

# Fluid and Electrolyte Imbalances: Interpretation and Assessment

## ABSTRACT

Maintaining the balance of fluid and electrolytes is crucial to the care of patients across the continuum. To do this, a practitioner must be cognizant of key monitoring and assessment parameters. Key electrolytes, their function within the body, normal values, signs and symptoms of imbalances, key treatment modalities, and other considerations are discussed.

**Key words:** calcium, electrolyte imbalances, fluid balance, magnesium, phosphorus, potassium, sodium

An understanding of the normal balance of fluid and electrolytes in the body is essential not only to understand normal functioning of the human body but also to predict problems and to intervene during various disease processes. To do that, the practitioner must have a thorough understanding of the balance of fluids, homeostasis, electrolytes, and the shifts that can result when 1 element is no longer in balance.

## THE BALANCE OF FLUID

The human body is composed of approximately 70% water. Two-thirds of the water is found in the intracellular

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The author has no conflicts of interest to disclose.

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DOI:10.1097/NAN.000000000000193

fluid. The other one-third is found in the extracellular fluid and comprises water and sodium chloride. Extracellular fluid is further categorized as intravascular and interstitial fluid. The body maintains a balance of fluid to molecules, including electrolytes, which is known as homeostasis.<sup>1-9</sup>

Homeostasis is maintained through the transport of solutes through semipermeable membranes. This is accomplished using many different processes. The process of diffusion occurs when the ions and molecules in a solution spread out for uniform distribution. Filtration is the process through which a liquid or gas passes through a filter (membrane) by forces of pressure. Osmosis occurs when a solute passes through a membrane, leaving a solution of higher concentration to a solution of lower concentration, thereby balancing the solutes on either side of the membrane. Finally, active transport is the process of solutes moving against the electrochemical gradient, such as the sodium-potassium pump that moves the electrolytes back and forth depending on the need of the cell.<sup>1-10</sup>

For fluid in the body, normal serum osmolality—the concentration of solutes in intravascular fluid—is 275 to 295 mOsm/kg.<sup>8,9</sup> Therefore, any serum osmolality greater than 295 is known as hyperosmolar, while a serum osmolality less than 275 is known as hyposmolar. The osmolality of body fluids affects the tonicity of the fluid, the pressure or tension of solutes in the solution, and will have an impact on the concentration of various solutes and normal functioning in the cells.<sup>8,9</sup>

This is known as osmotic pressure, which is proportional to the osmolality of a solution. In reference to the human body, an isotonic solution has the same osmotic pressure as the intravascular fluid.<sup>1-10</sup> Examples of isotonic solutions include 0.9% sodium chloride, lactated Ringer's (LR), and 5% dextrose in water. For this reason, these solutions typically are the first line used to replace fluid lost in the body.<sup>8,9</sup> Hypertonic solutions, those with a higher osmotic pressure compared with intravascular fluid, include 5% dextrose in 0.9% sodium chloride or LR. Hypertonic solutions can be used to draw fluid from the intracellular and interstitium into

the intravascular space to help reverse fluid overload.<sup>8,9</sup> Hypotonic solutions, such as sodium chloride 0.45%, contain a lower osmotic pressure than intravascular fluid and can be used to promote fluid shifts out of the intravascular space and into the intracellular or interstitial spaces.<sup>8,9</sup>

There are several processes at work to maintain the balance of fluid in the body. When the body senses a decrease in circulating volume or an increase in serum osmolality, multiple pathways serve to correct the issue. First is the thirst mechanism, which is housed in the hypothalamus and is driven by either a decrease in intravascular volume or an increase in serum osmolality.<sup>1-9</sup> When the body senses either of these states, the body interprets the problem and responds by triggering the urge to drink. In addition, antidiuretic hormone (ADH) is released by the pituitary gland in response to a decrease in circulating volume and promotes retention of fluid in the body.<sup>1-9</sup> In the kidneys, the juxtaglomerular apparatus measures renal blood flow. When a decrease in renal flow is detected, it works to regulate both sodium and fluid levels in the body through the renin-angiotensin-aldosterone system (RAAS). The RAAS causes the kidneys to reabsorb sodium, which then pulls water from the intracellular fluid and into the intravascular fluid, increasing flow.<sup>1-9</sup>

