Hyperkalemia: A Potential Silent Killer

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Hyperkalemia is a common, silent, and potentially lethal clinical condition. The pathophysiologic factors that lead to hyperkalemia are well understood, and the treatment is straightforward. Clinical management requires exclusion of pseudohyperkalemia, assessment of the urgency for treatment, and institution of appropriate therapy. Long-term treatment requires identification of the etiology and prevention of recurrence.

Pathophysiology

Hyperkalemia develops when the regulation between potassium intake and excretion or the distribution between intracellular and extracellular potassium is disturbed. Figure 1 summarizes potassium distribution and movement in the body.

The vast majority of total body potassium is present in the intracellular fluid (1). The ubiquitous Na⁺-K⁺-ATPase transports potassium from the extracellular space into the intracellular space, against its electrochemical gradient. Estimates of total body potassium suggest that approximately 98% of total body potassium is present in the intracellular space, with 2% in the extracellular fluid (2,3).

Potassium Intake

The primary means of potassium intake is through food (1). Although all foods contain potassium, the relative amounts of potassium differ greatly. Fruits and vegetables tend to have the highest concentrations of potassium. A commonly overlooked dietary potassium source is salt substitutes; potassium chloride is the primary constituent of many salt substitutes. Another source of dietary potassium is enteral nutrition supplements. Table 1 summarizes the potassium content of a number of enteral food sources. Patients with renal failure receiving total nutritional support through enteral nutritional supplements frequently will develop hyperkalemia. Potassium is also a common component of hyperalimentation fluids. The potassium concentrations usually recommended for hyperalimentation fluids may be excessive for patients with renal insufficiency; close monitoring and frequent reevaluations may be necessary for this patient population.

Potassium Excretion

The second component of renal potassium homeostasis is excretion. Under normal conditions, 80 to 90% of potassium is eliminated via renal excretion. The colon is responsible for the majority of the remaining potassium excretion (7,8). Although colonic potassium secretion is regulated by physiologic stimuli, the absolute magnitude of colonic potassium secretion is sufficiently low that inadequate potassium secretion is not a cause of hyperkalemia. Sweat contributes quantitatively little to potassium elimination, although in hot climates and with exertion the amount of potassium lost in sweat may be significant, and may lead to hypokalemia.

Renal potassium elimination is regulated predominantly in the collecting duct. Although potassium is freely filtered by the glomerulus (9), approximately 90% of filtered potassium is reabsorbed by the proximal tubule and the loop of Henle (10,11). Potassium delivery to the distal convoluted tubule is relatively constant even in the face of widely varying systemic potassium levels (12,13). Figure 2 summarizes the major potassium transport mechanisms in the collecting duct. The principal cell secretes potassium, whereas the intercalated cell...
reabsorbs potassium (14,15). A basolateral Na\(^+\)-K\(^+\)-ATPase transports potassium against its electrochemical gradient from the peritubular space into the principal cell cytoplasm (16). Under most circumstances, cellular potassium is secreted into the luminal fluid, down its electrochemical gradient, via an apical potassium channel (17,18). An apical KCl cotransport mechanism may also contribute to potassium secretion (19,20). Whether this mechanism represents a KCl cotransporter or parallel K and Cl transporters is unknown.

Several factors are known to regulate principal cell potassium secretion. The mineralocorticoid aldosterone, via binding to the mineralocorticoid receptor, plays a central role in stimulating cortical collecting duct potassium secretion (21–23). Arachidonic acid metabolites are an intracellular signaling molecule regulating potassium secretion (24). Urine flow rates also regulate potassium secretion (25); however, oliguria is insufficient to yield hyperkalemia unless renal insufficiency is also present.

In addition to secreting potassium, the collecting duct also reabsorbs potassium (26). Collecting duct intercalated cells possess an apical H\(^+-\)K\(^+\)-ATPase ion pump (27–30). This transporter uses ATP as an energy source for proton secretion and potassium reabsorption (31,32). Potassium that enters can either recycle across the apical membrane via a barium-sensitive transporter, most likely a potassium channel, or exit across the basolateral membrane, also via a barium-sensitive mechanism (33). Net potassium reabsorption is determined by a variety of factors, including apical H\(^+-\)K\(^+\)-ATPase activity and the relative permeabilities of the apical and basolateral barium-sensitive transporters to potassium exit.

