- ABG
    - Components of ABG:
        - pH: 7.35-7.45
        - HCO3: 22-26
        - CO2: 35-45
        - PaCO2: 80-100
        - SPO2: 95-100
    - Interpreting results (Acid-Base imbalances)
        - Look at practice questions
    - EKG findings from imbalances
        - Metabolic acidosis:
            - Hyperkalemia:
                - Peaked T waves
        - Metabolic alkalosis:
            - Hypokalemia:
                - U wave present
        - Respiratory acidosis:
            - Hypokalemia:
                - U wave Present
        - Respiratory alkalosis:
            - Hypocalcemia:
                - Lengthened QT, shortened QRS
            - Hypokalemia:
                - U waves
- Respiratory Alkalosis
    - Nursing interventions:
        - This is due to hyperventilation
        - Lightheadedness, inability to concentrate, numbness and tingling, loss of consciousness.
        - Correct the cause of hyperventilation
- Septic shock: Circulatory shock state resulting from overwhelming infection causing relative hypovolemia.
    - Assessment findings:
        - Sepsis that has circulatory, cellular and metabolic abnorms.
        - Those who require vasopressors to maintain a MAP of >65 and have a lactate of <2
        - RR: >22
        - GCS: <15
        - Temp: >38.3
        - SBP: <100
        - HR: >90
        - WBC: >12,000
        - Characterized by persistent hypotension despite inadequate fluid resuscitation, vasodilation, maldistribution and myocardial depression.

- Nursing interventions:
  - Sepsis: Secure dilation and correct hypoxemia, establish venous, CBC, lactate, ABGs, blood cultures, imaging targeting at the suspected site of infection, procalcitonin, aggressive IVG 30 mL/kg in the first 3 hours, antibiotic within first hour.
  - Shock: IV vasopressor (norepinephrine), glucocorticoids if shock is refractory to adequate fluid resuscitation and vasopressor administration.

- MODS
  - Nursing interventions:
    - Aimed at supporting patient and monitor closely.
    - Provide info and support to the family, address end of life decisions.
    - If they survive talk about goals and expectations to progress to those goals
  - Priority:
    - Priority is to prevent and detect early.
    - If prevention fails treat measures to reverse are aimed at controlling the initiating event, promoting adequate organ perfusion, providing nutritional support and maximizing comfort.
  - S/Sx:
    - Resp: severe dyspnea, tachypnea , PaO2/FiO2 <200, bilateral fluffy filtrates, V/Q mismatch, refractory hypoxemia.
    - Cardio: myocardial depression, massive vasodilation, decrease in SVR, BP and MAP, increase in HR, bivent failure.
    - CNS: acute neuro changes, fever, seizure, failure to wean.
    - Endo: hypoglycemia.
    - Renal: pre: BUN/Cr >20:1
      - Intra: BUN/Cr <10:1
    - GI: Hypoperfusion (decrease in peristalsis and paralytic ileus), GI bleeds
    - Hepatic: Bili >2, increased LFTs, encephalopathy.
    - Hematology: Coagulation (increase PT/PTT), increased D-Dimer
- Cardiogenic shock
  - Causes:
    - Coronary:
      - Acute MI
    - Noncoronary:
      - Severe hypoxemia, tension pneumo, cardiomyopathies, valvular stenosis or regurgitation, cardia tamponade, dysrhythmias, blunt traumas.
  - Nursing interventions:
    - Limit further damage, preserve healthy myocardium, increase cardiac function, increase O2 supply, prevent cardiogenic shock, monitor hemodynamics, administer meds/IVF, maintain intra-aortic balloon counterpressure, ensure safety and comfort.

