N441 Concept Review Exam 2

1. **Total water composition**
   Intracellular (ICF)-fluid in the cells
   Extracellular (ECF)- fluid outside of the cells
   interstitial fluid-fluid in spaces between cell
   Intravascular-plasma
   Approximately ⅔ of the body is ICF, approximately ⅓ is ECF

c. Third spacing-excess fluid collects in the non-functional area between cells. Fluid is trapped where it is difficult or impossible to move back into the cells of blood vessels. EARLY SIGN: DECREASE IN URINE OUTPUT.OTHER S/S: INCREASED HEART RATE, DECREASED BP, DECREASED CVP, EDEMA, INCREASED BODY WEIGHT, & IMBALANCES OF FLUID INTAKE AND OUTPUT.

Occurs in patients with: hypocalcemia, decreased iron intake, severe liver disease, alcoholism, hypothyroidism, malabsorption, immobility, burns and 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 respirations) or continuous coughing, increase this loss
- lungs also play a major role in maintaining acid-base balance
- Eliminate water vapor (insensible loss) at rate of approximately 300ml/day
- Loss increases with increases RR or in dry climate

c. Kidneys

- regulates ECF
- selective retention and excretion of body fluids
- regulation of normal electrolyte levels by selective electrolyte retention and excretion
- regulation of metabolic wastes and toxic substances
- GIVEN THESE FUNCTIONS, FAILURE OF THE KIDNEYS RESULTS IN MULTIPLE FLUID AND ELECTROLYTE ABNORMALITIES
- Urine output- 1-2L (adult)

d. Pituitary

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

3. **Hypovolemia/Hypervolemia**

a. s/s

<u>Hypovolemia-</u> occurs when loss of ECF volume exceeds the intake of fluid. Occurs when water and electrolytes are lost in the same proportion as they exist in normal body fluids, thus the ratio of serum electrolytes to water remains the same. FVD should not be confused with dehydration, which refers to loss of water alone, with increased serum sodium levels. <u>Causes</u>: abnormal fluid losses (V/D, GI suctioning, sweating), decreased intake (nausea or lack of access to fluids), 3rd space fluid shifts (edema formation in burns, ascites with liver dysfunction)	**<u>Hypervolemia-</u>** refers to isotonic expansion of ECF caused by abnormal retention of water and sodium in the same proportions in which they normally exist in the ECF. R/T to simple fluid overload or diminished function of the homeostatic mechanisms responsible for regulating fluid balance. Contributing factors can include heart failure, renal failure and cirrhosis of the liver, excessive amounts of table or other sodium salts. Excessive administration of sodium containing fluids in a patient impaired regulatory mechanisms may predispose as well.
<u>S/S:</u> acute weight loss decreased skin turgor oliguria concentrated urine capillary filling time prolonged Low CVP decreased BP and increased pulse Flattened neck veins dizziness weakness thirst confusion muscle cramps sunken eyes nausea increased temperature cool, clammy, pale skin	**<u>S/S:</u>** acute weight gain peripheral edema and ascites JVD crackles elevated CVP shortness of breath increased BP bounding pulse increased RR increased urine output cough
<u>Labs:</u>elevated hemoglobin, hematocrit, serum urine osmolality and specific gravity, BUN, creatinine. decreased urine sodium.	**<u>Labs:</u>** decreased hemoglobin and hematocrit, serum and urine osmolality, urine sodium and specific gravity
<u>Nursing management:</u> -Isotonic electrolyte solutions (lactated ringers, 0.9% sodium chloride) 1ST CHOICE	**<u>Nursing management:</u>** -Diuretics: generally thiazide diuretics are prescribed for mild to moderate hypervolemia and

