N441 Concept Review Exam 2

1. **Total water composition**
   1. Intracellular (ICF) – fluid in the cells
      a. Approximately 2/3 of fluid in body

   2. Extracellular (ECF) – fluid outside the cells
      a. Approximately 1/3 of fluid in body

      1. interstitial fluid – fluid in spaces between cells
      2. intravascular – plasma

   c. Third spacing:

   - Loss of ECF into a space that does not contribute to equilibrium between the ICF and ECF
   - Early evidence: decreased urine output
   - s/s: increased HR, decreased BP, decreased CVP, edema, increased body weight, imbalances in I/O
   - Occur in patients with hypocalcemia, decreased iron intake, severe liver diseases, alcoholism, hypothyroidism, malabsorption, immobility, burns, cancer

2. **Regulation of Fluid**

   a. Heart/Blood vessels

   - Circulates blood through the kidneys
   - Failure of this pumping action interferes with renal perfusion and thus with water and electrolyte regulation

   b. Lungs

   - Through exhalation
   - Hyperpnea (abnormally deep respiration) or continuous coughing, increase this loss

- Lungs also play a major role in maintaining acid–base balance

c. Kidneys

- Regulates ECF
- Selective retention and excretion of body fluids
- Regulation of normal electrolyte levels by selective electrolyte retention and excretion
- Regulation of pH retention of hydrogen ions
- Excretion of metabolic wastes and toxic substances
- Given these functions, failure of the kidneys results in multiple fluid and electrolyte abnormalities.

d. Pituitary

- Hypothalamus manufactures ADH
- ADH controls the retention or excretion of water by the kidneys

3. **Hypovolemia/Hypervolemia**

Hypovolemia

a) **s/s** – 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

b) Nursing Management –

- I&O at least every 8 hours (sometimes hourly)
    - Observe for urine output <0.5-1 mL/kg/h in an adult
- Daily weights
    - Acute loss of 0.5 kg represents a fluid loss of approximately 500 mL (1L of fluid weighs approximately 1 kg, or 2.2 lb)
- Vital signs

        - Observes for weak, rapid pulse & orthostatic hypotension
        - Decrease body temperature often accompanies FVD unless concurrent infection
    - Skin and tongue turgor are monitored
        - turgor is best measured by pinching the skin over the sternum, inner aspects of the thighs, or forehead

c) Isotonic solutions – lactated Ringer's & sodium chloride

<u>Hypervolemia</u>

a) **s/s** – 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

b) Nursing Management –

- Measure I&O
- Daily weights
    - Gain of 1 kg (2.2 lb) = gain of approx 1L fluid
- Assess breath sounds.
    - Particularly if parenteral fluids are being administered
- Monitor degree of edema in dependent parts of body (i.e. – feet, ankles, & sacral region)
    - Pitting edema: assessed by pressing finger into affected part, creating a pit or indentation that is evaluated on a scale of 1+ (minimal) to 4+ (severe)
    - Peripheral edema is monitored by measuring the circumference of the extremity with a tape marked in millimeters

c) Hypotonic solutions – 0.45%, 0.33%, 0.225% NaCl & D2.5W

4. **Hyponatremia/Hypernatremia**

Hyponatremia

a) Contributing Factors – 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 combination of both

b) **s/s** – poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, **abdominal cramping**, neurologic changes, **CONFUSION**

c) Nursing Management – assessment and prevention, dietary sodium and fluid intake, identify and monitor at-risk patients, effects of medications (diuretics, lithium)

Hypernatremia

a) Contributing Factors – inadequate water intake, excess water loss, or rarely, sodium gain

b) s/s – thirst; elevated temperature; dry, swollen tongue; sticky mucosa; neurologic symptoms; restlessness; weakness

c) Nursing Management – assessment and prevention, assess for OTC sources of sodium, offer and encourage fluids to meet patient needs, provide sufficient water with tube feedings

5. **Hypokalemia/Hyperkalemia**

Hypokalemia

a) Contributing Factors – GI losses, medications, alterations of acid–base balance, hyperaldosterism, poor dietary intake

b) s/s – fatigue, anorexia, nausea, vomiting, dysrhythmias, muscle weakness and cramps, paresthesias, glucose intolerance, decreased muscle strength, DTRs

c) Nursing Management – assessment, severe hypokalemia is life-threatening, monitor ECG and ABGs, dietary potassium, nursing care related to IV potassium administration

