



Fluids and Electrolytes

FLUID AND ELECTROLYTE BALANCE

Maintenance of the proper amount, distribution, and composition of body fluids and electrolytes

Body Fluid Compartments

	location	major cation
ICF	inside cells	potassium
ECF	outside cells	sodium

ECF consists of:

Intravascular → inside blood vessels

Interstitial → between and around cells

Electrolytes

Substances that dissociate into electrically charged ions when dissolved in body fluids.

Major electrolytes: Sodium, Potassium, Calcium, Magnesium

Electrolyte	Normal range
Sodium	135-145 mEq/L
Potassium	3.5-5.0 mEq/L
Calcium	8.5-10.5 mg/dL
Magnesium	1-3-2.1 mEq/L

Regulation of Fluid Balance

1. Kidneys: primary regulators of water, sodium, potassium, urine concentration, and acid-base balance
2. Antidiuretic hormone: retains water by increasing renal water reabsorption
3. Aldosterone: retains sodium and excretes potassium
4. Thirst: promotes fluid intake when the body needs water.

FLUID MOVEMENTS

Process	What happens?
Osmosis	water moves toward higher solute concentration
Diffusion	particles move from high to low concentration
Filtration	fluid/solutes move due to pressure differences
Active Transport	substances move against concentration gradient using energy (ATP).



FLUID VOLUME IMBALANCES

Fluid Volume Deficit

The body loses more fluid than it takes in.

CAUSES:

Vomiting/Diarrhea, Fever/Excess sweating, Hemorrhage, Burns, Excessive diuresis, GI losses, Third spacing due to burns

Assessment findings:

Cardiovascular: ↑ Heart rate, weak pulse, ↓BP, orthostatic hypotension

Renal: ↓ urine output, concentrated urine

Neurologic: weakness, dizziness, confusion if severe

Skin: dry mucous membranes, ↓ skin turgor

Remember:

When there is low fluid volume, the body will compensate by increasing heart rate to increase cardiac output, and by keeping fluids in the body through decreased urination.

Nursing interventions:

1. Assess BP, HR, perfusion, and mental status
2. Monitor I&O and urine output
3. Obtain daily weights
4. Monitor electrolytes and renal function
5. Replace fluids as ordered:
 - Oral fluids if appropriate
 - IV fluids if needed
 - Blood products if indicated
6. Identify and treat the cause

Fluid Volume Excess

The body retains more fluid than it can eliminate.

CAUSES:

Heart failure, Renal failure, Excessive IV fluid therapy, Excess sodium, Liver disease, Corticosteroid therapy

Assessment findings:

Cardiovascular: ↑BP, Bounding pulse, Jugular Vein distention (JVD)

Respiratory: Dyspnea, Crackles, Pulmonary congestion, Orthopnea

Peripheral: Edema, Weight gain

Nursing interventions:

1. Assess respiratory status: Respiratory rate, SpO₂, Lung sounds, Work of breathing
2. Position accordingly: High Fowler's if dyspneic
3. Monitor I&O
4. Obtain daily weights
5. Assess edema and JVD
6. Administer diuretics and oxygen as prescribed; implement fluid/sodium restriction
7. Treat underlying cause

Sodium (Na)

Normal: 135-145 mEq/L

Sodium is the primary extracellular cation. It regulates fluid balance, water distribution, nerve impulses, muscle contraction and blood volume.

Remember: "Where sodium goes, Water follows."

Hyponatremia - $\text{Na}^+ < 135 \text{ mEq/L}$

Sodium is low relative to the amount of water inside the body

CAUSES:

Sodium loss

Sodium wasting diuretics (thiazide)

Vomiting/diarrhea

Excessive sweating

Adrenal insufficiency

Dilutional Hyponatremia- Too much water
SIADH

Excessive water intake

Heart failure

Renal dysfunction



Remember: Dilutional hyponatremia does not automatically mean the body has lost sodium. The sodium is diluted because there is too much water relative to sodium.

