

**Northwest Community EMS System
Paramedic Education Program**

ASTHMA

Connie J. Mattera, M.S., R.N., EMT-P

Reading assignment:

Aehlert Vol. 1 pp 711 - 725

SOPs: COPD/Asthma

Drugs to know: albuterol; epinephrine 1:1000; ipratropium, magnesium

OBJECTIVES:

Upon reading the textbook and completion of the class and homework questions, each participant will independently do the following with at least an 80% degree of accuracy without critical error:

1. Define asthma using the National Asthma Education and Prevention Program (AEPP) guidelines.
2. List major categories of asthma triggers.
3. Generalize the pathophysiology of asthma focusing on the immune system responses.
4. Highlight the three major clinical manifestations of asthma.
5. Cite key history questions to ask an asthma patient prior to starting therapy.
6. Discuss the physical exam parameters to obtain in an asthma patient.
7. Identify the indices that point to a severe asthma attack in adults.
8. Describe, sequence, and justify the rationale for EMS interventions for an asthma attack.
9. Differentiate the presentation of asthma in a child from an adult.
10. Define status asthmaticus.
11. List warning signals of a potentially fatal asthma attack.
12. Explain why steroids may be necessary in asthma management.
13. Discuss current maintenance regimens to treat chronic asthma.

Asthma

Connie J. Mattera, M.S., R.N., EMT-P

I. Introduction

A. Epidemiology

1. Incidence and morbidity: Over 16 million adults and 7 million children have asthma. Two-three percent of all E.D. visits are for asthma.
2. Recent estimates suggest 10% of Americans have asthma
 - a. Illinois: 7.8%
 - b. U.S.: 7.1%
3. 11 million outpatient visits, 2 million ED visits
4. Can develop anytime, although it usually strikes in childhood.

B. The term, asthma, comes from the Greek word, *aazein*, meaning “sharp breath” or to pant”.

C. **Definition:** Chronic lung disease characterized by **increased reactivity** of the trachea, bronchi, and especially bronchioles to some stimuli which causes widespread reversible airway obstruction (but not always completely so) initiated by an **inflammatory response** in the airways (edema or swelling). This results in **airway narrowing and copious mucous production**. Small airways are clogged by mucus and plugs made of dead cells and serum proteins that leak through inflamed airway walls that decrease air movement and gas exchange.

This refinement of the definition places emphasis on **inflammation and hyperresponsiveness** as the primary abnormalities and highlights the fact that relieving bronchospasm is no longer the primary treatment priority.

D. **Asthma triggers:** In poorly controlled asthma, airways can become more and more sensitized over time so allergens or triggers that were once tolerated begin to cause symptoms. More than 200 occupational triggers have been identified in the literature.

1. **Allergens:** Pollens (flowers, trees, grasses, hay, ragweed, mold spores), feather pillows, down comforters, animals with fur (rabbits, cats, dogs, hamsters, gerbils, chickens, birds), insect parts (between 23-60% of urban residents with asthma have a sensitivity to cockroach allergen (NEJM, 1997); and foods (nuts, chocolate, eggs, orange juice, fish, milk, peanut butter).
2. **Household products:** Vapors from solvents, paint, paint thinner, liquid chlorine bleach; sprays from furniture polish, starch, cleaners, room deodorizers, spray deodorants, perfumes, hair sprays, talcum powder, scented cosmetics.
3. **Dust/dust mites:** Cloth upholstered furniture, bedding, carpets, draperies that gather dust, brooms and dusters that raise dust, dirty filters on hot air furnaces and air conditions that put dust into the air.
4. **Work/school-related exposures:** Dusts (chalk), vapors or fumes from: wood products (western red cedar, some pine and birch woods, mahogany); flour, cereals, grains, coffee, tea, papain; metals (platinum, chromium, nickel, sulfate, soldering fumes); cotton, flax, hemp; mold from decaying hay.
5. **Smoke:** Cigarettes, cigars, pipes
6. **Air pollution:** Weather inversions, traffic jams, smoke-filled rooms. Two components of pollution:
 - a. Ozone, nitrogen dioxide, sulfur dioxide
 - b. Particulates: Small liquid or solid particles less than 10 microns in diameter can reach the lower airways
7. **Weather conditions:** Extremely cold air, excessive humidity, change in seasons

8. **Infections:** Colds, viral illnesses, sore throats, sinusitis
9. **Physical exertion:** Running, climbing stairs
10. **Excitement/emotional stress:** Laughing too hard, crying, or coughing; fear, anger, frustration
11. **Drugs:** Acetylsalicylic acid (ASA), non-steroidal anti-inflammatory agents (NSAIDs), beta-blockers, sulfites in sensitive patients, and food additives.
12. **Other factors:** Gastroesophageal reflux (GERD): Repetitive episodes of gastric aspiration results in airway inflammation and "irritant-induced" asthma.

E. New patterns

1. Children with one asthmatic parent contract asthma 3-6 times the rate of others. With two asthmatic parents, the child has a 10 X increased risk. However, trends show increasing sensitivity to allergens. Toddlers who live with at least one smoker are nearly 3 X as likely to wheeze as kids in smoke-free homes.
2. Found more commonly in urban children and blacks. Proposed causes: poor air quality, psychosocial problems, less access to medical care, increased exposure to allergens.

F. Morbidity/mortality

1. **Good news**
 - a. Death rates have declined
 - b. Close to 4000 deaths/year
2. Highest death rates in blacks; adolescents in 10-14 year age group
3. Highest in cities with economic and social variability from one neighborhood to the next
4. Accounts for 10 million lost work days for adults and 14 million lost school days for children
5. 80% die between 12 am - 6 am. Histamine is at peak levels during this time.
6. **High risk patients with potentially fatal asthma (PFA)**
 - a. Multiple hospitalizations within past year for severe asthma
 - b. A previous near-fatal attack
 - c. ≥ 2 episodes of pneumomediastinum/pneumothorax associated with status asthmaticus
 - d. ≥ 2 hospitalizations for status asthmaticus in the past year despite long-term steroid therapy
 - e. Current use, or recent withdrawal, from systemic steroids
 - f. Acute respiratory acidosis without intubation
 - g. History of intubation seizure, syncope, or respiratory failure (5 year mortality rate of 20%) due to asthma. Asphyxia and not arrhythmias is thought to be the cause of death in fatal asthma.
 - h. Concomitant psychiatric and psychosocial problems (alcohol abuse, bereavement, dysfunctional family) (Pollack, 1998).

G. Factors associated with an increased risk for a fatal asthma attack

1. **Patient factors**
 - a. Many patients cannot interpret the severity of their symptoms and underestimate the urgency despite airway obstruction; failure to seek care in a timely manner or poor access to medical care
 - b. Non-compliance with medications; poor compliance with prescribed treatment regimens
 - c. Conflict between family and healthcare provider regarding an asthma management plan
 - d. "Prednisone phobia": reluctance to use steroids due to Cushinoid S&S
 - e. Major psychiatric disorder; depression

- f. Allergy to the mold *Alternaria*
- g. Refusal to institute allergen control; ↑ environmental pollution; increased indoor time with exposure to dust mites and allergens (carpets, pets, cockroaches, indoor molds)
- h. Marked bronchial hyperreactivity as evidenced by significant changes in FEV1 with albuterol
- i. History of sudden, severe, asthma attack
- j. Tremendous link between patient education and reduced morbidity. Highest risk for adverse outcome in those with low income, poor access to medical care, or psychosocial problems and those who fail to understand or adhere to treatment regimens.

2. Physician factors

- a. Inaccurate assessment of disease severity by physicians due to lack of objective assessment and under-appreciation of:
 - (1) essential differences in management between the acute exacerbation and chronic control.
 - (2) limitations of aerosol treatment.
 - (3) differences between aerosol devices and the optimum techniques for inhalational therapy.
- b. Conflicting opinions in the literature on how to treat
- c. Underuse of steroids
- d. Overuse of Theophylline and beta agonist medications to the exclusion of steroids that are more appropriate for long-term management
- e. Excessively demanding regimens which lead to patient non-compliance (slow taper of steroids with detailed instructions)
- f. Poor follow-up and lack of patient education; inadequate counseling for the management of acute exacerbations

II. Pathophysiology of asthma

- A. Results from an abnormal immune response in the bronchial airways
- B. **Allergen** (stimuli) introduced which could be an antigen, virus, or an environmental pollutant and finds its way to the inner airways causing an immediate drop in pulmonary function. They are ingested by antigen presenting cells. These APCs “present” pieces of the allergen to other immune cells that usually ignore them.
- C. In asthmatics, the cells transform into T_H2 cells. T_H2 cells activate the humoral immune system to form IgE antibodies.
- D. IgE antibodies recognize an allergen. These antibodies sit on mast cells and basophils and block beta 2 receptors.
- E. **Early asthmatic reaction** or EAR occurs within 15 minutes - 3 hours
 - 1. Inflammatory cells, including lymphocytes, eosinophils, and mast cells, are found in the airways of patients with asthma even during periods of clinical stability.
 - 2. When antigens bind to the antibodies on these cells, they secrete substances that attract WBCs including Eosinophilic Chemotactic Factor of Anaphylaxis (ECF-A), leukotriene B (LTB), and Neutrophil Chemotactic Factor (NCF).
 - 3. These mediators initiate the migration of inflammatory cells, which stimulate the release of direct activating mediators, i.e., *histamine, bradykinin, and cysteinyl leukotrienes* (leukotrienes C₄, D₄, and E₄) before symptoms appear.
 - 4. **Histamine:** Irritating chemical attracts inflammation-generating cells (eosinophils) that move from blood to tissues causing inflammation.
 - a. **Bradykinin:** potent bronchoconstrictor

- b. **Leukotrienes:** Endogenous molecules formed by the breakdown of a membrane component (arachidonic acid) via the 5-lipoxygenase enzyme pathway. Leukotrienes are produced by a wide range of inflammatory cells in the airway, attach to sites in smaller bronchioles, and **cause potent bronchoconstriction**. Some scientists believe that they may be the **direct cause of airway irritability** through chemical triggers that cause asthma symptoms. They reduce the amount of stimulation needed to inflame airway walls and extend the response duration by acting similarly to histamine causing **irritation, inflammation, edema, and increased mucous production**. This local effect, combined with prostaglandins, enhances the action of histamine. (Females find that one week before each menstrual period is bad for hay fever due to the increased release of prostaglandins.)
- c. **Platelet Activating Factor (PAF):** Mast cells also secrete PAF, which is suspect in the etiology of asthma. It can trigger bronchial constriction, airway swelling and accumulation of eosinophils.

