Exam 2 Concept Review:

1. S/S of Hypovolemic Shock: Decreased intravascular volume d/t fluid loss
   - Weak and thready pulse
   - Dehydration
   - Thirst
   - ↓urine output
   - Dizziness
   - ↓central venous pressure

2. DIC Patho
   - In DIC, normal hemostatic mechanisms are altered.
   - The inflammatory response generated by the underlying disease initiates the process of inflammation and coagulation within the vasculature
   - The natural anticoagulant pathways within the body are simultaneously impaired, and the fibrinolytic system is suppressed so that a massive amount of tiny clots forms in the microcirculation
   - Initially, the coagulation time is normal. However, as the platelets and clotting factors form microthrombi, coagulation fails
     - Thus, the paradoxical result includes excessive clotting and bleeding
   - The clinical manifestations of DIC are primarily reflected in compromised organ function or failure
   - Decline in organ function is usually a result of excessive clot formation (with resultant ischemia to all or part of the organ) or, less often, of bleeding
   - The excessive clotting triggers the fibrinolytic system to release fibrin degradation products, which are potent anticoagulants, furthering the bleeding
   - The bleeding is characterized by low platelet and fibrinogen levels; prolonged PT, aPTT, and thrombin time; and elevated fibrin degradation products and D-dimers
   - Mortality rate: 80%

3. DIC S/S
   - Integumentary (skin)
     - ↓Temperature, sensation; ↑pain; cyanosis in extremities, nose, earlobes; focal ischemia, superficial gangrene
     - Petechiae, including periorbital and oral mucosa; bleeding: gums, oozing from wounds, previous injection sites, around catheters (IVs, tracheostomies); epistaxis; diffuse ecchymoses; subcutaneous hemorrhage; joint pain
   - Circulatory/Respiratory
     - ↓Pulses: capillary refill >3 seconds; hypoxia (secondary to clot in lung); dyspnea; chest pain with deep inspiration; ↓breath sounds over areas of large embolism

    - Tachycardia; high-pitched bronchial breath sounds; tachypnea; ↑consolidation; s/s of acute respiratory distress syndrome
- Gastrointestinal
    - Gastric pain; "heartburn"
    - Hematemesis; melena; retroperitoneal bleeding (abdomen firm and tender to palpation; distended; ↑abdominal girth)
- Renal/Neurologic
    - ↓Urine output; ↑creatinine, ↑blood urea nitrogen; ↓alertness and orientation; ↓pupillary reaction; ↓response to commands; ↓strength and movement
    - Hematuria
    - Anxiety; restlessness; ↓mentation, altered LOC; headache; visual disturbances; conjunctival hemorrhage

4. DIC labs
    - Decreased platelet count
    - Increased PT time
    - Increased aPTT time
    - Increased Thrombin time
    - Decreased fibrinogen
    - Increased D-dimer level
    - Increased Fibrin degradation products (FDPs)
    - Thromboelastography, can be performed at the bedside and can better assess platelet function as well as fibrinolytic activity
    - International Society on Thrombosis and Hemostasis has developed a highly sensitive and specific scoring system (See next slide)

5. ABGs Electrolyte imbalances S/S
    - **↓Hyponatremia**
        - **Seizures,** HYPOtension
        - Decreased skin turgor
        - Dry mucosa
        - HA, Confusion, **Lethargy**
        - Tachycardia
        - Nausea, Fatigue
        - Neuro changes, L. Headed
        - **Increased ICP**
        - Abdominal cramping
        - Anorexia
        - Muscle twitching
        - Hyperactive bowel sounds

- **Hypernatremia**
  - Thirst
  - Elevated temperature
  - Dry, swollen tongue
  - Sticky mucosa
  - Restlessness
  - Weakness, Anorexia
  - Neuro symptoms – Cerebral Edema
  - Muscle Twitching
  - Coma, Decreased DTRs
  - Lethargy
  - Decreased Urine output

- **↓Hypokalemia**
  - Fatigue
  - Anorexia
  - N/V
  - Dysrhythmias
  - Muscle weakness and cramps
  - Paresthesias
  - Glucose intolerance
  - Decreased muscle strength
  - Decreased DTRs
  - HYPOtension
  - Thready, weak pulse

- **Hyperkalemia**
  - Cardiac changes and dysrhythmias
  - Muscle weakness with potential respiratory impairment
  - Parashesia/Paralysis
  - Anxiety
  - Diarrhea/Nausea

