

FLUID AND ELECTROLYTE IMBALANCES

Body fluid is composed primarily of water and electrolytes. The body is equipped with homeostatic mechanisms to keep the composition and volume of body fluids within narrow limits. Organs involved in this mechanism include the kidneys, lungs, heart, blood vessels, adrenal glands, parathyroid glands, and pituitary gland. Body fluid is divided into two types: intracellular (within the cells) and extracellular (interstitial or tissue fluid, intravascular or plasma, and transcellular, such as cerebrospinal or synovial fluids).

RELATED CONCERNS

All plans of care specific to underlying health condition causing imbalance, e.g., DM, HF, upper GI bleeding, renal failure/dialysis.

Metabolic acidosis (primary base bicarbonate deficit)

Metabolic alkalosis (primary base bicarbonate excess)

Respiratory acidosis (primary carbonic acid excess)

Respiratory alkalosis (primary carbonic acid deficit)

NURSING PRIORITIES

1. Restore homeostasis.
2. Prevent/minimize complications.
3. Provide information about condition/prognosis and treatment needs as appropriate.

DISCHARGE GOALS

1. Homeostasis restored.
2. Free of complications.
3. Condition/prognosis and treatment needs understood.
4. Plan in place to meet needs after discharge.

Note: Because fluid and electrolyte imbalances usually occur in conjunction with other medical conditions, the following information is offered as a reference. The interventions are presented in a general format for inclusion in the primary plan of care.

FLUID BALANCE

Total body water, essential for metabolism, declines with age and also varies with body fat content and gender. It constitutes about 80% of an infant's body weight, 60% of an adult's, and as little as 40% of an older person's weight.

Hypervolemia (Extracellular Fluid Volume Excess)

PREDISPOSING/CONTRIBUTING FACTORS

Excess sodium intake including sodium-containing foods, medications, or fluids (PO/IV)

Excessive, rapid administration of hypertonic (or possibly isotonic) parenteral fluids

Increased release of antidiuretic hormone (ADH); excessive adrenocorticotropic hormone (ACTH) production, hyperaldosteronism

Decreased plasma proteins as may occur with chronic liver disease with ascites, major abdominal surgery, malnutrition/protein depletion

Chronic kidney disease/acute renal failure (ARF)

Heart failure (HF)

Patient Assessment Database

ACTIVITY/REST

May report: Fatigue, generalized weakness

CIRCULATION

May exhibit: Hypertension, elevated central venous pressure (CVP)

Pulse full/bounding; tachycardia usually present; bradycardia (late sign of cardiac decompensation)
Extra heart sounds (S₃)
Edema variable from dependent to generalized
Neck and peripheral vein distension

ELIMINATION

May report: Decreased urinary output, polyuria if renal function is normal
Diarrhea

FOOD/FLUID

May report: Anorexia, nausea/vomiting
Thirst (may be absent, especially in elderly)

May exhibit: Abdominal girth increased with visible fluid wave on palpation (ascites)
Sudden weight gain, often in excess of 5% of total body weight
Edema initially dependent, pitting may progress to facial/periorbital, general/anasarca

NEUROSENSORY

May exhibit: Changes in level of consciousness, from lethargy, disorientation, confusion to coma;
aphasia
Muscle twitching, tremors, seizure activity
Hyperreflexia, rigid paralysis (severe hypernatremia)

PAIN/DISCOMFORT

May report: Headache
Abdominal cramps

RESPIRATION

May report: Shortness of breath
May exhibit: Tachypnea with/without dyspnea, orthopnea; productive cough
Crackles

SAFETY

May exhibit: Fever
Skin changes in color, temperature, turgor, e.g., taut and cool where edematous

TEACHING/LEARNING

Refer to predisposing/contributing factors
Discharge plan considerations: **DRG projected mean length of inpatient stay: depends on underlying condition**
May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible postdischarge considerations.

DIAGNOSTIC STUDIES

Hematocrit: Elevated in dehydration, decreased in fluid overload.

Serum sodium: May be high, low, or normal (between 135 and 145 mEq/L).

Serum potassium and BUN: Normal, or decreased in fluid overload unless renal damage present.

Total protein: Plasma proteins/albumin may be decreased.

Serum osmolality: Usually unchanged, although hypo-osmolality may occur.

Urine sodium: May be low because of sodium retention.

Urine specific gravity: Decreased.

Chest x-ray: May reveal signs of congestion.

NURSING DIAGNOSIS: Fluid Volume excess

May be related to

Excess fluid or sodium intake
Compromised regulatory mechanism

Possibly evidenced by

Signs/symptoms noted in database

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Fluid Balance (NOC)

Demonstrate stabilized fluid volume as evidenced by balanced I&O, vital signs within patient's normal range, stable weight, and absence of signs of edema.

Knowledge: Treatment Regimen (NOC)

Verbalize understanding of individual dietary/fluid restrictions.
Demonstrate behaviors to monitor fluid status and prevent/limit recurrence.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hypervolemia Management (NIC)</p> <p>Independent</p> <p>Monitor vital signs, also CVP if available.</p> <p>Auscultate lungs and heart sounds.</p> <p>Assess for presence/location of edema formation.</p> <p>Note presence of neck and peripheral vein distension, along with pitting edema, dyspnea.</p> <p>Maintain accurate I&O. Note decreased urinary output, positive fluid balance (intake higher than output) on 24-hr calculations.</p>	<p>Tachycardia and hypertension are common manifestations. Tachypnea usually present with/without dyspnea. Elevated CVP may be noted before dyspnea and adventitious breath sounds occur. Hypertension may be a primary disorder or occur secondary to other associated conditions, e.g., HF.</p> <p>Adventitious sounds (crackles) and extra heart sounds (S₃) are indicative of fluid excess. Pulmonary edema may develop rapidly.</p> <p>Edema can be either a cause or a result of various pathological conditions reflecting four competing forces—blood hydrostatic and osmotic pressures, and interstitial fluid hydrostatic and osmotic pressures. The dynamic interaction of these four forces allows fluid to shift from one body compartment to another. Edema may be generalized or localized in dependent areas. Elderly patients may develop dependent edema with relatively little excess fluid. <i>Note:</i> Patients in a supine position can have an increase of 4–8 L of fluid before edema is readily detected.</p> <p>Signs of cardiac decompensation/HF.</p> <p>Decreased renal perfusion, cardiac insufficiency, and fluid shifts may cause decreased urinary output and edema formation.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hypervolemia Management (NIC)</p> <p>Independent</p> <p>Weigh as indicated. Be alert for acute or sudden weight gain.</p> <p>Give oral fluids with caution. If fluids are restricted, set up a 24-hr schedule for fluid intake.</p> <p>Monitor infusion rate of parenteral fluids closely; administer via control device/pump as necessary.</p> <p>Encourage coughing/deep-breathing exercises.</p> <p>Maintain semi-Fowler's position if dyspnea or ascites is present.</p> <p>Turn, reposition, and provide skin care at regular intervals.</p> <p>Encourage bedrest. Schedule care to provide frequent rest periods.</p> <p>Provide safety precautions as indicated, e.g., use of side rails, bed in low position, frequent observation, soft restraints (if required).</p>	<p>One liter of fluid retention equals a weight gain of 2.2 lb.</p> <p>Fluid restrictions, as well as extracellular shifts, can aggravate drying of mucous membranes, and patient may desire more fluids than are prudent.</p> <p>Sudden fluid bolus/prolonged excessive administration potentiates volume overload/risk of cardiac decompensation.</p> <p>Pulmonary fluid shifts potentiate respiratory complications.</p> <p>Gravity improves lung expansion by lowering diaphragm and shifting fluid to lower abdominal cavity.</p> <p>Reduces pressure and friction on edematous tissue, which is more prone to breakdown than normal tissue.</p> <p>Limited cardiac reserves result in fatigue/activity intolerance. In addition, lying down favors diuresis and reduction of edema.</p> <p>Fluid shifts may cause cerebral edema/changes in mentation, especially in the geriatric population. <i>Note:</i> Application of restraints can increase agitation, requiring alternative interventions (e.g., one-on-one monitoring, sedation). Use of side rails may place the elderly confused patient at risk for entrapment injury/death.</p>
<p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory studies as indicated, e.g., electrolytes, BUN, ABGs.</p> <p>Provide balanced protein, low-sodium diet. Restrict fluids as indicated.</p>	<p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Extracellular fluid shifts, sodium/water restriction, and renal function all affect serum sodium levels. Potassium deficit may occur with diuretic therapy. BUN may be increased as a result of renal dysfunction/failure. ABGs may reflect metabolic acidosis.</p> <p>In presence of decreased serum proteins (e.g., malnutrition), increasing level of serum proteins can enhance colloidal osmotic gradients and promote return of fluid to the vascular space. Restriction of sodium/water decreases extracellular fluid retention.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hypervolemia Management (NIC)</p> <p>Collaborative</p> <p>Administer diuretics: loop diuretic, e.g., furosemide (Lasix); thiazide diuretic, e.g., hydrochlorothiazide (Esidrix); or potassium-sparing diuretic, e.g., spironolactone (Aldactone).</p> <p>Replace potassium losses as indicated.</p> <p>Prepare for/assist with dialysis or ultrafiltration, if indicated.</p>	<p>To achieve excretion of excess fluid, either a single diuretic (e.g., thiazide) or a combination of agents (e.g., thiazide and spironolactone) may be selected. The combination can be particularly helpful when two drugs have different sites of action, allowing more effective control of fluid excess.</p> <p>Potassium deficit may occur, especially if patient is receiving potassium-wasting diuretic. This can cause lethal cardiac dysrhythmias if untreated.</p> <p>May be done to rapidly reduce fluid overload, especially in the presence of severe cardiac/renal failure.</p>

