

Study Guide Chapters 22-25

Chapter 22

Respiratory Emergencies

Extremely common: two categories, acute and chronic, both can present as life-threatening.

Respiratory System: filters, warming. Humidifying and exchanging more than 10,000L of air per day (adult).

Upper: mouth, pharynx (nasal and oral) larynx and vocal cords.

Lower: trachea, bronchi, bronchioles and alveoli

Respiration= inspiring O₂ and exhaling CO₂

Ventilation = exchanging CO₂

Diffusion= crossing the capillary/alveolar junction

Perfusion = oxygenated blood pumped out to the tissues

Pathophysiology: problems that affect gas exchange (ventilation)

- Upper airway obstruction (trauma, epiglottitis, tonsillitis, choking)
- Lower airway obstruction (trauma, obstructive lung disease, mucus, bronchospasm airway edema)
- Impaired chest movement (trauma, hemothorax, pneumothorax, empyema (pus))
- Neurological control problems, depressed CNS function, stroke, disease

Diffusion-Related problems:

- Low O₂ in air
- Alveolar pathology (COPD, blebs)
- Interstitial space pathology, high water pressure, edema

Perfusion-Related Problems

- Inadequate blood volume, or hemoglobin levels
- Impaired circulation
- Chest wall trauma

Assessment Findings: Major focus is recognizing life-threats

- Altered mental status
- Severe cyanosis
- Absent breath sounds
- Stridor
- 1-2 word dyspnea
- Tachycardia > 130/min
- Pallor, diaphoresis
- Use of accessory muscles
- Focused History
 - Ever had this before, known pulmonary problems, ever been intubated,
 - Medications, changes in medications

- Possible toxic exposures
- General Impression, evaluate the following
 - Position- tripod position = moderately severe respiratory distress
 - Mentation
 - Ability to speak
 - Respiratory Effort
 - Skin color and appearance
- Vital Signs: obtain baseline vitals, Pulse, BP, Respiratory rate/patterns
- Head and Neck: pursed lips breathing, JVD, sputum suggests infection
- Chest: symmetry, trauma, barrel chest (COPD), retractions
- Extremities: carpopedal spasm = low CO2 levels
- Diagnostic testing: Pulse Ox, Peak flow, Capnometry (end of ET tube)

Obstructive Airway Diseases are Asthma (4-5%) and COPD: sometimes divided into emphysema and chronic bronchitis (20% of adult males). Contributing factors:

- Stress
- Infection
- Exercise
- **Tobacco smoke** can cause asthma and COPD
- **Allergens**: food, animal dander, dust, mold, pollen
- Drugs: reactions to beta blockers
- Occupational hazards: latex allergy

Pathophysiology of obstructive lung disease:

- Smooth muscle spasm/bronchospasm: beta receptors respond to sympathetic stimulation>bronchodilation. Beta blockers are bronchoconstrictors
 - Aminophylline
 - Theodur
 - Somophylline, Elixophylline, Brethine
 - Proventil, Ventolin, Alupent, Albuterol
- Mucus: cilia moves mucus out
- Inflammation

Asthma: 8.9 million people have this acute airflow obstructive disease of the lower airway. Hypersensitive bronchial airways that are easily irritated>bronchospasm> limits movement of air>increased mucus> swelling and edema> inflammatory cell proliferation

Most common onset in children and young adults. 1/3 develop it before age 5, 1/3 of children outgrow it, adult onset asthma is usually persistent. 1/4 diagnosed after age 50.

- Extrinsic asthma: some specific outside substance causes bronchioles to narrow, more common in children
- Intrinsic asthma: no substance identified as causing narrowing. More commonly adult onset

Triggers to Asthma Attack:

- Respiratory infections

- Allergens
- Drugs
- Irritants
- Exercise
- Emotions/Stress
- Chemicals
- Changes in environmental conditions

Status Asthmaticus: severe, prolonged asthma attack that does not respond to standard medications. Monitor closely, transport immediately

COPD: progressive and irreversible disease, marked by decreased inspiratory and expiratory capacity of the lungs. Usually a combination of bronchitis and emphysema. Caused by:

- Overgrowth of airway mucus glands, excess mucus
- Emphysema = destruction of the elastic walls of the alveoli (they trap air and eventually pop)
- Airway has a marked resistance to air movement
- COR PULMONALE = Right Side heart failure due to effort required to move blood to diseased lungs
- Major cause is cigarette smoking
- *Quiet sounding chest in a patient who is obviously short of breath is ominous – airways may be too tight to wheeze-----*
- CO2 Retainers = patient who have lost their CO2 drive to breathe, low O2 (hypoxic) is their only drive to breathe. Quite unusual

