fluids & electrolytes

KEY ELEMENTS OF F&E

1. Cell Membrane
2. Body Fluid Composition
    a. Water
    b. Electrolyte
        b.1 Anions (-)
        b.2 Cations (+)

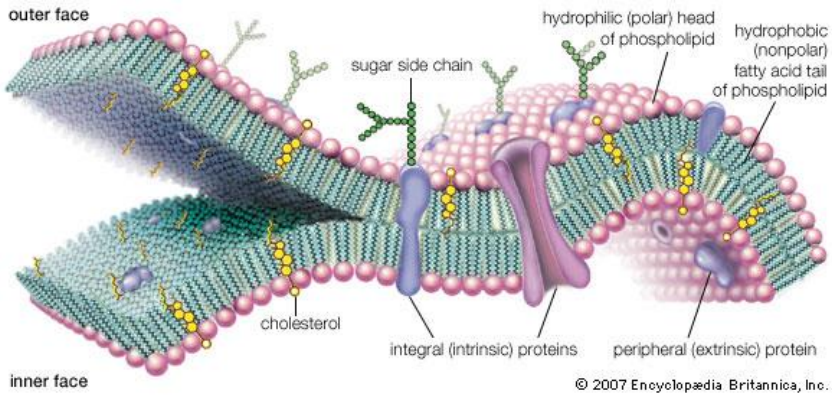
CELL MEMBRANE

Characteristic:

- Semipermeable membrane

Composition:

- Double phospholipid layer
- Proteins – second major component of the cell membrane
- Cell coat – long chains of complex carbohydrates (glycoproteins, glycolipids, and lectins); fxn: in cell-to-cell recognition and adhesion



BODY FLUID COMPOSITION

WATER	Medium for the transport and exchange of nutrients and other substances
	Medium for metabolic reactions
	Assists in regulating body temperature
	Total body water constitutes 60% of the total body weight
	45-50% Total body water decreases with obesity and aging

OTHER FUNCTIONS OF WATER

- Acts as a solvent in which solute are available for cell function
- Maintain blood volume
- Medium of waste excretion
- Cushion body parts from injury

FACTORS AFFECTING BODY WATER

1. Age
    Infant 70-80%
    Adult 50-60%
    Elderly 45-50%
2. Sex
    Male 60%
    Female 50%
3. Body fats
    Fat cells contain little water

NOTE: To maintain fluid balance, body water intake and output should be approximately equal. The average fluid intake and output is about 2500mL over a 24 hour period

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INSENSIBLE WATER LOSS

- Occurs through the skin, lungs, and feces
- Can increase significantly during: exercise; high environmental temperatures, during illness

ELECTROLYTES

- These are minerals in your body that have an electric charge. They are in your blood, urine, tissues, and other body fluids. Electrolytes help balance the amount of water in the body.

4 MAJOR FUNCTIONS

- ✓ Assist with regulation of water balance
- ✓ Regulating and maintaining acid-base balance
- ✓ Contributing to enzyme reactions
- ✓ Essential for neuromuscular activity

ELECTROLYTE DISTRIBUTION

Cations	Plasma	Interstitial	ICF
Sodium (Na+)	142	146	15
Potassium (K+)	5	5	150
Calcium (Ca++)	5	3	2
Magnesium (Mg++)	2	1	27

Anions	Plasma	Interstitial	ICF
Chloride (Cl)	102	14	1
Bicarb (HCO3)	27	30	10
Phosphate (HPO4-2)	2	2	100
Sulfate (SO4-2)	1	1	20
Organic Acid	5	8	0
Proteinate (Prot-)	16	1	73

ELECTRO-LYTE ION	DISTRIBUTION IN BODY FLUID		BASIC FUNCTIONS	DIETARY SOURCES
	ECF (mEq/L)	ICF (mEq/L)		
Sodium (Na+)	135-154	15-20	• regulates fluid volume within ECF compartment • regulates vascular osmotic pressure • controls water distribution between ECF and ICF compartments • participates in conduction of nerve impulses • maintains neuromuscular excitability	• table salt • cheese, milk, processed meat, poultry, shellfish, fish, eggs and foods preserved with salt (i.e. ham and bacon)
Potassium	3.5-5	150 - 155	• regulates osmolality of ICF • participates in transmission of nerve impulses • promotes contraction of skeletal and smooth muscles • regulates acid-base balance by cellular exchange of hydrogen ions	• Fruits, especially bananas, oranges, and dried fruits • vegetables • meats • nuts

ELECTRO-LYTE ION	DISTRIBUTION IN BODY FLUID		BASIC FUNCTIONS	DIETARY SOURCES
Calcium	4.5-5.5	1-2	• provides strength and durability to bones and teeth • establishes thickness and strength of cell membranes • promotes transmission of nerve impulses • maintains neuromuscular excitability • essential for blood coagulation • activates enzyme reactions and hormone secretions	• dairy products (milk, cheese, and yogurt • sardines, whole grains and green leafy vegetables
Magnesium	4.5-5.5	27-29	• activates enzyme systems, mainly those associated with vit. B metabolism and the use of K, Ca, and CHON • promotes regulation of serum Ca, P, Ca levels • promotes neuromuscular activity	• green leafy vegetables, whole grains, fish and nuts

SERUM COMPONENT	VALUES	
	Conventional	SI millimoles
Sodium	135-145 mEq/L	135-145 mmol/L
Chloride	98-106 mEq/L	98-106 mmol/L
Bicarbonate	22-26 mEq/L	22-26 mmol/L
Calcium	8.5- 10.0 mEq/L	2.1-2.6 mmol/L
Potassium	3.5-5.0 mEq/L	3.5-5.0 mmol/L
Phosphate/inorganic phosphorus	1.7-2.6 mEq/L (2.5-4.5 mg/dl)	0.8-1.5 mmol/L
Magnesium	1.6-2.6 mg/dl (1.3-2.1 mEq/L)	0.8-1.3 mmol/L
Serum osmolality	275-295 mOsm/kg (milliosmole)	275-295 mmol/kg

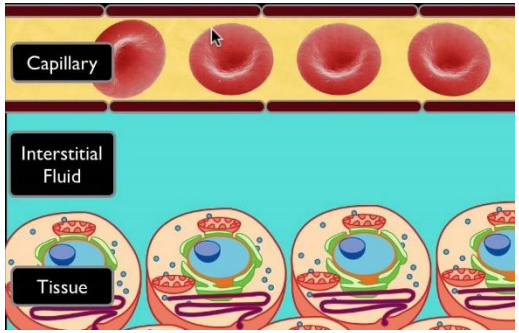
BODY FLUID COMPARTMENT

1. Intracellular
2. Extracellular
   a. Intravascular
   b. Interstitial
   c. Transcellular

BODY FLUID COMPARTMENT

- Body fluid is classified by its location inside or outside the cells.
- Capillary and cell membranes separate total body fluids into two main compartments: the intracellular and extracellular

Interstitial fluid



MECHANISM OF FLUID TRANSPORT

1. Active Transport
   a. Sodium Potassium Pump
   b. Endocytosis
      b.1 Phagocytosis
      b.2 Pinocytosis
      b.3 Receptor-Mediated endocytosis (RME)
   c. Exocytosis
2. Passive Transport
   a. Osmosis
   b. Diffusion
      b.1 Facilitated
      b.2 Simple
   c. Filtration
   d. Hydrostatic Pressure

ACTIVE TRANSPORT

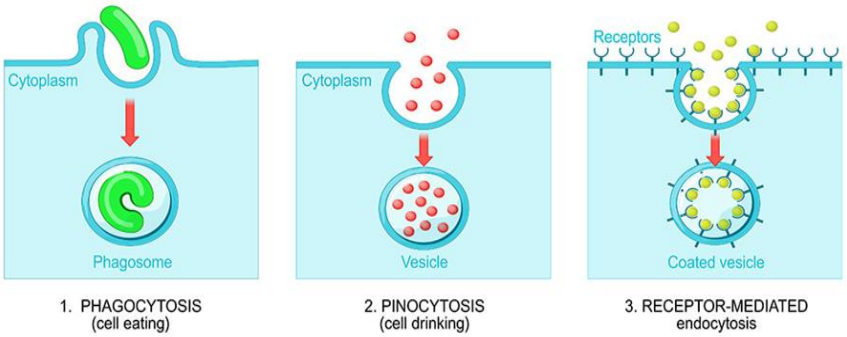
- Transport substances that are unable to pass by diffusion:
  - They may be too large
  - They may not be able to dissolve in the fat core of the membrane
  - They may have to move against a concentration gradient
- Two common forms of active transport:
  - Solute pumping
  - Bulk transport

SOLUTE PUMPING

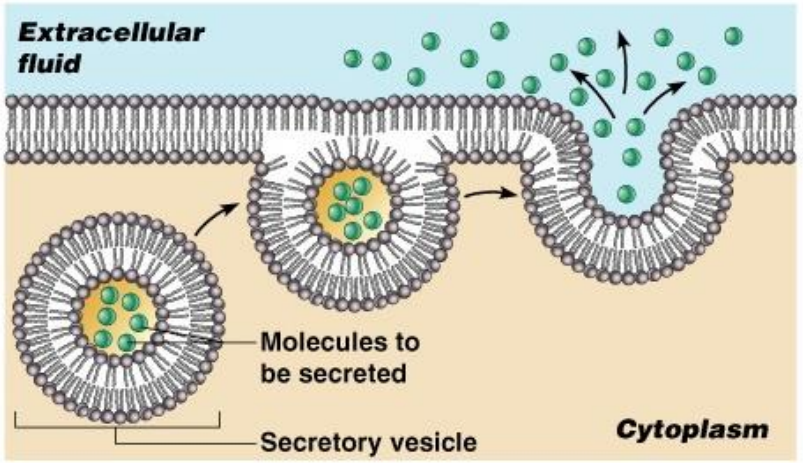
- Amino acids, some sugars and ions are transported by solute pumps
- ATP energizes protein carriers, and in most cases, moves substances against concentration gradients

BULK TRANSPORT

- Endocytosis
  - Extracellular substances are engulfed by being enclosed in a membranous vesicle
  - Types of endocytosis
    - Phagocytosis – cell eating; dissolved materials enter the cell → plasma membrane engulfs the solid material → phagocytic vesicle
    - Pinocytosis – cell drinking; plasma membrane folds inward to form a channel allowing dissolved substances to enter the cell → pinocytic vesicle
    - RME – a process by which cells absorb metabolites, hormones, proteins – and in some cases viruses – by the inward budding of the plasma membrane



- Exocytosis
  - Moves materials out of the cell
  - Material is carried in a membranous vesicle
  - Vesicle migrates to plasma membrane
  - Vesicle combines with plasma membrane
  - Material is emptied to the outside



PASSIVE TRANSPORT

A. Diffusion

- Simple diffusion
  - Unassisted process
  - Solutes are lipid-soluble materials or small enough to pass through membrane pores
- Facilitated diffusion
  - Substances require a protein carrier for passive transport

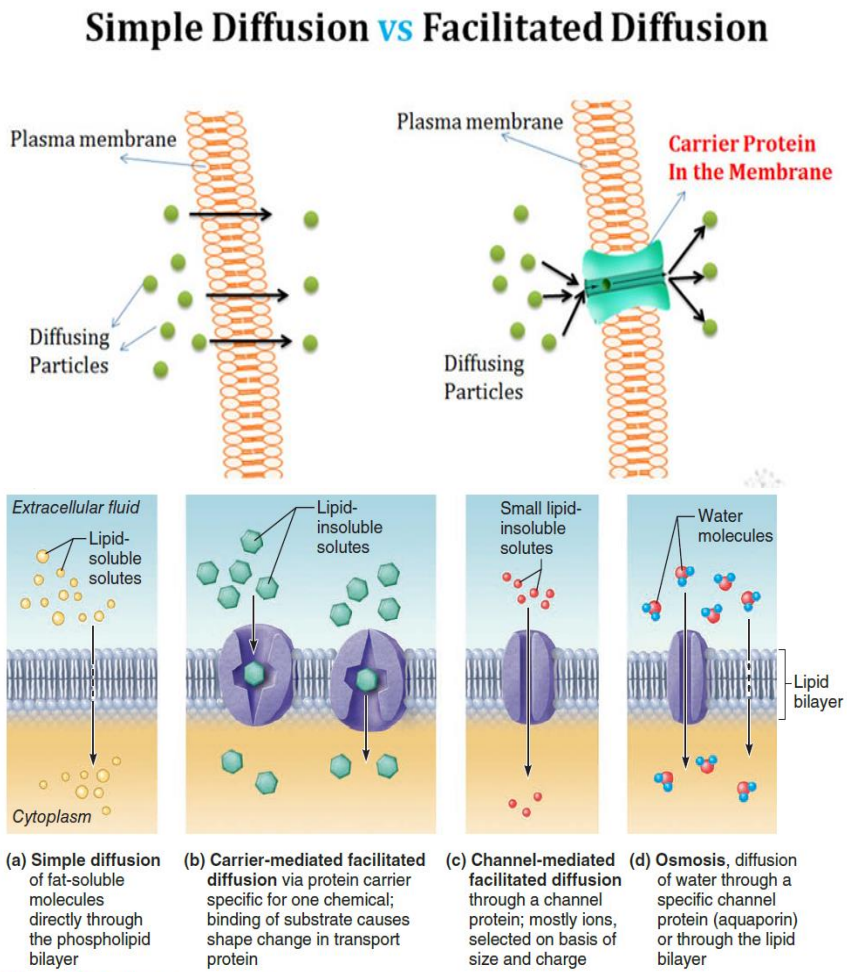
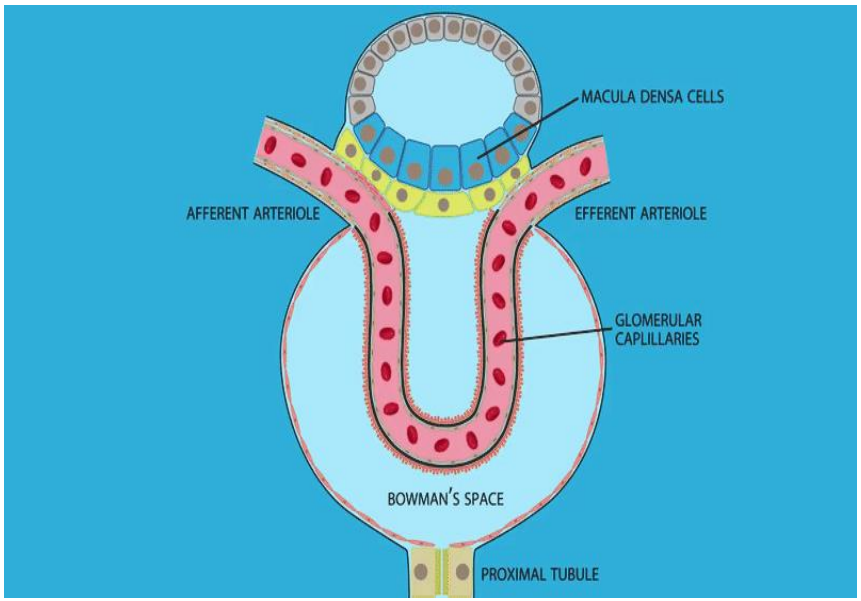


Figure 3.10 Diffusion through the plasma membrane.