Hypovolemia (Extracellular Fluid Volume Deficit)

PREDISPOSING/CONTRIBUTING FACTORS

Excessive fluid losses: Vomiting, gastric suctioning, diarrhea, polyuria, diaphoresis, wounds or burns, intraoperative fluid loss, hemorrhage

Insufficient/decreased fluid intake, e.g., preoperative/postoperative NPO status

Systemic infections, fever

Intestinal obstruction or fistulas

Pancreatitis, peritonitis, cirrhosis/ascites; adrenal insufficiency

Kidney disease, diabetic ketoacidosis, hyperglycemic hyperosmotic nonketotic coma (HHNC), diabetes insipidus, syndrome of inappropriate antidiuretic hormone (SIADH)

Patient Assessment Database

ACTIVITY/REST

May report: Fatigue, generalized weakness

CIRCULATION

May exhibit: Hypotension, including postural changes
Pulse weak/thready; tachycardia
Neck veins flattened; CVP decreased

ELIMINATION

May report: Constipation or occasionally diarrhea, abdominal cramps

May exhibit: Urine volume decreased, dark/concentrated color; oliguria (severe fluid depletion)

FOOD/FLUID

May report: Thirst, anorexia, nausea/vomiting

May exhibit: Weight loss often exceeding 2%–8% of total body weight
Abdominal distension
Mucous membranes dry, furrows on tongue; decreased tearing and salivation
Skin dry with poor turgor; or pale, moist, clammy (shock)

NEUROSENSORY

May report: Tingling of the extremities, vertigo, syncope
May exhibit: Behavior change, apathy, restlessness, confusion

RESPIRATION

May exhibit: Tachypnea, rapid/shallow breathing

SAFETY

May exhibit: Temperature usually subnormal, although fever may occur

TEACHING/LEARNING

Refer to predisposing/contributing factors
Discharge plan **DRG projected mean length of inpatient stay: depends on underlying cause**
considerations: May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

Serum sodium: May be normal, high, or low.

Urine sodium: Usually decreased (less than 10 mEq/L when losses are from external causes; usually higher than 20 mEq/L if the cause is renal or adrenal).

CBC: Hb/Hct and RBC usually increased (hemoconcentration); decrease suggests hemorrhage. In presence of mild anemia, Hct may appear to be WNL.

Serum glucose: Normal or elevated.

Serum protein: Increased.

BUN and Cr: Increased BUN and normal Cr level resulting in a ratio greater than 20:1.

Urine specific gravity: Increased.

NURSING DIAGNOSIS: Fluid Volume, deficient

May be related to

Active fluid loss, e.g., hemorrhage, vomiting/gastric intubation, diarrhea, burns, wounds, fistulas
Regulatory failure, e.g., adrenal disease, recovery phase of ARF; diabetic ketoacidosis (DKA), HHNC; diabetes insipidus, systemic infections

Possibly evidenced by

Signs/symptoms noted in patient database

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Fluid Balance (NOC)

Maintain fluid volume at a functional level as evidenced by individually adequate urinary output with normal specific gravity, stable vital signs, moist mucous membranes, good skin turgor, and prompt capillary refill.

Knowledge: Treatment Regimen (NOC)

Verbalize understanding of causative factors and purpose of therapeutic interventions.
Demonstrate behaviors to monitor and correct deficit as appropriate.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hypovolemia Management (NIC)</p> <p>Independent</p> <p>Monitor vital signs and CVP. Note presence/degree of postural BP changes. Observe for temperature elevations/fever.</p> <p>Palpate peripheral pulses; note capillary refill, skin color/temperature. Assess mentation.</p> <p>Monitor urinary output. Measure/estimate fluid losses from all sources, e.g., gastric losses, wound drainage, diaphoresis.</p> <p>Weigh daily and compare with 24-hr fluid balance. Mark/measure edematous areas, e.g., abdomen, limbs.</p> <p>Evaluate patient's ability to swallow.</p> <p>Ascertain patient's beverage preferences, and set up a 24-hr schedule for fluid intake. Encourage foods with high fluid content.</p> <p>Turn frequently, massage skin, and protect bony prominences.</p> <p>Provide skin and mouth care. Bathe every other day using mild soap. Apply lotion as indicated.</p> <p>Provide safety precautions as indicated, e.g., use of side rails, bed in low position, frequent observation, soft restraints (if required).</p>	<p>Tachycardia is present along with a varying degree of hypotension, depending on degree of fluid deficit. CVP measurements are useful in determining degree of fluid deficit and response to replacement therapy. Fever increases metabolism and exacerbates fluid loss.</p> <p>Conditions that contribute to extracellular fluid deficit can result in inadequate organ perfusion to all areas and may cause circulatory collapse/shock.</p> <p>Fluid replacement needs are based on correction of current deficits and ongoing losses. <i>Note:</i> A diaphoretic episode requiring a full linen change may represent a fluid loss of as much as 1 L. Decreased urinary output may indicate insufficient renal perfusion/hypovolemia, or polyuria can be present, requiring more aggressive fluid replacement.</p> <p>Although weight gain and fluid intake higher than output may not accurately reflect intravascular volume, e.g., third-space fluid accumulation cannot be used by the body for tissue perfusion, these measurements provide useful data for comparison.</p> <p>Impaired gag/swallow reflexes, anorexia/nausea, oral discomfort, and changes in level of consciousness/cognition are among the factors that affect patient's ability to replace fluids orally.</p> <p>Relieves thirst and discomfort of dry mucous membranes and augments parenteral replacement.</p> <p>Tissues are susceptible to breakdown because of vasoconstriction and increased cellular fragility.</p> <p>Skin and mucous membranes are dry, with decreased elasticity, because of vasoconstriction and reduced intracellular water. Daily bathing may increase dryness.</p> <p>Decreased cerebral perfusion frequently results in changes in mentation/altered thought processes, requiring protective measures to prevent patient injury. <i>Note:</i> Application of restraints can increase agitation, requiring alternative interventions (e.g., one-on-one monitoring, sedation). Use of side rails may place the elderly confused patient at risk for entrapment injury/death.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Hypovolemia Management (NIC)</p> <p>Independent</p> <p>Investigate reports of sudden/sharp chest pain, dyspnea, cyanosis, increased anxiety, restlessness.</p> <p>Monitor for sudden/marked elevation of BP, restlessness, moist cough, dyspnea, basilar crackles, frothy sputum.</p> <p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory studies as indicated, e.g., electrolytes, glucose, pH/Pco₂, coagulation studies.</p> <p>Administer IV solutions as indicated:</p> <p>Isotonic solutions, e.g., 0.9% NaCl (normal saline), 5% dextrose/water;</p> <p>0.45% NaCl (half-normal saline), lactated Ringer's (LR) solution;</p> <p>Colloids, e.g., dextran, Plasmanate/albumin, hetastarch (Hespan);</p> <p>Whole blood/packed RBC transfusion, or autologous collection of blood.</p> <p>Administer sodium bicarbonate, if indicated.</p> <p>Provide tube feedings, including free water as appropriate.</p>	<p>RATIONALE</p> <p>Hemoconcentration (sludging) and increased platelet aggregation may result in systemic emboli formation.</p> <p>Too rapid a correction of fluid deficit may compromise the cardiopulmonary system, especially if colloids are used in general fluid replacement (increased osmotic pressure potentiates fluid shifts).</p> <p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Depending on the avenue of fluid loss, differing electrolyte/metabolic imbalances may be present/require correction; e.g., use of glucose solutions in patients with underlying glucose intolerance may result in serum glucose elevation and increased urinary water losses.</p> <p><i>Note:</i> Hypodermoclysis (rehydration via subcutaneous infusion) may be indicated to correct fluid deficits without the stress of IV therapy/enteral tubes or hospitalization, especially for elderly patients.</p> <p>Crystalloids provide prompt circulatory improvement, although the benefit may be transient (increased renal clearance).</p> <p>As soon as patient is normotensive, a hypotonic solution (0.45% NaCl) may be used to provide both electrolytes and free water for renal excretion of metabolic wastes. <i>Note:</i> Buffered crystalloids (LR) are used with caution because they may potentiate the risk of metabolic acidosis.</p> <p>Corrects plasma protein concentration deficits, thereby increasing intravascular osmotic pressure and facilitating return of fluid into vascular compartment.</p> <p>Indicated when hypovolemia is related to active blood loss.</p> <p>May be given to correct severe acidosis while correcting fluid balance.</p> <p>Enteral replacement can provide proteins and other needed elements in addition to meeting general fluid requirements when swallowing is impaired.</p>

SODIUM

Sodium (Na) is the major cation of extracellular fluid and is primarily responsible for osmotic pressure in that compartment. Sodium enhances neuromuscular conduction/transmission of impulses and is essential for maintaining acid-base balance. Normal serum range is 135–145 mEq/L; intracellular, 10 mEq/L. Chloride is carried by Na and will display the same imbalances. Normal serum chloride range is 95–105 mEq/L.