F. **Immediate physical response**

- 1. **Spasm:** Inflammatory mediators constrict bronchial smooth muscle causing marked airway narrowing which results in wheezing, prolonged expiration, and poor air movement.
- 2. **Swelling:** Inflammatory response increases capillary permeability and mucosal edema in bronchial walls.
- 3. **Secretions:** Goblet cells produce large amounts of thick, tenacious mucus that plugs the tracheobronchial tree. Creates a life-threatening environment. These secretions are difficult to remove and become rock hard mucus plugs.
- 4. **What do we see?**
 - a. Increased WOB
 - b. Increase in distending pressure required significant increase in inspiratory muscle forces resulting in
 - (1) Marked negative pleural pressures
 - (2) Dyspnea
 - (3) Tachypnea
 - (4) Accessory muscle use
- 5. **Dynamic hyperinflation of chest:** Airflow obstruction allows mechanical inspiratory positive pressure to inflate the lungs while slow emptying caused by ↑ expiratory resistance, coupled with early airway closure, produces air trapping and lung hyperinflation. This increases residual volume and total lung capacity.
 - a. During an attack, some airways are occluded and some are distended with old, stale air resulting in **ineffective gas exchange** with ventilation/perfusion mismatching. Areas of poor ventilation increase the dead space, progressively decrease alveolar ventilation, and lead to CO₂ retention (**hypercarbia**) and **hypoxemia**. PaCO₂ may reach 70-80.
 - b. Severe air trapping → hypercapnia → **pneumothorax, pneumo-mediastinum, and sub-q emphysema**.

G. **Late asthmatic reaction** (3-8 hours after early reaction)

In 40% of the patients, the acute reaction is followed 3-8 hours later by airway obstruction, depending on the extent of lung tissue damage. More potent inflammatory cells invade airways, promoting further inflammation and hyperresponsiveness. May persist for hours to several days.

- H. **Chronic inflammatory process** is the final phase in the mechanism of asthma and is perpetually worsened over time unless treatment is initiated. Elastin fibrils are disrupted, airways hyperdistend, and there is hypersecretion of mucus causing irreversible lung damage.
- III. **Patient assessment:** Goal: identify high-risk patients
- A. **Primary assessment:** Evaluate degree of airway obstruction, oxygenation, gas exchange, work of breathing, and cerebral function as affected by fatigue, hypoxia or carbon dioxide narcosis and cardiac status.
1. **Position:** Usually sitting, leaning forward. **BEWARE IF THEY ARE TIRED!** – Implies impending respiratory failure. Patient may be fatigued from the work of breathing and energy needed to exhale. Prepare intubation supplies and other ventilatory support devices.
 2. **Degree of ventilatory distress/**breathlessness, chest tightness. Observe chest wall movement; general ventilatory rate; observe for shallow or deep breaths; Intercostal and supraclavicular **retractions** with inspiration; prominent use of accessory muscles: sternocleidomastoids during inspiration and abdominals during expiration.
 3. **Speech** fragmented by rapid breathing. Assess their ability to speak a full sentence of 4 - 5 words or only 1 - 2 words (word clusters) indicating severe distress. Infants stop feeding.
 4. **Dangers of hypoxemia**
 - a. Aggravates pulmonary hypertension
 - b. Produces increased airway resistance
 - c. Alters cerebral function
 - d. Prevents adequate renal perfusion
 - e. Alters cardiac contractility
 - f. Dysrhythmias
 5. **Skin color/temperature/moisture/turgor:** Observe for hypoxia/dehydration
 - a. **Factors that influence the detection of cyanosis**
 - (1) Rate of blood flow
 - (2) Degree of desaturation
 - (3) Type of light
 - (4) Observer skill
 - (5) Thickness and color of skin
 - b. Need 5 Gm of desaturated hemoglobin to develop cyanosis
 6. S&S of hypoxia (not quantifiable; skin color not reliable in patients who are anemic or have peripheral vasoconstriction)
 - a. Restlessness, anxiety
 - b. Disorientation, confusion
 - c. Dyspnea, hypoventilation
 - d. Cyanosis
 - e. Tachycardia, dysrhythmia
 - f. Hypotension
 7. Quantifiable: pulse oximetry – measures oxygenation
 - a. Many patients have unrecognized hypoxia by physical exam alone and 85% do not complain of respiratory distress, indicating a need for prehospital SpO₂ as an objective measure.
 - b. Limitation due to lack of sensitivity can be offset by adding clinical correlation (artificial elevation of pO₂ and SpO₂ when hyperventilating on room air).

- c. Low SpO₂ (91% or less) is a predictor of poor outcomes
 - d. While peak flow rates correlate with SpO₂ values, do not depend solely on SpO₂ to indicate degree of distress! Beware an SpO₂ < 90% as it correlates to a pO₂ of 60 torr.
8. Capnography (also quantifiable) measures ventilation
- a. In asthma, incomplete or obstructed exhalation is evidenced by a shark-fin pattern on the waveform. This may also be a sign that the ET tube is kinked or obstructed.
 - b. **RED FLAG:** R ≥ 40 and pCO₂ > 40. Tachypnea should result in a pCO₂ in the 20s. Increased work of breathing with normal pCO₂ is poor sign.
9. **Hydration status**
10. **Mental status:** Alertness, patient may appear anxious. Observe for AMS. Syncope or near syncope indicates severe attack.

B. **Secondary assessment**

1. **Chief complaint**

- a. Perception of dyspnea
- b. Orthopnea
- c. Light headedness
- d. Tightness in the chest
- e. Rapid breathing
- f. Cough: productive or non-productive; may have white, thick sputum.
- g. Wheezing

Cough variant asthma: The patient usually (but not always) coughs caused by irritation and/or constriction of the airways. As it progresses, coughing increases spasmodically, causing a lack of O₂ due to bronchospasm.

2. **History of present illness**

- a. **Onset:** How rapidly did the attack progress?
- b. What **provoked** this attack? What are the patient's triggers?
- c. **Recurrence;** prior episodes of respiratory insufficiency due to asthma (loss of consciousness, intubation, or need for mechanical ventilation). Prior hospitalizations and ED visits.
- d. **Repeat visit?** Has the patient visited or been transported to an E.D. or a physician with this attack within the last 24-48 hours? Second visit for same attack indicates severe disease; prepare for patient deterioration.
- e. **Severity** of symptoms, including exercise limitation and disturbance of speech and sleep. Has the patient vomited? How do they rate their symptom severity? Patient's gauge of symptom severity is notoriously inaccurate. **Exercise intolerance:** Difficulty walking 100 feet or more.
- f. **Time** of onset and duration of current attack

3. **SAMPLE history:** Try to determine the severity and chronicity of the patient's asthma

- a. Known **allergies** to food, drugs, environment; recent exposure to the allergen
- b. **Medications**
 - (1) What medications are customarily used to treat their asthma?

- (a) **Prescription beta agonists:** terbutaline (Brethine, Brethaire); albuterol (Proventil, Ventolin); metaproterenol (Alupent, Metaprel); salmeterol xinafoate (Serevent). Regular use of inhaled beta-agonists show an association with risk of hospitalization or death from asthma (O'Hollaren, 2005)
 - (b) **NSAIDs:** Cromolyn Sodium (MAST cell inhibitor); Nedocromil sodium (Tilade) - inhibits histamine and prostaglandin release
 - (c) **Steroids:** beclomethasone (Beclovent, Beconase), triamcinolone acetonide (Azmecort), flunisolide (Aerobid, Aerobid M), fluticasone propionate (Flovent)
 - (2) What medications have been used to treat *this* attack in the last 48 hours? Failure to improve symptoms using outpatient asthma regimen indicates moderate to severe attack. Last and total doses? Route?
 - c. **PMH:** High risk patient? Co-morbid factors: patients > 55 are at ↑ risk of dying due to co-existing cardiac or pulmonary disease. Those on chronic steroids to control symptoms are at high risk of severe disease as are those with psychiatric or psychosocial problems.
 - (1) Previous respiratory arrests or similar episodes;
 - (2) History of seizure during an acute asthma attack;
 - (3) Psychosocial factors and personality traits;
 - d. **Last oral intake;** consider possibility of dehydration
 - e. **Events surrounding this incident (history of present illness)**
4. **Role of infections**
- a. May be responsible for a severe asthma attack
 - b. Ask patient if they have experienced runny nose, posterior nasal discharge, sore throat, hoarseness, or sputum production
 - c. Severely dyspneic patient with marked ↓ in airflow may not have adequate tussive force to produce purulent sputum to indicate lower respiratory infection
 - d. High doses of steroids to treat asthma may obscure a febrile response
 - e. Hyperinflation of chest may obscure pulmonary infiltrates on chest x-ray and make diagnosis difficult
5. **Full set of vital signs**
- a. Tachypnea with prolonged expiration
 - b. Pulse rate, quality, rhythmicity. Anticipate tachycardia: > 120 minutes (> 160 for infants). Tachycardia may suggest fear, hypoxia, stress or dehydration. Bradycardia is far worse, and may suggest an increase in parasympathetic tone or severe hypoxia.
 - c. **BP:** Relative vs. absolute hypotension. Causes of hypotension
 - (1) Tachy/bradycardia
 - (2) Positive intrathoracic pressure
 - (3) Hypovolemia
 - (4) Dysrhythmia
 - d. **Pulsus paradoxus:** BAD SIGN: Hyperinflation and increased negativity of pleural pressures on inspiration cause significant stress on the cardiovascular system. Increased venous return to a stretched right ventricle leads to **pulmonary hypertension** and interventricular septal deviation. The compromised LV must pump blood from a negative

pressure in the thorax to the systemic circulation (elevated transmural pressure) leading to a fall of systolic BP or a weaker pulse during inspiration.