        - Meds: Dobutamine, nitro, dopamine, vasoclusive meds, antiarrhythmics.
    - Treatment goals:
        - Limit further damage, preserve healthy myocardium, increase cardiac function by increasing cardiac contractility, decrease ventricular afterload or both, increase O2 supply.
- Anaphylactic shock
    - Treatment:
        - Always prevent first
        - Patent airway (ETT or cricothyroidotomy)
        - IM epinephrine is drug of choice
        - Diphenhydramine and ranitidine (zantac) are given as adjunctive
        - Nebulized bronchos
        - Fluid restriction
- Neurogenic shock
    - Manifestations:
        - Occur within 30 minutes of a spinal injury and can last up to 6 weeks
        - Loss of SNS vasoconstrictor tone
        - Massive vasodilation -> pooling of blood -> tissue hypoperfusion -> impaired circular metabolism
        - Hypotension and bradycardia make this different form others
- Hypovolemic shock
    - Manifestations:
        - Increase HR, CO, RR
        - Decreased BP
        - Anxious
        - Decreased urine output
        - SV, CVP, PAWP are decreased due to decreased circulating blood volume
    - Resuscitation fluid options:
        - Crystalloid solutions (LR or NS) are commonly used to treat hypovolemic shock
        - If hypovolemia d/t blood loss → administer 3 mL of crystalloid solution for each mL of EBL (3:1)
        - Colloid solutions (e.g., albumin) may also be used
        - Blood productions (also colloids) may need to be administered if cause is hemorrhage
            - Decision to give blood is based on pt's lack of response to crystalloid resuscitation, the volume of blood lost, the need for hemoglobin to assist with oxygen transport, and the necessity to correct the patient's coagulopathy
            - Pts who receive massive blood transfusions to achieve near-normal hemoglobin levels tend to have poorer outcomes than those with low hemoglobin levels (e.g., less than 7 g/dL)

        - PRBCs are administered to replenish the patient's oxygen-carrying capacity in conjunction with other fluids that will expand volume
        - Need for transfusions is based on pt's oxygenation needs, which are determined by vital signs, blood gas values, chemistry, coags, and clinical appearance rather than an arbitrary laboratory values
- Stages of shock
    - Manifestations:
        - Compensatory:
            - BP is normal, HR is >100, RR is >20 BPM/PaCo2 <32, skin is cold and clammy, urinary output is decreased, confused and agitated, in respiratory alkalosis
            - Inadequate perfusion = Anaerobic metabolism = buildup of lactic acid producing metabolic acidosis
            - Increased RR in response to need to increase oxygen & to compensate for metabolic acidosis
            - Pt may experience change in affect, express feeling anxious, or be confused
            -
        - Progressive:
            - Systolic is <90, MAP <65, requires fluid resuscitation, HR >150, RR is shallow and rapid, crackles, PaO2 <80, PaCO2 >45, skin is mottled and has petechiae, urinary output is less than 0.5 mL, lethargic, metabolic acidosis.
            - Resp: Respirations are rapid and shallow, crackles are heard over the lung fields
                - Decreased pulmonary blood flow causes arterial oxygen levels to decrease and CO2 levels to increase
                - Hypoxemia and biochemical mediators cause an intense inflammatory response and pulmonary vasoconstriction, perpetuating pulmonary capillary hypoperfusion and hypoxemia
                - Hypoperfused alveoli stop producing surfactant and subsequently collapse
                - Pulmonary capillaries begin to leak, causing pulmonary edema, diffusion abnormalities (shunting), and additional alveolar collapse → this condition is called acute lung injury (ALI)
                - Interstitial inflammation and fibrosis are common consequences, leading to acute respiratory distress syndrome (ARDS)
            - CV: A lack of adequate blood supply leads to dysrhythmias and ischemia, HR is rapid, sometimes exceeding 150 bpm
                - May complain of chest pain and even suffer a myocardial infarction (MI)

        - Levels of cardiac enzymes and biomarkers (e.g. troponin) increase
        - Myocardial depression and ventricular dilation may further impair the heart's ability to pump enough blood to the tissues to meet oxygen requirements
    - Neuro: Blood flow to brain becomes impaired (cerebral hypoperfusion) & mental status deteriorates
        - Initially may exhibit subtle changes in behavior (agitation and confusion), later may become lethargy & begin to lose consciousness
    - Renal: MAP falls <65 mmHg = GFR drops
        - Acute kidney injury (AKI) → increase BUN & Cr, fluid & electrolyte shifts, acid-base imbalances, & loss of renal-hormonal regulation of BP
        - Urinary output decreases to <0.5 mL/kg per hour (or <30 mL per hour)
    - Hepatic: Decreased blood flow to liver impairs ability of liver cells to perform metabolic and phagocytic functions; consequently, patient less able to metabolize medications and metabolic waste products, such as ammonia and lactic acid.
        - Pt more susceptible to infection as liver fails to filter bacteria from blood
        - Liver enzymes (AST, ALT, LDH), and bilirubin levels are elevated, and pt develops jaundice
    - GI: GI ischemia can cause stress ulcers in stomach = risk for GI bleeding
        - In small intestine, mucosa can become necrotic and slough off, causing bloody diarrhea
        - GI ischemia leads to bacterial toxin translocation, in which bacterial toxins enter the bloodstream through the lymphatic system
        - Net result is interference with healthy cellular functioning and ability to metabolize nutrients
    - Hemotological: Inflammatory cytokines activate the clotting cascade, causing deposition of microthrombi in multiple areas & consumption of clotting factors
        - Disseminated intravascular coagulation (DIC)
        - Bruises (ecchymoses) and bleeding (petechiae) may appear
        - Coagulation times (e.g., PT, aPTT) are prolonged
- Irreversible:
    - Mechanical or pharmological support needed for BP, HR is irrative, need intubation, mechanical ventilation, manual ventilation, jaundice, anuric and needing dialysis, unconscious, profound acidosis.

