

Northwest Community EMS System
Paramedic Education Program
ALLERGIC REACTIONS/ANAPHYLAXIS
Connie J. Mattera, M.S., R.N., EMT-P

Reading assignments

Text-Vol.1 pp. 235, 1272-1276

SOP: Allergic Reactions/ Anaphylactic Shock

Assumed knowledge:

Drugs: Epinephrine 1:1,000, 1:10,000; albuterol, ipratropium, dopamine, glucagon

KNOWLEDGE OBJECTIVES

Upon reading the assigned text assignments and completion of the class and homework questions, each participant will independently do the following with at least an 80% degree of accuracy and no critical errors:

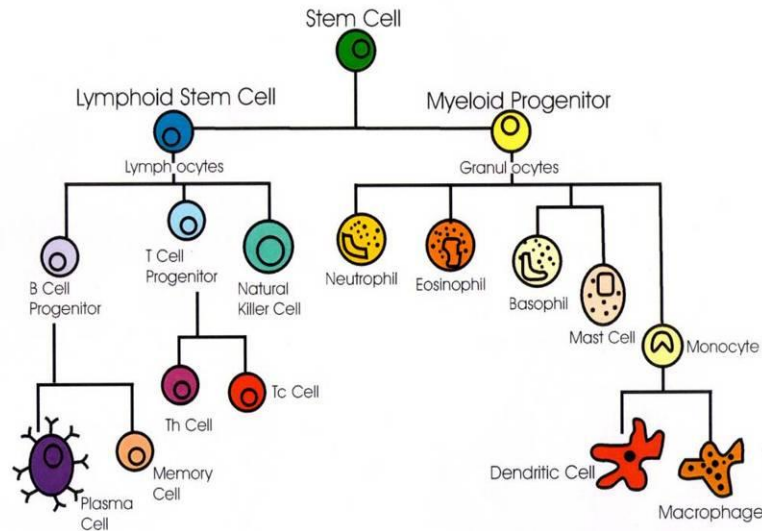
1. Define allergic reaction.
2. Describe the incidence, morbidity and mortality of allergic reactions and anaphylaxis.
3. Identify risk factors that predispose a patient to anaphylaxis.
4. Explain the physiology of the immune system following exposure to an allergen including activation of histamine receptors and the formation of antibodies.
5. Discuss the pathophysiology of allergic reactions and anaphylaxis.
6. Describe the common modes by which allergens enter the body.
7. Compare and contrast natural and acquired and active vs. passive immunity.
8. Identify antigens most frequently associated with anaphylaxis.
9. Differentiate the clinical presentation and severity of risk for a mild, moderate and severe allergic reaction with an emphasis on recognizing an anaphylactic reaction.
10. Integrate the pathophysiologic principles of anaphylaxis with treatment priorities.
11. Sequence care per SOP for patients with mild, moderate and severe allergic reactions.

NWC EMSS Paramedic Education Program
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I. **Immune system**

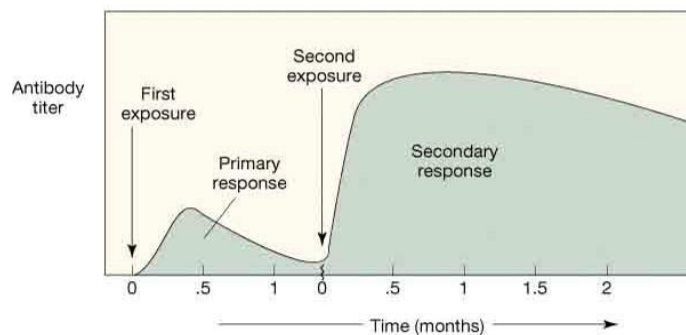
- A. Principal body system involved in allergic reactions. Others include the cutaneous, cardiovascular, respiratory, nervous, and gastrointestinal systems.
- B. The immune system defends against foreign substances, including antigens (foreign proteins) and combats infection.
- C. **Components:** Blood, bone marrow, lymphatic system

Cells of the Immune System



1.
 - D. **Immune response:** Series of events that occur following activation by an invading **antigen** (foreign protein) or **pathogen** (disease-producing agent or invading substance). The goal is to destroy or inactivate the pathogens, abnormal cells, or foreign molecules, such as **toxins** (poisonous chemical secreted by bacteria or released following destruction of the bacteria).
- II. The body does this through two mechanisms: **cellular and humoral immunity**
- A. **Cellular immunity mechanisms:** Involves a direct attack on the foreign substance by specialized cells in the immune system. These cells physically engulf and destroy the invaders. The primary immune system cells are the **T lymphocytes** that originate in the thymus gland (located in the upper chest) and lymphokines. T cells are primarily responsible for fighting infections of biological agents including TB, many viral infections, and most fungal infections.
 - B. **Humoral or antibody-mediated immunity mechanisms**
 1. More complicated. This is a chemical attack on the invaders.
 2. **Four distinct stages lead to classic reactions**
 - a. **Antigen** is introduced into the body by ingestion, injection, inhalation, etc.
 - (1) Defined as anything capable, under appropriate conditions, of inducing a specific immune response
 - (2) Antibody (AB) generator molecules
 - (3) Proteins, nucleoproteins, polysaccharides and some glycolipids
 - (4) Large molecules: size usually > 10,000 daltons
 - b. **Antibodies** are produced in response to previous infection, vaccination, transfer from mother to fetus in utero, or without known antigenic stimulation as a result of an accidental exposure.