6. Auscultation of lung sounds

- a. Wheezes may be audible without a stethoscope
- b. Listen right away if patient in distress.
- c. When listening for lung sounds with a stethoscope, detect the amount and quality of air being moved. Inspiratory/expiratory wheezes are often not a reliable sign of asthma severity.
- d. Wheezes are harmonic, musical sounds produced when air passes rapidly through a narrowed bronchus as bronchial walls fluctuate between closed and barely open.
- e. **Decision tree: All that WHEEZES is not asthma!** Consider the presence of other diseases or complications.

A: Asthma

S: Stasis

Pulmonary embolism

T: Toxins

Toxic gasses, smoke inhalation, insecticides, chemical irritants, cholinergic poisonings

H: Heart

HF pulmonary edema - "cardiac asthma", noncardiogenic pulmonary edema – ARDS

M: Mechanical

Foreign body: upper or lower airways

A: Allergy/aspiration

Anaphylaxis, laryngeal edema, organic particle exposure, extrinsic allergic alveolitis, aspiration of gastric contents, near drowning

T: Trauma/tumor

Upper airway, pneumothorax, endobronchial tumor

I: Infection

Bronchitis, pneumonia, bronchiolitis, croup, epiglottitis, pertussis, fungal diseases

C: Chronic

COPD, alpha-1 antitrypsin deficiency, cystic fibrosis, congenital abnormalities, bronchopulmonary dysplasia

- f. **Silent chest:** Wheezing may be absent if attack is severe due to bronchospasm and ↓ airflow. If not reversed, asthma patients will stop wheezing because they are not moving enough air, thus they are called a **silent asthmatic**. A silent chest is an **ominous sign** indicating marked airflow obstruction and may portend respiratory arrest. The patient will start to wheeze again once they start moving enough air, but they are still in severe to moderate respiratory distress.
- g. Assess for pneumonia, atelectasis, and pneumothorax.

C. Objective diagnostic indicators

1. **Peak expiratory flow rate (PEFR)** determined by peak flow meter or Forced expiratory volume in one second (FEV₁) by spirometry after age 5. Expressed in L/minute.
 - a. FEV₁ is measured by an individual taking the deepest possible breath and blowing out as much of that as possible in 1 second.
 - b. Peak flow is obtained by taking the deepest breath possible and blowing out as much as possible into the meter. This measures the fastest rate at which air moves through the airways during a forced expiration.

- c. Many patients (60%) cannot predict their degree of obstruction by clinical symptoms. They report their distress as mild, yet have poor peak flow readings. Patients with moderate persistent or severe persistent asthma may have peak flow meters at home and keep a peak flow diary.
- d. **Advantages:** Gross assessment of obstruction severity, evaluates response to Rx, and detects changes in airflow prior to symptoms. Evaluates response to treatment.
 - (1) **Technique**
 - (a) Place mouthpiece on meter
 - (b) Set meter to zero
 - (c) Take deepest breath possible
 - (d) Blow out as hard and as fast as possible into the meter for 2-3 seconds
 - (e) Keep meter horizontal while taking the reading
- e. **Peak flow limitations:** PEFR reflects obstruction (airflow limitation) mainly in the larger airways. Easiest to obtain but not as sensitive as spirometry and may underestimate the degree of airflow obstruction. Results depend on consistent, proper patient effort and technique. It has limited utility in children less than 5.
- f. **Precautions**
 - (1) Do not force a patient to take a reading before they are moving enough air to get a useful measurement. Peak flow attempts can precipitate bronchospasm.
 - (2) Patients need to be coached to provide maximum effort and provide a daily diary of PEFR taken upon rising and at bedtime.
 - (3) The highest of 3 readings are recorded. Personal best values are calculated when not having an attack.
 - (4) **Best predicted values:** Predicted normal values are determined by a nomogram that takes into consideration age, sex, and height. Personal best is the highest reading after bronchodilator medication when well. Peak flow variability > 20%/day = poor asthma control
- g. Significant reversibility is indicated by an increase of $\geq 12\%$ and 200 ml in FEV_1
- h. **Asthma severity classifications**
 - (1) **Mild:** "Green zone" (doing well): $\geq 80\%$ of predicted; PEFR diurnal variations < 20%
 - (2) **Moderate:** "Yellow zone": 50-80% of predicted; Needs quick relief treatments
 - (3) **Severe:** "Red zone" (medical alert!): $\leq 50\%$ predicted or personal best **or** failure of PEFR to improve at least 100 l/min or 10% after initial treatment. Needs quick relief treatments and immediate transport to hospital.

IV. Indices of sudden asphyxic asthma (signs of crashing)

A. Most reliable physical exam findings

1. Altered mental status (most ominous); agitated, drowsy, confused, syncope
2. Inability to speak more than one to three syllables at a time
3. Markedly diminished or absent breath sounds (silent chest)

4. Central cyanosis: arterial $pO_2 < 50$ mmHg (V_A/Q disturbance)

B. Usually reliable

1. Use of accessory muscles: marked sternocleidomastoid retractions (associated with $FEV_1 < 1$ L)
2. Diaphoresis due to SOB: if absent, suspect dehydration
3. Exhaustion; weak respiratory effort
4. Inability to lie flat (ability to lie supine predictive of peak flow of at least 150 L/min and $PaCO_2$ of no more than 45 mmHg) (Pollack, 1998)
5. Significantly altered VS: HR > 120 /min or < 60 ; RR > 25 -30/minute; RR is variable and is not always correlated to the severity of the attack

C. Less reliable

1. Degree of dyspnea; c/o chest tightness
2. Presence of cough
3. Severity of wheezing; prolonged expiratory phase
4. ECG abnormalities

V. Emergency management

- A. Initial treatment should begin at point of patient contact

B. Goals of exacerbation therapy

1. Early recognition of worsening lung function by patient/EMS team
2. Prompt entry into the emergency medical system
3. Rapid relief of airflow obstruction
4. Prevention/correction of hypoxemia while avoiding intubation and mechanical ventilation if possible
5. Reduce work of breathing
6. Appropriate intensification of anti-asthma medications to provide direct and in-direct bronchodilation
7. Reduce inflammation

C. Initial airway access

1. Patient positioning; non-invasive adjuncts
2. Have patient breathe out past pursed lips to replicate PEEP

- D. **Supplemental oxygen:** The presence of hypoxemia correlates poorly with the severity of airflow obstruction

1. Provide supplemental oxygen to all patients with moderate to severe attacks as large doses of sympathomimetic agents induce ventilation/perfusion mismatches that oxygen therapy can offset. O_2 also helps address the issue of respiratory muscle fatigue.
2. **Mild to moderate distress:** Apply appropriate device/Liter flow depending on pulse ox reading pending administration of bronchodilators. Supplement 6 L nebulized O_2 with nasal cannula if moderate distress or hook nebulizer to mask. May provide in-line nebulizer using BVM if ventilatory assistance is necessary.
3. Attempt to keep $SpO_2 > 92\%$ -94% (95% in peds, pregnant women, and patients with heart disease). Hypoxemia aggravates pulmonary hypertension, produces \uparrow airway resistance, alters cerebral function, prevents adequate renal perfusion and alters cardiac contractility. Dysrhythmias are more prevalent in presence of hypoxia.
4. Severe distress: Once the crashing asthmatic patient is intubated, mortality is reported at 10%-40%. Use permissive hypercapnia whenever possible with non-invasive ventilatory support (C-PAP/Bi-PAP) to prevent intubation. Goal is to keep them off the ventilator. Mechanical ventilation is strongly associated with barotrauma in these patients.

Noninvasive pressure support ventilation: C-PAP

- a. Muscles maintaining autopeep (expiratory effort) can be rested when external PEEP is applied and they can be recruited into inspiratory effort, thereby reducing inspiratory work of breathing.
 - b. Analogous to pursed lip breathing
 - c. Use with caution, both may cause barotrauma
 - d. Purpose is to reduce subjective dyspnea, work of breathing
 - e. Best comfort levels: C-PAP started with PEEP at 5 cm ↓ pulmonary resistance and most patients feel better. Studies with status asthmaticus show benefits at 8 cm H₂O. Don't approach 10 cm H₂O, which is where auto-PEEP occurs.
 - f. If SBP falls under 90: Remove C-PAP.
5. Drug assisted **intubation** should be considered for those who present with the following:
- a. RR 40 or more;
 - b. Exhaustion (patient telling you they're tired of breathing)
 - c. Severe hypoxia (SpO₂ < 90); failure to improve with maximal initial therapy;
 - d. Near apnea; coma or depressed mental status;
 - e. Peak flow in red zone
 - f. Severe hypercapnia (EtCO₂ > 60 mmHg); pH < 7.3
 - g. Hemodynamic instability, worsening pulses (bradycardia)
 - h. Impending respiratory failure or arrest.
 - i. If assisted ventilation/intubation required: **ventilate at 6 - 8 BPM** [slower rate, smaller tidal volume (6-8 mL/kg), This rate allows longer expiration time and complete exhalation (1:4 ratio).
 - j. Troubleshooting after intubation
 - (1) Breath stacking from rapid or deep BVM ventilations can result in hyperinflation; hypotension; & tension pneumo
 - (2) If BP falls or resistance is noted:
 - (a) Verify tube position; ✓displacement
 - (b) Eliminate obstructions
 - (c) R/O pneumothorax & equip. failure
 - (3) If tension pneumo – decompress

E. Circulation/cardiac status

1. Venous access and optimal hydration: initiate IV NS
 - a. No evidence that large volumes of IV fluids alter the consistency or viscosity of secretions to promote their clearance. Hydration has not been shown to affect pulmonary secretions in patients who have normal access to liquids.
 - b. Multiple cases of acute pulmonary edema have been reported in the setting of asthma. High negative intrapleural pressures predispose to capillary leakage.
 - c. Infants and young children may become dehydrated more rapidly from ↑ RR and ↓ oral intake. They may be unable to access fluids themselves and may require IV fluids if objectively depleted.
2. Monitor **ECG**

F. Drugs

1. Drug therapy has changed greatly in the past decade. Medications are used based on the severity of disease. In the past, most treatment was aimed at controlling symptoms and reversing bronchospasm. Now it is focused on the inflammatory

component. However, anti-inflammatory medications will not stop an attack already in progress. Medications are classified as either **quick relief** or **long-term control/preventive** agents.