Hyperkalemia

a) Contributing Factors – usually treatment related, impaired renal function, hypoaldosteronism, tissue trauma, acidosis

b) s/s – cardiac changes and dysrhythmias, muscle weakness with potential respiratory impairment, paresthesias, anxiety, GI manifestations

c) Nursing Management – assessment of serum potassium levels, mix IVs containing K^+ well, monitor medication affects, dietary potassium restriction/dietary teaching for patients at risk

6. **Hypocalcemia/Hypercalcemia**

Hypocalcemia

a) Contributing Factors – hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications

b) s/s – tetany, circumoral numbness, paresthesias, hyperactive DTRs, Trousseau's sign, Chovstek's sign, seizures, respiratory symptoms of dyspnea and laryngospasm, abnormal clotting, anxiety

c) Nursing Management – assessment, severe hypocalcemia is life-threatening, weight-bearing exercises to decrease bone calcium loss, patient teaching related to diet and medications, and nursing care related to IV calcium administration

Hypercalcemia

a) Contributing Factors – malignancy and hyperparathyroidism, bone loss related to immobility

b) s/s – muscle weakness, incoordination, anorexia, constipation, nausea and vomiting, abdominal and bone pain, polyuria, thirst, ECG changes, dysrhythmias

c) Nursing Management – assessment, hypercalcemic crisis has high mortality, encourage ambulation, fluids of 3 to 4 L/d, provide fluids containing sodium unless contraindicated, fiber for constipation, ensure safety

7. **Hypomagnesemia/Hypermagnesemia**

Hypomagnesemia

a) Contributing Factors – alcoholism, GI losses, enteral or parenteral feeding deficient in magnesium, medications, rapid administration of citrated blood; contributing causes include diabetic ketoacidosis, sepsis, burns, hypothermia

b) s/s – neuromuscular irritability, muscle weakness, tremors, athetoid movements, ECG changes and dysrhythmias, alterations in mood and level of consciousness

c) Nursing Management – assessment, ensure safety, patient teaching related to diet, medications, alcohol use, and nursing care related to IV magnesium sulfate

Hypermagnesemia

a) Contributing Factors – renal failure, diabetic ketoacidosis, excessive administration of magnesium

b) s/s – flushing, lowered BP, nausea, vomiting, hypoactive reflexes, drowsiness, muscle weakness, depressed respirations, ECG changes, dysrhythmias

c) Nursing Management – assessment, do not administer medications containing magnesium, patient teaching regarding magnesium-containing OTC medications

8. **Hypophosphatemia/Hyperphosphatemia**

Hypophosphatemia

a) Contributing Factors – alcoholism, refeeding of patients after starvation, pain, heat stroke, respiratory alkalosis, hyperventilation, diabetic ketoacidosis, hepatic encephalopathy, major burns, hyperparathyroidism, low magnesium, low potassium, diarrhea, vitamin D deficiency, use of diuretic and antacids

b) s/s – neurologic symptoms, confusion, muscle weakness, tissue hypoxia, muscle and bone pain, increased susceptibility to infection

c) Nursing Management – assessment, encourage foods high in phosphorus, gradually introduce calories for malnourished patients receiving parenteral nutrition

Hyperphosphatemia

a) Contributing Factors – renal failure, excess phosphorus, excess vitamin D, acidosis, hypoparathyroidism, chemotherapy

b) s/s – few symptoms; soft-tissue calcifications, symptoms occur due to associated hypocalcemia

c) Nursing Management – assessment, avoid high-phosphorus foods; patient teaching related to diet, phosphate-containing substances, signs of hypocalcemia

9. **Laboratory Testing (Normal/Abnormal Values)**

a. Creatinine – 0.7-1.4

b. Hematocrit – M: 42-52% | F: 35-47%

c. Urine sodium – 40-220

10. **Acid Base Balance (Normal/abnormal)**

a. pH – 7.35-7.45

b. $PaCO_2$ – 35-34

c. HCO_3 – 22-26

d. PaO_2 – 80-100

e. O_2 Sat > 94%

11. **Acidosis/Compensation**

a. Metabolic – ↓ pH; normal PaCO2; ↓ HCO3

- s/s: headache, confusion, drowsiness, increased respiratory rate/depth, decreased bp, decreased cardiac output, dysrhythmias, shock

b. Respiratory – ↓ pH; ↑ PaCO2; normal HCO3

- s/s: sudden increase in pulse/respiratory rate/bp, mental changes, feeling of fullness in head

