FLUID AND ELECTROLYTE BALANCE is critical for maintaining body functions. In previous articles in this series, we discussed the normal functions of sodium, potassium, and magnesium. In this article, we review the roles of calcium and phosphorus, including assessment points and nursing considerations for patients experiencing imbalances of these important electrolytes.

**Note:** Normal value ranges may vary slightly according to age group, gender, and lab reference values. Always refer to reference lab data to verify normal serum electrolyte ranges used in your institution.

**Calcium: Stored in bones and teeth**
Calcium is the major divalent cation (a positively charged ion) found in the body. About 99% of calcium is found in the bones and teeth. The remaining 1% is found in the cells and fluid compartments, mainly in the extracellular fluid (ECF) compartment.\(^1\)

Approximately 40% of calcium in the blood is in the ionized or free state. About 45% is transported in the blood, bound to plasma proteins and nonprotein anions.\(^2\)

Serum calcium levels are measured in two ways: total and ionized. A total serum calcium level measures both bound and ionized calcium; the normal range is 8.5 to
10.5 mg/dL. The normal range of ionized calcium is 4.65 to 5.25 mg/dL.2

Although all serum calcium serves a purpose, ionized calcium most directly influences physiologic functioning of nerves and muscles. Consequently, free calcium ion concentration is a better diagnostic indicator than total calcium.3

Aided by vitamin D, calcium is absorbed from the gastrointestinal (GI) tract and stored in bone or excreted via the kidneys.1 Dairy products, especially milk, yogurt, and cheese, are the major dietary sources of calcium. Other sources include salmon, sardines, tofu, turnip greens, bread (whole wheat and white), broccoli, and kale. Many food items, such as orange juice and ready-to-eat cereals, are fortified with calcium.4

Besides being a major component of teeth and bones, calcium has other functions, including:
• regulating skeletal, cardiac, and smooth muscle contraction
• facilitating nerve impulse transmission
• activating enzymes that stimulate crucial body chemical reactions
• contributing to the coagulation system
• influencing cardiac automaticity and contractility.1,3
Regulating calcium concentrations
The body regulates ECF calcium concentration through the action of two antagonistic hormones: parathyroid hormone (PTH), and calcitonin. For example, when serum calcium levels are low, the parathyroid gland releases PTH, which stimulates release of calcium and phosphorus from bones into the ECF. (See How PTH regulates serum calcium concentration.) When serum calcium levels are high, the thyroid releases calcitonin (sometimes called thyrocalcitonin), which inhibits the release of calcium from the bones into the ECF and reduces the renal tubular reabsorption of calcium and phosphate.1

Vitamin D and its metabolites are steroid hormones, not true vitamins. The two forms, vitamin D2 and vitamin D3, have the same effect in the body.5 Vitamin D promotes bone resorption and calcium absorption through the GI tract and kidneys, raising the serum calcium level. Phosphorus acts in opposition to vitamin D, inhibiting calcium absorption in the GI tract.

For a look at how these hormones affect calcium and phosphorus levels, see How PTH, calcitonin, and vitamin D interact.

Additional factors affecting serum calcium levels
As you can see from the above discussion, calcium and phosphorus have an inverse relationship: when calcium levels increase, phosphorus levels decrease, and vice versa. pH also affects the level of ionized calcium. As pH rises and blood becomes more alkalotic, calcium binds more easily with protein, causing the level of ionized calcium to drop. Conversely, when pH falls, causing acidosis, less calcium binds with protein, which raises the ionized calcium level.1

As discussed earlier, almost half of calcium in the ECF is bound to plasma proteins (especially albumin), so serum albumin abnormalities affect total serum calcium levels. When assessing total calcium levels, also take serum albumin levels into account. Before hypocalcemia can be diagnosed, for example, total calcium levels must be corrected for hypoalbuminemia.1

Because many factors affect calcium regulation, both hypocalcemia and hypercalcemia are fairly common disorders.3,6 Let’s take a closer look at hypocalcemia.