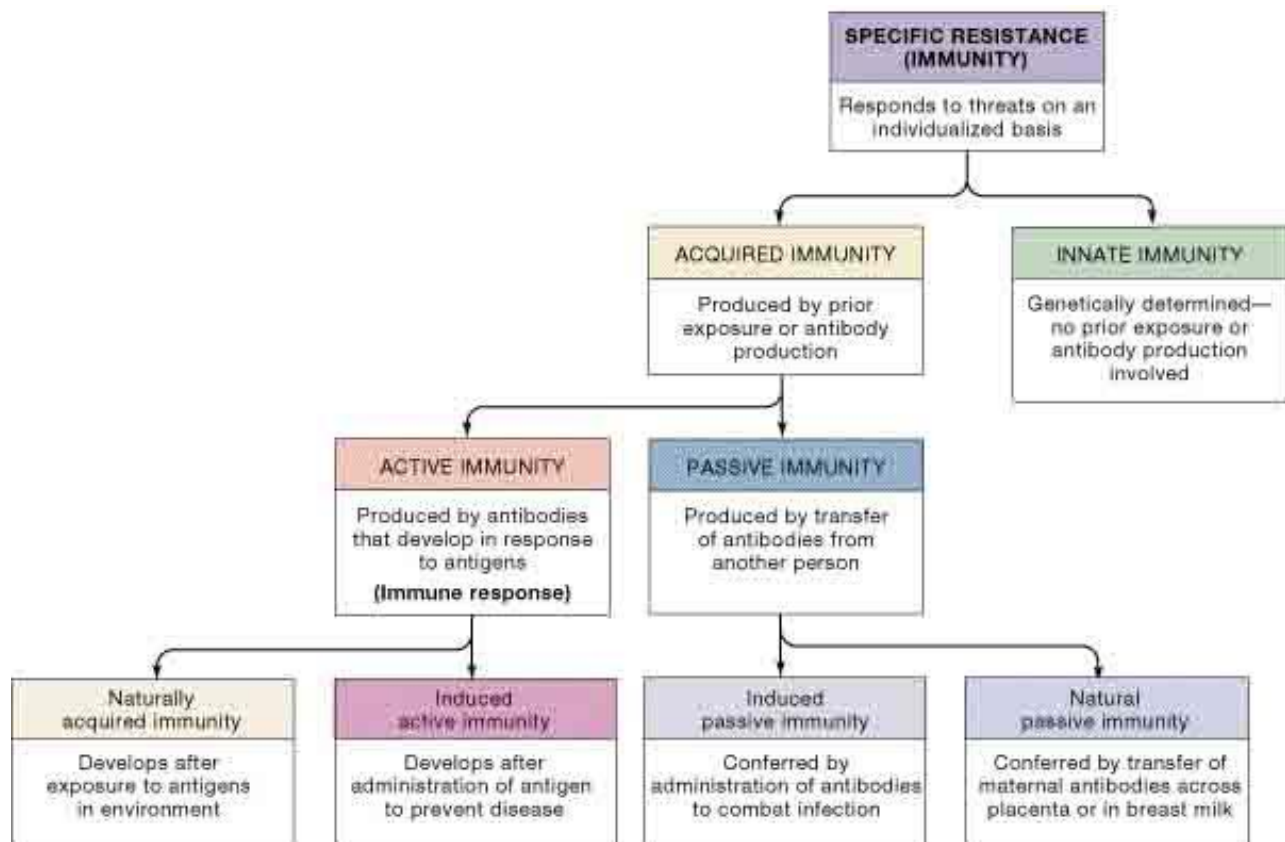
- (1) Antibodies are also called **immunoglobins**
 - (2) Special proteins manufactured by **B lymphocytes**.
 - (3) **Antibody diversity**
 - (a) A human makes 10,000,000 different kinds of antibodies
 - (b) Each AB is composed of identical light and heavy chains
 - (c) Each B cell makes 1 kind of AB
 - (4) **Five classes of human antibodies (immunoglobins)**
 - (a) IgA: Present in the mucous membranes
 - (b) IgD: Present in the lowest concentration
 - (c) **IgE: Contributes to allergic & anaphylactic responses**
 - (d) IgG: Has "memory" and recognizes a repeat invasion
 - (e) IgM: Responds immediately
 - (5) **Antibody functions**
 - (a) Attach to invading substance to facilitate removal of that substance from the body.
 - (b) Neutralization
 - (c) Agglutination and precipitation
 - (d) Activation of complement
 - (e) Attraction of phagocytes
 - (f) Enhancement of phagocytes
 - (g) Stimulation of inflammation
 - (6) They either circulate on **basophils** or **mast cells** or are fixed within target organs.
- c. **Sensitization phase:** If the body has never been exposed to a particular antigen, the response is different than if it has been previously exposed. The initial exposure to an antigen is called **sensitization** or the **primary response**.
- (1) Following first time exposures to an antigen, several days are required before both the cellular and humoral components of the immune system respond.
 - (2) IgG and IgM (generalized antibodies) are first released to help fight the antigen.
 - (3) At the same time, **antibodies specific to the antigen are formed**. They develop a *memory* for the antigen.
- d. **Secondary response:** If the body is exposed to that antigen again, it will respond much more quickly with a much stronger secondary response.

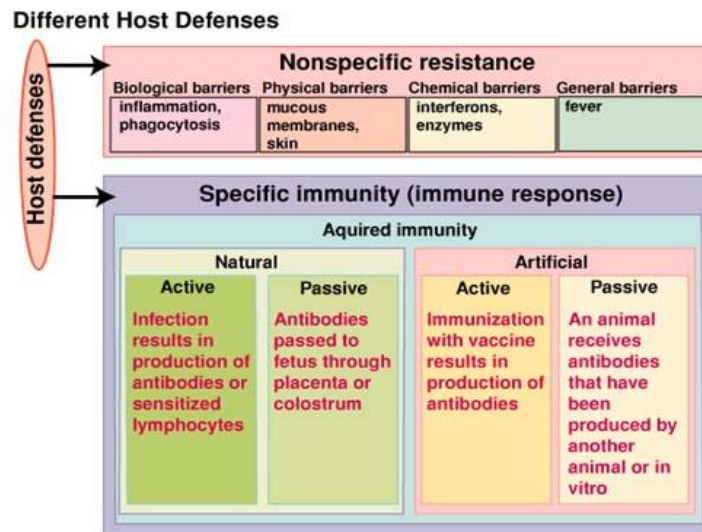


C. **Types of immunity**

1. **Natural** (innate) immunity is genetically predetermined. It is present at birth and has no relation to previous exposures to a particular antigen. Everyone is born with some innate immunity.

2. **Acquired:** Develops over time and results from exposure to antigens and production of specific antibodies. See process explained above. Subsequent exposures to that antigen will have an immune response.
- a. **Naturally acquired:** Begins to develop after birth and is continually enhanced by exposure to new pathogens and antigens throughout life. Example: life-long immunity to chicken pox after having the disease.
 - b. **Induced active immunity (*Artificially acquired immunity*):** Designed to provide protection from exposure to an antigen at some time in the future. Achieved through **vaccination** where an antigen is injected into the body to generate an immune response. This results in the creation of antibodies specific for that antigen and provides protection against future infection. Ex: tetanus, diphtheria, pertussis, Hepatitis B, measles, polio vaccines. Re-emerging: smallpox vaccine.
 - c. **Active:** Occurs following exposure to an antigen and results in the production of antibodies specific for the antigen. It takes some time for these antibodies to develop and provide protection. (See above)
 - d. **Passive:** Administration of the antibodies to provide protection until active immunity can take place.
 - (1) **Natural passive immunity:** Antibodies cross the placenta from mother to fetus to provide protection against embryonic or fetal infections.
 - (2) **Induced (artificial) passive immunity:** Administration of antibodies. Examples: Tetanus immune globulin (Hypertet) or hepatitis-B immunity globulin (H-BIG).





III. Allergies and hypersensitivity reactions

- A. Some persons become **hypersensitive** to a particular antigen. *Hypersensitivity* is an unexpected, exaggerated, or pathologic immune system response. Rather than harming the foreign material, the body either attacks its own cells or mounts such a large response that it causes harm (Aehlert, 988). Often used interchangeably with the term, **allergy**. An antigen that causes production of IgE antibodies is an **allergen**.

B. Type I: Immediate hypersensitivity

1. Epidemiology

- Most familiar; allergic or atopic reaction (predisposition to allergic responses)
- Caused by contact with an antigen (allergen) against which the host has a large number of existing IgE antibodies. These patients have **elevated IgE levels** (10-100 times normal)
- 20%-30% of population has a type I hypersensitivity to common environmental substances
- Genetic component – inherited sensitivity from parents
- Rapid, immediate reaction as well as a late phase component
- Initiation time: 2-30 minutes
- Symptoms depend on site of contact

2. Examples IgE mediated hypersensitivity (Classic - first isolated in 1966)

- Inhaled allergens: Hayfever (allergic rhinitis), pollen, rag weed
- Hymenoptera venom:** Honeybees (*Apoidea*), bumblebees, wasps, hornets (*Vespidae*), yellow jackets, imported fire ants (*Formicoidea*), and others. Prevalence of sensitivity 4%. 50-100 deaths/year.
- Drug allergies:** Most common culprits

- (1) **Antibiotics:** Risk of death from anaphylaxis secondary to drugs is extremely low; however, all drugs should be considered as having this potential.

Penicillin was the first drug implicated in fatal anaphylaxis in 1949 and it has remained the leading cause of anaphylactic death. Other antibiotics of concern are synthetic analogues, cephalosporins, tetracyclines, streptomycin, erythromycin, nitrofurantoin; resulting in 400-800 deaths/year.

