Exam 2 Concept Review: Shannon O'Malley

S/S of Hypovolemic Shock (MOST COMMON TYPE):

- Shock state resulting from decreased intravascular volume due to fluid loss
    - External and internal fluid loss example:
        - EXTERNAL: trauma, surgery, vomiting, diarrhea, diuresis, diabetes insipidus
        - INTERNAL: Hemorrhage, burns, ascites, peritonitis, dehydration, necrotizing pancreatitis

DIC Patho:

- Not an actual disease but a sign of an underlying condition
    - May be triggered by sepsis, trauma, cancer, shock, abruptio placentae, toxins, and allergic reactions
    - Potentially life threatening (mortality rate is 80%)
    - Excessive clotting and bleeding
- The inflammatory response generated by the underlying disease initiates the process of inflammation and coagulation within the vasculature
- Natural anticoagulant pathways in the body all become impaired and causes a massive amount of tiny clots that form in the microcirculation
- Bleeding is characterized by low platelet and fibrinogen levels, prolonged PT, aPPT, and thrombin time, elevated fibrin degradation products and D-dimers

DIC S/S:

- Bleeding from mucous membranes, venipuncture sites, and GI and urinary tracts
- Decreased temperature, increased pain, decreased pulses, hypoxia, decreased urine output, increased creatinine, and increased BUN, decreased alertness and orientation
- Bleeding can range from minimal occult internal bleeding to profuse hemorrhage from all orifices
- Typically develop MODS
- Initial phase: only manifestation may be decrease in platelet count
- The patient experiences S/S of thrombosis in the organs involved
- As clotting factors and platelets are consumed to form thrombi, bleeding occurs
- initially subtle but can develop into frank hemorrhage

DIC labs:

- Decreased platelet count
- increased PT time
- Increased aPTT time
- Increased Thrombin time
- Decreased fibrinogen
- Increased D-dimer level
- Increased Fibrin degradation products

ABGs

- pH 7.35–(7.4)–7.45
- PaCO2 35–(40)–45 mm Hg
- HCO3- 22–(24)–26 mEq/L
- PaO2 80–100 mm Hg

Disorder	Initial Event	Compensation
Respiratory acidosis	↓ pH, ↑ or normal HCO3–, ↑ PaCO2	↑ Renal acid excretion (↑ PaCO2) and ↑ serum HCO3– >26 mEq/L
Respiratory alkalosis	↑ pH, ↓ or normal HCO3–, ↓ PaCO2	↓ Renal acid excretion (↓ PaCO2) and ↓ serum HCO3– <21 mEq/L
Metabolic acidosis	↓ pH, ↓ HCO3–, ↓ or normal PaCO2	Hyperventilation with resulting ↓ PaCO2 (>45 mm Hq) ↓ HCO3
Metabolic alkalosis	↑ pH, ↑ HCO3–, ↑ or normal PaCO2	Hypoventilation with resulting ↑ PaCO2 (<35 mm Hq) ↑ HCO3

Electrolyte imbalances S/S

- ★ Hyponatremia: poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, abdominal cramping, neurologic changes and CONFUSION
- ★ Hypernatremia: Thirst, elevated temperature, dry, swollen tongue, sticky mucosa, neurologic symptoms, restlessness, weakness
- ★ Hypokalemia: Fatigue, anorexia, nausea, vomiting, dysrhythmias, muscle weakness and cramps, paresthesias, glucose intolerance, decreased muscle strength and DTR's
- ★ Hyperkalemia: cardiac changes and dysrhythmias, muscle weakness with potential respiratory impairment, paresthesias, anxiety, GI manifestations
- ★ Hypocalcemia: Tetany, circumoral numbness, paresthesias, hyperactive DTR, Trousseau's sign, chvostek sign, seizures, respiratory symptoms of dyspnea and laryngospasm, abnormal clotting anxiety
- ★ Hypercalcemia: muscle weakness, incoordination, anorexia constipation, nausea and vomiting, abdominal and bone pain, polyuria, thirst, ECG changes, dysrhythmias
- ★ Hypomagnesemia: neuromuscular irritability, muscle weakness, tremors, athetoid movements, ECG changes, alterations in mood and LOC
- ★ Hypermagnesemia: Flushing, lowered BP, nausea, vomiting, hypoactive reflexes, drowsiness, muscle weakness, depressed respirations, ECG changes, dysrhythmias
- ★ Hypophosphatemia: neurologic symptoms, confusion, muscle weakness, tissue hypoxia, muscle and bone pain, increased susceptibility to infection
- ★ Hyperphosphatemia: few symptoms, soft tissue calcifications, symptoms occur due to associated hypocalcemia

