Advanced Emergency Nursing Journal
Vol. 46, No. 1, pp. 12–24

APPLIED
PHARMACOLOGY

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NCPD

Acute Hyperkalemia Management in the Emergency Department

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ABSTRACT
Acute hyperkalemia is characterized by high concentrations of potassium in the blood that can potentially lead to life-threatening arrhythmias that require emergent treatment. Therapy involves the utilization of a constellation of different agents, all targeting different goals of care. The first, and most important step in the treatment of severe hyperkalemia with electrocardiographic (ECG) changes, is to stabilize the myocardium with calcium in order to resolve or mitigate the development of arrythmias. Next, it is vital to target the underlying etiology of any ECG changes by redistributing potassium from the extracellular space with the use of intravenous regular insulin and inhaled beta-2 agonists. Finally, the focus should shift to the elimination of excess potassium from the body through the use of intravenous furosemide, oral potassium-binding agents, or renal replacement therapy. Multiple nuances and controversies exist with these therapies, and it is important to have a robust understanding of the underlying support and recommendations for each of these agents to ensure optimal efficacy and minimize the potential for adverse effects and medication errors.
Key words: arrhythmias, calcium, furosemide, hyperkalemia, insulin, potassium, renal dialysis

NORMAL SERUM concentrations of potassium generally range between 3.5 and 4.5 mEq/L with fluctuations of no more than 10%. This is only a small representation of total body potassium, however, as there is a high ratio of intracellular (98%) compared with extracellular potassium concentrations (Gennari, 1998; Gumz et al., 2015; Rafique et al., 2019). This balance of concentrations is regulated by a variety of endogenous processes, including insulin and beta-adrenergic catecholamines, that stimulate cell-membrane sodium-potassium-ATPase (Na-K-ATPase) and increase potassium uptake. Further, feedback mechanisms exist such that reduced potassium concentrations inhibit insulin secretion and hyperkalemia increases insulin secretion. In addition, potassium concentrations are influenced by both serum pH, with an acidotic pH increasing potassium concentrations, and thyroid hormone, which stimulates the synthesis of Na-K-ATPase.

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Disclosure: *The authors report no conflicts of interest.*

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DOI: 10.1097/TME.0000000000000504

Hyperkalemia, often defined as a serum potassium above 5 milliequivalents per liter (mEq/L), is a medical emergency frequently seen in emergency departments (EDs) that can lead to fatal arrhythmias if left untreated (Abuelo, 2017; Singer et al., 2017; Sterns et al., 2010). To assist in decision-making, some have suggested stratifying hyperkalemia into mild (5.5–5.9 mEq/L), moderate (6.0–6.4 mEq/L), and severe (>6.5 mEq/L) (Rafique et al., 2019). This electrolyte abnormality can be secondary to a multitude of etiologies including decreased excretion of potassium, increased consumption of potassium, medications, and endocrine abnormalities (see Table 1). As the excretion of potassium is primarily through the kidneys (80%–90%), it follows that the most common comorbidity contributing to this presentation is chronic kidney disease (~70%), although diabetes mellitus and heart failure are also frequent contributors (Gumz et al., 2015; Rafique et al., 2019). The most concerning consequence of hyperkalemia is the potential for the patient to develop bradycardia and electrocardiographic (ECG) changes (e.g., peaked T-waves, prolonged PR segment, and QRS widening) (Rafique et al., 2019).

Table 1. Frequent patient characteristics associated with emergency department hyperkalemia presentations[a]

Acute/chronic kidney disease
Diabetes mellitus
Rhabdomyolysis
Hypoaldosteronism
Heart failure
Sickle cell disease
Drug-induced
• Angiotensin-converting enzyme inhibitors
• Angiotensin II receptor blockers
• Spironolactone
• Beta-blockers
• Trimethoprim
• Nonsteroidal anti-inflammatory agents
• Digoxin
• Potassium supplementation
• Tacrolimus

Note. From "Hyperkalaemia in the Emergency Department: Epidemiology, Management and Monitoring of Treatment Outcomes," by K. Pollack, K. R. Manning, J. Balassone, C. Bui, D. M. Taylor, and S. E. Taylor, 2022, *Emergency Medicine Australasia, 34*(5), pp. 751–757.
[a]Selective list.

In addition to identifying and correcting modifiable causes of hyperkalemia (e.g., potassium-containing supplementation), the treatment of acute hyperkalemia involves the sequential implementation of multiple therapies targeting different therapeutic goals (see Figure 1) (Abuelo, 2017; ECC Committee & Task Forces of the American Heart, 2005; Kamel & Wei, 2003). The first goal is to ensure myocardial stability by using intravenous (IV) calcium gluconate or calcium chloride to reduce cardiac excitability in patients at risk. Next, it is important to implement pharmacotherapy targeted at temporarily mobilizing extracellular potassium into the intracellular space by activating the Na-K-ATPase pump through the use of IV regular insulin and a beta-2 agonist (e.g., albuterol). The last step in treatment provides a more enduring reduction in potassium concentrations through enhancing excretion of potassium from the body with the diuretics (e.g., IV furosemide), oral potassium-binding agents (e.g., sodium zirconium cyclosilicate), or the use of renal replacement therapy (Abuelo, 2017; ECC Committee & Task Forces of the American Heart, 2005; Kovesdy, 2015). Although seemingly straightforward, multiple controversies and nuances currently exist with these proposed therapies potentially leading to variations in practice and suboptimal care. When rapid reduction and normalization of potassium in the ED is achieved however, it can be associated with a 50% reduction in mortality (Singer et al., 2020). The intent of this review is to provide the most up-to-date evidence and recommendations for proposed options and highlight areas in need of further investigation to allow practitioners to appropriately weigh the risks and benefits of each therapy.

