asthma and ran out of his emergency inhaler a couple weeks ago. The previous year, on two separate occasions, he was admitted to the hospital after going to the ER for severe asthma exacerbation. He has no other medical history.

Asthma pathophysiology

Asthma is a complex disease that can entail chronic inflammation, airway remodeling, bronchial smooth muscle hypertrophy, chronic increased mucous production and epithelium cell

injury. Many cells associated with the immune system have a role, including histamine containing mast cells, macrophages and eosinophils. Asthma is a chronic condition but, when an asthmatic is exposed to a trigger, they can suffer an acute asthma exacerbation and the lungs of an asthmatic are excessively responsive to triggers. An exposure causes an increased inflammatory response that leads to edema formation, increased mucous production, tissue swelling and constriction of bronchial smooth muscle. The increased production of mucous leads to the formation of mucous plugs and subsequent bronchial plugging. As the bronchial tree divides and narrows, the amount of smooth muscle available to constrict increases; this causes an increased resistance to airflow in the smaller airways. The lungs of asthmatics have altered elastic recoil and, possibly, more collapsible airways than non-asthmatics.⁵ Structural changes seen in asthmatics do not only occur in the lungs but also stretch into the peripheral areas outside of the reach of inhaled medications.⁵ Air trapping, and hyperinflation of the thoracic cavity, can occur due to airway obstruction from bronchoconstriction, airway remodeling and mucous plugging. Besides hyperinflation, this also results in the longer inspiratory phase than expiratory phase sometimes seen in these patients. Air trapping and hyperinflation are more common in a severe asthma exacerbation than mild or moderate one.⁶

The call

Your patient appears alert and terrified. He is unable to speak and is gesturing wildly to keep your attention. His respiratory rate is 38 breaths per minute. He has a palpable and regular radial pulse and is tachycardic at a rate of 136 bpm. There is sinus tachycardia on the monitor without ectopy. The patient's skin is pale and diaphoretic. There is no evidence of fever. When you auscultate the chest you hear a slight wheeze in both apexes during inspiration and expiration and the bases are both silent. The pulse oximetry value is 88 percent on room air.

Classifying the asthma exacerbation

An acute asthma exacerbation can be categorized as mild, moderate or severe depending on the presenting signs and symptoms.

Mild: A patient who is able to speak in full sentences, walk around and is able to lie down. She may experience shortness of breath when walking, have no accessory muscle use and no wheezing or only slight expiratory wheezes will be present

during auscultation. This patient can be tachypneic with a normal heart rate (60 to 100 beats per minute in an adult).⁶

Moderate: A patient who speaks in phrases and prefers to sit. He is often breathless while sitting and may engage accessory muscles while breathing. During a moderate attack the wheezing may be audible without the aid of a stethoscope and is usually noted through the expiratory phase. Respiratory rates are increased and he is tachycardic but the rate, for an adult, is usually less than 120 bpm.⁶ Infants may have trouble feeding. Mild or moderate acute asthma attacks can dissipate without medication or progress to a severe attack.

Severe: This is a true medical emergency requiring immediate medical treatment. These patients will often be agitated or confused, due to hypoxia, and they may be fighting with responders. Typically they have audible wheezing on inspiration and expiration or they may have progressed to the "silent chest" stage. Due to mucous plugging, bronchial constriction and air trapping ventilation is compromised; patients may pass little or no air through their lungs. These patients will have a remarkably increased work of breathing, noticeable accessory muscle use and will only speak in one or two word sentences. Skin will often be diaphoretic and pale. Cyanosis is an ominous sign. In an attempt to facilitate ventilations, these patients will often be sitting in the tripod position. The increased energy these patients must expend in order to breathe will cause them to fatigue quickly; this is especially true in pediatrics. Adult patients experiencing a severe exacerbation will usually have respiratory rates greater than 30 and a heart rate greater than 120 bpm.⁶ Infants having a severe exacerbation will be unable to feed.

Pulsus paradoxus, an abnormally large drop in systolic pressure during inspiration, is another indicator of a severe asthma exacerbation. In healthy patients systolic pressure normally falls less than 10 mm Hg during inspiration; however, adults suffering from a severe attack may experience a systolic pressure drop of more than 25 mm Hg. In children this number typically falls between 20 to 40 mm Hg. Pulsus paradoxus is caused by decreased ventricular preload. There is increased pressure in the thoracic cavity due to air trapping; this impedes blood flow back to the heart from the low-pressure venous system.

Status asthmaticus is a severe asthma exacerbation that does not respond to initial standard treatment.