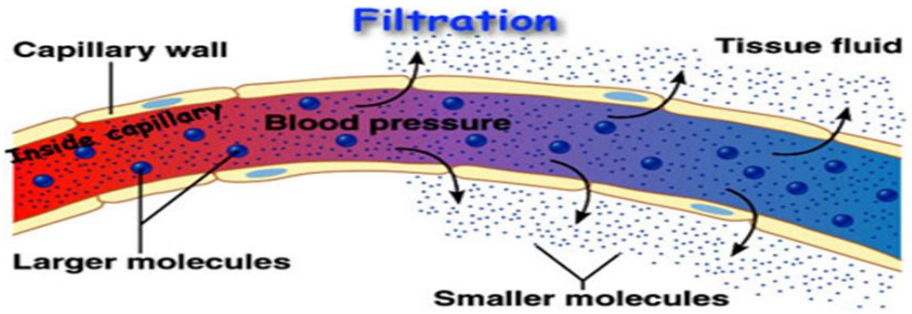
B. Filtration

- process by which water and dissolved substances (solutes) move from an area of high **hydrostatic pressure** to an area of low hydrostatic pressure
- usually occurs across capillary membranes



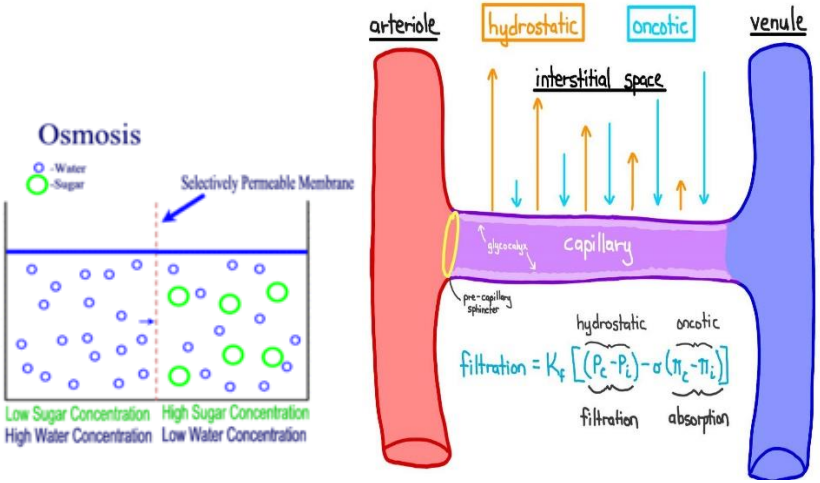
C. Hydrostatic pressure

- created by the pumping action of the heart and gravity against the capillary wall
- filtration occurs in the glomerulus of the kidneys, as well as at the arterial end of capillaries



D. Osmosis

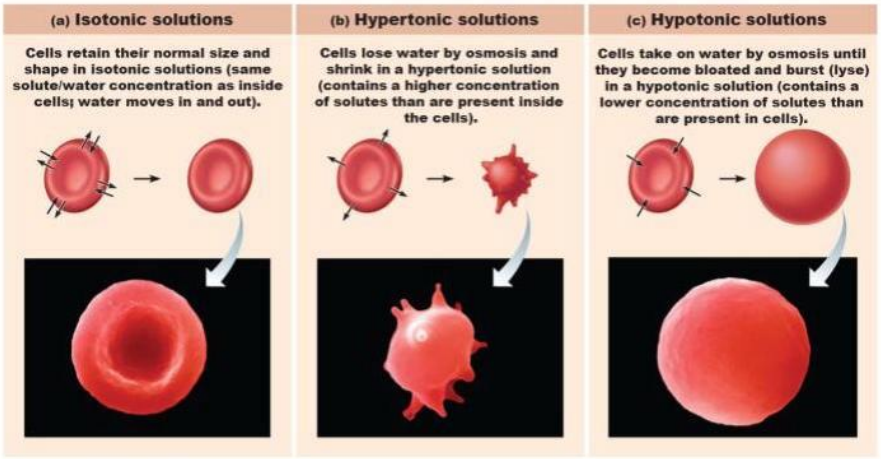
- process by which water moves across a selectively permeable membrane from an area of lower solute concentration to an area of higher solute concentration
- Osmolality - number of solutes per Liter of fluid
- Osmotic Pressure - power of a solution to draw water across a membrane



E. Oncotic Pressure

- also called colloid osmotic pressure (COP)
- osmotic pressure is exerted by plasma proteins in the vessels (e.g., albumin)
- proteins in the bloodstream exert oncotic pressure to pull fluid out of the interstitial space into the intravascular space to maintain fluid balance and osmolality
- average COP is 28mmHg, a pressure that remains constant across the capillary
- Tonicity: refers to the effect a solution's osmotic pressure has on water movement across the cell membrane of cells within that solution

TONICITY



ELECTROLYTE IMBALANCE

ELECTROLYTE

- substances that dissociate in solution to form charged particles called ions
- cations are positively charged electrolytes
- anions are negatively charged electrolytes
- Function:
  - assisting with the regulation of water balance
  - regulating and maintaining acid-base balance
  - contributing to enzyme reactions
  - essential for neuromuscular activity
- Major roles:
  - Maintain body fluid osmolality - regulate water distribution
  - Nervous System - Propagation of Action Potential
  - Cardiovascular System - Cardiac conduction & contraction

SODIUM

- one of the most important elements in the body
- accounts for 90% of extracellular fluid cations (positively charged ions) and is the most abundant solute in extracellular fluid
- 135 to 145 mEq/L
- maintain proper extracellular fluid osmolality (concentration)
- imbalances affect the osmolality of ECF and water distribution between the fluid compartments
- Hyponatremia → water is drawn into the cells of the body, causing them to swell
- Hypernatremia → draw water out of body cells, causing them to shrink
- kidney is the primary regulator of sodium balance in the body
- excretes or conserves sodium → changes in vascular volume

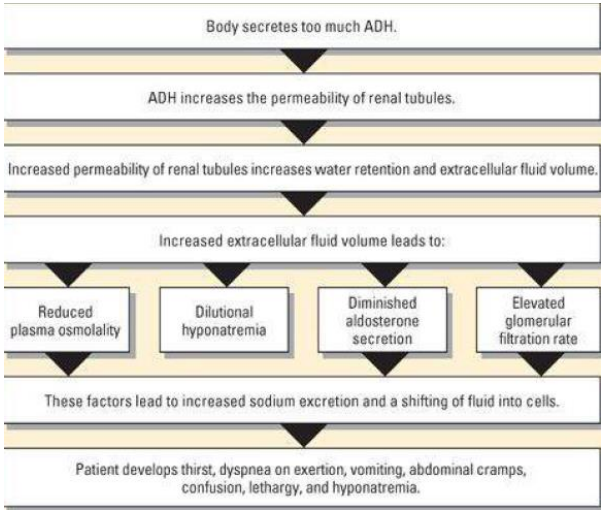
HYPONATREMIA

- common electrolyte imbalance
- usually results from a loss of sodium from the body
- may also be caused by water gains that dilute extracellular fluid (ECF)

CLASSIFICATIONS OF HYPONATREMIA

- Hypovolemic - both sodium and water levels decrease in the extracellular area, but sodium loss is greater than water loss
  → Causes: vomiting, diarrhea, fistulas, gastric suctioning, excessive sweating, cystic fibrosis, burns, wound drainage, osmotic diuresis, adrenal insufficiency, diuretic use
- Hypervolemic - both water and sodium levels increase in the extracellular area, but the water gain is more impressive
  → Causes: heart failure, liver failure, nephrotic syndrome, excessive administration of hypotonic IV fluids, hyperaldosteronism
- Isovolemic - sodium levels may appear low because too much fluid is in the body; Patients may not exhibit signs of fluid volume excess, and total body sodium remains stable
  → glucocorticoid deficiency, hypothyroidism, renal failure

PATHOPHYSIOLOGY (ADH)



MANIFESTATIONS (HYPONATREMIA)

- depend on the rapidity of onset, the severity, and the cause of the imbalance
- If the condition develops slowly, manifestations are usually not experienced until the serum sodium levels reach 125 mEq/L
- Poor skin turgor
- Dry mucosa
- Decrease saliva production
- Orthostatic hypotension
- Nausea, abdominal cramping
- Neurologic changes such as: lethargy, confusion, signs of increase ICP, muscle twitch, seizure

DIAGNOSTIC (HYPONATREMIA)

- Serum osmolality less than 280 mOsm/kg (dilute blood)
- Serum sodium level less than 135 mEq/L (low sodium level in blood)
- Urine specific gravity less than 1.010
- Elevated hematocrit and plasma protein levels

MEDICAL MANAGEMENT (HYPONATREMIA)

- Hypervolemia/Isovolemia
  - Fluid restrictions
  - Oral sodium supplements
- Hyponatremia
  - Give isotonic IV fluids (e.g. normal saline)
  - Offer High sodium foods
- In SEVERE cases:
  - Infusion of hypertonic saline solution (3% or 5% saline)

NURSING MANAGEMENT (HYPONATREMIA)

- Monitor and record vital signs, especially blood pressure and pulse, and watch for orthostatic hypotension and tachycardia
- Monitor neurologic status frequently → Report any deterioration in level of consciousness.
- Accurately measure and record intake and output.
- Assess skin turgor at least every 8 hours for signs of dehydration.
- Restrict fluid intake as ordered (fluid restriction is the primary treatment for dilutional hyponatremia).
- Administer oral sodium supplements as ordered.
- Provide a safe environment for patients with altered thought processes
- Seizure precautions

HYPERNATREMIA

- Hypernatremia, a less common problem than hyponatremia, refers to excess of sodium relative to the amount of water in the body

PATHOPHYSIOLOGY (HYPERNATREMIA)

- The cells play a role in maintaining sodium balance. When serum osmolality increases because of hypernatremia, fluid moves by osmosis from inside the cell to outside the cell. As fluid leaves the cells, they become dehydrated and shrink.

MANIFESTATIONS (HYPERNATREMIA)

- Thirst is the first manifestation
- If thirst is not relieved, the primary manifestations relate to altered neurologic function (lethargy, weakness, and irritability can progress to seizures, coma, and death in severe hypernatremia)
- Low grade fever
- Flushed skin
- Dry mucous membranes
- Oliguria
- Orthostatic hypotension

DIAGNOSTIC (HYPERNATREMIA)

- Serum sodium level: >145 mEq/L
- Serum osmolality: >295 mOsm/kg

MEDICAL MANAGEMENT (HYPERNATREMIA)

- Treatment for hypernatremia varies with the cause. The underlying disorder must be corrected, and serum sodium levels and related diagnostic tests must be monitored.
- Note that the fluids should be given gradually over 48 hours to avoid shifting water into brain cells.

NURSING MANAGEMENT (HYPERNATREMIA)

- Intravenous fluid replacement (dextrose 5% in water) → to return serum sodium levels to normal
- Sodium intake restrictions
- Administer diuretics to increase sodium loss
- Monitor and record vital signs, especially blood pressure and pulse
- Check neurologic status frequently.
- Report any deterioration in the level of consciousness
- Carefully measure and record intake and output.
- Assess skin and mucous membranes for signs of breakdown and infection
- Monitor serum sodium level and report any increase.
- Assist with oral hygiene. Lubricate the patient's lips frequently.
- Provide a safe environment for confused or agitated patients

POTASSIUM

- major cation (ion with a positive charge) in intracellular fluid
- affects nerve impulse transmission
- 3.5 to 5.0 mEq/L
- Aldosterone helps regulate potassium elimination by the kidneys
- Function: (Potassium directly affects how well the body cells, nerves, and muscles function by:)
  - Maintaining cells' electrical neutrality and osmolality
  - Aiding neuromuscular transmission of nerve impulses
  - Assisting skeletal and cardiac muscle contraction and electrical conductivity
  - Affecting acid-base balance in relationship to the hydrogen ion.

HYPOKALEMIA

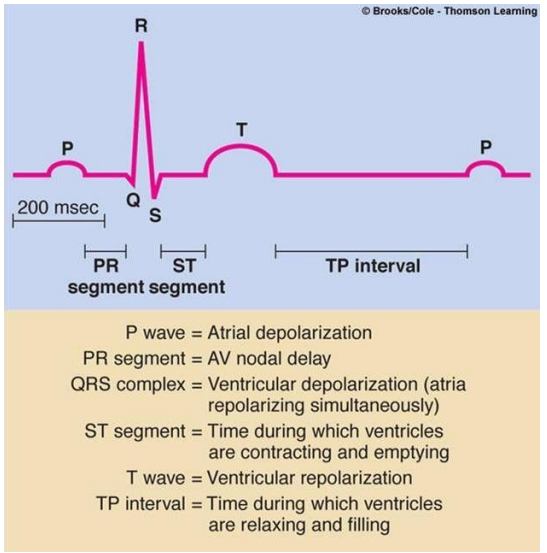
- an abnormally low serum potassium level
- usually results from excess potassium loss
- hospitalized patients may be at risk for hypokalemia because of inadequate potassium intake

PATHOPHYSIOLOGY (HYPOKALEMIA)

- Inadequate intake and excessive output of potassium can cause a moderate drop in its level
- Causes:
  - Inadequate potassium intake
  - Severe GI losses, e.g. suction, lavage, prolonged vomiting can deplete the body's potassium supply as a result potassium levels drop
  - Drug associated, e.g. diuretics,(esp. thiazides and furosemides), corticosteroids, insulin
  - Decrease bowel motility

MANIFESTATIONS (HYPOKALEMIA)

- Skeletal muscle weakness, especially in the legs → a sign of a moderate loss of potassium. This also includes paresthesia and leg cramps
- Decreased bowel sounds, constipation, paralytic ileus
- Weak and irregular pulse
- Orthostatic hypotension and palpitations
- ECG changes, flattened or inverted T wave, prominent U wave



LABORATORY FINDINGS (HYPOKALEMIA)

- The following test results may develop to confirm the diagnosis of hypokalemia:
  - Serum potassium level <3.5 mEQ/L
  - Increased 24-hour urine level
  - Characteristic ECG changes

MEDICAL MANAGEMENT (HYPOKALEMIA)

- Identify and treat the cause → treatment for hypokalemia focuses on restoring a normal potassium balance, preventing serious complications, and removing or treating the underlying causes
- Oral and IV replacements (40-80 meq/day – Kalium durule or K IV), it can be safely given at the same time

NURSING MANAGEMENT (HYPOKALEMIA)

- Monitor vital signs, especially blood pressure → hypokalemia is commonly associated with hypovolemia, which can cause orthostatic hypotension
- Check heart rate and rhythm and ECG tracings in patients with serum potassium level less than 3 meQ/L → hypovolemia causes tachyarrthymias
- Assess respiratory rate, depth and pattern → hypokalemia may weaken or paralyze respiratory muscles. Notify the doctor immediately if respirations become shallow and rapid
- Monitor serum potassium levels→ changes in serum potassium level can lead to serious cardiac complications
- Administer potassium infusions cautiously

HYPERKALEMIA

- Hyperkalemia can result from inadequate excretion of potassium, excessively high intake of potassium, or a shift of potassium from the ICF to the ECF. Hyperkalemia affects neuromuscular and cardiac function