Assessment findings:

Mild: Headache, N&V, Lethargy, Muscle cramps

Severe: Confusion, Altered mental status, Seizure, ↓LOC, Coma

Remember:

Hyponatremia: Water moves into the brain cells → cerebral edema → neurologic changes = Seizure

HIGHEST PRIORITY: RISK FOR SEIZURE



Nursing interventions:

1. Monitor neurological status and report any changes
2. Implement seizure precautions
3. Monitor serum sodium and fluid status
4. Monitor for signs of fluid excess (crackles, edema, distended jugular vein)
5. If caused by FLUID EXCESS (dilutional hyponatremia) : The goal is to remove excess water
6. If caused by SODIUM/FLUID LOSS: The goal is to replace sodium and volume

Hypernatremia - $\text{Na}^+ > 145 \text{ mEq/L}$

This generally means: too little water relative to sodium. The problem is often water loss, rather than simply excessive sodium intake.

CAUSES

Dehydration (diarrhea), inadequate water intake, excessive sweating, fever, Diabetes Insipidus, osmotic diuresis, excess sodium administration

Assessment findings:

Intense thirst, restlessness, irritability, weakness, confusion, neurologic changes from cerebral dehydration, seizures, decreased LOC.

Remember:

Hypernatremia → cellular dehydration → neurologic changes
Because brain cells lose water, severe hypernatremia can lead to confusion, seizures, and decreased LOC

NURSING INTERVENTIONS: Same as hyponatremia + slow fluid replacement.

Potassium - K^+

Normal: 3.5-5.0 mEq/L

Potassium is essential for nerve transmission, skeletal muscle contraction, and cardiac electrical activity

Potassium Regulation:

- Primarily regulated by the kidneys
- Aldosterone: Retains Sodium and promotes potassium excretion
- Loop and thiazide diuretics: promote potassium wasting
- Renal failure: Impaired potassium excretion leads to hyperkalemia

Remember: Abnormalities in potassium can result in potentially fatal cardiac dysrhythmias and muscle weakness. When potassium abnormalities are substantial or symptomatic, ECG evaluation is especially crucial.

Hypokalemia - $\text{K}^+ < 3.5 \text{ mEq/L}$

Think: ↓ K^+ = Muscle weakness + ↓ GI motility + Cardiac dysrhythmia

CAUSES

Loop/Thiazide diuretics, Vomiting, Diarrhea, GI suction, Increased renal K^+ losses, Inadequate potassium intake

**Assessment Findings:**

Musculoskeletal: Muscle weakness, muscle cramps, fatigue

GI: Decreased intestinal motility, constipation, hypoactive bowel sounds, abdominal distention, N&V

Cardiac: Dysrhythmias

ECG may show:

- Flattened T waves
- Prominent U waves
- ST- segment depression

**Nursing Interventions**

1. (#1 priority) Monitor cardiac rhythm when indicated
 - Assess ECG/cardiac rhythm
 - Watch out for dysrhythmias, particularly if the client has severe or symptomatic hypokalemia.
2. Replace potassium as ordered
 - Oral potassium is commonly used for mild/moderate cases when the client can tolerate it.
 - IV replacement may be necessary for severe or symptomatic hypokalemia or when oral therapy is not appropriate. Hold potassium supplements if urine output is less than 30 mL/hr.
 - In accordance with the prescribed order, potassium must be diluted and infused at a controlled rate.
 - **Rapid administration can cause fatal cardiac dysrhythmias/cardiac arrest. NEVER give potassium IV push.**
3. Monitor response to replacement
3. Encourage potassium-rich foods when appropriate: Banana, Oranges, Potatoes, Raisins/dried fruits.

Remember: Diet alone is generally not enough to rapidly correct significant hypokalemia

Hyperkalemia- $K^+ > 5.0$ mEq/L

Think: $\uparrow K^+$ = muscle weakness + cardiac dysrhythmias

CAUSES

Renal failure/ Renal impairment

Potassium-sparing diuretics
(Spironolactone)

Adrenal insufficiency

Excess potassium administration

Cellular/Tissue breakdown

Assessment findings:

Musculoskeletal: Muscle weakness, Flaccid paralysis in severe cases

GI: Increased intestinal motility, Diarrhea, Abdominal cramping

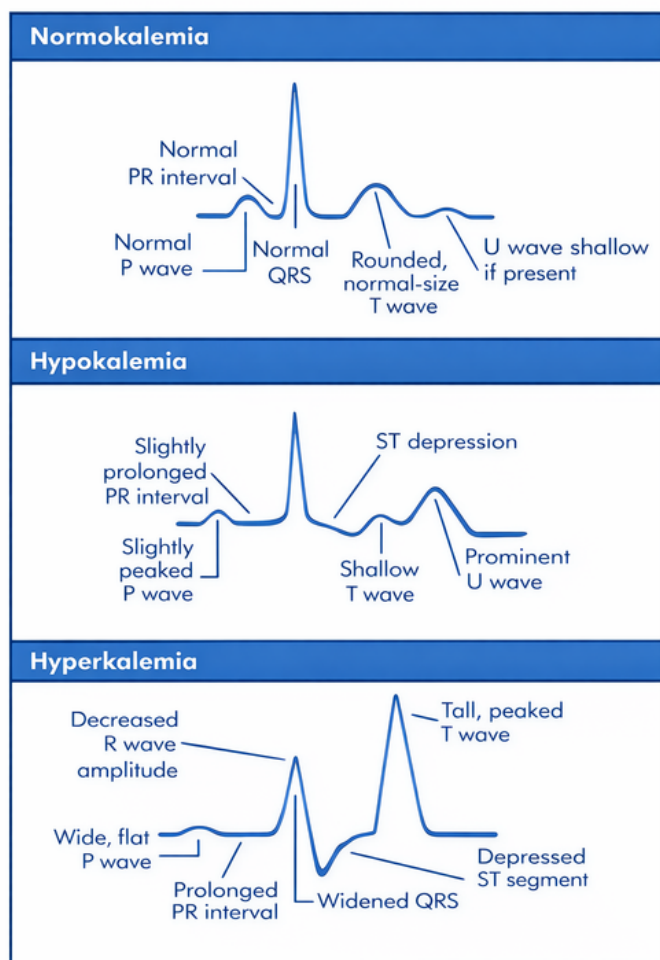
Cardiac: Dysrhythmias

ECG may show:

- Tall/ Peaked T waves
- Prolonged PR
- Widened QRS
- Severe toxicity- Potential fatal rhythm disturbances

**Nursing Interventions**

1. (#1 priority) Assess cardiac status immediately
2. Stop potassium sources as prescribed
3. Provide IV calcium gluconate to stabilize heart
4. Shift potassium INTO cells
5. Insulin (moves potassium into cells) + Dextrose IV (reduces the risk of hypoglycemia)
5. Remove excess potassium from the body
 - Potassium-binding medications -Sodium polystyrene sulfonate (Kayexalate)
 - Loop diuretics (furosemide) when appropriate
 - Hemodialysis- especially important in severe hyperkalemia with significant renal impairment

**Calcium - Ca^{2+}** **Normal: 8.5-10.5 mg/dL**

Calcium is essential for muscle contraction, nerve transmission, blood clotting, and bone/teeth strength

Calcium regulation

- Mainly regulated by the parathyroid hormone (PTH), vitamin D, kidneys and bones.
- PTH increases blood calcium by promoting calcium release from bone, increasing renal reabsorption, activating vitamin D.
- Calcitonin lowers blood calcium by promoting calcium deposition in bone

Remember: Calcium abnormalities particularly affect the neuromuscular system and cardiac function
 Calcium Imbalance $\rightarrow$ muscle/nerve changes + possible cardiac dysrhythmias

Hypocalcemia $\text{Ca}^{2+} < 8.5 \text{ mg/dL}$

Think: $\downarrow$ calcium $\rightarrow$ increased nerve/muscle excitability

CAUSES

Hypoparathyroidism, Vitamin D deficiency, Chronic kidney disease, pancreatitis, malabsorption, low calcium intake, massive blood transfusion, hyperphosphatemia (phosphates are binding calcium)

Assessment findings

Neuromuscular: Tingling/paresthesias around mouth, fingers or toes, Muscle cramps, muscle spasms, tetany, hyperactive deep tendon reflexes (DTRs), seizures if severe; osteoporosis and pathologic fractures if chronic.

Classic signs:

- Chvostek sign – facial twitch when facial nerve is tapped
- Trousseau sign – Carpal spasm when BP cuff is inflated

Cardiac: prolonged QT interval, dysrhythmias

Remember:

Low Ca^{2+} = Twitchy

Tingling > Twitching > Tetany > seizures

Nursing interventions

1. Assess neuromuscular and cardiac status. Monitor for muscle spasms, tetany, seizures.
2. PRIORITIZE SAFETY. Implement seizure precautions.
3. Administer IV calcium gluconate for severe cases
4. Provide Vitamin D supplements.