The physical examination

As with every call, the paramedic's physical exam will start prior to ever touching the patient by evaluating level of consciousness, airway patency and work of breathing. Take notice of whether the patient is able to speak in full sentences, his posture and any accessory muscle use. Patients who exhibit somnolence, an inability to speak, a decreased reaction to painful stimuli and have a PCO₂ greater than or equal to 42 mm Hg are in impending danger of complete respiratory failure.⁶

Follow the standard head-to-toe physical examination given to every medical patient, taking special care to evaluate breath sounds and look for the presence of subcutaneous emphysema. Due to air trapping, the asthmatic has an increased risk for a pneumothorax. Evaluate all vital signs, including oxygen saturation and ECG monitoring; Supraventricular tachycardia is the most common arrhythmia seen during an asthma exacerbation, all other arrhythmias are rare.⁷ A pulse oximetry reading of less than 92 percent (or less than 90 percent depending on source) is indicative of a severe asthma attack.^{6,7}

Medical history

Most of these patients are aware of their asthma and recognize the symptoms of an asthma exacerbation; if able to speak they will likely tell you they are having an attack. If the patient is able to communicate, or if a family member is present, certain historical events will aid in patient assessment and help the paramedic prepare for further treatment. It is important to ask about previous intubations, hospitalization and ICU stays. A patient history that includes recent exposure to a known trigger will help confirm the diagnosis of asthma. Inquire about medications, compliance with medications and stimulus exposure. It will also be helpful to note whether the patient has self-administered an emergency short-acting beta agonist (SABA) since the onset of symptoms and whether any relief was obtained. Find out the duration of the present symptoms, any concomitant medical history, allergies and social history (including smoking). Has the patient recently been ill, had any incident of fever or increased temperature, had a productive cough or change in sputum color? A positive answer to any of these questions may indicate pneumonia or a viral infection, which are both common intrinsic stimuli. It may be helpful to find out if the patient has an at-home asthma action plan, which helps track symptoms, pulmonary function tests and treatment medications.

Important risk factors

Being familiar with the risk factors that are associated with an increased chance of death in the patient with a severe asthma exacerbation may help guide the patient's treatment. The Expert Panel Report 3 issued by the National Asthma Education and Prevention Program (NAEPP) and the National Heart Lung and Blood Institute (NHLBI) provide an detailed list of identifiable risk factors.⁶ Besides previous intubations, ICU admissions and emergency rooms visits, other factors include another chronic pulmonary, cardiovascular disease, illicit drug use and a history of using more than two SABA canisters per month. Another factor that increases the risk of death due to an asthma exacerbation is the inability to recognize the symptoms associated with a worsening of the disease. Patients who may be unable to realize the severity of their symptoms include those with a psychiatric illness or those under the influence of drugs (legal or illicit) or alcohol.

The drugs

All drugs and dosages discussed are based on the NAEPP and the NHLBI recommendations in their Expert Panel Report 3, Section 5, Managing Exacerbations of Asthma.⁶ Emergency medical technicians and paramedics should follow their local protocols.

In the emergency medical field oxygen is the first drug indicated for any patient with a patent airway in respiratory distress who displays signs of hypoxia. The asthmatic is no exception to this rule. In the dyspneic patient presenting with any signs indicative of hypoxia, 100 percent oxygen is appropriate.

After supplemental oxygen, standard treatment for an acute asthma exacerbation is the short-acting beta agonist. In most EMS systems paramedics administer the bronchodilator albuterol. Albuterol is a beta-2 agonist, meaning it binds with beta-2 receptors in the lungs. This leads to decreased pulmonary resistance through smooth muscle relaxation and bronchodilation. Albuterol is selective for beta-2, so it has only minimal effect on heart rate (which contains beta 1 receptors). Usually, it is given prehospitally in two forms: the metered dose inhaler (MDI) and nebulized. For patients having a severe exacerbation nebulized is the most appropriate route. This may be true depending on the severity of the moderate attack as well, the age of the patient and the patient's ability to follow directions. The adult dose for albuterol is 2.5mg to 5mg in 3cc normal saline administered via a nebulizer mask with 6 to 8 lpm



(liters per minute) oxygen. If the oxygen flow is too high the medication droplets will be too small, if the rate is too slow they will not reach their target. The rate of 6 to 8 lpm has been determined to provide the best albuterol droplet size.⁶ The pediatric dose is 0.15mg/kg with a minimum dose of 2.5 mg. In both adults and pediatrics, albuterol can be repeated every twenty minutes to a max of three times in one hour. Albuterol's onset of action is quick (less than five minutes) and peak effects occur 30 to 60 minutes post administration. It is only contraindicated in patients with a known hypersensitivity. Adverse side effects may include anxiety, nervousness, tremors and tachycardia; however, patients in respiratory distress will usually demonstrate a paradoxical bradycardia when albuterol is administered. This occurs because with bronchial dilation the patient experiences a decrease in their work of breathing, their heart rate slows down as they are able to ventilate better and they become less anxious. Albuterol also causes a decrease in serum potassium levels of 0.4mEq/L; however, the benefits of treatment far outweigh the slight risk of arrhythmia due to the decrease potassium.⁷