- (2) Chemotherapeutic agents
- (3) **Anesthetics** "caine" agents: Important differences between the amides (lidocaine) and the esters (cocaine, procaine, and tetracaine). Allergic reactions to local anesthetics belonging to the amide family (lidocaine) are extremely rare if they occur at all. In patients who have reacted, preservatives used with the lidocaine have been implicated as the true antigen. Cardiac lidocaine does not contain preservatives.
- (4) Amides (lidocaine) – rare
- (5) Esters (cocaine, procaine, tetracaine)
- (6) Opioids – histamine releasers
- (7) Streptokinase

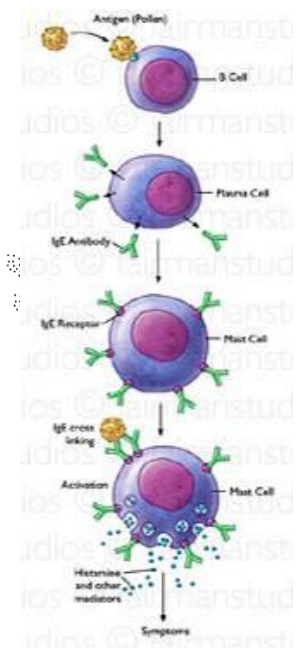
d. **Food allergies**

- (1) 35%-55% anaphylaxis caused by food allergies
- (2) 6%-8% of children have food allergies
- (3) 3%-4% of adults have food allergies
- (4) Incidence increasing
 - (a) 150 deaths/year
 - (b) Usually caused by a known allergy
- (5) Accidental food exposures are common and unpredictable
- (6) Onset of S&S usually occur w/in minutes but may be delayed several hours. Most reactions to foods are actually reactions to substances present within the food.
- (7) Symptoms may be isolated to areas around the mouth, lips and digestive tract or may involve other areas of the body
- (8) Children & adults (usually not outgrown)
 - (a) Peanuts, tree nuts
 - (b) Seafood, shellfish, fish
 - (c) Bisulfites, MSG
 - (d) Chocolate
- (9) Additional triggers in children (commonly outgrown) if food is eliminated from diet for 1-2 years
 - (a) Cow's milk
 - (b) Eggs
 - (c) Soy products
 - (d) Wheat
- (10) Bronchospasm & asphyxia most frequent mechanisms; many exhibit biphasic reaction

e. **Foreign proteins** (hormones, seminal fluid, vaccines)

3. **Mechanism of Type I allergic reaction**

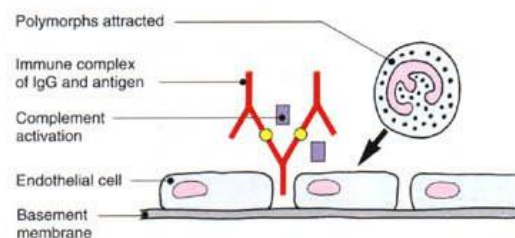
- a. Re-exposure to an allergen with previous sensitivity causes antigen cross-bridging on a pair of IgE antibodies affixed to a mast cell which activates the cell. The blood counterpart to the MAST cell is the **basophil or eosinophil**. Blood counts show eosinophilia in allergy and anaphylaxis.
- b. Mast cells are found in all organs, especially around nerves, lymphatics, and blood vessels. They are most concentrated in the skin, respiratory tract, GI tract, and cardiovascular system - target organs of hypersensitivity reactions.



Important in cellular defense mechanisms including blood coagulation following injury or infection.

4. When the allergen binds to MAST cells or eosinophils, they break down or *degranulate* releasing **histamine**, heparin, bradykinin, leukotrienes, and thromboxanes from granules within the cells. Because of this feature, MAST cells and basophils are called *granulocytes*. This release produces the clinical features of an allergic reaction which may range from mild to severe.
5. **Histamine**: Most important chemical released in an allergic reaction.
 - a. **3 histamine receptors**
 - (1) **H₁**: Responsible for coronary artery vasoconstriction, bronchoconstriction, **vasodilation** (flushing), **increased vascular permeability resulting in fluid shifts** (hypotension) and **tissue edema**, and increased secretion of airway and nasal mucus, intestinal smooth muscle contraction (diarrhea, abdominal pain and cramping) and prostaglandin production. Decreases AV conduction time, so can lead to dysrhythmias. Is also responsible for the pruritis (**itching**), pain and burning seen in many hypersensitivity reactions as well as airway Vagal nerve stimulation. Positively, stimulation of H₁ receptors help to modulate the reaction. Further histamine release from H₁ receptors is **blocked by diphenhydramine** (Benadryl).
 - (2) **H₂**: Stimulates ventricular and atrial contraction (+ inotropic effect), increases HR (+ chronotropic effect), coronary and peripheral vasodilation, **gastric acid secretion**, lower airway mucus production, inhibits basophil and neutrophil activity, suppresses T-cell stimulation, and ↑ cyclic AMP in the cells. Causes nausea, vomiting,. (Reversed w/ H₂ blockers like Tagamet)
 - (3) **H₃**: Inhibits central and peripheral NS neurotransmitter release and may inhibit histamine formation.
 - b. **Histamine takes only 2½ minutes to appear** in the blood, reaches peak levels in 5 minutes, and returns to baseline in 15-30 minutes. The actual goal of histamine release is to limit the body's exposure to the allergen. Bronchoconstriction decreases the amount that can be inhaled through the respiratory tract. Increased gastric acid helps destroy an ingested antigen. Increased intestinal motility moves it through the GI tract faster minimizing absorption. Vasodilation and capillary permeability help to remove allergens from the circulation where they have the most potential for doing the most harm (Bledsoe, 351).
6. **Heparin**
 - a. Produced by the MAST cells of the liver and by basophils.
 - b. **Inhibits coagulation** by preventing liberation of thromboplastin from blood platelets.
7. **Bradykinin** causes ↑ vascular permeability, vasodilation, cough and mucosal edema.
8. **Leukotrienes** cause mucosal edema, mucus secretion, airway inflammation, bronchospasm (coughing, sneezing, wheezing) not inhibited by antihistamines, vasodilation, vascular permeability, and urticaria (hives).
9. **Thromboxanes** cause vascular and airway constriction and platelet and leukocyte aggregation (clumping).
10. **Cumulative result** when there is an exaggerated histamine release is a relative hypovolemia due to **vasodilation** and **peripheral pooling** of blood.

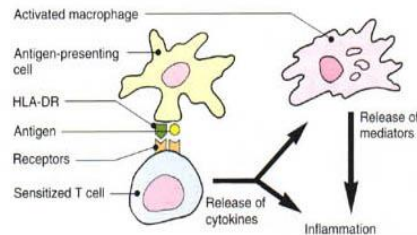
- C. **Direct, non-IgE-mediated drug induced anaphylactoid reaction** that independently stimulates the MAST cell. Mimics anaphylaxis but does not involve IgE antibodies. Thus, may occur with first exposure.
1. Iodinated contrast media (IVP dye), Telepaque (gall bladder dye), sulfobromophthalein (BSP), sodium dehydrocholate (Decholin), Congo red. (May induce Vagal reaction). 1/10,000 chance of fatal reaction or 1000 deaths/year.
 2. Opiates; Mannitol
 3. Antibiotics: vancomycin, aminoglycosides
 4. **ACE inhibitors** (alter bradykinin metabolism). Angioedema reaction first reported in 1984. Significant potential for airway compromise. Angioedema due to tissue accumulation of bradykinin.
 5. Cannot be distinguished in the field from IgE mediated – treat the same.
- D. **Type II: Cytotoxic hypersensitivity**
1. Antigens on target skin cells or structures attract specific IgM or IgG AB. Target cells usually foreign to the host.
 2. Destroys cells by recruiting killer T cells & initiating the **complement cascade creating a membrane-attack complex**. Cell membranes are damaged or destroyed.
 3. Major reaction **may occur with first or subsequent exposures**
 - a. Blood products (transfusions): ABO incompatibility reaction
 - b. Hemolytic disease of newborn
 - c. Acute rejection of a transplanted organ
 - d. Dialysis membranes
- E. **Type III: Immune complex hypersensitivity**
1. Antigen and antibodies form **immune complexes** of IgG AB and soluble antigens when not promptly removed by the reticuloendothelial system (Aehlert, 991).
 2. Deposit in tissues and cause an inflammatory response. Wherever deposited, activate the complement cascade with results similar to cytotoxic hypersensitivity.
 3. Much in common with Type I except AB involved is IgG and is not prebound to mast cells