Hyponatremia (Sodium Deficit)

Predisposing/contributing factors

Primary hyponatremia (loss of sodium): Lack of sufficient dietary sodium, severe malnutrition, infusion of sodium-free solutions; excessive sodium loss through heavy sweating (e.g., heat exhaustion), wounds/trauma (hemorrhage), burns, gastric suctioning, vomiting, diarrhea, small-bowel obstruction, peritonitis, salt-wasting renal dysfunction, adrenal insufficiency (Addison's disease)

Dilutional hyponatremia (water gains): Excessive water intake, electrolyte-free IV infusion, water intoxication (IV therapy, tap-water enemas), gastric irrigations with electrolyte-free solutions, presence of tumors or CNS disorders predisposing to SIADH, HF, renal failure/nephrotic syndrome, hepatic cirrhosis, DM (hyperglycemia), freshwater near-drowning; use of certain drugs, e.g., hypoglycemia medications, barbiturates, antipsychotics, aminophylline, morphine (may stimulate pituitary gland to secrete excessive amounts of ADH), anticonvulsants, some antineoplastic agents, or NSAIDs *Note:* A pseudohyponatremia may occur in presence of multiple myeloma, hyperlipidemia, or hypoproteinemia but does not reflect an actual abnormality of water metabolism.

Patient Assessment Database

(Patient may be asymptomatic until serum sodium level is less than 125 mEq/L, depending on rapidity of onset.)

General

ACTIVITY/REST

May report: Malaise
Generalized weakness, faintness, muscle cramps

EGO INTEGRITY

May report: Anxiety
May exhibit: Restlessness, apprehension

FOOD/FLUID

May report: Nausea, anorexia, thirst
Low-sodium diet
Diuretic use

NEUROSENSORY

May report: Headache, blurred vision, vertigo
May exhibit: Loss of coordination, stupor, personality changes

TEACHING/LEARNING

Refer to predisposing/contributing factors
Use of oral hypoglycemic agent, potent diuretics, NSAIDs, other drugs that impair renal water excretion

Discharge plan considerations: **DRG projected mean length of inpatient stay: depends on underlying cause**
May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

Sodium/Water Deficit

(Na less than 135 mEq/L; urine specific gravity elevated, serum osmolality normal)

CIRCULATION

May exhibit: Hypotension, tachycardia
Peripheral pulses diminished
Pallid, clammy skin

ELIMINATION

May report: Abdominal cramping, diarrhea
May exhibit: Urinary output decreased

FOOD/FLUID

May report: Anorexia, nausea/vomiting
May exhibit: Poor skin turgor; soft/sunken eyeballs
Mucous membranes dry, decreased saliva/perspiration

NEUROSENSORY

May report: Dizziness
May exhibit: Muscle twitching
Lethargy, restlessness, confusion, stupor

RESPIRATION

May exhibit: Tachypnea

SAFETY

May exhibit: Skin flushed, dry, hot
Fever

Sodium Deficit/Water Excess

(Na less than 135 mEq/L; urine specific gravity low; serum osmolality decreased)

CIRCULATION

May exhibit: Hypertension
Generalized edema

ELIMINATION

May exhibit: Urinary output increased

NEUROSENSORY

May exhibit: Muscle twitching, restlessness, changes in mentation (more severe when problem is acute/develops rapidly)

PAIN/DISCOMFORT

May report: Headache, abdominal cramps

Severe Sodium Deficit

(Na less than 120 mEq/L)

CIRCULATION

May exhibit: Hypotension with vasomotor collapse
Rapid thready pulse
Cold/clammy skin, fingerprinting on sternum; cyanosis

NEUROSENSORY

May exhibit: Hyporeflexia
Convulsions/coma

DIAGNOSTIC STUDIES (DEPEND ON ASSOCIATED FLUID LEVEL)

Serum sodium: Decreased, less than 135 mEq/L. However, signs/symptoms may not occur until level is less than 120 mEq/L.

Urine sodium: Less than 15 mEq/L indicates renal conservation of sodium because of sodium loss from a nonrenal source unless sodium-wasting nephropathy is present. Urine sodium higher than 20 mEq/L indicates SIADH.

Serum potassium: May be decreased as the kidneys attempt to conserve sodium at the expense of potassium.

Serum chloride/bicarbonate: Levels are decreased, depending on which ion is lost with the sodium.

Serum osmolality: Commonly low, but may be normal (pseudohyponatremia) or high (HHNC).

Urine osmolality: Usually less than 100 mOsm/L unless SIADH present, in which case it will exceed serum osmolality.

Urine specific gravity: May be decreased (less than 1.010) or increased (higher than 1.020) if SIADH is present.

Hct: Depends on fluid balance, e.g., fluid excess versus dehydration.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:
Electrolyte & Acid/Base Balance (NOC)
 Display heart rate, BP, and laboratory results within normal limits (WNL) for patient; absence of muscle weakness, neurological irritability.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyponatremia (NIC)</p> <p>Independent</p> <p>Identify patient at risk for hyponatremia and the specific cause, e.g., sodium loss or fluid excess.</p> <p>Monitor I&O. Calculate fluid balance. Weigh daily.</p> <p>Assess level of consciousness/neuromuscular response.</p> <p>Maintain quiet environment; provide safety/seizure precautions.</p> <p>Note respiratory rate and depth.</p> <p>Encourage foods and fluids high in sodium, e.g., milk, meat, eggs, carrots, beets, and celery. Use fruit juices and bouillon instead of plain water.</p>	<p>Provides clues for early intervention. Hyponatremia is a common imbalance (especially in the elderly) and may range from mild to severe. Severe hyponatremia can cause neurological damage or death if not treated promptly.</p> <p>Indicators of fluid balance are important, because either fluid excess or deficit may occur with hyponatremia.</p> <p>Sodium deficit may result in decreased mentation (to point of coma), as well as generalized muscle weakness/cramps, convulsions.</p> <p>Reduces CNS stimulation and risk of injury from neurological complications, e.g., seizures.</p> <p>Co-occurring hypochloremia may produce slow/shallow respirations as the body compensates for metabolic alkalosis.</p> <p>Unless sodium deficit causes serious symptoms requiring immediate IV replacement, patient may benefit from slower replacement by oral method or removal of previous salt restriction.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyponatremia (NIC)</p> <p>Independent</p> <p>Irrigate NG tube (when used) with normal saline instead of water.</p> <p>Observe for signs of circulatory overload as indicated.</p> <p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor serum and urine electrolytes, osmolality.</p> <p>Provide/restrict fluids depending on fluid volume status.</p> <p>Administer medications as indicated, e.g.:</p> <ul style="list-style-type: none"> Furosemide (Lasix); Sodium chloride; Potassium chloride; Demeclocycline (Declomycin); Captopril (Capoten). <p>Prepare for/assist with dialysis as indicated.</p>	<p>Isotonic irrigation will minimize loss of GI electrolytes.</p> <p>Administration of sodium-containing IV fluids in presence of HF increases risk.</p> <p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Evaluates therapy needs/effectiveness.</p> <p>In presence of hypovolemia, volume losses are replaced with isotonic saline (e.g., normal saline), or, on occasion, hypertonic solution (3% NaCl) when hyponatremia is life-threatening. In the presence of fluid volume excess, or SIADH, fluid restriction is indicated. <i>Note:</i> Too rapid/excessive administration of hypertonic solutions can be lethal.</p> <p>Effective in reducing fluid excess to correct sodium/water balance.</p> <p>Used to replace deficits/prevent recurrence in the presence of chronic/ongoing losses.</p> <p>Corrects potassium deficit, especially when diuretic is used.</p> <p>Useful in treating chronic SIADH, or when severe water restriction may not be tolerated, e.g., COPD. <i>Note:</i> May be contraindicated in patients with liver disease, because nephrotoxicity may occur.</p> <p>May be used in combination with a loop diuretic (e.g., Lasix) to correct fluid volume excess, especially in the presence of HF.</p> <p>May be done to restore sodium balance without increasing fluid level when hyponatremia is severe or response to diuretic therapy is inadequate.</p>

Hypernatremia (Sodium Excess)

Predisposing/contributing factors

Excessive water losses: Polyuria (as may occur with diabetes insipidus); use of osmotic diuretics (such as mannitol); presence of fever, profuse sweating, vomiting, diarrhea; extracellular fluid volume excesses: e.g., renal disease, HF, primary aldosteronism, excessive steroids/Cushing's disease; excessive ingestion or infusion of sodium; salt-water near-drowning

Insufficient water intake: Administration of tube feedings/high-protein diets with minimal fluid intake, self-medication/ "ulcer diets" primarily using half-and-half/whole milk

Patient Assessment Database

Sodium Excess/Water Deficit

(Na higher than 145 mEq/L; elevated urine specific gravity)

ACTIVITY/REST

May report: Weakness
May exhibit: Muscle rigidity/tremors, generalized weakness

CIRCULATION

May exhibit: Decreased blood pressure, postural hypotension
Tachycardia

ELIMINATION

May exhibit: Decreased urinary output

FOOD/FLUID

May report: Thirst
May exhibit: Mucous membranes dry, sticky; tongue dry, swollen, rough

NEUROSENSORY

May exhibit: Irritability, lethargy/coma (depending on rapidity of onset rather than actual serum sodium level)
Delusions, hallucinations
Muscle irritability, seizure activity

SAFETY

May exhibit: Hot, dry, flushed skin
Fever

Sodium/Water Excess

(Na higher than 145 mEq/L; urine specific gravity decreased)

CIRCULATION

May exhibit: Elevated BP, hypertension

ELIMINATION

May exhibit: Polyuria

FOOD/FLUID

May report: Thirst
May exhibit: Skin pale, moist, taut with pitting edema
Weight gain