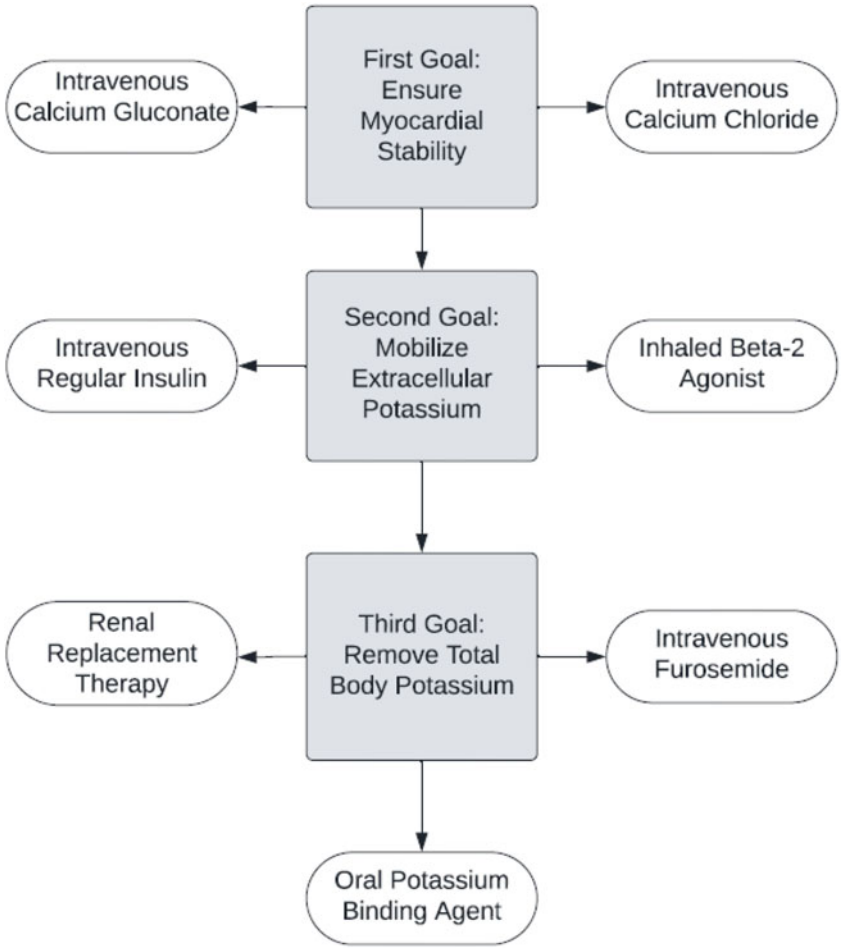

Figure 1. Acute hyperkalemia management goals. From "Hyperkalemia Management in the Emergency Department: An Expert Panel Consensus," by Z. Rafique, F. Peacock, T. Armstead, J. J. Bischof, J. Hudson, M. R. Weir, and J. Neuenschwander, 2021, *Journal of the American College of Emergency Physicians Open, 2*(5), p. e12572.

CALCIUM

The first, and most important, step in the management of acute hyperkalemia is to limit the morbidity and mortality associated with any cardiac manifestations that may be present (Sterns et al., 2016). Calcium concentrations antagonize the myocardial excitability induced by hyperkalemia and can result in a rapid improvement or reversal of ECG changes in less than 5 min (Hoffman & Suckling, 1956). Calcium is typically administered IV in this setting as 10–20 ml of 10% calcium gluconate or 10 ml of 10% calcium chloride, and further doses can be administered if no effect is seen after 5–10 min (see Table 2) (Carvalhana et al., 2006; Mahoney et al., 2005; Pang et al., 2004; Quick & Bastani, 1994; Soar et al., 2010). Both formulations are considered equally effective in this scenario and no randomized controlled trials have compared the efficacy of these two agents for the treatment of hyperkalemia. It is important to note that the elemental calcium contained in 10 ml of 10% calcium gluconate (93 mg) is substantially less than

Table 2. Pharmacotherapy options for treating acute hyperkalemia in the emergency department

Pharmacotherapy	Dose	Onset of action of therapeutic effect	Adverse effects
Calcium chloride, 10%	1 g IV (10 ml)	5–10 min	Extravasation; hypotension; flushing; bradycardia; nausea; vomiting
Calcium gluconate, 10%	1 g IV (10 ml)	5–10 min	
Insulin, regular	5 units IV	15 min	Hypoglycemia
Albuterol	10–15 mg inh/neb	30 min	Tachycardia; tremor; palpitations
Furosemide	20 mg IV[a]	Unknown	Hypotension; hypovolemia; hyponatremia; ototoxicity
Bumetanide	1 mg IV[a]	Unknown	Hypotension; hypovolemia; hyponatremia; ototoxicity
Sodium zirconium cyclosilicate	10 mg orally	60 min	Gastrointestinal upset

Note. inh = inhaled; iv = Intravenous; neb = nebulized. From "Management of Severe Hyperkalemia Without Hemodialysis: Case Report and Literature Review," by V. Carvalhana, L. Burry, & S. E. Lapinsky, 2006, *Journal of Critical Care, 21*(4), pp. 316–321, and "Hyperkalemia Management in the Emergency Department: An Expert Panel Consensus," by Z. Rafique, F. Peacock, T. Armstead, J. J. Bischof, J. Hudson, M. R. Weir, and J. Neuenschwander, 2021, *Journal of the American College of Emergency Physicians Open, 2*(5), p. e12572.
[a]Or home dose intravenously if applicable.

that of 10 ml of 10% calcium chloride (272 mg), and hence two to three doses of calcium gluconate are necessary to achieve an equivalent calcium dose to 10 ml of 10% calcium chloride. No difference in the speed or extent of calcium concentration elevations has been found when administering these two agents at equivalent doses (Cote et al., 1987; Martin et al., 1990).

Although calcium administration is a critical mainstay of acute hyperkalemia therapy, it does carry the potential for adverse effects (Gupta et al., 2022). Adverse effects of IV calcium use can include vasodilation leading to hypotension, bradycardia, and arrhythmias (Rafique et al., 2019). The physical administration of calcium also has potential to cause serious adverse events if extravasation occurs, which can lead to tissue necrosis (Heckler & McCraw, 1976; Pacheco Compana et al., 2017; Semple & Booth, 1996). Although extravasation is a risk with both calcium gluconate and calcium chloride, there are no randomized controlled trials comparing the safety of these two agents in the treatment of this condition. The risk appears to be increased with higher calcium concentrations; thus, calcium gluconate is potentially a safer option when administering this agent through a peripheral vein, and opting to use calcium chloride for patients in cardiac arrest or when central access is available (Panchal et al., 2020). The speed of administration can also impact adverse effect occurrence as rapid administration of IV calcium has the potential to generate additional adverse effects (e.g., flushing, nausea, vomiting, and hypotension), so when possible it is recommended to infuse calcium over 5 min (Truhlář et al., 2015). However, in the setting of hemodynamic instability or cardiac arrest, IV calcium chloride should always be administered as rapidly as possible.