2. **Quick relief short-acting beta-2 agonists:** Bronchodilators act to stop an asthma attack that has already begun but do not reverse airway hyperresponsiveness or inflammation. They are prescribed to be used on an as needed basis but are not usually meant to be used every day.
 - a. First line drugs **selectively stimulate Beta-2 receptors** that promote production of cyclic AMP in the cells. Cyclic AMP stimulates the sodium/potassium pump, which moves sodium into the extracellular fluid and potassium into the intracellular fluids. This action prevents calcium from entering the contractile elements of smooth muscle cells, resulting in **bronchodilation**. In addition, they enhance mucociliary clearance.
 - b. **Advantages:** Act in < 5 minutes and may last up to 6 hours; inhalation route has least cardiac side effects. Optimal dose is titratable and patient-specific
 - c. **Fast acting beta agonists**
 - (1) **Albuterol 2.5 mg/3 ml NS (Proventil, Ventolin)**
 - (2) Metaproterenol sulfate (Alupent, Metaprel) 15 mg/3 ml,
 - (3) pirbuterol (Maxair)
 - (4) isoetharine (Bronkosol, Bronkometer)
 - (5) bitolterol (Tornalate)
 - (6) levalbuterol (Xopenex)
 - d. **Devices used to deliver inhaled medications**
 - (1) Pressurized metered dose inhalers (MDIs)
 - (2) Breath-actuated metered dose inhalers
 - (3) Dry powder inhalers (Accuhalers, Turbuhalers): require an inspiratory effort that may be difficult during severe attacks and for children under 5
 - (4) Nebulizer pumps
 - (5) **Hand held nebulizer (HHN):** Convert liquid medication into a cloud of aerosol particles. Usually driven by compressed air (oxygen). Can deliver smaller particles than an inhaler, and can penetrate deeper into the airways.
 - e. **MDI precautions**
 - (1) If you see any vapor escaping from the mouth, they have not inhaled the full dose.
 - (2) Do not poke anything into the hole in the canister. If you think it may be blocked, wash under warm water.
 - (3) If you cannot hear a liquid moving inside when you shake the canister, it is probably empty. The canister delivers the same dose, even if it is nearly empty.
 - (4) There is impaired MDI function in cold conditions (increased particle size with decreased airway penetration). Duration of action is unknown and varies with severity of disease.
3. Studies have shown **no clear advantage of nebulized drug over use of MDIs** in mild to moderate attacks as long as the patient can coordinate hand motion and breathing. Nebulizers are most often used when the patient is having a severe attack, is dyspneic and tachypneic and may have difficulty in coordinating inhalation of an MDI.

4. This can be corrected through the use of a **spacer** device (AeroChamber, AeroChamber with mask; small and large) in all patients < 10 and patient coaching. The spacer must fit the inhaler. The size of the spacer must increase as a child grows and lung size increases.
 - a. Children < 2 should use spacer and face mask or nebulizer
 - b. Children 2-5 should use an MDI with spacer or a nebulizer prn
 - c. Children > 5 who have difficulty using an MDI should use a pressurized MDI with spacer, a breath-actuated inhaler, a dry powder inhaler, or nebulizer
5. **Albuterol** 2.5 mg /3 mL via HHN with O₂ at 6 L. Supplement O₂ with a nasal cannula. Repeat every 20 minutes X 3 or provide continuous nebulizer therapy for 1 hour.
6. **Xopenex** (levalbuterol HCl) has removed the (s)-isomer that causes the cardiac side effects. More expensive than albuterol with no clear clinical advantage to most patients.
7. **Side effects of beta agonists::** ANS stimulation producing rapid or irregular HR, shakiness, or nervousness, nausea, sweating; dry mouth, heartburn, altered taste.
8. **Use with caution** if patient has a history of overactive thyroid, heart disease, hypertension, epilepsy, diabetes, or is pregnant or breast feeding.
9. **Drug interactions**
 - a. **Beta blockers:** propranolol, labetalol, timolol
 - b. **Drugs containing ephedrine, epinephrine, or pseudoephedrine**
 - c. **Drugs for depression: MAO inhibitors** (furazolidone, phenelzine, selegiline, tranylcypromine)
10. **Parenteral adrenergics**
 - a. If a patient fails to respond sufficiently to inhaled beta₂ agonists, the drugs may be unable to penetrate the smaller airways effectively and may be largely deposited in the mouth.
 - b. Local protocols may prescribe parenteral non-selective alpha and beta adrenergic agonists such as epinephrine, terbutaline, and/or isoproterenol if a patient presents with a severe attack and deteriorating ventilations and/or life-threatening asthma.
 - c. An individualized risk-benefit analysis is needed prior to administration of any parenteral drugs.
 - d. **Epinephrine** has been used to treat acute asthma since 1910. Some physicians are reluctant to order this drug for adults over 40 or in patients with coexistent heart disease because of the potential cardiovascular side effects. Cydulka (1988) studied 95 patients (108 episodes) ranging in age from 15-96 years of age who were treated with three subcutaneous doses of 0.3 mL epinephrine 20 minutes apart and found that there was no significant difference in the occurrence of ventricular dysrhythmias in patients less than or more than 40 years old. She found that the mean diastolic BP, mean HR, and mean respiratory rate decreased with treatment in the older population.
 - (1) Epi relaxes bronchial smooth muscles and constricts bronchial arterioles. It reduces congestion and edema and thus improves pulmonary function. It has an onset of detectable systemic levels within five to ten minutes after sub-q injection and reaches peak blood levels between 20-40 minutes. Its bronchodilating effects last up to four hours.

- (2) If the patient is conscious and oriented, is not exhausted, and has adequate baseline cardiopulmonary reserves, sub-q epi may eliminate the need for intubation.
- (3) **Adults: Epinephrine 1:1000 0.3 mg sub-q. or IM May repeat X 1** in 10 min. Do not repeat if patient develops VT, three or more consecutive PVCs accompanied by hypotension, or angina. **Peds:** Epi 1:1000 0.01 mg/kg up to 0.3 mg sub-q. May repeat in 10 min.
- (4) Precautions: Elderly or those with HR > 100, CVD/HTN due to epi's beta-1 & alpha effects, those who are pregnant or experiencing significant SE to albuterol.
- (5) Epi increases myocardial irritability and may precipitate cardiac dysrhythmias. Sinus tach and isolated PVCs are the most common dysrhythmias in hospitalized asthmatic patients and both disappear with correction of arterial oxygen tension and pulmonary function.
- (6) Note on **SODIUM BISULFITES** - Common preservative used in foods, beer, wine, shellfish, salads and many medications including 99% of marketed epinephrine. It is estimated that 1% to 2% of asthmatics are allergic to this preservative. American Regent Labs markets an epinephrine 1:1000 ampule 1 mg with no preservatives.

e. **Terbutaline sulfate** (Brethaire, Brethine, Bricanyl) (Not our System)

- (1) Selective Beta-2 adrenergic agent. It causes a significant increase in HR, cardiac output, and systolic BP when given in therapeutic doses. When compared to equivalent doses of epinephrine, terbutaline caused a significantly greater and more prolonged increase in HR (Cydulka et al, 1988).
- (2) Should be reserved for patients < 30 years of age due to beta-1 side effects (Pollack, 1998).
- (3) **Sub-q dose:** 0.01 mg/kg up to 0.3 mg sub-q every 2-6 hours
- (4) **IV dose:** 10 mcg over 10 minutes loading dose. 0.4 mcg/kg/min; increase as necessary by 0.2 mcg/kg/min and expect to use 3-6 mcg/kg/min.

11. **Anticholinergics (parasympatholytic)**

Ipratropium bromide (Atrovent): Less potent and slower acting bronchodilator than a beta-2 agonist through its action of decreasing the muscarinic vagal constriction of the airways, but may have an additive effect when used simultaneously (shown more conclusively for COPD than asthma). Qureshi (1997) demonstrated improvement when used in combination over Albuterol alone. Dose: HHN 0.5 mg. Peak action at 30-90 min after inhalation. Does not need to be repeated in the field.

12. **Magnesium sulfate** 2 Gm mixed with 16 mL NS slow IVP over 5 minutes. Possible bronchial smooth muscle relaxant effects due to Ca blocking properties. May also interfere with acute inflammatory response. May potentiate beta agonists and offset beta tachyphylaxis. Used by some physicians when conventional therapy is ineffective as a small subset of patients have a strikingly prompt response when refractory to other therapies. Side effects are dose related (pain at infusion site, facial flushing, sweating, nausea, vomiting, hypertension and occasionally, CNS depression).

13. **Anti-inflammatory drugs:** Early **IV or nebulized steroids** are crucial at the hospital. Steroids interfere with the synthesis of inflammatory agents and prevent migration and activation of inflammatory cells. **Steroids treat the disease, beta-agonists only treat the symptoms.**

- a. **Methylprednisolone** sodium succinate (A-Methapred, Solu-Medrol 125 mg IV q. 6-8 hours for first 36-48 hours. Time to peak effect: 6-12 hours. Alternative: hydrocortisone sodium succinate (Hydrocortone Phosphate, A-Hydrocort, Solu-Cortef) 2 mg/kg IV q. 4 h or 100-500 mg in a single injection followed by continuous infusion of 0.5 mg/kg/h.
- b. **Oral alternative:** either Prednisone 2 mg/kg (max 60 mg) or oral Methylprednisolone (Medrol) 60 mg initially, then 60-120 mg q. 24 hours in divided doses with tapering based on response. Prevailing thought: start high and rapidly taper to avoid unwanted side effects.