NEUROSENSORY

May exhibit: Confusion, lethargy
Delusions, hallucinations

RESPIRATION

May exhibit: Dyspnea

Sodium Excess/Water Deficit or Excess

TEACHING/LEARNING

Discharge plan considerations: Refer to predisposing/contributing factors
DRG projected mean length of inpatient stay: depends on underlying cause
 May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

Serum sodium: Increased, higher than 145 mEq/L. Serum levels higher than 160 mEq/L may be accompanied by severe neurological signs.
Serum chloride: Increased, higher than 106 mEq/L.
Serum potassium: Decreased.
Serum osmolality: Higher than 295 mOsm/L when dehydrated; lower in presence of extracellular fluid excess, and less than 200 mOsm/L with excessive polyuria.
Hct: May be normal or elevated depending on fluid status.
Urine sodium: Less than 50 mEq/L.
Urine chloride: Less than 50 mEq/L.
Urine osmolality: Higher than 800 mOsm/L.
Urine specific gravity: Increased, higher than 1.015, if water deficit present; or less than 1.010 when hyponatremia is due to polyuria.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:
Electrolyte & Acid/Base Balance (NOC)
 Display BP, heart rate, and laboratory results WNL for patient; absence of neuromuscular irritability, cognitive impairment.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyponatremia (NIC)</p> <p>Independent</p> <p>Monitor BP.</p> <p>Identify patient at risk for hyponatremia and likely cause, e.g., water deficit, sodium excess.</p> <p>Note respiratory rate, depth.</p> <p>Monitor I&O, urine specific gravity. Weigh daily. Assess presence/location of edema.</p>	<p>Either hypertension or hypotension may be present, depending on the fluid status. Presence of postural hypotension may affect activity tolerance.</p> <p>Early identification and intervention prevents serious complications associated with this problem.</p> <p>Deep, labored respirations with air hunger suggest metabolic acidosis (hyperchloremia), which can lead to cardiopulmonary arrest if not corrected.</p> <p>These parameters are variable, depending on fluid status, and are indicators of therapy needs/effectiveness.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyponatremia (NIC)</p> <p>Independent</p> <p>Evaluate level of consciousness and muscular strength, tone, movement.</p> <p>Maintain safety/seizure precautions, as indicated, e.g., bed in low position, use of padded side rails.</p> <p>Assess skin turgor, color, temperature, and mucous membrane moisture.</p> <p>Provide/encourage meticulous skin care and frequent repositioning.</p> <p>Provide frequent oral care. Avoid use of mouthwash/rinse that contains alcohol.</p> <p>Offer debilitated patient fluids at regular intervals. Give free water to patient receiving enteral feedings.</p> <p>Recommend avoidance of foods high in sodium, e.g., canned soups/vegetables, processed foods, snack foods, and condiments.</p>	<p>Sodium imbalance may cause changes that vary from confusion and irritability to seizures and coma. In presence of water deficit, rapid rehydration may cause cerebral edema.</p> <p>Sodium excess/cerebral edema increases risk of convulsions.</p> <p>Water-deficit hypernatremia manifests by signs of dehydration.</p> <p>Maintains skin integrity.</p> <p>Promotes comfort and prevents further drying of mucous membranes.</p> <p>May prevent hyponatremia in patient who is unable to perceive or respond to thirst.</p> <p>Reduces risk of sodium-associated complications.</p>
<p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor serum electrolytes, osmolality, and ABGs as indicated.</p> <p>Increase PO/IV fluid intake, e.g., 5% dextrose/water in presence of dehydration; 0.9% NaCl if extracellular deficit is present.</p> <p>Restrict sodium intake and administer diuretics as indicated.</p>	<p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Evaluates therapy needs/effectiveness. <i>Note:</i> Co-occurring hyperchloremia may cause metabolic acidosis, requiring buffering, e.g., sodium bicarbonate.</p> <p>Replacement of total body water deficit will gradually restore sodium/water balance. <i>Note:</i> Rapid reduction of serum sodium level with corresponding decrease in serum osmolality can cause cerebral edema/convulsions.</p> <p>Restriction of sodium intake while promoting renal clearance lowers serum sodium levels in the presence of extracellular fluid excess.</p>

POTASSIUM

Potassium is the major cation of the intracellular fluid and is responsible for maintaining intracellular osmotic pressure. Potassium also regulates neuromuscular excitability, aids in maintenance of acid-base balance, synthesis of protein, and metabolism of carbohydrates. Normal serum range is 3.5–5.0 mEq/L (body total of 42 mEq/L).

Hypokalemia (Potassium Deficit)

PREDISPOSING/CONTRIBUTING FACTORS

Renal loss: Use of potassium-wasting diuretics, diuretic phase of acute tubular necrosis (ATN), healing phase of burns; diabetic acidosis; Cushing's syndrome; nephritis, hypomagnesemia; use of sodium penicillins, amphotericin B, carbenicillin steroids; licorice abuse

GI loss: Profuse vomiting, excessive diarrhea, laxative abuse, prolonged gastric suction, inflammatory bowel disease, fistulas

Inadequate dietary intake: Anorexia nervosa, starvation, high-sodium diet

Shift into cells: TPN, alkalosis, or excessive secretion or administration of insulin

Other: Sweat losses (heavily perspiring person acclimated to heat); liver disease

Patient Assessment Database

ACTIVITY/REST

May report: Generalized weakness, lethargy, fatigue

CIRCULATION

May exhibit: Hypotension
Pulses weak/diminished, irregular
Heart sounds distant
Dysrhythmias, e.g., premature ventricular contractions (PVCs), ventricular tachycardia/fibrillation

ELIMINATION

May exhibit: Nocturia, polyuria if factors contributing to hypokalemia include HF or DM
Bowel sounds diminished, decreased bowel motility, paralytic ileus
Abdominal distension

FOOD/FLUID

May report: Anorexia, nausea/vomiting
Thirst

NEUROSENSORY

May report: Paresthesias
May exhibit: Depressed mental state/confusion, apathy, drowsiness, irritability, coma
Hyporeflexia, tetany, paralysis (flaccid quadriplegia)

PAIN/DISCOMFORT

May report: Muscle pain/cramps

RESPIRATION

May exhibit: Hypoventilation/decreased respiratory depth due to muscle weakness/paralysis of diaphragm; apnea, cyanosis

TEACHING/LEARNING

Refer to predisposing/contributing factors
Discharge plan considerations: **DRG projected mean length of inpatient stay: depends on underlying cause**
May require assistance with changes in therapeutic regimen, dietary management

Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

- Serum potassium:*** Decreased, less than 3.5 mEq/L.
- Serum chloride:*** Often decreased, less than 98 mEq/L.
- Serum glucose:*** May be slightly elevated.
- Serum magnesium:*** Levels often decreased when potassium deficit is present.
- Plasma bicarbonate:*** Increased, higher than 29 mEq/L.
- Urine osmolality:*** Decreased.
- ABGs:*** pH and bicarbonate may be elevated (metabolic alkalosis).
- ECG:*** Low voltage; flat or inverted T wave, appearance of U wave, depressed ST segment, peaked P waves; prolonged QT interval, ventricular dysrhythmias.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:
Electrolyte & Acid/Base Balance (NOC)
 Display heart rhythm and laboratory results WNL for patient; absence of muscle weakness, paresthesias, cognitive impairment.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypokalemia (NIC)</p> <p>Independent</p> <p>Monitor heart rate/rhythm.</p> <p>Monitor respiratory rate, depth, effort. Encourage cough/deep-breathing exercises; reposition frequently.</p> <p>Assess level of consciousness and neuromuscular function, e.g., strength, sensation, movement.</p> <p>Auscultate bowel sounds, noting decrease/absence or change.</p> <p>Maintain accurate record of urinary, gastric, and wound losses.</p> <p>Monitor rate of IV potassium administration using microdrop or pump infusion devices. Check for side effects. Provide ice pack as indicated.</p>	<p>Changes associated with hypokalemia include abnormalities in both conduction and contractility. Tachycardia may develop, and potentially life-threatening atrial and ventricular dysrhythmias, e.g., PVCs, sinus bradycardia, atrioventricular (AV) blocks, AV dissociation, ventricular tachycardia.</p> <p>Respiratory muscle weakness may proceed to paralysis and eventual respiratory arrest.</p> <p>Apathy, drowsiness, irritability, tetany, paresthesias, and coma may occur.</p> <p>Paralytic ileus commonly follows gastric losses through vomiting/gastric suction, protracted diarrhea.</p> <p>Guide for calculating fluid/potassium replacement needs.</p> <p>Ensures controlled delivery of medication to prevent bolus effect and reduce associated discomfort, e.g., burning sensation at IV site. When solution cannot be administered via central vein and slowing rate is not possible/effective, ice pack to infusion site may help relieve discomfort.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypokalemia (NIC)</p> <p>Independent</p> <p>Encourage intake of foods and fluids high in potassium, e.g., bananas, oranges, dried fruits, red meat, turkey, salmon, leafy vegetables, peas, baked potatoes, tomatoes, winter squash, coffee, colas, tea. Discuss use of potassium chloride salt substitutes for patient receiving long-term diuretics.</p> <p>Review drug regimen for potassium-wasting drugs, e.g., furosemide (Lasix), hydrochlorothiazide (Esidrix), Acetazolamide (Diamox), IV catecholamines, gentamicin (Garamycin), carbenicillin (Geocillin), amphotericin B (Fungizone).</p> <p>Discuss preventable causes of condition, e.g., nutritional choices, proper use of laxatives.</p> <p>Dilute liquid and effervescent potassium supplements (K-Tab, K-Lyte/Cl) with 4 oz water/juice and give after meals.</p> <p>Watch for signs of digitalis intoxication when used (e.g., reports of nausea/vomiting, blurred vision, increasing atrial dysrhythmias, and heart block).</p> <p>Observe for signs of metabolic alkalosis, e.g., hypoventilation, tachycardia, dysrhythmias, tetany, changes in mentation.</p> <p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory studies, e.g.: Serum potassium;</p> <p>ABGs;</p>	<p>Potassium may be replaced/level maintained through the diet when patient is allowed oral food and fluids. Dietary replacement of 40–60 mEq/L/day is typically sufficient if no abnormal losses are occurring.</p> <p>If alternative agents, e.g., potassium-sparing diuretics such as spironolactone (Aldactone), triamterene (Dyrenium), amiloride (Midamor), cannot be administered or when high-dose sodium drugs are administered (e.g., carbenicillin), close monitoring and replacement of potassium are necessary.</p> <p>Provides opportunity for patient to prevent recurrence. Also, dietary control is more palatable than oral replacement medications.</p> <p>May prevent/reduce GI irritation and saline laxative effect.</p> <p>Low potassium enhances effect of digitalis, slowing cardiac conduction. <i>Note:</i> Combined effects of digitalis, diuretics, and hypokalemia may produce lethal dysrhythmias.</p> <p>Frequently associated with hypokalemia and requiring additional correction.</p> <p>Refer to listing of predisposing/contributing factors to determine treatment needs. <i>Note:</i> Hypokalemia is life-threatening, early detection is crucial.</p> <p>Levels should be checked frequently during replacement therapy, especially in the presence of insufficient renal function. Sudden excess/elevation may cause cardiac dysrhythmias.</p> <p>Correction of metabolic alkalosis raises serum potassium level and reduces replacement needs. Correction of acidosis drives potassium back into cells, resulting in decreased serum levels and increased replacement needs.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypokalemia (NIC)</p> <p>Collaborative</p> <p>Serum magnesium;</p> <p>Serum chloride.</p> <p>Administer oral and/or IV potassium.</p>	<p>Hypomagnesemia occurs with and exacerbates potassium loss and sodium retention, altering cell membrane excitability (affects cardiac and neuromuscular function).</p> <p>Use of diuretics, e.g., furosemide (Lasix), hydrochlorothiazide (HydroDIURIL), may cause chloride and potassium depletion.</p> <p>May be required to correct deficiencies when changes in medication, therapy, and/or dietary intake are insufficient. <i>Note:</i> Even in severe deficit, parenteral replacement should not exceed 40 mEq/2 hr. Dietary supplementation may also be used to produce a gradual equilibration if patient is able to take oral food and fluids.</p>