Under normal conditions, the kidney can excrete large quantities of potassium. The daily ingestion of 400 mEq of KCl, which is severalfold greater than the usual daily intake, increases serum potassium on average by less than 1 mEq/L if renal function is normal and potassium excretion mechanisms are intact (34). Under almost all conditions, hyperkalemia not due to redistribution between the intra- and extracellular compartments is related, at least in part, to impaired renal potassium excretion. This may be due to either renal insufficiency, which decreases the number of nephron units available for potassium secretion, or to factors that impair the rate of collecting duct potassium secretion.

The colon also secretes small amounts of potassium. The mechanisms through which this occurs are less well defined, but similar to the kidney. Some regulation of colonic potassium secretion occurs, particularly in renal insufficiency, although the absolute amounts of potassium secretion remain low.

**Adverse Effects**

Hyperkalemia can cause adverse effects that range from subtle and difficult to detect to those that are life-threatening. The majority of these effects are related to effects of potassium on cellular membrane potential or voltage. The primary determinant of cell membrane potential in most cells is the ratio of intracellular to extracellular potassium concentration. Normally, intracellular potassium concentration is approximately 120 mEq/L, whereas extracellular potassium concentration is approximately 4 mEq/L. The contribution of potassium to resting membrane potential is related to this ratio of intracellular to extracellular potassium; thus, small changes in extracellular potassium can result in large changes in the intracellular to extracellular potassium ratio, and hence large changes in resting membrane potential. Resting membrane potential is important in all electrically active cells, including neurons, voluntary muscles, and involuntary muscles.

The most prominent effect of hyperkalemia is on the myocardium (35–38). Decreases in resting membrane potential decrease myocardial cell conduction velocity and increase the rate of repolarization. The decreased conduction velocity leads
Figure 2. Summary of potassium transport in the collecting duct. The principal cell secretes potassium via an apical potassium channel in concert with basolateral Na\(^+\)-K\(^+\)-ATPase. An apical KCl cotransporter may also be present in the principal cell. The A-type intercalated cell (A cell) and the B-type intercalated cell (B cell) both reabsorb potassium via an apical H\(^+\)-K\(^+\)-ATPase in concert with a peritubular barium-sensitive mechanism, most likely a potassium channel. An apical barium-sensitive transporter, most likely a potassium channel, allows potassium recycling when necessary.

the progression from benign to lethal arrhythmias in hyperkalemia is unpredictable, and the presence of any EKG findings of hyperkalemia should be considered a medical emergency. Treatment methods are described below.

Hyperkalemia also affects other cells throughout the body. Skeletal muscles are particularly sensitive to hyperkalemia, resulting in increased weakness and fatigue. This may be related to the importance of membrane potential for normal contraction of skeletal muscles. Smooth muscles are also sensitive to hyperkalemia; hyperkalemia has been reported to cause severe respiratory depression (39).

Evaluation

The evaluation of hyperkalemia involves verifying that hyperkalemia is present in vivo (and is not an artifact of the in vitro measurement of serum potassium), determining the severity of the hyperkalemia and the rapidity of treatment needed, and correcting the condition(s) that led to the hyperkalemia.

The plasma potassium level may be artificially high in some conditions and not reflect actual in vivo potassium levels, a condition known as “pseudohyperkalemia.” Potassium levels are measured typically in blood that has been allowed to clot, and then centrifuged to obtain the serum. Potassium release from any of the cellular elements of blood can artificially elevate the serum potassium level.

The most common cause of pseudohyperkalemia is hemolysis, and this is usually easily noted in the laboratory due to a pink tinge to the plasma resulting from release of hemoglobin from damaged red blood cells. Centrifuging the specimen before the clot has completely formed predisposes the red blood cells to membrane damage, leading to potassium leakage. Alternatively, an excessively tight tourniquet surrounding an exercising extremity (e.g., opening and closing a hand) can increase plasma potassium by more than 2 mEq/L (40).

Potassium release from cells other than red blood cells can also cause pseudohyperkalemia. Excessive numbers of either leukocytes (41-43), greater than 70,000/cm\(^3\), or platelets (44-46), greater than 1,000,000/cm\(^3\), also can lead to pseudohyperkalemia. The degree of elevation of serum potassium is related to the severity of the leukocytosis or thrombocytosis, and can occur at clinically frequent levels of thrombocytosis; 34% of patients with reactive thrombocytosis and platelet counts between 500,000/cm\(^3\) and 1,000,000/cm\(^3\) exhibit pseudohyperkalemia, with a significant correlation between the degree of pseudohyperkalemia and the platelet count (47).

