In Brief: Hypokalemia
Treva Caraway Ingram and John M. Olsson
*Pediatrics in Review* 2008;29;e50
DOI: 10.1542/pir.29-9-e50

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://pedsinreview.aappublications.org/content/29/9/e50
Hypokalemia

Treva Caraway Ingram, MD
John M. Olsson, MD
Brody School of Medicine
East Carolina University
Greenville, NC

Potassium, the most abundant cation in the intracellular space, is involved in a number of homeostatic processes, including regulation of cellular metabolism and maintenance of intracellular volume and cellular resting membrane potential. Proper functioning of all tissues, especially muscles and nerves, depends on normal concentrations of potassium.

Potassium concentrations are regulated principally by the kidneys, which secrete or reabsorb potassium in response to various signals. Aldosterone is the primary hormone regulating potassium excretion, but other agents and processes also affect it. Glucocorticoids, antidiuretic hormone, high urinary flow rate, and increased sodium delivery to the distal nephron increase potassium excretion; insulin and catecholamines decrease its excretion. Insulin and catecholamines also increase the cellular uptake of potassium.

Hypokalemia is defined as a potassium concentration less than 3.5 mEq/L (3.5 mmol/L). The causes of hypokalemia may be divided into the following categories: prolonged decreased intake, extrarenal losses, renal losses, and transcellular shifts.

Hypokalemia resulting from prolonged decreased intake of potassium is rare because potassium is available in most foods. Generally, reduced intake must coincide with potassium loss for a patient to develop hypokalemia. For example, a patient who has anorexia nervosa may decrease intake of potassium-containing foods while increasing extrarenal losses by using diuretics or laxatives.

The gastrointestinal (GI) tract is a major site of extrarenal potassium loss. Specifically, the most common cause of hypokalemia in children is gastroenteritis with its accompanying fecal loss of potassium. Less commonly, patients who have bulimia and use laxatives may develop hypokalemia. Additional GI causes include aggressive nasogastric suctioning and protracted vomiting. On occasion, excessive sweating may lead to hypokalemia.

Renal losses can result from the use of certain medications or from intrinsic renal disease. Medications that increase urinary potassium loss include diuretics, certain antibiotics (eg, amphotericin, gentamicin, clindamycin), and corticosteroids. Examples of intrinsic renal disease include Bartter syndrome (hyperreninemia, hyperaldosteronism) and renal tubular acidosis (types 1 and 2 lead to hypokalemia as a result of increased urinary potassium loss).

Transcellular shifts of potassium from serum to cells, such as those that occur during episodes of acute alkalosis, can result in low concentrations of serum potassium. For every 0.1 increase in blood pH, the serum potassium may decrease by 0.3 to 1.3 mEq/L (0.3 to 1.3 mmol/L). Because medications such as albuterol and insulin also can cause cellular shifting, hypokalemia may be anticipated when treating children who have asthma exacerbations or when correcting diabetic ketoacidosis (DKA).

In the latter scenario, plasma potassium does not reflect total body depletion because DKA, with its metabolic acidosis and lack of insulin, favors movement of potassium from the intracellular to the extracellular space. Therefore, in early DKA, potassium values may appear to be high or normal, whereas in reality, total body potassium almost always is decreased. As acidosis is corrected and insulin is provided, causing potassium to shift back into the cell, the patient requires ongoing potassium supplementation to maintain normokalemia.

Clinical manifestations of hypokalemia vary with the degree and the rate of potassium loss. Mild potassium deficit manifests as weakness and cramping of skeletal muscles. In more severe depletion, when potassium concentrations are lower than 2.5 mEq/L (2.5 mmol/L), leg, arm, and finally diaphragmatic paralysis may ensue. Other consequences of hypokalemia include constipation or ileus, urinary retention, arrhythmias, and myocardial cell necrosis.

An important adjunct to determining serum potassium values is electrocardiography (ECG). Common ECG findings associated with hypokalemia include...
flattened T waves, depressed ST segments, and prominent U waves. In patients who have underlying heart disease, ventricular fibrillation and torsades de pointes also may occur. Patients taking digoxin are more sensitive to digitalis-related arrhythmias when hypokalemia is present.

Treatment for the child who has hypokalemia depends on the patient’s symptoms and the degree of hypokalemia. In most settings, replacement can be managed less emergently, with oral supplementation using potassium-rich foods or potassium preparations. The regimen is tailored to the cause of the hypokalemia. This approach minimizes the risk of hyperkalemia. Urgent correction of hypokalemia is needed only for patients who are severely affected. In these cases, administration of 0.5 to 1.0 mEq/L per kilogram of potassium chloride intravenously over 1 hour, to a maximum of 40 mEq, is indicated. Those who have acidosis may be supplemented with potassium acetate or citrate salts. Potassium phosphate is indicated if the patient is experiencing hypophosphatemia. When hypokalemia occurs in the setting of dehydration and volume depletion, fluid administration with added potassium chloride generally suffices.
In Brief: Hypokalemia
Treva Caraway Ingram and John M. Olsson
Pediatrics in Review 2008;29;e50
DOI: 10.1542/pir.29-9-e50

Updated Information & Services
including high resolution figures, can be found at:
http://pedsinreview.aappublications.org/content/29/9/e50

References
This article cites 3 articles, 0 of which you can access for free at:
http://pedsinreview.aappublications.org/content/29/9/e50#BIBL

Subspecialty Collections
This article, along with others on similar topics, appears in the following collection(s):
Endocrinology
http://pedsinreview.aappublications.org/cgi/collection/endocrinology_sub

Permissions & Licensing
Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at:
/site/misc/Permissions.xhtml

Reprints
Information about ordering reprints can be found online:
/site/misc/reprints.xhtml