6. Electrolyte imbalances causes
   - **Normal Electrolyte Ranges:**
     - **(Na+) 135 – 145**
     - **(K+) 3.5 – 5**
     - **(Ca+) 9.0 – 10**
     - **(Mg) 1.3 – 2.1**
     - **(Phos) 3 – 4.5**
     - **(Cl-) 98 – 106**
   - **↓Hyponatremia (Neuro): Respiratory arrest; Seizures; Coma**
     - Diuretics
     - NG tube suction

- Hyperglycemia
- Kidney disease
- SIADH

- **Hypernatremia (Neuro):Convulsions; Death; Seizures**
  - Diabetes Insipidus
  - Kidney failure
  - Cushing's syndrome
  - Aldosteronism
  - Glucocorticosteroids

- **↓Hypokalemia (Cardiac): Respiratory failure; Cardiac arrest**
  - Vomiting/Diarrhea
  - NG suctioning
  - Excessive use of laxatives
  - Tap water enemas
  - Kidney disease
  - Diuretics
  - Corticosteroids

- **Hyperkalemia (Cardiac): Cardiac arrest**
  - Chronically ill patients
  - Decrease in renin & aldosterone
  - Increased use of salt substitutes
  - ACE inhibitors
  - K+ sparing diuretics
  - DKA
  - Sepsis, trauma, surgery, fever, MI

7. S/S of Hypovolemia
   - **Fluid volume deficit (Hypovolemia) – Think HIPPO = SLOWWWWWS down**
   - Manifestations
     - ↑**Tachy**cardia, ↓ **HYPO**tension
     - **↓Decreased skin turgor**
     - **Concentrated urine**
     - **Tachypnea**
     - **Low grade fever**
     - Acute weight loss
     - Cool, clammy, pale skin
     - Oliguria
     - Prolonged capillary refill
     - Flat neck veins
     - Dizziness

- Weakness
- Thirst + Confusion
- Muscle Cramps
- Sunken eyes
- Nausea

8. Blood products/fluids in hypovolemic shock
   - Resuscitation fluid options (During Hypovolemic Shock → **Replacing FLUIDS is #1 priority!!**)
     - Packed RBC's ** Most common given (hemorrhaging)
     - FFP
     - Platelets
     - 0.9% Nacl (crystalloid)
     - Lactated Ringers (LR) → best option when pt. is in metabolic acidosis
     - Albumin (colloids 5, 25%) → best option for burn patients

9. Types of fluids
   - Crystalloids
     - Contain the same concentration of electrolytes as the extracellular fluid
       - Does not alter the concentrations of electrolytes in the plasma
     - Fluids utilized in Shock
       - **0.9% NS**
       - **LR**
     - Disadvantage: some of the volume administered is lost to the interstitial compartment and some remains in the intravascular compartment; this occurs as consequence of cellular permeability that occurs during shock
     - Avoid both underresuscitating and overresuscitating the patient in shock
   - Colloids
     - Similar to plasma proteins, contain molecules that are too large to pass through capillary membrane
     - Expand intravascular volume by exerting oncotic pressure, thereby pulling fluid into the intravascular space
     - In addition, colloids have a longer duration of action than crystalloids, because the molecules remain within the intravascular compartment longer
     - Typically, **albumin** is the agent used
     - Disadvantage of albumin is its high cost compared to crystalloid solutions.
   - Blood components (PRBC's, FFP, & platelets)
     - Packed RBC's ** Most common given (hemorrhaging)
     - FFP
     - Platelets
     - 0.9% Nacl (crystalloid)

- Lactated Ringers (LR) → best option when pt. is in metabolic acidosis
- Albumin (colloids 5, 25%) → best option for burn patients

10. Pulse pressure and what it indicates
- Pulse pressure correlates well with stroke volume
  - Pulse pressure is calculated by subtracting diastolic measurement from systolic measurement; the difference is the pulse pressure: Normal 30-40 mmHg
  - Narrowing or decreased pulse pressure is an earlier indicator of shock than a drop in systolic BP
  - Elevation of diastolic BP w/release of catecholamines & attempts to increase venous return through vasoconstriction is an early compensatory mechanism in response to decreased stroke volume, BP, and overall cardiac output
  - **Note**: By the time BP drops, damage has already been occurring at the cellular and tissue levels!