CAUSES OF HYPERKALEMIA

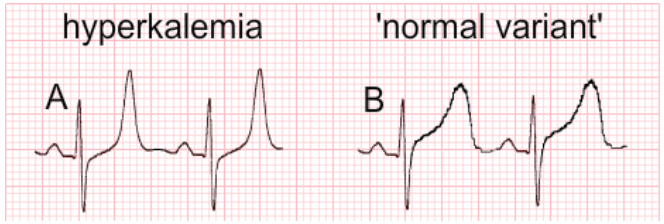
- Increased dietary intake
- Blood transfusion (stored blood)→ serum potassium level increases longer the blood is stored.
- Rapid IV potassium administration
- Renal insufficiency
- Metabolic acidosis, potassium shift to ECF in exchange of H ions

PATHOPHYSIOLOGY (HYPERKALEMIA)

- Potassium move from the extracellular to the intracellular compartment and increases cell excitability, so that cells respond to stimuli of less intensity and may actually discharge independently without a stimulus

MANIFESTATIONS (HYPERKALEMIA)

- Signs and symptoms of hyperkalemia reflect its effects on neuromuscular and cardiac functioning in the body
  - Nausea, abdominal cramping and diarrhea→ the early signs of hyperkalemia due to smooth muscle hyperactivity
  - Muscle weakness that in turn lead to flaccid paralysis
  - Decreased heart rate, irregular pulse, decreased cardiac output, hypotension
  - ECG changes, elevated T wave (Tall or peaked T wave)



LABORATORY FINDINGS (HYPERKALEMIA)

- Serum potassium level >5 mEq/L
- Decreased arterial pH, indicating acidosis
- ECG abnormalities

MEDICAL MANAGEMENT (HYPERKALEMIA)

- Treatment for hyperkalemia is aimed at lowering the potassium level, treating its cause, stabilizing the myocardium, and promoting renal and gastrointestinal excretion of potassium. The severity of hyperkalemia dictates how it should be treated.
  - Diet restrictions
  - May administer loop diuretics for mild hyperkalemia → to increase potassium loss from the body or to resolve any acidosis present
  - Sodium polystyrene sulfonate (Kayexalate) → a cation-exchange resin (common treatment for hyperkalemia)
  - Hemodialysis, if patient has renal failure

NURSING INTERVENTIONS (HYPERKALEMIA)

- Assess vital signs
- Monitor the patient's intake and output→ Report an output of less than 30 mL/hour.
- Prepare to administer a slow calcium chloride or gluconate IV infusion to counteract the myocardial depressant effects of hyperkalemia
- Keep in mind when giving Kayexalate that serum sodium levels may rise. Watch for signs of heart failure.
- Monitor ECG changes

CALCIUM

- 4.5-5.5 mEq/L
- Calcium is a positively charged ion or cation
- found in both extracellular and intracellular fluid
- About 99% of the body's calcium is found in the bones and teeth
- Only 1% is found in serum and in soft tissue
- Function:
  - Skeletal and heart muscle relaxation, activation, excitation and contraction
  - Maintains cellular permeability
  - Promotes blood coagulation
  - Nerve impulse transmission
- three forms of calcium in the body:
  - 45% is bound to protein, mostly albumin
  - 40% is ionized calcium, it is the calcium that is physiologically active and clinically important for neuromuscular transmission
  - 15% is bound to other substances such as phosphate, citrate, or carbonate
- system interactions:
  - Parathyroid Hormone (PTH) → raises the plasma calcium
  - Calcium is dependent upon calcitriol, the most active from of vitamin D
  - Calcitonin, a calcium-lowering hormone produced by the thyroid gland

HYPOCALCEMIA

- Hypocalcemia can result from decreased total body calcium stores or low levels of extracellular calcium with normal amounts of calcium stored in bone. The systemic effects of hypocalcemia are caused by decreased levels of ionized calcium in extracellular fluid.

CAUSES (HYPOCALCEMIA)

- Inadequate intake
- Hypoparathyroidism, resulting from surgery (parathyroidectomy, thyroidectomy, radical neck dissection)
- Electrolyte imbalances → Low serum albumin (most common cause)

- Malabsorption → can result from increased intestinal motility
- Vitamin D deficiency → due to lack sun exposure or malabsorption
- Massive blood transfusion, liver unable to metabolize citrate (added to prevent clotting)
- Chronic diarrhea
- Diuretic phase of ARF
- Severe burns

PATHOPHYSIOLOGY (HYPOCALCEMIA)

- Extracellular calcium acts to stabilize neuromuscular cell membranes. This effect is reduced in hypocalcemia, increasing neuromuscular irritability. Thus, electrical activity occurs spontaneously and continuously.

MANIFESTATIONS (HYPOCALCEMIA)

- Signs and symptoms of hypocalcemia reflect calcium's effects on nerve transmission and muscle and heart functions; therefore, neuromuscular and cardiovascular findings are most evident
  - Anxiety, confusion, and irritability that can progress to seizures
  - Paresthesia, muscle twitching, cramps or tremors
  - Diarrhea
  - Hyperactive tendon reflexes
  - Fractures may occur
  - Brittle nails, dry skin and hair
  - Decreased cardiac output and subsequent arrhythmias
  - Prolonged QT segment on electrocardiogram (ECG)

TROUSSEAU'S SIGN

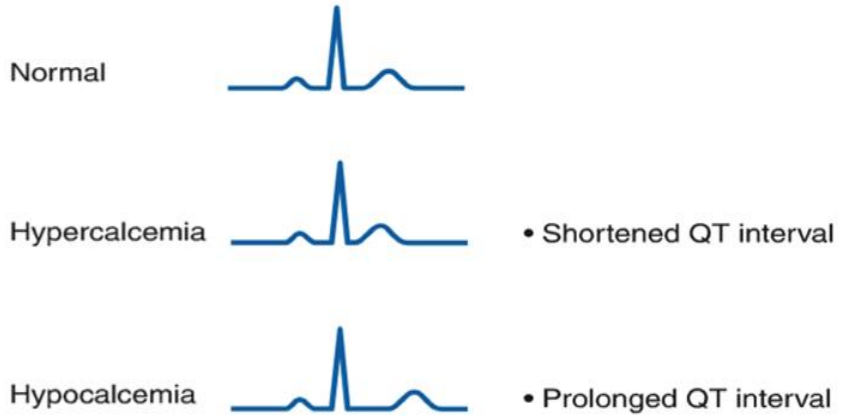
- *carpal spasm induced by inflating a blood pressure cuff on the upper arm to above systolic blood pressure for 2 to 5 minutes due to increase nerve excitability*

CHVOSTEK'S SIGN

- *contraction of facial muscles produced by tapping the facial nerve in front of the ear*

LABORATORY FINDINGS (HYPOCALCEMIA)

- Elevated total serum calcium level
- Low Ionized calcium level (ionized calcium measurement is the definitive method to diagnose hypocalcemia)
- Low albumin level
- Characteristic ECG changes, prolonged ST segment



MEDICAL MANAGEMENT

- Treatment for hypocalcemia focuses on correcting the imbalance as quickly and safely as possible. The underlying cause should be addressed to prevent recurrence.
  - Dietary supplement, e.g. milk, cheese, cereal
  - Oral/IV calcium, e.g. Calcium carbonate/gluconate
  - Vitamin D supplements, facilitates GI absorption of calcium

NURSING MANAGEMENT (HYPOCALCEMIA)

- Monitor vital signs
- Place patient on a cardiac monitor, and evaluate for changes in heart rate and rhythm
- Check for Chvostek's and Trousseau's sign
- Serum levels monitoring

HYPERCALCEMIA

- Hypercalcemia is a common metabolic emergency that occurs when serum calcium level rises, or the rate of calcium entry into extracellular fluid exceeds the rate of calcium excretion by the kidneys

CAUSES OF HYPERCALCEMIA

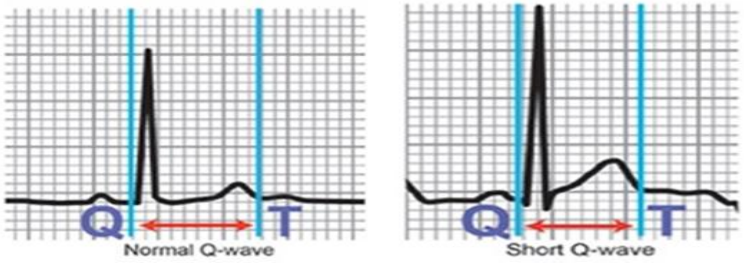
- Hyperparathyroidism (most common cause) the body excretes more PTH than normal, which greatly strengthens the effects of the hormone
- Metastatic malignancy→ causes bone destruction as malignant cells invade the bones and cause the release of a hormone similar to PTH
- Thiazide diuretics → potentiates PTH and decrease excretion in kidneys
- Increase Vitamin D (can prompt an increase in serum calcium levels) & prolong use of alkaline antacid (calcium carbonate)
- Prolong immobility

MANIFESTATIONS (HYPERCALCEMIA)

- Muscle weakness
- Bradycardia
- Shortened QT interval
- Decreased gastrointestinal motility (anorexia, n/v)
- Stone formation

LABORATORY FINDINGS (HYPERCALCEMIA)

- Elevated serum calcium level
- Elevated Ionized calcium level
- Digoxin toxicity if patient is taking digoxin
- Characteristic ECG changes (Shortened QT interval)



MEDICAL MANAGEMENT (HYPERCALCEMIA)

- ECG monitoring
- Treat with IV saline→ the sodium in the solution is typically used for hydration in these cases
- Loop diuretics (Lasix)→ promotes calcium excretion
- Administer Calcitonin (IM/SC)

NURSING MANAGEMENT (HYPERCALCEMIA)

- Be sure to monitor the calcium levels of patients who are at risk for hypercalcemia, such as those who have cancer or parathyroid disorders, are immobile, or are receiving a calcium supplement. For a patient who develops hypercalcemia, you can take the following actions:
  - Monitor vital signs
  - Watch the patient for arrhythmias
  - ECG monitoring
  - Calcium levels monitoring
  - Encourage Mobilization
  - Dietary limits of vitamin D/Calcium

MAGNESIUM

- 1.5-2.5 mEq/L
- Promotes enzyme reactions within the cell during carbohydrate metabolism
- Influences vasodilation and irritability and contractility of the cardiac muscles, thereby helping the cardiovascular system function normally
- Aids in neurotransmission and hormone-receptor binding
- Makes production of parathyroid hormone possible

HYPOMAGNESEMIA

- Hypomagnesemia is a common problem in critically ill patients. It is may be caused by deficient magnesium intake, excessive losses, or a shift between the intracellular and extracellular compartment

CAUSES OF HYPOMAGNESEMIA

- Poor dietary intake of magnesium
- Poor magnesium absorption by the GI tract
- Excessive magnesium loss from the GI tract
- Excessive magnesium loss from the urinary tract

PATHOPHYSIOLOGY (HYPOMAGNESEMIA)

- Hypomagnesemia causes increased neuromuscular excitability which commonly occurs with hypokalemia and hypocalcemia

MANIFESTATIONS (HYPOMAGNESEMIA)

- Signs and symptoms of hypomagnesemia can range from mild to life-threatening. Generally speaking, your patient's signs and symptoms may resemble those you would see with a potassium or calcium imbalance. However, you can't always count on detecting hypomagnesemia from clinical findings alone
  - Altered level of consciousness, tetany, seizures
  - Emotional lability
  - Vomiting
  - Tremors, twitching, tetany, hyperactive deep tendon reflexes
  - Rapid heart rate
  - Chvostek's and Trousseau's sign

LABORATORY FINDINGS (HYPOMAGNESEMIA)

- Serum magnesium level below 1.5 mEq/L (possibly with a below normal serum albumin level)
- Low potassium and calcium level
- Characteristic ECG changes
- Elevated serum levels of digoxin in a patient receiving the drug

MEDICAL MANAGEMENT (HYPOMAGNESEMIA)

- Treatment for hypomagnesemia depends on the underlying cause of the condition and the patient's clinical findings:
  - Dietary management, (green leaf vegetables, nuts, legumes, seafood, wholegrain, chocolates)
  - Intravenous infusion/ intramuscular injection, before magnesium administration, renal function should be assessed

NURSING MANAGEMENT (HYPOMAGNESEMIA)

- Assess the patient's mental status and report changes
- Evaluate the patient's neuromuscular status regularly by checking for hyperactive Deep Tendon Reflexes, tremors, and tetany.
- Check for Chvostek's and Trousseau's sign
- Monitor patients who have lost an excessive amount of fluid, they are at risk for magnesium deficiency
- Urine output should be monitored at least every 4 hours. Magnesium generally isn't administered if urine output is less than 100 mL in 4 hours.
- If patient id receiving digoxin, monitor him closely for signs and symptoms of digoxin toxicity (such as nausea, vomiting and bradycardia
- During magnesium replacement, check the cardiac monitor frequently and assess the patient closely for signs of magnesium excess, such as hypotension and respiratory distress. Keep calcium gluconate at the bedside in case signs occur

HYPERMAGNESEMIA

- Having too much magnesium in the serum can be just as bad as having too little. Hypermagnesemia occurs when the body's serum magnesium level rises above the normal range. However, hypermagnesemia is uncommon; typically, the kidneys can rapidly reduce the amount of excess magnesium in the body.

CAUSES OF HYPERMAGNESEMIA

- Impaired magnesium excretion, e.g. renal dysfunction, most common cause of hypermagnesemia
- Excessive magnesium intake

PATHOPHYSIOLOGY (HYPERMAGNESEMIA)

- Elevated serum magnesium levels suppress cellular excitability, resulting to muscular flaccidity and suppression of electrical impulses

LABORATORY FINDINGS (HYPERMAGNESEMIA)

- Serum magnesium level above 2.5 mEq/L
- ECG changes (prolonged PR interval, widened QRS complex, tall T wave.

MEDICAL MANAGEMENT (HYPERMAGNESEMIA)

- Correct underlying cause
- Intravenous infusion of calcium gluconate, to antagonize magnesium effect

NURSING MANAGEMENT (HYPERMAGNESEMIA)

- Monitor vital signs
- Checked for flushed skin and diaphoresis
- Check for deep tendon reflexes
- Evaluate for changes in mental status

PHOSPHOROUS

- The primary anion, or negatively charged ion, found in the intracellular fluid. It's contained in the body as phosphate.
- 1.2 to 3.0 mEq/L
- Integral part of acid base buffer system
- Regulate ATP use for muscle contraction, nerve transmission, electrolyte transport
- Regulates 2,3 DPG (diphosphoglycerate) a compound in red blood cells that facilitates oxygen delivery from the red blood cells to the tissues.
- Regulates 2,3 DPG (diphosphoglycerate) a substance in RBC affecting oxygen affinity

HYPOPHOSPHATEMIA

- Hypophosphatemia occurs when the serum phosphorous level falls below 1.2 mEq/L. although this condition generally indicates a deficiency of phosphorous, it can occur under various circumstances when total body phosphorous stores are normal

CAUSES OF HYPOPHOSPHATEMIA

- Respiratory alkalosis → one of the most common cause of hypophosphatemia, can stem from a number of conditions that produce hyperventilation
- Malabsorption syndromes
- Diuretic use, (Thiazides, Loop diuretics, Acetazolamide)

PATHOPHYSIOLOGY (HYPOPHOSPHATEMIA)

- Most effects of hypophosphatemia result from depletion of ATP and impaired oxygen delivery to the cells due to a deficiency of the red blood cell enzyme 2,3-DPG. Severe hypophosphatemia affects virtually every major organ system

MANIFESTATIONS (HYPOPHOSPHATEMIA)

- Muscle weakness, most common symptom
- malaise, weakened hand grasp, myalgia (pain in the muscles)
- Rhabdomyolysis (skeletal muscle destruction), can occur with altered muscle cell activity
- Osteomalacia/ Fracture, due to loss of bone density
- Bruising and bleeding

LABORATORY FINDINGS (HYPOPHOSPHATEMIA)

- serum phosphorous level less than 1.2 mEq/L
- elevated Creatinine Kinase level if Rhabdomyolysis is present
- X-ray studies that reveal skeletal changes typical of osteomalacia or bone fractures
- Abnormal electrolytes (decreased magnesium levels and increased calcium levels)

MEDICAL MANAGEMENT (HYPOPHOSPHATEMIA)

- Treat the underlying cause
- Oral and intravenous supplements

NURSING INTERVENTIONS (HYPOPHOSPHATEMIA)

- Monitor vital signs.
- Assess the patient frequently for evidence of decreasing muscle strength, such as weak hand grasps or slurred speech, and document findings regularly.
- Assist in ambulation and activities of daily living, if needed, and keep essential objects near the patient to prevent accidents.

