1. What is TPA? What are at least 2 conditions where you might see it given as a medication? Why would we not give it?
   tPA: clot busting agent used to reduce the effects of CVA (stroke) in some individuals. It is contraindicated for anticoagulant drugs.
   The release of tissue plasminogen (tPA) from injured blood vessels and tissues, converts plasminogen to plasmin. Plasmin is a potent enzyme that digests fibrin strands (this leads to clot dissolution).

2. Know the triad that would lead you to conclude your patient is experiencing cardiac tamponade
   Fluid compresses the heart, prevents stretching & filling, reduces CO
   Arterial pressure falls, venous pressures rise, pulse pressure narrows
   <u>Beck's Triad:</u> Distant, muffled heart sounds (like under water, fluid), JVD, decreased BP
   =EMERGENCY
   pericardiocentesis with surgical repair as appropriate is needed (16-18 gauge needle inserted into pericardial space to relieve pressure and analyze fluid)
   Manifestations include heavy feelings over the chest, shortness of breath, tachycardia, cough, dysphagia, hiccups, hoarseness, nausea, vomiting, excessive perspiration, decreased level of consciousness, pulses paradoxes, distant or muted heart sounds, and extreme anxiety, neck vein distension

3. The 3 Ps of diabetes
   Polydipsia: osmolality, polyuria: osmotic diuresis, and polyphagia: cell starvation

4. What electrolyte is of most concern with kidney failure patients?
   Potassium: cardiac

5. Review the signs of hypokalemia and hyperkalemia
   Hypokalemia: muscle cramps, weakness, cardiac arrhythmias, flattening of the T wave, confusion and drowsiness, and irritability
   Hyperkalemia: peaked T wave, v-fib, cardiac arrest, muscle weakness, respiratory distress

6. Right sided heart failure versus left sided heart failure
   CHF: Occurs when the heart is unable to pump sufficient blood to meet the metabolic needs of the body; usually occurs secondary; may present as an acute episode but usually is a chronic condition; may result from an infarction or a valve defects may arise from increased demands on the heart, such as those imposed by hypertension or lung disease;
   <u>Left-Sided Heart Failure</u>: Characterized by impaired pumping ability of left side of heart; Eventually backs blood up into pulmonary circulation (crackles)- THIS IS BAD-FLUID IN THE LUNGS MEANS LESS AIR IN THE

LUNGS; Results in elevated pressure and congestion in pulmonary veins/capillaries; Symptoms: Fatigue, activity intolerance, shortness of breath, cough, orthopnea, paroxysmal nocturnal dyspnea
****tripod position (leaning over: usually seen); **Pulmonary congestion: pink frothy sputum, cough, crackles, wheezes, tachypnea**
<u>Right-Sided Heart Failure</u>: Characterized by impairment of pumping ability on right side of heart; Backup of blood followed by congestion and elevated pressure in systemic veins and capillaries; Most common cause is left-sided dysfunction; Symptoms result from volume overload leading to ascites and edema; Patients may complain of abnormal bloating and discomfort, with poor appetite and sometimes nausea and vomiting

7. Can you tell the difference between diabetes insipidus, diabetes mellitus type 1 & type 2?
   DI: lack of ADH action, dehydration
   DM1: complete lack of insulin, usually diagnosed young; Absolute absence of insulin – must have injections or insulin pump for life. NOT oral meds; Two types: immune mediated (most common, especially in children; Latent autoimmune diabetes (LADA) in adults
   Genetic disposition + trigger (eg infection) + T cell hypersensitivity to beta cell antigen) and idiopathic (less common, no autoimmune component)
   DM2: lifestyle changes, accounts for 90% of diabetes, strong correlation with diabetes, genetic component, cellular resistance to insulin, deranged insulin secretion: beta cells fail, increased glucose production by liver; diagnosed later d/t insidious onset; frequent infections, slow wound healing, blurred vision; Usually oral meds as initial pharmacotherapy but often require insulin therapy later; often require insulin when acutely ill on temporary basis

