Monitoring a patient’s fluid and electrolyte status is critical to nursing assessment. When fluctuations in homeostasis occur, the patient’s acuity may quickly change. Abnormal potassium levels may lead to a patient crisis and even death. While searching for the underlying cause of the fluid and electrolyte imbalance, prompt intervention is necessary. This article describes common sources of potassium imbalances, identifies corrective actions for abnormal levels, and provides key points for nurses to consider during the process of restoring balance.

**The role of potassium**

Potassium is a positively charged ion and functions as an irritant to neuromuscular junctions. Neuromuscular junctions are communication points for motor neuron axons and muscle fibers, ultimately resulting in contraction or relaxation of skeletal, smooth, and cardiac muscles. In addition, potassium affects nerve impulse transmission, acid-base equilibrium, enzymatic action, intracellular fluid (ICF) tonicity, and renal function. Ninety-eight percent of potassium is located in the ICF, and the remaining 2% is located outside of cells in the extracellular fluid (ECF).

A normal plasma sample of potassium measures only 3.5 to 5 mEq/L. Maintaining a normal potassium balance depends on adequate intake of potassium-containing foods, the movement of electrolytes between the ICF and ECF compartments, and the renal system being able to excrete any excess potassium. The body rids excess potassium through the kidneys, the bowel, and sweat. Renal function is critical, however, because the kidneys filter 80% of the body’s excess potassium.

Potassium also serves an important role in preserving the normal depolarization and repolarization of the heart; abnormal potassium levels can lead to cardiac dysrhythmias. (See ECG changes that can be noted with low versus high potassium levels.) Although the ECF contains a small percentage of the body’s total potassium, nurses must be proactive in monitoring and correcting potassium imbalances, as even slight changes may result in the development of significant clinical problems.

**Reviewing the sodium-potassium pump**

The initial article in this series (“Understanding hyponatremia,” in the March issue of Nursing2013 Critical Care) focused on sodium. Having a clear understanding of how sodium and potassium work together to maintain homeostasis is important, and the sodium-potassium pump serves as the body’s mechanism for maintaining this balance. The site of action is in the cellular membranes and involves a continuous flux of potassium and sodium from the two primary fluid compartments. The pump essentially works to counteract the results of diffusion. Remember that water chases sodium. If sodium becomes more abundant inside the cells, the increase in fluid causes swelling. Through energy from adenosine triphosphate, sodium is moved...
from the ICF to the ECF, and simultaneously, potassium is pumped from the ECF into the ICF.\textsuperscript{2} Under normal conditions, fluid and electrolyte balance is achieved.

**Hypokalemia causes and patient assessment**

Hypokalemia is a common problem that occurs when the serum potassium level is less than 3.5 mEq/L.\textsuperscript{6} Levels between 2.5 and 3 mEq/L are considered moderate, and levels below 2.5 mEq/L are considered severe.\textsuperscript{6,7} Potassium is a vital electrolyte, and an inadequate level that goes untreated may quickly lead to a life-threatening condition.

Potassium loss may result from many causes, but the most common is medications that increase renal excretion of potassium, such as diuretics, corticosteroids, and digitalis preparations.\textsuperscript{7} Closely monitor the patient’s lab results when the patient is taking medications that directly affect the potassium level. Remember that patients with hypokalemia are at increased risk of digitalis toxicity.\textsuperscript{1,3} Inadequate potassium in one’s diet, diarrhea, vomiting, gastric suctioning, hemorrhage, an excess of insulin, and conditions that cause hypersecretion of aldosterone from the adrenal glands may also result in increased renal excretion of potassium.\textsuperscript{7,8}

Metabolic alkalosis is another possible cause of hypokalemia.\textsuperscript{2,8} Serum pH and potassium levels have an inverse relationship. When metabolic alkalosis occurs, the body’s compensatory mechanism causes potassium levels to decrease in response to increases in serum pH, and hydrogen ions move from the ICF to the ECF in an attempt to preserve normal pH. Additional potassium then moves into the ICF, resulting in serum hypokalemia. Patients who lose gastric fluid from vomiting or gastric suctioning may develop metabolic alkalosis and a declining potassium level.\textsuperscript{2,7}