-Monitor I&O (every 8 hours), daily weights vital signs, CVP, LOC, breath sounds, skin color, skin and tongue turgor. -Monitor for fluid overload -If oliguric: consider prerenal azotemia vs VTN -Volumes of fluid are administered at specific rates and intervals while monitoring their bodies response. -Prevent hypovolemia -Correct hypovolemia- offer small volumes of oral rehydration solutions	loop diuretics for severe hypervolemia. -Dialysis: when pharmacological agents arent effective -Nutritional therapy: sodium restricted diet -Measure I&O: gain of 1 kg -Daily weights -Assess breath sounds -Monitor degree of edema in dependent parts of body

c. Isotonic solutions → The concentration of solutes is the same both inside and outside the cell

0.9% normal saline & lactated ringers

4. **Sodium**

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

Hyponatremia

- •Serum sodium <135 mEq/L
- •Causes: loss of sodium-containing fluids (draining woundcs, 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
- •Manifestations: poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, abdominal cramping, neurologic changes, CONFUSION
- •Medical management: water restriction, sodium replacement
- •Level should not increase more than 8-12 mEq/L in the 1st 24 hrs
- •Nursing management: assessment and prevention, dietary sodium and fluid intake, identify and monitor at-risk patients, effects of medications (diuretics, lithium)
    - Hypertonic saline
        - Used in emergency settings when patient requires 4-6 mEq/L increase in serum sodium as soon as possible
        - •Example à 3% saline
        - •Administer slowly because it may cause volume overload and pulmonary edema

Hypernatremia

- •Serum sodium >145 mEq/L
- •Causes: inadequate water intake, excess water loss, or rarely, sodium gain
- •Manifestations: thirst; elevated temperature; dry, swollen tongue; sticky mucosa; neurologic symptoms; restlessness; weakness
- •Note: thirst may be impaired in elderly or the ill
- •Medical management: Depends on underlying cause
- –If water deficit à fluid replacement (oral or IV)
- –If sodium excess à sodium free IV fluids (D5W), diuretics, and restrict dietary sodium
- –Serum sodium should not decrease by more than 8-15 mEq/L in an 8 hr period à too fast and result is cerebral edema & neurologic complications (risk is greatest if hypernatremia developed over several days or longer)
- •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

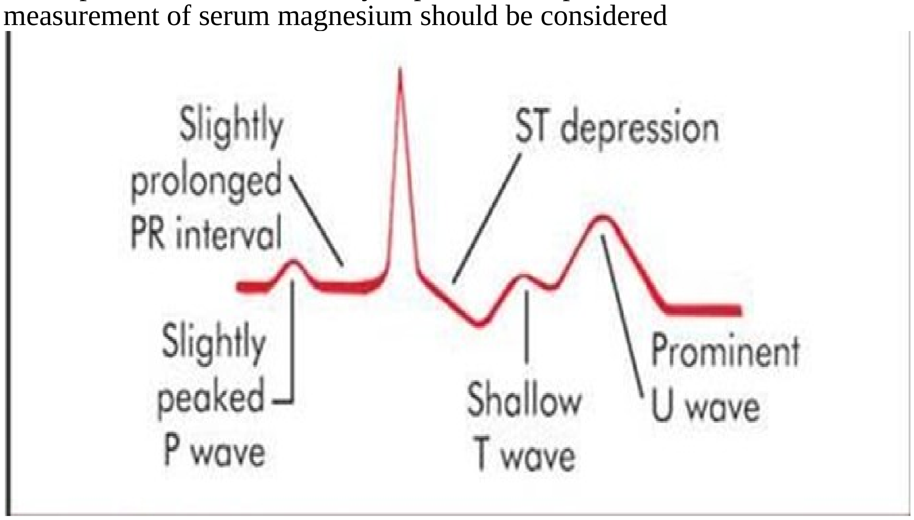
5. **Hypokalemia/Hyperkalemia**

Hypokalemia

- •Below-normal serum potassium (<3.5 mEq/L), may occur with normal potassium levels with alkalosis due to shift of serum potassium into cells
- •Causes: GI losses, medications, alterations of acid–base balance, hyperaldosterism, poor dietary intake
- •Manifestations: fatigue, anorexia, nausea, vomiting, dysrhythmias, muscle weakness and cramps, paresthesias, glucose intolerance, decreased muscle strength, DTRs
- •Medical management: increased dietary potassium, potassium replacement, IV for severe deficit
- •Nursing management: assessment, severe hypokalemia is life-threatening, monitor ECG and ABGs, dietary potassium, nursing care related to IV potassium administration
    - **Potassium deficit**