14. **Less conventional therapies (also hospital-based)**

- a. **Heliox** (helium/oxygen mixture) 70/30% or 80/20% (this O₂ is lower than room air, must have extra O₂ support). Improves efficiency of gas flow in distal airways and oxygen/carbon dioxide diffusion.
 - (1) Heliox makes airflow less turbulent, more laminar, and more normal to improve gas exchange
 - (2) Dramatically decreases pCO₂ (ave. 36 mm) in a few minutes, peaking at 20 minutes.
 - (3) Decreases peak airway pressures (average 33 cm H₂O); improves peak flow
 - (4) Improves retention of beta agonists
- b. **Methylxanthines** (Aminophylline - theophylline, ethylenediamine)
 - (1) 3-4 times less effective alone than beta agonists but promotes bronchodilation in small airways that may not be affected by inhaled medications. May also have favorable effects on diaphragm function and relieve subjective complaints of dyspnea in patients with COPD.
 - (2) Prevailing medical thought: **Do not use aminophylline as a first line drug for either acute or chronic asthma.** Reserve for severe bronchospasm that does not respond to beta agonists and subsequent steroids. For chronic asthma, Theophylline (Theo-dur, Uni-phyl, Slo-bid and others is considered a third line choice, but may be helpful with nocturnal asthma due to its sustained duration.
 - (3) Has narrow therapeutic index. Causes increased adverse side effects without effecting additive bronchodilation including cardiac dysrhythmias, nausea, vomiting, severe weakness, confusion, irregular heart beat and seizures. Ingesting large amounts of caffeine may increase side effects.
 - (4) Some drugs may increase theophylline levels
 - (a) Cimetidine (Tagamet)
 - (b) Erythromycin (E-Mycin, E.E.S.)
 - (c) Ciprofloxacin (Cipro)
 - (5) Some drugs may decrease theophylline levels
 - (a) Phenytoin (Dilantin)
 - (b) Carbamazepine (Tegretol)
 - (6) Detoxified poorly in smokers and in those with HF and liver disease and quickly results in toxicity.
 - (7) Contraindications
 - (a) Allergies to Methylxanthines
 - (b) Those with abnormal heart rhythms that are poorly controlled
 - (c) Those with seizures that are poorly controlled

(d) Those with hyperactive thyroid

(e) Those with peptic ulcer disease

15. No diphenhydramine! Big problem in asthma = mucous plugs. Diphenhydramine will dry mucous plugs and make them rock hard. Can't oxygenate past plugs.

G. On-going assessment: Monitor the following:

1. Mental status
2. Degree of dyspnea; speech
3. Use of accessory muscles; WOB
4. Respiratory rate, effort, orthopnea
5. Presence and intensity of breath sounds
6. Degree of wheezing
7. Peak flows, SpO₂, capnography, ECG

VI. Chronic asthma control medications (long-term preventive medications)

- A. In February 1997, the National Institutes of Health issued the National Asthma Education and Prevention Program's Expert Panel Report II: Guidelines for the Diagnosis and Management of Asthma. This document was prepared by a multidisciplinary group of clinicians and scientists with expertise in asthma management.
- B. The guidelines refined the previous expert panel report (1991) and provided five sections to address the panel's goals: pathogenesis, assessment and monitoring, contributing factors and control of asthma triggers, pharmacotherapy, and patient education. Those guidelines were updated again in 2002.
- C. New practice parameters
1. Published 11/06 in the Journal of Allergy & Clinical Immunology
 2. Joint effort of three professional organizations
 - a. Am Academy of Allergy, Asthma, and Immunology
 - b. Am College of Allergy, Asthma, and Immunology
 - c. Joint Council of Allergy, Asthma, and Immunology
 3. Important concept is now asthma CONTROL
 4. Control is achieved through a step-wise approach to meds with a strong emphasis on anti-inflammatory drugs
- D. **Current asthma classifications:** Mild, moderate, or severe exacerbations can occur in patients in any category.

Classification of Asthma Severity				
Severity prior to initiation of therapy				
	Mild intermittent	Mild persistent	Moderate persistent	Severe persistent
Symptoms	≤ 2 / week	> 2 / week	Daily symptoms	Continual
Nighttime symptoms	≤ 2 / month	> 2 / month	> 1 / week	Frequent
Lung function	≥ 80% predicted	≥ 80% predicted	> 60% - ≤ 80%	≤ 60%
Peak flow variability	< 20%	20-30%	> 30%	> 30%

VII. Achieving control of asthma requirements

A. Component 1: Assessment and monitoring

1. Asthma severity
2. Daytime symptoms

3. Nighttime symptoms
4. Lung function (peak flow measurements, pulmonary function tests)
5. Asthma control
6. Current degree of impairment
7. Risk of future impairment

B. Component 2: Education

1. Should occur at every point of care
2. Importance of partnering for effective care
3. Assess and teach self-monitoring skills
4. Develop an asthma action plan

C. Component 3: Control of environmental factors

1. Identify allergic sensitivities/triggers
2. Incorporate measures to reduce indoor and outdoor allergens/irritants
3. Manage comorbidities

D. Component 4: Medication therapy

1. Step-wise approach to treatment based on achieving good asthma control
2. Long-term controller medication (corticosteroids, LABAs, mast cell stabilizers, immunomodulators, leukotriene modifiers, methylxanthines)
3. Short-term rescue medication (anticholinergics, SABAs, systemic corticosteroids) (McCormick, 2010)

E. Goals of long-term drug use

1. Control chronic and nocturnal symptoms
2. Maintain normal activity levels, including exercise
3. Maintain near-normal pulmonary function
4. Prevent acute episodes of asthma
5. Require little or no quick-relief medication
6. Avoid adverse effects of asthma medications

F. Asthma is well controlled if

1. asthma S/S are experienced 2 times a week or less.
2. rescue bronchodilators therapy is used 2 times/week or less.
3. there is no night time or early morning awakening for asthma or dyspnea.
4. there are no limitations for work, school or exercise.
5. the patient and their physician consider it well controlled.
6. the patient's PEF or FEV₁ is normal or personal best.

G. Corticosteroids

1. If the patient uses a bronchodilator puffer more than 2 times per week, they need preventative medications
2. Inhaled steroids reduce airway inflammation
3. They must be taken regularly even if asthma is under good control
4. Inhaled drugs reduce the need for prolonged or chronic use of oral steroids and facilitate withdrawal when short courses of oral steroids are used during acute exacerbations.
5. Inhaled steroids such as **triamcinolone (Azmecort)**, **beclomethasone (Vanceril, Beclovent)**, **budesonide (Pulmicort Turbuhaler)**, **flunisolide (AeroBid)**, **fluticasone propionate (Flovent)** may prevent progression of the disease, provide a better long-term prognosis, and reduce or eliminate the needs for oral systemic steroids. Before starting on steroids, some children respond well to a 4 to 6 week trial with cromolyn sodium. Adults may respond to nedocromil (Tilade). If successful, they do not need inhaled steroids.
6. **Typical dose:** 4 - 20 puffs/24 hours. If disease worsens: 16-24 puffs/24 hours.

- a. **Local adverse effects:** Headache, hoarseness, cough, upper respiratory infection, GI upset, oral thrush (candidiasis), which may be lessened by the use of a spacer device (AeroChamber, InspirEase, or Optihaler) and rinsing the mouth after inhalation.
- b. **Systemic adverse effects:** A small amount of inhaled steroids are swallowed with each dose, but it is much less than that contained in oral preparations, so there are fewer side effects of long-term use. Doses above 1 mg/day may be associated with skin thinning, easy bruising, and adrenal suppression. Long-term use of oral corticosteroids may lead to osteoporosis, arterial hypertension, diabetes, cataracts, hypothalamic-pituitary-adrenal axis suppression, obesity, or muscle weakness.

H. **Leukotriene modifiers**

1. Leukotrienes promote the inflammatory response caused by exposure to allergens. Less leukotriene = less inflammation = fewer symptoms
2. Advantage: Available as tablets, chewable tablets, and oral granules; once daily dosing
3. Contraindications: Individuals with phenylketonuria (PKU) should not take the chewable tablets that contain aspartame as this artificial sweetener contains phenylalanines (eMedicine, 2003)
4. **Singulair^R** (montelukast)
 - a. **Indications:** Alternative, but not preferred treatment, for mild persistent asthma in adults and children 6 years of age and older.
 - b. **Action:** Selective leukotriene receptor antagonist similar to Accolate.
 - c. **Advantages over Accolate:** Once daily dosing of 10 mg PO in the evening (peds: 5 mg chewable cherry-flavored tabs); fewer drug interactions, and administration without regard to meals. Efficacy has been reported in chronic stable asthma, exercise-induced asthma, and aspirin intolerance.
 - d. Side effects (generally mild): Headache, fatigue, fever, GI upset, laryngitis, and pharyngitis. An FDA notice in March 2008 reported a link between Singulair and psychiatric adverse events such as agitation, aggression, and suicidal behavior.
5. **Accolate** (zafirlukast)
 - a. **Indications:** Prophylaxis and chronic treatment of asthma in adults and children 7 years and older.
 - b. **Action:** Blocks receptors needed for leukotrienes to work. Blocks daytime and nighttime symptoms, decreases beta-agonist use, and improves pulmonary function in patients with mild-to-moderate asthma (Fish et al, 1997).
 - c. **Precautions:** Levels increase when given with ASA and decrease if taken with erythromycin and theophylline. Potentiates coumadin, patients should have their PTs/INRs monitored and dose adjusted.
 - d. **Dose:** Adults: 20 mg P.O. BID. Take one hour before or two hours after meals. Peds 7-11 years: 10 mg BID.
 - e. Low **side effect** profile: headache, infection (respiratory tract), GI upset, pain, fever, elevated liver enzymes (rare)
6. **Zyflo** (zileuton): Long-term use drug which inhibits an enzyme needed to make leukotrienes (5-lipoxygenase inhibitor). Dose: 600 mg QID. Not to be taken concurrently with Seldane due to risk of cardiac dysrhythmias. Causes Inderal levels to double, potentiates theophylline. Increases bleeding times when given with Coumadin. Extensively metabolized in the liver and early studies show

incidence of ↑ liver enzymes requiring lab assessments monthly for three months, then every two to three months for the next year, then periodically. Patients must be taught when to take additional medication. Alert them to seek help for respiratory distress, increased breathlessness, increased need for inhalers, and decreased peak flow levels below 50% best predicted.