Signs and symptoms:

- Shortness of breath, 1-2 word dyspnea
- Coughing
- *Cyanosis ----- Serious*
- *Anxiety, feeling of suffocation, too tired to breath ----- Problems*
- *Altered LOC----- Transport*
- *Diaphoresis and pallor----- Priority*
- Tachycardia
- Hypertension
- Tachypnea
- Cigarette stains on fingers
- Barrel chest
- Use of accessory breathing muscles
- Audible abnormal breath sounds
- Decreased pulse ox (low 90s is good for COPDers)
- Neck vein distension
- Leg edema

Management of COPD/Asthma

- Intubate if: severe respiratory difficulty, cyanosis, BP<70
- Or provide high flow O2 on a non-rebreather (85-100% O2) or nasal cannula 6L/min (24-44%)
- Transport upright, keep calm, don't exert
- Monitor vital signs, pulse ox, ECG
- Treat bronchospasm with albuterol (beta 2 agonist) and/or epinephrine
- IV solution of normal saline or lactated Ringers or 5% dextrose in water
- Transport priority patients

Pneumonia: acute inflammatory condition of the lungs, Bacterial, fungal or viral, 5th leading cause of death. Group of infections due to many different agents. Disorder of ventilation due to infection of the lung parenchyma> sometimes atelectasis.

- Community acquired: not as severe
- Hospital acquired: pseudomonas = gram negative bacteria

Risk factors:

- Cigarette smokers
- Alcoholics
- Cold exposure (chronic hypothermia – homeless people)
- Extremes of age
- Abnormal immune systems

Assesment: typically acute fever and chills, cough, pleuritic chest pain, crackles

Management: Contagious! Airway support, O2, IV to rehydrate, watch for septic shock, reduce fever, inhaled Beta-2 agonists may be helpful.

Pulmonary Edema: filling of the lungs with fluid in the interstitial spaces, the alveoli or both. Classified as either high pressure (cardiogenic, resulting from AMI) or high permeability (noncardiogenic, hypoxemia, near-drowning, shock etc.) pulmonary edema.

Pathophysiology: result is impaired gas diffusion, particularly O2

In high pressure (cardiogenic) pulmonary edema:

- Ischemia leads to left ventricle failure> increased ventricular pressure> pushes left atrium>pushes back into the pulmonary veins
- Increased pulmonary capillary pressure>engorged vessels leak>accumulates in the interstitial spaces>impairs gas diffusion> alveoli rupture

In high Permeability (noncardiogenic) pulmonary edema the alveolar/capillary membrane is disrupted

- Severe hypotension
- Severe hypoxemia (post drowning, post cardiac arrest, severe seizure)
- High altitude
- Environmental toxins
- Septic shock

Assessment: Most common presentation is Acute SHORTNESS OF BREATH. Evaluate any chest pain, cardiac history, hypoxic episode, shock, chest trauma, toxic gas inhalation, high altitude. Sign and symptoms: dyspnea, orthopnea (worse lying down),

fatigue, reduced exercise capacity. Wet sounding lungs, crackles, wheezes, decreased pulse ox, and cardiac dysrhythmias. 12-lead ECG may show MA or stress.
Management: High-flow O₂, keep calm, they don't need any more adrenaline, IV if local protocol but KEEP FLOW LOW or TKO. Transport upright.

Pulmonary Thrombo(form a clot) Embolism(break off and float up): blockage of a pulmonary artery by foreign matter. Usually it's a blood clot formed in a pelvic or deep leg vein. 50,000 death annually, 5% of sudden deaths. Around 10% die, and that 10% in less than an hour. Risks:

- Sedentary lifestyle
- Obesity
- Infection
- Cancer
- Thromboplebitis
- BCPs
- Fracture of long bone
- Pregnancy
- Recent surgery
- Blood diseases

Pathophysiology: blood supply to some lung is blocked. Clot breaks off in leg, follows blood through right artia and ventricle and becomes lodged in the lung where it can go no farther> lung tissue ischemic> blood pools>right ventricle pumping against very high pressures> acute cor pulmonale>decreased blood supply

Assessment: Massive PE= cardiac arrest/syncope, altered mentation, cyamosis profound hypotension. Smaller = sudden chest pain, increases with deep breaths, pleuritic chest pain, shortness of breath, respiratory distress, wheezing, hemoptysis, anxiety, shock. Similar to MI or spontaneous pneumothorax. Tachypnea, tachycardia
Management: ABC, hig flow O₂, IV, watch for shock, cardiac monitor, transport in position of comfort

Spontaneous Pneumothorax: sudden accumulation of air in the pleural space. Lung on the involved side collapses. Tension pneumo can also develop.