Verifying that pseudohyperkalemia is not present is important if there is a family history of hyperkalemia or if conditions associated with extreme leukocytosis or thrombocytosis are present to avoid unnecessary treatment.

There are rare reports of pseudohyperkalemia from other causes. It has been described in association with rheumatoid arthritis (48) and mononucleosis (49). Occasional families have abnormal red blood cell membrane potassium permeability, which leads to excessive potassium leakage rates and pseudohyperkalemia (50-52).

Pseudohyperkalemia can be excluded by simultaneously
measuring plasma and serum potassium concentrations. Plasma potassium can be measured by obtaining a heparinized blood specimen, either in a "green-top" tube or in a "blood-gas syringe." If the serum potassium is abnormal and exceeds the plasma potassium by more than 0.3 mEq/L, then pseudohyperkalemia should be diagnosed, and all further potassium measurements should be made on the basis of the plasma potassium value.

Several classification methods have been developed for the evaluation of "true" hyperkalemia. We recommend one that is based on the mechanisms that govern potassium homeostasis, but utilizing common clinical principles. In this schema, hyperkalemia results from either excess intake, redistribution between the intra- and extracellular fluid compartments, or inadequate renal excretion. A careful history and physical examination is sufficient to differentiate most causes.

**Excess Intake**

Excessive potassium ingestion is an infrequent cause of hyperkalemia without other contributing factors. As described above, the normal kidney can excrete hundreds of milliequivalents of potassium daily (34). However, if renal potassium excretion is impaired, whether through drugs, renal insufficiency, or other causes, then excess potassium intake can produce hyperkalemia. Common causes of hyperkalemia are potassium supplements and salt substitutes. As many as 4% of patients receiving potassium chloride supplements develop hyperkalemia (53). Typical salt substitutes contain 10 to 13 mEq of potassium per gram, or 200 mEq per tablespoon (54). Many enteral nutrition products contain 40 mEq/L KCl or more; administration of 100 ml/h of such products can result in a potassium intake of approximately 100 mEq per day. Some studies estimate that 50% of all cases of hyperkalemia are related to potassium supplements (46,55–57).

** Redistribution**

The vast majority of total body potassium is located in the intracellular fluid compartment; even small changes in the distribution between intra- and extracellular compartments can result in marked hyperkalemia. Several common clinical conditions are known to cause redistribution. These include acidosis, membrane-depolarizing anesthetics, and extracellular hyperosmolarity if due to "effective osmols."

Metabolic acidosis due to mineral acids, such as HCl or NH₄Cl, is commonly associated with hyperkalemia. In contrast, metabolic acidosis due to organic acids, such as β-hydroxybutyric acid or lactic acid, is an infrequent cause of hyperkalemia (58,59). The contrasting effects of organic and mineral acids on plasma potassium are attributed to differences in cellular potassium release. Hydrochloric acid, but not lactic acid or β-hydroxybutyric acid, causes cellular potassium release. Mineral acids are largely dissociated and cause intracellular acidification by electrogeneric proton uptake, which results in membrane depolarization and a more favorable gradient for conductive potassium exit. In contrast, organic acids are incompletely dissociated in solution and relatively permeable in the undissociated state across cell membranes. This results in cellular uptake with subsequent dissociation to protons and the weak base (60,61). Because this acid uptake step predominately involves diffusion of a neutral molecule, membrane potential is largely unaffected, which prevents conductive cellular potassium exit.

In practice, the factors that determine whether hyperkalemia occurs in response to acidosis are much more complicated than just the nature of the acid load. A potassium-reabsorbing H⁺-K⁺-ATPase is present in the apical membrane of collecting duct intercalated cells, and contributes to a significant component of acid secretion and potassium reabsorption (31,32). Stimulation of H⁺-K⁺-ATPase by acidosis increases potassium reabsorption across the apical membrane (27,28,30).