8. Can you summarize the difference between multiple sclerosis and ALS? How about how do they differ from Parkinson's?
   <u>MS: (Multiple Sclerosis)</u> involves a progressive demyelination of the neurons in the brain, spinal cord, and cranial nerves; characterized by remissions and periods of exacerbations; considered a disease of young to middle-aged adults with the onset usually being between 20 and 50 years of age; Onset is usually insidious and gradual; course is unpredictable; debilitating autoimmune disorder; Clinical manifestations: fatigue, ataxia, muscle spasms, paresthesia or abnormal sensations, difficulty moving arms or legs, unsteady gait, tremor, bowel and bladder issues, decreased attention span, difficulty reasoning, dizziness, hearing loss; Predisposing factors include being between 20-40 years old, smoking, women, and caucasian ethnicity
   <u>ALS: (Amyotrophic Lateral Sclerosis)</u> progressive degenerative disease affecting both upper motor neurons in the cerebral cortex and lower

motor neurons in the brain stem and spinal cord, no indication of inflammation around the nerves; The Sensory neurons, cognitive function, and cranial nerves III, IV, and VI are not affected; Unlike MS, ALS will lead to death; Clinical manifestations include: foot drop (difficulty lifting the front of the foot and toes), lower extremity weakness, hand weakness or clumsiness, slurred speech or dysphagia, muscle cramps and twitching in upper extremities and tongue;**
It will eventually affect chewing, swallowing, speaking, & breathing
Parkinson's: Progressive condition involving destruction of substantia nigra in brain which results in a lack of dopamine. Dopamine is responsible for smooth, coordinated muscle movement; It is a disorder of the basal ganglia; has Lewy bodies: unusual protein deposits; Clinical manifestations – slowing or stopping of automatic movement like blinking, constipation, dysphagia, drooling, unsteady gait, masklike appearance to face, myalgias, difficulty initiating walking, lose of fine hand movements (difficulty with handwriting), shuffling gait, bradykinesia (slow movements), tremors, finger-thumb rolling (pill-rolling), monotone voice

9. Can you summarize the difference between osteoarthritis and Rheumatoid arthritis?
(chart from webMD)

Characteristic	**Rheumatoid arthritis**	**Osteoarthritis**
Age at which the condition starts	It may begin any time in life.	It usually begins later in life.
Speed of onset	Relatively rapid, over weeks to months	Slow, over years
Joint symptoms	Joints are painful, swollen, and stiff.	Joints ache and may be tender but have little or no swelling.
Pattern of joints that are affected	It often affects small and large joints on both sides of the body **(symmetrical),** such as both hands, both wrists or elbows, or the balls of	Symptoms often begin on one side of the body and may spread to the other side. Symptoms begin gradually and are often limited to one set of

	both feet.	joints, usually the finger joints closest to the fingernails or the thumbs, large weight-bearing joints (hips, knees), or the spine.
Duration of morning stiffness	Morning stiffness lasts longer than 1 hour.	Morning stiffness lasts less than 1 hour. Stiffness returns at the end of the day or after periods of activity.
Presence of symptoms affecting the whole body (systemic)	Frequent fatigue and a general feeling of being ill are present.	Whole-body symptoms are not present.

***Osteoarthritis is caused by mechanical wear and tear on joints. Rheumatoid arthritis is an autoimmune disease in which the body's own immune system attacks the body's joints.**
RA: mainly affects middle joints and OA: mainly affects end joints

10. What cholesterol is the good cholesterol? (Hint - remember LDL = lousy)
HDL: the "good" lipoprotein; has a low lipid content and is used to transport cholesterol AWAY from the peripheral cells to the liver, where it undergoes catabolism and excretion; is increased with exercise; protects women until after menopause; smoking decreases HDL levels;

11. What kind of immunity do we get from vaccines? How about from mother to baby? How do you get active immunity?
Vaccines: Acquired, Active, ARTIFICIAL (immunization)
Mother to baby: Acquired, Passive, NATURAL (maternal antibodies)
Active immunity: refers to our own antibodies; not ready-made antibodies (Passive: either from mother to baby or artificial: antibodies from another source); Natural active: exposure to infectious agent (creating our own antibodies) and Artificial active: from immunizations

12. What does Starling's law tell you about fluid/blood volume and the contractile force of the heart?
Frank Starling's law states that, to a point, the more myocardial fibers are stretched, the greater their force of contraction.