Causes of hypovolemia include decreased intake; decreased absorption, such as from a bowel resection; hemorrhage; excessive diuresis, such as with diabetes insipidus (DI); and prolonged vomiting and diarrhea.<sup>1-9</sup> Clinical manifestations include thirst, dry mucous membranes, tachycardia, poor skin turgor, and the later signs of hypotension and oliguria.<sup>1-9</sup> Treatment should focus first on stopping the loss and then on replacing the volume.<sup>1-9</sup>

When the heart senses an increase in blood volume, atrial natriuretic peptide is released, which then blocks the RAAS, resulting in the loss of both sodium and fluid in an attempt to normalize blood pressure and circulating volume.<sup>1-9</sup>

Causes of hypervolemia include inadequate sodium and water excretion (such as with Cushing's syndrome, heart failure, or renal failure) or excessive sodium intake.<sup>1-9</sup> Hallmarks of fluid overload include peripheral and pulmonary edema, hypertension, venous distension, and acute weight gain.<sup>1-9</sup> Treatment should be focused on correcting the underlying cause and possibly restricting intake.<sup>1-9</sup>

## THE BALANCE OF ELECTROLYTES

The shift and flux of electrolytes are how the body relays messages and conducts normal functioning within the cells. Cations are positively charged electrolytes, while anions are negatively charged. Although there are others, sodium, potassium, calcium, phosphorus, and

magnesium are key electrolytes that have an important effect on normal functioning in the cells.

### Sodium

Sodium ( $\text{Na}^+$ ) is the primary cation in the body's extracellular fluid. Its role is to regulate fluid volume, osmolality, and acid-base balance, as well as to conduct muscle and nerve activity.<sup>1-10</sup> The body regulates sodium through dietary intake, excretion by the kidneys, and hormonal responses to aldosterone and ADH.<sup>1-10</sup> The normal range for sodium is 135 to 145 mEq/L.<sup>11,12</sup>

#### Critical Thinking Exercise 1

As you read the following information regarding sodium imbalances, consider this patient scenario: You are caring for a 26-year-old autistic patient who was just admitted for sudden onset of seizures and a decreased level of consciousness. His mother says he "really likes water" and "drinks at least a couple of gallons a day." What could be the cause of his symptoms, and how should you treat him?

Hypernatremia ( $\text{Na} > 145$ ) can be caused either by excessive intake—whether from dietary intake or from rapid infusion of isotonic or hypertonic solutions—or from excessive loss of water through, for example, DI, an impaired thirst center, decreased intake, or an inability to concentrate urine. Clinical manifestations of hypernatremia include thirst and dry mucous membranes, disorientation and hallucinations, oliguria or anuria, and a rapid, weak pulse.<sup>1-12</sup>

The key to treating hypernatremia is to identify and treat the underlying cause. For example, DI should be treated with vasopressin to cause the kidneys to retain water. Additionally, an infusion of a hypotonic solution should be used to help replace fluid without increasing sodium levels. If the hypernatremia is an acute condition, aggressive therapy to reduce sodium levels by 1 mEq/L/h should be instituted. If the hypernatremia is a chronic condition (in existence for more than 48 hours), care should be taken to reduce sodium levels slowly. During hypernatremia, the body's initial response is to pull water from the intracellular fluid, including the cells in the brain. A rapid decrease in sodium during a chronic condition will then cause a massive influx of water back into the cells and can result in cerebral edema. The reduction of sodium should be slow, about 0.5 mEq/L/h (or 10-12 mEq/L over 24 hours).<sup>1-12</sup>

Hyponatremia ( $\text{Na} < 135$ ) can be caused by excessive diuresis, adrenocorticoid insufficiency, increased fluid intake, ketoacidosis, and syndrome of inappropriate antidiuretic hormone (SIADH). Signs and symptoms include muscle cramps and weakness, lethargy and/or agitation,

orthostatic hypotension, anorexia, and in extreme cases, seizures. Sodium levels less than 115 mEq/L can cause permanent neurological dysfunction.<sup>1-12</sup>

