



HYPOKALEMIA AND ACID BASE BALANCE

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Potassium is one of the major ions found in the body. Almost 98% of the body's potassium is intracellular. Potassium is often first thought of in relation to cell membrane potential, where the ratio of intracellular to extracellular potassium is important. The cardiovascular and neuromuscular systems can be severely affected by small changes in the extracellular potassium level. The kidney plays a key role in potassium homeostasis and is also responsible for the removal of excess potassium from the body via the urine.

Normal potassium levels are 3.5-5.5 mEq/L. Blood levels of less than 2.5-3.0 mEq/L are considered moderate hypokalemia. Levels less than 2.5 mEq/L are considered severe hypokalemia. Most of these can and will affect the arterial blood gas.

Epidemiological studies indicate that hypokalemia can be found in as many as 20% of hospitalized patients, but only 4-5% of these are clinically significant. Approximately 14% of outpatients are mildly hypokalemic, with approximately 80% of patients who receive diuretics become hypokalemic.

Causes for extrarenal potassium loss are probably the ones we are most familiar with as respiratory therapists

There are a number of potentially serious problems associated with hypokalemia such as: Neuromuscular manifestations such as fatigue, paralysis, weakness, respiratory muscle dysfunction, and rhabdomyolysis. Gastrointestinal problems include constipation and ileus. There are a number of cardiac arrhythmias and ECG changes that can occur such as prominent U waves, T wave flattening, and ST segment changes.

The causes for hypokalemia can be grouped into four main categories: Spurious hypokalemia, redistribution hypokalemia, extrarenal potassium loss, and renal potassium loss.

Spurious hypokalemia is an artificial decrease in potassium which can occur in two ways. First, if a patient has a very high white count of over 100,000, and a tube of blood drawn from the patient is allowed to sit at room temperature for a prolonged period of time, the white cells may take up the potassium, thus causing a decrease in the plasma portion of the specimen. Also, if the patient receives a dose of insulin before blood is drawn, the insulin could cause a temporary movement of potassium into the blood cells in the tube, thus lowering the potassium level in the serum. This type of movement is rather small in the range of only 0.3 mEq/L or so, however.

Redistribution hypokalemia is caused by the entry of potassium into cells. Only a small amount of total body potassium is located in the extracellular compartment. As a result, a small shift of potassium from the extracellular space to the intracellular space can

cause a change in plasma potassium concentration. During this shift, alkalosis may occur. A rough guide to the degree of shift is that the serum potassium concentration falls by about 0.3 mEq/L for each 0.1 increase in pH. Alkalosis often results from disorders that deplete total body potassium. As a result, true depletion of total body stores is usually present as well as a redistribution hypokalemia when metabolic alkalosis is present.

Increased beta2-adrenergic activity is an important one for therapists to keep in mind. Treating asthmatic patients, particularly with prolonged continuous therapy, may cause a transient shift of potassium into cells and decrease the serum potassium concentration. This occurs because of an increase in activity of sodium-potassium ATPase. This also occurs in situations where there is sympathetic hyperresponsiveness, such as myocardial infarction or delirium tremens or in cases of major head trauma. A potassium shift into the cells may also result from another common respiratory drug, theophylline. This generally occurs only during theophylline toxicity (>20 mg%). The mechanism for this is unknown; however, this hypokalemia may aggravate serious arrhythmias that sometimes occur in severe theophylline toxicity. Hypokalemia has also been seen within a few days or so after factor replacement therapy for severe megaloblastic anemias.

Causes for extrarenal potassium loss are probably the ones we are most familiar with as respiratory therapists. Severe diarrhea results in loss of potassium with HCO₃⁻ resulting in hypokalemia and metabolic acidosis. Chronic laxative abuse can result in severe potassium depletion and metabolic alkalosis. Sweat has a potassium concentration of roughly 9 mEq/L; heavy athletic training or hard exercise on a hot, humid day may produce up to 12 L/day of sweat and significant potassium deficits. This could be an initiating event in cardiac arrhythmias. Fasting or inadequate intake of fluids generally only result in a moderate potassium depletion because the kidneys normally have a very good potassium conservation mechanism.

Renal potassium depletion conditions also tend to be associated with acid-base disorders. It is useful to classify the various causes of renal potassium loss according to whether they typically occur together with metabolic acidosis, metabolic alkalosis, or have no specific acid-base disorder. Common causes of renal potassium depletion with metabolic acidosis are diabetic ketoacidosis, carbonic anhydrase inhibitor therapy and renal tubular acidosis, type I if it is distal or type II if it is proximal.

Metabolic alkalosis is usually associated with hypokalemia since conditions that cause metabolic alkalosis also lead to potassium depletion. In many types of metabolic alkalosis, the excess



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Evidence-Based Medicine... *Continued from previous page*

Too, where evidence-based medicine gets its evidence raises some serious questions. Remember that "evidence" is derived after anecdotal results have been published and it seems that many current medical and surgical practices lack substantial evidence in support of their use. There may be tons of excellent research out there, but if it doesn't get published, it may as well not exist. The types of trials considered "gold standard" (i.e. randomized double-blind placebo controlled trials) are prohibitively expensive. Funding sources are the final determinant in what and who gets investigated. There are certain groups that have historically been under-represented e.g. racial minorities and people with coexistent disorders. And of course, research studies are notoriously subject to conflicts of interest and bias.

Does evidence based medicine have its own evidential base? Ironically, for a topic which speaks to the best evidence, there's not a whole lot evidence – best or otherwise - that EBM actually works.

Whether you're a true believer or not, you can bet you'll be hearing more and more on this subject as time goes on - simply because so many have jumped on the EBM bandwagon.

This is no longer a subject just for docs. Evidence-based thinking is now being tied to nursing, the public health community and to the education of allied health practitioners.

What role if any, EBM should play in the teaching and learning of nursing and allied health students is currently a hot topic in education circles. It will be the topic for another Focus article.

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HCO₃⁻ acts as a poorly reabsorbable anion and "carries" more sodium to the collecting tubule, leading to increased sodium-potassium exchange and urinary potassium loss.

Recovery from acute renal failure, postobstructive diuresis, and osmotic diuresis can all lead to renal potassium loss. Magnesium depletion is a very important cause of renal potassium loss. It is difficult to correct the potassium loss until the magnesium deficit is corrected first. If magnesium depletion is not corrected, urinary potassium loss will continue despite large replacement doses of potassium.

Antibiotics may also be culprits in potassium regulation. Penicillins act as poorly reabsorbable anions, which thereby increase distal sodium delivery and sodium-potassium exchange. Gentamicin and cisplatin have direct tubular toxic effects that induce potassium loss.

Treatment of hypokalemia can be done orally or intravenously. A variety of salts of potassium are available for oral administration; however, potassium chloride is used most often. In type I and type II renal tubular acidosis and in diarrhea, potassium bicarbonate or potassium citrate is used.

Intravenous administration of potassium is used for profound, life-threatening hypokalemia and in patients who are unable to tolerate oral administration. Potassium is very irritating veins and must be given slowly over time. There is also an increased risk of acute hyperkalemia with the intravenous route and the patient must be closely monitored.

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