13. If 2 patients came in with BG of 750 and looked dehydrated and delirious, what signs and symptoms would you look for to differentiate between DKA and HHNS?
    DKA: Hyperglycemia; Often presenting at time of DMI diagnosis; Must have insulin to prevent death; Kussmaul's respirations, fruity acetone breath, metabolic acidosis
    HHNS: Hyperglycemic, Hyperosmolar, Non-Ketotic Coma; Action of insulin is severely inhibited; Most typical in elderly DMII; Minimal or no ketosis; S/S: Polyuria then oliguria, lethargy progressing to coma, increased temp, HR, and decreased BP, signs of severe fluid deficit, seizures, blood glucose level often > 600 mg, increased serum osmolality; same symptoms as DKA except more dehydrated (and no respirations, acidosis)

14. Can you explain diverticulosis, appendicitis, cholelithiasis, choleycystitis?
    Diverticulosis: Diverticulum development in the large intestine; Diverticula: small pouch-like projections of through the muscular wall of the intestine; Risk factors: age, obesity, low-fiber/highly refined diet; Typically asymptomatic- patients may report h/o (history of) chronic constipation alternating with diarrhea
    Appendicitis: Inflammation and infection of the appendix
    Signs/Symptoms: Abrupt onset (rapid), Abdominal pain (epigastric, periumbilical; as advances LRQ) & tenderness, Rebound tenderness, Nausea and vomiting, Elevated temp, Elevated CBC;
    Complication: Rupture, which can lead to peritonitis, which can lead to sepsis;
    Tx: removal of appendix
    Appendicitis tends to present in younger populations (5 years to 30 years
    Cholelithiasis: Also known as gallstones; Incidence increases with age, other risk factors include obesity, DM, cirrhosis, Crohn's disease
    3 factors pathological factors contribute; 1) abnormal composition of bile (high cholesterol), 2) stasis of bile, 3) infammation of gallbaldder
    Gallstones classified as cholesterol, pigment or mixed content
    Gallstones often asymptomatic unless they block the bile duct
    Symptoms with obstruction: Indigestion, colicky abdominal pain, jaundice; Pain usually in RUQ (often referred to back); Found more in Caucasians, females, after pregnancy; Antibiotics: preventive measure first against infection; After excising the gallbladder: on low fat diet at home because the liver emulsifies fat, wont get broken down as easily
    Cholecystitis: Inflammation of the gallbladder
    Types: acute, chronic; Partial or complete obstruction of the common bile leads to inflammation which leads to ischemia, therefore an increased risk for infection (sepsis); Risk factors: sedentary lifestyle, overweight, gallstones

Signs & symptoms: RUQ pain & tenderness, epigastric pain, referred right scapular pain, N/V, low grade fever, elevated WBC; Usually go and take gallbladder out (microscopically)

15. Why do we say to put patients on the lowest amount of oxygen that meets the body's needs?
    Higher flow rates usually do not help and can increase the risk of harmful carbon dioxide buildup in the blood, especially in people who also have lung disease. (Provider will set flow rate)

16. Most common childhood leukemia, most common adult leukemia
    ALL: most common childhood leukemia; proliferation of more immature lymphoid cell-linecells
    AML: most common adult leukemia; proliferation of more immature myeloid cell-line cells
    (CML: associated with Philadelphia chromosome, 3 phases: chronic, accelerated and blast)
    (Hodgkin Lymphoma: Reed-Sternberg cells: transformed lymph cells; Non-Hodgkin's: less predictable)
17. What lab values will tell you the patient has progressed from HIV to AIDs?
    In people with HIV, CD4 count the most important laboratory indicator of how well your immune system is working and the strongest predictor of HIV progression; Once a person is infected with HIV, the virus begins to attack and destroy the CD4 cells;
    ***A very low CD4 count (less than 200 cells/mm3) is one of the ways to determine whether a person living with HIV has progressed to stage 3 infection (AIDS)**

18. What does the liver make? What are the consequences of liver dysfunction?
    The liver serves many functions. It <u>produces bile</u>, metabolizes hormones and drugs, synthesizes proteins, glucose and clotting factors, stores vitamins and minerals, changes ammonia to urea and converts fatty acids to ketones.
    Jaundice: abnormally high bilirubin level, in hepatitis jaundice results from inability of the liver to conjugate bilirubin
    ALT and AST are liver enzymes that are frequently assessed to determine liver function. ALT is liver specific, AST is also found in other organs. Elevated ALT and AST values are found in hepatitis, cirrhosis and other liver disorders.
    Portal hypertension, contributes to ascites in the abdomen (accumulation of fluid in the peritoneal cavity). It produces spleen enlargement; an enlarged spleen can retain more blood (leading to anemia, low platelets and leukocyte count). Blood that is shunted to collateral veins leads to varicosities in the anal area (hemorrhoids) and esophageal varices (these esophageal veins can hemorrhage and the patient will bleed to death).