- More common in men
- Young tall male smokers
- Congenital defect
- Menstruation
- Lung disease
- COPD

Assessment: sudden onset, sharp chest pain, shortness of breath, decreased lung sounds, increased respirations, coughing anxious agitated.

Signs and Symptoms of a Tension Pneumothorax

- Weak pulse

- Cyanosis
- Hypotension
- Decreased breath sounds
- JVD
- Tracheal deviation (late sign)
- Subcutaneous emphysema, crepitus

Management:

- Maintain airway, highflow O₂
- IV
- Cardiac monitor
- Transport in position of comfort
- Needle decompression as allowed by protocol

Hyperventilation Syndrome: respiratory rate greater than that required for normal body function. Disease states resulting in hyperventilation:

- Asthma attack
- COPD
- MI
- Spont. Pneumo
- CHF
- Diabetic ketoacidosis
- Drugs
- Psychogenic factors

Assessment: Chest pain, dizzy, faint, numbness, tingling in extremities, altered mental status, tachycardia, palpitations.

Management: don't assume it is just anxiety, there are lots of disease states that cause hyperventilation. Assume illness until proven otherwise. Give O₂, Take a pulse Ox, try to help them slow breathing, if chest pain > IV, transport according to protocols.

Chapter 23

Cardiovascular Emergencies

Epidemiology: CVD is number 1 killer, 2600 die each day, High BP, Coronary Heart Disease, AMI, Angina, Stroke, Rheumatic heart disease and CHF, 1 in 5 adults has some form. Risk Factors:

- Age: death rates rise with each year of life
- Family history: predisposition
- Hypertension: major risk factor $BP > 170/95 = 5$ times higher risk
- Lipids: high bad cholesterol
- Smoking
- Gender: males have higher risks
- Diabetes

Cardiovascular system

Stroke volume: amount of blood pumped into the cardiovascular system in one heart contraction Amount is usually around 70mL. Depends on:

- Contractility: the extent and velocity of muscle fiber shortening
- Preload: the passive stretching force on the ventricular muscle at the end of diastole. More blood returning increases the preload, less blood returning decreases it. If the container (vessels) is greater than the fluid, you'll have inadequate preload and a decrease in cardiac output.
- Afterload: the pressure the ventricular muscles must generate to overcome the higher pressure in the aorta, and eject the blood out.

Myocardium: Heart muscle

Epicardium: outside of the heart

Pericardium: thick set of two membranes surrounding the heart, inner is visceral, outer is perietal

Blood Pressure: the force that blood exerts against the walls of the arteries as it passes.

Systole: pressure within the arteries during heart contraction, concomitant pumping

Diastole: Relaxation phase, also indicates myocardial perfusion

Cardiac Output: amount of blood pumped each minute. Heart rate x stroke volume.

Cardiovascular system is closed so increasing cardiac output or vascular resistance increases blood pressure. Decreasing cardiac output or vascular resistance decreases BP.

Pulmonary Circulation: transports deoxygenated blood through the lungs, oxygenate it, and return it to the left side of the heart

Systemic Circulation: left ventricle pumps blood out to the body through SEMI-LUNAR (AORTIC) valve to the aorta

Coronary Circulation: where the heart muscle receives its blood supply, right main, left main etc. etc.. Five coronary veins empty into the right atrium via the coronary sinus.

Blood Vessels: closed system, sometimes called "the container"

- Elastic, always adjusting their diameter
- Responding to local tissue needs, bypassing less important tissues for vitals
- Sympathetic nervous system activates dilation and contraction

Inherent Pacemaker Rates of the Heart:

“Foci” or the focus: where electrical impulses start, Organized rhythm is required for perfusion

Complexes: blocks of rhythms

Isoelectric line: midline on an ECG, up is depolarizing, down in repolarizing

Normal Sinus rhythm: (NSR) originates in the uppermost node, the SA (SinoAtrial) node, rate is usually between 60 and 100 beats per minute

AV Junction: if SA or atria fail, the junctional node starts. Usually 40-60 beats per min. (no P-wave will show on the chart)