The patient with hypokalemia may have a myriad of complaints, such as fatigue, muscle weakness (particularly in the legs), leg cramping, paresthesias, and constipation. Further assessment may reveal decreased deep tendon reflexes, dilute urine, excessive thirst, and ECG changes, including dysrhythmias.\textsuperscript{3,5} Clinical manifestations of hypokalemia may not become apparent until the level decreases below 3 mEq/L.\textsuperscript{8} Remember that the patient’s vague complaints may not always include signs or symptoms that suggest a potassium imbalance or cardiac involvement. Until lab and ECG results are assessed, a potentially serious potassium imbalance may go unrecognized. Check the patient’s magnesium level as well, as low magnesium levels may lead to increased renal excretion of potassium.\textsuperscript{3} The potential for fatal dysrhythmias, respiratory arrest, and coma increases as the patient’s potassium level decreases.\textsuperscript{7} Use your knowledge and critical thinking skills when considering assessment findings and anticipating interventions.

**Hypokalemia interventions**

Patients with hypokalemia should be given supplemental potassium; oral or I.V. forms may be used in the clinical setting. Dietary changes (see Examples of high-potassium-containing foods) or over-the-counter supplements may be adequate in some cases. Advise patients who use salt substitutes to use them cautiously, as each teaspoon may contain up to 60 mEq of potassium, and excess use could lead to hyperkalemia.\textsuperscript{3} Remember that whether administered via the oral or I.V. route, supplemental potassium is an irritant. Oral supplements may irritate the gastrointestinal (GI) mucosa and may cause discomfort, abdominal distention, and GI bleeding.\textsuperscript{3} Medication is usually prescribed by the healthcare provider to help prevent such problems.

Also consider the patient’s swallowing ability. Oral administration, for tablets or solutions, shouldn’t be the supplementation route if the patient has dysphagia. The large size of potassium tablets puts patients at risk for respiratory compromise because of aspiration, choking, and laryngospasm.

Potassium administered via a peripheral I.V. may lead to vessel irritation and pain at the I.V. site and in the extremity used, especially proximal to the I.V. site. Frequently assess the patient’s I.V. site and in the extremity used, especially proximal to the I.V. site. Frequently assess the patient’s I.V. site and in the extremity used, especially proximal to the I.V. site. Frequently assess the patient’s I.V. site and in the extremity used, especially proximal to the I.V. site. Frequently assess the patient’s I.V. site and in the extremity used, especially proximal to the I.V. site.

### Examples of high-potassium-containing foods\textsuperscript{3,4,7}

- Avocados
- Beans
- Dark leafy greens
- Fruit juices
- Fruits (fresh and dried)
- Grains
- Meats
- Milk
- Potatoes
- Salmon
- Squash
- Yogurt
site and extremity. Potassium is not an I.V.-push or I.V.-bolus medication and must always be administered as a diluted solution.\(^7\) To ensure a consistent administration rate, use an infusion pump. The recommended peripheral venous access administration concentration for potassium is less than or equal to 20 mEq/100 mL with a rate no greater than 20 mEq/h.\(^1,3\) A central venous access administration route is recommended for concentrations greater than 20 mEq/100 mL.\(^3\) Although the pharmacy may prepare small bags of premixed potassium solutions for piggyback administration, liter bags of I.V. solutions with 20 to 40 mEq of potassium are usually included in the unit stock.

Be sure you know your facility’s standard of care for I.V. potassium administration, and take care when selecting the appropriate solution. Consult the healthcare provider if you have concerns about the type of solution prescribed, and teach patients and families about potassium and the precautions to consider.

**Hyperkalemia causes and patient assessment**

Hyperkalemia is defined as a potassium level greater than 5.5 mEq/L. From 5.5 to 6.5 mEq/L is mild hyperkalemia, 6.5 to 7.5 mEq/L is moderate, and levels above 7.5 mEq/L are severe hyperkalemia.\(^4\) The source lab’s normal reference range values may reflect slight variations.\(^2\) Hyperkalemia is a critical finding, and prompt assessment and intervention is paramount for optimal patient outcomes.