11. Anaphylactic shock medication
- **Epinephrine IV/Pen (#1 first!!)**
  - Rapid acting medication that promotes effective oxygenation and used to treat anaphylactic shock (addresses ABC's)
- Diphenhydramine
  - Used to treat itching, rash, pruritis
- Albuterol (nebulized)
- Prednisone

12. Hypervolemia S/S
- ↑Tachycardia, ↑**HTN**
- **Dyspnea**
- **Weakness**
- **Edema**
- Bounding pulse
- ↑ RR
- SOB
- Acute weight gain
- Acites
- Distended jugular veins
- Cough
- Crackles
- ↑urine output

13. Stages of shock and S/S: Earlier interventions = an increased chance of survival. Initial aggressive therapy w/in 3 hrs of identifying shock for best outcomes.
    - Initial
        - No visible changes in client parameters, only changes on cellular level
    - Compensatory (non-progressive): Normal BP; HR >100 bpm; RR >20 breaths/min (PaCO2 <32 mmHg); Skin is cold & clammy; Decreased urinary output; Confusion & agitation; Respiratory alkalosis
        - Measures to increase CO to restore tissue perfusion and oxygenation
        - SNS causes vasoconstriction, increased HR, increased heart contractility
            - This 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
            - Resulting in cool, clammy skin, hypoactive bowel sounds, decreased urine output
        - Perfusion of tissues is inadequate
        - Acidosis occurs from anaerobic metabolism
        - Respiratory rate increases due to acidosis, may cause compensatory respiratory alkalosis
        - Confusion may occur
    - Progressive: Systolic <90 mmHg; MAP <65 mmHg & requires fluids resuscitation to support BP; HR >150 bpm; RR rapid, shallow breaths & crackles; PaO2 <80 mmHg; PaCO2 >45 mmHg; Skin is mottled & petechiae; Urinary output <0.5 mL/kg/hr; Lethargy; Metabolic acidosis
        - Compensatory mechanisms begin to fail
    - Refractory
        - Irreversible shock and total body failure

14. Types of distributive shock
    - Anaphylactic shock
    - Neurogenic shock
    - Septic shock

15. DIC Treatment
    - Treat underlying condition
    - Correct secondary effects of tissue ischemia by
        - 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
    - Caution w/ FFP
        - Replaces coagulation factors but can exacerbate capillary leak, further compromising pulmonary function
- Controversial treatment strategy can be used to interrupt the thrombosis process through the use of heparin infusion
- Heparin may inhibit the formation of microthrombi and thus permit perfusion of the organs (skin, kidneys, or brain) to resume
    - Prophylactic dose – prevent
    - Therapeutic dose – severe thrombosis
- Effectiveness of heparin
    - Normalization of plasma fibrinogen concentration
    - Diminished signs of bleeding

16. MODS Nursing Management
    - General plan of nursing care for patients with MODS is the same as that for patients with septic shock
    - Primary nursing interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted
    - Providing information and support to family members is a critical role of the nurse → health care team must address end-of-life decisions to ensure that supportive therapies are congruent with the patient's wishes
    - Patients who survive MODS must be informed about the goals of rehabilitation and expectations for progress toward these goals, b/c massive loss of skeletal muscle mass makes rehabilitation a long, slow process

17. Cardiogenic Shock Nursing interventions
- Goal: Increase oxygen supply to the heart muscles while reducing oxygen demands
- Oxygenation: supplemental oxygen
- Pain control
    - IV morphine (reduces workload of the heart by both decreasing cardiac filling pressure (preload) & reducing pressure against which heart muscle has to eject blood (afterload)
- Hemodynamic monitoring: arterial line
- Laboratory marker monitoring: BNP, cardiac profile
- Fluid Therapy
- Pharmacologic Therapy
    - Dobutamine
    - Nitroglycerin
    - Dopamine
    - Other vasoactive medications

    - Antiarrhythmic medications
- Nursing Management
    - Preventing cardiogenic shock
    - Monitoring hemodynamic status
    - Administering medications, IV fluids
    - Maintain/monitor IABP
    - Ensuring safety, comfort

18. Neurogenic S/S and causes
    - Hemodynamic phenomenon that can occur within 30 min of a spinal cord injury and can last up to 6 weeks
    - Injury results n massive vasodilation w/o compensation b/c of the loss of SNS vasoconstrictor tone
        - Remember that SNS stimulation causes vascular smooth muscle to constrict and PNS stimulation causes vascular smooth muscle to relax or dilate
    - Massive vasodilation → pooling of blood → tissue hypoperfusion → impaired cellular metabolism
    - Most important clinical manifestations are hypotension (from massive vasodilation) and bradycardia (from unopposed parasympathetic stimulation)
    - Can be caused by spinal cord injury, spinal anesthesia, or other nervous system damage; may also result from the depressant action of medications or from lack of glucose (e.g., insulin reaction)
    - Treatment based on cause
        - If spinal cord injury → promote spinal stability (spinal precautions, c-collar)
    - Maintain BP and organ perfusion: Use vasopressors
    - Bradycardia: Use atropine
    - Infuse fluids cautiously as cause of hypotension is not r/t to fluid loss
    - Monitor for hypothermia d/t hypothalamic dysfunction
    - Corticosteroids do not have an effect in neurogenic shock and current guidelines no longer recommend the use of methylprednisone for patients w/ spinal cord injury