If administering albuterol with an MDI, the dose is 90mcg per puff with a recommended four to eight puffs every 20 minutes up to four hours in adults. In pediatrics the four to eight puffs can be repeated every 20 minutes to a maximum of three doses. Albuterol delivered via MDI has been shown to be just as effective as nebulized albuterol when administered for a mild or moderate exacerbation as long as the patient is properly coached on proper administration techniques. A spacer or value-holding chamber may help ensure the MDI is used correctly.

After the first dose of albuterol, many systems will move on to a nebulized mixture of albuterol and atrovent, often referred to as an A&A. Atrovent (ipratropium bromide) is an anticholinergic, it works to dry secretions in the airway and possesses bronchodilating effects. Atrovent will increase the effectiveness of albuterol. The nebulized adult dose for atrovent is 0.5mg and the pediatric dose is 0.25mg to 0.5mg. Like albuterol, atrovent can be repeated every 20 minutes to a maximum of three doses in one hour; however, it is not recommended as a front-line treatment because its onset of action time of 20 minutes is not as fast-acting as albuterol.⁸ Also, atrovent should not be considered appropriate therapy with in every asthma exacerbation; the administration of atrovent is only indicated in patients suffering a severe asthma exacerbation.^{6,7} Depending on patient status, it is

appropriate to repeat the albuterol without atrovent.

Terbutaline (Brethine) is another bronchodilator and beta-2 agonist used in some EMS systems. Terbutaline has a slower onset of action than albuterol and is indicated after the administration of albuterol. The adult dose is 0.25mg given as a subcutaneous injection; this may be repeated in 20 minutes and administered a total of three times. The pediatric dose is 0.01mg/kg and this may be repeated every 20 minutes to a maximum of three doses. Terbutaline is contraindicated in patients with a known hypersensitivity to the drug.

The expert panel recommends oral systemic corticosteroids for moderate to severe exacerbations that do not respond to initial treatments with a short-acting beta agonist. If gastrointestinal absorption is not adversely affected parenteral administration offers no known benefits over oral therapy.^{6,8} If respiratory arrest is imminent intravenous corticosteroids would be appropriate. Corticosteroid are anti-inflammatory drugs that mimic the corticosteroid hormones naturally secreted by the adrenal cortex. These stress hormones suppress inflammation, which works to relieve asthma symptoms by reducing swelling in the airway and decreasing edema formation. Inhaled or systemic glucocorticoids have been shown to decrease hospitalization rates and increase pulmonary function when administered to treat an acute asthma exacerbation.⁹ Due to a delayed onset of action time paramedics will not see the beneficial effects offered by this drug in the field; corticosteroids produce their anti-inflammatory effects six to 12 hours post administration.⁸ There are a number of steroid drugs used in the treatment of asthma, including prednisone, methylprednisolone and dexamethasone. Paramedics should follow local protocols and drug dosage for the corticosteroid administer in their system.

Epinephrine is a sympathomimetic with bronchodilating effects. It is indicated for severe asthma exacerbations which do not respond appropriately to albuterol and atrovent. The adult dose for epinephrine is 0.3mg to 0.5mg IM given subcutaneously. The pediatric dose for epinephrine is 0.01mg/kg not to exceed 0.5mg.⁶ Unlike albuterol, epinephrine is a non-selective beta agonist—it will affect beta receptors in the lungs and the heart so side effects may include cardiac irritability, tachycardia and nervousness.

The Expert Panel 3 report recommends considering magnesium sulfate and/or heliox for severe exacerbations that do not respond to the previously listed treatments. Magnesium is believed to have a desirable affect due to its smooth-muscle

relaxant properties and by reducing histamine release; the recommended adult dose is 2 grams intravenously. The pediatric dose is 25 to 75mg/kg to a maximum of 2 grams. Heliox is not typically administered by prehospital responders and will not be reviewed in this article.

Mechanical ventilation

Due to an increased risk of nosocomial infections, barotrauma and other complications, intubation should be used as a last resort for these patients. Intubation is not considered a treatment for a severe asthma exacerbation—it only allows a means of ventilation until the disease process is resolved through medication. There have been some reports supporting the use of noninvasive ventilation techniques, such as CPAP, to treat asthma exacerbation, however this is still considered an experimental treatment due to the paucity of data.^{6,10,11} When mechanically ventilating these patients, either by intubation or by bag-valve mask assisted ventilations, hypoventilation is recommended.⁶ A slow ventilation rate may help minimize airway pressures.

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

About the author

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