- •Potassium deficit varies directly with the severity of hypokalemia
- •A potassium deficit of 200-400 mEq is required to lower the serum potassium concentration by 1 mEq/L
- •However, these estimates are only an approximation of the amount of potassium replacement required to normalize the serum potassium concentration and careful monitoring is required

- **Potassium Replacement**
  - •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 à pain and phlebitis during infusion, primarily occurs at rate >10mEq/hr
  - •Must use an infusion pump
  - •Increase intake of potassium-rich foods
  - –Highest à dried figs, molasses, seaweed
  - –Very High à dried fruits, nuts, avocados, wheat germ, lima beans
  - –High à spinach tomatoes, broccoli, beets, carrots, cauliflower, potatoes, bananas, cantalouse, kiwis, oranges, mangos, ground beef, steak, pork, veal, lamb
  - •If continued potassium losses à rate of potassium administration must be increased by the rate of potassium loss to produce the desired rate of potassium repletion
  - **IV Potassium Safety Alert**
  - •IV KCl must always be diluted and never given in concentrated amounts
  - •Never give KCl via IV push or as a bolus
  - •Invert IV bags containing KCl several times to ensure even distribution in the bag
  - •Do not add KCl to hanging IV bag to prevent giving a bolus dose
  - Patients with hypokalemia may also have hypomagnesemia d/t concurrent loss w/ diarrhea or diuretic therapy
  - •Such patients can be refractory to potassium replacement alone; thus measurement of serum magnesium should be considered