The risks associated with this therapy should always be balanced with the potential benefits of IV calcium administration, keeping in mind it may not be indicated in all acute hyperkalemic patients. Although it is indicated for life-threatening ECG abnormalities even in the setting of unknown potassium concentrations, there is limited data as to whom would benefit from this therapy

beyond this indication (Mahoney et al., 2005; Pang et al., 2004; Quick & Bastani, 1994; Soar et al., 2010). Some have suggested that in those presenting without ECG changes, regardless of potassium concentration, it may be reasonable to withhold calcium therapy as these patients are at a lower risk for subsequent hyperkalemia-induced ECG changes (Alfonzo et al., 2006; Durfey et al., 2017; Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, 2000; Heckler & McCraw, 1976; Montague et al., 2008; Pacheco Compana et al., 2017; Semple & Booth, 1996).

PROMOTING POTASSIUM UPTAKE

The extensive intracellular stockpile of potassium is maintained through sodium exchange and represents the critical extrarenal mechanism for maintaining homeostatic serum potassium concentrations (Sterns et al., 2016). In the acute management of hyperkalemia, this endogenous mechanism is a therapeutic target to emergently reduce serum potassium concentrations. Three potential therapies have been suggested to take advantage of this endogenous process: insulin, beta-2 agonists, and sodium bicarbonate.

INSULIN

Insulin reduces serum potassium concentrations through increasing the activity of the Na-K-ATPase and exchanging extracellular potassium for intracellular sodium, often within 15 min (Rafique et al., 2021; Sterns et al., 2016). One of the earliest studies examining the use of insulin for this purpose found a significant correlation between potassium concentration lowering and increasing insulin doses (DeFronzo et al., 1980). Traditionally, it has been recommended to administer 10 units of regular IV insulin for this purpose, which is a recommendation cited from a 1995 review article, which evaluated seven studies demonstrating the efficacy of insulin in lowering serum potassium concentrations (Allon, 1995; Allon & Copkney, 1990; Alvestrand et al., 1984; Blumberg et al., 1988; DeFronzo et al., 1980; Kovesdy, 2014; Lens et al., 1989; Massara et al., 1975; Minaker & Rowe, 1982). However, as patients with renal insufficiency are one of the most frequent patient populations presenting with clinically significant hyperkalemia, and renal function also plays a critical role in insulin metabolism and excretion, this therapy can have an extended duration of action and an increased risk of iatrogenic hypoglycemia in this population (Harel & Kamel, 2016; Kovesdy, 2014; LaRue et al., 2017; Li & Vijayan, 2014; McNicholas et al., 2018; Pierce et al., 2015; Wheeler et al., 2016). Additional research has identified that patients with no history of diabetes mellitus, lower pretreatment glucose concentrations (<140 mg/dL), lower body weights, and female sex also appear to be at a higher risk of hypoglycemia (Apel et al., 2014; Coca et al., 2017; Garcia et al., 2020; Moussavi et al., 2019; Schafers et al., 2012; Wheeler et al., 2016). To help address this issue, studies have sought to evaluate a variety of reduced dose and weight-based regimens in those with different severities of renal dysfunction (Garcia et al., 2020; Harel & Kamel, 2016; LaRue et al., 2017; McNicholas et al., 2018; Pierce et al., 2015; Scott et al., 2019; Wheeler et al., 2016). These studies have found that one standardized dose of IV regular insulin is not appropriate for all patients and that lower insulin doses have the potential to have equivalent potassium-lowering ability while minimizing hypoglycemia risks. Although an optimal dose has yet to be identified, multiple studies have found that using an IV bolus of 5 units of regular insulin may be better in the setting of renal dysfunction and confer a lower risk of hypoglycemia with no difference in serum potassium reduction compared to 10 units (Garcia et al., 2020; Keeney et al., 2020; LaRue et al., 2017; McNicholas et al., 2018).

Regardless of the insulin-dosing strategy chosen however, there remains a subsequent risk of hypoglycemia several hours later (i.e., 1–6 hr) (Harel & Kamel, 2016). As such,

unless the patient has a substantially elevated baseline glucose, it is typically recommended that IV dextrose (e.g., 25–50 g of 10%–50%) be administered prior to IV regular insulin administration to decrease the incidence of iatrogenic hypoglycemia (Moussavi et al., 2019). It is also important to reobtain glucose and potassium concentrations following insulin administration, regardless of dextrose administration, to ensure both safety and efficacy of this therapy. It is generally recommended that blood glucose concentrations be monitored at least every hour for the first 4–6 hr and potassium concentrations should be reassessed 2–4 hr following insulin administration (Moussavi et al., 2019; Rafique et al., 2021). From a functional standpoint at bedside, it is often helpful to build computerized order sets for hyperkalemia management with linked hypoglycemia order sets that include both repeat glucose monitoring and hypoglycemia management therapy options (e.g., oral glucose) in a busy ED to minimize patient harm.

As with any emergent therapy there is risk of medication errors, however the unique aspects of both insulin itself and its use in this scenario exacerbate these risks dramatically. This therapy represents a substantial deviation from traditional insulin use, both in terms of the drug used (i.e., not short-acting insulin) and route of administration (i.e., intravenous). An analysis of 200 adverse events associated with hyperkalemia treatment found that delayed treatment and administration of insulin by the wrong route or the wrong dose (most commonly overdoses) were the most frequent errors (Institute for Safe Medication Practices, 2018). The etiology of the wrong route errors was most frequently founded in the automatic default of systems to order the subcutaneous route for insulin administration, and the dosing errors were most commonly caused by insulin doses being measured in milliliters instead of units. To mitigate the occurrence of these errors, it is recommended that hyperkalemia order sets be developed to ensure the most appropriate route of administration is utilized and that Luer-compatible insulin syringes without needles be available for use to help limit dosing errors.

BETA-2 AGONISTS

Beta-2 agonists (e.g., albuterol), most commonly administered through inhalation/nebulization, work to reduce serum potassium concentrations similar to insulin by enhancing Na-K-ATPase activity by binding to beta-2 receptors (McClure et al., 1994; Rafique et al., 2021; Sterns et al., 2016). Although urgent, the administration of these agents is less emergent compared with previously discussed therapies as their onset is somewhat delayed at 30 min, and the peak effect is not noted until around 60 min (Allon & Copkney, 1990; Allon et al., 1989; Lens et al., 1989). It is important to note that the dose used for this purpose (i.e., 10–15 mg) is higher than most traditional recommendations for asthma management. As a result, of the need for this higher dose, it also has an increased risk of side effects, potentially leading to tachycardia, tremor, and palpitations (Allon et al., 1989; Mandelberg et al., 1999).

