N441 Concept Review Exam 2

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1. **Total water composition**
   ICF- ⅔ of fluid in body, inside the cells
   ECF- ⅓ of fluid, outside cells
   Interstitial- fluid between cells and outside vessels
   Intravascular- fluid within vessels
   Third spacing- loss of fluid into spaces that don't contribute to balancing the ICF and ECF
   s/s- early evidenced by oliguria, ↑HR, ↓BP, ↓central venous pressure, ↑weight, imbalance fluid I/O
   Occurs with hypocalcemia, ↓iron intake, liver disease, alcoholism, hypothyroidism, malabsorption, immobility, burns, and cancer

2. **Regulation of Fluid**
   Cardiac moves fluids, Lungs breath out moist air, Kidneys excrete fluid in urine along with electrolytes, hypothalamus releases ADH/vasopressin

Crystalloids	Advantages	Disadvantages
Crystalloids • 0.9% sodium chloride (normal saline solution)	Widely available, inexpensive	Requires large volume of infusion; can cause hypernatremia, pulmonary edema, abdominal compartment syndrome
• Lactated Ringer's	Lactate ion that helps buffer metabolic acidosis	Requires large volume of infusion; can cause metabolic acidosis, pulmonary edema, abdominal compartment syndrome
Colloids • Albumin (5%, 25%)	Rapidly expands plasma volume of crystalloid (or colloid equivalent).	Expensive; requires human donors; limited supply; can cause heart failure

3. **Hypo/Hypervolemia**
   Hypovolemia
   Decreased fluid volume from low sodium, low fluid intake, dehydration, trauma, 3rd space, edema, burns, ascites
   S/S - weight loss, >3 sec skin turgor, flat neck viens, dizzy, weak, thirst, confused, ↓LOC, pale skin, muscle cramp, oliguria
   LABS - ↑hct, ↑BUN, ↓osmolarity,↑ specific gravity, ↑creatinine,

↓sodium

Management - daily weight, I/O vitals, tongue turgor

Hypervolemia

Increased fluid volume from retention, too much vassopressin/ADH, heart failure, poor perfusion, renal failure, liver cirrhosis, high sodium

S/S - weight gain, <3 sec skin turgor, ↑BP, bounding pulse, distended veins, crackles, sob, tachypnea, polyruria

LABS - ↓hct, osmolarity/gravity/cretinine/burn ↓↓↓

Management - daily weight, diuretics (loop/thiazide), dialysis, sodium restriction, heart sounds, assess for edemas

Imbalance	Contributing Factors	S/Sx & Laboratory Findings
Fluid Volume Deficit (hypovolemia)	Loss of water and electrolytes, as in vomiting, diarrhea, fistulas, fever, excess sweating, burns, blood loss, gastrointestinal suction, and third-space fluid shifts; and decreased intake, as in anorexia, nausea, and inability to gain access to fluid. Diabetes insipidus and uncontrolled diabetes both contribute to a depletion of extracellular fluid volume.	• 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

Imbalance	Contributing Factors	S/Sx & Laboratory Findings
Fluid Volume Excess (hypervolemia)	Compromised regulatory mechanisms, such as renal failure, heart failure, and cirrhosis; overzealous administration of sodium-containing fluids; and fluid shifts (i.e., treatment of burns). Prolonged corticosteroid therapy, severe stress, and hyperaldosteronism augment fluid volume excess.	• 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

Fluids

Hypo - isotonic .9%, 3% with severely low sodium levels

Hyper - D5W is sodium free fluid

4. **Hypo/hypernatremia**

Hyponatremia - low sodium caused by either low intake or loss through trauma, water excess, renal sailure, SIADH

S/S - poor skin turgor, dry mucosa, headache, low salivation, low BP, nausea, abdominal cramping, changes in LOC/confusion

LABS - sodium levels

Management - dietary, fluid intake with sodium, 3%given slowly, diuretics, lithium

Hypernatremia - too much salt caused by low water, too much salt intake, diuretic use

S/S - thirst, high temp, dry swollen tongue, sticky mucosa, changes in LOC, restless, weakness

Labs- sodium levels

Management - D5W is sodium free, diuretics, assess for sources of sodium to avoid /education

Electrolyte	Functions	Sources & Losses	Regulation
Sodium (Na+) • Chief electrolyte of ECF • Normal serum sodium: 135-145 mEq/L	• Regulates extracellular fluid volume; Na+ loss or gain accompanied by a loss or gain of water • Affects serum osmolality • Role in muscle contraction and transmission of nerve impulses • Regulation of acid–base balance as sodium bicarbonate	• Normally enters the body through the GI tract from dietary sources, such as salt added to processed foods, sodium preservatives added to processed foods • Lost from gastrointestinal tract, kidneys, and skin	• Transported out of the cell by the sodium-potassium pump • Regulated by renin-angiotensin-aldosterone system • Elimination and reabsorption regulated by the kidneys • Sodium concentrations affected by salt and water intake

5. **Hypo/hyperkalemia**

Hypo - GI loss, medications, alterations of acid base balance, high aldosterone, poor intake

S/s - fatigue, anorexia, nasuea, vomiting, heart dyshrythm, muscle

weakness, cramps, paresthesia, glucose intolerance, Deep tendon reflexes up↑ low T wave, narrow QRS

Management - diet, assessment, monitor ECG ABGs, care with IV potassium (it hurts a ton!)