- Family education:
  - Families may be overwhelmed and frightened
  - Make sure family is comfortably situated and kept informed about pt's status
  - Encourage rest and inform that they will be notified of any significant changes in pt's status
  - Offer visit from hospital chaplain

- Shock
  - Manifestations:
    - Low BP
    - High HR
    - High RR
  - Interventions:
    - Fluid resuscitation
    - Medication administration
    - Blood administration
    -
  - Key assessments:
    - HR, BP, RR, CO

- Fluid volume deficit
  - Manifestations:
    - Causes include:
      - abnormal fluid losses (V/D, GI suctioning, sweating)
      - decreased intake (nausea or lack of access to fluids)
      - 3rd space fluid shifts
      - movement of fluid from vascular system to other body spaces
        - Examples: edema formation in burns, ascites with liver dysfunction
    - S/Sx: Acute weight loss, ↓ skin turgor, oliguria, concentrated urine, capillary filling time prolonged, low CVP, ↓ BP, flattened neck veins, dizziness, weakness, thirst and confusion, ↑ pulse, muscle cramps, sunken eyes, nausea, increased temperature; cool, clammy, pale skin
    - Labs indicate: ↑ hemoglobin and hematocrit, ↑ serum and urine osmolality and specific gravity, ↓ urine sodium, ↑ BUN and creatinine, ↑ urine specific gravity and osmolality
    - Contributing Factors: Loss of water and electrolytes via:
      - Vomiting
      - Diarrhea
      - Fistulas
      - Fever
      - Excess sweating
      - Burns
      - Blood loss
      - GI suction

        - Third-space fluid shifts
        - Decreased intake via:
        - Anorexia
        - Nausea
        - inability to gain access to fluid
        - Diabetes insipidus and uncontrolled diabetes both contribute to a depletion of extracellular fluid volume.
    - Diagnostic findings:
        - BUN & its relation to serum Cr concentration
            - Volume-depleted patient has BUN elevated out of proportion to serum Cr (ratio greater than 20:1)
        - Hematocrit (Hct)
            - Elevated d/t decreased plasma volume
        - Serum electrolyte changes may also exist
            - Hypokalemia & hyponatremia or hyperkalemia, hypernatremia
            - Hypokalemia w/ GI & renal losses
            - Hyperkalemia w/ adrenal insufficiency
            - Hyponatremia w/ increased thirst & ADH release
            - Hypernatremia w/ increased insensible losses & DI
        - Urine specific gravity
            - Increased d/t kidneys' attempt to conserve water and decreased w/ DI
        - Urine sodium
            - Aldosterone is secreted when fluid volume is low causing reabsorption of sodium and chloride = decreased urinary sodium
        - Urine osmolality
            - >450 mOsm/kg because the kidneys try to compensate by conserving water
- Fluid volume overload
    - Manifestations:
        - Acute weight gain, peripheral edema and ascites, distended jugular veins, crackles, elevated CVP, shortness of breath, ↑ BP, bounding pulse and cough, ↑ respiratory rate, ↑ urine output
        - Labs indicate: ↓ hemoglobin and hematocrit, ↓ serum and urine osmolality, ↓ urine sodium and specific gravity
        - Compromised regulatory mechanisms:
            - renal failure
            - heart failure
            - Cirrhosis
            - Overzealous administration of sodium-containing fluids
            - Fluid shifts (i.e., treatment of burns)
            - Prolonged corticosteroid therapy
            - Severe stress
            - Hyperaldosteronism