SODIUM BICARBONATE

The theory behind the use of sodium bicarbonate in the management of acute hyperkalemia is that it increases intracellular sodium and subsequently increases Na-K-ATPase activity (Rafique et al., 2021; Sterns et al., 2016). Although the theoretical mechanism for the use of sodium bicarbonate would be effective in reducing extracellular potassium concentrations, there is substantial debate regarding the utility of this agent in reducing potassium concentrations and multiple studies have failed to demonstrate efficacy (Allon & Shanklin, 1996; Greenberg, 1998; Gutierrez et al., 1991; Mahoney et al., 2005; Ngugi et al., 1997). This lack of efficacy may be related to the activity of the sodium-hydrogen ion exchanger, the initial step leading to enhanced Na-K-ATPase activity, being in an inactive state in the absence

of acidosis (Kamel & Wei, 2003). Therefore, currently the most likely role for this agent is in the setting of hyperkalemia associated with metabolic acidosis. Although the threshold for identifying a metabolic acidosis that may benefit from sodium bicarbonate is not firmly established, a classically referenced investigation found a large benefit in those with a pH less than 7.2 (Schwarz et al., 1959).

As with other agents, this therapy does not come without its own risks, most notably the risk of extravasation (Wax & Haynes, 2019). Although multiple concentrations of sodium bicarbonate are available, ranging from 4.2% to 8.4%, all are extremely hyperosmolar with the 8.4% solution having an osmolality of 2,000 mOsm/L for example (Wax & Haynes, 2019). Therefore, similar to IV calcium, this agent should be used at the lowest concentration necessary and should be used judiciously in those whom the benefits outweigh this extravasation risk (Keidan et al., 2015; Le & Patel, 2014). In addition, although metabolic acidosis carries with it multiple deleterious physiological effects (e.g., impaired myocardial contractility), there are some beneficial effects such as the reduced hemoglobin affinity for oxygen and increased tissue oxygen availability as well as increased availability of ionized calcium (Schaer & Bachmann, 1974; Stengl et al., 2013; Wardi et al., 2023). Therefore, administration of sodium bicarbonate could inadvertently cause decreased oxygen availability and a reduction in ionized calcium concentrations by increasing albumin binding, thus resulting in an antagonistic mechanism to IV calcium administration (Douglas et al., 1979).

ENHANCING POTASSIUM ELIMINATION

Although all these therapies may be effective at reducing potassium concentrations acutely, correction of the underlying problem of elevated total body potassium content is also necessary (Rafique et al., 2021; Sterns et al., 2016). Shifting potassium intracellularly is a temporizing measure, albeit the most significant measure implemented emergently in the ED, and so this should be followed by efforts to eliminate systemic potassium from the body whether it be through pharmacotherapy targeted at eliminating potassium or removal through renal replacement therapy. Pharmacotherapies aimed at increasing potassium elimination from the body include potassium-wasting diuretics (e.g., furosemide) and oral potassium-binding agents (e.g., sodium polystyrene sulfonate) (Sterns et al., 2016).

LOOP DIURETICS

Loop diuretics (i.e., furosemide, bumetanide) reduce systemic potassium concentrations through their action in the loop of Henle to block sodium and potassium reabsorption (Ismail et al., 2021; Rafique et al., 2021). Unfortunately, evidence for the use of these agents in the setting of acute hyperkalemia or their onset of action is limited and is dependent on the patient being able to make urine. Nevertheless, these provide functional options in patients presenting with hyperkalemia secondary to heart failure who are also fluid overloaded and should be administered IV to maximize their diuretic effect (Ismail et al., 2021). Individual doses of furosemide IV up to 80 mg can be administered slowly over 1–2 min; however, larger doses should be diluted and infused at a rate no greater than 4 mg/min (Fresenius Kabi, 2015). Individual doses of bumetanide IV should also be administered over 1–2 min (Hospira Inc, 2017). Adverse effects of these agents are similar to their utilization in other disease states such as hypotension secondary to hypovolemia, hyponatremia, and ototoxicity (Rafique et al., 2021).

SODIUM POLYSTYRENE SULFONATE

Although the primary route of potassium elimination from the body is through the urine, the colon also excretes a small amount of potassium each day and sodium polystyrene sulfonate (SPS) is an agent that has been historically used to take advantage

of this fact (Gupta et al., 2022; Rafique et al., 2021). Theoretically, SPS exchanges sodium for potassium in the intestines to enhance the rectal elimination of potassium (Rafique et al., 2021). To date, three successive Cochrane reviews of the literature regarding SPS in this setting have concluded that high-quality studies are lacking and that this agent does not have sufficient support that it lowers serum potassium (Batterink et al., 2015; Mahoney et al., 2005; Natale et al., 2020).

As further deterrence for using this agent, although there is an absence of literature supporting the efficacy with SPS, there is evidence suggesting harm with its use (Gupta et al., 2022; Rafique et al., 2021). A systematic review of the literature in 2013 of 58 cases of adverse events found a significant association between SPS use and gastrointestinal adverse events, most commonly intestinal necrosis and a mortality rate of 33% (Harel et al., 2013). A subsequent systematic review also found a similar association with gastrointestinal adverse effects and an elevated mortality among patients experiencing them (Wu et al., 2021). Further, a study of 20,020 individuals identified an almost twofold higher risk of hospitalization for gastrointestinal events within 30 days of being prescribed SPS (Noel et al., 2019). Due to the lack of efficacy and identified risk of adverse effects, this agent is not recommended for use in the treatment of acute hyperkalemia (Lindner et al., 2020; Panchal et al., 2020).