4. Examples: Circulating immune complexes with certain autoimmune diseases
 - a. Serum sickness: Seen historically when foreign serum was injected. If not cleared rapidly is bound with antibodies to form immune complexes
 - b. Now seen with certain drugs (penicillin)
 - c. Post-infection complications of arthritis and glomerulonephritis, systemic lupus erythematosus
5. Initiation time: 2-8 hours

F. Type IV Delayed hypersensitivity reaction

1. **Not caused by antibodies. Cell-mediated hypersensitivity by CD4 lymphocytes** such as those targeted by HIV (Aehlert, 991).
2. Only class triggered by antigen-specific memory **TH1 cells** that release cytokines – involves IgM AB and probably complement
3. Cytokines recruit and activate macrophages, other T cells, and to lesser effect, neutrophils



- a. Initiation time: Time frame for lymphocyte response is hour to days after contact with the antigen.

b. Examples

(1) Contact dermatitis

- (a) Poison oak or ivy
- (b) Chemical skin allergies (nickel), dyes, fragrances, cosmetics, soaps

(c) Latex-induced

- (i) Proteins in natural rubber latex
- (ii) Component of ~40,000 commonly used items
 - (a) Rubber bands
 - (b) Elastic (undergarments)
 - (c) Hospital/dental equipment
- (iii) Latex-dipped products that stretch are biggest culprits: Balloons, gloves, bandages, hot water bottles
- (iv) 1%-6% of U.S. pop. (16 million) affected
- (v) 8%-17% incidence in healthcare workers
- (vi) Repeated exposure leads to a higher risk
- (vii) Incidence increased after mid 1980s d/t use of latex gloves especially powdered gloves

(viii) Reactions to latex

- (a) Irritant contact dermatitis: Dry, itchy, irritated hands
- (b) Allergic contact dermatitis: delayed hypersensitivity
- (c) Latex allergy S&S: Hives, itching, sneezing, rhinitis, dyspnea, cough, wheezing. Greatest risk with mucosal contact

(d) Prevention

- (i) Use latex-free products and equipment
- (ii) Wear Medic-alert® bracelet

(iii) Awareness of cross-sensitivity with foods:

- (a) Banana
- (b) Avocado
- (c) Chestnuts
- (d) Kiwi
- (e) Stone fruit

(2) **TB skin test reaction:** purified protein derivative (tuberculin) is injected intradermally. Local reaction may occur if patient has been infected in the past with TB. Greater reaction indicated prior sensitization.

(3) **Chronic rejection of a transplanted organ**

G. **Inhibition of the arachidonic acid pathway**

1. **Aspirin:** Inhibits cyclooxygenase production, thereby ↑ the number of leukotrienes which **causes either a local or generalized reaction**. Five percent of asthmatics have an exacerbation and other adverse reaction after taking ASA.
2. **Non-steroidal anti-inflammatory drugs (NSAIDs):** Advil etc.

H. A **combined reaction** associated with an alternate complement pathway in which the C₁ complement component fixes the antigen to the antibody complex to the MAST cell.

I. **Idiopathic:** Rashes may be caused by heat, cold, vibration, pressure, light, or exercise. **Exercise** is increasingly being recognized as a precipitator of anaphylaxis. Dedicated athletes are usually involved and symptoms often abate if the patient rests as soon as he notes pruritus (itching). Other causes need to be ruled out before this etiology is considered.

J. **Risk of allergic reactions/anaphylaxis**

1. Family history is not considered a reliable predictor. However, the same clinical pattern of allergic response does tend to recur in the same individual.
2. **Severity of a reaction is affected by**
 - a. the quantity of the antigen;
 - b. the route and rapidity of absorption: Most risky is parenteral as the allergen is distributed throughout the body, the least risk is topical, and oral ingestion is somewhere in between;
 - c. a PMH of asthma or cardiac disease; and/or
 - d. patients taking beta blocker drugs.

IV. **Patient assessment**

A. **Maintain a high index of suspicion**

1. May involve only one body system or multiple systems.
2. Can have an abrupt onset with variable course ranging from mild to fatal.
3. Most reactions will be evident within 30-60 seconds to minutes following exposure although a delay of several hours may occur in rare situations.
4. In general, the sooner the syndrome manifests, the more severe the reaction. Fatal outcomes usually occur within 30 minutes of antigen exposure but some patients have developed symptoms up to 2 hours after exposure.
5. Some patients may need definitive treatment before a thorough exam has been completed. Drug interventions may be needed on the basis of rapid initial assessments. You may not have time to identify the etiologic agent to substantiate your paramedic impression.

B. Scene size up; make sure it is safe for you to enter - rampaging bees???

C. Primary assessment

1. General level of consciousness
2. Airway; ability to speak
3. Breathing; gas exchange
 - a. General RR (tachypnea), effort (labored, use of accessory muscles), prolonged expiration
 - b. Skin color temperature, moisture
 - c. SpO₂: O₂ sat > 90% indicates that the PaO₂ is > 60. Use to follow general trends. Patient may be or become hypoxic.
 - d. Monitor capnography if available. As anaphylaxis progresses CO₂ levels rise due to respiratory and metabolic acidosis.

4. Signs of airway/ventilatory impairment

a. Upper airway

(1) Angioneurotic edema, also known as **angioedema**

(a) Involves the deep dermis, sub-q or submucosal tissues and can be either hereditary or acquired.

(b) Well-demarcated, non-pitting, non-pruritic edema commonly found in the periorbital, oral, lingual (tongue), and pharyngeal areas, although the hands, feet, penis and scrotum may also be involved. Does not itch.



(c) May persist for days. Classified as acute if lasts less than 6 weeks. Chronic if it lasts longer.

(2) Hypersalivation

(3) Laryngeal edema (hoarseness), change in voice (hoarseness) may progress to stridor

(4) ↑ secretions, nasal congestion, rhinitis, sneezing

b. Lower airways

(1) Dyspnea; sensation of tightness in chest and throat

(2) Coughing

(3) Retractions

(4) Wheezing due to intense bronchial swelling and spasm. May be evident without a stethoscope. No wheezing or diminished breath sounds may mean no breathing! Bronchospasm and laryngeal edema may induce swift respiratory arrest.

(5) Tachypnea

5. Circulatory status; ECG

a. Assess: General pulse rate (fast or slow), quality, rhythmicity; ECG

b. **Signs of cardiovascular decompensation/impairment**

- (1) Chest pain
- (2) Tachycardia
- (3) Dysrhythmias: ST, AF, AV and IV conduction delays, transient left bundle branch block (LBBB)
- (4) ↓ BP due to massive peripheral vasodilation and 3rd space losses due to ↑ permeability of capillaries **resulting in marked loss of plasma from the circulation.**
- (5) Acute coronary syndromes
- (6) Reversible cardiomyopathy (disease of heart muscular wall)
- (7) This **leads to cardiovascular collapse, hypotension (BP ≤ 90),** cardiac dysrhythmias, shock and coma.

D. **Secondary assessment**

1. **Full set of vital signs**

- a. BP: Important to determine stability or instability. Will fall with significant vasodilation and capillary leak.
- b. P: rate, rhythmicity, quality, location (anticipate reflex tachycardia in most patients). HR will fall late in anaphylaxis. Ominous sign.
- c. Respiratory rate, pattern, depth, and effort. Will initially increase, but severe obstruction can reduce rate.