I. **Long-term anti-inflammatory agents (MAST cell inhibitors)**

1. Inhibit early and late-phase reactions that are associated with asthma symptoms and the development of severe asthma by preventing the release of histamine and other proinflammatory mediators. The drug is not effective until 4-7 days after the first dose.
2. Nedocromil (**Tilade**) 1.75 mg/spray, MDI: Inhaled pyranoquinolines provide a wider span of anti-inflammatory activity by inhibiting various airway inflammatory cells and reactions; is more efficacious than Cromolyn. Side effects: unpleasant taste, upper respiratory symptoms, GI upset.
 - a. Not recommended in children < 2
 - b. Inhibits late-phase reactions
 - c. Less potent than steroids and less effective for severe asthma
 - d. *Not a bronchodilator so is not effective in an acute attack*
 - e. Can be used 15 minutes before exercise or allergen exposure
 - f. Nedocromil may have steroid-sparing actions; can use smaller dose of steroid
3. Cromolyn sodium (**Intal**): NSAID by inhalation (nebulizer or MDI)
 - a. **Indications:** Usually safe and effective for preventing immune response attacks in patients with mild to moderate asthma
 - b. **Action:** Prevents degradation of MAST cells. Variable onset of action. Not very effective for patients with severe disease.
 - c. **Dose/route:**
 - (1) Adults and children > 5: MDI 0.8 mg/inh 2 puffs QID
 - (2) 2 years and older: Nebulizer solution: 20 mg/2 ml QID
 - d. **Side effects:** Bronchospasm, throat irritation, bad taste, cough, wheezing, nasal congestion, anaphylaxis

J. **Long-acting Beta-2 agonists (LABAs)**

1. Maintenance therapy does not stop progressive inflammatory changes which are responsible for chronic asthma and development of severe disease. Prior use of beta agonists for maintenance may ↓ the effectiveness of anti-inflammatory agents. Some data imply that regular daily therapy w/ beta agonists may ↑ hyperresponsiveness and worsen asthma.
2. Most experts agree that regular daily therapy with beta agonists should be in combination with anti-inflammatory agents (steroids, cromolyn or nedocromil).
3. Patients with mild asthma should need to use these inhalers only three to four times a week. A pattern of regular or increasing use approaching eight to 12 puffs each day reflects poor asthma control and should trigger further evaluation.
4. Feb 18, 2010: FDA notified healthcare professionals and consumers that, due to safety concerns, FDA is requiring a risk management strategy (REMS) and class-labeling changes for all LABAs. The REMS will require a revised Medication Guide written specifically for patients, and a plan to educate healthcare professionals about the appropriate use of LABAs. These changes are based on FDA's analyses of studies showing an increased risk of severe exacerbation of asthma symptoms, leading to hospitalizations in pediatric and adult patients as well as death in some patients using LABAs for the treatment of asthma.

Healthcare professionals are reminded that to ensure the safe use of these products:

- a. Single-ingredient LABAs should only be used in combination with an asthma controller medication; they should not be used alone.
- b. LABAs should only be used long-term in patients whose asthma cannot be adequately controlled on asthma controller medications.
- c. LABAs should be used for the shortest duration of time required to achieve control of asthma symptoms and discontinued, if possible, once asthma control is achieved. Patients should then be maintained on an asthma controller medication.
- d. Pediatric and adolescent patients who require the addition of a LABA to an inhaled corticosteroid should use a combination product containing both an inhaled corticosteroid and a LABA, to ensure compliance with both medications.
- e. Black box warning: FDA has determined that the benefits of LABAs in improving asthma symptoms outweigh the potential risks when used appropriately with an asthma controller medication in patients who need the addition of LABAs. FDA believes the safety measures recommended will improve the safe use of these drugs. Increased death rate from asthma reported.
- f. <http://www.fda.gov/Safety/MedWatch/SafetyInformation/SafetyAlertsforHumanMedicalProducts/ucm201003.htm>

5. **Salmeterol xinafoate (Serevent)** 21 mcg/inh MDI. Adult dose: 2 puffs (42 mcg) taken by metered dose inhaler BID about 12 hours apart. Not recommended for children. It provides nighttime control of asthma symptoms, prevents exercise-induced bronchospasm, and is an alternative to prolonged-release theophylline agents. Not an anti-inflammatory. It is not a replacement for inhaled corticosteroids, which should be continued at the same dose and not stopped or reduced when treatment with salmeterol is initiated. Recommended for use in combination with a steroid for those not controlled by steroid monotherapy.

Onset of action too slow for use in an acute attack. Side effects: headache (10%), tremor (3%), and cough (3%), respiratory tract infection, nasopharyngitis, and GI disturbances.

6. **Formoterol (Foradil)** - q. d. dosing. Considerable anti-inflammatory activity. Seems to inhibit late-phase asthma reaction in vitro.
7. **Pirbuterol (Maxair autohaler and Maxair inhaler):** Breath-actuated metered dose inhaler and MDI. Dose: 200 mcg/inh 1-2 inh q. 4-6 hours. Adverse reactions: Paradoxical bronchospasm, nervousness, tremors, headache, palpitations, tachycardia, dizziness, nausea, and cough.
8. **Sustained release tablets:** terbutaline, salbutamol, Proventil, Volmax (albuterol). Side effects may include tachycardia, anxiety, pyrosis, skeletal muscle tremor or cramps, headache, hyperactivity, insomnia, nausea, bronchospasm, or hypokalemia.
9. **Xolair (omalizumab)** for subcutaneous use is an injectable prescription medicine for patients 12 years of age and older with moderate to severe persistent allergic asthma caused by year-round allergens in the air. XOLAIR is not a rescue medicine and should not be used to treat sudden asthma attacks. It has been found to cause a slightly elevated risk of cardiovascular and cerebrovascular serious adverse events and warnings have been put on the label.

K. **Immunomodulators: Monoclonal antibodies**

1. **Newest asthma medication:** omalizumab (Xolair) – prevents the binding of IgE to the receptors on basophils and mast cells. Black box warning to be ready to treat anaphylaxis.
2. Considered for those with persistent, moderate to severe asthma due to seasonal allergies that is not controlled by inhaled steroids
3. Very expensive: \$12,000 to 15,000 per year
4. For children 12 and over. Given as an injection every 2 to 4 weeks. Dose depends on body's IgE levels.

L. **On the horizon**

1. **Bronchial thermoplasty**

- a. First non-drug treatment for asthma
- b. Dr. John Miller and Gerald Cox participated in the Asthma Intervention Research (AIR) Trial published in the NEJM. This was a global clinical study of the effectiveness and safety of bronchial thermoplasty to treat moderate to severe asthma.
- c. It enrolled 112 patients; aged 18 to 65 at 11 centers in 4 countries.
- d. Recipients showed significant positive changes: decreased attacks, increase in number of days with no S/S, improved quality of life, reduced use of medication, and improved asthma control at one year following procedure.
- e. How it works
 - (1) A flexible tube with an expandable wire basket and the end is inserted through the mouth or nose and fed into the airways of the lungs
 - (2) The four arms of the basket are expanded so each comes into contact with the airway wall. Basket wires emit heat to reduce the airway smooth muscle and thus widen the airway.

VIII. **Patient resources**

- A. Allergy and Asthma Network/Mothers of Asthmatics, Inc. (800-878-4403)
- B. American Academy of Asthma, Allergy, and Immunology (800-822-2762)
- C. American College of Allergy, Asthma & Immunology
- D. American Lung Association (212-315-8700)
- E. Asthma and Allergy Foundation of America (800-7-ASTHMA)
- F. National Asthma Education Program Information Center (301-951-3260)
- G. Asthma in America (202) 736-1658 or www.asthmainamerica.com

- IX. Healthcare provider resources available from the National Institutes of health, National Heart, Lung, and Blood Institute, Bethesda, Maryland 20824 (301-592-8573), and the Global Initiative for Asthma Secretariate, Department of Respiratory Diseases, University Hospital, Ghent, Belgium.

References

- American Heart Association. (October, 2010). Guidelines for CPR and Emergency Cardiovascular Care. *Circulation*, S829-832.
- Corbridge, S. & Corbridge, T.C. (May 2010). Asthma in adolescents and adults. *AJN*, 110 (5), 28-38.
- Cydulka, R., Davison, R., Grammer, L., Parker, M., & Mathews, J. (1988). The use of epinephrine in the treatment of older adult asthmatics. *AEM*, 17(4), 322-326.
- McCormick, M.J. (March-April 2010). Asthma update – four components of care. Nursing Made Incredibly Easy. Accessed on-line: www.NursingMadeIncrediblyEasy.com .
- National Asthma Education and Prevention Program. (2002). *Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma*. U.S. Dept. of Health and Human Services, National Institutes of Health. www.nhlbi.nih.gov/guidelines/asthma/index.htm.
- O'Hollaren, M.T. (Sept. 12, 2005). Sudden death from asthma: Is your patient at risk? www.medscape.com