PATIROMER AND SODIUM ZIRCONIUM CYCLOSILICATE

Although SPS does not appear to be effective for the treatment of acute hyperkalemia, two other promising agents have been developed to take advantage of the same route for potassium elimination, patiromer, and sodium zirconium cyclosilicate (SZC) (Meaney et al., 2017). Patiromer is a cation exchange resin that exchanges calcium for potassium in the gastrointestinal tract, whereas SZC is also a cation exchange agent that works in a modality similar to SPS by exchanging potassium for sodium and hydrogen (Meaney et al., 2017; Rafique et al., 2021). Secondary to its delayed onset of action (i.e., 7–48 hr), patiromer's role is primarily relegated to the management of chronic hyperkalemia and is not recommended for use in the acute setting (Meaney et al., 2017). In contrast, SZC has a far more rapid onset of action (i.e., 1–6 hr) and hence can be a valuable asset in the management of acute hyperkalemia in the ED. However, it is important to point out once again that this therapy should be initiated following the previously mentioned emergent measures as the average reduction in potassium concentration at 1 h following SZC administration is only around 0.5 mEq/L (Meaney et al., 2017).

HEMODIALYSIS

Renal replacement therapy is the most effective means of reducing serum potassium concentrations by directly and effectively filtering the serum (Rafique et al., 2021). Although effective, its utility is somewhat limited in the ED for acute management due to the need for appropriate vascular access and machine availability. This limits its use to individuals with preexisting end-stage renal disease and established access, although the emergent placement of access and dialysis is an option in life-threatening settings (Palmer & Clegg, 2018).

CONCLUSION

Acute hyperkalemia is a common presentation to the ED and can carry with it substantial morbidity and mortality if not emergently corrected. Multiple agents exist to help manage this presentation, all with a specific goal and sequence of implementation. However, there are multiple nuances and controversies that exist regarding these agents and it is important to tailor therapies to the individual patient and balance the risks and benefits of each therapy to ensure optimal patient outcomes.

REFERENCES

Abuelo, J. G. (2017). Treatment of severe hyperkalemia: Confronting 4 fallacies. *Kidney International Reports*, *3*(1), 47–55. https://doi.org/10.1016/j.ekir.2017.10.001

Alfonzo, A. V., Isles, C., Geddes, C., & Deighan, C. (2006). Potassium disorders—clinical spectrum and emergency management. *Resuscitation*, *70*(1), 10–25. https://doi.org/10.1016/j.resuscitation.2005.11.002

Allon, M. (1995). Hyperkalemia in end-stage renal disease: Mechanisms and management. *Journal of the American Society of Nephrology*, *6*(4), 1134–1142. https://www.ncbi.nlm.nih.gov/pubmed/8589279

Allon, M., & Copkney, C. (1990). Albuterol and insulin for treatment of hyperkalemia in hemodialysis patients. *Kidney International*, *38*(5), 869–872. https://www.ncbi.nlm.nih.gov/pubmed/2266671

Allon, M., Dunlay, R., & Copkney, C. (1989). Nebulized albuterol for acute hyperkalemia in patients on hemodialysis. *Annals of Internal Medicine*, *110*(6), 426–429. https://doi.org/10.7326/0003-4819-110-6-426

Allon, M., & Shanklin, N. (1996). Effect of bicarbonate administration on plasma potassium in dialysis patients: Interactions with insulin and albuterol. *American Journal of Kidney Diseases*, *28*(4), 508–514. https://doi.org/10.1016/s0272-6386(96)90460-6

Alvestrand, A., Wahren, J., Smith, D., & DeFronzo, R. A. (1984). Insulin-mediated potassium uptake is normal in uremic and healthy subjects. *American Journal of Physiology*, *246*(2, Pt. 1), E174–E180. https://doi.org/10.1152/ajpendo.1984.246.2.E174

Apel, J., Reutrakul, S., & Baldwin, D. (2014). Hypoglycemia in the treatment of hyperkalemia with insulin in patients with end-stage renal disease. *Clinical Kidney Journal*, *7*(3), 248–250. https://doi.org/10.1093/ckj/sfu026

Batterink, J., Cessford, T. A., & Taylor, R. A. (2015). Pharmacological interventions for the acute management of hyperkalaemia in adults. *Cochrane Database of Systematic Reviews*, *10*(10), CD010344. https://doi.org/10.1002/14651858.CD010344.pub2

Blumberg, A., Weidmann, P., Shaw, S., & Gnädinger, M. (1988). Effect of various therapeutic approaches on plasma potassium and major regulating factors in terminal renal failure. *The American Journal of Medicine*, *85*(4), 507–512. https://www.ncbi.nlm.nih.gov/pubmed/3052050

Carvalhana, V., Burry, L., & Lapinsky, S. E. (2006). Management of severe hyperkalemia without hemodialysis: Case report and literature review. *Journal of Critical Care*, *21*(4), 316–321. https://doi.org/10.1016/j.jcrc.2005.10.001

Coca, A., Valencia, A. L., Bustamante, J., Mendiluce, A., & Floege, J. (2017). Hypoglycemia following intravenous insulin plus glucose for hyperkalemia in patients with impaired renal function. *PLoS ONE*, *12*(2), e0172961. https://doi.org/10.1371/journal.pone.0172961

Cote, C. J., Drop, L. J., Daniels, A. L., & Hoaglin, D. C. (1987). Calcium chloride versus calcium gluconate: Comparison of ionization and cardiovascular effects in children and dogs. *Anesthesiology*, *66*(4), 465–470. https://www.ncbi.nlm.nih.gov/pubmed/3565811

DeFronzo, R. A., Felig, P., Ferrannini, E., & Wahren, J. (1980). Effect of graded doses of insulin on splanchnic and peripheral potassium metabolism in man. *American Journal of Physiology*, *238*(5), E421–E427. https://doi.org/10.1152/ajpendo.1980.238.5.E421

Douglas, M. E., Downs, J. B., Mantini, E. L., & Ruiz, B. C. (1979). Alteration of oxygen tension and oxyhemoglobin saturation. A hazard of sodium bicarbonate administration. *The Archives of Surgery*, *114*(3), 326–329. https://doi.org/10.1001/archsurg.1979.01370270096018

Durfey, N., Lehnhof, B., Bergeson, A., Durfey, S. N. M., Leytin, V., McAteer, K., Schwam, E., & Valiquet, J. (2017). Severe hyperkalemia: Can the electrocardiogram risk stratify for short-term adverse events? *The Western Journal of Emergency Medicine*, *18*(5), 963–971. https://doi.org/10.5811/westjem.2017.6.33033

ECC Committee, Subcommittees and Task Forces of the American Heart Association. (2005). 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*, *112*(24 Suppl.), IV1–203. https://doi.org/10.1161/CIRCULATIONAHA.105.166550

Fresenius Kabi (per DailyMed). (2015). *Product information: Furosemide intravenous, intramuscular injection, furosemide intravenous, intramuscular injection*.