2. **History important if time permits**

- a. OPQRST of chief complaint
- b. **Allergies:** History of allergies to food, medications, plants insect stings environmental triggers, bites or others?
- c. **Medications**
 - (1) Does the patient have an epi-pen prescribed?
 - (2) Have they taken anything already to relieve their symptoms?
 - (3) Has the patient taken any NEW medication?
 - (4) Are they on ace-inhibitors, beta blockers or allergy meds?
- d. **Past history**
 - (1) Have they ever had an allergic reaction in the past?
 - (2) If so, when was their last reaction?
 - (3) What was their response to therapy? Risk of recurrent systemic reactions about 28%.
 - (4) How severe was the last reaction? Have they ever needed to be intubated or admitted to ICU?
 - (5) Do they have any significant illnesses? History of asthma, CVD
- e. **Last oral intake:** When was the last time the patient had anything to eat or drink? What did they consume? How much?
- f. **Event specifics**
 - (1) What was the patient doing prior to the onset of their symptoms?
 - (2) What was the patient exposed to that could have caused a reaction? Any new soaps, foods, household products, etc.
 - (3) What was the route of exposure - injection, ingestion, inhalation, or contact?

3. **Physical exam**

- a. **Neurological** - level of consciousness
 - (1) Anxiety, apprehension, restlessness
 - (2) Altered mental status; confusion, disorientation, decreased level of consciousness
 - (3) Dizziness
 - (4) Hypotension or dysrhythmias may manifest by c/o lightheadedness or syncope
 - (5) Headache
 - (6) Perioral tingling
 - (7) Seizures, syncope, coma
- b. **Cutaneous** (skin - 90%)
 - (1) Warmth, redness (flushing), tingling
 - (2) **Pruritus** (itching): Itching of palms of hands, soles of feet, or back of throat may be an early sign
 - (3) Fine, red, rash appears diffusely involving face, chest, back, and abdomen.
 - (4) **Urticaria**: Histamine release will cause fluid to diffuse from leaky capillaries resulting in a wheal and flare reaction characterized by red, raised bumps that may appear and disappear across the body. Also called hives.
 - (5) Edema
 - (6) Diaphoresis
 - (7) Cyanosis
- c. **Mucus membranes**
 - (1) Edema
 - (2) Burning
 - (3) ↑ secretions (drooling)
 - (4) Rhinorrhea
 - (5) Ocular itching and lacrimation (↑ tearing)
- d. **Gastrointestinal**
 - (1) Bloating, abdominal pain, cramping
 - (2) Hyperactive bowel sounds
 - (3) Nausea, vomiting, diarrhea
 - (4) Difficulty swallowing
 - (5) Loss of bowel control
- e. **Renal**: Urgency, incontinence or decreased urinary output
- f. **Reproductive**: Uterine cramping

V. **Differential diagnosis**

- A. Stridor: Signs of airway obstruction may be caused by FB aspiration, epiglottitis, croup
- B. Wheezing: Asthma/COPD, HF, AMI, pneumonia, FB aspiration
- C. Hypotension: Shock: cardiogenic, hypovolemic, septic
- D. Urticaria: Infection
- E. Skin and/or pulmonary S&S: MSG syndrome, scombroid fish poisoning
- F. In the unconscious patient, vasovagal syncope is the most common differential. Classically, the patient is bradycardic, with hypotension and pallor in vasovagal reactions as opposed to the tachycardia and flush seen in anaphylaxis.

VI. **Clinical syndromes – See SOP p. 11**

A. **Goals of interventions**

1. Eliminate inciting agent if possible
2. Resolve any immediate life-threats
3. Impede further mediator release
4. Inhibit target organ effects
5. Reverse target organ effects
6. Identify the underlying cause to avoid future incidents

B. **IMC special considerations**

1. Ask about history of allergies vs. asthma
2. Apply venous constricting band proximal to bite or injection site if swelling is ↑ rapidly.
3. Attempt to identify and/or remove inciting cause: scrape off honeybee stingers with tongue blade or credit card. **DO NOT SQUEEZE**. Other stinging insects do not leave stingers in skin, but can sting repeatedly.
4. Apply ice/cold pack to bite or injection site for 15-20 minutes to induce vasoconstriction unless contraindicated (snake bite).
5. Do NOT start IV, give meds, or take BP in same extremity as a bite or injection site.
6. Keep extremity dependent - do not elevate

- C. **LOCAL reaction:** Only MAST cells and basophils in the specific area of exposure are involved. **S&S:** A&O X 3, **urticaria** [red, edematous blanching papules (superficial solid raised areas or wheals commonly called hives) involving the superficial dermis (skin) associated with vasodilation] **confined to the site of exposure** or GI distress after food ingestion.. They are associated with intense itching (pruritus) that generally lasts 3-4 hours. Local reactions are also characterized by rhinitis (runny nose), and contact dermatitis.



BP ≥ 90; Massive local reaction results in swelling beyond 2 joints of an extremity.

Treatment: Observe for progression and transport.

- D. **MILD SYSTEMIC REACTION:** MAST cells and basophils all over the body are involved. **S&S** may include: peripheral tingling, warm sensation, fullness in the mouth and throat, nasal congestion, periorbital swelling, rash, itching, tearing of the eyes, and sneezing. **BP ≥ 90; airway and ventilations OK.**

Treatment: diphenhydramine 1 mg/kg (max 50 mg) IM or slow IVP

1. H-1 blocking effects of diphenhydramine bind to histamine receptors and **prevent additional histamine from being released**. It does not eliminate, reverse, or bind histamine already in circulation
2. **Use caution with** alcohol intoxication, drug intoxication, Hx asthma, nursing mothers
3. **SE: Sedation;** drowsiness, blurred vision, ataxia, dry mouth, ↑ HR; ↓ BP; will thicken bronchial secretions due to its drying effects.



- E. **MODERATE SYSTEMIC REACTION** - S&S may include any of the above plus bronchospasm, dyspnea, **wheezing**, edema of airways, larynx, or soft tissues; cough, flushing, N&V, warmth, or anxiety. **BP ≥ 90.**

Treatment:

1. **EPINEPHRINE (1:1000) 0.3 mg (mL) IM** in vastus lateralis muscle
Catecholamine; sympathomimetic; Beta -2 effects promote bronchodilation by reversing bronchospasm, beta 1 effects increase cardiac output through positive inotropic effects, and ↑ cAMP production which stabilizes mast cell membranes and inhibits further mediator release.

 - a. Caution: Consider age, existing conditions; other causes of wheezing; 12 L EG if CVD or + risk factors; apply O₂ to offset ↑ demand; may need to ↓ dose; monitor closely for SE; start IV in case needed
 - b. P > 100, CVD/HTN; on beta blockers (pure alpha response), digoxin, MAO inhibitors; or pregnant
 - c. May repeat in 5-10 min: **DO NOT DELAY TRANSPORT waiting for a response**
 - d. **SE: SNS stimulant:** HA, dizziness, tremors, restlessness, anxiety; tachycardia, palpitations, HTN, vasoconstriction, ↑ myocardial O₂ consumption; *Do not give to patients in HF!* ; can worsen myocardial ischemia & HTN; N / V
2. **Diphenhydramine 50 mg IVP**; if no IV give IM
3. If wheezing: **ALBUTEROL 2.5 mg** (beta-2 stimulant) & **IPRATROPIUM (Atrovent)** (parasympatholytic) **0.5 mg** via HHN or mask. See SOP for drug profiles.
Supplement w/O₂ 6 L/NC if patient is hypoxic & using a HHN.