Comparison of Inhaled Beta-2 agonist Bronchodilators					
Agent	Adrenergic Receptor	Usual dose	Max dose	Onset (Min)	Duration of Activity
albuterol [salbutamol]: (Proventil, Proventil HFA, Ventolin, Asmol, Airtel)	$\beta_1 < \beta_2$	2 puffs q. 4-6 h prn	12 puffs/d	< 5	3-8 hrs.
Ventolin rotocaps	$\beta_1 < \beta_2$	1 capsule q. 4-6 h prn	12 caps/d		
bitolterol mesylate (Tornalate)	$\beta_1 < \beta_2$	2 puffs q. 6-8 h prn	12 puffs/d	3 - 4	$5 \geq 8$ hrs.
metaproterenol sulfate (Alupent, Metaprel): Also in tabs and syrup	$\beta_1 < \beta_2$	2 puffs q. 4-6 h prn	12 puffs/d	5 - 30	2 - 6 hrs.
pirbuterol acetate (Maxair inhaler)	$\beta_1 < \beta_2$	2 puffs q. 4-6 h prn	12 puffs/d	< 5	5 hrs.
Maxair Autohaler	$\beta_1 < \beta_2$	1-2 puffs q. 4-6 h	12 puffs/d		
levalbuterol hydrochloride (Xopenex)	$\beta_1 < \beta_2$	0.63-1.25 mg/neb TID			
salmeterol xinafoate (Serevent); formoterol (Foradil) (LABA)	$\beta_1 < \beta_2$	2 puffs q. 12 h	4 puffs/d	5 - 14	12 hrs.
terbutaline sulfate (Brethaire, Brethine, Bricanyl)	$\beta_1 < \beta_2$	2 puffs q. 4-6 h prn	12 puffs/d	5 - 30	3 - 6 hrs.
isoetharine mesylate (Bronkometer)	$\beta_1 < \beta_2$	1-2 puffs/aerosol q. 4 h 3-7 inhal/HHN q. 4 h	12 puffs/d		
Anticholinergic bronchodilator					
ipratropium bromide (Atrovent) Combivent is an anticholinergic + beta ₂ agonist		1-2 puffs q 4-6 h		12 puffs/d	
Corticosteroids					
budesonide (Pulmicort turbuhaler): dry-powder metered dose inhaler.		200 mcg/inhalation Adult dose: 1-2 puffs 1-2 times per day Children 6 and older: 1 puff BID		4 puffs/BID	
triamcinolone acetonide (Azmecort MDI oral inhaler)		100 mcg/puff 2-4 puffs BID - QID		12-16 puffs/d	
flunisolide: (Aerobid, Aerobid M MDI aerosol - mint flavored, contains menthol)		250 mcg/puff 2-4 puffs BID		8 puffs/d	
fluticasone propionate (Flovent MDI inhalation aerosol)		44 mcg, 110 mcg, 220 mcg 2-4 puffs BID 1 inhalation BID		440-880 mcg/d Depends on current dose of steroid	
prednisone (Deltasone, Liquid pred, Orasone)		5-60 mg/day (adult) 10-40 mg q. 12 h Q.O.D. (peds)			
Beclomethasone (QVAR)					
Mometasone (Asmanex)					
Combination Fluticasone-salmeterol (Advair discus) 100/50; 250/50; 500/50 inhaled powder) Budesonide-formoterol (Symbicort)					
Non-steroidal anti-inflammatory agents (MAST cell stabilizers may soon be unavailable)					
cromolyn sodium (Intal and others))		0.8 mg/puff 2 puffs QID or 20-30 min pre exercise		8 puffs/d	
nedocromil sodium (Tilade)		2 puffs QID		8 puffs/d	
Xanthines					
theophylline (Slo-BID)		16 mg/kg/day in 2-3 divided doses q/ 8-12 hours		400 mg/day	
Theo-24		Ext. rel. caps 100 mg, 200 mg, 300 mg, 400 mg; Once daily			
Theo-dur		200 mg q/ 12 hours		450 mg q. 12 hrs	
Uni-dur				13 mg/kg/d or 900 mg/d whichever is less	
Uniphyll		400 or 600 mg tabs once daily			

dyphylline (Dilor, Lufyllin)	15 mg/kg PO QID 500 mg IM X 1 then 20-500 mg slow IM q/ 2-6 hr	15 mg/kg q. 6 hr
Long-term control medications		Quick-relief medications
*** Corticosteroids ** Cromolyn/nedocromil ** Leukotriene modifiers ** Methylxanthines * Long-acting beta-agonists	* Short-acting beta-agonists * Anti-cholinergics *** Systemic glucocorticosteroids	

*** Most potent and effective anti-inflammatory agents * No demonstrated anti-inflammatory activity

** Some anti-inflammatory activity

Guide for Severity of Asthma Attacks				
Parameter	Mild	Moderate	Severe	Resp arrest imminent
Breathlessness	Walking Can lie down	Talking infant: softer, shorter cry; difficulty feeding DOE; prefers sitting	Dyspnea at rest Infant stops feeding Hunched forward	Exhausted from work of breathing
Talks in	Sentences	Phrases	Words	(Unable to speak)
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
Respiratory rate	Increased	Increased	Often > 30 /minute	Very rapid
Guide to respiratory rates associated with respiratory distress in awake children <div> <div>Age</div> <div> <div>< 2 months</div> <div>2-12 months</div> <div>1-5 years</div> <div>6-8 years</div> </div> </div> <div> <div>Normal rate</div> <div> <div>< 60/minute</div> <div>< 50/minute</div> <div>< 40/minute</div> <div>< 30/minute</div> </div> </div>				
Accessory muscles & suprasternal retractions	Usually not	Usually	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Moderate; often only end expiratory	Loud	Usually loud	Absence of wheeze
Pulse rate	< 100	100-120	> 120	Bradycardia
Guide to limits of normal pulse rate in children <div> <div>Age</div> <div> <div>Infants</div> <div>Preschool</div> <div>School age</div> </div> </div> <div> <div>Normal rate</div> <div> <div>2-12 months</div> <div>1-4 years</div> <div>5-8 years</div> </div> </div> <div> <div>< 160/minute</div> <div>< 120/minute</div> <div>< 110/minute</div> </div>				
Pulsus paradoxus	Absent < 10 mmHg	May be present 10-25 mmHg	Often present > 25 mmHg adult 20-40 mmHg child	Absence suggests respiratory muscle fatigue
PEF after initial bronchodilator; % predicted or % of personal best	Over 80%	60-80%	< 60% predicted or personal best (100 L/min adults) or response lasts < 2 h	Can't measure
PaO₂ room air and/or pCO₂	Not usually necessary to obtain < 45 mmHg	> 60 mmHg and < 45 mmHg	< 60 mmHg Possible respiratory failure	Respiratory failure
SaO₂ on room air	> 95%	91-95%	< 90%	< 90%

Note: The presence of several parameters, but not necessarily all, indicate the general classification of the attack

ALBUTEROL (Proventil)

Classifications	Selective Beta 2 agonist
Actions	<ul style="list-style-type: none">- Selective beta-2 agonist causes smooth muscle relaxation in lungs- Bronchodilator
Indications	Reversible bronchospasm associated with COPD, asthma, or moderate/severe allergic reactions; croup; cystic fibrosis
Dose & route	<p>For bronchospasm: 2.5 mg in 3 mL (0.083%) via HHN with O₂ at 6-8 L depending on unit until mist stops (5-15 min). May use HHN, mask or BVM.</p> <p>Continue/repeat enroute.</p> <p>SE from MDIs are blunted by using a spacer device.</p>
Onset & duration	Within minutes
Special considerations	Face mask may be preferred for those having difficulty breathing with the mouthpiece
General precautions	<p>Caution in patients w/ ACS, dysrhythmias, symptomatic tachycardia, diabetes, HTN, or seizures; or in active labor.</p> <p>Note: DO NOT wait at scene to determine patient response. Begin the neb treatment and transport as soon as possible.</p>
Contraindications	<ul style="list-style-type: none">- Hypersensitivity
Side effects	<ul style="list-style-type: none">- Anxiety, tremors, nervousness- Tachycardia, palpitations- ↑ BP- Dizziness- Angina- Headache, vomiting
Use in pregnant women	Considered relatively safe for use during pregnancy. Management of asthma in pregnant patients should be the same as in non-pregnant patients, as poor oxygenation due to uncontrolled asthma represents a greater danger to the fetus than any potential harm associated with the drugs used to treat the disease.

IPRATROPIUM BROMIDE (Atrovent)

Classifications	<ul style="list-style-type: none">➤ Anticholinergic➤ Bronchodilator
Actions	Cholinergic antagonist (parasympathetic blocker) of acetylcholine at the cholinergic receptors. It suppresses the increase of cyclic guanosine monophosphate (cyclic GMP) levels due to the interaction of acetylcholine with the muscarinic receptor on bronchial smooth muscle, thus blocking the bronchoconstrictor action of vagal impulses. This inhibition of vagal tone produces dilation of the large central airways and small airways.
Indications	<ul style="list-style-type: none">➤ Bronchospasm associated with COPD, asthma, or moderate/severe allergic reactions in adult patients. General note: Ipratropium is useful primarily in the long-term treatment of chronic obstructive pulmonary disease, including chronic bronchitis and emphysema. Drug therapy will not reverse, slow, or prevent progressive decline in lung function, but can relieve symptoms. It is generally not recommended for the long-term treatment of asthma. It may offer additive benefit to inhaled beta-2 agonists in moderate to severe acute asthma attacks, particularly in patients with obstructive airway disease. It may also be an alternative for patients who cannot tolerate inhaled beta-2 agonists.➤ Considered the treatment of choice for beta-blocker induced bronchospasm. Contact medical control for order.
Dose & route	0.5 mg (500 mcg) in 2.5 mL NS added to 1 st albuterol dose/HHN.
Onset & duration	<p>Ipratropium is poorly absorbed into the systemic circulation following inhalation and does not readily cross the blood-brain barrier and does not cause the CNS effects observed with atropine or beta-2 agonists. However, it has a slower onset of action.</p> <p>Initial response: Asthma: 3 to 30 minutes Peak response: Asthma: 90 to 180 minutes</p>
Special considerations	<ul style="list-style-type: none">➤ Nebulizer mouthpiece preferred over face mask to avoid contact w/ eyes.➤ Unit doses are stored in foil pouches to protect from light.
General precautions	<ul style="list-style-type: none">➤ Bladder neck obstruction➤ Glaucoma, narrow angle➤ Prostate hypertrophy
Contraindications	<ul style="list-style-type: none">➤ Peds patients < 12 yr (Not FDA approved for children < 12, but commonly used. Consult medical control.)➤ Hypersensitivity to atropine or ipratropium products
Side effects	<p>Ipratropium's toxic profile is more acceptable than that of atropine or the beta 2 adrenergic agents. It is relatively free of side effects even in large doses.</p> <ul style="list-style-type: none">➤ GI: Dry mouth (most common), abnormal taste in mouth (bitter), nausea; paralytic ileus (rare).➤ Eyes: Blurred vision, dilated pupil (mist leak exposing eyes)➤ Immunologic: Hypersensitivity reaction, angioedema, bronchospasm, urticaria, anaphylaxis, oropharyngeal edema (rare)➤ Cardiovascular effects are minimal as the drug is absorbed poorly following inhalation. Should have no change in heart rate or ECG; should not have a significant effect on cardiac vagal tone.
Use in pregnant women	Considered relatively safe for use during pregnancy. Management of asthma in pregnant patients should be the same as in non-pregnant patients, as poor oxygenation due to uncontrolled asthma represents a greater danger to the fetus than any potential harm associated with the drugs used to treat the disease.