Hyper - lasix use, impaired renal function, low aldosterone (invers relation) trauma, acidosis

S/S - cardiac, HIGH T WAVE wide QRS, muscles weak, potential respiratory and GI, *anxiety*

Managment - assessment, mix IV soutions well, thiazide potassium sparing, avoid salt substitutes

Electrolyte	Functions	Sources & Losses	Regulation
Potassium (K+) •Major cation of ICF •Normal serum potassium: 3.5-5.0	• Controls intracellular osmolality • Regulator of cellular enzyme activity • Role in the transmission of electrical impulses in nerve, heart, skeletal, intestinal, and lung tissue; protein and carbohydrate metabolism; and cellular building • Regulation of acid–base balance by cellular exchange with H+	• Adequate quantities via a well-balanced diet • Leading food sources: fruits and vegetables, dried peas and beans, whole grains, milk, meats • Lost via kidneys, stool, sweat, emesis • Gastrointestinal (GI) secretions contain potassium in large quantities, so can be lost through vomitus	• Regulated by aldosterone • Eliminated by the kidneys (no effective method of conserving potassium) • Additional regulation via transcellular shift between the ICF and ECF compartments

6. **Hypo/hypercalcemia**

Hypo - hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications

S/S - tetany, chovstek, trueso, hyperactive DTR, seizures, dyspnea, laryngospasm, abormal clotting, anxiety

Managment - IV calcium gluconate, vitamin D, diet, weight bearing exercises

Hyper - cancer, hyperparathyroidism, bone loss from immobility

S/S - muscles weak, uncoordinated, anorexia, constipation, nausesa, vomitting, bone pain, polyruia, thirst, ECG, dysyhtmias

Management - fluids, furosemide, phashpates, calcitonin, biophsophates, encourage ambulation, fluids with sodium 3-4L

Electrolyte	Functions	Sources & Losses	Regulation
Calcium • Most abundant electrolyte in the body • 99% of body calcium is stored in bone • 1% inside cells • Normal total serum calcium level: 8.6– 10.2 mg/dL • Normal ionized serum calcium level: 4.5–5.1 mg/dL	• Role in blood coagulation and in transmission of nerve impulses • Helps regulate muscle contraction and relaxation • Activates enzymes that stimulate essential chemical reactions in the body • Major component of bones and teeth	• Absorbed from foods in the presence of normal gastric acidity and vitamin D • Lost via feces and urine • Sources include milk, milk products, and cheese; dried beans; fortified orange juice; green, leafy vegetables; small fish with bones; and dried peas and beans	• Primarily excreted by GI tract; lesser extent by kidneys • Regulated by parathyroid hormone and calcitonin • High serum phosphate results in decreased serum calcium level; low serum phosphate leads to increased serum calcium

7. **Hypo/hypermagnesemia**

Hypo - alcoholism, GI losses, feeding deficit, medications, rapid administration og citrated blood, diabetic ketoacidosis, sepsis, burns, hypothermia

S/S - neuromuscular irritability, muscles weak, tremors, athetoid movements, ECF changes, dysrythmias, altered mood LOC

Management - diet oral mag, magnesium sulfate IV, ensure safety with IV care, teaching diet, education alcohol use

Hyper - renal failure, diabetic ketoacidosis, exceccise administration of magnesium

S/S - flushing, low BP, nausea, vomiting, hypoactive reflexes, drowsiness, muscle weakness, depressed respirations, ECG changes, dysthrmias

Management - IV calcium gluconate, loop diruetics (Lasix furosemide) IV NS or lactated ringers, hemodialysis, dont admin meds with mag, teaching regarding OTC medications

Electrolyte	Functions	Sources & Losses	Regulation
Magnesium (Mg^{2+}) • 2nd most abundant ICF cation after potassium • Normal serum concentration: 1.3–2.3 mEq/L	• Metabolism of carbohydrates and proteins • Activator for many intracellular enzyme systems • Role in neuromuscular function • Acts on cardiovascular system, producing vasodilation	• Enters the body via GI tract • Found in green, leafy vegetables; nuts; sea- food; whole grains; dried peas and beans; cocoa • Lost via urine with use of loop diuretics	• Eliminated by kidneys • Regulated by parathyroid hormone

8. **Hypo/hyperphosphatemia**

Hypo - alcoholism (again), refeeding after starvation, pain, heatstroke, respiratory alkalosis, hyperventilation, diabetic ketoacidosis, hepatic encephalopathy, major burns, hyperparaythyroidism, low magnesium, low potassium, diarrhea, vitamin D deficiency, use of diuretics and antacids **(many contributing factors!!)**

S/S - neurological symptoms, confusion, muscles weak, tissue hypoxia, muscle/bone pain, increased change to be infection

Managment - oral or IV phopsorus, encourage foods with phosphates, gradual feeding