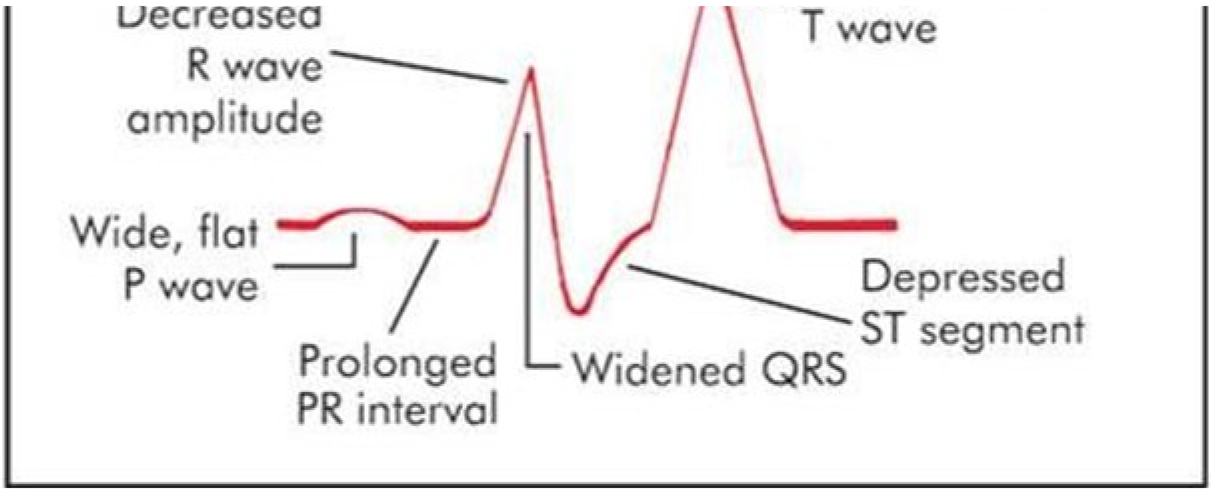
Potassium

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

Hyperkalemia

- •Serum potassium >5.0 mEq/L
- •Causes: usually treatment related, impaired renal function, hypoaldosteronism, tissue trauma, acidosis
- •Manifestations: cardiac changes and dysrhythmias, muscle weakness with potential respiratory impairment, paresthesias, anxiety, GI manifestations
- •Medical management: monitor ECG, limitation of dietary potassium, cation-exchange resin (Kayexalate), IV sodium bicarbonate, IV calcium gluconate, regular insulin and hypertonic dextrose IV, b-2 agonists, dialysis
- •Nursing management: 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 of Hyperkalemia**
    - **Antagonism of membrane action potentials**
        - Calcium
        - Calcium gluconate vs calcium carbonate → calcium chloride contains 3x the concentration of elemental calcium however calcium gluconate is generally preferred d/t calcium chloride causing local irritation at the injection site
    - Drive extracellular potassium into cells
        - Insulin & glucose
        - Sodium bicarbonate (primarily if metabolic acidosis)
        - Beta 2 adrenergic agonists (Albuterol)
    - Removal of potassium from the body
        - Loop or thiazide diuretics (if no severe renal impairment)
        - Gastrointestinal cation exchange (ie – patiromer, Kayexalate)
        - Dialysis, preferably hemodialysis if severe

- Dixogin like drugs and B-adrenergic blockers (e.g., propranolol) can impair entry of potassium into cells, resulting in a higher ECF potassium concentration
- •Several drugs, such as heparin, potassium-sparing diuretics, angiotensin II receptor blockers (e.g., losartan), and angiotensin-converting enzyme (ACE) inhibitors (e.g., lisinopril) can contribute to hyperkalemia by reducing the kidneys ability to excrete potassium
- Because patients on digoxin therapy have an increased risk of toxicity if their serum potassium level is low, monitor the patient for digitalis toxicity
- •Digoxin toxicity occurs as plasma concentration rises above 2.0, however, signs of toxicity may occur at levels below 1.3-1.5 in the presence of hypokalemia



6. **Hypocalcemia/Hypercalcemia**

- Hypocalcemia
  - Contributing factors: hypoparathyroidism, malabsorption, pancreatitis, alkalosis, massive transfusion of citrated blood, renal failure, medications, other
  - s/s: tetany, circumoral numbness, paresthesias, hyperactive DTRs, Trousseau's sign, Chovstek's sign, seizures, respiratory symptoms of dyspnea and laryngospasm, abnormal clotting, anxiety
  - 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
  - Contributing Factors: malignancy and hyperparathyroidism, bone loss related to immobility
  - s/s: muscle weakness, incoordination, anorexia, constipation, nausea and vomiting, abdominal and bone pain, polyuria, thirst, ECG changes, dysrhythmias

  - 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**
  - 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
  - s/s: neuromuscular irritability, muscle weakness, tremors, athetoid movements, ECG changes and dysrhythmias, alterations in mood and level of consciousness
  - Nursing Management: assessment, ensure safety, patient teaching related to diet, medications, alcohol use, and nursing care related to IV magnesium sulfate
- **Hypermagnesemia**
  - Contributing Factors: renal failure, diabetic ketoacidosis, excessive administration of magnesium
  - s/s: flushing, lowered BP, nausea, vomiting, hypoactive reflexes, drowsiness, muscle weakness, depressed respirations, ECG changes, dysrhythmias
  - Nursing Management: assessment, do not administer medications containing magnesium, patient teaching regarding magnesium-containing OTC medications

8. **Hypophosphatemia/Hyperphosphatemia**

Electrolyte	**Functions**	**Sources & Losses**	**Regulation**

Phosphate (PO4−) • 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

Hypophosphatemia

- •Serum level below 2.5 mg/DL
- •Causes: 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
- •Manifestations: neurologic symptoms, confusion, muscle weakness, tissue hypoxia, muscle and bone pain, increased susceptibility to infection
- •Medical management: oral or IV phosphorus replacement
- •Nursing management: assessment, encourage foods high in phosphorus, gradually introduce calories for malnourished patients receiving parenteral nutrition