Again, the key to treatment is to identify and correct the underlying cause. In addition, fluids may be restricted to help correct the imbalance. The aggressiveness of sodium replacement is dependent on signs and symptoms. In an asymptomatic patient, replacement of 8 to 10 mEq/L over 24 hours is sufficient. In a symptomatic patient, more aggressive replacement should increase sodium levels by 1 to 2 mEq/L/h.<sup>1-12</sup>

### Critical Thinking Answer 1

Your patient is suffering from hyponatremia related to excessive water intake. Given the severity of the symptoms, rapid sodium replacement should occur to prevent recurrence of seizures and to normalize sodium levels.

## Potassium

Potassium ( $K^+$ ) is the primary cation in intracellular fluid; 98% of potassium can be found in the intracellular fluid. Its key functions include conduction related to neuromuscular, cardiac, and skeletal muscle activity. Intake of potassium is through diet. Eighty percent of potassium is excreted through the distal tubules of the kidneys, while 20% is excreted through sweat and the bowel.<sup>1-10</sup> Its normal range is 3.5 to 5 mEq/L.<sup>11,12</sup>

### Critical Thinking Exercise 2

As you read the following information regarding potassium imbalances, consider this patient scenario: You are caring for a 56-year-old patient in end-stage renal failure. She says she started feeling ill 3 days before and missed her regularly scheduled dialysis appointment. She came into the emergency department because of “palpitations.” Her electrocardiogram shows widened QRS and tall, peaked T waves. What could be her diagnosis? What would be the most appropriate intervention?

Hyperkalemia ( $K > 5$  mEq/L) can be caused by excessive intake; certain pharmaceuticals, such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta blockers, and potassium-sparing diuretics; conditions, such as Addison’s disease; and impaired renal function and metabolic acidosis. In cases of extreme cellular trauma, such as burns and crush injuries, large quantities of intracellular potassium can be released into extracellular fluid. Key clinical

manifestations of hyperkalemia include muscle cramps, weakness and paresthesias, hyperreflexia, and changes to the electrocardiogram (ECG), including widened QRS and tall, peaked T waves, and, in severe cases, ventricular fibrillation.<sup>1-12</sup>

Treatment of hyperkalemia should include changing any contributory medications and decreasing intake. Short-term or immediate treatment may include the administration of sodium polystyrene sulfonate, intravenous (IV) insulin and a 50% dextrose solution, sodium bicarbonate, or calcium chloride. Dialysis also may be used to decrease potassium levels rapidly.<sup>1-12</sup>

Hypokalemia can be caused by inadequate dietary intake, excessive diuresis, vomiting and diarrhea, metabolic acid-base disorders, Cushing’s disease, the administration of beta-adrenergic agonists (such as epinephrine, dobutamine, and albuterol), and large doses of IV insulin given for the treatment of diabetic ketoacidosis (DKA) or hyperglycemic hyperosmolar nonketotic syndrome. Clinical manifestations include muscle weakness, fatigue, hyporeflexia, metabolic alkalosis, and ECG changes (prolonged PR interval and flattened T waves).<sup>1-12</sup>

Potassium can be replaced orally or intravenously, depending on the severity of symptoms. IV potassium replacement should not exceed 20 mEq/h to prevent complications similar to hyperkalemia resulting from rapid infusion.<sup>1-12</sup>

### Critical Thinking Answer 2

Your patient was suffering from hyperkalemia. Given the patient’s history and symptoms, the most appropriate intervention would be to dialyze her acutely to return her potassium levels to within acceptable limits.