F. SEVERE systemic reaction / ANAPHYLACTIC SHOCK

1. **Definition:** Severe allergic reaction with systemic involvement (skin, GI, resp, CV) with S&S of **low resistance shock .Life threatening** - *needs immediate interventions*. Can develop within 30-60 sec and cause death within minutes after exposure to a substance that sets off a biochemical chain of events. Shorter the interval between exposure & reaction, the more likely the reaction will be severe.
2. **Incidence**
 - a. Anaphylaxis is underreported and incidence seems to be increasing. Up to 41 million Americans are at risk.
 - b. 1:3000 persons suffers from anaphylaxis to some allergen which accounts for 1 of every 2650 ED visits with 63,000 new cases per year.
 - c. 5% of adults may have a Hx of anaphylaxis.
3. **Etiology:** Caused by contact with a substance to which the person is hypersensitive. The route of exposure is often injection where the allergen is widely distributed throughout the body.
4. **Course of anaphylaxis**
 - a. **Uniphasic course:** Majority of patients - develop S&S of anaphylaxis, recover with appropriate therapy, and remain asymptomatic.
 - b. **Biphasic reaction (20%):** Second wave of S&S may recur 4-8 hours after the initial remission of anaphylaxis. Small percentage rebound at 24-48 hours.
5. **Morbidity factors**
 - a. Estimated 500-1000 deaths/year
 - (1) 70% of deaths due to airway compromise
 - (2) 30% due to circulatory collapse and refractory hypotension.
 - b. 1% risk

- c. Risk factors
 - (1) Failure to give epinephrine immediately
 - (2) Beta blocker, ACEI therapy
 - (3) Hx asthma, cardiac disease
 - (4) Rapid IV allergen: Large doses of IV agents are more likely to result in severe reactions than small doses given by another route

6. **Diagnosing anaphylaxis**

- a. Based on clinical S&S, exposure history
- b. Skin, respiratory, CV S&S most common
- c. Some cases may be difficult to diagnose: vasovagal syncope

Signs/symptoms	Incidence (%)
Urticaria, angioedema	88
Upper airway edema	56
Dyspnea & wheezing	47
Flush	46
Dizziness, syncope, hypotension	33
GI	30
Rhinitis	16
Headache	15
Substernal pain	6
Itch without rash	4.5
Seizure	1.5

7. **S&S:** Above plus intense bronchospasm, decreased/absent breath sounds or diffuses wheezes, laryngeal edema, hoarseness, stridor, severe dyspnea, cyanosis, respiratory failure, cardiovascular collapse, dysrhythmias, and coma. GI edema results in dysphagia, intense abdominal cramping, diarrhea, and vomiting. **BP < 90.**

8. **Treatment: IMC special considerations: Time sensitive patient**

9. **If airway/ventilations severely compromised:**

Consider the need for early intubation (DAI). If intubation is delayed, patients can deteriorate over a brief period of time (0.5 to 3 hours) with development of progressive stridor, severe dysphonia or aphonia (difficulty or inability to speak), laryngeal edema, massive lingual (tongue) swelling, facial and neck swelling, and hypoxemia. At this point both tracheal intubation and cricothyrotomy may be difficult or impossible.



Attempts at tracheal intubation may only further increase laryngeal edema or compromise the airway with bleeding into the oropharynx and narrow glottic opening (AHA 2010). Crics may be impossible because severe swelling will obliterate landmarks. In these desperate circumstances consider the need for the following:

- a. Digital tracheal intubation if the patient is unconscious and non-responsive
- b. King airway if unconscious and no gag reflex
- c. Needle cric followed by surgical cric

10. **IV NS consecutive 200 mL fluid challenges to attain BP of 90** or above. Support of circulation requires rapid volume expansion and administration of vasopressor agents to support BP. Very large volumes of IVF may be needed over short periods of time. Typically 2 to 4 L of NS should be given as rapidly as possible. Use a large bore IV catheter and apply a pressure infuser around the IV bag to hasten fluid delivery.

11. **EPINEPHRINE (1:10,000) titrate in 0.1 mg increments q. 1 min up to 1 mg slow IVP/IO**

- a. **Epinephrine** is the drug of choice for treatment of vasodilation/hypotension and cardiac arrest in these patients. The alpha stimulating effects of epinephrine 1:10,000 slow IVP/IO in 0.1 mg increments promote resolution of angioedema, urticaria, and hypotension by reversing vasodilation. Reassess after each 0.1 mg increment.



- b. **Risk/benefit analysis:** Insufficient initial doses of epi may permit a severe allergic reaction to progress to airway obstruction. Excessive doses can lead to severe hypertension, AMI or ventricular dysrhythmias in the pharmacologically vulnerable patient (i.e. on beta blockers as alpha receptors remain the only sites available for Epi binding), or pathophysiologically vulnerable patient (i.e., elderly or those with CHD)

- c. No IV/IO: **EPI (1:1,000) 1 mg IM**

12. If BP remains < 90: **DOPAMINE IVPB** Start at **10 mcg/kg/min** to activate alpha receptors and trigger vasoconstriction; May increase to a maximum of 20 mcg/kg/min. Titrate to maintain SBP > 90. See SOP for drug profile.

13. If patient is on beta blockers & not responding to Epi & Dopamine: **GLUCAGON 1 mg IVP/IN/IO/IM**. May repeat X1. This agent is short-acting; give 1 to 2 mg every 5 minutes. Nausea, vomiting, and hyperglycemia are common side effects.

14. **Diphenhydramine 50 mg IVP**; if no IV give IM

15. If wheezing: **ALBUTEROL 2.5 mg & IPRATROPIUM (Atrovent) 0.5 mg /HHN**
Continue nebulizer therapy while enroute. May repeat **ALBUTEROL 2.5 mg/HHN**

16. **If cardiac arrest occurs:**

- a. **Prolonged CPR** Cardiac arrest associated with anaphylaxis may respond to longer therapy than usual. Patients with anaphylaxis are often young with healthy hearts and cardiovascular systems, and they may respond to rapid correction of vasodilation and low intravascular volume. Effective CPR may maintain sufficient oxygen delivery until the catastrophic effects of the anaphylactic reaction resolve. (AHA IV-145)

- b. **Aggressive volume expansion.** Start 2nd vascular access line; give IVF as rapidly as possible (up to 8 L) (use pressure infusers if available). Near-fatal anaphylaxis produces profound vasodilation that significantly increases intravascular capacity. Massive IVF replacement is needed. Use at least 2 large-bore IVs with pressure bags to give large volumes (typically between 4 -8 L) of NS as quickly as possible.

- c. **EPINEPHRINE 1:10,000 1 mg IVP/IO every 2 minutes** (high dose)

- d. **Treat dysrhythmias** per appropriate SOP for cardiac arrest.

VII. **Ongoing assessment**

- A. **DANGER! Biphasic Reaction:** Second wave of multi-organ failure 6-8 hours following initial remission seen in 20% of patients. Crashes may come without warning
- B. Monitor mild or moderate reactions for sudden deterioration
- C. Determine effectiveness of drug administration
- D. Reassess ABCs and VS q. 5-15 minutes while unstable

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Patient resources

- Allergy and Asthma Network/Mothers of Asthmatics, Inc. (800-878-4403)
- American Academy of Asthma, Allergy, and Immunology (800-822-2762)
- American College of Allergy, Asthma & Immunology
- American Lung Association (212-315-8700)
- Asthma and Allergy Foundation of America (800-7-ASTHMA)

The Truth About Poison Ivy

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Poison ivy is not in the least contagious. Nor is it caused by any kind of “poison.”

The culprit is an oleoresin known as *urushiol*. This oily resin is contained in the stems, leaves, and flowers of plants from the *Toxicodendron* genus, which includes poison ivy (by far the most common source), poison oak (found almost exclusively west of the Rockies) and poison sumac (found in limited areas of the southeastern United States). Similar reactions can be caused by exposure to other botanicals, including ficus, fern, fig, mango, and Japanese lacquer trees. (Another member of the *Toxicodendron* family is the Rhus tree, found mostly in Australia, where it is notorious for causing rashes.)