This results in net potassium reabsorption if potassium exits basolaterally, and can contribute to the development of hyperkalemia. One factor known to regulate potassium exit mechanisms present at the apical and basolateral membranes is the prior potassium intake. Increased dietary potassium loads increase potassium recycling across the apical membrane, thereby decreasing net potassium reabsorption, whereas dietary potassium restriction enhances potassium exit basolaterally and potassium reabsorption (33).

Acidosis inhibits potassium secretion in addition to stimulating potassium reabsorption. Both metabolic and respiratory acidosis cause intracellular acidosis, and intracellular acidosis decreases the open probability (Pₒₒ) of collecting duct apical potassium channels (62,63), which should decrease potassium secretion. Chronic acidosis stimulates ammoniagenesis, and increased extracellular ammonia levels inhibit collecting duct potassium secretion. This effect may be due to ammonium-mediated inhibition of sodium reabsorption (64) and the resulting gradient for potassium secretion, or it may reflect direct regulation of the apical potassium channel by ammonium (65).

Another cause of redistribution-induced hyperkalemia is hyperosmolarity. Increases in extracellular osmolality, when caused by effective osmoles, can increase extracellular potassium concentration by 1 to 2 mEq/L or more (66–69). Mannitol is the most commonly administered exogenous osmole, whereas hyperglycemia, in the absence of sufficient insulin or insulin responsiveness, is the most common endogenous cause. The increase in extracellular osmolality, when caused by effective osmoles, is attributed to cell shrinkage, which increases intracellular potassium concentration and stimulates potassium exit. Ineffective osmoles are those that rapidly cross plasma membranes, such as urea or glucose in the presence of sufficient insulin, do not lead to cell shrinkage, and do not lead to hyperkalemia. The hyperkalemia associated with diabetic ketoacidosis may, in part, be related to the significant increase in extracellular osmolality that occurs in response to the hyperglycemia and lack of insulin.

Drugs may interfere with the hormonal systems that regulate the distribution of potassium between the intra- and extracellular fluid compartments. The major hormones that regulate potassium distribution are insulin, aldosterone, and β-adrenergic agonists. The normal response to increased potassium intake is increased aldosterone synthesis, and increased aldosterone can increase the intracellular potassium content.
bition of aldosterone production or action can lead to significant hyperkalemia, at least in part, due to decreased cellular potassium uptake. Many drugs in common use affect aldosterone synthesis or action. Aldosterone synthesis is regulated in large part by renin-stimulated angiotensin II production. Inhibitors of renin secretion, such as β-adrenergic antagonists and atrial natriuretic peptide analogues, angiotensin-converting enzyme inhibitors, and angiotensin II receptor antagonists, inhibit angiotensin II-mediated stimulation of adrenal aldosterone synthesis. Heparin inhibits adrenal aldosterone synthase, thereby inhibiting aldosterone production. Spironolactone, a mineralocorticoid receptor antagonist, inhibits aldosterone action at the cellular level. Drugs from any of these classes can cause hyperkalemia in part through alterations in the cellular distribution of potassium.

Renal Potassium Secretion

The kidney possesses such a remarkable ability to excrete large amounts of potassium that chronic hyperkalemia is almost impossible to produce unless renal potassium secretion is impaired. Because the kidney primarily regulates potassium excretion via potassium secretion in the collecting duct, factors that affect potassium excretion generally can be classified into those due to reduced nephron mass (number of functioning collecting ducts) and intrinsic impairment of active potassium secretion. Because the number of collecting ducts is directly related to the glomerular filtration rate, renal insufficiency or renal failure, whether acute or chronic, leads to impaired renal potassium secretion. Patients with impaired renal function have a significantly greater risk of hyperkalemia.

A large number of medications in clinical use interact with collecting duct potassium secretion. Aldosterone directly increases potassium secretion, independent of its effects on potassium distribution. Any drug that interferes with aldosterone production or effect will inhibit collecting duct potassium secretion.

Arachidonic acid metabolites play an important role in collecting duct potassium secretion. This may be due in part to their necessity for stimulation of renin release. Arachidonic acid metabolites also regulate potassium channels; nonsteroidal anti-inflammatory drugs reduce arachidonic acid metabolite production and decrease potassium channel $P_o$, thereby decreasing potassium secretion and potentially causing hyperkalemia.