Remember: IV Calcium must be administered carefully, because rapid administration can cause cardiac dysrhythmias

**Hypercalcemia - $\text{Ca}^{2+} > 10.5 \text{ mg/dL}$**

Think: increased calcium = sluggish neuromuscular activity + kidney/GI problems

CAUSES

hyperparathyroidism, malignancy, Prolonged immobilization, Excess vitamin D, Certain medications

Assessment findings

Neuromuscular: muscle weakness, fatigue, decreased reflexes, confusion, lethargy

GI: constipation, abdominal discomfort, nausea and vomiting, decreased bowel motility

Renal: kidney stones, polyuria, polydipsia, dehydration

Cardiac: shortened QT interval, dysrhythmias

Think: HIGH Calcium = sluggish

Weak > slow gut > constipation; Kidney stones

Nursing interventions:

1. Monitor cardiac status, including ECG and watching out for dysrhythmias
2. Promote hydration: encourage oral fluids, if appropriate, IV isotonic fluids may be prescribed
3. Promote mobility
4. Administer prescribed medications depending on the cause/severity: loop diuretics after adequate hydration when appropriate, calcitonin, bisphosphonates
5. Monitor: calcium levels, renal function, I&O, ECG, Neurologic status

Magnesium - Mg^{2+}

Normal: 1.3-2.1 mEq/L

Magnesium helps regulate neuromuscular activity, cardiac conduction, muscle contraction, and electrolyte balance.

Magnesium regulation:

- Mainly regulated by kidneys
- Renal impairment: magnesium retention - > hypermagnesemia
- GI losses and certain medications can cause magnesium deficiency.

Remember: magnesium imbalances can cause neuromuscular changes and potentially dangerous cardiac effects.

Hypomagnesemia - $\text{Mg}^{2+} < 1.3 \text{ mEq/L}$

Think: Decreased magnesium → Increased neuromuscular excitability

CAUSES

Diarrhea/vomiting, malnutrition, alcohol use disorder, malabsorption, diuretics, prolonged poor intake

Assessment findings:

Neuromuscular: tremors, muscle cramps, weakness, hyperactive deep tendon reflexes (DTRs), tetany, seizure if severe

Cardiac: dysrhythmias, prolonged QT, Torsades de pointes can occur

Other: Irritability, confusion

Think: Low magnesium = twitchy

Tremors → hyperreflexia → tetany → seizures

Nursing interventions:

1. Monitor cardiac status: assess ECG/Cardiac rhythm when indicated, and watch for dysrhythmias and QT prolongation
2. Assess neuromuscular status: Monitor reflexes, muscle cramps, tetany, and seizures
3. Replace magnesium as prescribed: **Magnesium Sulfate**
 - Oral for mild deficiency when appropriate
 - IV for severe/symptomatic. Monitor for signs of magnesium toxicity (respiratory depression, decreased/absent DTRs)

Remember: IV magnesium must be administered carefully, and according to the prescribed rate. Keep calcium gluconate readily available.



Hypermagnesemia $Mg^{2+} > 2.1 \text{ mEq/L}$

CAUSES

Renal failure/renal impairment, excessive magnesium-containing medications or supplements, Magnesium-containing antacids/laxatives, especially with renal impairment

Assessment findings:

Neuromuscular: decreased deep tendon reflexes, muscle weakness, lethargy, decreased LOC, flaccid paralysis if severe

Cardio: hypotension, bradycardia, dysrhythmias

Respiratory: respiratory depression, respiratory arrest if severe

Think: High magnesium= slow

Slow reflexes -> slow heart -> slow breathing



Nursing Interventions:

1. Monitor respiratory and cardiac status first: assess respiratory rate and effort, SpO₂, Monitor BP and HR, monitor ECG when indicated
2. Stop magnesium sources: hold/discontinue magnesium-containing medications or supplements as prescribed. (laxatives, antacids)
3. monitor renal function and urine output.
4. Administer prescribed treatment
5. IV calcium gluconate may be given to counteract magnesium's cardiac and neuromuscular effects.
6. IV fluids and loop diuretics may be used when appropriate and renal function is adequate.
7. Hemodialysis may be required for severe hypermagnesemia, particularly with significant renal impairment.

Remember:

Calcium does NOT remove magnesium. It antagonizes/stabilizes its effects, especially on the heart and neuromuscular system.