A history of recent exposure to the great outdoors can therefore usually be obtained, although exposure can also occur through contact with pets or with the clothing of family members who have been exposed. A live plant is not necessary. Many a victim has acquired poison ivy from clearing brush or handling firewood in the dead of winter. (The antigen can also be acquired through an airborne route, such as from the smoke generated by burning brush—even at a considerable distance.) Repeated exposure to poison ivy over several years' time is required, which explains why children and city-dwelling adults may appear to be immune.

Typically, the blisters begin to appear within 12 to 48 hours of exposure (with some notable exceptions). Much to the consternation of the patient and family, new lesions can continue to manifest for up to two weeks after initial exposure, which is probably why so many people think poison ivy is contagious. The truth is, there is no urushiol in the fluid from the blisters, nor is the antigen “poison” in any way. In short, poison ivy cannot be transmitted from one person to another.

Poison ivy rashes manifest in several different ways. The most common is a type IV delayed hypersensitivity reaction, with pathognomic linear streaks of erythematous patches of edematous, blistery skin. **Figure 1** shows a classic linear lesion on the face of a 69-year-old woman who had cleaned out her fence line a few days previously. This particular patient had a similar rash on her chest.



Figure 2 shows a more atypical form of poison ivy, entirely lacking linear lesions or even blisters. The patient's rash was



widespread, but concentrated on popliteal and antecubital areas and medial thighs. It comprised large sheets of highly erythematous skin, in the centers of which were targetoid reddish blue patches. This is a variant of erythema multiforme, which is by definition a secondary condition usually triggered by bugs (strep, herpes simplex virus) or drugs (sulfa, tetracycline, aspirin, penicillin) but occasionally by an exaggerated reaction to antigens such as poison ivy. In this case, this 185-pound 47-year-old woman and her family acquired poison ivy while picking wildflowers in wet weeds—bringing a rapid end to the family vacation.

The most effective treatment for severe, symptomatic poison ivy is the use of systemic glucocorticoids (eg, prednisone) and/or intramuscular injection of a corticosteroid (eg, triamcinolone). Patient 2's condition was so severe that she couldn't sleep, eat, or even drive a car. (Interestingly enough, she was the only one who sought medical evaluation. The rest of her family was content to clean out the local pharmacy's supply of calamine lotion and diphenhydramine capsules.) Severity of this nature demands serious medication—in this case, a two-week taper of prednisone (from 60 mg down to 20 mg) plus an IM injection of triamcinolone (60 mg).

We made this treatment decision with the knowledge that such doses might produce adverse effects, including an increase in appetite, fluid accumulation, irritability, and sleeplessness. Before prescribing these medications, the potential effects were thoroughly discussed with the patient and a history was

taken to rule out antecedent diabetes, severe hypertension, intercurrent infection, peptic ulcer disease, or bipolar affective disorder, all of which could be worsened by the use of corticosteroids.

So-called “dosepaks” of corticosteroids are far too weak for such a serious condition, and topical medications—even the most powerful—will be of little benefit. I gave Patient 2 one more thing: a prescription for hydroxyzine hydrochloride (25 mg tablets, to be taken at bedtime), both for sedative and antipruritic effects. For milder cases of poison ivy, I often advise no treatment except topical because the one thing that can be depended on is that poison ivy will resolve on its own, eventually.

I also educate patients about the appearance of the poison ivy plant (see **Figure 3**). In my part of the country (Oklahoma), where poison ivy is found in virtually every fence line, every creek bank, and every backyard, most people don’t really know what it looks like. Part of that confusion results from the fact that it can take a number of forms, including a vine, often wrapped around trees, a small tree, or even a root, coursing along the ground. Its distinguishing features include the “leaves of three” in typical shapes (darts, with notches, or “thumbs” along the leaf margins), a shiny surface, and white flowers turning into berries.



TAKE-AWAY LEARNING POINTS

- Poison ivy is predominantly found east of the Rocky Mountains and poison oak almost exclusively to the west of them. Poison sumac is limited to the southeastern United States.
- Poison ivy is also known as a form of phytodermatitis, with other botanical sources such as ficus, fig, fern, mango, rhus, and Japanese lacquer trees.
- Poison ivy can grow in the form of a bush, a vine, or even a small tree. However, it also demonstrates the “leaves of three.”
- Linear blisters on an erythematous base is the classic description of a poison ivy reaction, but it can also present in a more diffuse form that concentrates on the popliteal areas and medial thighs.
- Poison ivy is not “poison.” It is the quintessential contact dermatitis, a type IV delayed hypersensitivity reaction to the oil contained in the plant.
- Poison ivy is not contagious and cannot be spread on the patient’s body or to anyone else.

Homework questions

1. What chemical released from MAST cells in an allergic reaction is responsible for causing bronchoconstriction, vasodilation, and tissue edema?
 - A. Histamine
 - B. Aldosterone
 - C. Angiotensin II
 - D. Thromboxane
2. What is the most common cause of fatal anaphylaxis?
 - A. Tetanus vaccine
 - B. Injected penicillin
 - C. Ingestion of bisulfites
 - D. Bee and wasp stings
3. Which immunity develops over time and results from exposure to an antigen?
 - A. Natural
 - B. Cellular
 - C. Humoral
 - D. Acquired
4. Which of these best describes induced active immunity?
 - A. Is genetically predetermined and is present at birth.
 - B. Results from a direct attack of a foreign substance by specialized cells of the immune system.
 - C. Begins to develop after birth and is continually enhanced by exposure to new pathogens and antigens throughout life.
 - D. Is achieved through a vaccination given to generate an immune response that results in the development of antibodies specific for the injected antigen.
5. Which antibody contributes to allergic and anaphylactic responses?
 - A. IgA
 - B. IgD
 - C. IgE
 - D. IgG
 - E. IgM
6. Which histamine receptor is responsible for coronary artery vasoconstriction, bronchoconstriction, cutaneous vascular permeability, intestinal smooth muscle contraction and prostaglandin production?
 - A. H1
 - B. H2
 - C. H3
7. Which of these best described urticaria?
 - A. Itchy, watery eyes
 - B. Sneezing or a "runny" nose
 - C. Raised areas or wheals that occur on the skin
 - D. An itching or tickling sensation in the upper airway
8. Define **Angioedema**:

9. What is pruritis?
- A. Bloating and cramping in the GI track
 - B. Increased secretions from the eyes and nose
 - C. Itching of palms of hands, soles of feet, or throat
 - D. Fine, red, rash involving face, chest, back, and abdomen
10. Describe the physical S&S experienced by the varying degrees of allergic reactions.

S&S	Localized	Mild systemic	Moderate systemic	Anaphylaxis
CNS				
HEENT				
Resp				
Cardio-vascular				
GI				
Skin				

11. What additional problems should a paramedic consider if a pt presents with each of the following?
- Stridor:
 - Wheezing:
 - Hypotension:
 - Urticaria
12. If a patient has been stung by a bee and the only S&S are a red, swollen area at the site of injection, what should be done locally to the area of the bee sting?