Junctional Rhythms: any rhythm starting in the AV Node or the AV Junction

Ventricular: If SA, atria and AV junction fail, bundle branches and purkinje’s fibers, 20-40 beats/min

V-Fib: Totally unorganized, causes the heart to quiver (get the AED)

V-Tac: wide rhythms, electrical impulses starting from the lower bundles (get the AED)

A-Fib: Abnormal heart rhythm

PVC: Premature Ventricular Contraction. Will show as a big fat wave in the middle of all the regular complexes

Trigeminy: PVC one out of every three contractions

Bigeminy: PVC one out of every two contractions

Unifocal PVC: one focal point is firing

Multifocal PVC: multiple focal points are firing

The EKG is just showing the electrical impulses. This is not proof that the heart is, in fact, beating. Check the pulse, there may be no mechanical capture of the electrical signal.

EMD: Electromechanical disassociation

PEA: Pulseless electrical activity

Electrophysiology:

Automaticity: self-generate electrical activity

Excitability: respond to appropriate electrical stimulus

Conductivity: transmits stimulus from cell to cell

Contractility: Contract when stimulated by the appropriate electrical stimulus

Regulation of heart function: comes partially from the brain (autonomic), from hormones if the endocrine system and from the heart tissue

Baroreceptors: sensory nerve endings that sense changes in BP from vaso dilation/constriction

- Lower BP in response to increased arterial pressure
 - Inhibit medulla
 - Excite vagal center

- Decrease force of cardiac contraction
- Increase BP in response to decreased arterial pressure
 - Inhibit vagal center
 - Activate sympathetic nervous system > norepinephrine and epinephrine

Chemoreceptors: walls of atria of heart, vena cava, aortic arch and carotid sinus. Increase in CO₂/decreases in O₂ initiate a sympathetic response to increase rate and depth of respiration.

Chronotropic State: control of the heart rate
 Less than 60 = bradycardia
 More than 100 = tachycardia

Dromotropic State: rate of electrical conduction

Inotropic State: Strength of contraction

Electrocardiographic Monitoring: a record of the electrical activity in the heart, transferred to the ECG machine and displayed or printed on paper

Boxes on the paper represent time: .2 sec largest lines

Positive impulses go upward, negative go downward

Flatline: Isoelectric line is produced if no electrical impulse is present

P-Wave: occurs first and represents depolarization moving through the atria, resulting in atrial contraction. Upright and round, usually 60-100

PR Segment: pause as the impulse passes through the AV node

QRS Complex: depolarization moving through the ventricles, ventricle contraction/systole

ST Segment: Repolarization of the ventricles, shows flat

T wave: Complete repolarization

U wave: sometime a small wave before the next P wave

Dysrhythmias : irregularities of the heart rhythm: including

- Myocardial ischemia/necrosis
- Autonomic nervous system imbalance
- Distention of heart chambers
- Acid-base abnormalities
- Hypoxemia
- Electrolyte Imbalance
- Drugs
- Electrical injury
- Hypothermia
- CNS injury

Atrial dysrhythmias

- Ischemia
- Hypoxia
- Atrial stretching due to CHF
- Increased pulmonary artery pressure
- Wandering atrial pacemaker > pacemaker site switches from beat to beat (also MAT multifocal atrial tachycardia)

- Atrial flutter 250-350 beats a minute\
- Atrial fibrillation> multiple area fire simultaneously

Assessment: LOC, ABCs, SAMPLE, OPQRST

Detailed Physical Exam: JVD, Thorax, Epigastrium (AAA?)

Vitals: BP, heart rate, skin temp, color, condition, pulse O_x

EKG: The EKG is just showing the electrical impulses. This is not proof that the heart is, in fact, beating. Check the pulse, there may be no mechanical capture of the electrical signal.

Management: ABCs, is the patient stable?

Asystole: prompt CPR, O₂, intubation, IV line, epinephrine, atropine, transport

PEA: prompt CPR, O₂, intubation, IV line, epinephrine, atropine, transport

V-Tac: AED

V-Fib: AED

Other causes of chest pain – potentially life threatening problems

Angina: Angina Pectoris is an intermittent attack of chest pain due to a reduction in blood flow to the heart muscle> exertion, stress, cold weather

- Stable
- Unstable
- Progressive
- Preinfarction

AMI: Acute Myocardial Infarction: death of an area of heart muscle due to blockage of blood flow in a coronary artery

Aneurysm: Abnormal dilation of the aorta

Aortic Dissection: sudden tear in the wall of the aorta

Blunt Trauma

Cholecystitis: inflammation of the gall bladder

Pancreatitis

Pericarditis

Pneumothorax

Pulmonary Embolism

Congestive Heart Failure: circulatory congestion due to inadequate flow of blood.