Portal hypertension: cirrhosis leads to elevated portal vein pressure, will back up into spleen and blood from spleen will not be able to drain, enlarge, veins start to dilate and become tortuous on spleen, stomach, esophagus (esophageal varices)**

19. Can you differentiate very plainly between hypothyroid, hyperthyroid, Graves, Hashimoto's, Addison's, Cushing's, and adrenal insufficiency?
    Addison's: refers to a deficiency of adrenocortical secretions, the glucocorticoids, mineralocorticoids, and androgens (can not make all 3 adrenal hormones); an autoimmune reaction is the common cause(TB was the common cause before: JFK); high risk of infection, poor stress response, weight loss, fatigue, anorexia, nausea, diarrhea, hypotension, syncope, hyperpigmentation, (increased ACTH)
    Cushing's: Oversecretion of Cortisol – tumor; Overstimulation of Adrenal Cortex; buffalo hump, central adiposity, truncal obesity (thin extremities), PUD or GI bleeding, acne, moon face, bruises and petechiae;
    Graves: most common cause of hyperthyroidism: weight loss, clubbing of fingers, intolerance to heat, facial flushing, tachycardia, tremors; *everything speeds up except for weight; bulging eyes (exopth.): do NOT go away; goiter; Thyroid Storm – crisis precipitated by stress, trauma, infection, or thyroidectomy. High fever, tachycardia, CHF, angina, agitations, delirium
    Hypothyroidism: myxedema coma: medical crisis: CO2 retention, F/E imbalance, and hypothermia; intolerance to cold, fatigue, bradycardia, *everything slows down except for weight
    Hashimoto's: autoimmune disorder, hypothyroidism

20. Inflammation and accompanying consequences in a burn patient
    Partial thickness burns: damage the epidermis and may involve the upper dermis; usually appear red and painful (sunburn. mild scald)
    Deep partial thickness: involves the destruction of the epidermis and part of the dermis; area is red, edematous, blistered, and often hypersensitive and painful during the inflammatory stage
    Full thickness burns: result in destruction of all skin layers and often underlying tissues as well
    Burn victims should increase intake of protein and carbs; use non-stick dressings; shock frequently follows due to fluid/protein loss and infection is a HIGH threat; there will be localized temperature increase and local inflammation with burns.

21. What major hormone does kidney make? What does it cause? Why will a patient in CKD often develop anemia?
    ? Aldosterone: (hormone from adrenal cortex) Increases reabsorption of Na and excretion of K; Controlled by renin-angiotensin-aldosterone system

CKD: decreased erythropoietin production= anemia: feel very weak and tired because of this;

22. What structures are in the kidney? Where do many diuretics work? What happens in glomerulonephropathy?
Major structures of the kidney:
1) Renal Capsule
2) Renal Parenchyma: Cortex & Medulla
3) Renal pyramid→renal calyx→renal pelvis
4) Ureters→bladder→urethra
5) Renal arteries
6) Renal veins
Diuretics work within the kidney tubules (inhibit reabsorption, therefore increasing urine output)
<u>Glomerulonephropathy</u>: any noninflammatory disease of the renal glomeruli;
<u>Glomerulonephritis</u>: inflammation of glomerular structures
Significance: leading cause of chronic renal failure in US
Etiology: exact cause unknown

23. What's the tie between DVT (VTE) and pulmonary embolism? Major s/s each.
<u>Pulmonary Embolism:</u> Occlusion of a pulmonary blood vessel by an embolus; Patients at risk: post-surgical patients, patients with arrhythmias; Recognition & prompt treatment of a PE is ESSENTIAL! Once a thrombus dislodges & becomes an emboli & lodges in a pulmonary vein, blood flow is obstructed, leads to: atelectasis, decreased perfusion, right-sided HF, and cardiopulmonary arrest;
S/S: tachypnea, dyspnea, chest pain, cough, hemoptysis, diaphoresis, anxiety, impending sense of doom; Diagnostic tests: D-dimer, V/Q scan, CT, pulmonary angiography
<u>Deep Vein Thrombosis:</u> DVT can move into pulmonary vasculature; the formation of a thrombus in association with inflammation in the vein; the most common disorder of the veins;
S/S: leg pain or swelling may occur but there may be no symptoms