## Calcium

Calcium ( $Ca^+$ ) is an essential cation, 99% of which can be found in the skeletal system. Its functions include the transmission of nerve impulses, muscle contraction and relaxation, certain enzyme activation, coagulation, and bonding with proteins to pass through capillary walls, as well as being the primary component of bones and teeth.<sup>1-10</sup> Regulation of calcium is through dietary intake, and excretion is through calcitonin and parathyroid hormone.<sup>1-10</sup> The normal range for total calcium is 8.5 to 10.5 mg/dL.<sup>11,12</sup>

Calcium has an inverse relationship with phosphorus, meaning an increase in one will result in a decrease in the other, and vice versa. In addition, because of its ability to bond with proteins to assist with transport across cell walls, not all serum calcium is bioavailable for other functions. For this reason, practitioners should

use either a corrected calcium level or an ionized calcium level. Corrected calcium is a calculation done to correct bioavailable calcium based on serum albumin levels. The formula for corrected calcium is  $\text{Ca} + 0.8 (4 - \text{serum albumin})$ . Ionized calcium is a laboratory test for calcium not bound to protein (normal 1.15-1.34 mg/dL). Ionized calcium may be preferred in critical care settings or in disease processes causing protein deficiencies. An ionized calcium greater than 1.35 mg/dL carries a 50% mortality rate.<sup>1-12</sup>

### Critical Thinking Exercise 3

As you read the following information regarding calcium imbalances, consider this patient scenario: You are caring for a 34-year-old patient who was just admitted for muscle spasms, particularly in the face and hands. She complains of an inability to control the “twitching” that tends to occur when she grips heavy items and the facial tics that come and go. She reports a recent bout of gastrointestinal upset with 2 to 3 days of vomiting and diarrhea. What could be the cause of her symptoms? How should you treat her?

Hypercalcemia ( $\text{Ca} > 10.5 \text{ mg/dL}$ ) can be caused by acidosis; renal failure; hyperparathyroidism; thyrotoxicosis, including Graves’ disease; and certain cancers, such as squamous cell carcinoma, breast and ovarian cancer, and bone metastasis. Its clinical manifestations include constipation, hypotonicity, renal calculus, confusion and stupor, bradycardia with wide T waves, and, in severe cases, cardiac arrest.<sup>1-12</sup>

Treatment includes identifying and treating the underlying cause and administration of loop diuretics and IV fluids.<sup>1-12</sup>

Hypocalcemia ( $\text{Ca} < 8.5 \text{ mg/dL}$ ) can be caused by disease processes such as pancreatitis, hypoparathyroidism, hypoalbuminemia, metabolic alkalosis, hypomagnesemia, and large-volume administration of citrated blood products (blood products treated with preservatives). Signs and symptoms include numbness or tingling of the hands, toes, and mouth; tetany; emotional instability; seizures; hypotension; and a short QT interval on an ECG. Assessments for hypocalcemia include Trousseau’s sign, an inflated blood pressure cuff causing muscle spasm in the thumb or hand, and Chvostek’s sign (tapping 1 inch below the zygomatic arch at the angle of the jaw will cause a contraction of facial muscles).<sup>1-12</sup>

In addition to treating the underlying cause, calcium replacement may be administered orally or intravenously. Calcium gluconate is preferred over calcium chloride for calcium replacement.<sup>1-12</sup>

### Critical Thinking Answer 3

It’s likely your patient had hypocalcemia related to metabolic alkalosis. The treatment would include ensuring the gastrointestinal issues are resolved and administering calcium replacement.

## Phosphorus

Phosphorus ( $\text{P}^-$ ) is a major intracellular anion, 85% of which can be found in bone.<sup>1-10</sup> Phosphorus acts as a buffer for acid-base balance, is essential for neuromuscular and red blood cell function, and is a primary structure of teeth and bones.<sup>1-10</sup> Phosphorus levels are regulated through the kidneys, parathyroid gland, and other hormone levels.<sup>1-10</sup> The normal range for phosphorus is 2.5 to 4.5 mg/dL.<sup>11,12</sup>

### Critical Thinking Exercise 4

As you read the following information regarding phosphorus imbalances, consider this patient scenario: You are caring for a 40-year-old construction worker who was admitted with a left femur fracture after being run over at work by a forklift. He has just returned from the operating room following a fasciotomy for compartment syndrome of the left leg. He calls out and complains of numbness and tingling in all extremities; his blood pressure is lower than when he first returned from the operating room. What could be the cause of his symptoms? How should you treat him?