A third mechanism essential for renal potassium secretion is collecting duct sodium reabsorption. Collecting duct sodium reabsorption increases potassium secretion by increasing luminal electronegativity, thereby increasing the electrochemical gradient for potassium secretion. Several drugs in common use inhibit sodium reabsorption by blocking the principal cell apical sodium channel. The potassium-sparing diuretics amiloride and triamterene are specific inhibitors of this sodium channel (70). More recently, the antibiotics trimethoprim and pentamidine have been found to block the principal cell apical sodium channel (71–73) and lead to hyperkalemia (74–76). High-dose trimethoprim, frequently used with sulfamethoxazole to treat Pneumocystis carinii pneumonia, routinely causes a mild increase in serum potassium, averaging 1.1 mEq/L (77), and occasionally results in severe hyperkalemia (77–80). Those with renal insufficiency and the elderly may develop hyperkalemia even when receiving conventional doses of sulfamethoxazole-trimethoprim (81,82).

Inhibiting the principal cell basolateral Na$^+$-K$^+$-ATPase with digitalis and its analogues, such as digoxin, can cause hyperkalemia (83,84) by inhibiting both sodium reabsorption and potassium secretion. Digoxin may cause hyperkalemia in predisposed patients, such as those with end-stage renal failure, even in the absence of toxic ingestion of a cardiac glycoside (85). This may relate to both impaired renal excretion and to impaired extrarenal cellular uptake of potassium. Some poisoning deaths have been attributed to bufadienolides, naturally occurring cardioactive steroids that have digoxin-like effects (86).

Obstructive uropathy frequently leads to hyperkalemia, but the mechanism is incompletely understood (87,88). The hyperkalemia may occur either in association with metabolic acidosis or separately. These patients have a degree of mineralocorticoid resistance; whether this is a receptor deficiency or is related to the interstitial nephritis that occurs is unclear (87,88). Many patients with obstructive uropathy-induced hyperkalemia will remain hyperkalemic for periods of up to several weeks after relief of the obstruction.

Therapy

Therapies for hyperkalemia can be divided into those that minimize the cardiac effects of hyperkalemia, those that induce potassium uptake by cells resulting in a decrease in plasma potassium, and those that remove potassium from the body.

Blocking Cardiac Effects

Intravenous calcium administration specifically antagonizes the effects of hyperkalemia on the myocardial conduction system and on myocardial repolarization (89). Calcium is the most rapid way to treat hyperkalemia, and is effective even in normocalemic patients. Calcium can be administered as either calcium gluconate or calcium chloride, and should be given via an intravenous route. Effects can be documented on the EKG within 1 to 3 min, and last for 30 to 60 min. A second dose may be given if no effect is seen within 5 to 10 min. Because of the rapid onset of its effect, intravenous calcium administration should be the initial treatment for individuals with EKG abnormalities related to hyperkalemia.

Several precautions should be observed with intravenous calcium. First, it should not be administered in solutions containing NaHCO$_3$ because CaCO$_3$ precipitation can occur. Second, hypercalcemia that occurs during rapid calcium infusion may potentiate the myocardial toxicity of digitalis. Hyperkalemic patients taking digoxin should be given calcium as a slow infusion over 20 to 30 min to avoid hypercalcemia.

Cellular Potassium Uptake

The second fastest way to treat hyperkalemia is to alter potassium distribution by increasing cellular uptake with either insulin or β$_2$-adrenergic agonist administration. Insulin rapidly
Calcium stimulates cellular potassium uptake by extrarenal cells, primarily hepatocytes and myocytes (90,91). Ten units of insulin should be administered intravenously to ensure rapid and consistent bioavailability, and will begin to affect serum potassium levels within 10 to 20 min, with effects lasting for 4 to 6 h (91). Glucose is generally coadministered to avoid hypoglycemia, but should not be given to hyperglycemic individuals. Glucose-induced hyperglycemia can lead to further increases in the potassium concentration due to hypertonicity-induced potassium redistribution.