Hyperkalemia (Potassium Excess)

PREDISPOSING/CONTRIBUTING FACTORS

Potassium retention: Decreased renal excretion (e.g., renal disease/acute failure, hypoaldosteronism, Addison's disease), hypovolemia, use of potassium-conserving diuretics, especially when associated with potassium supplements, use of NSAIDs

Excessive potassium intake: Salt substitutes, nutrition supplementation shakes, drugs containing potassium (e.g., penicillin), improper use of oral potassium supplements, too-rapid IV administration of potassium, massive transfusion of banked blood

Shift or release of potassium out of cells: Severe catabolism, burns, crush injuries, myocardial infarction (MI), severe hemolysis, rhabdomyolysis, chemotherapy with cytotoxic drugs, respiratory or metabolic acidosis, anoxia, hyperglycemia with insulin deficiency, use of some [beta]-adrenergic blockers, profound digitalis toxicity

Other: Use of certain medications such as captopril, heparin, cyclosporine

Patient Assessment Database

Data depend on degree of elevation and length of time condition has existed.

ACTIVITY/REST

May report: Vague muscular weakness

May exhibit: Restlessness, irritability

CIRCULATION

May exhibit: Irregular pulse, bradycardia, heart block, asystole

EGO INTEGRITY

May report: Apprehension

ELIMINATION

May report: Intermittent abdominal cramps, diarrhea

May exhibit: Urine volume decreased
Hyperactive bowel sounds

FOOD/FLUID

May report: Nausea/vomiting

NEUROSENSORY

May report: Paresthesias (often of face, tongue, hands, feet)
Slurred speech

May exhibit: Decreased deep-tendon reflexes; progressive, ascending flaccid paralysis; twitching, seizure activity
Apathy, confusion

PAIN/DISCOMFORT

May report: Muscle cramps/pain

TEACHING/LEARNING

Discharge plan considerations: Refer to predisposing/contributing factors
DRG projected mean length of inpatient stay: depends on underlying cause
May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

Serum potassium: Increased, higher than 5.1 mEq/L.

Serum magnesium: Levels may be elevated if renal failure is present.

Renal function studies: May be altered, indicating failure.

Leukocyte or thrombocyte count: Elevation may cause a pseudohyperkalemia, affecting choice of interventions.

ECG changes: T waves tall and peaked/tented, prolonged PR interval, loss of P waves, widening of QRS complex, shortened QT interval, and ST segment depression; atrial/ventricular dysrhythmias, e.g., bradycardia, atrial arrest, complete heart block, ventricular fibrillation, cardiac arrest.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:
Electrolyte & Acid/Base Balance (NOC)
Display heart rate/rhythm and laboratory results WNL for patient; absence of muscle weakness, paresthesias, cognitive impairment.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyperkalemia (NIC)</p> <p>Collaborative</p> <p>Identify patient at risk; or the cause of the hyperkalemia, e.g., excessive intake of potassium or decreased excretion.</p> <p>Instruct patient in use of potassium-containing salts (salt substitutes), taking potassium supplements safely.</p>	<p>Influences choice of interventions. Early identification and treatment can prevent complication. <i>Note:</i> A major cause of hyperkalemia is decreased renal excretion.</p> <p>Patient is often able to prevent hyperkalemia through management of supplements, diet, and other medications.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyperkalemia (NIC)</p> <p>Collaborative</p> <p>Monitor respiratory rate and depth. Elevate head of bed. Encourage cough/deep-breathing exercises.</p> <p>Monitor heart rate/rhythm. Be aware that cardiac arrest can occur.</p> <p>Monitor urinary output.</p> <p>Assess level of consciousness, neuromuscular function, e.g., movement, strength, sensation.</p> <p>Encourage/assist with ROM exercises as tolerated.</p> <p>Encourage frequent rest periods; assist with care activities, as indicated.</p> <p>Review drug regimen for medications containing/affecting potassium excretion, e.g., penicillin G, spironolactone (Aldactone), amiloride (Midamor), hydrochlorothiazide (Dyazide, Maxzide).</p> <p>Identify/discontinue dietary sources of potassium, e.g., tomatoes, broccoli, orange juice, bananas, bran, chocolate, coffee, tea, eggs, dairy products, dried fruits.</p> <p>Recommend an increase in carbohydrates/fats and foods low in potassium, e.g., canned fruits, refined cereals, apple/cranberry juice.</p> <p>Stress importance of patient's notifying future caregivers when chronic condition potentiates development of hyperkalemia, e.g., oliguric renal failure.</p>	<p>Patients may hypoventilate and retain CO₂, leading to respiratory acidosis. Muscular weakness can affect respiratory muscles and lead to complications of respiratory infection/failure.</p> <p>Excess potassium depresses myocardial conduction. Bradycardia can progress to cardiac fibrillation/arrest.</p> <p>In kidney failure, potassium is retained because of improper excretion. Potassium should not be given if oliguria or anuria is present.</p> <p>Patient is usually awake and alert; however, muscular paresthesia, weakness, and flaccid paralysis may occur.</p> <p>Improves muscular tone and reduces muscle cramps and pain.</p> <p>General muscle weakness decreases activity tolerance.</p> <p>Requires regular monitoring of potassium levels, and may require alternative drug choices or changes in dosage/frequency.</p> <p>Facilitates reduction of potassium level and may prevent recurrence of hyperkalemia.</p> <p>Reduces exogenous sources of potassium and prevents catabolic tissue breakdown with release of cellular potassium.</p> <p>May help prevent recurrence.</p>
<p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory results, e.g., serum potassium, ABGs, BUN/Cr, glucose as indicated.</p>	<p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Evaluates therapy needs/effectiveness. <i>Note:</i> Hypoventilation may result in respiratory acidosis, thereby increasing serum potassium levels.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hyperkalemia (NIC)</p> <p>Collaborative</p> <p>Administer medications as indicated, e.g.:</p> <ul style="list-style-type: none"> Diuretics, e.g., furosemide (Lasix); IV glucose with insulin, sodium bicarbonate; Calcium gluconate; Sodium polystyrene sulfonate (Kayexalate, SPS suspension), orally, per NG tube, or rectally; [beta]-adrenergic agonist, e.g., albuterol (Proventil). Infuse potassium-based medication/solutions slowly. Provide fresh blood or washed RBCs (when possible) if transfusions required. Prepare for/assist with dialysis (peritoneal or hemodialysis). 	<p>RATIONALE</p> <p>Loop or thiazide diuretics promote renal clearance and excretion of potassium.</p> <p>Short-term emergency measure to move potassium into the cell, thus reducing toxic serum level. <i>Note:</i> Use with caution in presence of HF or hyponatremia. Use of glucose is contraindicated in patients who are hyperkalemic.</p> <p>Temporary stopgap measure that antagonizes toxic potassium depressant effects on heart and stimulates cardiac contractility. <i>Note:</i> Calcium is contraindicated in patients on digitalis because it increases the cardiotoxic effects of the drug and may cause dysrhythmias.</p> <p>Resin removes potassium by exchanging potassium for sodium or calcium in the GI tract. Sorbitol enhances evacuation. <i>Note:</i> Use cautiously in patients with HF, edema, and in the elderly because it increases sodium level. In addition, Kayexalate may cause hyperchloremia.</p> <p>Nebulizer administration has been effective in patients receiving hemodialysis, and may also attenuate the hypoglycemic effect of insulin administration.</p> <p>Prevents administration of concentrated bolus, allows time for kidneys to clear excess free potassium.</p> <p>Fresh blood has less potassium than banked blood, because breakdown of older RBCs releases potassium.</p> <p>May be required when more conservative methods fail or are contraindicated, e.g., severe HF.</p>

