Week 5: Breanna Schoonover

Fluid volume deficit

Manifestations

- Increased HR, decreased BP, decreased CVP, edema, increased body weight, & imbalances in fluid intake and output (I&O)

Imbalance	Contributing Factors	S/Sx & Laboratory Findings
Fluid Volume Deficit (hypovolemia)	Loss of water and electrolytes via: Vomiting Diarrhea Fistulas Fever Excess sweating Burns Blood loss GI suction Third-space fluid shifts Decreased intake via: Anorexia Nausea 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

Diagnostic findings

- Laboratory data:
- BUN & its relation to serum Cr concentration
- Volume-depleted patient has BUN elevated out of proportion to serum Cr (ratio greater than 20:1)
- Hematocrit (Hct)
- Elevated d/t decreased plasma volume
- Serum electrolyte changes may also exist
- Hypokalemia & hyponatremia or hyperkalemia, hypernatremia
- Hypokalemia w/ GI & renal losses
- Hyperkalemia w/ adrenal insufficiency
- Hyponatremia w/ increased thirst & ADH release
- Hypernatremia w/ increased insensible losses & DI
- Urine specific gravity
- Increased d/t kidneys' attempt to conserve water and decreased w/ DI
- Urine sodium

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- Aldosterone is secreted when fluid volume is low causing reabsorption of sodium and chloride = decreased urinary sodium
- Urine osmolality

Fluid volume overload

- Manifestations

Imbalance	Contributing Factors	S/Sx & Laboratory Findings
Fluid Volume Excess (hypervolemia)	Compromised regulatory mechanisms: renal failure heart failure Cirrhosis Overzealous administration of sodium-containing fluids Fluid shifts (i.e., treatment of burns) Prolonged corticosteroid therapy Severe stress Hyperaldosteronism	• 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

Nursing interventions

- Measure I&O
- Daily weights
- Gain of 1 kg (2.2 lb) = gain of approx 1L fluid
- Assess breath sounds
- Monitor degree of edema in dependent parts of body (i.e. – feet, ankles, & sacral region)
- promote rest
- restrict sodium intake
- monitor parenteral fluid therapy
- administer appropriate medications
- document I&O and body weight changes

Complications

- Sodium imbalances (hypo/hyper)

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- Hypo: **Manifestations**: poor skin turgor, dry mucosa, headache, decreased salivation, decreased blood pressure, nausea, abdominal cramping, neurologic changes
- **Hyper: Manifestations**: thirst; elevated temperature; dry, swollen tongue; sticky mucosa; neurologic symptoms; restlessness; weakness
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- Potassium imbalances (hypo/hyper)
  - Manifestations
  - **Manifestations**: fatigue, anorexia, nausea, vomiting, dysrhythmias, muscle weakness and cramps, paresthesias, glucose intolerance, decreased muscle strength, DTRs
  - **Manifestations**: cardiac changes and dysrhythmias, muscle weakness with potential respiratory impairment, paresthesia, anxiety, GI manifestations

Nursing interventions

- **Nursing management**: assessment, severe hypokalemia is life-threatening, monitor ECG and ABGs, dietary potassium, nursing care related to IV potassium administration
- **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

- **Medical management**: increased dietary potassium, potassium replacement, IV for severe deficit
- **Medical management**: monitor ECG, limitation of dietary potassium, cation-exchange resin (Kayexalate), IV sodium bicarbonate, IV calcium gluconate, regular insulin and hypertonic dextrose IV, β-2 agonists, dialysis
- Insulin & glucose
- Sodium bicarbonate, primarily if metabolic acidosis
- Beta-2 adrenergic agonists (albuterol