        - BUN and hematocrit levels
            - Both decreased d/t plasma dilution, low protein intake, and anemia
        - Serum osmolality
            - Decreased owing to excessive retention of water
        - Urine sodium
            - Increased if the kidneys are attempting to excrete excess volume
        - Hypervolemia occurs when aldosterone is chronically stimulated (i.e., cirrhosis, heart failure, and nephrotic syndrome); therefore, the urine sodium level does not increase in these conditions.
        - CXR
        - May reveal pulmonary congestion
    - Nursing interventions:
        - Measure I&O
        - Daily weights
        - Gain of 1 kg (2.2 lb) = gain of approx 1L fluid
        - Assess breath sounds
        - Monitor degree of edema in dependent parts of body (i.e. – feet, ankles, & sacral region)
        - Pharmacologic therapy
            - Diuretics
            - Thiazide diuretics (HCTZ or chlorthalidone) block sodium reabsorption in distal tubule
            - Loop diuretics (furosemide, bumetanide, or torsemide) cause greater loss of both sodium and water because they block sodium reabsorption in the ascending limb of Henle's loop
            - Generally, thiazide diuretics, such as are prescribed for mild to moderate hypervolemia and loop diuretics for severe hypervolemia
            - Electrolyte imbalances may result from diuretic use
            - Dialysis
            - If renal function is so severely impaired that pharmacologic agents cannot act efficiently → hemodialysis or peritoneal dialysis to remove nitrogenous wastes and control potassium and acid–base balance, and to remove sodium and fluid
            - Continuous renal replacement therapy (CRRT) may also be required
        - Nutritional therapy
            - Dietary restriction of sodium
            - Avg daily diet NOT restricted in Na+ contains 6-15g of salt
            - Low-sodium diets as little as 250mg of sodium/day
            - May range from light salting to no addition of salt to commercially prepared foods that are already seasoned
            - Avoid foods high in salt

            - Seasoning substitutes → lemon juice, onions, and garlic are excellent substitute flavorings
            - Salt substitutes → contain K+ and must therefore be used cautiously by patients taking potassium-sparing diuretics (e.g., spironolactone, triamterene, amiloride) or NOT AT ALL w/ any condition associated w/ K+ retention (ie – advanced renal disease)
    - Complications:
        - Pericarditis
        - Heart failure
        - Delayed wound healing
        - Tissue breakdown
        - Decreased bowel function
        - Edema
        - Third spacing
- Normal Electrolyte Ranges
    - Sodium: 135-145
    - Potassium: 3.5-5.5
    - Calcium: 8.5-10
    - Mag: 1.3-2.3
    - Chloride: 98-107
    - Phosphate: 2.5-4.5
- Electrolyte imbalances
    - General risk factors
        - Hyponatremia:
            - Loss of sodium containing fluids (draining wounds, V/D, primary adrenal insufficiency), water excess in relation to amount of sodium, SAIDH, combo of all
        - Hypernatremia:
            - Inadequate water intake, excess water loss or rarely sodium gain
        - Hypokalemia:
            - GI losses, medications, alterations of acid-base balance, hyperaldosterism, poor dietary intake
        - Hyperkalemia:
            - Usually treatment related, impaired renal function, hyperaldosteronism, tissue trauma, acidosis
        - Hypocalcemia:
            - Hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications
        - Hypercalcemia:
            - Malignancy and hyperparathyroidism, bone loss related to immobility
        - Hypomagnesia:

            - Alcoholism, GI losses, enteral or parenteral feeding deficient in magnesium, medications, rapid administration of citrated blood, contributing causes include diabetic ketoacidosis, sepsis, burns, hypothermia
        - Hypermagnesia:
            - Renal failure, diabetic ketoacidosis, excessive administration of magnesium
        - Hypophosphate:
            - Alcoholism, refeeding of patient after starvation, pain, heat stroke, respiratory alkalosis, hyperventilation, diabetic ketoacidosis, hepatic encephalopathy, major burns, hyperparathyroidism, low magnesium, low phosphate, diarrhea, vitamin D def, use of diuretics and antacids
        - Hyperphosphate:
            - Renal failure, excess phosphorus excess vitamin D, acidosis, hypoparathyroidism, chemo
    - Understand which elevated or decreased values can cause severe conditions
        - Sodium:
            - Hypo: Neurologic changes
            - Hyper: neuro
        - Potassium:
            - Hypo: Dysrhythmias
            - Hyper: Respiratory impairment, muscle weakness
        - Calcium:
            - Hypo: Hyperactive DTRs, trousseau's, chovsteks, seizures, respiratory impairment
            - Hyper: ECG changes, dysthymias
        - Magnesium:
            - Hypo: ECG changes, LOC, neuromuscular irritability
            - Hyper: Low BP, ECG changes, dysrhythmias
        - Phosphate:
            - Hypo: neuro changes, tissue hypoxia
            - Hyper: n/a
        - Chloride:
            - Hypo: dysrthymias, seizures, coma
            - Hyper: respiratory troubles, cognitive changes
- Sodium imbalances (hypo/hyper)
    - Manifestations:
        - Hypo: poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, abdominal cramping, neurologic changes
        - Hyper: thirst; elevated temperature; dry, swollen tongue; sticky mucosa; neurologic symptoms; restlessness; weakness
            - Note: thirst may be impaired in elderly or the ill
- Potassium imbalances (hypo/hyper)

- Manifestations:
  - Hypo: fatigue, anorexia, nausea, vomiting, dysrhythmias, muscle weakness and cramps, paresthesias, glucose intolerance, decreased muscle strength, DTRs
  - Hyper: cardiac changes and dysrhythmias, muscle weakness with potential respiratory impairment, paresthesia, anxiety, GI manifestations
- Nursing interventions:
  - Hypo: assessment, severe hypokalemia is life-threatening, monitor ECG and ABGs, dietary potassium, nursing care related to IV potassium administration

- Hyper: assessment of serum potassium levels, mix IVs containing K^+ well, monitor medication affects, dietary potassium restriction/dietary teaching for patients at risk
  - Hemolysis of blood specimen or drawing of blood above IV site may result in false laboratory result
  - Salt substitutes, medications may contain potassium
  - Potassium-sparing diuretics may cause elevation of potassium
    - Should not be used in patients with renal dysfunction
  - Treatment:
    - Hyper:
      - Diuretics
      - Calcium Gluconate
      - Insulin and glucose
      - Sodium bicarb
      - Beta-2 adrenergic
    - Hypo:
      - Potassium chloride
        - Preferred
      - Raises serum potassium concentration at a faster rate
      - Oral can be given in crystalline form (salt substitutes), as a liquid, or in a slow-release table or capsule
      - IV route → pain and phlebitis during infusion, primarily occurs at rate >10mEq/hr
      - Must use an infusion pump
      - Increase intake of potassium-rich foods
      - Highest → dried figs, molasses, seaweed
      - Very High → dried fruits, nuts, avocados, wheat germ, lima beans
      - High → spinach tomatoes, broccoli, beets, carrots, cauliflower, potatoes, bananas, cantaloupe, kiwis, oranges, mangos, ground beef, steak, pork, veal, lamb
      - If continued potassium losses → rate of potassium administration must be increased by the rate of potassium loss to produce the desired rate of potassium repletion
  - Patient education:

        - Hyper:
            - Decrease the amount of potassium you get in your diet
        - Hypo:
            - Increase the amount of potassium in your diet
    - EKG changes:
        - Hyper: ST depression, Peaked T wave
        - Hypo: ST depression, U wave
- Calcium imbalances (hypo/hyper)
    - Manifestations:
        - Hypo:
            - tetany, circumoral (around the mouth) numbness, paresthesia, hyperactive DTRs, Trousseau’s sign, Chovstek's sign, seizures, respiratory symptoms of dyspnea and laryngospasm, abnormal clotting, anxiety
        - Hyper:
            - muscle weakness, incoordination, anorexia, constipation, nausea and vomiting, abdominal and bone pain, polyuria, thirst, ECG changes, dysrhythmias
    - EKG changes:
        - Hypo:
            - K: T waves becomes wider, ST segment depression, P wave increase, U waves, flat T waves,
            - Ca:Lengthened QT, Shortened QRS
        - Hyper:
            - K: Earliest sign is pointed T waves, moderate P wave decreases, QRS becomes wider
            - Ca: Shortened QT, Lengthened QRS, bradycardia