A repeat lab test may be wise in some cases, such as for an asymptomatic patient who has no clinical manifestations of hyperkalemia. Perhaps an error occurred during the lab processing, or the phlebotomy was poorly performed. A tourniquet left on the arm for too long or a mishandled specimen tube may cause the potassium level to rise.\(^9\) Remember that trauma to blood cells results in hemolysis, letting potassium leak out of the ICF into the ECF, which raises the serum potassium level.\(^4\)

Dietary increases in potassium usually don’t result in the patient becoming hyperkalemic, as long as renal function is normal. When the kidneys are unable to excrete excess potassium, or the uptake of potassium into the ICF compartment is impaired, hyperkalemia will result.\(^6,8\) For example, patients with renal failure, as well as patients who require dialysis, are challenged with hyperkalemia.\(^4,8\) Uremic toxins and a weakened sodium–potassium pump mechanism contribute to diminished ICF potassium uptake.\(^10\)

Hyperkalemia is common to certain medical conditions; diabetes and rhabdomyolysis are two examples.\(^4\) Diabetes causes insufficient insulin availability and a hyperosmotic state related to increased fluid losses secondary to polyuria, which affects normal uptake of ICF potassium levels. The result is a rise in the serum (ECF) potassium.\(^10\) Rhabdomyolysis is a condition that results in increased serum potassium because of muscle fiber breakdown. Several common causes include trauma and crush injuries, drug abuse, burns, compartment syndrome, and alcoholism.\(^5,8\) When muscle tissue breaks down, myoglobin is released into the blood. Myoglobin is excreted by the kidneys and damages the renal tubules, hindering excretion of potassium.\(^5\) A classic sign of rhabdomyolysis is dark, tea-colored urine. The acid-base balance and inverse relationship between acidosis and potassium must also be recognized. The lower the pH level is, the higher the potassium level.\(^2,3\)

Numerous medications (reportedly 60%) contribute to hyperkalemia by interfering with urinary excretion or by affecting the influx of potassium from the ICF to the ECF.\(^2\) Potassium-sparing diuretics, such as spironolactone and triamterene, are common examples of medications that inhibit potassium excretion via the kidneys, while beta-blockers may affect ICF uptake.\(^1,4\) Remember, too, that many patients choose to take over-the-counter potassium supplements without understanding the potential danger.

Weakness, fatigue, hyperactive bowel sounds, abdominal cramping, skeletal muscle twitching, and palpitations are signs and symptoms of hyperkalemia.\(^1,5,9\) Assessment findings may reveal ECG abnormalities, and in some cases, an elevated potassium level may not be obvious until a dysrhythmia occurs. Dysrhythmias are common with moderate and severe hyperkalemia, and treatment is essential to prevent ventricular fibrillation and cardiac arrest.\(^5,9\)
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Hyperkalemia intervention

Interventions to consider for hyperkalemia include medication administration, continuous cardiac monitoring, and accurate intake and output. Serial ECGs and serum electrolyte assessments are very important, as ECG changes and increases in the potassium level are key indicators for how aggressive treatment must be. Perform frequent vital sign checks and conduct close observation for changes in the patient’s condition. In addition, any potassium supplementation must be discontinued from the medication regime.

While calcium gluconate doesn’t affect potassium level, this medication may be administered to help stabilize the cardiac muscle and lessen the possibility of dysrhythmias. Insulin and dextrose may be infused to reduce serum potassium. Insulin causes potassium to shift to the ICF, lowering the ECF level. The blood glucose level is also reduced, and dextrose must be given to prevent hypoglycemia. Blood glucose monitoring is critical.

Infusing insulin and dextrose is a temporary treatment for acute hyperkalemia because the excess potassium merely moves into the cell. When the insulin/dextrose infusion is discontinued, the potassium will move back into the ECF, increasing the potassium level once again. I.V. or nebulized beta-adrenergic agonists may also be used to cause potassium to move from the ECF to the ICF. In addition to searching for the underlying cause, additional interventions are necessary that will rid the body of excess potassium. A common medication used for this effect is sodium polystyrene (with no sorbitol), which causes potassium to be excreted through the bowel. Sorbital isn’t recommended because of the severe, even fatal, adverse reaction of intestinal necrosis.

Remember that stored blood may contain an increased potassium concentration. Blood cells deteriorate as the storage period increases, releasing the intracellular potassium into the serum. Fresh is best!

Numerous physiologic changes occur with aging and must be considered when focusing on correcting fluid and electrolyte imbalances. Decreased renal function results in greater susceptibility for potassium imbalances in the older adult. With the normal changes and multiple medical conditions that accompany aging, polypharmacy is common. As with all patients, treating potassium imbalances may be challenging and caution must be given to corrective interventions, whether focused upon increased or decreased levels.