Hyperphosphatemia ( $\text{P} > 4.5 \text{ mg/dL}$ ) can be caused by use of laxatives; renal impairment; hypoparathyroidism; tumor lysis syndrome; and extreme muscle necrosis, as seen in compartment syndrome. Clinical manifestations include tetany; paresthesias; hypotension; and dysrhythmias, in particular, torsade de pointes from an associated hypomagnesemia.<sup>1-12</sup>

Treatment for hyperphosphatemia includes decreasing phosphate intake; administering calcium-based phosphorus binders, because phosphorus has an inverse relationship with calcium levels; certain diuretics; and dialysis.<sup>1-12</sup>

Hypophosphatemia ( $\text{P} < 2.5 \text{ mg/dL}$ ) can be caused by excessive antacid intake; severe protein-calorie malnutrition, as seen in severe burn patients; DKA; hyperparathyroidism; and salicylate poisoning. Signs and symptoms include ataxia, intention tremors, weakness, paresthesias, confusion, seizures, hemolysis and significant bleeding, and, in severe cases, coma and respiratory failure.<sup>1-12</sup>

### Critical Thinking Answer 4

Your patient may be suffering from hyperphosphatemia. You can treat it by increasing phosphorus excretion through the administration of a diuretic, such as acetazolamide.

Treatment includes identification of the underlying cause and phosphorus replacement.<sup>1-12</sup>

### Magnesium

Magnesium (Mg<sup>+</sup>) is a significant intracellular cation. It plays a role in carbohydrate and protein metabolism,

### Critical Thinking Exercise 5

As you read the following information regarding magnesium imbalances, consider this patient scenario: You are caring for an 88-year-old female patient who arrived lethargic and confused. She says she “couldn’t remember if she had had a bowel movement recently” and that she had been taking multiple products to help facilitate moving her bowels. While you are assessing her, you notice her cardiac rhythm converts into a wide, complex, chaotic rhythm, and she loses consciousness. What could be the cause, and how should you treat her?

protein and deoxyribonucleic acid synthesis, and electrical conductivity.<sup>1-10,13</sup> It is regulated through dietary intake, the kidneys, and the parathyroid gland.<sup>1-10,13</sup> The normal range for magnesium is 1.5 to 2.4 mg/dL.<sup>11,12</sup>

Hypermagnesemia (Mg > 2.4 mg/dL) is a rare imbalance caused by renal failure, DKA, Addison’s disease, hyperparathyroidism, and hypothyroidism. It is usually concurrent with hypocalcemia and hyperkalemia. Clinical manifestations include confusion, seizures, weakness and hyporeflexia, dysphagia, flushed face, and atrioventricular blocks on an ECG.<sup>1-13</sup>

Treatment includes administration of IV fluids, calcium, and possibly dialysis in the case of renal dysfunction.<sup>1-13</sup>

Hypomagnesemia (Mg < 1.5 mg/dL) can be caused by diuretics; increased calcium intake, which competes with magnesium to bind with proteins; alcoholism; excessive laxative use; small-bowel bypass; and SIADH. Signs and symptoms include confusion, dizziness and headache, nystagmus, hypertension, tetany, and ventricular dysrhythmias (torsade de pointes). Assessment parameters may include positive Chvostek’s, Trousseau’s, and Babinski’s signs.<sup>1-13</sup>

Treatment includes magnesium infusion, usually 1 to 2 g. Caution should be taken in magnesium replacement for patients in renal failure.<sup>1-13</sup>

### Critical Thinking Answer 5

Your patient most likely has hypomagnesemia and is displaying torsade de pointes. Emergent treatment for this includes the immediate administration of IV magnesium.

## CONCLUSION

Understanding the balance of fluid and electrolytes in the body is essential to predicting complications and planning care in acutely ill patients. The ability to predict imbalances based on disease processes, identify signs and symptoms of imbalance and key assessment parameters, and formulate treatment modalities will allow practitioners to provide optimal care to patients who have a variety of medical issues.

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