24. What are the intrinsic rates for SA and AV nodes.
SA node: fires an impulse between 60-100bpm (depolarizes faster than other cells in the heart and is therefore responsible for pacing the heart)
AV node: 40-60bpm (important that the conduction is slower the further you go down the conduction system)

25. Know about ammonia, bilirubin, BUN, creatinine, uric acid, who gets rid of them, if they are elevated what conditions might they be associated with?
<u>Ammonia:</u> a nitrogen waste resulting from protein metabolism in the intestine or liver, is removed from the blood and converted to urea,

enabling it to be excreted by the kidneys; correlates with hepatic encephalopathy;
<u>BUN:</u> BUN stands for "blood urea nitrogen." Urea is a nitrogenous end product of protein metabolism. Urea is filtered by the kidneys and found in urine. The serum concentration of urea nitrogen serves as index of renal function. BUN test values are affected by protein intake, tissue breakdown, and fluid volume changes, however. (When elevated: not good kidney function) could be falsely elevated if dehydrated, exercised, or increased protein
<u>Creatinine:</u> endogenous waste product of skeletal muscle = filtered actively and passively at the glomerulus, passed thru tubules with little change, then excreted in the urine. Measuring creatinine clearance is a good measure of the glomerular filtration rate (GFR= rate of creating initial filtrate which is called "ultrafiltrate"); so it is a good measure of overall renal function. As renal function declines, creatinine clearance decreases indicating less filtration or removal of waste products
<u>Uric Acid:</u> Uric acid is a byproduct of purine metabolism (can accumulate in the case of gout).

26. Most numerous first responder WBC, which cells are lymphocytes, where do they mature?
Neutrophils: first responders
Lymphocytes: T cells and B cells; T cells: produced in bone marrow, mature in thymus; responsible for cell-mediated immunity ("direct attack"), types: helper T (CD4), cytotoxic T (CD8), suppressor T; B cells: produced in bone marrow, responsible for humoral immunity, when stimulated, become plasma cells which produce antibodies, they also produce memory cells (faster secondary response)

27. Amylase, lipase, ALT, AST - what are they, what organ do they come from?
ALT and AST are liver enzymes that are frequently assessed to determine liver function. ALT is liver specific, AST is also found in other organs. Elevated ALT and AST values are found in hepatitis, cirrhosis and other liver disorders.
Amylase: present in saliva, begins the chemical process of digestion;
Lipase: produced in pancreas, mouth, and stomach; used to break down fats

28. Difference between PAD and venous disease and acute arterial occlusion.
<u>Acute Arterial Occlusion:</u> Medical Emergency, acute onset
The Ps: Pain, Pallor, Pulseless, Polar (cold), Paresthesias, and Paralysis (Table from Cardiac Lecture)

	ARTERIAL	VENOUS

Pulses	**Weak or absent**	**Present**
Cap refill	**> 3 sec**	**< 3 sec**
Edema	**Minimal or absent**	**Present**
Hair	**No**	**Present or no**
Ulcers	**Toe tips, heels, lateral ankle**	**Medial ankle or lower leg**
Skin color	**Rubor, elevate and turns pale**	**Brown with varicose veins**
Skin Texture	**Thin, shiny**	**Thick, hardened**
	Intermittent claudication – pain with walking	

Arterial Insufficiency: Skin cool, shiny, onion-like, pain with cold, pale with elevation, distal pulses decreased or absent, decreased or absent hair, ischemic ulcers, thick nails
Venous insufficiency: dry, flaky (brown/blotchy), purple: dependent, elevation decreases dependent edema, edema may obliterate pulses, venous stasis ulcers, paresthesias

29. Remember the 5 Ps, intermittent claudication, which diseases cause legs to look which color.
Intermittent Claudication: pain with walking
6 P's? =Question 28; See table for color