CALCIUM

Calcium is involved in bone formation/reabsorption, neural transmission/muscle contraction, regulation of enzyme systems, and is a coenzyme in blood coagulation. Normal serum levels are 4.5–5.3 mEq/L, 8.5–10.5 mg/dL (total) or 2.1–2.6 mEq/L (ionized). The ionized calcium is physiologically active and clinically important, especially in critically ill patients. The total serum calcium is directly related to the serum albumin, follows it, and must be considered if only total serum readings are available. Some factors that alter the percentage of ionized calcium are changes in pH (affects how much calcium is bound to protein) or increased serum levels of fatty acids, lactate, and bicarbonate.

Hypocalcemia (Calcium Deficit)

PREDISPOSING/CONTRIBUTING FACTORS

Primary or surgical hypoparathyroidism; transient hypocalcemia following thyroidectomy; hyperphosphatemia, hypomagnesemia
Massive subcutaneous tissue infections, acute pancreatitis, burns, peritonitis, malignancies
Excessive GI losses: Draining fistula, diarrhea, fat malabsorption syndromes, chronic laxative use (particularly phosphate-containing laxatives/enemas)
Extreme stress situations with mobilization and excretion of calcium
Diuretic and terminal phase of renal failure
Inadequate dietary intake, lack of milk/vitamin D, excessive protein diet
Alcoholism: Primary effect of ethanol, plus intestinal malabsorption, hypomagnesemia, hypoalbuminemia, and pancreatitis
Use of anticonvulsants, antibiotics, corticosteroids; loop diuretics, drugs that lower serum magnesium (e.g., cisplatin, gentamicin)
Infusion of citrated blood, calcium-free infusions; rapid infusion of Plasmanate
Malignant neoplasms with bone metastases
Alkalotic states
Decreased ultraviolet exposure

Patient Assessment Database

Data depend on duration, severity, and rate of onset of hypocalcemia.

CIRCULATION

May exhibit: Hypotension
Pulses weak/decreased, irregular (weak cardiac contraction/premature dysrhythmias)

ELIMINATION

May report: Diarrhea, abdominal pain
May exhibit: Abdominal distension (paralytic ileus)

FOOD/FLUID

May report: Nausea/vomiting
May exhibit: Difficulty swallowing

HYGIENE

May exhibit: Coarse, dry skin; alopecia (chronic)

NEUROSENSORY

May report: Circumoral paresthesia, numbness and tingling of fingers and toes; muscle cramps
May exhibit: Anxiety, confusion, irritability, alteration in mood, impaired memory, depression, hallucinations, psychoses
Muscle spasms (carpopedal and laryngeal), increased deep-tendon reflexes; tetany, tonic/clonic seizure activity, positive Trousseau's and Chvostek's signs

RESPIRATION

May exhibit: Labored shallow breathing; stridor (spasm of laryngeal muscles)

SAFETY

May exhibit: Bleeding with no or minimal trauma

TEACHING/LEARNING

Discharge plan considerations: Refer to predisposing/contributing factors
DRG projected mean length of inpatient stay: depends on underlying cause
May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

Serum calcium: Decreased, less than 4.5 mEq/L or 8.5 mg/dL (total), 2.1 mEq/L (ionized)

Urine Sulkowitch test: Shows light or no precipitate.

ECG: Prolonged QT interval (characteristic but not necessarily diagnostic). In severe deficiency, T waves may flatten or invert, giving appearance of hypokalemia or myocardial ischemia; ventricular tachycardia may develop.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Electrolyte & Acid/Base Balance (NOC)

Display heart rhythm and laboratory results WNL for patient; absence of neuromuscular irritability, respiratory impairment.

ACTIONS/INTERVENTIONS	RATIONALE
Electrolyte Management: Hypercalcemia (NIC) Independent Monitor heart rate/rhythm.	Calcium deficit along with associated hypomagnesemia weakens cardiac muscle/contractility.
Assess respiratory rate, rhythm, effort. Have tracheostomy equipment available.	Laryngeal stridor may develop and result in respiratory emergency/arrest.
Observe for neuromuscular irritability, e.g., tetany, seizure activity. Assess for presence of Chvostek's/Trousseau's signs.	Calcium deficit causes repetitive and uncontrolled nerve transmission, leading to muscle spasms and hyperirritability.
Provide quiet environment and seizure precautions as appropriate.	Reduces CNS stimulation and protects patient from potential injury.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypercalcemia (NIC)</p> <p>Independent</p> <p>Encourage relaxation/stress reduction techniques, e.g., deep-breathing exercises, guided imagery, visualization.</p> <p>Check for bleeding from any source (mucous membranes, puncture sites, wounds/incisions, and so on). Note presence of ecchymosis, petechiae.</p> <p>Review patient's drug regimen, e.g., use of insulin, plicamycin (Mithracin), parathyroid injection, digitalis.</p> <p>Discuss use of laxatives/antacids.</p> <p>Review dietary intake of vitamins and fat.</p> <p>Identify sources to increase calcium and vitamin D in diet, e.g., dairy products, beans, cauliflower, eggs, oranges, pineapples, sardines, shellfish. Restrict intake of phosphorus, e.g., barley, bran, whole wheat, rye, liver, nuts, chocolate.</p> <p>Encourage use of calcium-containing antacids if needed (e.g., Titalac, Tums).</p> <p>Stress importance of meeting calcium needs.</p>	<p>Tetany can be potentiated by hyperventilation and stress. <i>Note:</i> Direct pressure on the nerves (e.g., tightening BP cuff) may also cause tetany.</p> <p>Alterations in coagulation can occur as a result of calcium deficiency.</p> <p>Some drugs can lower magnesium levels, affecting calcium level. The effect of digitalis is enhanced by calcium, and, in patients receiving calcium, digitalis intoxication may develop.</p> <p>Those containing phosphate may negatively affect calcium metabolism.</p> <p>Insufficient ingestion of vitamin D and fat impairs absorption of calcium.</p> <p>Vitamin D aids in absorption of calcium from intestinal tract. Phosphorus competes with calcium for intestinal absorption.</p> <p>Possible sources for oral replacement to help maintain calcium levels, especially in patients at risk for osteoporosis.</p> <p>Adverse effects of long-term deficiency include tooth decay, eczema, cataracts, and osteoporosis.</p>
<p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory studies, e.g.:</p> <ul style="list-style-type: none"> Serum calcium and magnesium; serum albumin, ABGs; <p>PT, platelets.</p>	<p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Evaluates therapy needs/effectiveness. <i>Note:</i> Low serum albumin levels or serum pH affects calcium levels, e.g., a low albumin level causes a deceptively low calcium level; alkalosis causes surplus bicarbonate to bind with free calcium, impairing function; acidosis frees calcium, potentiating hypercalcemia.</p> <p>Calcium is an essential part of the clotting mechanism and deficit may lead to excessive bleeding.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypercalcemia (NIC)</p> <p>Collaborative</p> <p>Administer the following:</p> <p> Calcium gluconate/gluceptate/chloride IV;</p> <p> Oral preparations, e.g., calcium lactate/carbonate;</p> <p> Magnesium sulfate IV/PO if indicated;</p> <p> Vitamin D supplement (e.g., calcitriol).</p>	<p>Provides rapid treatment in acute calcium deficit (especially in presence of tetany/convulsions). <i>Note:</i> Calcium chloride is not used as often because it is irritating to the vein and can cause tissue sloughing if it leaks into tissues.</p> <p>Oral preparations are useful in correcting subacute deficiencies.</p> <p>Hypomagnesemia is a precipitating factor in calcium deficit.</p> <p>May be used in combination with calcium therapy to enhance calcium absorption once concomitant phosphate deficiency is corrected.</p>

Hypercalcemia (Calcium Excess)

PREDISPOSING/CONTRIBUTING FACTORS

Hyperparathyroidism, hyperthyroidism, multiple myeloma/other malignancies (e.g., cancer of breast, lung); renal disease, skeletal muscle paralysis, parathyroid tumor, sarcoidosis, adrenal insufficiency, TB
Excessive/prolonged use of vitamins A and D and calcium-containing antacids; prolonged use of thiazide diuretics, theophylline, lithium
Multiple fractures, bone tumors, osteoporosis, osteomalacia, prolonged immobilization causing imbalance between the rate of bone formation and resorption
Milk-alkali syndrome as a side effect of prolonged milk/antacid self-medication for gastric pain/ulcer
Hypophosphatasia, hyperproteinemia
Anticancer drugs, e.g., tamoxifen, androgens/estrogens