HYPERPHOSPHATEMIA

- Hyperphosphatemia is a serum phosphate level greater than 4.5mg/dL. As with other electrolyte imbalances, it may be the result of impaired phosphate excretion, excess intake, or a shift of phosphate from the intracellular space into extracellular fluids.

CAUSES OF HYPERPHOSPHATEMIA

- Renal Failure
- Hypoparathyroidism→ impairs less synthesis of parathyroid hormone(PTH), when less PTH is synthesized, less phosphorous is excreted from the kidneys.
- Respiratory acidosis
- Increase tissue breakdown

PATHOPHYSIOLOGY (HYPERPHOSPHATEMIA)

- Effects of high phosphorous level are actually due to hypocalcemia; calcium suppresses cellular excitability

MANIFESTATIONS (HYPERPHOSPHATEMIA)

- Hyperphosphatemia causes few symptoms (tetany, tissue calcification)
- Symptoms occurring result from decrease calcium secondary to reciprocity

LABORATORY FINDINGS (HYPERPHOSPHATEMIA)

- Serum phosphorous above normal
- Low serum calcium level

MEDICAL MANAGEMENT (HYPERPHOSPHATEMIA)

- Treat underlying cause
- Restrict dietary intake
- Administer phosphate binding antacid, this may decrease absorption of phosphorous in the gastrointestinal system

NURSING MANAGEMENT (HYPERPHOSPHATEMIA)

- Monitor vital signs
- Monitor intake and output.(if urine output falls below 30 mL/hour, immediately notify the doctor) → Decreased urine output can seriously affect renal clearance of excess serum phosphorous
- Monitor serum phosphorous and calcium levels
- Monitor signs of tetany, such as positive Trousseau's and Chvostek's sign

FLUID SHIFTS

FLUID SHIFTS

- Plasma to Interstitial
  - Edema – palpable swelling produced by expansion of the interstitial fluid volume
    - May be localized or generalized; pitting or nonpitting, depending on the cause
    - Can cause fluid shifts in other VULNERABLE areas of the body → termed as third space shifts
    - Where can your fluid shift?
      - PLEURAL SPACE (PERITONEAL SPACE)
- Interstitial to Plasma
  - Movement back of edema to circulatory volume
  - e.g. excessive administration of hypertonic solution

MAJOR CAUSES OF EDEMATOUS STATE

- Decreased colloid osmotic pressure in the capillary
  - e.g. burns, liver failure
- Increased capillary hydrostatic pressure
  - e.g. CHF
- Increased capillary permeability
  - e.g. burns, allergic reaction
- Lymphatic obstruction or increased interstitial colloid osmotic pressure
  - e.g. surgical removal of lymph structures

MANAGEMENT OF EDEMA

- Diuretic therapy
- Elevating the affected extremity
- Elastic support stockings in the morning
- Albumin IV

FLUID IMPAIRMENT

- Two types of fluid imbalance
  - Fluid volume deficit
  - Fluid volume excess
- Both types can be life threatening
- Often seen in acute care settings

FLUID VOLUME DEFICIT (FVD)

FLUID VOLUME DEFICIT (FVD)

- is a decrease in intravascular, interstitial, and/or intracellular fluid in the body
- CAUSE: excessive fluid losses, insufficient fluid intake, or failure of regulatory mechanisms and fluid shifts within the body
- a relatively common problem that may exist alone or in combination with other electrolyte or acid-base imbalances

ISOOSMOLAR FLUID VOLUME DEFICIT

- Occurs when sodium and water are lost in equal amounts

HYPEROSMOLAR FLUID VOLUME DEFICIT

- Occurs when more fluid is lost than sodium, resulting in higher serum osmolality than normal

HYPOOSMOLAR FLUID VOLUME DEFICIT

- Occurs when electrolyte loss is greater than fluid (rare)

ETIOLOGY OF FVD

- Common cause of fluid volume deficit is excessive loss of GI fluids
  - Vomiting
  - Diarrhea
  - GI suctioning
  - Intestinal fistulas
  - Intestinal drainage
- Other causes
  - Diuretics
  - renal disorders
  - endocrine disorders
  - excessive exercise
  - hot environment
  - Hemorrhage
  - chronic abuse of laxatives and/or enemas
- Other factors
  - inadequate fluid intake
  - inability to access fluids
  - inability to request or to swallow fluids
  - oral trauma
  - altered thirst mechanisms

PATHOPHYSIOLOGY

Loss of extracellular fluid volume
↓
Hypovolemia
↓
decreased circulating blood volume
(Electrolytes often are lost along with fluid – ISOOSMOLAR FVD)
↓
serum sodium level remains normal (levels of other electrolytes such as potassium may fall)
↓
Fluid is drawn into the vascular compartment from the interstitial spaces
(COMPENSATORY MECHANISM to maintain tissue perfusion)
↓
eventually depletes fluid in the intracellular compartment
↓
Severe fluid loss (i.e. hemorrhage) → shock cardiovascular collapse

MANIFESTATIONS

- Rapid weight loss
  - loss of 2% of body weight represents a mild FVD
  - 5%, moderate FVD
  - 8% or greater, severe FVD
- skin turgor to diminish D/T loss in interstitial fluid
- Postural or orthostatic hypotension
- flat neck veins D/T falling of venous pressure

- Tachycardia
- pale, cool skin (vasoconstriction)
- decreased urine output

MULTISYSTEM EFFECTS
- Mucous Membranes
  - Dry; may be sticky
- Decrease tongue size, longitudinal furrows increase
- Urinary
  - Decrease urine output
  - Oliguria (severe FVD)
  - Increase in urine specific gravity
- Neurologic
  - Altered mental status
  - Anxiety, restlessness
  - Diminished alertness/condition
  - Possible coma (severe FVD)
- Integumentary
  - Diminished skin turgor
  - Dry skin
  - Pale, cool extremities
- Cardiovascular
  - Tachycardia
  - Orthostatic hypotension (moderate FVD)
  - Falling systolic/diastolic pressure (severe FVD)
  - Flat neck veins
  - Decrease venous filling
  - Decrease capillary refill
  - Increase hematocrit
- Potential Complication
  - Hypovolemic shock
- Musculoskeletal
  - Fatigue
- Metabolic Processes
  - Decrease body temperature (isotonic FVD)
  - Increase body temperature (dehydration)
  - Thirst
  - Weight loss
- 2-5% mild FVD
- 6-9% moderate FVD
- >10% severe FVD

DIAGNOSTIC
- Serum Electrolytes
- Serum Osmolality
- Hemoglobin and HCT
- Urine Specific Gravity and Osmolality
- CVP

SERUM ELECTROLYTES
- Test measures the levels of electrolytes such as Sodium, Potassium and Chloride
- Isotonic fluid deficit → sodium levels are within normal limits
- Water loss → sodium levels are high
- Decreases in potassium are common

SERUM OSMOLALITY
- measures the amount of chemicals dissolved in the liquid part (serum) of the blood.
- Chemicals that affect serum osmolality include sodium, chloride, bicarbonate, proteins, and sugar (glucose).
- This test is done on a blood sample taken from a vein
- Helps to differentiate isotonic fluid loss from water loss

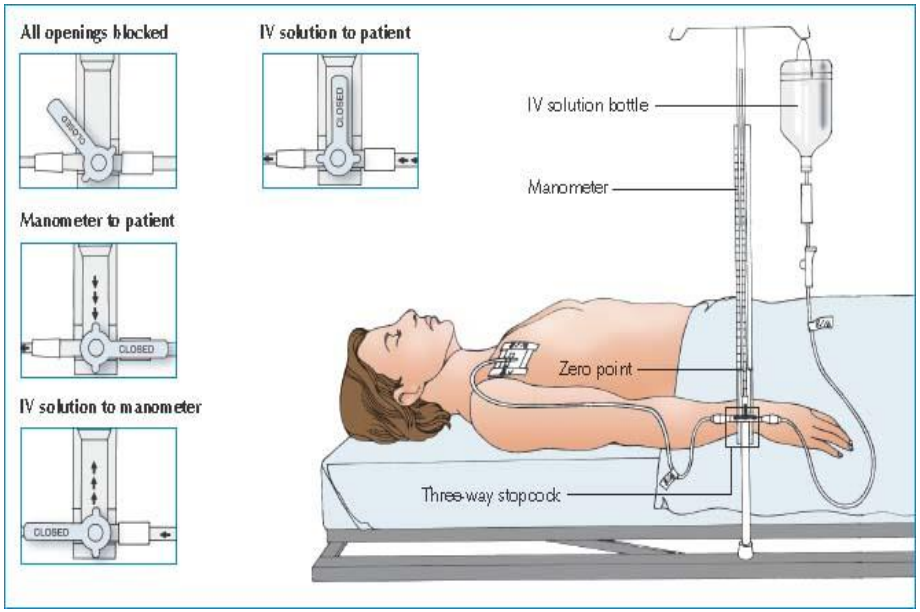
HEMOGLOBIN AND HCT
- Hemoglobin
  - When the hemoglobin level is low, the patient has *anemia*. An *erythrocytosis* is the consequence of too many red cells; this results in hemoglobin levels above normal.
- HCT
  - measures the volume of red blood cells compared to the total blood volume (red blood cells and plasma)
  - Both the hemoglobin and the hematocrit are based on whole blood and are therefore dependent on plasma volume. If a patient is severely dehydrated, the hemoglobin and hematocrit will appear higher than if the patient were normovolemic; if the patient is fluid overloaded, they will be lower than their actual level

URINE SPECIFIC GRAVITY AND OSMOLALITY

- shows the concentration of all chemical particles in the urine
- specific gravity and osmolality of urine increase → compensatory mechanism

CENTRAL VENOUS PRESSURE (CVP)

- Measures the mean pressure in the superior vena cava or right atrium, providing an accurate assessment of fluid volume status
- Normal CVP is 2-6 mm Hg
- CVP decreases with:
  - hypovolemic shock from hemorrhage, fluid shift, dehydration
  - negative pressure breathing which occurs when the patient demonstrates retractions or mechanical negative pressure which is sometimes used for high spinal cord injuries
- CVP increases with:
  - overhydration which increases venous return
  - heart failure or PA stenosis which limit venous outflow and lead to venous congestion
  - positive pressure breathing, straining



The 3-way stopcock is opened so that IV fluid from a container fills the manometer (10- to 20-cm). Then the tap is closed to the fluid bag and opened to the patient. Fluid in the manometer will fall to the level of the CVP and an average reading is taken (as level pulsates up and down during the reading). This system can be set up for long periods and the reading taken intermittently (for example, every hour).

MEDICAL MANAGEMENT

- Correction of fluid loss depends on the acuteness and severity of the fluid deficit. Goals are to replace F/E (Na primarily) that have been loss.
- Fluid Restoration
  - Oral Rehydration
  - IV Rehydration
- Monitoring complications of fluid restoration
- Correction of Underlying Problem

Oral Rehydration

- safest and most effective treatment for fluid volume deficit in alert patients who are able to take oral fluids
- Fluids are replaced gradually, particularly in older adults, to prevent rapid rehydration of the cells

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NOTE:

- For mild fluid deficits in which a loss of electrolytes has been minimal (e.g., moderate exercise in warm weather), water alone may be used for fluid replacement
- For more severe fluid deficits and when electrolytes have also been lost e.g. vomiting and/or diarrhea, strenuous exercise for longer than an hour or two → carbohydrate/electrolyte solution such as a sports drink, ginger ale, or a rehydrating solution (e.g., Pedialyte or Rehydralyte) is more appropriate. These solutions provide sodium, potassium, chloride, and calories to help meet metabolic needs

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IV Rehydration

- When the fluid deficit is severe or the patient is unable to ingest fluids, the IV route is used to administer replacement fluids.
- Types of Fluid Volume Deficit:
  - **Isotonic:** Caused by losing fluids and solutes about equally; solute concentration in the remaining extracellular fluid then remains relatively unchanged
  - **Hypertonic:** Caused by losing more fluids than solutes, leading to increased solute concentration in the remaining fluid.
  - **Hypotonic:** Caused by losing more solutes than fluid leading to decreased solute concentration in remaining fluid. This is the rarest type.