Garcia, J., Pintens, M., Morris, A., Takamoto, P., Baumgartner, L., & Tasaka, C. L. (2020). Reduced versus conventional dose insulin for hyperkalemia treatment. *Journal of Pharmacy Practice*, *33*(3), 262–266. https://doi.org/10.1177/0897190018799220

Gennari, F. J. (1998). Hypokalemia. *The New England Journal of Medicine*, *339*(7), 451–458. https://doi.org/10.1056/NEJM199808133390707

Greenberg, A. (1998). Hyperkalemia: Treatment options. *Seminars in Nephrology*, *18*(1), 46–57. https://www.ncbi.nlm.nih.gov/pubmed/9459288

Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. (2000). Part 8: Advanced challenges in resuscitation: Section 1: Life-threatening electrolyte abnormalities. The American Heart Association in collaboration with the International Liaison Committee on Resuscitation. *Circulation*, *102*(8 Suppl.), I217–I222. https://www.ncbi.nlm.nih.gov/pubmed/10966675

Gumz, M. L., Rabinowitz, L., & Wingo, C. S. (2015). An integrated view of potassium homeostasis. *The New*

England Journal of Medicine, *373*(1), 60–72. https://doi.org/10.1056/NEJMra1313341

Gupta, A. A., Self, M., Mueller, M., Wardi, G., & Tainter, C. (2022). Dispelling myths and misconceptions about the treatment of acute hyperkalemia. *American Journal of Emergency Medicine*, *52*, 85–91. https://doi.org/10.1016/j.ajem.2021.11.030

Gutierrez, R., Schlessinger, F., Oster, J. R., Rietberg, B., & Perez, G. O. (1991). Effect of hypertonic versus isotonic sodium bicarbonate on plasma potassium concentration in patients with end-stage renal disease. *Mineral and Electrolyte Metabolism*, *17*(5), 297–302. https://www.ncbi.nlm.nih.gov/pubmed/1668124

Harel, Z., Harel, S., Shah, P. S., Wald, R., Perl, J., & Bell, C. M. (2013). Gastrointestinal adverse events with sodium polystyrene sulfonate (Kayexalate) use: A systematic review. *The American Journal of Medicine*, *126*(3), 264.e9–24. https://doi.org/10.1016/j.amjmed.2012.08.016

Harel, Z., & Kamel, K. S. (2016). Optimal dose and method of administration of intravenous insulin in the management of emergency hyperkalemia: A systematic review. *PLoS ONE*, *11*(5), e0154963. https://doi.org/10.1371/journal.pone.0154963

Heckler, F. R., & McCraw, J. B. (1976). Calcium-related cutaneous necrosis. *Surgical Forum*, *27*(62), 553–555. https://www.ncbi.nlm.nih.gov/pubmed/1019976

Hoffman, B. F., & Suckling, E. E. (1956). Effect of several cations on transmembrane potentials of cardiac muscle. *American Journal of Physiology*, *186*(2), 317–324. https://doi.org/10.1152/ajplegacy.1956.186.2.317

Hospira Inc (per DailyMed). (2017). *Product information: Bumetanide intravenous injection, intramuscular injection, bumetanide intravenous injection, intramuscular injection*.

Institute for Safe Medication Practices. (2018). *Medication errors during insulin adminisntration for patients with hyperkalemia*. https://www.ismp.org/resources/medication-errors-during-insulin-administration-patients-hyperkalemia

Ismail, U., Sidhu, K., & Zieroth, S. (2021). Hyperkalaemia in heart failure. *Cardiac Failure Review*, *7*, e10. https://doi.org/10.15420/cfr.2020.29

Kamel, K. S., & Wei, C. (2003). Controversial issues in the treatment of hyperkalaemia. *Nephrology Dialysis Transplantation*, *18*(11), 2215–2218. https://www.ncbi.nlm.nih.gov/pubmed/14551344

Keeney, K. P., Calhoun, C., Jennings, L., Weeda, E. R., & Weant, K. A. (2020). Assessment of intravenous insulin dosing strategies for the treatment of acute hyperkalemia in the emergency department. *American Journal of Emergency Medicine*, *38*(6), 1082–1085. https://doi.org/10.1016/j.ajem.2019.158374

Keidan, I., Ben-Menachem, E., & Greenberger, S. (2015). Safety of extravasated sodium bicarbonate. *Resuscitation*, *97*, e7. https://doi.org/10.1016/j.resuscitation.2015.04.014

Kovesdy, C. P. (2014). Management of hyperkalaemia in chronic kidney disease. *Nature Reviews Nephrology*, *10*(11), 653–662. https://doi.org/10.1038/nrneph.2014.168

Kovesdy, C. P. (2015). Management of hyperkalemia: An update for the internist. *The American Journal of Medicine*, *128*(12), 1281–1287. https://doi.org/10.1016/j.amjmed.2015.05.040

LaRue, H. A., Peksa, G. D., & Shah, S. C. (2017). A comparison of insulin doses for the treatment of hyperkalemia in patients with renal insufficiency. *Pharmacotherapy*, *37*(12), 1516–1522. https://doi.org/10.1002/phar.2038

Le, A., & Patel, S. (2014). Extravasation of noncytotoxic drugs: A review of the literature. *Annals of Pharmacotherapy*, *48*(7), 870–886. https://doi.org/10.1177/1060028014527820

Lens, X. M., Montoliu, J., Cases, A., Campistol, J. M., & Revert, L. (1989). Treatment of hyperkalaemia in renal failure: Salbutamol v. insulin. *Nephrology Dialysis Transplantation*, *4*(3), 228–232. https://doi.org/10.1093/oxfordjournals.ndt.a091860

Li, T., & Vijayan, A. (2014). Insulin for the treatment of hyperkalemia: A double-edged sword? *Clinical Kidney Journal*, *7*(3), 239–241. https://doi.org/10.1093/ckj/sfu049

Lindner, G., Burdmann, E. A., Clase, C. M., Hemmelgarn, B. R., Herzog, C. A., Malyszko, J., Nagahama, M., Pecoits-Filho, R., Rafique, Z., Rossignol, P., & Singer, A. J. (2020). Acute hyperkalemia in the emergency department: A summary from a kidney disease: Improving Global Outcomes conference. *European Journal of Emergency Medicine*, *27*(5), 329–337. https://doi.org/10.1097/MEJ.0000000000000691