30. Differences between asthma, chronic bronchitis, emphysema. restrictive versus obstructive, respiratory distress and failure
<u>Emphysema:</u> the destruction of the alveolar walls and septae, which leads to large, permanently inflated alveolar air spaces; onset is insidious and dyspnea occurs first on exertion and then progresses until it is marked even at rest; hyperventilation with a prolonged expiratory phase, use of accessory muscles, and hyperinflation leading to development of a "barrel chest"; anorexia and fatigue contributing to weight loss; clubbed fingers and secondary polycythemia may develop as compensations; air trapping; obstructive
<u>Asthma:</u> hyper-responsive airway disease; obstructive disorder; characterized by: inflammation, edema, and mucus hypersecretion; Bronchospasm: progresses, limits airflow; harder for pt to breath; has

to work a lot harder to get air in and out; increased use of accessory muscles; as inflammatory process progresses, will hear wheezing= air moving through inflamed bronchial, anxiety goes up, HR goes up
Extrinsic asthma: hypersensitivity (IgE mediated) Mast cells inflammatory mediators cause acute response within 10–20 minutes.
Intrinsic asthma: triggers: exercise, respiratory infections, aspirin/NSAIDS, inhaled irritants (smoke)
Chronic Bronchitis: increased mucus, obstruction small airways, and chronic productive cough; Inflammation and fibrosis of bronchial wall; main trigger: smoking; main symptom: productive cough; obstructive

31. First lecture ever - adaptation, homestasis, hypertrophy versus hyperplasia versus dysplasia, shock (also in cardiac lecture), factors that impair wound healing.
Homeostasis: stability or equilibrium; The body needs a relatively constant environment in order to function; self-regulated and occurs without thought on our part;
Hypertrophy: cells increase in size, due to increased demand; an increase in the muscle mass and cardiac wall thickness in response to overwork and strain; occurs slowly because it takes time for increased muscle tissues to develop; generally follows persistent or chronic dilation and thus further increases the contractile power of the muscle fibers
Hyperplasia: cells increase in number
Dysplasia: cells mutate into cells of different size, shape, and appearance. Example: cancer
Metaplasia: one type of cell will change into another type of cell. Example: different type of epithelial cell
Factors affecting wound healing: oxygenation, infection, age and sex hormones, stress, diabetes, obesity, medications, alcoholism, smoking, and nutrition

32. What's a fat embolism? What's compartment syndrome?
Fat embolism: A disruption to blood supply caused by fat globules in a blood vessel; fat tissue that passes into bloodstream and lodges within a blood vessel;
Compartment syndrome: A painful and dangerous condition caused by pressure buildup from internal bleeding or swelling of tissues. It impedes blood flow to and from the affected tissue; sever tissue damage can result;

33. Know your usual values for platelets, hemoglobin, hematocrit, what's a d-dimer, what's DIC, what's thrombocytopenia, what's leukocytosis, what's leukopenia, what's a normal potassium and sodium level
Platelets: 140,000-340,000/mm3 (another ppt: 150,00-400,000)

Hemoglobin: Women: 12-16mg/dl Men: 14-18mg/dl; values vary based on age and gender
Hematocrit: Women: 38-46% Men: 42-54%; values vary based on age and gender
Potassium: 3.5-5.3
Sodium: 135-145
Disseminated Intravascular Coagulation (DIC): severe failure to maintain homeostatic balance; usually increased clotting then increased bleeding; Associated with: Infection, malignancy, trauma, shock, burns, transfusion reactions, obstetric emergencies; treatment is controversial;
Thrombocytopenia: decreased number of platelets: increased bleeding; 3 main causes: Diminished Production, Altered Distribution, and Increased Destruction;
Leukocytosis: increase in number of white blood cells, especially during an infection; (Epstein Barr Virus)
Leukopenia: decrease in number of white blood cells (particularly T cells), immunocompromised patients; HIV/AIDS; immune system can not effectively respond;
D-Dimer: D-dimer tests are used to help rule out the presence of an inappropriate blood clot (thrombus);

34. What role do sodium and albumin have in blood volume?
    Where sodium goes, water goes; protein (albumin) pulls water;

35. Can you interpret an ABG? What will the patient look like who's in respiratory acidosis? Metabolic acidosis? (Respiratory rate wise)
    Respiratory acidosis: Causes: slow, shallow respirations; Headache, blurred vision, confusion, tremors
    Metabolic Acidosis: Rapid deep respirations (causes: shock, tissue hypoxia), coma, Kussmaul's respirations, hypotension
    Respiratory alkalosis: hyperventilation, pain and anxiety; paresthesia
    Metabolic alkalosis: vomiting, hypovolemia, slow shallow respirations; mental confusion, hyperactive reflexes
    ABG values:
    pH: 7.35-7.45
    PaCO2: 35-45 (a)
    HCO3: 22-26
    PaO2: 80-100