MONITORING COMPLICATIONS OF FLUID RESTSORATION

- Client with severe ECFVD accompanied by severe heart, liver and kidney disease can't tolerate large volumes of fluid or sodium without the risk of development of heart failure
- Unstable clients needs to be monitored to detect ↑ pressure from fluids
  - Monitor:
    - Fluid volume status by CVP insertion
    - Lab values (Na, K, BUN, Osmolarity)
    - Body Weight
    - Urine output

CORRECTION OF UNDERLYING PROBLEMS

- Medication
  - Antiemetics
  - Antidiarrheals
  - Antibiotics
  - Antidiuretics to reduce body temperature

NURSING MANAGEMENT

Nurses are responsible for (1) identifying patients at risk for fluid volume deficit, (2) initiating and carrying out interventions to prevent and treat fluid volume deficit, and (3) monitoring the effects of therapy

1. I & O every 8 hours or hourly (Record all output accurately)
2. VS every 2-4 hours, report changes from baseline VS; Assess CVP every 4hrs (if patient has CVP access)
3. Weight patient daily and record.
4. Peripheral vein filling (Capillary refill 3-5 sec)
5. Renal client / relatives to report urine output < than 30 m/L x 2 consecutive hours or < 240 ml x 8O period
6. Assess for dryness of mucous membrane and skin turgor (check should be done)
7. Oral care to ↓ discomfort related to mucous membrane dryness
8. Monitor plasma sodium, BUN, Glucose, HCT to determine osmolality
9. Assess the client for confusion, easy indication of ICF involvement
10. Keep fluids easily accessible
11. Administer and monitor intake of oral fluids as prescribed
12. Administer IV fluids as prescribed using an infusion pump. Monitor for indicators of fluid overload if rapid fluid replacement is ordered: dyspnea, tachypnea, tachycardia, increased CVP, jugular vein distention, and edema

NURSING DIAGNOSIS

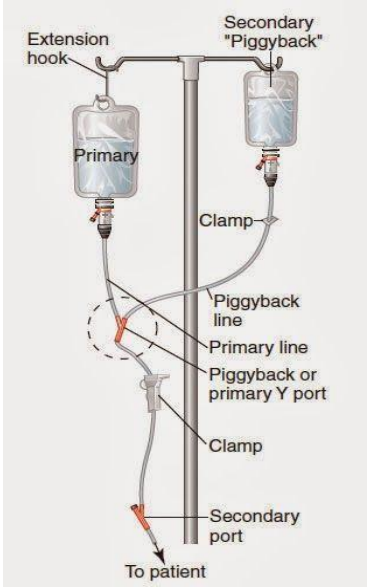
- Fluid Volume Deficit
  - Patients with a fluid volume deficit due to abnormal losses, inadequate intake, or impaired fluid regulation require close monitoring as well as immediate and ongoing fluid replacement
- Ineffective Tissue Perfusion
  - A fluid volume deficit can lead to decreased perfusion of renal, cerebral, and peripheral tissues. Inadequate renal perfusion can lead to acute renal failure. Decreased cerebral perfusion leads changes in mental status and cognitive function, causing restlessness, anxiety, agitation, excitability, confusion, vertigo, fainting, and weakness
- Risk for Injury
  - The patient with fluid volume deficit is at risk for injury because of dizziness and loss of balance resulting from decreased cerebral perfusion secondary to hypovolemia

IV THERAPY

IV THERAPY

- Indications for IV Therapy
  - unable to eat or drink adequate amounts
  - rapid delivery of medication in an emergency
  - Patients with anemia or blood loss can receive lifesaving IV transfusions
  - unable to eat for an extended period can have their nutritional needs met with total parenteral nutrition (TPN)

TYPES OF INFUSION



- Continuous Infusion
  - infusion is kept running constantly until discontinued by the physician
- Intermittent Infusion
  - Intermittent IV lines are "capped off" with an injection port and used only periodically
- Bolus (IV push or IVP Drug)
  - bolus drug (sometimes called an IV push or IVP drug) is injected slowly via a syringe into the IV site or tubing port
  - can be dangerous if they are given incorrectly, and a drug reference should always be checked to determine the safe amount of time over which the drug can be injected
- Piggyback/Secondary Infusion
  - Some IV medications, such as antibiotics, need to be infused over a short period of time
  - In order for the piggyback medication to infuse, it must hang higher than the primary infusion

FACTORS AFFECTING FLOWRATES

- Change in catheter position
- Height of the solution
- Patency of the catheter

TYPES OF IVF SOLUTIONS

- COLLOIDS
  - Fluids that expand the circulatory volume due to particles that cannot cross a semipermeable membrane. They pull fluid from the interstitial space into the intravascular space, increasing fluid volume
  - ADVANTAGE: is beneficial to large losses of fluid
  - DISADVANTAGE: costly and the risk of volume overload, including pulmonary edema
- CRYSTALLOIDS
  - Work much like colloids but do not stay in the intravascular circulation as well as colloids do, so more of them need to be used
  - ADVANTAGE: cheaper and more convenient to use; provide hydration and calories to patients and include dextrose, normal saline, and Ringer's and lactated Ringer's solution
  - DISADVANTAGE: lacks large molecules

Table 1. **Comparative summary of crystalloid and colloid solutions**

Crystalloid solution	Colloid sollution
Half-life of 30-60 minutes	Half-life of several hours or days
Three times the volume needed for replacement	Replaces fluid volume for volume
Excessive use can cause peripheral and pulmonary oedema	Excessive use can precipitate cardiac failure
Molecules small enough to freely cross capillary walls, so less fluid remains in the intravascular spaces	Molecules too large to cross capillary walls, so fluid remains in intravascular spaces for longer
Inexpensive	More expensive than crystalloids
Non-allergenic	Risk of anaphylactic reactions
Suitable for vegetarian or vegan patients	Some preparations unsuitable for vegetarian or vegan patients

TONICITY OF SOLUTIONS

ISOTONIC SOLUTIONS • 0.9% Saline • Lactated Ringer's solution	• Monitor for fluid overload; discontinue fluids and notify the healthcare provider • Do not administer lactated ringer's solution to patients with severe liver disease as the liver may be unable to convert the lactate to bicarbonate and the patient may become acidotic. • If administering lactated ringer's solution, monitor potassium levels and cardiac rhythm; if abnormalities are present, notify the health care provider
HYPOTONIC SOLUTIONS • 0.45% Saline or 0.25% Saline • D5W	• Monitor for inflammation and infiltration at IV insertion site as hypotonic solutions may cause cells to swell and burst, including those at the insertions site; this narrows the lumen of the vein • Monitor blood sodium levels • Do not administer to patients at risk for increased intracranial pressure (e.g head trauma, stroke, neurosurgery) • Do not administer to patients at risk for third-space shifts (burns, trauma, liver disease, malnutrition)
HYPERTONIC SOLUTIONS • Hypertonic fluids have a tonicity >350 mEq/L and include the ff: • Fluids containing medications • D5W sodium chloride • D5W in lactated ringer's solution • Total parenteral solutions	• Monitor for inflammation and infiltration at IV insertion site as hypertonic solutions cause cells to shrink, exposing the basement membrane of the vein • Monitor blood sodium levels • Monitor for circulatory overload • Do not administer to patients with diabetic ketoacidosis or impaired cardiac or kidney function.

IV Solution Cheat Sheet

Type	Description	Osmolality	Use	Miscellaneous
Normal Saline (NS)	0.9% NaCl in Water Crystalloid Solution	Isotonic (308 mOsm)	Increases circulating plasma volume when red cells are adequate	• Replaces losses without altering fluid concentrations. • Helpful for Na+ replacement
1/2 Normal Saline (1/2 NS)	0.45% NaCl in Water Crystalloid Solution	Hypotonic (154 mOsm)	Raises total fluid volume	• Useful for daily maintenance of body fluid, but is of less value for replacement of NaCl deficit. • Helpful for establishing renal function. • Fluid replacement for clients who don't need extra glucose (diabetics)
Lactated Ringer's (LR)	Normal saline with electrolytes and buffer	Isotonic (275 mOsm)	Replaces fluid and buffers pH	• Normal saline with K+, Ca++, and lactate (buffer) • Often seen with surgery
D_5W	Dextrose 5% in water Crystalloid solution	Isotonic (in the bag) *Physiologically hypotonic (260 mOsm)	Raises total fluid volume. Helpful in rehydrating and excretory purposes.	• Provides 170-200 calories/1,000cc for energy. • **Physiologically hypotonic** - the dextrose is metabolized quickly so that only water remains - a hypotonic fluid
D_5NS	Dextrose 5% in 0.9% saline	Hypertonic (560 mOsm)	Replaces fluid sodium, chloride, and calories.	• Watch for fluid volume overload
D_5 1/2 NS	Dextrose 5% in 0.45% saline	Hypertonic (406 mOsm)	Useful for daily maintenance of body fluids and nutrition, and for rehydration.	• Most common postoperative fluid
D_5LR	Dextrose 5% in Lactated Ringer's	Hypertonic (575 mOsm)	Same as LR plus provides about 180 calories per 1000cc's.	• Watch for fluid volume overload
Normosol-R	Normosol	Isotonic (295 mOsm)	Replaces fluid and buffers pH	• pH 7.4 • Contains sodium, chloride, calcium, potassium and magnesium • Common fluid for OR and PACU

TYPES OF IV SOLUTIONS ACCORDING TO PURPOSE

- Hydrating
  - Replace water loss; Dilute meds; Keep veins open
- Nutritional
  - Promotes faster recuperation
- Maintenance
  - Replace electrolyte loss at ECF level; Maintenance in patients with no oral intake; Replace fluid loss; Treatment for dehydration
- Volume Expander
  - Increase osmotic pressure thus maintain circulatory volume

Local Complications of IV Therapy	Signs and Symptoms	Nursing Interventions
Hematoma collection of blood outside of blood vessels	• Ecchymoses • Swelling • Inability to advance catheter • Resistance during flushing	• Remove catheter • Apply pressure with 2x2 • Elevate extremity
Thrombosis formation of a blood clot	• Slowed or stopped infusion • Fever/malaise • Inability to flush catheter	• Discontinue catheter • Apply cold compress to site • Assess for circulatory impairment
Phlebitis inflammation of the walls of a vein	• Redness at site • Site warm to touch • Local swelling • Pain • Palpable cord • Sluggish infusion rate	• Discontinue catheter • Apply cold compress initially; • then warm • Consult physician if severe
Infiltration (Extravasation) the accidental leakage of non-vesicant solutions out of the vein into the surrounding tissue	• Coolness of skin at site • Taut skin • Dependent edema • Backflow of blood absent • Infusion rate slowing	• Discontinue catheter • Apply cool compress • Elevate extremity slightly • Follow extravasation guidelines • Have antidote available
Local Infection	• Redness and swelling at site • Possible exudate • Increase WBC count • Elevated T lymphocytes	• Discontinue catheter and culture • site and catheter • Apply sterile dressing • over site • Administer antibiotics if ordered
Venous Spasm occurs due to severe vein irritation, administration of cold fluids or blood, and a very rapid flow rate	• Sharp pain at site • Slowing of infusion	• Apply warm compress to site • Restart infusion in new site if • spasm continues

SYSTEMIC COMPLICATIONS OF PERIPHERAL IV THERAPY

Complication	Signs and Symptoms	Nursing Interventions
Septicemia blood poisoning, especially that caused by bacteria or their toxins	• Fluctuating temperature • Profuse sweating • Nausea/vomiting • Diarrhea • Abdominal pain • Tachycardia • Hypotension • Altered mental status	• Restart new IV system • Obtain cultures • Notify physician • Initiate antimicrobial therapy as • ordered • Monitor patient closely
Fluid Overload is a medical condition where there is too much fluid in the blood	• Weight gain • Puffy eyelids • Edema • Hypertension • Changes in input and output (I&O) • Rise in central venous pressure (CVP) • Shortness of breath • Crackles in lungs • Distended neck veins	• Decrease IV flow rate • Place patient in high Fowler's position • Keep patient warm • Monitor vital signs • Administer oxygen • Use microdrip set or controller
Air Embolism also known as a gas embolism, is a blood vessel blockage caused by one or more bubbles of air or other gas in the circulatory system	• Lightheadedness • Dyspnea, cyanosis, tachypnea, expiratory wheezes, cough • chest pain, hypotension • Changes in mental status • Coma	• Call for help! • Place patient in Trendelenburg's • position • Administer oxygen • Monitor vital signs • Notify physician

FLUID VOLUME EXCESS

FLUID VOLUME EXCESS

- results when both water and sodium are retained in the body
- may be caused by fluid overload (excess water and sodium intake) or by impairment of the mechanisms that maintain homeostasis
- excess fluid can lead to excess :
  - intravascular fluid (HYPERVOLEMIA)
  - excess interstitial fluid (EDEMA)

ETIOLOGY

- results from conditions that cause retention of both sodium and water
- conditions include:
  - heart failure
  - cirrhosis of the liver
  - renal failure
  - adrenal gland disorders
  - corticosteroid administration
  - stress conditions causing the release of ADH and aldosterone

PATHOPHYSIOLOGY

extracellular compartment is expanded
↓
increase in volume increases the pressure in the vasculature
↓
Baroreceptors sense the increase in pressure
↓
increase in their firing to the central nervous system (CNS)
↓
SNS is inhibited
↓
RAAS function declines = vasodilation, lowering of blood pressure, reduced reabsorption of Na^+, increase urine output

MANIFESTATIONS

- Excess extracellular fluid leads to hypervolemia and circulatory overload
- Excess fluid in the interstitial space causes peripheral or generalized edema
- Peripheral edema, or if severe, anasarca (severe generalized edema)
- Full bounding pulse, distended neck and peripheral veins, increased central venous pressure, cough, dyspnea (labored or difficulty breathing), orthopnea (difficult breathing when supine)
- Dyspnea at rest
- Tachycardia and hypertension
- Reduced oxygen saturation
- Moist crackles on auscultation of the lungs, pulmonary edema
- Increased urine output (polyuria)
- Ascites (excess fluid in the peritoneal cavity)
- Decreased hematocrit and BUN
- Altered mental status and anxiety

COMPARTMENTS AFFECTED

- Fluid overload can occur in either the extracellular or intracellular compartments

EXTRACELLULAR FLUID OVERLOAD

- Occurs in either the intravascular compartment or in the interstitial area
  - EDEMA - most common term associated with fluid overload found in the interstitial or lung tissue
  - HYPERVOLEMIA - when an overabundance of fluid occurs in the intravascular compartment
  - ISOTONIC FLUID VOLUME EXCESS - type of fluid overload wherein sodium and water remain in equal proportions with each other. Also results from a decreased elimination of sodium and water
  - ANASARCA - generalized edema

CAUSES OF EXTRACELLULAR FLUID OVERLOAD

- Excessive sodium intake through diet
- administration of hypertonic fluids
  - D5.45 normal saline solution
  - D5.9 normal saline solution
  - 10% Dextrose
  - 3% normal saline solution
- Diabetes insipidus
- Congestive heart failure
- Cirrhosis
- Renal failure
- Cushing's syndrome
- Hyperaldosteronism

MANIFESTATIONS OF EXTRACELLULAR OVERLOAD

- Pitting peripheral edema
- Periorbital edema
- Shortness of breath
- Shift of interstitial fluid to plasma
- Bounding pulse and jugular venous distention
- Anasarca
- Rapid weight gain
- Moist crackles
- Tachycardia
- hypertension

INTRACELLULAR FLUID OVERLOAD

- also known as water intoxication
- Hypotonic fluid from the intravascular space moves by osmosis to an area of higher solute concentration inside the cell
- Cells run the risk of rupturing if they become too overloaded with fluid

CAUSES OF INTRACELLULAR FLUID OVERLOAD

- Hypotonic intravenous administration
  - 0.45% normal saline solution
  - 5% dextrose in water
- Excessive nasogastric tube irrigation with free water
- Excessive administration of free water via enteral tube feedings
- Syndrome of inappropriate antidiuretic hormone
- Psychogenic polydipsia

MANIFESTATIONS OF INTRACELLULAR FLUID OVERLOAD

- Neurological
  - Cerebral edema
- Headache
- Irritability
- Confusion
- Anxiety
  - Muscle weakness
  - Twitching
- Respiratory
  - Dyspnea on exertion
  - Increased respirations
- Gastrointestinal
  - Nausea and vomiting
- Increased thirst
- Cardiac
  - Elevated blood pressure
  - Decreased pulse

DIAGNOSTICS FOR FVE

- Serum electrolytes and serum osmolality are measured, but usually remain within normal limits
- Serum hematocrit and hemoglobin often are decreased due to plasma dilution from excess extracellular fluid
- Renal and liver function
- Chest radiograph
- ABG - fluid in the alveoli impairs gas exchange resulting in hypoxia as evidenced by a low PO2

MEDICAL MANAGEMENT

- Managing fluid volume excess focuses on prevention in patients at risk, treating its manifestations, and correcting the underlying cause.

DIURETICS

- Commonly used to treat fluid volume excess. They inhibit sodium and water reabsorption, increasing urine output.
- The three major classes of diuretics, each of which acts on a different part of the kidney tubule, are as follows:
  - Loop diuretics - Inhibit sodium and chloride reabsorption in the ascending loop of Henle
    - Furosemide
    - Ethacrynic acid
    - Bumetanide
    - torsemide
  - Thiazide-type diuretics – Promote the excretion of sodium, chloride, potassium and water by decreasing absorption in the distal tubule
    - Bendroflumethiazide
    - Chlorothiazide
    - Hydrochlorothiazide
    - Metolazone
    - Polythiazide
    - Chlorthalidone
    - Trichlormethiazide
    - Indamide
    - Xipamid
  - Potassium-sparing diuretics – Promote excretion of sodium and water by inhibiting sodium-potassium exchange in the distal tubule
    - Spironolactone
    - Amioride
    - Triamterene

FLUID MANAGEMENT

- Fluid intake may be restricted in patients who have fluid volume excess. The amount of fluid allowed per day is prescribed by the primary care provider. All fluid intake must be calculated, including meals and that used to administer medications orally or IV

DIETARY MANAGEMENT

- Because sodium retention is a primary cause of fluid volume excess, a sodium-restricted diet often is prescribed. The primary dietary sources of sodium are the salt shaker, processed foods, and foods themselves.