Hyperphosphatemia

- •Serum level above 4.5 mg/DL
- •Causes: renal failure, excess phosphorus, excess vitamin D, acidosis, hypoparathyroidism, chemotherapy
- •Manifestations: few symptoms; soft-tissue calcifications, symptoms occur due to associated hypocalcemia
- •Medical management: treat underlying disorder, vitamin-D preparations, calcium-binding antacids, phosphate-binding gels or antacids, loop diuretics, NS IV, dialysis
- •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: 42-52 % (Males) 35-47% (Females)

c. Urine sodium: 20 mEq/L

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

a. pH: 7.35-7.45

b. PaCO2: 25-45

c. HCO3: 22-26

d. PaO2: 80-100

e. O2 Sat: >94%

11. **Acidosis/Compensation**

a. Metabolic:

- Low pH <7.35
- PaCO2 35-45
- Low bicarbonate <22 mEq/L
- Most commonly due to renal failure; other causes include: DKA, lactic acidosis, starvation, inadequate elimination of hydrogen ions
- Manifestations: headache, confusion, drowsiness, increased respiratory rate and depth, decreased blood pressure, decreased cardiac output, dysrhythmias, shock; if decrease is slow, patient may be asymptomatic until bicarbonate is 15 mEq/L or less
- Correct underlying problem, correct imbalance
- Bicarbonate may be administered
- Correct underlying problem, correct imbalance
- Bicarbonate may be administered
- With acidosis, hyperkalemia may occur as potassium shifts out of cell
- As acidosis is corrected, potassium shifts back into cell, potassium levels decrease
- Monitor potassium levels
- Serum calcium levels may be low with chronic metabolic acidosis
- Must be corrected before treating acidosis

b. Respiratory:

- Low pH <7.35
- PaCO2 >45 mm Hg
- HCO3 22-26
- Always due to respiratory problem with inadequate excretion of CO2
- With chronic respiratory acidosis, body may compensate, may be asymptomatic
- Symptoms may be suddenly increased pulse, respiratory rate and BP, mental changes, feeling of fullness in head
- Potential increased intracranial pressure

- Treatment aimed at improving ventilation

12. **Alkalosis/Compensation**

a. Metabolic:

- High pH >7.45
- PaCO2 35-45
- High bicarbonate >26 mEq/L
- Most commonly due to vomiting or gastric suction
- May also be due to medications, especially long-term diuretic use; excess use of antacids
- Hypokalemia will produce alkalosis
- Manifestations: symptoms related to decreased calcium, respiratory depression, tachycardia, symptoms of hypokalemia
- Correct underlying disorder, supply chloride to allow excretion of excess bicarbonate, restore fluid volume with sodium chloride solutions

b. Respiratory:

- High pH >7.45
- PaCO2 <35 mm Hg
- HCO3 22-26
- Always due to hyperventilation
- Manifestations: lightheadedness, inability to concentrate, numbness and tingling, sometimes loss of consciousness
- Correct cause of hyperventilation

13. Shock

a. Cardiogenic

1. s/s: Tachycardia. Low BP, narrow pulse pressure, Low CO, crackles, tachypnea, signs of peripheral hypoperfusion (cyanosis, pallor, diaphoresis, weak pulse, delayed cap refill, cool clammy skin), decreased urine output, anxiety, confusion, agitation

2. Nursing Management: goal is into increase oxygen supply to heart muscle while reducing oxygen demands, O2 therapy, pain control- IV morphine, hemodynamic monitoring with A-line, Monitor labs- BNP and cardiac profile, fluid therapy, ensuring safety and comfort

b. Hypovolemic

1. s/s: appears anxious, decreased urine output, decreased cardiac output and stroke volume, hypotension, decreased tissue perfusion

