FLUID CIRCULATING throughout the body contains electrolytes: elements or minerals that, when dissolved in water or another solvent, dissociate into ions and carry an electrical charge, either positive (cations) or negative (anions). Electrolyte concentrations differ in extracellular and intracellular fluids, but overall the total concentration of cations and anions in each fluid compartment should be equal or balanced. Electrolyte abnormalities occur when this balance is upset.

This article reviews the normal functions of two key electrolytes, sodium and potassium, and discusses nursing assessment and intervention when imbalances occur. For a review of how various I.V. fluids act within the body, see the first article in this series, "I.V. Fluids: What Nurses Need to Know," in the May issue of Nursing2011.

Note: Normal value ranges may vary slightly for different age groups, between males and females, and among different analytical labs. Always refer to...
individual reference lab data to verify normal serum electrolyte ranges used in your institution.

**Sodium: Abundant cation**
The normal serum value for sodium, the most abundant cation in extracellular fluid, is 135 to 145 mEq/L. Sodium is the primary determinant of extracellular fluid osmolality, so it has a principal role of controlling water distribution and fluid balance throughout the body. (See *Osmolality or osmolarity: What’s the difference?*) Remember that water follows sodium, so high levels of sodium in a fluid compartment will draw water with it. Some diuretics utilize this principle to achieve diuresis. Sodium also functions to:
- promote transmission of nerve impulses
- maintain intracellular osmolality
- activate several enzymatic reactions
- assist with regulation of acid-base balance
- promote myocardial, skeletal, and smooth muscle contractility.\(^1\,^2\)
Sodium is actively absorbed by the intestines and excreted by the kidneys. The body possesses an intricate system of safeguards and feedback mechanisms to monitor and maintain the sodium level in the extracellular fluid. When sodium levels begin to rise, the body makes adjustments by stimulating a thirst mechanism, which encourages the person to drink additional water.

Sodium levels are influenced by antidiuretic hormone (ADH) and aldosterone through their sodium and water conservation and excretion feedback mechanisms. Increased secretion of ADH causes more water to be reabsorbed in the kidneys; decreased ADH secretion allows more water to be excreted.

Aldosterone is a mineralocorticoid produced in the adrenal cortex. High aldosterone levels promote the reabsorption of sodium, along with water and chloride, in the kidneys’ distal tubules. Because the body is so adept at protecting its sodium, people don’t need to ingest large quantities of sodium.1,2

Sodium functions to maintain acid-base balance by readily combining with chloride and bicarbonate ions. This facilitates the maintenance of balance between cations and anions to help prevent pH imbalances. As a mainly extracellular ion, sodium works with potassium (which is primarily intracellular) to maintain the normal balance of electrolytes in the intracellular and extracellular fluids through the sodium-potassium pump, an active transport mechanism. (See How the sodium-potassium pump works.) Because it helps promote excitability of nerve impulses, this pump system has an important role in conducting impulses in muscle and nerve fibers.

Now let’s look at what happens when imbalances of these electrolytes occur.

**HYponATREMIA**

A serum sodium level below 135 mEq/L is considered hyponatremia. This condition can be due to low levels of sodium or to excess water in relation to the amount of sodium, sometimes referred to as dilutional hyponatremia. Some common causes of hyponatremia include profuse diaphoresis, draining wounds, excessive diarrhea or vomiting, trauma with significant blood loss, low sodium intake, hormonal changes associated with Addison disease or hypothyroidism, and overuse of thiazide diuretics. Low sodium levels may be seen in patients with aldosterone deficiency due to adrenal insufficiency and in patients diagnosed with the syndrome of inappropriate secretion of antidiuretic hormone. Hyponatremia is also a common electrolyte imbalance in postoperative patients.

**Assessment**

A drop in sodium level causes cellular edema, which affects the central nervous system (CNS), leading to CNS depression and cerebral edema. Monitor patients for signs and symptoms of CNS depression (mental status changes, headache, personality changes, and irritability). Gastrointestinal (GI) symptoms of hyponatremia include nausea, vomiting, abdominal discomfort, and diarrhea. Severe hyponatremia (115 mEq/L or less) causes muscle twitching and tremors, focal weakness, seizures, signs of increasing intracranial pressure, and coma.3

**Treatment**

The treatment goal is to determine and treat the underlying cause of hyponatremia and replace the lost sodium. Because sodium is plentiful in a normal diet, patients who can eat and drink can easily replace low levels. But those who can’t take sodium orally may require I.V. administration of lactated Ringer’s solution or 0.9% sodium chloride solution.4 The patient may also need to restrict water intake.

Tolvaptan is an oral medication used to treat clinically significant hypervolemic and euvolemic hyponatremia (serum sodium less than 125 mEq/L or less marked hyponatremia that causes symptoms and hasn’t responded to correction with fluid restriction).