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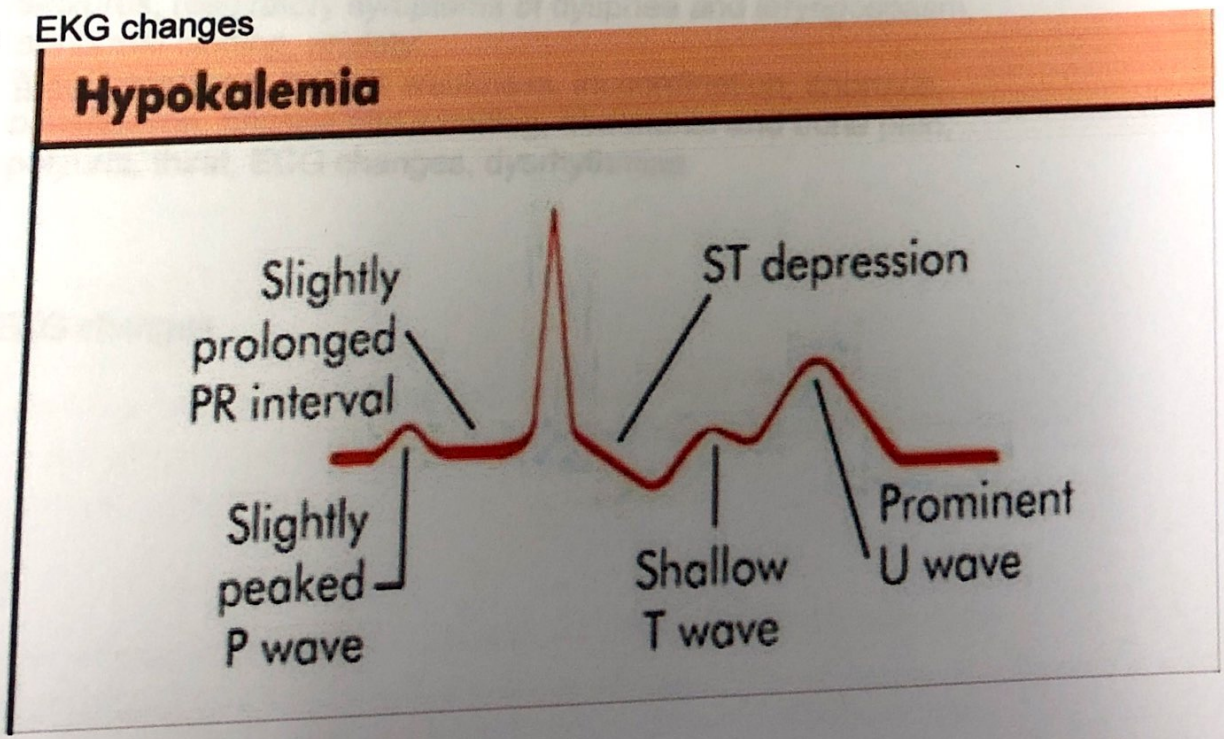
- **Antagonism of membrane action potentials**
  - **Calcium**
  - **Calcium gluconate vs calcium chloride → calcium chloride contains 3x the concentration of elemental calcium however calcium gluconate is generally preferred d/t calcium chloride causing local irritation at the injection site**
- **Removal 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

Patient education

- EKG changes



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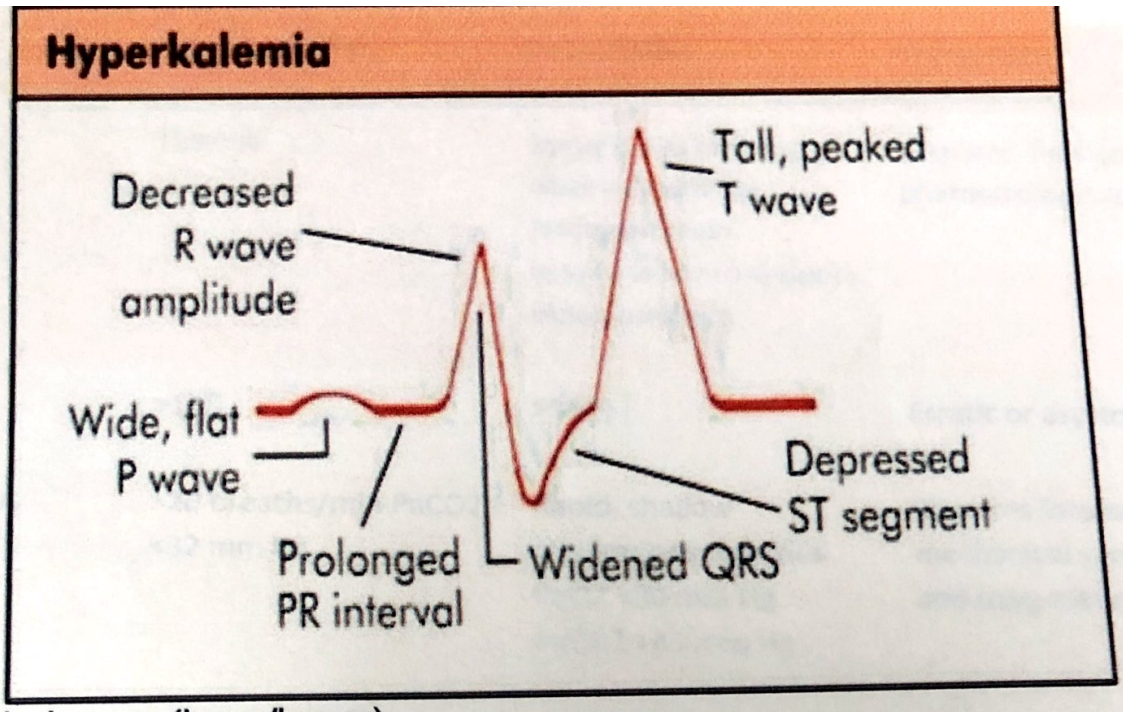