HYPOCALCEMIA
A patient is hypocalcemic when total serum calcium level is less than 8.5 mg/dL or the ionized calcium level is below 4.65 mg/dL. Possible causes of hypocalcemia include inadequate intake of calcium, hypomagnesemia (which can reduce PTH secretion or cause PTH resistance), malabsorption (as in celiac disease), hyperphosphatemia, or excessive renal excretion of calcium. Disorders often associated with hypocalcemia include renal failure, pancreatitis, and primary and surgical hypoparathyroidism. Pseudohypocalcemia is caused by hypoalbuminemia.2

Assessment
The major signs and symptoms of hypocalcemia are related to increased neuromuscular excitability. Mild hypocalcemia may cause few if any signs and symptoms, but severe hypocalcemia, which can cause
seizures, heart failure, and laryngospasm, can be life-threatening.\(^7\)

During physical assessment, you may elicit a positive Chvostek sign or Trousseau sign.\(^1,3,7\) See Two signs of trouble. For more signs and symptoms of hypocalcemia, see Comparing hypocalcemia and hypercalcemia.

**Treatment**

Interventions for hypocalcemia depend on various factors, such as the underlying cause. Treatment includes administration of oral or I.V. calcium preparations, depending on the severity of the condition.

Acute symptomatic hypocalcemia, a medical emergency, requires prompt administration of I.V. calcium gluconate or calcium chloride (calcium gluconate is usually preferred because extravasation is less likely to cause tissue necrosis).\(^7\) Use extreme caution in patients taking digoxin because calcium solutions can cause digoxin toxicity.\(^9\) Oral vitamin D supplementation is indicated for patients with vitamin D deficiency or hypoparathyroidism.\(^7\) Because hypocalcemia and hypomagnesia often occur together, the patient may also need magnesium supplementation.

**Nursing implications**

Implement safety measures and monitor for tetany and seizure activity. Initiate seizure precautions if appropriate. Instruct the patient/family to call for assistance when the patient wants to get out of bed.

Monitor vital signs, including apical pulse, and assess heart sounds. Closely monitor for dysrhythmias and QT prolongation. Calcium solutions are highly irritating to veins, so administer the solution slowly as prescribed, and frequently assess the I.V. site for signs and symptoms of extravasation, such as pain or burning at the site, erythema, and edema. Closely monitor the patient because too-rapid administration can produce cardiac dysrhythmias and cardiac arrest. Give oral calcium after meals or at bedtime with a full glass of water.\(^8\) Monitor serial electrolyte results and report abnormalities to the healthcare provider.

Before discharge, teach the patient to eat foods with sufficient calcium, vitamin D, and protein. A patient with high phosphate levels may be prescribed aluminum hydroxide antacids to bind with the excess phosphate; because phosphate and calcium are reciprocally regulated, this helps raise calcium levels.\(^9\) Educate the patient about all prescribed medications. Teach the patient about signs and symptoms of both hypocalcemia and hypercalcemia, and discuss when to call the healthcare provider.

**HYPERCALCEMIA**

By definition, hypercalcemia is a total serum calcium level above 10.5 mg/dL, or an ionized calcium level above 5.25 mg/dL. Although less common than hypocalcemia, hypercalcemia can be dangerous if severe; mortality for hypercalcemic crisis is high if the condition isn’t treated promptly.\(^9\)

Hypercalcemia is usually related to conditions that trigger an increase in the reabsorption of calcium that exceeds urinary calcium excretion or bone deposition. The most common causes of hypercalcemia are hyperparathyroidism, in which the parathyroid gland secretes higher than normal levels of PTH, and certain malignancies.\(^10\) Other causes of hypercalcemia include hyperthyroidism, prolonged immobilization, hypophosphatemia, multiple fractures, excessive vitamin D intake, thiazide diuretic or lithium use, and high use of medications containing calcium.\(^11\)

**Assessment and treatment**

Clinical signs and symptoms of hypercalcemia depend on its severity and rate of development. ECG changes include a shortened QT
interval and prolonged PR interval.\textsuperscript{12} (See Comparing hypocalcemia and hypercalcemia.)