Case studies

Case study A

Mr. K is a 62-year-old man with a history of hypertension. He takes a beta-blocker and baby aspirin every day. Mr. K has his mind set that 2013 will be the year for improving his health through diet and exercise. He’s eating more fresh fruits, green leafy vegetables, and has added a glass of 1% milk to the whole grain toast he has each day. So far, he walks around the block four times each morning. Several days ago, he noticed puffiness in his ankles and began experiencing slight muscle cramping in his legs. From the advice of his neighbor, he began taking potassium tablets twice daily to help relieve the discomfort in his legs.

Today, Mr. K is admitted to the hospital with complaints of generalized weakness, fatigue, severe leg cramps, and difficulty urinating. Assessment findings include a BP of 160/82, pulse of 88, 2+ edema in his lower extremities, and a potassium level of 6.5 mEq/L. His ECG findings include tented T waves, and his arterial blood gas (ABG) analysis indicates metabolic acidosis. Given Mr. K’s history and clinical presentation, the underlying cause of his hyperkalemia is apparent.

Mr. K clearly needs patient education; however, the priorities are to stabilize his cardiac status, reduce his serum potassium, and correct his fluid imbalance. Elevated potassium levels increase the risk of cardiac dysrhythmias, and administration of I.V. calcium gluconate will lower the myocardium threshold potential, which helps reduce this risk. As an urgent intervention, regular insulin will aid in potassium reduction by causing increased cellular uptake of potassium. Because insulin will also reduce the blood glucose, dextrose administration will also be necessary. The effect of insulin and glucose may be recognized within 1 hour. Sodium polystyrene (Kayexalate) will also promote a decrease in potassium through bowel excretion, but with a slower onset. Loop or thiazide diuretics may also be used to increase potassium excretion in the urine; however, given Mr. K’s complaint of difficulty urinating, further investigation of his renal function is warranted. Accurate intake and
output, continuous cardiac monitoring, serial ECGs, frequent vital sign checks, and serial serum chemistry levels are essential for assessing the patient’s response to care measures. When you educate Mr. K, provide him with information about dietary needs and medications. He should stop taking the potassium supplements and talk to his healthcare provider for future health guidance.

Case study B
Mrs. T, 48, is admitted to the ED with dehydration. Her husband states that she sees a psychologist related to an eating disorder. Her symptoms include fatigue, lethargy, and nausea. She states that she has vomited several times over the past 2 days and has no appetite. She takes a daily multivitamin.

Assessment findings include a BP of 98/58, pulse of 120, dry mucous membranes, and minimal and concentrated urine output. Her blood work results include the following: sodium 149 mEq/L, potassium 3.2 mEq/L, and glucose 148 mg/dL. Her ECG findings include a slightly flattened T wave, and ABG analysis reveals metabolic alkalosis.

Mrs. T needs ongoing therapy for her patient’s eating disorder, but the current priority is correcting her potassium level and increasing her fluid volume. I.V. potassium and fluids are ordered (0.9% sodium chloride solution with 40 mEq of potassium). I.V. potassium and fluids are ordered (0.9% sodium chloride solution with 40 mEq of potassium). I.V. potassium and fluids are ordered (0.9% sodium chloride solution with 40 mEq of potassium).

Mrs. T’s potassium level to ensure adequate potassium replacement and to reduce the risk of overcorrection during treatment, a common occurrence during hospitalization. While the patient’s ECG reflects an abnormality, continuous cardiac monitoring will track any decline in cardiac status that would result in a more aggressive course of treatment. A repeat ECG may also be obtained. Administer an antietemic as prescribed and encourage Mrs. T to drink clear fluids. Arrange a dietary consult to assess the patient’s nutritional status and to provide recommendations for a dietary plan, taking into consideration her eating disorder.

Staying in balance
Understanding the numerous causes and effects of potassium imbalances can help you anticipate patient care needs. By promptly identifying abnormal potassium levels and implementing corrective measures, you may be able to avert a life-threatening crisis. A patient’s clinical presentation is always a key assessment factor; however, serum blood testing must be performed to confirm electrolyte values. An ECG may help you determine how urgently intervention is needed. You’ll also want to stay up-to-date on medications that can cause potassium excretion or retention, as well as dietary modifications that may affect the body’s potassium level. Finally, recognizing age-related changes and the importance of patient and family teaching can help prevent negative patient outcomes.

REFERENCES

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