NURSING MANANGEMENT

Nursing care focuses on preventing fluid volume excess in patients at risk and on managing problems resulting from its effects

- Assess vital signs, heart sounds, and volume of peripheral arteries.
- Auscultate lungs for presence or worsening of crackles and wheezes; auscultate heart for extra heart sounds.
- Place in Fowler's position if dyspnea or orthopnea is present.
- Monitor oxygen saturation levels and arterial blood gases(ABGs) for evidence of impaired gas exchange (SaO2 < 92% to 95%; PaO2 < 80 mmHg). Administer oxygen as indicated.
- Assess for the presence and extent of edema, particularly in the lower extremities and the back, sacral, and periorbital areas.
- Obtain daily weights at the same time of day, using approximately the same clothing and a balanced scale.
- Administer oral fluids cautiously, adhering to any prescribed fluid restriction.
- Discuss the restriction with the patient and significant others, including the total volume allowed, the rationale, and the importance of reporting all fluid taken.
- Provide oral hygiene at least every 2 hours. Oral hygiene contributes to patient comfort and keeps mucous membranes intact; it also helps relieve thirst if fluids are restricted.
- Teach patient and significant others about the sodium-restricted diet.
- Administer prescribed diuretics as ordered, monitoring the patient's response to therapy.
- Promptly report significant changes in serum electrolytes or osmolality or abnormal results of tests done to determine contributing factors to the fluid volume excess.

NURSING DIAGNOSIS

- Fluid Volume Excess
  - Nursing care for the patient with excess fluid volume includes collaborative interventions such as administering diuretics and maintaining a fluid restriction, as well as monitoring the status and effects of the excess fluid volume
- Risk for Impaired Skin Integrity
  - Tissue edema decreases oxygen and nutrient delivery to the skin and subcutaneous tissues, increasing the risk of injury
- Impaired Gas Exchange
  - With fluid volume excess, gas exchange may be impaired by edema of pulmonary interstitial tissues. Acute pulmonary edema is a serious and potentially life-threatening complication of pulmonary congestion

ACID AND BASE BALANCE

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pH (potential of hydrogen)
Acid base balance depends on the regulation of H^+ ions.
pH is inversely proportional to H^+ levels.
Normal: 7.35 – 7.45

REGULATORY MECHANISM

- Chemical Buffers
  - Carbonic Bicarbonate System
  - Phosphate
  - CHON buffer
- Respiratory System
  - Lungs
- Kidneys
  - Elimination of H^+
  - Regeneration of HCO_3

NOTE: CHECMICAL BUFFERS - do not correct pH deviations, but only serve to reduce the extent of the change that would otherwise occur

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COMPONENTS OF ABG

- $PaCO_2$ or partial pressure of carbon dioxide shows the adequacy of the gas exchange between the alveoli and the external environment (alveolar ventilation)
- PaO_2 or partial pressure of oxygen or PAO2 indicates the amount of oxygen available to bind with hemoglobin
- SO_2 or oxygen saturation, measured in percentage, is the amount of oxygen in the blood that combines with hemoglobin
- HCO_3 or bicarbonate ion is an alkaline substance that comprises over half of the total buffer base in the blood

KEY ELEMENTS IN DETERMINING ACID BASE IMBALANCE

NORMAL GAS VALUES	
pH	7.35 – 7.45
PaO2	80 – 100%
PaCO2	35 – 45 mmHg
HCO3	22 – 26 mEq/L

Note: Not a clinical diagnosis or disease, rather they are clinical syndromes associated with a wide variety of diseases.

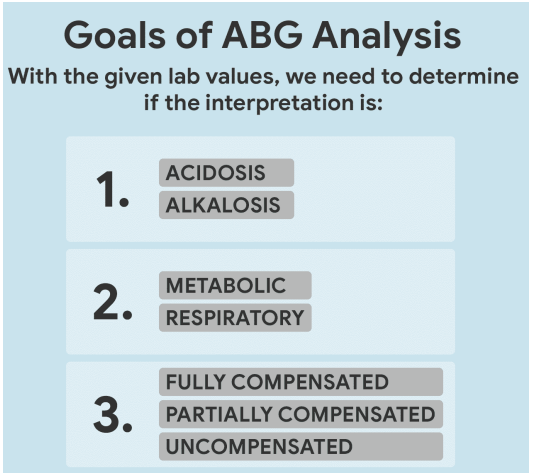
TYPES OF ACID BASE BALANCE

ACIDOSIS	ALKALOSIS

• pH is below 7.35
  • Respiratory
  • Metabolic
  • Fully Compensated
  • Partially Compensated
  • Uncompensated
	• pH is above 7.45
	  • Respiratory
	  • Metabolic
	  • Fully Compensated
	  • Partially Compensated
	  • Uncompensated

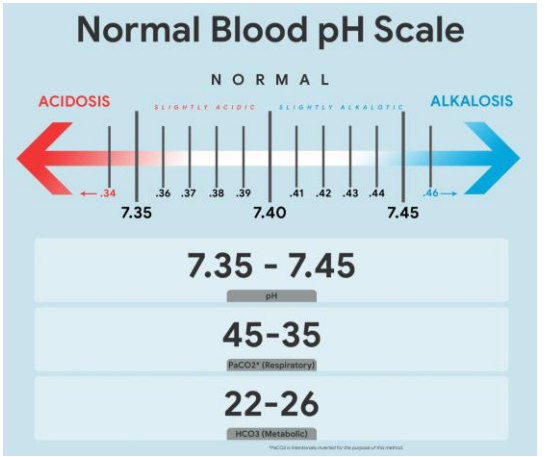
COMBINED	

DETERMINING ACID-BASE IMBALANCE



STEPS IN ABG ANALYSIS USING THE TIC-TAC-TOE METHOD

1. Memorize the normal values.



2. Create your tic-tac-toe grid.

Tic-Tac-Toe Grid		
ACIDOSIS	NORMAL	ALKALOSIS

3. Determine if pH is under NORMAL, ACIDOSIS, or ALKALOSIS.
4. Determine if $PaCO_2$ is under NORMAL, ACIDOSIS, or ALKALOSIS.
5. Determine if HCO_3 is under NORMAL, ACIDOSIS, or ALKALOSIS.
6. Solve for goal #1: ACIDOSIS or ALKALOSIS.
7. Solve for goal #2: METABOLIC or RESPIRATORY.
8. Solve for goal #3: COMPENSATION.

EXERCISES

1. pH=7.44 | $PaCO_2$=30 | HCO_3=21 RESP. ALK., FULLY COMPENSATED
2. pH=7.38 | $PaCO_2$=49 | HCO_3=30 RESP. ACID., FULLY COMPENSATED
3. pH=7.10 | $PaCO_2$=40 | HCO_3=18 META. ACID., UNCOMPENSATED
4. pH=7.20 | $PaCO_2$=57 | HCO_3=26 RESP. ACID., UNCOMPENSATED
5. pH=7.48 | $PaCO_2$=30 | HCO_3=22 RESP. ALK., UNCOMPENSATED
6. pH=7.41 | $PaCO_2$=19 | HCO_3=30 COMBINED ALKALOSIS
7. pH=7.50 | $PaCO_2$=20 | HCO_3=20 RESP. ALK., PARTIALLY COMPENSATED
8. pH=7.30 | $PaCO_2$=50 | HCO_3=20 COMBINED ACIDOSIS
9. pH=7.60 | $PaCO_2$=20 | HCO_3=30 COMBINED ALKALOSIS

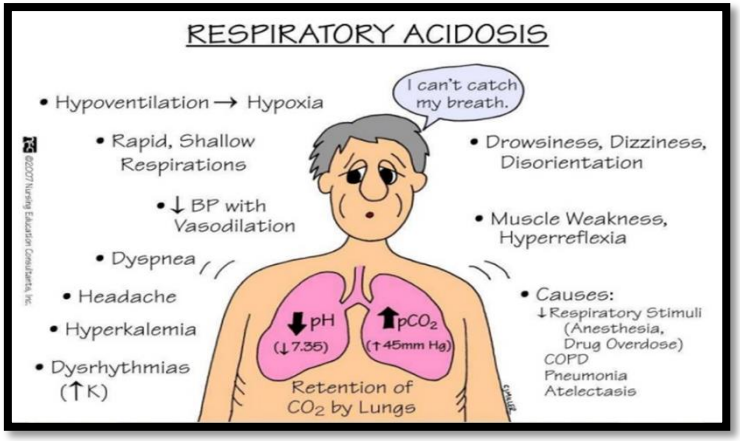
RESPIRATORY ACIDOSIS

CAUSES OF RESPIRATORY ACIDOSIS

- Acute respiratory conditions
- Depression of respiratory center
- Iatrogenic cause

MANIFESTATIONS (RESPIRATORY ACIDOSIS)

- Hypercapnia, due to rapid rise of PaCO2 level
- Headache, CO2 dilates cerebral blood vessels
- Warm and flushed skin, related to the peripheral vasodilation as well as to impaired gas exchange
- Fine flapping tremors
- Decreased reflexes
- Rapid, shallow respirations; elevated pulse rate; tachycardia
- Decreasing level of consciousness



MEDICAL MANAGEMENT (RESPIRATORY ACIDOSIS)

- ABG analysis
- Chest X-rays, can help pinpoint some cause, eg COPD, pneumonia
- Serum electrolytes level, in acidosis potassium leaves the cell, so expect serum level to be elevated
- Bronchodilators, to open constricted airways
- Supplemental oxygen
- Drug therapy to treat hyperkalemia

NURSING DIAGNOSIS (RESPIRATORY ACIDOSIS)

- Ineffective breathing Pattern related to hypoventilation
- Impaired gas exchange related to alveolar hypoventilation
- Anxiety related to breathlessness
- Risk for injury related to decreased level of consciousness

NURSING MANAGEMENT (RESPIRATORY ACIDOSIS)

- Maintain patent airway
- Monitor vital signs
- Monitor neurologic status and report significant changes
- Administer oxygen as ordered
- Accurate intake and output records
- Report any variations in ABG levels
- Coughing and deep breathing exercises

METABOLIC ACIDOSIS

- The underlying mechanisms in metabolic acidosis are a loss of bicarbonate from extracellular fluid, an accumulation of metabolic acids, or a combination of the two.

CAUSE OF METABOLIC ACIDOSIS

- Diabetic ketoacidosis
- Renal insufficiency
- Severe diarrhea

MANIFESTATIONS (METABOLIC ACIDOSIS)

- Weakness, fatigue, general malaise
- Anorexia, nausea, vomiting, abdominal pain
- Decrease level of consciousness
- Rapid, deep, labored breathing (Kussmaul's respirations) → the first clue to metabolic acidosis
- Decrease cardiac output and blood pressure
- Skin is warm and dry, as a result of peripheral vasodilation
- Diminished muscle tone and reflexes

NOTE: Increase respiratory rate as means of compensatory mechanism

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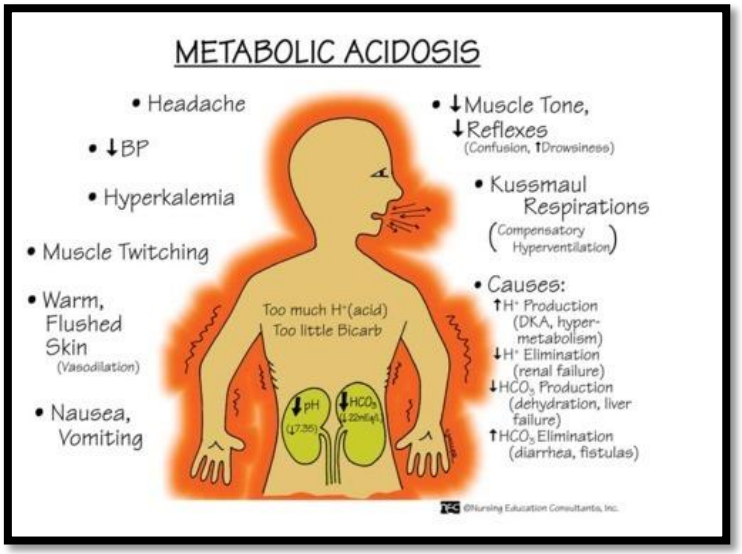


MEDICAL MANAGEMENT (METABOLIC ACIDOSIS)

- ABG analysis
- Serum potassium levels → usually elevated as hydrogen ions move into the cells and potassium moves out to maintain electroneutrality
- Rapid acting insulin to reverse diabetic ketoacidosis and drive potassium back into the cell.
- Intravenous Sodium bicarbonate → to neutralize blood acidity in patients with bicarbonate loss
- Fluid replacement

NURSING DIAGNOSIS (METABOLIC ACIDOSIS)

- Decreased cardiac output secondary to dysrhythmias and / or fluid volume deficits
- Risk for sensory/ perceptual alterations related to changes in neurological functioning secondary to acidosis
- Risk for fluid volume deficit related to excessive loss from the kidneys or gastrointestinal system

NURSING MANAGEMENT (METABOLIC ACIDOSIS)

- Monitor vital signs
- Monitor neurologic status
- Maintain patent IV line
- Careful administration of sodium bicarbonate
- Proper positioning to promote chest expansion and facilitate breathing
- Record intake and output

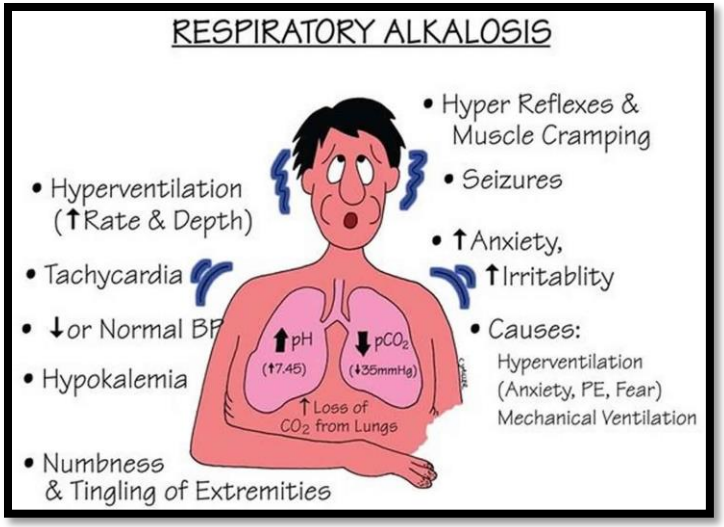
RESPIRATORY ALKALOSIS

CAUSES OF RESPIRATORY ALKALOSIS

- Vomiting → loss of hydrochloric acid from the stomach
- Diuretic therapy (thiazides, loop diuretics)→ can lead to a loss of hydrogen, potassium from the kidneys
- Cushing's disease → causes retention of sodium and chloride and urinary loss of potassium and hydrogen
- Hyperventilation → most common cause of acute respiratory alkalosis
- Severe anemia, acute hypoxia 20 high altitude → overstimulation of the respiratory system causes to breathe faster and deeper