19. Medications:
    - Norepinephrine
        - IV vasopressor
    - Epinephrine
    - Nitroglycerin
    - Dopamine
    - Vasopressin

20. Cardiogenic Shock Causes
- Decreased cardiac contractility
    - Decreased stroke volume and cardiac output
        - Pulmonary congestion
        - Decreased systemic tissue perfusion
        - Decreased artery perfusion

21. What is MODS – Multiple Organ Dysfunction Syndrome
    a. Failure of 2 or more organ systems in an acutely ill patient such that homeostasis cannot be maintained w/o intervention
    b. May be a complication of any form of shock
    c. Precise mechanism remains unknown → frequently occurs toward the end of the continuum of septic shock when tissue perfusion cannot be effectively restored
    d. It is not possible to predict which patients who experience shock will develop MODS, partly b/c much of the organ damage occurs at the cellular level and therefore cannot be directly observed or measured
    e. Organ failure usually begins in the lungs, and cardiovascular instability as well as failure of the hepatic, GI, renal, immunologic, and CNS follow
- Clinical assessment tools attempt to predicts pts at risk
    - APACHE (Acute Physiology and Chronic Health Evaluation)
    - SAPS (Simplified Organ Failure Assessment)
    - PIRO (Predisposing factors, the Infection, the host Response, and Organ dysfunction)
- Manifestations of MODS:
    - Respiratory
        - Severe dyspnea, tachypnea
        - PaO2/FiO2 ration <200
        - Bilateral fluffy infiltrates on CXR
        - V/Q mismatch
        - Refractory hypoxemia
    - Cardiovascular
        - Myocardial depression
        - Massive vasodilation
        - Decrease SVR, BP, MAP
        - Increase HR
        - Biventricular failure
    - Central Nervous System
        - Acute change in neurologic status → confusion, disorientation, delirium
        - Fever
        - Seizures
        - Failure to wean, prolonged rehabilitation
    - Endocrine System
        - Hyperglycemia
    - Renal System

        - Pre-renal
            - BUN/Creatinine rate >20:1
        - Intrarenal
            - BUN/Creatinine rate <10:1
    - Gastrointestinal System
        - Hypoperfusion → decrease peristalsis, paralytic ileus
        - GI bleeding
    - Hepatic System
        - Bilirubin >2, increased LFTs
        - Hepatic encephalopathy
    - Hematologic System
        - Coagulopathy (increased PT & PTT, decreased platelet count)
        - Increased D-dimer
- Clinical Manifestations
    - Typically lungs are first (progressive dyspnea & respiratory failure
    - Usually hemodynamically stable but may require increasing amounts of IV fluids and vasoactive agents to support BP and cardiac output
    - Signs of a hypermetabolic state, hyperlactic academia, and increased BUN are present
    - Metabolic rate may be 1.5-2x the BMR resulting in loss of skeletal muscle mass (autocatabolism)
    - 7-10 days later, hepatic and renal dysfunction occur
        - Increased Bilirubin and LFTs
        - Increased Creatinine and Anuria
    - As lack of tissue perfusion continues, the hematologic system becomes dysfunctional
        - Worsening immunocompromised
        - Increasing the risk of bleeding
    - CV system becomes unstable and unresponsive to vasoactive agents, and the patient's neurologic response progresses to a state of unresponsiveness or coma
    - The goal of all shock states is to reverse the tissue hypoperfusion and hypoxia
    - If effective tissue perfusion is restored before organs become dysfunctional, the patient's condition stabilizes
    - Along the septic shock continuum, the onset of organ dysfunction is a warning prognostic sign
        - The more organs that fail, the worse the outcome
- Medical Management
    - Prevention remains the top priority!
    - Early detection & documentation of initial signs of infection are essential
    - Subtle changes in mentation and a gradual rise in temperature are early warning signs
    - If preventative measures fail, treatment measures to reverse MODS are aimed at
        - Controlling the initiating event
        - Promoting adequate organ perfusion

        - Providing nutritional support
        - Maximizing patient comfort
- Nursing Management
    - General plan of nursing care for patients with MODS is the same as that for patients with septic shock
    - Primary nursing interventions are aimed at supporting the patient and monitoring organ perfusion until primary organ insults are halted
    - Providing information and support to family members is a critical role of the nurse → health care team must address end-of-life decisions to ensure that supportive therapies are congruent with the patient's wishes
    - Patients who survive MODS must be informed about the goals of rehabilitation and expectations for progress toward these goals, b/c massive loss of skeletal muscle mass makes rehabilitation a long, slow process

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