Patient Assessment Database

ACTIVITY/REST

May report: General malaise, fatigue/weakness
Lethargy

May exhibit: Incoordination, ataxia

CIRCULATION

May exhibit: Hypertension
Irregular pulse, dysrhythmias, bradycardia

ELIMINATION

May report: Constipation or diarrhea

May exhibit: Polyuria, nocturia
Kidney stones/calculi

FOOD/FLUID

May report: Anorexia, nausea/vomiting
Thirst
Abdominal pain

May exhibit: Poor skin turgor, dry mucous membranes

NEUROSENSORY

May report: Headache
May exhibit: Hypotonicity/muscular relaxation, flaccid paralysis, depressed/absent deep-tendon reflexes
Drowsiness, apathy, paranoia, personality changes, decreased attention span, memory loss,
depression, inappropriate/bizarre behaviors, psychosis, confusion, stupor/coma
Slurred speech

PAIN/DISCOMFORT

May report: Epigastric, deep flank pain, or bone/joint pain

TEACHING/LEARNING

Discharge plan considerations: Refer to predisposing/contributing factors
DRG projected mean length of inpatient stay: depends on underlying cause
May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

Serum calcium: Increased, higher than 2.6 mEq/L (ionized) or 10.5 mg/dL (total).
BUN: Increased (calculi can damage kidney).
Serum phosphorus: Decreased levels may be noted.
Urine Sulkowitch test: Shows heavy precipitate.
Urine calcium: Increased.
Urine osmolality: Decreased.
Urine specific gravity: Decreased.
X-ray: May reveal evidence of bone cavitation, pathological fracture, osteoporosis, urinary calculi.
ECG changes: Shortened QT interval, inverted T waves. In severe deficit, QRS may widen, PR interval lengthen, and ventricular prematurities develop.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:
Electrolyte & Acid/Base Balance (NOC)
Display heart rhythm, muscle strength, cognitive status, and laboratory results WNL for patient.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypercalcemia (NIC)</p> <p>Independent</p> <p>Monitor cardiac rate/rhythm. Be aware that cardiac arrest can occur in hypercalcemic crisis.</p>	<p>Overstimulation of cardiac muscle occurs with resultant dysrhythmias and ineffective cardiac contraction. Sinus bradycardia, sinus dysrhythmias, wandering pacemaker, and AV block may be noted. Hypercalcemia creates a predisposition to cardiac arrest.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypercalcemia (NIC)</p> <p>Independent</p> <p>Assess level of consciousness and neuromuscular status, e.g., muscle movement, strength, tone.</p> <p>Monitor I&O; calculate fluid balance.</p> <p>Encourage fluid intake of 3–4 L/day, including fluids (within cardiac tolerance) and use of acid-ash juices, e.g., cranberry and prune if kidney stones present or suspected.</p> <p>Strain urine if flank pain occurs.</p> <p>Auscultate bowel sounds.</p> <p>Maintain bulk in diet.</p> <p>Encourage frequent repositioning and ROM and/or muscle-setting exercises with caution. Promote ambulation if patient is able.</p> <p>Provide safety measures, e.g., gentle handling when moving/transferring patient.</p> <p>Review drug regimen, noting use of calcium-elevating drugs, e.g., heparin, tetracyclines, methicillin, phenytoin.</p> <p>Identify/restrict sources of calcium intake, e.g., dairy products, eggs, and spinach; calcium-containing antacids (Titalac, Tums).</p>	<p>Nerve and muscle activity is depressed. Lethargy and fatigue can progress to convulsion/coma.</p> <p>Efforts to correct original condition may result in secondary imbalances/complications.</p> <p>Reduces dehydration, encourages urinary flow and clearance of calcium, reduces risk of stone formation. <i>Note:</i> Sodium favors calcium excretion and can be used if not contraindicated by other conditions.</p> <p>Large amount of calcium present in kidney parenchyma may lead to stone formation.</p> <p>Hypotonicity leads to constipation when the smooth muscle tone is inadequate to produce peristalsis.</p> <p>Constipation may be a problem because of decreased GI tone.</p> <p>Muscle activity may reduce calcium shifting from the bones that occurs during immobilization. <i>Note:</i> Increased risk for pathological fractures exists because of calcium shifts out of the bones.</p> <p>Reduces risk of injury/pathological fractures.</p> <p>May affect drug choice or require reduction in oral sources of calcium.</p> <p>Foods or drugs containing calcium may need to be limited in chronic conditions causing hypercalcemia.</p>
<p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory studies, e.g., calcium, magnesium, phosphate.</p> <p>Administer isotonic saline and sodium sulfate IV/orally.</p>	<p>Refer to listing of predisposing/contributing factors to determine treatment needs.</p> <p>Monitors therapy needs/effectiveness. <i>Note:</i> Phosphate levels may be low when parathyroid hormone inversely promotes calcium uptake and calcium competes with phosphate for absorption/transport with vitamin D.</p> <p>Emergency measures in severe hypercalcemia used to dilute extracellular calcium concentration and inhibit tubular reabsorption of calcium, thereby increasing urinary excretion.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypercalcemia (NIC)</p> <p>Collaborative</p> <p>Administer medications as indicated:</p> <ul style="list-style-type: none"> Diuretics, e.g., furosemide (Lasix); Sodium bicarbonate; Phosphate; Glucocorticoid therapy; Plicamycin (Mithracin); Disodium edetate (EDTA); Calcitonin; Neutra-Phos, Fleet Phospho-Soda. <p>Prepare for/assist with hemodialysis.</p>	<p>Diuresis promotes renal excretion of calcium and reduces risks of fluid excess from isotonic saline infusion.</p> <p>Induces alkalosis, thereby reducing the ionized calcium fraction.</p> <p>Rapid-acting agent that induces calcium excretion and inhibits resorption of bone.</p> <p>Inhibits intestinal absorption of calcium and reduces inflammation and associated stress response that mobilizes calcium from the bone.</p> <p>Cytotoxic antibiotic that lowers serum calcium by inhibiting inappropriate bone resorption, typically seen in malignancies or hyperparathyroidism.</p> <p>Chelating action lowers serum calcium level.</p> <p>Promotes movement of serum calcium into bones, temporarily reducing serum calcium levels, especially in the presence of increased parathyroid hormone.</p> <p>These drugs bind calcium in the GI tract, promoting excretion.</p> <p>Rapid reduction of serum calcium may be necessary to correct life-threatening situation.</p>

MAGNESIUM

Magnesium influences carbohydrate metabolism, secretion of parathyroid hormone, sodium/potassium transport across the cell membrane, and synthesis of protein and nucleic acid. Magnesium activates adenosine triphosphate (ATP) and mediates neural transmission within the CNS. Magnesium deficit is often associated with hypokalemia and promotes intracellular potassium loss and sodium accumulation, altering and exacerbating membrane excitability. Normal serum range is 1.5–2.5 mEq/L or 1.8–3.0 mg/dL.

Hypomagnesemia (Magnesium Deficit)

PREDISPOSING/CONTRIBUTING FACTORS

GI losses: Biliary/intestinal fistula; surgery (bowel resection, small-bowel bypass); severe, protracted diarrhea, laxative abuse, impaired GI absorption/malabsorption syndrome, gastric/colon cancer, prolonged gastric suction
Protein/calorie malnutrition; feeding (enteral or parenteral) without adequate magnesium replacement
Prolonged IV infusion of magnesium-free solutions, multiple transfusions with citrated blood products
Chronic alcoholism, alcohol withdrawal; pancreatitis
Hyperaldosteronism: Primary or secondary (e.g., cirrhosis or HF)
Toxemia of pregnancy
Renal losses: Severe renal disease/diuretic phase of ARF; vigorous and/or prolonged diuresis with mercurial thiazides or loop diuretics; SIADH
Drugs that affect magnesium balance: Aminoglycosides (gentamicin, tobramycin), antifungals (amphotericin B); chemotherapy agents (cisplatin); antirejection agents (cyclosporine), and excessive doses of calcium or vitamin D supplements
Diabetic ketoacidosis, malignancies causing hypercalcemic states, severe burns, sepsis, hypothermia; hypoparathyroidism, hypercalcemia, hyperthyroidism

Patient Assessment Database

ACTIVITY/REST

May report: Generalized weakness, insomnia
Ataxia, vertigo

CIRCULATION

May exhibit: Tachycardia, dysrhythmias
Hypotension (vasodilation); occasional hypertension

FOOD/FLUID

May report: Anorexia, nausea/vomiting, diarrhea

NEUROSENSORY

May report: Paresthesia (legs, feet)
Vertigo
May exhibit: Nystagmus
Musculoskeletal fasciculations/tremors, neuromuscular irritability/spasticity, spontaneous carpopedal spasms, hyperactive deep-tendon reflexes, clonus
Tetany, convulsions; positive Babinski's, Chvostek's, and Trousseau's signs
Disorientation, apathy, depression, irritability, agitation, hallucinations/psychoses, coma

TEACHING/LEARNING

Refer to predisposing/contributing factors
Discharge plan considerations: **DRG projected mean length of inpatient stay: depends on underlying cause**
May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible postdischarge considerations.

DIAGNOSTIC STUDIES

Serum magnesium: Decreased, less than 1.5 mEq/L or 1.8 mg/dL. (Usually symptoms do not appear until level is less than 1 mEq/L.)

Calcium: May be decreased, unless there is a hypercalcemic condition causing the magnesium deficit.

Potassium: Decrease associated with severe hypomagnesemia.