Mahoney, B. A., Smith, W. A., Lo, D. S., Tsoi, K., Tonelli, M., & Clase, C. M. (2005). Emergency interventions for hyperkalaemia. *Cochrane Database of Systematic Reviews*, *2005*(2), CD003235. https://doi.org/10.1002/14651858.CD003235.pub2

Mandelberg, A., Krupnik, Z., Houri, S., Smetana, S., Gilad, E., Matas, Z., & Priel, I. E. (1999). Salbutamol metered-dose inhaler with spacer for hyperkalemia: How fast? How safe? *Chest*, *115*(3), 617–622. https://doi.org/10.1378/chest.115.3.617

Martin, T. J., Kang, Y., Robertson, K. M., Virji, M. A., & Marquez, J. M. (1990). Ionization and hemodynamic effects of calcium chloride and calcium gluconate in the absence of hepatic function. *Anesthesiology*, *73*(1), 62–65. https://doi.org/10.1097/00000542-199007000-00010

Massara, F., Camanni, F., & Molinatti, G. (1975). Effect of propranolol on some adrenaline- and insulin-induced metabolic changes in man. *Acta diabetologica Latina*, *12*(1), 41–51. https://www.ncbi.nlm.nih.gov/pubmed/1229805

McClure, R. J., Prasad, V. K., & Brocklebank, J. T. (1994). Treatment of hyperkalaemia using intravenous and nebulised salbutamol. *Archives of Disease in Childhood*, *70*(2), 126–128. https://doi.org/10.1136/adc.70.2.126

McNicholas, B. A., Pham, M. H., Carli, K., Chen, C. H., Colobong-Smith, N., Anderson, A. E., & Pham, H. (2018). Treatment of hyperkalemia with a low-dose insulin protocol is effective and results in reduced hypoglycemia. *Kidney International Reports*, *3*(2), 328–336. https://doi.org/10.1016/j.ekir.2017.10.009

Meaney, C. J., Beccari, M. V., Yang, Y., & Zhao, J. (2017). Systematic review and meta-analysis of patiromer and sodium zirconium cyclosilicate: A new armamentarium for the treatment of hyperkalemia. *Pharmacotherapy*, *37*(4), 401–411. https://doi.org/10.1002/phar.1906

Minaker, K. L., & Rowe, J. W. (1982). Potassium homeostasis during hyperinsulinemia: Effect of insulin level, beta-blockade, and age. *American Journal of Physiology*, *242*(6), E373–377. https://doi.org/10.1152/ajpendo.1982.242.6.E373

Montague, B. T., Ouellette, J. R., & Buller, G. K. (2008). Retrospective review of the frequency of ECG changes in hyperkalemia. *Clinical Journal of the American Society of Nephrology*, *3*(2), 324–330. https://doi.org/10.2215/CJN.04611007

Moussavi, K., Fitter, S., Gabrielson, S. W., Koyfman, A., & Long, B. (2019). Management of hyperkalemia with insulin and glucose: Pearls for the emergency clinician. *Journal of Emergency Medicine*, *57*(1), 36–42. https://doi.org/10.1016/j.jemermed.2019.03.043

Natale, P., Palmer, S. C., Ruospo, M., Saglimbene, V. M., & Strippoli, G. F. (2020). Potassium binders for chronic hyperkalaemia in people with chronic kidney disease. *Cochrane Database of Systematic Reviews*, *6*(6), CD013165. https://doi.org/10.1002/14651858.CD013165.pub2

Ngugi, N. N., McLigeyo, S. O., & Kayima, J. K. (1997). Treatment of hyperkalaemia by altering the transcellular gradient in patients with renal failure: Effect of various therapeutic approaches. *East African Medical Journal*, *74*(8), 503–509. https://www.ncbi.nlm.nih.gov/pubmed/9487416

Noel, J. A., Bota, S. E., Petrcich, W., Garg, A. X., Carrero, J. J., Harel, Z., Tangri, N., Clark, E. G., Komenda, P., & Sood, M. M. (2019). Risk of hospitalization for serious adverse gastrointestinal events associated with sodium polystyrene sulfonate use in patients of advanced age. *JAMA Internal Medicine*, *179*(8), 1025–1033. https://doi.org/10.1001/jamainternmed.2019.0631

Pacheco Compana, F. J., Midon Miguez, J., & de Toro Santos, F. J. (2017). Lesions associated with calcium gluconate extravasation: Presentation of 5 clinical cases and analysis of cases published. *Annals of Plastic Surgery*, *79*(5), 444–449. https://doi.org/10.1097/SAP.0000000000001110

Palmer, B. F., & Clegg, D. J. (2018). Hyperkalemia across the continuum of kidney function. *Clinical Journal of the American Society of Nephrology*, *13*(1), 155–157. https://doi.org/10.2215/CJN.09340817

Panchal, A. R., Bartos, J. A., Cabañas, J. G., Donnino, M. W., Drennan, I. R., Hirsch, K. G., Kudenchuk, P. J., Kurz, M. C., Lavonas, E. J., Morley, P. T., O'Neil, B. J., Peberdy, M. A., Rittenberger, J. C., Rodriguez, A. J., Sawyer, K. N., & Berg, K. M.; Adult Basic and Advanced Life Support Writing Group. (2020). Part 3: Adult basic and advanced life support: 2020 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*, *142*(16 Suppl. 2), S366–S468. https://doi.org/10.1161/CIR.0000000000000916

Pang, P. S., Look, R. B., Brown, D. F., & Nadel, E. S. (2004). Wide complex rhythm and cardiac arrest. *Journal of Emergency Medicine*, *26*(2), 197–200. https://doi.org/10.1016/j.jemermed.2003.12.001

Pierce, D. A., Russell, G., & Pirkle, J. L., Jr. (2015). Incidence of hypoglycemia in patients with low eGFR treated with insulin and dextrose for hyperkalemia. *Annals of Pharmacotherapy*, *49*(12), 1322–1326. https://doi.org/10.1177/1060028015607559

Pollack, K., Manning, K. R., Balassone, J., Bui, C., Taylor, D. M., & Taylor, S. E. (2022). Hyperkalaemia in the emergency department: Epidemiology, management and monitoring of treatment outcomes. *Emergency Medicine Australasia*, *34*(5), 751–757. https://doi.org/10.1111/1742-6723.13971