MANIFESTATIONS (RESPIRATORY ALKALOSIS)

- Slow, shallow respirations
- nausea, vomiting
- polyuria
- Twitching, weakness and tetany
- Hyperactive reflexes
- Numbness and tingling sensation
- Confusion or syncope → lack of carbon dioxide in the blood may lead to hyperventilation
- Dysrhythmia: related to hypokalemia and hypocalcemia



MEDICAL MANAGEMENT (RESPIRATORY ALKALOSIS)

- Identify and eliminate causative factor if possible
- Sedative or Anxiolytics agents may be given→ to relieve anxiety and restore a normal breathing pattern.
- Respiratory support, e.g. oxygen therapy to prevent hypoxemia; breathe into a paper bag → this forces the patient to breathe exhaled carbon dioxide, thereby raising the carbon dioxide
- ABG analysis → key diagnostic test in identifying respiratory alkalosis
- ECG → may indicate arrhythmias or the changes associated with hypokalemia or hypocalcemia

NURSING DIAGNOSIS (RESPIRATORY ALKALOSIS)

- Ineffective breathing pattern related to hyperventilation
- Altered thought processes related to altered cerebral functioning

NURSING MANAGEMENT (RESPIRATORY ALKALOSIS)

- Allay anxiety whenever possible to prevent hyperventilation
- Monitor vital signs, and report changes
- Report variations in ABG and ECG
- Maintain a calm, quiet environment

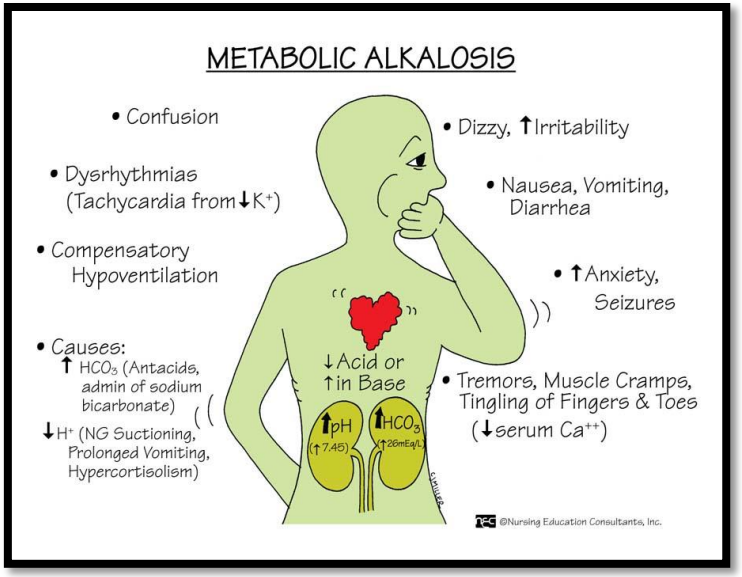
METABOLIC ALKALOSIS

CAUSES OF METABOLIC ALKALOSIS

- diuretic therapy → cause loss of H+, A-, K+ but precipitates ↑HCO3 level
- ingestion of NaHCO3 or excessive NaHCO3 to correct acidosis
- aldosterone excess → ↑ Na retention, ↑ H+ and bicarbonate regeneration
- prolonged steroid therapy → same with aldosterone effects
- prolonged gastric suctioning or vomiting → loss or H+ ions; Sengstaken, Blakemore tube (a thick catheter with triple lumen with 2 balloons; inflated at the orifice of the stomach and esophagus to apply pressure thus prevent bleeding, the 3rd lumen is for suctioning gastric contents)
- Massive blood transfusion (whole blood) → (citrate anticoagulant which is use for storing blood is metabolize to bicarbonate)

MANIFESTATIONS (METABOLIC ALKALOSIS)

- Increased myocardial activity, palpitations
- Increased heart rate
- Rapid , shallow breathing
- Dizziness, lightheadedness
- Hyperactive reflexes
- Nausea, vomiting



MANAGEMENT (METABOLIC ALKALOSIS)

- Replacement of electrolytes
- Antiemetics may be administered to treat underlying nausea and vomiting
- Acetazolamide (Diamox) → to increase renal excretion

NURSING DIAGNOSIS (METABOLIC ALKALOSIS)

- Ineffective breathing pattern related to hypoventilation
- Impaired gas exchange related to alveolar hypoventilation
- Anxiety related to breathlessness

NURSING MANAGEMENT (METABOLIC ALKALOSIS)

- Monitor vital signs
- Assess patient's level of consciousness
- Administer oxygen → treat hypoxemia
- Monitor Intake and output

BURNS

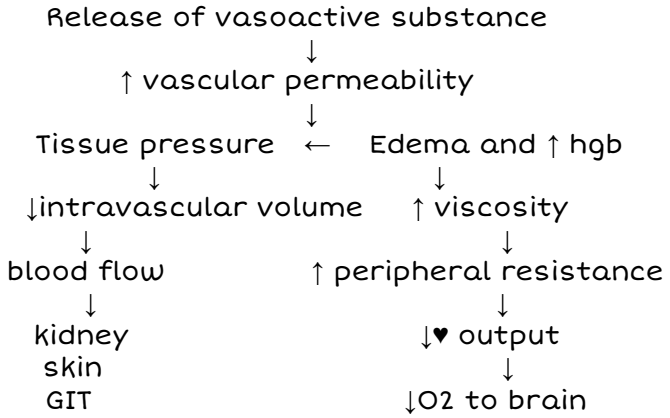
BURN DEFINITION

- A burn is an injury from exposure to heat, chemicals, radiation or electric current leading to sequence of physiologic events.
- can lead to irreversible tissue damage (IF UNTREATED)

4 MAJOR GOALS

- Prevention
- Institution Of Life-Saving Measures For The Severely Burned Person
- Prevention Of Disability And Disfigurement Through Early Specialized Individual Treatment
- Rehabilitation Through Constructive Surgery And Rehabilitative Programs

PHYSIOLOGIC RESPONSE TO BURN INJURY



SYSTEMIC PHYSIOLOGIC CHANGES WITH BURNS (>25% TBSA)

STAGE CHANGE	HYPOVOLEMIC STAGE MECHANISM	24-48- 72 HOURS RESULT	DIURETIC STAGE MECHANISM	RESULT
ECF SHIFT	vascular to interstitial	Hemocon-centration Edema at the affected site	Interstitial to vascular	hemodilution
Renal function	Decrease urine due to decrease BP, and cardiac output	oliguria	increase renal blood flow, thus urine	diuresis
Na	Trapped in exudate or edema	low	Lost in diuresis	low
K level	Cell injury releases potassium decrease potassium excretion due to decrease renal function	Hyper-kalemia	Lost in diuresis stabilize after 3-5 days	Hypo-kalemia
CHON balance	Escape from poor vascular integrity and trapped in interstitial space	Low Negative nitrogen balance	Continued catabolism	Low Negative nitrogen balance
Acid base balance	Anaerobic metabolism related to hypo-perfusion, decrease renal function	Metabolic acidosis	Bicarb lost in diuriesis Increase acid end product (hyper-metabolism)	Metabolic acidosis

CATEGORIES OF BURN

- Mild
  - Partial thickness <15% adult
  - Full thickness burns <2% adult
- Moderate
  - Partial thickness burn 15 to 25 % adult tbsa
  - Full thickness 2% to 10 % adult tbsa
  - Plus minor category criteria
- Severe
  - Partial thickness >25% adult tbsa
  - Full thickness burns are >10% adult tbsa
  - Burns are accompanied by other injuries
  - Presence of other criteria in the previous categories
- Other criteria
  - Does not involve eyes, ears, nose, hands, face, feet, perineum
  - No electrical burns / inhalation injuries
  - Adult younger than 60 yrs. Old
  - No pre-existing disease and other injury with the burn

FACTORS DETERMINING BURN SEVERITY

- Burning Agent/Cause
- Location/Body parts involve
- Age
- Depth of the burn
- Size of the burn
- History of cardiac, pulmonary, renal, hepatic disease
- Injuries sustained during burn injury

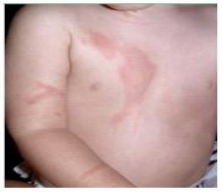
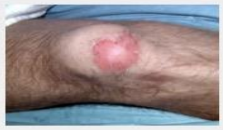
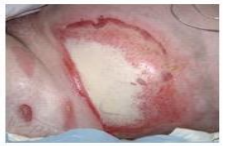
BURNING AGENT AND LOCATION

- The higher the temperature of the burning agent and longer duration of contact can cause more severe injury
- Burns sustained in the head, neck and chest is associated with higher mortality rate because of:
  - bronchoconstriction secondary to histamine release causing edema
  - carbon monoxide poisoning secondary to smoke inhalation
  - chest constriction secondary to circumferential burns
  - Burns sustained in the perineum area requires special care (RISK FOR INFECTION)

TYPES OF BURN INJURY

Cause /Type	Causative Agent	Priority Treatment
Thermal	open flame steam hot liquids (water, grease, tar, metal)	Extinguish flame (stop, drop, and roll) Flush with cool water Consult fire department
Chemical	Acids Strong alkalis Organic compounds	Neutralize or dilute chemical Remove clothing Consult poison control center
Electrical	Direct current Alternating current Lightning	Disconnect source of current Initiate CPR if necessary Move to area of safety Consult electrical experts
Radiation	Solar X-rays Radioactive agents	Shield the skin appropriately Limit time of exposure Move the patient away from the radiation source Consult a radiation expert

AGE, DEPTH, AND SIZE OF BURN

Degree of Burn	Signs & Symptoms	Action Plan	
First Degree Damage to top layer of skin only. Takes up to a week to heal	1) Mild swelling 2) Red 3) Painful Note: The area turns from red to white when you press them	1) Run or soak the affected area(s) in cool water for 5 to 20 minutes. Do not use cold water or ice baths 2) Apply Burnaid gel 3) Cover with an appropriate dressing 4) Clean the wound and change the dressing everyday	
Second Degree Damage to top layer of skin and the layer underneath. Takes about 3 weeks to heal	1) Very swollen 2) Very red 3) Very painful Note: Blisters may form and turn white when you press them	1) Run or soak the affected area(s) in cool water for 20 minutes. Do not use cold water or ice baths 2) Seek medical assistance Note: Do not pop any blisters	
Third Degree Damage to first, second and underlying layers of tissues. Nerves are affected and possibly the bones and muscles. Takes more than 3 weeks to heal	1) Skin looks white or charred 2) Little or no pain at the site of burn 3) Surrounding skin will be very painful Note: 1) Skin do not turn pale when touched 2) The skin does not hurt when you touch it because it cannot feel anything	1) Seek medical assistance immediately Note: If possible, raise the burned area above the level of the heart to reduce swelling and pain	

HISTORY OF CARDIAC, PULMONARY, RENAL, HEPATIC DISEASES AND INJURIES SUSTAINED DURING BURN INJURY

- Pre-existing disease conditions would reduce normal compensatory responses to minor hypovolemia
- Optimum systemic functioning is very vital for the burned client to respond to the burn management such as fluid resuscitation, nutritional correction and infection prevention
- Injuries sustained during burn injury like fractures requires prolong hospitalization and additional management

STAGES OF INTERDISCIPLINARY CARE

- clinical course of treatment for the burn patient are divided into three stages
- stages are useful to determine the clinical needs of the patient

STAGES OF BURN CARE

Phase	Duration	Priorities
Emergent / immediate Resuscitative	• onset of injury to completion of fluid resuscitation 48 -72 HRS post injury	• Ist aid, prevention of shock • Prevention of respiratory distress • Detection and treatment of injury • Wound assessment/initial care
Acute	• Beginning of diuresis to need completion of wound closure	• Wound care and closure prevention or treatment, of complication nutritional support
Rehabilitative	• From major wound closure to return of individual's optimal level of physical and psychosocial adjustment	• Prevention of scars and contractures • Physical, occupational, vocational rehabilitation • Functional cosmetic reconstruction, psychosocial counseling

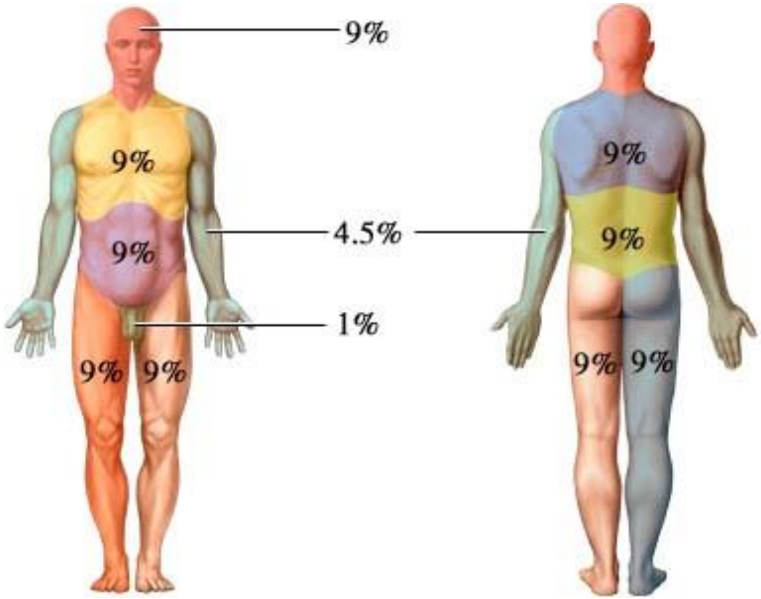
EMERGENT OR RESUSCITATIVE PHASE

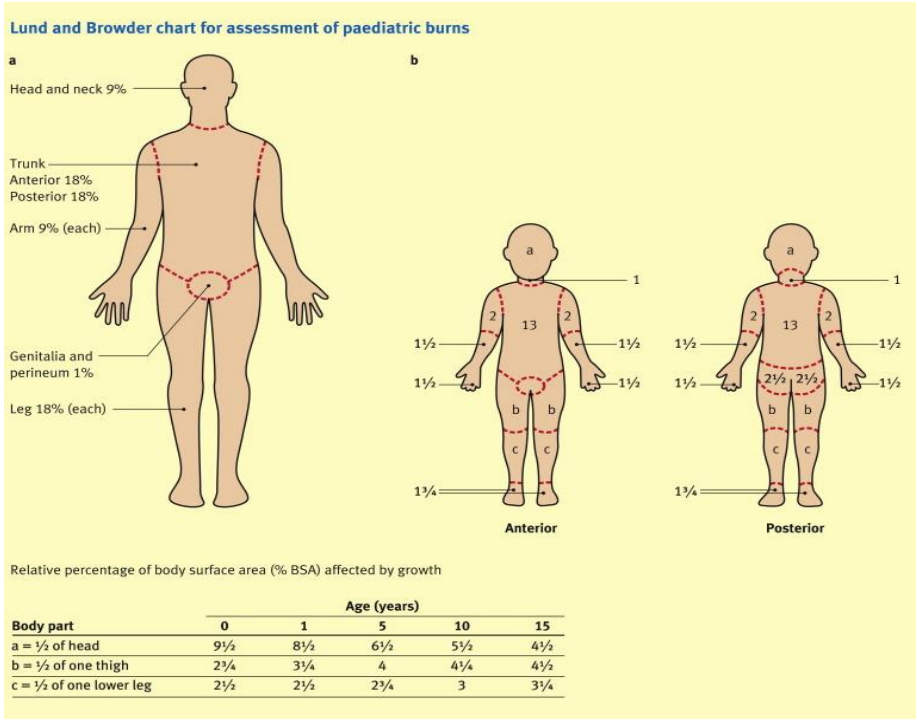
- Pre -Hospital Care
  - Remove person from source of burn
  - Assess ABC and trauma
  - Cover burn with sterile or clean cloth
  - Remove constricting clothes and jewelry
  - Transport immediately
- Emergency Care for Minor Burn
  - Administer pain medication
  - Administer Tetanus prophylaxis
  - Wound care
  - Apply topical antibiotics