- Calcium imbalances (hypo/hyper)

Manifestations

- **Manifestations**: tetany, circumoral (around the mouth) numbness, paresthesia, hyperactive DTRs, Trousseau's sign, Chovstek's sign, seizures, respiratory symptoms of dyspnea and laryngospasm, abnormal clotting, anxiety
- **Manifestations**: muscle weakness, incoordination, anorexia, constipation, nausea and vomiting, abdominal and bone pain, polyuria, thirst, ECG changes, dysrhythmias

- EKG changes

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- 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

Septic shock

Assessment Findings

- Persisting hypotension requiring vasopressors to maintain MAP ≥65 mm Hg
- Blood lactate >2 mmol/L despite adequate volume resuscitation
- Characterized by persistent hypotension despite adequate fluid resuscitation requiring vasopressors, along with inadequate tissue perfusion resulting in tissue hypoxia

Nursing interventions

- 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

MODS

Nursing Interventions

- Early detection & documentation of initial signs of infection are essential
- Subtle changes in mentation and a gradual rise in temperature are early warning signs

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- Other patients at risk for MODS are those with chronic illness, malnutrition, immunosuppression, or surgical or traumatic wounds
- If preventive measures fail, treatment measures to reverse MODS are aimed at (1) controlling the initiating event, (2) promoting adequate organ perfusion, (3) providing nutritional support, and (4) maximizing patient comfort
- 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

TOP Priority of MODS

- Prevention remains the top priority!

Cadriogenic Shock

Systolic dysfunction • **Inability of the heart to pump blood forward**	**Myocardial infarction, cardiomyopathy, blunt cardiac injury, severe systemic or pulmonary hypertension, myocardial depression from metabolic problems**
Diastolic dysfunction • Inability of the heart to fill	Cardiac tamponade, ventricular hypertrophy, cardiomyopathy
Dysrhythmias	Bradydysrhythmias, tachydysrhythmias

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Anaphylactic shock

- 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

Neurogenic

- Most important clinical manifestations are hypotension (from massive vasodilation) and bradycardia (from unopposed parasympathetic stimulation)

Hypovolemic shock

- Fluid & Blood Replacement
  - 2 large-gauge IV lines are inserted
    - Multiple IV lines allow simultaneous administration of fluid, medications, and blood component therapy if required
  - If an IV catheter cannot be quickly inserted, an intraosseous catheter (IO) may be used for 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 the intravascular compartment into the intracellular compartment
  - Crystalloid solutions (LR or NS) are commonly used to treat hypovolemic shock
  - If hypovolemia d/t blood loss → administer 3 mL of crystalloid solution for each mL of EBL (3:1)
  - Colloid solutions (e.g., albumin) may also be used
  - Blood productions (also colloids) may need to be administered if cause is hemorrhage

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Structural factors	Valvular stenosis or regurgitation, ventricular septal rupture, tension pneumothorax

- Systolic dysfunction
  - Heart's inability to pump the blood forward
  - Primarily affects the LV
  - If affects RV → blood flow through pulmonary circulation is reduced
  - Most common cause is acute MI
- Diastolic dysfunction
  - Heart's inability to fill
  - Causes include cardiac tamponade, ventricular hypertrophy, cardiomyopathy

Goals

- Goals are to:
- limit further myocardial damage
- preserve healthy myocardium
- improve cardiac function by increasing cardiac contractility, decreasing ventricular afterload, or both
- In general, these goals are achieved by increasing oxygen supply to the heart muscle while reducing oxygen demand
- Nursing Interventions
- Preventing cardiogenic shock
- Monitoring hemodynamic status
- Administering medications, IV fluids
- Maintaining intra-aortic balloon counterpulsation
- Ensuring safety, comfort

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- Decision to give blood is based on pt's lack of response to crystalloid resuscitation, the volume of blood lost, the need for hemoglobin to assist with oxygen transport, and the necessity to correct the patient's coagulopathy
- Pts who receive massive blood transfusions to achieve near-normal hemoglobin levels tend to have poorer outcomes than those with low hemoglobin levels (e.g., less than 7 g/dL)
- PRBCs are administered to replenish the patient's oxygen-carrying capacity in conjunction with other fluids that will expand volume
- Need for transfusions is based on pt's oxygenation needs, which are determined by vital signs, blood gas values, chemistry, coags, and clinical appearance rather than an arbitrary laboratory values

Shock

- Syndrome characterized by ***decreased or inadequate tissue perfusion*** and impaired cellular metabolism
- Results in an imbalance between the supply of and demand for O2 and nutrients
- Adequate blood flow to tissues/cells requires:
- (1) effective cardiac pump
- (2) adequate vasculature or circulatory system
- (3) sufficient blood volume

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