Quick, G., & Bastani, B. (1994). Prolonged asystolic hyperkalemic cardiac arrest with no neurologic sequelae. *Annals of Emergency Medicine*, *24*(2), 305–311. https://doi.org/10.1016/s0196-0644(94)70144-x

Rafique, Z., Chouihed, T., Mebazaa, A., & Frank Peacock, W. (2019). Current treatment and unmet needs of hyperkalaemia in the emergency department. *European Heart Journal Supplements*, *21*(Suppl. A), A12–A19. https://doi.org/10.1093/eurheartj/suy029

Rafique, Z., Peacock, F., Armstead, T., Bischof, J. J., Hudson, J., Weir, M. R., & Neuenschwander, J. (2021). Hyperkalemia management in the emergency department: An expert panel consensus. *Journal of the American College of Emergency Physicians Open*, *2*(5), e12572. https://doi.org/10.1002/emp2.12572

Schaer, H., & Bachmann, U. (1974). Ionized calcium in acidosis: Differential effect of hypercapnic and lactic acidosis. *British Journal of Anaesthesia*, *46*(11), 842–848. https://doi.org/10.1093/bja/46.11.842

Schafers, S., Naunheim, R., Vijayan, A., & Tobin, G. (2012). Incidence of hypoglycemia following insulin-based acute stabilization of hyperkalemia treatment. *Journal of Hospital Medicine*, *7*(3), 239–242. https://doi.org/10.1002/jhm.977

Schwarz, K. C., Cohen, B. D., Lubash, G. D., & Rubin, A. L. (1959). Severe acidosis and hyperpotassemia

treated with sodium bicarbonate infusion. *Circulation*, *19*(2), 215–220. https://doi.org/10.1161/01.cir.19.2.215

Scott, N. L., Klein, L. R., Cales, E., & Driver, B. E. (2019). Hypoglycemia as a complication of intravenous insulin to treat hyperkalemia in the emergency department. *American Journal of Emergency Medicine*, *37*(2), 209–213. https://doi.org/10.1016/j.ajem.2018.05.016

Semple, P., & Booth, C. (1996). Calcium chloride: A reminder. *Anaesthesia*, *51*(1), 93. https://doi.org/10.1111/j.1365-2044.1996.tb07673.x

Singer, A. J., Thode, H. C. Jr., & Peacock, W. F. (2017). A retrospective study of emergency department potassium disturbances: Severity, treatment, and outcomes. *Clinical and Experimental Emergency Medicine*, *4*(2), 73–79. https://doi.org/10.15441/ceem.16.194

Singer, A. J., Thode, H. C. Jr., & Peacock, W. F. (2020). Rapid correction of hyperkalemia is associated with reduced mortality in ED patients. *American Journal of Emergency Medicine*, *38*(11), 2361–2364. https://doi.org/10.1016/j.ajem.2019.12.012

Soar, J., Perkins, G. D., Abbas, G., Alfonzo, A., Barelli, A., Bierens, J. J., Brugger, H., Deakin, C. D., Dunning, J., Georgiou, M., Handley, A. J., Lockey, D. J., Paal, P., Sandroni, C., Thies, K. C., Zideman, D. A., & Nolan, J. P. (2010). European Resuscitation Council Guidelines for Resuscitation 2010 Section 8. Cardiac arrest in special circumstances: Electrolyte abnormalities, poisoning, drowning, accidental hypothermia, hyperthermia, asthma, anaphylaxis, cardiac surgery, trauma, pregnancy, electrocution. *Resuscitation*, *81*(10), 1400–1433. https://doi.org/10.1016/j.resuscitation.2010.08.015

Stengl, M., Ledvinova, L., Chvojka, J., Benes, J., Jarkovska, D., Holas, J., Soukup, P., Sviglerova, J., & Matejovic, M. (2013). Effects of clinically relevant acute hypercapnic and metabolic acidosis on the cardiovascular system: An experimental porcine study. *Critical Care*, *17*(6), R303. https://doi.org/10.1186/cc13173

Sterns, R. H., Grieff, M., & Bernstein, P. L. (2016). Treatment of hyperkalemia: Something old, something new. *Kidney International*, *89*(3), 546–554. https://doi.org/10.1016/j.kint.2015.11.018

Sterns, R. H., Rojas, M., Bernstein, P., & Chennupati, S. (2010). Ion-exchange resins for the treatment of hyperkalemia: Are they safe and effective? *Journal of the American Society of Nephrology*, *21*(5), 733–735. https://doi.org/10.1681/ASN.2010010079

Truhlář, A., Deakin, C. D., Soar, J., Khalifa, G. E., Alfonzo, A., Bierens, J. J., Brattebø, G., Brugger, H., Dunning, J., Hunyadi-Antičević, S., Koster, R. W., Lockey, D. J., Lott, C., Paal, P., Perkins, G. D., Sandroni, C., Thies, K. C., Zideman, D. A., & Nolan, J. P.; Cardiac arrest in special circumstances section Collaborators. (2015). European Resuscitation Council Guidelines for Resuscitation 2015: Section 4. Cardiac arrest in special circumstances. *Resuscitation*, *95*, 148–201. https://doi.org/10.1016/j.resuscitation.2015.07.017

Wardi, G., Holgren, S., Gupta, A., Sobel, J., Birch, A., Pearce, A., Malhotra, A., & Tainter, C. (2023). A review of bicarbonate use in common clinical scenarios. *Journal of Emergency Medicine*, *65*(2), e71–e80. https://doi.org/10.1016/j.jemermed.2023.04.012

Wax, P. M., & Haynes, A. (2019). Sodium bicarbonate. In L. S. Nelson, M. A. Howland, N. A. Lewin, S. W. Smith, L. R. Goldfrank, & Robert S. Hoffman (Eds.), *Goldfrank's Toxicologic Emergencies* (pp. 567–573). McGraw-Hill Education.

Wheeler, D. T., Schafers, S. J., Horwedel, T. A., Deal, E. N., & Tobin, G. S. (2016). Weight-based insulin dosing for acute hyperkalemia results in less hypoglycemia. *Journal of Hospital Medicine*, *11*(5), 355–357. https://doi.org/10.1002/jhm.2545

Wu, Y. H., Chou, J. W., Lai, H. C., Su, G. S., Cheng, K. S., & Chen, T. W. (2021). Adverse gastrointestinal effects with kayexalate or kalimate: A comprehensive review. *Clinical and Experimental Gastroenterology*, *14*, 1–18. https://doi.org/10.2147/CEG.S278812

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