ECG: Prolonged PR and QT intervals, widened QRS complex, ST-segment depression, T-wave inversion.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Electrolyte & Acid/Base Balance (NOC)

Display heart rate/rhythm, muscle strength, cognitive status, and laboratory results WNL for patient, absence of neuromuscular irritability.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypomagnesemia (NIC)</p> <p>Independent</p> <p>Monitor cardiac rate/rhythm, noting tachydysrhythmias and characteristic ECG changes.</p> <p>Monitor for signs of digitalis intoxication when used (e.g., reports of nausea/vomiting, blurred vision; increasing atrial dysrhythmias and heart block).</p> <p>Assess level of consciousness and neuromuscular status, e.g., movement, strength, reflexes/tone; note presence of Chvostek's/Trousseau's signs.</p> <p>Monitor status of airway and swallowing.</p> <p>Take seizure/safety precautions, e.g., padded side rails, bed in low position, frequent observation as indicated.</p> <p>Provide quiet environment and subdued lighting.</p> <p>Encourage ROM exercises as tolerated.</p> <p>Place footboard/cradle on bed.</p> <p>Auscultate bowel sounds.</p> <p>Encourage intake of dairy products, whole grains, green leafy vegetables, meat, and fish.</p>	<p>Magnesium influences sodium/potassium transport across the cell membrane and affects excitability of cardiac tissue.</p> <p>Magnesium deficit may precipitate digitalis toxicity.</p> <p>Confusion, irritability, and psychosis may occur. However, more common manifestations are muscular, e.g., hyperactive deep-tendon reflexes, muscle tremors, spasticity, generalized tetany.</p> <p>Laryngeal stridor and dysphagia can occur when depletion is moderate to severe.</p> <p>Changes in mentation or the development of seizure activity in severe hypomagnesemia increases the risk of patient injury.</p> <p>Reduces extraneous stimuli; promotes rest.</p> <p>Reduces deleterious effects of muscle weakness/spasticity.</p> <p>Elevation of linens may reduce spasms.</p> <p>Muscle weakness/spasticity may reduce peristalsis and bowel function.</p> <p>Provides oral replacement for mild magnesium deficits; may prevent recurrence.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypomagnesemia (NIC)</p> <p>Independent</p> <p>Instruct patient in proper use of laxatives and diuretics.</p> <p>Observe for signs of magnesium toxicity during replacement therapy, e.g., thirst, feeling hot and flushed, diaphoresis, anxiety, drowsiness, hypotension, increased muscular and nervous system irritability, loss of patellar reflex.</p> <p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p> <p>Monitor laboratory studies, e.g., serum magnesium, calcium, and potassium levels.</p> <p>Administer medications as indicated: Magnesium sulfate or magnesium chloride IV, monitoring administration closely;</p> <p>Magnesium sulfate IM, or magnesium hydroxide PO (Milk of Magnesia);</p> <p>Magnesium-based antacids, e.g., Mylanta, Maalox, Gelusil, Riopan.</p>	<p>Deficit may be the result of abuse of these drugs.</p> <p>Rapid, excessive IV replacement may lead to toxicity and life-threatening complications.</p> <p>Refer to listing of predisposing/contributing factors. <i>Note:</i> Studies have shown that chronic alcoholism with malnutrition is the most common cause of hypomagnesemia in the United States.</p> <p>Evaluates therapy needs/effectiveness. <i>Note:</i> These electrolytes are interrelated; symptoms may be similar, and deficits of more than one may be present.</p> <p>IV replacement is preferred in severe deficit because absorption of magnesium from intestinal tract varies inversely with calcium absorption. However, potential for drug interaction with digitalis preparations may lead to increased cardiac dysrhythmias/heart block. <i>Note:</i> Calcium gluconate is the antidote should hypermagnesemia be evidenced by depressed deep-tendon reflexes or respiratory depression and hypotension (late sign).</p> <p>May be given for mild deficit or in nonemergent situations. Injections should be deep IM to decrease local tissue reaction.</p> <p>Can supplement dietary replacement. <i>Note:</i> Use of these products may cause diarrhea, which can be alleviated by concurrent use of aluminum-containing products, e.g., Amphojel, Basaljel.</p>

Hypermagnesemia (Magnesium Excess)

PREDISPOSING/CONTRIBUTING FACTORS

Reduced renal function (e.g., acute processes or age), chronic renal disease/failure; or dialysis with hard water
Excessive intake/absorption: e.g., too-rapid replacement of magnesium (as in pregnancy-induced hypertension or premature labor), excessive use of magnesium-containing drugs/products, e.g., Maalox, Milk of Magnesia, Epsom salts
Untreated diabetic ketoacidosis
Hyperparathyroidism, aldosterone deficiency, adrenal insufficiency
Extracellular fluid volume depletion (e.g., after diuretic abuse)
Salt-water near-drowning, hypothermia, shock
Chronic diarrhea; diseases that interfere with gastric absorption

Patient Assessment Database

ACTIVITY/REST

May report: Generalized weakness, fatigue
May exhibit: Drowsiness, lethargy, stupor

CIRCULATION

May exhibit: Hypotension (mild to severe)
Pulses weak/irregular, bradycardia (12–15 mEq/L), cardiac arrest (higher than 25 mEq/L)

FOOD/FLUID

May report: Nausea/vomiting

NEUROSENSORY

May exhibit: Depressed deep-tendon reflexes (7–10 mEq/L) progressing to flaccid paralysis
Decreased level of consciousness, lethargy progressing to coma
Slurred speech

RESPIRATION

May exhibit: Hypoventilation progressing to apnea (12–15 mEq/L)

SAFETY

May exhibit: Skin flushing, sweating

TEACHING/LEARNING

Refer to predisposing/contributing factors
Discharge plan **DRG projected mean length of inpatient stay: depends on underlying cause**
considerations: May require assistance with changes in therapeutic regimen, dietary management
Refer to plan of care concerning underlying medical/surgical condition for possible considerations after discharge.

DIAGNOSTIC STUDIES

Serum magnesium: Symptomatic at levels higher than 3 mEq/L (increase to 10–20 mEq/L results in respiratory depression, coma, and cardiac arrest).

ECG: Prolonged PR and QT intervals, wide QRS, elevated T waves, development of heart block, cardiac arrest.

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Electrolyte & Acid/Base Balance (NOC)

Display heart rhythm, muscular strength, cognitive status, and laboratory results WNL for patient, absence of respiratory impairment.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Electrolyte Management: Hypermagnesemia (NIC)</p> <p>Independent</p> <p>Monitor cardiac rate/rhythm.</p> <p>Monitor BP.</p> <p>Assess level of consciousness and neuromuscular status, e.g., reflexes/tone, movement, strength.</p> <p>Monitor respiratory rate/depth/rhythm. Encourage cough/deep-breathing exercises. Elevate head of bed as indicated.</p> <p>Check patellar reflexes periodically.</p> <p>Encourage increased fluid intake if appropriate.</p> <p>Monitor urinary output and 24-hr fluid balance.</p> <p>Promote bedrest, assist with personal care activities as needed.</p> <p>Recommend avoidance of magnesium-containing antacids, e.g., Maalox, Mylanta, Gelusil, Riopan, in patient with renal disease. Caution patients with renal disease to avoid OTC drug use without discussing with healthcare provider.</p> <p>Collaborative</p> <p>Assist with identification/treatment of underlying cause.</p>	<p>Bradycardia and heart block may develop, progressing to cardiac arrest as a direct result of effect of hypermagnesemia on cardiac muscle.</p> <p>Hypotension unexplained by other causes is an early sign of toxicity.</p> <p>CNS and neuromuscular depression can cause decreasing level of alertness, progressing to coma, and depressed muscular responses, progressing to flaccid paralysis.</p> <p>Neuromuscular transmissions are blocked by magnesium excess, resulting in respiratory muscular weakness and hypoventilation, which may progress to apnea.</p> <p>Absence of these reflexes suggests magnesium levels about 7 mEq/L or higher. If untreated, cardiac/respiratory arrest can occur.</p> <p>Increased hydration enhances magnesium excretion, but fluid intake must be cautious in event of renal/cardiac failure.</p> <p>Renal failure is the primary contributing factor in hypermagnesemia; and, if it is present, fluid excess can easily occur.</p> <p>Flaccid paralysis, lethargy, and decreased mentation reduce activity tolerance/ability.</p> <p>Limits oral intake to help prevent hypermagnesemia.</p> <p>Refer to listing of predisposing/contributing factors to determine treatment needs. <i>Note:</i> Most frequently occurs in patients with advanced renal failure.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p data-bbox="235 279 740 338">Electrolyte Management: Hypermagnesemia (NIC)</p> <p data-bbox="235 359 407 386">Collaborative</p> <p data-bbox="235 405 732 459">Monitor laboratory studies as indicated, e.g., serum magnesium and calcium levels.</p> <p data-bbox="235 489 773 516">Administer IV fluids and thiazide diuretics as indicated.</p> <p data-bbox="235 577 724 604">Administer 10% calcium chloride or gluconate IV.</p> <p data-bbox="235 665 529 693">Assist with dialysis as needed.</p>	<p data-bbox="824 373 1195 401">Evaluates therapy needs/effectiveness.</p> <p data-bbox="824 462 1382 516">Promotes renal clearance of magnesium (if renal function is normal).</p> <p data-bbox="824 548 1341 602">Antagonizes action/reverses symptoms of magnesium toxicity to improve neuromuscular function.</p> <p data-bbox="824 634 1357 688">In the presence of renal disease/failure, dialysis may be needed to lower serum levels.</p>