Chapter 24 Diabetic Emergencies

Counterregulatory Hormones: glucagons and epinephrine raise the blood sugar – opposite of insulin

D₅₀ – Medication used to treat hypoglycemia, contains 25 grams of glucose in 50 mL of water

Diabetic Keto Acidosis (DKA): metabolic condition consisting of hyperglycemia, dehydration and accumulation of ketones and ketoacids.

Fingerstick Blood Sugar: testing blood sugar on a glucometer with a drop of blood from the finger. Results in 15-45 seconds.

Glucose: main nutrient for all cells, transported in the blood (blood sugar) approx. 75-100 is normal

Hyperglycemia: elevation of blood sugar level above normal, most common cause is diabetes

Hyperglycemic Hypersmolar Nonketotic Coma (HHNC): blood sugar is elevated but no acidosis is present. Dehydration and lowered level of consciousness

Hypoglycemia: abnormally low blood sugar level, sometime called insulin shock

Insulin: released from the pancreas and together with epinephrine and glucagons, regulated the blood sugar level. Insulin stimulates the production of glucose transport proteins to facilitate the passage of glucose into the cells to be broken down into energy (*Facilitated Diffusion*). Insulin also prevents the breakdown of fatty tissue in the body.

Ketoacids/Ketones: formed when the body metabolizes fats/fatty tissue
Kussmaul Respirations: rapid, deep sighing breaths that help a diabetic blow off excess CO₂, and lower the levels of acid>ketoacidosis

Type 1 Diabetes: require insulin injections to live, usually onset at a younger age

Type 2 Diabetes: non-insulin dependent, onset usually after teenage years, less prone to DKA, most can maintain blood sugar levels with diet.

Diabetes: a chronic disease of the endocrine system caused by a decrease in the secretion or activity of insulin. Diabetics don't make enough insulin to regulate the blood sugar level – it goes too high or too low. When it is too low, the cells start to starve.

Complications Of Diabetes

- Eye Disease: diabetic retinopathy causes bleeding in the vitreous humor
- Kidney disease: diabetic nephropathy>abnormal function> complete renal failure
- Nerve disease: chronic pain and decreased sensation, especially in the lower extremities
- Increased risk of cardiovascular disease: increased risk and at a younger age

Hypoglycemia: occurs as a result of an imbalance in the amounts of insulin and glucose. Most commonly caused by a patient taking insulin and then not eating enough food. causes include:

- Medications
- Excessive exercise
- Alcohol
- Poor diet
- Hypothermia
- Liver disease

Assessment: hypoglycemia develops rapidly, over a few minutes to a few hours.

Counterregulatory hormones are secreted causing early warning signs:

- Shakiness
- Weakness
- Diaphoresis
- Rapid pulse and respirations

Severe hypoglycemia

- Altered level of consciousness
- Slurred speech
- Neurological deficit
- Seizure

HYPOTENSION is NOT a sign of hypoglycemia. Look for another cause.

Emergency Care:

- Control the airway
- Give O₂ via nasal cannula at 3/4 L/min
- Monitor the ECG
- Give OJ with 2 packs of sugar, oral glucose, corn syrup or candy
- Start an IV and draw a blood sample
- Local protocols may allow D50, 50% dextrose (25 g dextrose in 50mL water) intravenously
- Always assume hypoglycemia and give sugar. Never give insulin
- Provide psychological support

Diabetic Ketoacidosis – DKA: diabetic person has inadequate insulin circulating to control blood sugar levels. Also there's excess epinephrine and glucagons present. Blood sugar level rises, fatty tissue breaks down forming ketones and ketoacids> changes blood acid/base balance>frequent urination>dehydration>loss of body chemicals (potassium)> chain of events leads down hill. Takes longer/more problems than hypoglycemia. Usually starts with an infection. Too small dose of (or forgot to take) insulin. Pretty rare. Signs and Symptoms:

- Weakness, nausea, vomiting (of course, more vomiting)
- Abdominal pain
- Polyurea, polydipsia
- Kussmaul respirations
- Altered LOC

- Fruity, acetone odor to breath (not always)
- Normal BP or mild hypotension
- Rapid, weak pulse

Emergency Care:

- Control the airway
- Give High concentration O₂
- Monitor the ECG
- Start an IV and draw a blood sample. Fluid bolus usually 500-1000mL normal saline or lactated Ringer's
- Watch for shock
- Nothing by mouth, Never give insulin
- Transport
- Psychological support

Hyperglycemic Hyperosmolar Non ketotic Coma (HHNC): Relative insulin deficiency, high levels of glucose in the CSF > but no ketones present > dehydration of the brain and decreased level of consciousness. Most at risk:

- People over 60
- Precipitated by infection, extreme cold or dehydration
- Gradual deterioration over 4-5 days
- No Kussmaul breathing, no fruity breath (because no acidosis)

Managing Diabetic Patients: Always ask?

1. What did you eat today?
2. Did you take your insulin?
3. Has the dosage changed?
4. Are you under any unusual stress?

Physical Assessment: look for:

- Altered Mental Status
- Kussmaul respirations
- Tachycardia
- Hypotension
- Skin color, temperature
- Hydration status

Chapter 25

Allergic Reactions

Allergic Reaction: result from exposure to any substance to which an individual is sensitive

Anaphylaxis: Specific type of allergic reaction caused by the interaction of an allergen (called an antigen) and one antibody (IgE)

Allergen: Generic term for any substance to which a person is sensitive

Antigen: an Allergen

Antibody: Antibodies respond to antigens. There are five types of antibodies, part of the immune system that recognizes antigens and stimulates an immune system response

Diphenhydramine: Benedryl. An antihistamine that blocks the effects of histamine

Epinephrine: Adreneline. Stimulates alpha and beta blockers

Histamine: Cellular substance released into the body during anaphylactic shock, causes bronchospasm, vasodilation and leakage of fluid from vessels

IgE: Ig=immunoglobins. IgE is the only antibody involved in anaphylaxis

Pathology of Anaphylaxis:

- Antibodies attack antigens. The severity of the symptoms range from progressive hives to cardiac arrest.
- Histamine release occurs first
 - Bronchospasm
 - Vasodilation
 - Leaking of fluid
- Histamine stimulates the release of other mediators
 - Unpredictable spiral of events
- Biphasic (early and late) anaphylactic response is 1 out of 5 people
 - Reoccurrence 4-5 hours later> may lead to shock

Presentation, Signs and Symptoms

- Upper Airway: hoarse, stridor, edema, runny nose
- Lower Airway: bronchospasm, mucus, wheezing, decreased breath sounds
- Cardiovascular system: tachycardia, hypotension, dysrhythmia, chest tightness
- GI: nausea, vomiting, cramps, diarrhea
- Neurological system: anxiety, dizziness, syncope, weakness, headache, seizure, coma
- Cutaneous: angioedema, urticaria, pruritus, erythema, edema, tearing of the eyes

MOST COMMON: hives, wheezing, abdominal pain

Methods of Entry into the Body:

- Inhalation
- Absorption
- Injection
- Ingestion

Assessment:

Get a good history ask about recent changes in foods, drugs, detergents, cosmetics etc.

Signs and Symptoms of Anaphylactic Shock

- Sense of agitation, confusion, decreased LOC
- Swelling of soft tissues like hands tongue and pharynx
- Wheezing, rales, rhonchi
- Tingling, burning, itching skin
- Abdominal pain
- Tachycardia
- Weak, thready pulse
- Profound hypotension (late sign)
- Weakness
- Diaphoresis
- Cyanosis
- Peripheral edema

Mild Reactions:

Treatment: Benadryl 10-50 mg slow IV push or IM if vital signs are normal, no respiratory symptoms, itching rash/swelling on outside of body.

Moderate and Severe Reactions:

- Aggressive airway management
- Ventilatory support
- Oxygen therapy
- Circulatory support
- Epinephrine .3-.5 mL subcu if:
 - Wheezing or stridor
 - Edema of the pharynx, tongue or soft palate
 - Vascular compromise (hypotension, weak thready pulse, confusion, tachycardia)

Anaphylactic shock:

- Provide reassurance, keep them calm
- Ensure an adequate airway
- Intubate if the patient can't maintain an airway
- Apply EKG pads to monitor
- High flow O2 by non-rebreather
- Start an IV, large bore cannula, normal saline or lactated Ringers, macro drip, BP<90 run it wide open
- Administer epinephrine .3-.5 mg subcu, or IM with an Epi-Pen
- Bronchodilators may be used for stridor or wheezing
- Transport priority patients