**Nursing implications**

Keep in mind that misuse of hypertonic saline can be extremely dangerous, so it should be administered cautiously. A possible result of correcting hyponatremia too rapidly is osmotic demyelination syndrome, which involves destruction of the myelin sheath of axons in the brain stem. This syndrome can cause severe brain damage and death.4

Closely monitor intake and output. Assess for changes in level of consciousness and monitor for seizure activity. Institute patient safety measures and monitor serum electrolyte levels and urine and serum osmolality as indicated.

Educate the patient and family about the role of sodium in the body and what a low blood level means. Teach signs and symptoms of hyponatremia and when to notify the healthcare provider. If fluid intake is restricted, tell the patient how much water he or she can drink and formulate a plan for spreading out intake during the day. Ask the patient to keep track of what he or she drinks.
and to let you know so you can document oral intake accurately.

**HYPERNATREMIA**

By definition, hypernatremia is an excess of sodium concentration in serum (levels above 145 mEq/L). It’s generally associated with a hyperosmolar state where a fluid volume deficit exists, although it also can exist in normal fluid volumes with excess sodium ions. The increase in extracellular sodium causes intracellular fluid to shift out into the extracellular space, causing cellular dehydration. Some common causes of hypernatremia include inadequate water intake or excessive fluid loss, administration of tube feedings and other high solute solutions without adequate water supplements, diarrhea, and excessive steroid use. Medical conditions associated with hypernatremia include hyperaldosteronism, Cushing syndrome, diabetes insipidus, and renal failure. Administration of hypertonic saline solutions or excessive use of sodium bicarbonate may also cause hypernatremia.1,2

**Assessment**

Clinical manifestations of hypernatremia include muscle weakness and twitching, personality changes, agitation, hallucinations, and decreased level of consciousness. Cardiac output is reduced due to decreased myocardial contractility, which may lead to heart failure.1 Because of this, assess capillary bed refill time of nail beds (normal is within 3 seconds). Patients experiencing hypernatremia may also experience signs and symptoms associated with hypovolemia, including dry/sticky mucus membranes, intense thirst, flushed skin, oliguria, tachycardia, postural hypotension, and fever. Dehydration with hypernatremia is a primary reason for behavior changes in older adults. Because blood vessels become fragile, severe hypernatremia may also result in intracerebral, subarachnoid, and subdural hemorrhage and permanent brain damage.1,2,5

**Treatment**

As with hyponatremia, the key is to treat the underlying cause of hypernatremia and restore fluid balance. The usual treatment is to increase salt-free fluids either orally or I.V. (with isotonic or hypotonic solutions) to normalize serum sodium levels. Rapid reduction of sodium may cause the serum osmolality in brain tissue to be temporarily greater than that of plasma osmolality, causing dangerous cerebral edema, so take care to administer I.V. fluids slowly as prescribed. Hypotonic sodium solutions (such as 0.45% sodium chloride solution) are considered safer than D5W because they allow a more gradual reduction of serum sodium, reducing the risk of cerebral edema.1,2

**Nursing implications**

Due to the possibility of mental status changes, initiate and maintain appropriate safety measures. Monitor serum sodium levels, hematocrit, and hemoglobin. Monitor intake and output and assess the patient for subtle changes in mental status. Educate the patient and family about the role of sodium in the body and what a high blood level means. Teach the signs and symptoms of hypernatremia and instruct the patient and family to immediately report any changes in mental status. Tell the patient to keep track of oral intake so that you can document intake accurately.

**Potassium: Many metabolic functions**

The normal range of potassium is 3.5 to 5.0 mEq/L. The major cation in intracellular fluid, it plays an important role in cellular metabolism, especially in protein and glycogen synthesis and in the enzymatic processes necessary for cellular energy. In addition, it aids in maintaining cellular electrical neutrality and osmolality. Potassium is critical to many body functions, including acid-base balance, nerve impulse conduction, maintenance of normal cardiac rhythm, and skeletal and smooth
muscle contraction. Potassium must be consumed daily because the body has no effective way to store it.

Potassium levels are regulated by kidney excretion, with remaining excess levels removed through the feces and sweat. Additionally, potassium levels are affected by the sodium-potassium pump and pH levels. The sodium-potassium pump regulates extracellular potassium levels by pumping sodium out of cells and allowing potassium to flow back into cells.