Hyper - renal failure, excess phosphorus, excess vitamin D, acidosis, hypoparathyroidism, chemotherapy

S/S - few symptoms, soft tissue calcification, symptoms occur due to associated hypocalcemia

Management - treatment of underyling cause, vitamin D,

Electrolyte	Functions	Sources & Losses	Regulation
Phosphate (PO_4^{-}) • Major ICF anion; a buffer anion in both ICF and ECF • Normal serum phosphate level: 2.5–4.5 mg/dL	• Role in acid–base balance as a hydrogen buffer • Promotes energy storage; carbohydrate, protein, and fat metabolism • Bone and teeth formation • Regulation of hormone and coenzyme activity • Role in muscle and red blood cell function	• Enters body via gastrointestinal tract • Sources include all animal products (meat, poultry, eggs, milk, bread, ready-to-eat cereal) • Absorption is diminished by concurrent ingestion of calcium, magnesium, and aluminum	• Eliminated by kidneys • Regulation by parathyroid hormone and by activated vitamin D • Phosphate and calcium are inversely proportional; an increase in one results in a decrease in the other

9. **Hypo/hyperchloremia**

Electrolyte	Functions	Sources & Losses	Regulation
Chloride (Cl−) • Major ECF anion • Normal serum level of chloride: 97–107 mEq/L	• Major component of interstitial and lymph fluid; gastric and pancreatic juices, sweat, bile, and saliva • Acts with sodium to maintain the osmotic pressure • Role in the body's acid–base balance; combines with hydrogen ions to produce hydrochloric acid	• Enters body via gastrointestinal tract • Almost all chloride in diet comes from salt • Found in foods high in sodium, processed foods	• Normally paired with sodium; excreted and conserved with sodium by the kidneys • Regulated by aldosterone alongside sodium • Low potassium level leads to low chloride level

10. **Lab values**

BUN - 7-20
Creatinine 0.6-1.2

Specific gravity - 1.010-1.030
osmolarity

HCT M - (42-52) F - (37-47)
Hgb M - (14-18) F - (12-16)

(reminder HBG and HCT follow each other with hypovolemia)

Electrolyte levels

Sodium	135-145
Potassium	3.5-5.0
Calcium	8.6-10.2
Magnesium	1.3-2.3
Phosphate	2.5-4.5
Chlorine	97-107

11. **Acid-bases**

PH	7.35-7.45
PaCo2	35-45

HCo3 22-26
PaO2 80-100
O2 stat >94
Base excess/deficit +- 2 mEq/L

12. **Acidosis compensation**
    Respiratory -bradypnea
    Metabolic -increased secretion of HCo3

13. **Alkalosis compensation**
    Respiratory - tachypnea
    Metabolic -decreased levels of HCo3

14. **Shock**
    6 Types total
        1. Cardiogenic - shock from failure of heart to pump effectively from cardio factor
        2. Hypovolemic - shock from reduced fluid volume levels
        3. *Distributive*/ Neurogenic - spinal injury or brain damage resulting in massive vasodilation
        4. *Distributive*/ Anaphylactic - Shock from allergic reaction causing allergic response
        5. *Distributive*/Septic - Shock from massive infection resulting in dramatic vasodilation
        6. Obstructive - Shock from physical blocking of blood flow or cardiac output

15. **Compensatory Mechanisms in Shock**
        Stages!
            1. Initial, no changes visible
            2. Compensatory, notable measures to compensate
            3. Progressive, compensation is unable to cope
            4. Refractory, irreversible failure

        Bodily responses to compensate for reduced tissue perfusion/circulation/oxydation
        -tachypnea, tachycardia, acidosis compensation, renin-angiotensin system to ↑BP and SVR, oliguria to maintain fluid volume levels, hyperglycemia to assist with respiration in cells, cortisol levels to cause ↑glucose↑

    (!give therapies within 3 hours of identifying shock for best outcomes!)

16. **Progressive Stage of Shock**
    Measures to increase cardiac output no longer effectively compensate, BP↓

and MAP↓, vasoconstriction still present but now acts with low BP to further hamper perfusion

LOC↓↓↓ from lower cerebral perfusion, hypoxia

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

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

17. **Irreversible Stage**
    The body can no longer recover and is in an irreversible state of shock
    - BP low, renal/liver failure, severe acidosis, MODS

18. **Tissue Perfusion**
    Assess with - fluid output/urine labs, base/acids, skin, lungs, electrolyte levels, oxygen sat

19. **Vasoactive Agents Used in Shock (meds)**

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 • Nitroprusside	Reduce preload and afterload, reduce oxygen demand of heart	Cause hypotension
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

20. **SIRS**

    - systemic inflammatory response to bodily insults such as infection, ischemia, infarction, and trauma

    Monitor for infection carefully

21. MODS

    Failure of 2 or more organ systems in acute illness in which homeostasis is not maintainable without intervention

    S/S - components of system failures,

    Managment - prevention is priority, support and monitor organ perfusion, support and educate client/family, advanced directives/advocacy, with survival support and educate on long rehabilition