13. An adult presents with peripheral tingling, scratchiness in the back of the mouth and throat, nasal congestion, periorbital swelling, tearing, and persistent sneezing following yard work. VS: BP 130/80; P 84; R 16; SpO₂ 98% on room air; lung sounds are clear. What severity of allergic reaction is evident?
- A. Local
 - B. Mild systemic
 - C. Moderate systemic
 - D. Severe systemic
14. Which of these is indicated for the above patient?
- A. Epinephrine 1:1,000 IM
 - B. Epinephrine 1:10,000 IVP
 - C. Albuterol & ipratropium/HHN
 - D. Diphenhydramine IM or slow IVP
15. An adult presents with dyspnea, anxiety, facial swelling, watery eyes, and sneezing following exposure to a cat. VS: BP 110/70; P 100; R 24; RA SpO₂ 94%; lung sounds: diffuse wheezing. Which of these is indicated first?
- A. Diphenhydramine IM
 - B. Epinephrine 1:1,000 IM
 - C. Epinephrine 1:10,000 IVP
 - D. Albuterol & ipratropium via HHN
16. An A&OX3 adult presents with urticaria and pruritis of the feet and ankles after exposure to poison ivy. There is no angioedema, wheezing, or dyspnea. VS are WNL. Which of these is indicated per SOP?
- A. Epinephrine 1:1000 IM
 - B. Epinephrine 1:10,000 IM
 - C. Diphenhydramine IM or slow IVP
 - D. Observe for progression and transport
17. What is the action diphenhydramine?
- A. Causes rapid bronchodilation
 - B. Reverses the action of macrophages
 - C. Prevents further histamine from being released
 - D. Immediately reverses the effects of histamine already in circulation
18. Which histamine receptor is blocked by diphenhydramine?
19. What is the dose & route of diphenhydramine for a pt with a mild systemic allergic reaction?
- A. 50 mg IN
 - B. 25-50 mg PO
 - C. 50 mg IVP/IO
 - D. 1 mg/kg IM or slow IVP
20. Which of these is a side effect of diphenhydramine?
- A. Palpitations & dysrhythmias
 - B. Thickened bronchial secretions
 - C. Respiratory depression & irritability
 - D. Postural hypotension & increased RR

21. What is the desired action of epinephrine when given for a moderate allergic reaction?
22. What is the initial dose and route for epinephrine when given for a moderate allergic reaction?
 - A. 1:1,000 1 mg IM
 - B. 1:1,000 0.3 mg IM
 - C. 1:10,000 0.1 mg IVP
 - D. 1:10,000 1 mg IVP/IO
23. In what populations should epinephrine be used cautiously for a moderate allergic reaction?
24. How often can epinephrine be repeated for a moderate allergic reaction?
25. List two side effects of giving epinephrine to a patient with a moderate allergic reaction.
 -
 -
26. What is the second drug (name, dose & route) to be given for a moderate systemic reaction?
27. If the patient with a moderate systemic reaction is still wheezing after the first two drugs, what additional drugs should a paramedic give?
28. What are the actions and doses of each?
29. Under what circumstances can an anaphylactic reaction occur with a patient's first exposure to a substance?
30. Which is true of anaphylaxis?
 - A. Signs and symptoms begin within 30-60 seconds.
 - B. Angioneurotic edema is a rare clinical presentation.
 - C. Reactions that develop more slowly tend to be much more severe.
 - D. The release of histamine causes gastrointestinal motility to decrease.
31. A conscious adult presents with severe dyspnea, angioedema, significant voice changes, chest tightness, retractions, absent breath sounds, BP 80/50; HR 116; RR 40 & SpO₂ 88% after eating peanuts 15 minutes ago. Which of these is indicated?
 - A. Drug assisted intubation
 - B. Insertion of an OPA & O₂ 15 L/NRM
 - C. Assist ventilations w/ BVM & transport immediately
 - D. Provide 1st line drugs & wait for response before definitive airway management

32. What causes patients with anaphylaxis to experience hypotension and a relative hypovolemia?
- A. Massive vasodilation & 3rd space losses
 - B. Increased sympathetic tone to the arteries
 - C. Decreased stroke volume & osmotic diuresis
 - D. Endotoxin release causing severe bradycardia?
33. How much IV fluid should a patient in anaphylactic shock receive?
34. Which of these is indicated first for a patient in anaphylactic shock?
- A. Epinephrine 1:1,000 0.3 mg IM
 - B. Epinephrine 1:10,000 0.1 mg IVP/IO
 - C. Albuterol & ipratropium via nebulizer
 - D. Benadryl (diphenhydramine) 50 mg IM
35. If a pt in anaphylaxis does not respond to IV fluid challenges and epi and the BP remains < 90, what drug is indicated next?
- A. Glucagon
 - B. Dopamine
 - C. Diphenhydramine
 - D. Albuterol & ipratropium

What is the minimum starting dose of this drug for anaphylactic shock?

Why? Which sympathetic receptors must be activated?

36. If a patient in anaphylactic shock is on beta blockers and is NOT responding to initial drug therapy, what drug (name, dose, route) should be given?

What is the action of this drug for anaphylaxis?

37. What is the desired action of epinephrine when given to a patient in anaphylaxis?
- A. Anticholinergic agent to dry secretions
 - B. Alpha stimulant resulting in vasoconstriction
 - C. Antiinflammatory agent to decrease hyperreactivity
 - D. H2 histamine blocker to reverse the immune response
38. How often should epinephrine 1:10,000 be repeated IVP/IO when treating a patient with a severe systemic allergic reaction/anaphylactic shock who is NOT in cardiac arrest?
- A. Every 5 to 10 minutes up to 3 mg
 - B. Every 3 to 5 minutes no dose limit
 - C. Every 1 minute up to a total of 1 mg
 - D. As rapidly as possible to attain the desired therapeutic effect

39. What is the desired action of dopamine when given to a pt with anaphylactic shock?
- A. Vasoconstriction & increased BP
 - B. Increased pulse & force of contraction
 - C. Bronchodilation and reduced air trapping
 - D. Increased renal blood flow & allergen elimination
40. What concentration, dose, route, and timing of epinephrine is indicated for a patient in anaphylactic shock who goes into cardiac arrest?
- A. 1:000 0.3 mg IM every 2 minutes
 - B. 1:000 1 mg IVP every 3 to 5 minutes
 - C. 1:10,000 1 mg IVP/IO every 2 minutes
 - D. 1:10,000 1 mg IVP/IO every 3 to 5 minutes
41. How should IV fluids be given to an adult patient in cardiac arrest from anaphylactic shock?
- A. 40 mL/kg IV bolus over 15 minutes
 - B. 2 lines with IVF as rapidly as possible up to 8 L
 - C. 1 line wide open with pressure infuser up to 2 L
 - D. 200 mL fluid challenges over 30 minutes up to 1 L
42. Is a patient in anaphylactic shock who experiences a cardiac arrest a good candidate for prolonged CPR?
- [] Yes [] No
- Why?
43. When treating a patient in anaphylaxis who goes into cardiac arrest, should Paramedics only give epi or should they additionally give the patient antidysrhythmics per SOP for the rhythm found e.g., atropine, amiodarone?

Glossary of terms

Antigen:	A substance that induces the formation of antibodies.
Antibody:	Proteins produced by plasma cells in lymphoid tissue. Antibodies may be present due to previous infection, vaccination, transfer from mother to fetus in utero, or may occur without known antigenic stimulus, usually as a result of an accidental exposure.
Basophils:	Are thought to bring anticoagulant substances to inflamed tissues. Increased numbers are found during the healing phase of inflammation and in chronic inflammation.
Bradykinin:	Causes increased vascular permeability, vasodilation.
Histamine	Is released from injured cells. The red flush of a burn is due to the local production of histamine. Histamine causes gastric secretion, flushing of skin, lowered blood pressure, and headache, brochospasms, laryngeal edema. Functions of histamine include increasing gastric secretion, dilatation of capillaries, and constriction of bronchial smooth muscle.
Heparin:	Is produced by the mast cells of the liver and by basophil leukocytes. It inhibits coagulation by preventing liberation of thromboplastin from blood platelets.
Mast cells:	Connective tissue cells that contain heparin and histamine in their granules. Important in cellular defense mechanisms including blood coagulation needed during injury or infection.