In the kidneys, sodium and potassium have a reciprocal relationship. Acting on the distal tubules, the hormone aldosterone triggers potassium excretion and resorption of sodium. High extracellular potassium levels cause the kidneys to excrete more potassium. However, the kidneys don’t have an effective mechanism to detect a potassium deficit, and they continue to excrete potassium even when levels are low.¹,⁶

Acid-base changes affect serum potassium levels because nearly all of the body’s potassium is freely exchangeable with hydrogen ions. Thus, shifts in hydrogen ions can change serum potassium levels. Acidosis causes an increase in hydrogen ions in extracellular fluid; to maintain pH, some hydrogen ions shift to intracellular fluid. To maintain intracellular electrical neutrality, some potassium ions shift to the extracellular fluid, which may cause hyperkalemia. Conversely, in alkalosis, more hydrogen ions are present in the intracellular fluid, so some hydrogen ions move to the extracellular fluid to buffer and maintain pH. This again produces inequities in the intracellular ion electrical charges; potassium ions compensate by moving from the extracellular fluid to the intracellular fluid, causing hypokalemia.

**HYPOKALEMIA**

Defined as serum potassium blood levels below 3.5 mEq/L, hypokalemia is a common electrolyte imbalance, and GI loss is the most common cause. Almost all of the body’s potassium content is freely exchangeable. So, when extracellular potassium is lost from conditions such as diarrhea, vomiting, diaphoresis, laxative or diuretic overuse, gastric suctioning, or alkalosis, the body compensates by shifting potassium from the intracellular space. In addition, potassium can be lost through kidney excretion in association with metabolic alkalosis and hyperaldosteronism.

Clinical manifestations of hypokalemia develop when the serum potassium level drops below 3 mEq/L. Potassium deficiency can cause alterations in normal cardiovascular and neuromuscular function. Respiratory function can be compromised due to respiratory muscle involvement. Prolonged hypokalemia impairs the kidneys’ ability to concentrate urine, resulting in polyuria and urine with a low specific gravity.⁴ Hypokalemia also depresses insulin release from the pancreas, resulting in glucose intolerance. Patients with

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**Potassium imbalances: Telltale ECG changes**

- **Normal**
  - PR prolongation
  - Delay in AV node
  - P wave
  - T wave
  - U wave

- **Hypokalemia**
  - PR prolongation
  - Depressed ST segment
  - Flattened T wave
  - Prominent U wave

- **Hyperkalemia**
  - Widening of QRS
  - Peaked T wave
  - PR prolongation
  - Low-amplitude P wave

severe hypokalemia can die from cardiac or respiratory arrest.1,2,6

Assessment
Focus on the cardiac, neuromuscular, GI, and urinary systems, as these are the ones most affected by a potassium imbalance.
- Cardiac assessment includes assessing the apical pulse for irregularities (especially new-onset), and monitoring the ECG for flattened T waves, depressed ST segments, U waves, and dysrhythmias.7 (See Potassium imbalances: Telltale ECG changes.) Closely monitor vital signs, especially if the patient is hypotensive.
- Neuromuscular assessment includes monitoring for diminished or absent deep tendon reflexes and myalgias. Other signs and symptoms involving this body system include skeletal muscle cramps and weakness, progressing from mild lower extremity weakness to more generalized paresis, fatigue, and paralysis. Monitor respiratory function, which may be impaired due to respiratory muscle involvement.
- Common GI effects include nausea/vomiting, anorexia, constipation, hypotonic or absent bowel sounds, or paralytic ileus. Assess bowel sounds, bowel movement patterns, and character and consistency of stools.
- Because prolonged hypokalemia affects the kidneys’ ability to concentrate urine, closely monitor intake and output.

Maintain safety measures and monitor other serum electrolytes. When indicated, digoxin levels should also be followed closely. The loss of potassium in extracellular fluid increases myocardial sensitivity to digitalis by potentiating digoxin’s effects. Signs of digoxin toxicity include anorexia, nausea, vomiting, headache, malaise, and cardiac dysrhythmias. The patient may report yellow-green halos in his field of vision. If you suspect digoxin toxicity, temporarily withhold the drug, notify the prescriber, and adjust the dosage as prescribed.

Treatment
Focus on restoring a normal potassium level in the body while treating the underlying cause of the deficiency and monitoring for potential complications. Patients are placed on a high potassium diet. Good sources of potassium include lean meats, whole grains, green leafy vegetables, potatoes, beans, and fruits such as bananas and oranges. Depending on the severity of the imbalance, oral or parental replacement potassium therapy may also be prescribed.

Administer I.V. potassium with extreme caution as prescribed and according to facility policy. It should be given slowly using an infusion pump while the patient is on continuous ECG monitoring. Never administer potassium I.V. push or by I.M. injection. Closely watch the I.V. site for tenderness and erythema because potassium is very irritating.

When administering oral potassium, dilute it in water or fruit juice and give with or before meals to decrease the risk of GI upset. Be alert for signs and symptoms of hyperkalemia (as discussed in the next section), such as dysrhythmias and other ECG changes or muscle pain.