2. Nursing Management: Treat underlying cause- stop bleeding, treat D/V, increase fluids intake. Start 2 large gauge IV lines for simultaneous administration of fluids (LR or .9% NS). Increase O2 to increase the amount of oxygen carried by available Hgb. MODIFIED TRENDELENBURG

Primary prevention: close monitoring of patients at risk for fluid deficits.
Complications: Monitor for CV overload (difficulty breathing, pulmonary edema)
Monitor older adults and patients with pre-existing cardiac disease closely
Monitor: VS, ABGs, Lactate levels, H/H, I&O, Temperature
Assessment: JVD
Report changes in BP, pulse pressure, CVP, HR, and lung sounds

c. Distributive three types neurogenic, anaphylactic, septic

1**.** Neurogenic

a. s/s: Hypotension, Bradycardia, hypothermia treat based on the cause.

b. Nursing Management: treat based on the cause. EX: if it is a spinal cord injury stabilize the spine.

2. Anaphylactic

a. s/s: food allergies, latex sensitivities, environmental allergies

b. Nursing Management: Epinephrine- Airway 1st

3. Septic

a. s/s immunosuppressed, hypoxia, hypotension

b. Nursing Management: watch for decreased LOC, abnormal VS

d. Obstructive

a. s/s decreased cardiac output EX: PE

b. Nursing Management: monitor circulation, VS, Lung Sounds

14**. Compensatory Mechanism in Shock (EARLY signs)**

a. S/S

- Blood pressure normal
- Heart rate >100
- RR >20 breaths/min and PaCO2 <32
- Skin cold and clammy’
- Urinary output decreased
- Confused and/ or agitation
- Respiratory alkalosis

b. Nursing Management

Early interventions along the continuum of shock is key to improving the patient's prognosis

- Assess the patient at risk for shock
- Recognizing subtle clinical signs of the compensatory stage before the patients BP drops

EARLY Interventions

- Identifying the cause of shock
- Administering IV fluids and oxygen
- Obtaining necessary lab tests to rule out and treat metabolic imbalances or infection

15. **Progressive Stage of Shock (2nd stage)**

S/s

- BP systolic <90, MAP <65 and requires fluids resuscitation to support BP
- HR >150
- RR rapid, shallow respirations, crackles, PaO <80, PaCO2 >45
- Skin mottled, petechiae
- Output <0.5 mL/kg/h
- Lethargy
- Metabolic acidosis

Nursing Management

- Requires expertise in assessing and understanding shock and the changes in assessment data
- The earlier the interventions the better for the clients chance if survival
- Close monitoring in a intensive care setting to monitor
  - Hemodynamic monitoring
  - Electrocardiographic monitoring (ECG)
  - Arterial blood gasses
  - Serum electrolyte levels
  - Physical and mental status changes
- Rapid and frequent administration of various prescribed medications and fluids
- Possibly interventions with supportive devices
  - Mechanical ventilation
  - Dialysis
  - Intra-aortic balloon pump

The nurse's role

- Document treatments, medications, and fluids that are given
  - Recording the time
  - Dosage or volume
  - Patient response
- Coordinates both scheduling of diagnostic procedures that may be at bedside and flow of health care personnel involved in the care of patients
- Provides essential support through ongoing communication with the patient and the family

16. **Irreversible Stage-** organ damage is so severe that the patient does not respond to treatment and cannot survive

S/S

- BP remains low and requires mechanical or pharmacologic support
- HR Erratic
- RR requires intubation and mechanical ventilation and oxygenation
- Skin Jaundice
- Output anuric; requires dialysis
- Unconscious
- Profound acidosis

Nursing Management

- Carrying out prescribed treatments
- Monitoring the patient
- Preventing complications
- Protecting the patient from injury
- Providing comfort
  - Offering brief explanations of what is happening even if it is uncertain the client hears or understands
  - Reassuring touches