Interventions for hypercalcemia include identifying and treating the underlying cause and decreasing the serum calcium level. Mild hypercalcemia may be treated conservatively by decreasing the patient's dietary intake of calcium, stopping medications contributing to calcium excess, and encouraging the patient to drink fluids for hydration.\textsuperscript{12}

Patients with more severe hypercalcemia require immediate treatment. Those with normal renal and cardiac function initially receive an I.V. infusion of 0.9% sodium chloride at 200 to 300 mL/hour to dilute the serum calcium concentration and facilitate renal excretion.\textsuperscript{12} Administered intramuscularly or subcutaneously, calcitonin lowers the serum calcium level by increasing renal calcium excretion and by decreasing bone resorption.\textsuperscript{13} Biphosphonates inhibit calcium release by interfering with osteoclast-mediated bone resorption.\textsuperscript{12} Life-threatening hypercalcemia may require dialysis.\textsuperscript{9}

**Nursing implications**
Monitor the patient for muscle weakness and assess vital signs, including the apical pulse. Also monitor all electrolyte levels, intake and output, renal function, and neurologic status, and institute safety measures as indicated. Assess for signs and symptoms of dehydration related to vomiting, and fluid overload related to I.V. therapy.\textsuperscript{12} Instruct the patient/family to call for assistance when the patient wants to get out of bed.

**All about phosphorus**
Normal serum phosphate levels for adults range from 2.7 to 4.5 mg/dL.\textsuperscript{14} Most phosphate (80% to 85%) is located in the bones and teeth, with the remainder in the cells and body fluids.\textsuperscript{15} Foods high in dietary phosphorus include almonds, dried beans, barley, bran, pumpkin, cheese, eggs, chocolate, meats, poultry, peanuts, and wheat. Soft drinks provide phosphoric acid.\textsuperscript{1} GI absorption of phosphate requires vitamin D.

Phosphate works as a buffer to maintain acid-base balance, and is a component of adenosine triphosphate, an energy-storing source within the body, and 2,3-bisphosphoglycerate (BPG, also called diphosphoglycerate) a substance in RBCs that facilitates oxygen delivery to the tissues. Cell membranes are made of phospholipids, so phosphate is essential for cell membrane integrity.\textsuperscript{11,15} Phosphate also plays an important role in
- muscle function
- neurological function
- fat, carbohydrate, and protein metabolism
- phagocytosis
- platelet function
- structural support of bones and teeth.\textsuperscript{11,15}

Because most phosphate is stored in teeth and bones, serum levels don't always reflect the overall amount of phosphate in the body.\textsuperscript{11}

**HYPOPHOSPHATEMIA**
A serum phosphate level below 2.7 mg/dL is considered hypophosphatemia.\textsuperscript{14} It can accompany such conditions as hyperparathyroidism, renal insufficiency, vitamin D deficiency or resistance, gastrectomy, increased insulin secretion during refeeding syndrome (especially if phosphate isn't added to parenteral nutrition), steatorrhea and chronic diarrhea, and acute respiratory alkalosis.\textsuperscript{16} Other possible causes of hypophosphatemia include alcoholic cirrhosis, overuse of antacids containing magnesium or aluminum, and conditions creating hypercalcemic states. Major thermal burns may also contribute to hypophosphatemia.\textsuperscript{5,16}

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**Two signs of trouble\textsuperscript{1,3,7}**
Positive Chvostek and Trousseau tests are associated with hypocalcemia.
- Chvostek sign: contraction of ipsilateral facial muscles when the facial nerve is tapped just in front of the ear.
- Trousseau sign: carpopedal spasm elicited by inflating a sphygmomanometer above systolic BP for 3 minutes.
Assessment
Clinical manifestations depend on the severity and chronicity of phosphate deficiency. Patients with hypophosphatemia may be asymptomatic or experience vague or nonspecific signs and symptoms, such as weakness, malaise, nausea, vomiting, and diarrhea. Cardiopulmonary manifestations include tachypnea, decreased myocardial contractility, and vasodilation. Neurologic signs and symptoms include confusion, irritability, apprehension, and delirium, which may progress to seizures or coma.