Nursing implications
Monitor patients for worsening hypokalemia as well as signs and symptoms of hyperkalemia during replacement therapy. Monitor vital signs frequently and carefully assess patients on digoxin for signs of toxicity.

Instruct the patient and family to inform the staff of muscle pain and weakness, and to call for assistance if the patient develops chest discomfort or palpitations. Before discharge, teach the patient about foods that are high in potassium, but caution him not to take potassium supplements unless his healthcare provider recommends it. If a potassium supplement is prescribed, teach the patient how to take it. Warn the patient not to alter the dose independently.

HYPERKALEMIA
Defined as a serum potassium level above 5.0 mEq/L, hyperkalemia is most often related to renal failure. Hyperkalemia is unlikely to develop if renal function is adequate. Other causes include excessive administration of oral or parental potassium preparations to correct a deficit, excessive use of salt substitute products containing potassium, hypoaldosteronism, and Addison disease. Administration of medications such as potassium chloride, angiotensin-converting enzyme inhibitors, nonsteroidal anti-inflammatory drugs, and potassium-sparing diuretics are often contributing factors, especially in conjunction with renal insufficiency.

In addition, events causing potassium to move from the intracellular to the extracellular space, such as severe infections, burns, traumatic crush injuries, and metabolic acidosis, may
create an excess in serum potassium levels.

Be aware of conditions that may cause falsely high levels of serum potassium. The use of a tight tourniquet around an exercising extremity (as when a patient opens and closes a fist while giving blood), hemolysis of blood before analysis, marked leukocytosis, and thrombocytosis may cause a fictitious or pseudo hyperkalemia.

The most clinically significant manifestation of hyperkalemia is its effect on myocardial tissue. Increased serum potassium levels can cause disturbances in cardiac conduction. ECG changes include peaked T waves, a prolonged PR interval, and widening of the QRS complex. At any point along this progression, ventricular dysrhythmias and cardiac arrest are possible.

Assessment
As with hypokalemia, patients with hyperkalemia can exhibit changes in their cardiac status. Other adverse effects of hyperkalemia include skeletal muscle weakness and paralysis, respiratory muscle paralysis, speech deficits, nausea, diarrhea, and intestinal colic.1,2,6

Treatment
Determine the underlying cause of the high potassium level and intervene to bring serum levels back to the normal range. Mild hyperkalemia may be treated simply by restricting intake of dietary potassium and potassium-containing medications, as prescribed. More serious hyperkalemia may be treated with cation-exchange resins (such as sodium polystyrene sulfonate) either orally or rectally as a retention enema. Administration of I.V. diuretics such as furosemide may be prescribed as well.

Emergency measures may include I.V. administration of calcium chloride or calcium gluconate. Although it doesn’t directly affect the serum potassium levels, calcium antagonizes the toxic effects of hyperkalemia at the cellular level. Closely monitor BP because rapid administration may cause hypotension. Bradycardia is an indication to stop the infusion.

I.V. administration of sodium bicarbonate may be prescribed to help alkalinize the plasma and temporarily shift potassium into cells. The sodium ions provided by sodium bicarbonate help to antagonize the cardiac effects of the potassium. I.V. regular insulin and hypertonic dextrose (D50) also will cause potassium to shift into the cells.

Though effective in countereacting hyperkalemia, calcium, sodium bicarbonate, and insulin/glucose administration are all considered temporary treatments, and should be used in conjunction with other treatments aimed at decreasing the serum potassium levels. Extreme hyperkalemia not controlled with cation-exchange resins and the above additional treatments may require hemodialysis or peritoneal dialysis to remove the excess potassium.8

Nursing implications
Take appropriate precautions related to muscle weakness and potential cardiac dysrhythmias, such as continuous cardiac monitoring and frequent auscultation of heart sounds. When you monitor vital signs, take an apical pulse. Monitor all electrolyte levels and intake and output.

If insulin or sodium bicarbonate is prescribed, administer it I.V. as prescribed. Patients receiving sodium bicarbonate are at risk for developing hypernatremia, so watch for signs and symptoms such as muscle weakness and mental status changes. Monitor patients with heart failure who are receiving sodium polystyrene sulfonate. This drug contains sodium, which can exacerbate fluid overload.3

Ask patients to notify staff if they experience chest discomfort, shortness of breath, or any worsening symptoms. Teach them about the role of potassium in the body, what high levels of this electrolyte mean, and what signs and symptoms to report. Ask patients to keep track of fluid intake so that you can accurately document intake.

Maintaining a healthy balance
Because electrolyte imbalances can affect patients with various medical and surgical conditions, assessing patients for potential problems and monitoring lab values is part of routine nursing care. But the importance of your assessment findings is far from routine. Watching for trends and knowing early signs and symptoms of electrolyte imbalances will help you intervene appropriately to head off potentially serious complications.

REFERENCES

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