EPINEPHRINE (Adrenalin) for asthma

Classification:	Endogenous hormone (catecholamine)
Actions:	Nonselective alpha and beta adrenergic agonist; stimulates the release of norepinephrine (sympathomimetic)
Therapeutic	<ul style="list-style-type: none">➤ At low doses (< 0.3 mcg/kg/min) β dominates<ul style="list-style-type: none">- Bronchodilator- \uparrow HR (+ chronotropic) (\uparrow rate)- \uparrow CO (+ inotropic) (\uparrow contraction force)- \uparrow AV conduction (+ dromotropic)This increases myocardial work and \downarrow subendocardial perfusion.➤ At higher doses (IV), alpha effects override beta₂ effects
Indications:	<ul style="list-style-type: none">➤ Moderate and severe systemic allergic reactions➤ Asthma attack in severe distress➤ Very severe unresponsive bradycardia (used with extreme caution)
Dose & route:	<p>Asthma: 1:1,000 0.3 mg IM</p> <ul style="list-style-type: none">▪ Caution: P > 100, CVD/HTN; on beta blockers, digoxin, or MAO inhibitors; pregnant; or experiencing significant side effects to albuterol▪ Begin transport as soon as Epi is given. Do not wait for a response.▪ May repeat Epi X 1 in 10 min if minimal response.
Side effects:	<ul style="list-style-type: none">➤ CNS: Headache, dizziness, tremors, restlessness, anxiety➤ CV: \uparrow HR, tachyarrhythmias, high dose may produce vasoconstriction, may compromise perfusion; HTN, angina, \uparrow myocardial O₂ consumption; use with caution in patients with HF can cause worsened ischemia, dysrhythmias.➤ GI: Nausea, vomiting➤ Skin: Pallor
Precautions:	<ul style="list-style-type: none">➤ Epi's therapeutic effects usually begin about 90 seconds after administration. However, because these effects are short lived, it may need to be given again to maintain therapeutic levels.➤ The beta-adrenergic effects may cause or aggravate myocardial ischemia due to the \uparrow work load and O₂ demand that it places on the heart. Adequately ventilate the patient using 100% oxygen to minimize myocardial ischemia.➤ May cause or increase the severity of ventricular ectopic activity. This is of special concern if the patient is taking digitalis, as dig causes the heart to become sensitive to the effects of epinephrine.
Drug interactions:	<ul style="list-style-type: none">➤ Beta blockers may block the therapeutic response to epi and cause pure alpha responses➤ Use with oxytocics/MAO inhibitors can cause severe hypertension➤ Combination with other beta-adrenergic agonists can cause severe arrhythmias

MAGNESIUM SULFATE

Classifications:	<ul style="list-style-type: none">➤ Pharmacologic: Mineral➤ Therapeutic: Mg supplement, antiarrhythmic, anticonvulsant, bronchodilator
Actions:	<p>Magnesium sulfate is one of the major cations of the body responsible for metabolic processes and enzymatic reactions. It is critical in glycolysis and assists all enzymes involved in phosphate transfer reactions that use ATP. It is essential to maintain normal intracellular electrolyte composition through its membrane stabilizing role via the Na/K ATPase pump. It is one of the electrolytes responsible for blocking neuromuscular transmission and maintaining muscular excitability. Mg decreases calcium uptake (Mg and Ca live in reciprocal environments) and potassium outflow through myocardial cells. Hypomagnesemia can cause torsades de pointes, symptoms of cardiac insufficiency, and sudden cardiac death. Hypomagnesemia can impede the replenishment of intracellular potassium leaving an irritable cell and precipitate refractory VF.</p>
Therapeutic benefit:	<p>Magnesium resolves magnesium deficient states which are associated with cardiac dysrhythmias and sudden cardiac death. <i>Similar action to calcium channel blockers</i> in that it causes vasodilation and bronchodilation.</p>
Indications:	<ul style="list-style-type: none">➤ Severe asthma attack➤ Torsades de pointes (polymorphic VT with long baseline QT interval)➤ Dysrhythmias suspected to be caused by magnesium deficiency➤ Preeclampsia and eclampsia
Dose & route:	<p>EMS dose: Dilute 2 g magnesium with 16 mL NS and administer slowly over 5 min (no more than 1 g/min). Observe continually during infusion.</p>
Precautions:	<ul style="list-style-type: none">➤ Use caution in administering to patients with renal dysfunction as they are at risk for Mg toxicity➤ Mg may precipitate myasthenic crisis by decreasing patient's sensitivity to acetylcholine
Side effects:	<ul style="list-style-type: none">➤ CNS: Drowsiness; confusion, dizziness, diplopia, slurred speech. Hypermagnesemia depresses the CNS, causes decreased reflexes and blocks peripheral neuromuscular impulse transmission by decreasing the amount of available acetylcholine. This may result in flaccid paralysis.➤ CV: Mild bradycardia, dysrhythmias, hypotension, 3° AVB➤ Resp: Depression/paralysis➤ GI: Diarrhea➤ MS: Weakness➤ Skin: Flushing, sweating, rash➤ Metabolic: Hypothermia <p>At the hospital: To reverse these effects, ventilate the patient using 15 L oxygen and administer an IV bolus of 10% calcium gluconate at 5-10 mEq (10-20 ml).</p>
Contraindications:	<ul style="list-style-type: none">➤ Hypocalcemia; persistent hypertension➤ Heart block, myocardial damage, shock
Drug interactions:	<ul style="list-style-type: none">➤ Cardiac conduction changes may occur if MgSO₄ is administered with cardiac glycosides (digitalis)➤ Don't combine with bicarbonates, calcium, dobutamine, phosphates, salicylates, heavy metals, or other neuromuscular blocking agents

1. Asthma is caused by chronic inflammation of the airways, resulting in airway hyperreactivity.
True / False
2. List two environmental asthma triggers.

3. List two non-allergen triggers of asthma

4. What are the three pathophysiologic abnormalities that occur in asthma? (all start with an S...)

5. In asthma, the lungs become _____.(Hyperinflated / atelectatic)
6. Name one other condition besides asthma that may present with wheezing which the EMT-P must rule out before treating a patient for asthma?

7. Minimal wheezing, decreased breath sounds, inability to speak full sentences, inability to lie flat, and altered consciousness are signs of a _____ asthma attack.
(Mild / Moderate / Severe)
8. In a severe acute asthma attack, which breath sounds are the most alarming?
 - A. Inspiratory wheezes
 - B. Expiratory wheezes
 - C. Both inspiratory and expiratory wheezes
 - D. Diminished or absent breath sounds
9. What signs indicate the presence of air trapping in an asthma patient?
10. What is peak flow monitoring?
11. A patient whose peak flow reading is 60% of their best predicted would be in their _____ zone.
 - A. Red
 - B. Yellow
 - C. Green
 - D. White
12. A patient whose room air pulse oximetry reading is 89% should be classified as having a _____ attack.
 - A. Mild
 - B. Moderate
 - C. Severe

13. Hypoxemia with cyanosis is seen in the _____ stage of asthma. (Early / Late)
14. If a known patient with asthma presents with severe ventilatory distress, exercise intolerance, is speaking in word clusters and appears exhausted, what must an EMT-P anticipate and prepare?
-
15. How should oxygen be delivered to a patient with asthma who presents with spontaneous ventilations and a RA SpO₂ reading of 90% while preparing to give medication? (NC / NRM)
16. Why can high airway pressures/high volumes be harmful when assisting ventilations in an asthma patient?
17. Which of these should be given first to a 15 y/o with a mild asthma attack who has bilateral wheezing, an adequate SpO₂, and no exercise intolerance?
- A. Albuterol & ipratropium via MDI or HHN
 - B. Epinephrine IM
 - C. Magnesium sulfate IVP
 - D. Benadryl IVP
18. The majority of asthma patients use their inhalers correctly. (True / False)
- What device should be added to aid in better drug delivery when patients are using a metered dose inhaler?
19. What is the classification of albuterol?
- What are the anticipated side effects of albuterol?
- . How much of this drug may be given on the scene?
- . How quickly should the patient start to feel some relief?
20. What is the classification of ipratropium?
- Why does it aid in bronchodilation?
- What is the dose of ipratropium?
- Can it be repeated in the field?

21. Which drug should be considered first for a 30 y/o patient with a severe asthma attack who has bilaterally diminished breath sounds and signs of dehydration and exhaustion?
- What specific receptors does it activate?
- What is the intended action of this drug?
- What is the dose and route for administration?
- Why is the IVP route of this drug contraindicated in a patient with asthma?
- What side effects should a paramedic warn the patient about and anticipate?
- How quickly should the patient start to experience relief?
22. If an asthma patient is in severe distress that is unresponsive to initial drug administration, what electrolyte drug is indicated?
- How does this drug cause bronchodilation?
- What is the dose, route and timing when giving this drug?
- What are the most common side effects of this drug?
- What side effect should be anticipated if this drug was pushed too fast?
23. What type of medication should be taken by the patients with moderate and severe persistent asthma to control their disease? Short-acting Beta-2 stimulants / Steroids
- List three examples of this class of drugs that patients may be taking:
-
-
-
24. List one example of a mast cell inhibitor that patients may be taking to get them through the night without wheezing.

25. Which medication classification for long-term asthma control blocks the release of inflammatory mediators and is taken orally? (methylxanthines / leukotriene blockers)

List two of these agents:

26. What is the main cause of death in patients with asthma? (VF / hypoxia)