- ★ Hypochloremia: agitation, irritability, weakness, hyperexcitability of muscles, dysrhythmias, seizures, coma
- ★ Hyperchloremia: tachypnea, lethargy, weakness, rapid deep respirations, hypertension, cognitive changes

Electrolyte imbalances causes

- ★ Hyponatremia: Sodium less than 135
    - Loss of sodium containing fluids (draining wounds, V/D, primary adrenal insufficiency) water excess in relation to amount of sodium (dilutional hyponatremia such as renal failure, SIADH, psychiatric disorders) or combo of both
- ★ Hypernatremia: Sodium greater than 145
    - Inadequate water intake, excess water loss, or rarely sodium gain
- ★ Hypokalemia: below 3.5
    - GI losses, medications, alterations of acid-base balance, hyperaldosteronism, poor dietary intake
- ★ Hyperkalemia: above 5.0
    - Usually treatment related, impaired renal function, hypoaldosteronism, tissue trauma, and acidosis
- ★ Hypocalcemia: less than 8.6
    - Hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications
- ★ Hypercalcemia: malignancy and hyperparathyroidism, bone loss related to immobility
- ★ Hypomagnesemia: less than 1.3
    - alcoholism, GI losses, enteral or parenteral feeding deficient in mag, medications, rapid admin of citrated blood, contributing causes include DKA, sepsis, burns, hypothermia
- ★ Hypermagnesemia: greater than 2.3
    - Renal failure, DKA, excessive admin of mag
- ★ Hypophosphatemia: below 2.5
    - alcoholism, refeeding after starvations, pain, heat stroke, respiratory alkalosis, hyperventilation, DKA, hepatic encephalopathy, major burns, hyperparathyroidism, low mag, low potassium, diarrhea, vitamin D deficient, use of lasix and antacids
- ★ Hyperphosphatemia: above 4.5
    - renal failure, excess phosphorus, excess vitamin D, acidosis, hypoparathyroidism, chemo
- ★ Hypochloremia: less than 97
    - Addison's disease, reduced chloride intake, GI loss, DKA, excessive sweating, fever, burns, medications, metabolic alkalosis
- ★ Hyperchloremia: above 107
    - excess sodium chloride infusions with water loss, head injury, hypernatremia, dehydration, severe diarrhea, respiratory alkalosis, metabolic acidosis, hyperparathyroidism, medications

S/S of Hypovolemia:

- acute weight loss, decreased skin turgor, oliguria, concentrated urine, prolonged capillary refill, low CVP (pressure in preload of heart), decreased BP, flattened neck veins, dizziness, weakness, thirst and confusion, increased pulse, muscle cramps, sunken eyes, nausea, increased temperature, cool, clammy, pale skin

Blood products/fluids in hypovolemic shock:

- Crystalloid solutions (LR or NS) are used to treat shock
    - If hypovolemia d/t blood loss -> administer 3 mL of crystalloid solution for each mL of estimated blood loss (3:1)
    - Colloid solutions (albumin) can be used
        - 2 large- gauge IV lines are inserted to allow simultaneous administration of fluid, medications, and blood
        - IO used for quick access in the sternum, legs, or arms to facilitate rapid fluid replacement
        - Administer fluids that will remain in the intravascular compartment to avoid fluid shifts from intravascular compartment into the intracellular compartment

Types of fluids:

- Intracellular space (fluid in the cells)
- Extracellular space (fluid outside the cells)
- Interstitial fluid (fluid in spaces between cells)
- Intravascular fluid (plasma)

Pulse pressure and what it indicates:

- Tissue and organ perfusion depend on MAP, or the average pressure at which blood moves through the vasculature
- MAP: should be equal to or greater than 65
- BP is regulated by baroreceptors (pressure receptors)

Anaphylactic shock medications:

- Norepinephrine- causes peripheral vasoconstriction and bronchodilation and opposed the effect of histamine
- Diphenhydramine and ranitidine are given as adjunctive therapies to block the ongoing release of histamine from allergic reactions
- Nebulized bronchodilators
- Fluid resuscitation

Hypervolemia S/S:

- Acute weight gain, peripheral edema, ascites, distended jugular veins, crackles, elevated CVP, shortness of breath, increased BP, bounding pulse and cough, increased respiratory rate, increased urine output

Stages of shock and S/S:

- Shock progresses along a continuum and can be identified as early or late, depending on the S/S
- Earlier interventions equals higher rate of survival, initiate aggressive therapy within 3 HOURS of identifying shock for best outcomes
- Stage 1: Initial- no visible changes in client parameters, only changes on cellular level
- Stage 2: Compensatory (Non- progressive)- measures to increase CO to restore tissue perfusion and oxygenation
    - Causes vasoconstriction, increased HR, increased heart contractility
        - Maintains BP and cardiac output
    - Body shunts blood from skin, kidneys, GI tract to the heart, lungs, and brain to maintain blood supply of these vital organs
        - Results in cool, clammy skin, hypoactive bowel sounds, decreased urine output
    - Inadequate perfusion of tissues, acidosis occurs, respiratory rate increases due to acidosis, confusion may occur
- Stage 3: Progressive- Compensatory mechanisms begin to fail
    - Mechanisms that regulate BP can no longer compensate so BP drops
        - All organs suffer from hypoperfusion, vasoconstriction continues further compromising cell perfusion
        - Mental status further deteriorates from decreased perfusion and hypoxia
    - Lungs begin to fail, rapid shallow respirations, hypoxemia, increased CO2, alveoli collapse, and pulmonary edema occurs
        - Inadequate perfusion of the heart leads to dysrhythmias and ischemia
        - When MAP falls below 70, GFR can't be maintained
    - Liver function, GI function, and hematological function all affected
- Stage 4: Refractory- Irreversible shock and total body failure
    - Severe organ damage and PT cannot survive
    - Erratic heart rate, intubation, jaundice, anuric, unconscious
        - BP remains low
        - Renal and liver function fail
        - Anaerobic metabolism worsens acidosis
        - Complete organ failure

Types of distributive shock:

- Disruptive shock: Shock state resulting from widespread vasodilation and increased capillary permeability
- Septic shock, neurogenic shock, and anaphylactic shock are all disruptive shock

DIC Treatment:

- Treat underlying condition
- Correct secondary effects of tissue ischemia
    - Improve oxygenation
    - Replace fluids
    - Correct electrolyte imbalances
    - Administer vasopressor medications
- If serious hemorrhage occurs
    - Cryoprecipitate: replace fibrinogen and factors V and VII
    - Platelets: correct severely low platelet levels and can control bleeding
        - Replaces coagulation factors but can exacerbate capillary leak, further compromising pulmonary function
- Controversial treatment!!: interrupt the thrombosis process through the use of a heparin infusion
    - Heparin may inhibit the formation of the microthrombi and thus permit perfusion of the organs to resume

MODS Nursing Management:

- General plan is the same as patients with septic shock
- Primary interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted
- providing information and support to family is a critical role of the nurse
    - Must address end of life decisions to ensure supportive therapies are the patient's wishes
- Patients who survive must know about rehab and the expectations for progress as the skeletal muscle mass makes rehab a long slow process

Cardiogenic Shock Nursing interventions:

- Preventing cardiogenic shock
- Monitoring hemodynamic status
- Administering medications, IV fluids
- Maintain/Monitor IABP
- Ensure safety and comfort

Neurogenic S/S and causes:

- Shock state resulting from loss of sympathetic tone causing relative hypovolemia
- S/S:
- Maintain BP, bradycardia, monitor for hypothermia d/t hypothalamic dysfunction

Medications:

- Norepinephrine: Vasopressor agent that increases blood pressure by vasoconstriction
- Epinephrine: Inotropic agent that improves contractility, increase stroke volume, and increase cardiac output
- Nitroglycerin: Vasodilator that reduces preload and afterload, reducing oxygen demand of the heart

- Dopamine: Inotropic medication that improves contractility, increase the stroke volume, and increase cardiac output
- Vasopressin: Vasopressor agent that increases blood pressure by vasoconstriction

Cardiogenic Shock Causes:

- Shock state resulting from failure of the heart to pump effectively due to a cardiac factor
    - Coronary and non coronary examples:
        - Coronary- Acute MI which results in damage to a significant portion of LV myocardium
        - Non-coronary- Severe hypoxemia, tension pneumo, cardiomyopathies, valvular stenosis, valvular regurg, cardiac tamponade, dysrhythmias, and blunt cardiac injury

What is MODS:

- Multiple organ dysfunction syndrome- failure of 2 or more organ systems in an acutely ill patient such that homeostasis cannot be maintained without intervention
    - complication of any form of shot
    - frequently occurs toward the end of the continuum of septic shock when tissue perfusion cannot be effectively restored
    - organ failure usually begins in the lungs

Please note there are several ABGs and electrolyte abnormality questions on this exam!