- Emergency Care for Major Burns
  - Evaluate degree and extent of burn
  - Established patent airway and administer oxygen for burn victims in enclosed area
  - Venoclysis and assess for hypovolemia
  - Maintain NPO and insert NGT
  - Insert foley catheter
  - Tetanus prophylaxis and give pain medication
- Fluid management is one approach to treat burn patients
- Within minutes of burn injury, a massive amount of fluid shifts from the intracellular and Intravascular compartments into the interstitial (third spacing)

COMPUTATION OF BSA

- extent of the burn injury size is expressed as percentage of the total body surface area (TBSA)
- Methods:
  - The "Rule of Nines" → a rapid method of estimation of the burn size
  -





COMMON FORMULA FOR FLUID RESUSCITATION

- Fluid resuscitation is the administration of the intravenous fluids to restore the circulating blood volume during the acute period of capillary permeability → prevent burn shock
- Parkland and modified Brooke formula
  - infuse the 50% volume of fluid during the first 8 hours and the remaining 50% to be infused over the next 16 hours
- Parkland formula
  - lactated Ringer solution is administered 4ml X kg X % TBSA burn
- Modified Brooke
  - lactated Ringer solution is administered 2ml X kg X % TBSA burn
- Hourly urine output is measured to determine if fluid resuscitation is effective

OTHER INTERVENTIONS DURING RESUSCITATIVE PHASE

- Elevate the head of the bed to 30 degrees for facial and head burns
- Elevate circumferential burns of the extremities with a pillow above the level of the heart
- Assess for infection, tracheal or laryngeal edema
- Protective isolation techniques Shave hair around wound margins
- Monitor gastric output and Ph for stress ulcer (Curling's Ulcer)
- Administer anti-ulcer drugs
- Avoid IM and SQ administration
- NPO until with bowel sound
- Monitor daily weights

ACUTE PHASE

- Begins with hemodynamic stability
- capillary permeability restored and diuresis begun
- Primary concern: Restorative therapy to wound closure and infection control
  - Aseptic technique
  - adequate debridement of wound
  - tetanus immunization
  - IV antibiotics
  - topical anti-bacteria therapy
  - wound care
- Escharotomy - surgical incision through the eschar into the subcutaneous tissues to allow the extremity to continue to swell without compressing the underlying blood vessels
- Fasciotomy - Surgical procedure where the fascia is cut to relieve tension or pressure commonly to treat the resulting loss of circulation to an area of tissue or muscle. Fasciotomy is a limb-saving procedure when used to treat acute compartment syndrome
- Hydrotherapy

ANTIMICROBIAL AGENTS USED

- Silver Sulfadiazine cream
- Mafenide Acetate cream
- Silver Nitrate Solution

REHABILITATIVE PHASE

GOALS:

- gain independence
- achieve maximal use of the affected part
- promote wound healing
- minimize deformities
- increase strength and function
- provide emotional support

NURSING DIAGNOSIS AND MANAGEMENT

- Impaired gas exchange related to carbon monoxide poisoning, smoke inhalation upper airway loss
  - Maintain adequate perfusion
- Ineffective airway clearance related to edema and effects of smoke inhalation
  - Maintain patent airway
  - FVD related to ↑ capillary permeability and evaporative losses from burn wound
  - Restore optimal fluid balance and perfusion of vital organs
- Pain related to tissue/nerve/emotional impact injury
  - Assess level of discomfort
  - Administer pain relievers and antibiotics
  - Provide emotional support to allay fear and anxiety

RENAL FAILURE

RENAL FAILURE

- Condition wherein the kidneys cannot remove the body's metabolic waste products or when it cannot perform its regulatory functions
- Two types of renal failure:
  - Acute renal failure (ARF)/acute kidney injury (AKI)
  - Chronic renal failure

ACUTE RENAL FAILURE (ARF)

ACUTE RENAL FAILURE (ARF)

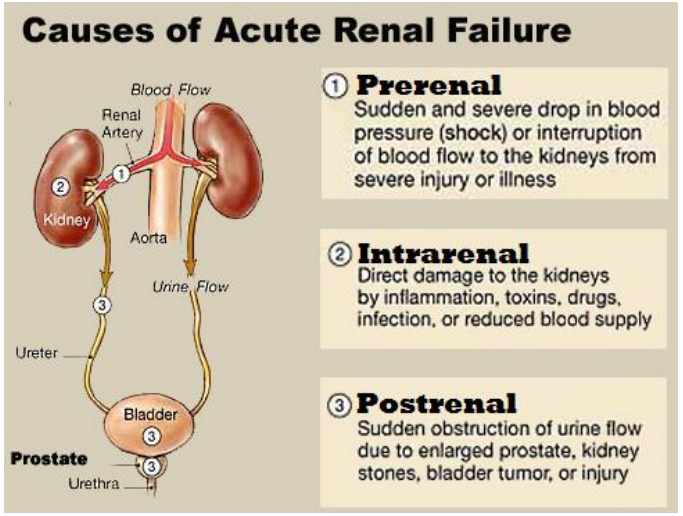
- reversible clinical syndrome wherein there is a sudden and almost complete loss of kidney function
- may last for days, weeks or even months
- some patients with ARF may die and for some may progress to chronic renal failure

Categories of ACUTE RENAL FAILURE (ARF)

I. Prerenal
- Occurs in 60% to 70% of cases which result from impaired blood flow
- If the kidneys do not receive sufficient blood that leads to hypoperfusion of the kidney and decreases gfr

II. Intrarenal
- Results from parenchymal damage to the glomeruli or kidney tubules, resulting from prolonged renal ischemia resulting from myoglobinuria

III. Postrenal
- result of an obstruction somewhere distal to the kidney
- pressure increases in the kidney tubules and eventually the GFR decreases
- blockage causes urine to back up and harm the kidneys
- causes are urinary tract obstruction like calculi, tumors, benign prostatic hyperplasia or an enlarged prostate, kinked ureter, and blood clots



STAGES OF ACUTE RENAL FAILURE

1. Initiation period
   - kidney begins with the initial injury and ends when oliguria develops
   - reduced blood flow to the nephrons
   - decreased reabsorption of water, electrolytes, and excretion of protein wastes and excess metabolic substances
   - can last from hours to days
   - urine output at 30 cc per hour or less
2. Oliguria period
   - reduction in the glomerular filtration rate
   - increase in the serum concentration of creatinine, urea, uric acid, and the intracellular cations like potassium and magnesium
   - uremic symptoms first appear and may develop life-threatening condition
   - hyperkalemia, hypernatremia, hyperphosphatemia, hypocalcemia, hypermagnesemia and metabolic acidosis
   - urine output of less than 400 ml /24 hours
   - may last for 1 to 2 weeks

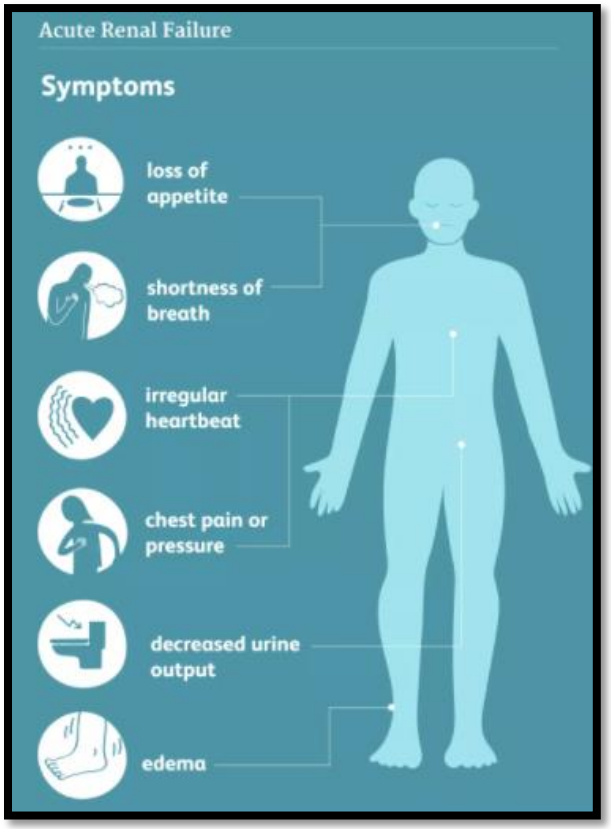
3. Diuresis period
   - gradual increase in urine output
   - glomerulus filtration has started to recover
   - increased BUN and creatinine result
   - urine output ranges from 3 to 5 liters per day due to partial regenerated tubule's inability to concentrate urine
   - lasts for 2 to 3 weeks
4. Recovery period
   - signals the improvement of renal function
   - may take 3 to 12 months
   - Laboratory values return to the patient's normal value

PATHOPHYSIOLOGY OF AKI

Figure 6-6 Pathophysiology of AKI

Pre	Intra	Post
↓ BLD supply	Precipitation occurs (CHON/HGB liberated)	obstruction
↓	↓	↓
renal ischemia	Interferes with urine excretion	↑intrarenal pressure
↓	↓	↓
tubular damage	kidney swells	↓ GFR
↓	↓	↓
↓ GFR	necrosis	↓ output /retention of waste
↓		
↓ urine output		
↑ retention of waste		

SIGNS AND SYMPTOMS OF AKI



DIAGNOSTIC MANAGEMENT
- History taking, physical exam
- Identify precipitating cause
- Creatinine, BUN, Serum electrolytes
- Urinalysis

MEDICAL MANAGEMENT
- Treatment of precipitating cause
- Fluid restriction
- Dietary management
- Dialysis
- Total Parenteral Nutrition (TPN) if indicated
- Measures to decrease potassium level (most life-threatening disturbance)
  A. Administer cation-exchange resin
     - Sodium polysterene sulfonate (Kayexalate)

- Calcium polysterene sulfonate (kalimate)

B. IV glucose and insulin
C. Ca gluconate
D. Bicarb IV
E. Dialysis
F. Dietary restriction

NURSING MANAGEMENT

Goal of treating patients with acute renal failure is to correct or eliminate any reversible causes of kidney failure

- Precise I/O monitoring
- Vital signs
- Dietary management
- Fluid management base on the clinical manifestation
- Daily weight
- Assess signs and symptoms of electrolyte imbalance such as:
  - mental status
  - neuromuscular status
  - cardiac rate and rhythm

NURSING DIAGNOSIS

- Fluid volume excess related to impaired kidney function
- Decrease cardiac output related to high output renal failure (diuretic phase)
- Fluid volume deficit related to high output renal failure (diuretic phase)
- Potential for complications of immobility related to therapeutic restrictions
- Deficient Knowledge related to condition and treatment
- Risk for Imbalanced Nutrition: Less Than Body Requirements

CHRONIC RENAL FAILURE

CHRONIC RENAL FAILURE

- AKA End Stage Renal Disease (ESRD)
- a progressive, irreversible deterioration in renal function in which the body's ability to maintain metabolic and fluid and electrolyte balance fails, resulting in uremia or azotemia
- Contributing factors:
  - Systemic factors
    - diabetes mellitus
    - Hypertension
    - chronic glomerulonephritis
    - Pyelonephritis
    - obstruction of the urinary tract
  - Hereditary lesions
    - polycystic kidney disease
    - vascular disorders
    - autoimmune disorders
    - Infections
    - medications which are nephrotoxic

STAGES OF CHRONIC RENAL FAILURE

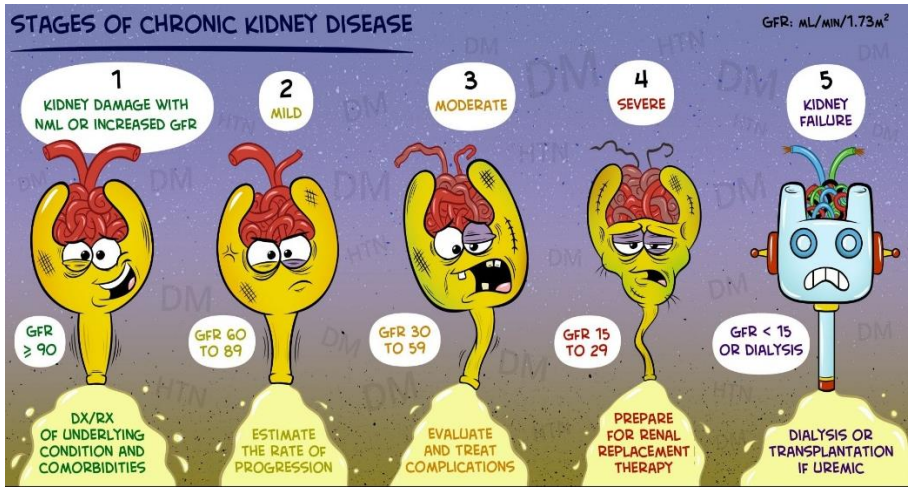
Stage 1. Decreased Renal Reserve
- residual renal function is 40-75%
- asymptomatic with normal BUN and plasma creatinine
- Excretory and regulatory renal functions are undamaged

Stage 2. Renal Insufficiency
- renal function is 20-40% marked decrease in glomerular filtration rate
- decreased solute clearances, ability to concentrate urine and hormone secretion
- signs and symptoms include rising BUN, plasma creatinine
- mild azotemia, polyuria, nocturia and anemia

Stage 3. Renal Failure
- Renal function is approximately 5% of normal
- Serum urea & creatinine levels rise rapidly
- Urine output is less than 500ml/day
- Symptoms of uremia develop

Stage 4. End Stage Renal Disease

- residual renal function less than 15% or normal
- Its excretory, regulatory and hormonal renal functions are severely impaired
- Unable to maintain homeostasis like fluids and electrolytes imbalance may occur as well as pH imbalances
- Noticeable are elevated BUN and plasma creatinine, anemia, hyperphosphatemia, hypocalcemia, hyperkalemia, fluid overload, usually oliguria . Uremic syndrome may occur and all body



MEDICAL MANAGEMENT OF CHRONIC RENAL FAILURE

- Laboratory:
  - Serum studies (nitrogenous wastes, electrolytes)
  - Complete blood count
  - Urinalysis / Culture
  - 24 hour creatinine clearance (to determine GFR)
- Medical Treatment Goals:
  - To prevent further damage to the kidneys
  - To promote recovery of renal function
  - To prevent complications
  - Pharmacologic (anti-hypertensive, erythropoietin, iron supplement, phosphate binding, calcium supplement, anti seizure, ketoanalogues, Sodium Bicarbonate, Potassium resin exchange (Kayexalate)
  - Nutritional - reduce protein potassium food sources, fluids
  - Blood transfusion to correct anemia
  - Correction of fluid and electrolyte imbalance
  - Dialysis
  - Kidney transplant