A second effective treatment for hyperkalemia is β-agonist administration. Intravenous albuterol, 0.5 mg, rapidly stimulates potassium uptake and can decrease serum potassium by approximately 1 mEq/L (92), but is not approved for intravenous use in the United States. Nebulized β2-agonists can be used, however. Albuterol, when administered by nebulizer at a dose of 10 or 20 mg, decreases serum potassium by 0.62 or 0.98 mEq/L, respectively, with an immediate onset of action and maximal effect at 90 to 120 min (93). The primary limitations of β2-agonist therapy include tachycardia when given intravenously (92) and lack of response in 20 to 33% of patients when given by nebulizer (93,94). In addition, albuterol may decrease potassium removal during subsequent hemodialysis (95). A frequent mistake when administering nebulized albuterol is underdosing; the dose required is 2 to 8 times that usually given by nebulizer and 50 to 100 times the dose administered by metered dose inhalers (96). In severe hyperkalemia, combined therapy with insulin and albuterol may be more effective than either alone (97).

Bicarbonate administration is probably less effective than either β-agonist administration or insulin. Recent studies show that the changes in serum potassium with intravenous bicarbonate are small and inconsistent (98–100). Moreover, the associated sodium load may worsen hypertension and contribute to the development of acute congestive heart failure. At present we do not recommend routine use of sodium bicarbonate for hyperkalemia. In patients with hyperkalemia, metabolic acidosis, and volume depletion, rehydration with 5% dextrose solutions with the addition of 150 mEq/L sodium bicarbonate (3 amps of sodium bicarbonate in 1 L D5W) may be a preferable intravenous solution for rehydration than normal saline solutions.

**Potassium Removal**

The definitive treatment for hyperkalemia is removal of potassium from the affected individual. Table 3 summarizes the available options for potassium removal. In selected cases, renal potassium elimination may be adequate for treatment of hyperkalemia. With chronic, mild hyperkalemia, stimulation of renal potassium excretion with either loop or thiazide diuretics may suffice. Acute hyperkalemia is generally not treated optimally with diuretics because the rate of potassium excretion usually will not be adequate. Most patients with hyperkalemia have underlying renal insufficiency as a contributing factor (101), limiting the effectiveness of diuretics. If a rapidly reversible cause of renal failure is identified, such as obstructive uropathy, then treatment of the underlying condition with close observation of the potassium level in association with continuous EKG observation may be adequate.

A second mode of potassium elimination is with the use of a resin, sodium polystyrene sulfonate. This resin exchanges sodium for potassium in the gastrointestinal tract, thereby allowing potassium elimination. In general, 1 g of sodium polystyrene sulfonate removes approximately 0.5 to 1.0 mEq of potassium in exchange for 2 to 3 mEq of sodium. It can be administered either orally or per rectum as a retention enema. The rate of potassium removal is relatively slow, requiring approximately 4 h for full effect. When given orally, sodium

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<td>β2-adrenergic agonist</td>
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* i.v., intravenously; p.o., postoperatively.
polystyrene sulfonate is generally administered with 20% sorbitol to avoid constipation. If given as an enema, avoiding the use of sorbitol may be prudent because several case reports suggest an association between the rectal administration of sodium polystyrene sulfonate with 20% sorbitol and subsequent colonic perforation (102–104). Animal models suggest that the sorbitol is responsible for the colonic perforation, possibly due to mucosal dehydration related to fluid loss into the colon lumen (103).

Dialysis should be considered the primary method of potassium removal when renal function is absent and hyperkalemia is persistent or severe. Hemodialysis is the most rapid method of potassium removal. If a potassium-free dialysate is used, serum potassium may decrease as much as 1.2 to 1.5 mEq/h (98), and should be monitored closely. The more severe the hyperkalemia, the more rapid should be the reduction in plasma potassium (105). However, care should be used with the use of 0 or 1 mEq/L K+ dialysate fluids to avoid precipitating hypokalemia. If a very low potassium dialysate is used, then the serum potassium should be rechecked after 2 h. Peritoneal dialysis, chronic arteriovenous hemodialysis, and chronic venovenous hemodialysis are effective in chronic hyperkalemia, but do not remove potassium fast enough to be recommended for use in acute, severe hyperkalemia. Although dialysis is the most rapid method available to treat most cases of hyperkalemia, other modes of treatment should not be delayed while waiting to institute dialysis.

Specific therapies may be quite valuable in certain causes of hyperkalemia. For example, digoxin-specific Fab fragments are beneficial in many cases of digitalis toxicity (106,107). Patients with acute urinary tract obstruction and subsequent hyperkalemia may be effectively treated with relief of the urinary tract obstruction. Since the rate of potassium excretion in the latter condition may be variable, frequent measurement of plasma potassium is necessary.

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