Evidence of long-term phosphate deficiencies may include bone pain, pathologic fractures, hyporeflexia, irritability, myalgia, and paresthesias. Many additional signs and symptoms are related to hypercalcemia. Chronic hypophosphatemia can cause ecchymoses and bleeding due to platelet dysfunction and may also predispose patients to infection because of leukocyte dysfunction.

Treatment
Interventions include replacing phosphate, usually with oral preparations, and identifying and correcting the underlying cause. For example, vitamin D supplementation is prescribed for patients with a vitamin D deficiency.

Severe hypophosphatemia is life threatening and requires prompt intervention. When needed, phosphate may be added to parenteral and enteral feeding solutions to maintain adequate levels.

If I.V. phosphate is required, administer it with caution as prescribed. Because it can precipitate with calcium, it can cause various potentially severe adverse reactions, including hypocalcemia, renal failure, and dysrhythmias.

Nursing implications
Assess patients with hypophosphatemia for malnutrition, alcoholism, antacid use, and infection. Monitor lab values, including complete blood cell count and electrolytes, dietary intake, changes in mental status, and bowel sounds. Closely monitor patients receiving parenteral nutrition for signs and symptoms of hypophosphatemia, such as myalgia and muscle weakness. Maintain patient safety measures.

Instruct the patient/family to call for assistance when the patient wants to get out of bed and to notify staff if the patient develops muscle weakness or pain. Before discharge, educate the patient about foods that are high in phosphorus.

**HYPERPHOSPHATEMIA**
Phosphate excess, known as hyperphosphatemia, occurs when the serum phosphate level is above 4.5 mg/dL. Hyperphosphatemia is rare when the kidneys are functioning properly. Common causes for an increase in phosphate levels are chronic renal failure, hypoparathyroidism, vitamin D intoxication, tumor lysis syndrome, rhabdomyolysis, lactic acidosis, hypoparathyroidism, ketoacidosis, and excessive use of laxatives containing phosphate.

In renal failure, calcium can’t be reabsorbed and phosphate can’t be excreted. In hypoparathyroidism, either deficient PTH secretion or renal resistance to PTH causes the kidneys to reabsorb phosphate, raising serum levels. Vitamin D intoxication increases both calcium and phosphate levels.

Assessment and treatment
Signs and symptoms of hyperphosphatemia are similar to those identified with hypocalcemia, including neuroexcitability, tetany, and seizures. Excess phosphate may precipitate into body tissue as phosphate salts, causing metastatic calcifications of soft tissue, joints, and arteries; consequently, the patient may exhibit ocular signs and symptoms (such as conjunctivitis), pruritis, and arthrosis. Renal deposits can lead to or exacerbate renal...
failure. Anorexia, nausea, vomiting, muscle weakness, hyperreflexia, and tachycardia may occur.  

Treatment of hyperphosphatemia depends on the underlying cause. Patients with chronic hyperphosphatemia may be prescribed a low-phosphate diet and phosphate binders. Critically ill patients may need dialysis to reduce the phosphate level.

**Nursing implications**

Maintain patient safety measures. Assess for vision changes and monitor lab values, including both serum phosphate and calcium levels. Assess for a history of hypoparathyroidism, renal failure, or excess intake of vitamin D.

Nursing interventions for hyperphosphatemia not related to renal failure include I.V. saline infusions to increase urinary phosphate excretion. Monitor intake and output, and restrict foods high in phosphorus.

Educate the patient on proper nutrition and avoiding foods high in phosphorus. Warn the patient not to take antacids containing phosphate unless directed otherwise by the healthcare provider.

Teach the patient/family to call for assistance before the patient gets out of bed. ■

**REFERENCES**


Ann Crawford is a professor at the College of Nursing at University of Mary Hardin-Baylor in Belton, Tex. Helene Harris is a clinical educator at Central Texas Veterans Healthcare System in Temple, Tex. The authors and planners have disclosed that they have no financial relationships related to this article.

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