Family - patient unlikely to survive

- Needs to be informed about the prognosis and likely outcome when the client has reached the point where it is obviously unlikely to survive
- Throughout care family should be be provided opportunities to
  - See
  - Touch
  - And talk to the patient
- When possible and appropriate ask about
  - Living wills
  - Advance directives
  - Other written or verbal wishes the patient may have shared in the event they cant make end-of-life decisions
- Conferences with family for effecting understand and communication

    - This is done so the family does not misinterpret the continued care provided by the health care team as a possibility of recovery which can cause anger when the client passes.
    - "During these conferences, it is essential to explain that the equipment and treatments being provided are intended for patient comfort and do not suggest that the patient will recover. Family members should be encouraged to express their views of life-support measures." (Ch 14, page 304)

17. **Tissue Perfusion**

- Observes for subtle changes in level of consciousness
- Vital Signs (Including pulse pressure)
- Urinary output
- RR
- Labs
    - Base deficit
    - Lactic acid levels
- Compensatory stage-release of aldosterone and catecholamines
    - Elevated serum sodium
    - Elevated blood glucose
- Blood cultures obtained in suspicion of infection prior to antibiotics
- Monitor hemodynamic status- BP indicator of hypoxia
- Assist in identifying and treating the underlying disorder by continuous in-depth assessment
- Administer prescribed fluids and medications
- Promote safety
- Report systolic <90 or drop of 40 from baseline
- Infection- Notify provider if patient exhibits 2 or 3 of the following
    - RR greater than or equal to 22
    - Altered mental status
    - Systolic less than or equal to 100
- Normal pulse pressure 30-40
    - Narrowing or decreased early indicator of shock

18**. Vasoactive Agents Used in Shock**

- Administer via central venous line
- Inotropic Agents- improve contractility, increase stroke volume, increase cardiac output
    - Dobutamine (Dobutrex)
    - Dopamine (Intropin)
    - Epinephrine (Adrenalin)
    - Milrinone (Primacor)
- Vasodilator- reduce preload and afterload, reduce oxygen demand of heart
    - Nitroglycerin (Tridil)
    - Nitroprusside (Nipride)
- Vasopressor Agents- Increase blood pressure by vasoconstriction
    - Norepinephrine (Levophed)

- Dopamine (Intropin)
- Phenylephrine (Neo- Synephrine)
- Vasopressin (Pitressin)
- Epinephrine (Adrenalin)

19**. SIRS- Systemic inflammatory response to a variety of insults, including infection, ischemia, infarction, and injury**

a. s/s

Temperature greater than 101F or less than 96.8F

Heart rate greater than 90 BPM

Respirations greater than 20 breaths per minutes or PaCO2 greater than 32 mm Hg

b. Nursing Management

IF SEPTIC:

Secure airway and correct hypoxemia

Establish venous access

Obtain CBC serum lactate, ABG, blood culture, imaging targeted at the suspected site of infection, procalcitonin

Aggressive administration of IVF given at 30 mL/Kg w? In the first 3 hours of presentation

Empiric antibiotic therapy administered within the first hour

IF SEPTIC SHOCK:

IV vasopressors- norepinephrine

Glucocorticoids if shock refractory to adequate fluid restriction and vasopressor administration

20**. MODS**

a. s/s

Severe dyspnea

Tachycardia

Myocardial depression

Massive vasodilation

Decreased SVR, BP, MAP

Increase heart rate

Biventricular failure

Acute change in neurologic status- Confusion, disorientation, delirium

Fever

Seizures

Failure to wean, prolonged rehabilitation

Hyperglycemia

GI bleeding

Hypoperfusion- decrease peristalsis, paralytic ileus

Increase d-dimer

Increases PT & PTT

Decreased platelet count

b. Nursing Management

General nursing plan of care is the same as that for patients with septic shock

Primary nursing interventions are aimed at supporting the patient and monitor organ perfusion until primary organs insults are halter.

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 patients wishes

Patients who survive MOD must be informed about the goals of rehabilitation and expectations for progress towards these goals because massive loss of skeletal muscle mass makes rehabilitation a long, slow process