12. **Alkalosis/Compensation**

a. Metabolic – ↑ pH; normal PaCO2; ↑ HCO3

- s/s: signs of decreased calcium/potassium, respiratory depression, tachycardia

b. Respiratory – ↑ pH; ↓ PaCO2; normal HCO3

- s/s: lightheadedness, inability to concentrate, numbness/tingling, loss of consciousness

13. **Shock**

1. <u>Cardiogenic</u> – failure of heart to pump effectively due to cardiac factor

1. s/s:

- Tachycardia, hypotension, narrowed pulse pressure, dysrhythmias
- Decreased cardiac output, cardiac index
- Tachypnea, crackles
- Peripheral hypo-perfusion
    - Cyanosis, pallor, diaphoresis, weak peripheral pulses, cool/clammy skin, delayed capillary refill
- Decreased urine output, Na/Water retention
- Anxiety, confusion, agitation

2. Nursing Management:

- Preventing cardiogenic shock
- Monitoring hemodynamic status
- Administering medications, IV fluids
- Maintaining intra-aortic balloon counterpulsation
- Ensuring safety, comfort

2. Hypovolemic – decreased intravascular volume due to fluid loss
   a) s/s – Pt may appear anxious, urine output decreases
   b) Nursing Management:

- Primary prevention of shock is essential focus of nursing care
  - Hypovolemic shock can be prevented in some instances by closely monitoring patients who are at risk for fluid deficits and assisting with fluid replacement before intravascular volume is depleted
- Nursing care focuses on:
  - assisting with treatment targeted at the cause of the shock
  - restoring intravascular volume

3. Distributive – widespread vasodilation and increased capillary permeability

1. Neurogenic – loss of sympathetic tone causing relative hypovolemia, occurs w/in 30 min of spinal injury

   a) s/s: bradycardia, hypotension, hypothermia
   b) Nursing Management:

- Treatment based on cause
  - if spinal cord injury → promote spinal stability (spinal precautions, c-collar)
- Use vasopressors (e.g., phenylephrine) to maintain BP and organ perfusion
- Bradycardia treated w/ 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

2. Anaphylactic – due to severe allergic reaction producing overwhelming systemic vasodilation, relative hypovolemia

a) s/s:
   - Present w/I 2-30 minutes of exposure
   - Rapid onset hypotension, neurologic compromise, respiratory distress, cardiac arrest, GI distress, skin/mucosal tissue irritation

b) Nursing Management:
   - Prevention
   - Maintain patent airway
     - Endotracheal intubation or cricothyroidotomy may be necessary
   - IM epinephrine is drug of choice
     - Causes peripheral vasoconstriction and bronchodilation and opposes the effect of histamine
   - Diphenhydramine and ranitidine (Zantac) are given as adjunctive therapies to block the ongoing release of histamine from allergic reactions
   - Nebulized bronchodilators
   - Fluid resuscitation

3. Septic – due to overwhelming infection causing relative hypovolemia, most common type of distributive shock.

a) s/s:
   - RR >22
   - Altered mentation (GCS <15)

- SBP <100
- Persisting hypotension requiring vasopressors to maintain MAP >/= 65
- Blood lactate >2 despite adequate volume resuscitation

b) Nursing Management:

- If septic:
  - Secure airway and correct hypoxemia
  - Establish venous access
  - Obtain CBC, serum lactate, ABG, blood cultures, imaging targeted at the suspected site of infection, procalcitonin
  - Aggressive administration of IVF given at 30mL/kg w/in the first 3 hrs of presentation
  - Empiric antibiotic therapy administered w/in first 1 hr
- If septic shock
  - IV vasopressors → norepinephrine
  - Glucocorticoids if shock refractory to adequate fluid resuscitation and vasopressor administration

4. <u>Obstructive</u> – due to physical obstruction impeding the filling/outflow of blood resulting in reduced CO

a) s/s:

- Anxiety, restlessness, altered mental state
- Hypotension (d/t decreased cardiac output/stroke volume)
- Rapid/weak/thready pulse
- Cool/clammy/mottled skin (d/t vasoconstriction/hypo-perfusion)
- Oliguria (d/t decreased renal perfusion)

b) Nursing Management:

- Early recognition and treatment to relieve or manage the obstruction

- Mechanical decompression for pericardial tamponade, tension pneumothorax, and hemopneumothorax
- If pulmonary embolism → thrombolytic therapy
- If superior vena cava syndrome → radiation, debulking, or removal of the mass or cause

14**. Compensatory Mechanism in Shock**

a) s/s:

- 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

b) Nursing Management:

- Early intervention!
- Assess for shock → recognizing subtle clinical signs of compensatory stage before BP drops
- Early interventions include:
    - identifying the cause of shock, administering IV fluids & oxygen, and obtaining necessary lab tests to rule out & treat metabolic imbalances or infection

15. **Progressive Stage of Shock** – compensatory mechanisms begin to fail

Body System	**Clinical Manifestations**

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

Body System	Clinical Manifestations
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
Hematologic	• 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

Nursing Management:

- Early interventions are essential to survival!
    - Suspecting that a patient may be in shock and reporting subtle changes in assessment are imperative
    - Hemodynamic monitoring, ECG monitoring, ABGs, serum electrolyte levels, physical & mental status changes
- Rapid and frequent administration of various prescribed medications and fluids
- Possibly interventions with supportive technologies, such as mechanical ventilation, dialysis (CRRT), &/or IABP

16. **Irreversible Stage** – refractory stage, total body failure

a) S/S:
    - BP – requires mechanical or pharmacologic support
    - HR – Erratic or asystole
    - Respiratory – Requires intubation and mechanical ventilation and oxygenation
    - Skin – Jaundice
    - Urinary output – Anuric, requires dialysis
    - Mentation – Unconscious
    - Acid-Base Balance – Profound acidosis

b) Nursing Management:

    - Nurse focuses on carrying out prescribed treatments, monitoring the patient, preventing complications, protecting the patient from injury, and providing comfort
    - Offering brief explanations to pt about what is happening is essential even if there is no certainty that the patient hears or understands what is being said

- Simple comfort measures, including reassuring touches, should continue to be provided despite the patient's non-responsiveness to verbal stimuli
- As it becomes obvious that pt is unlikely to survive, family must be informed about the prognosis and likely outcome
- Opportunities should be provided—throughout the patient's care—for family to see, touch, and talk to patient
- Close family friends or spiritual advisors may be of comfort to the family members in dealing with the inevitable death of their loved one

17. **Tissue Perfusion**

- In assessing tissue perfusion, the nurse observes for subtle changes in the following:
  - LOC
  - VS
  - urinary output
  - skin color/temperature
  - laboratory values
    - base deficit
    - lactic acid levels
    - In compensatory stage of shock, serum sodium & blood glucose levels are elevated in response to release of aldosterone & catecholamines
- Monitor hemodynamic status & promptly report deviations to HCP
- **Vital signs are key indicators of hemodynamic status**
  - BP is an indirect measure of tissue hypoxia

- Report SBP <90 mmHg or drop in SBP of 40 mmHg from baseline or MAP <65 mmHg
- Pulse pressure correlates well with stroke volume
    - Pulse pressure is calculated by subtracting diastolic measurement from systolic measurement; the difference is the pulse pressure
    - Normally, the pulse pressure is 30 to 40 mm Hg; 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!**

18**. Vasoactive Agents Used in Shock**

Medication	**Desired Action in Shock**	**Disadvantages**
Inotropic Agents • Dobutamine • Dopamine • Epinephrine • Milrinone (Primacor)	Improve contractility, increase stroke volume, increase cardiac output	Increase oxygen demand of the heart
Vasodilators • Nitroglycerin	Reduce preload and afterload, reduce oxygen demand of heart	Cause hypotension

• Nitroprusside		
Vasopressor Agents • Norepinephrine (Levophed) • Dopamine • Phenylephrine • Vasopressin	Increase blood pressure by vasoconstriction	Increase afterload, thereby increasing cardiac workload; compromise perfusion to skin, kidneys, lungs, gastrointestinal tract

19**. SIRS**

a) s/s:

- Temp >38.3°C (>101°F) or <36°C (<96.8°F)
- HR >90 bpm
- RR >20 breaths/min or PaCO2 >32 mm Hg
- WBC >12,000 cells/mm3, <4,000 cells/mm3, or >10% immature WBC (bands)
- Hypotension (decreased perfusion

b) Nursing Management:

- Assess for EKG changes
- Monitor:
- Oxygenation, vitals, EKG, urine output, LCO, skin color/temp/moisture/cap refill/turgor
- Explain procedures
- IV access
- Titrate IV drips to maintain hemodynamic parameters

- Monitor central venous pressure, pulmonary artery pressures, cardiac output, pulse pressure
- Educate patient, reassure family

20**. MODS**

a) s/s:

Body System	**Manifestations**
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/Cr rate >20:1 Intrarenal: • BUN/Cr <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

b) 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, because massive loss of skeletal muscle mass makes rehabilitation a long, slow process