Prehospital Electrolyte Care

A Review of Symptoms, Evaluation, and Management

Andrea Painter, BHS, 38BW4, SO-ATP 1; Brandon M. Carius, DSc, MPAS2*

ABSTRACT

Ongoing evolution of prehospital medical care continues to advance beyond tactical field care scenarios in the consideration of prolonged field care. This is even more important to consider in theaters with extended evacuation times and limited local medical assets. The critical regulatory functions of electrolytes such as sodium, potassium, calcium, and glucose require medics operating in these environments to have a strong, fundamental knowledge of the principles, manifestations, and initial stabilization measures to aid their patients prior to, or in lieu of evacuation. Continued development and access to point of care testing in increasingly forward deployed settings further enables medics to perform these tasks. Here, we provide a brief review of these vital electrolytes, as well as additional kidney function evaluation considerations, to assist medics in their treatment efforts. Specific concerns for battlefield and atraumatic presentations are addressed.

**KEYWORDS:** military; laboratory; sodium; potassium; calcium; glucose; electrolytes; creatinine

Introduction

Electrolyte regulation underlies the basic function and stability of most body processes, allowing for cellular and physiologic homeostasis. Electrolyte evaluation and monitoring may not be considered a priority in prehospital patient management, however when available its importance in completing an assessment and treatment plan cannot be overstated. Traumatic and atraumatic patients can present with significant electrolyte derangements, ranging from mild and asymptomatic to severe and life-threatening. The current use of urgent evacuation and prolonged field care settings further enables medics to perform these tasks. Here, we present a brief review of basic electrolyte evaluation and management considerations, to assist medics in their treatment efforts. Specific concerns for battlefield and atraumatic presentations are addressed.

**TABLE 1 Basic Electrolyte Measurements**

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Normal Range</th>
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<tbody>
<tr>
<td>Sodium (Na+)</td>
<td>135–145mEq/L</td>
</tr>
<tr>
<td>Potassium (K+)</td>
<td>3.5–5.0mEq/L</td>
</tr>
<tr>
<td>Ionized Calcium (iCa)</td>
<td>Hypocalcemia &lt; 1.20 mmol/L</td>
</tr>
<tr>
<td>Glucose</td>
<td>70–99mg/dL fasting</td>
</tr>
<tr>
<td></td>
<td>70–140mg/dL nonfasting*</td>
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*Correspondence to brandon.m.carius.mil@mail.mil

SSG Andrea Painter is affiliated with the 92nd Civil Affairs Battalion, 1st Special Forces Command, Fort Bragg, NC. 2MAJ Brandon M. Carius is affiliated with Madigan Army Medical Center Emergency Department, JBLM Fort Lewis, WA, and the 121 Field Hospital, Camp Humphreys, Republic of Korea.
Sodium

Sodium (Na⁺), most commonly found in table salt (NaCl) and seawater, is also the primary electrolyte found in most routinely used resuscitative fluids, such as normal saline (0.9% NaCl) and lactated Ringer’s (LR). It is also used as the diluent for administration of medications, e.g., tranexamic acid via “piggyback.” Normal range for sodium is generally 135–145mEq/L, with hyponatremia defined as below 135mEq/L and hypernatremia as above 145mEq/L.⁵

Hyponatremia (Low Sodium)

Hyponatremia (Na⁺ <135mEq/L) is the most common electrolyte disorder, found in up to 30% of acutely ill patients and often related to the intense physical nature of military training.⁶⁻¹¹ Hyponatremia can be classified as hyperosmolar (increased fluid volume from ICF to ECF without enough commensurate increase in sodium), iso-osmolar (a displacement of ECF fluid by high protein or lipid levels), or hypoposmolar (impaired water excretion and increased reabsorption with significant interstitial space redistribution). In most active-duty persons in extreme environmental conditions, hyperosmolar hyponatremia will be the most common presentation, resulting from improper hydration with electrolyte-poor fluids (instead of sports drinks or oral rehydration salts) during intense physical activity.⁶,⁸,⁹,¹²,¹³ This is commonly referred to as “water intoxication” or “exercise-associated hyponatremia.”⁶,⁸,⁹,¹²,¹³ In addition to vague symptoms common to electrolyte imbalances, presentations specifically concerning for severe hyponatremia include seizures, presenting initially or in the course of management.⁶,⁸ Conversely, some patients may be asymptomatic.⁶ Monitoring for hyponatremia should be considered in burn patients due to loss of extracellular sodium caused by increased cellular permeability. This should be managed as per Joint Trauma System (JTS) clinical practice guidelines (CPG), however they are not mentioned in JTS CPGs for prolonged care.¹⁴

Severity may be labeled as mild (130–134mEq/L), moderate (120–129mEq/L) or severe (<120mEq/L), but these cut-offs are not universal, and some equate symptomatic hyponatremia as severe hyponatremia.⁵ Others propose hyponatremia is only clinically significant below 130mEq/L, and these patients constitute only about 10% of all hyponatremia cases.¹,⁵,⁶,¹¹ Once established, initial hyponatremia treatment balances electrolyte disorder, found in up to 30% of acutely ill patients and often related to the intense physical nature of military training.⁶⁻¹¹ Hyponatremia can be classified as hyperosmolar (increased fluid volume from ICF to ECF without enough commensurate increase in sodium), iso-osmolar (a displacement of ECF fluid by high protein or lipid levels), or hypoposmolar (impaired water excretion and increased reabsorption with significant interstitial space redistribution). In most active-duty persons in extreme environmental conditions, hyperosmolar hyponatremia will be the most common presentation, resulting from improper hydration with electrolyte-poor fluids (instead of sports drinks or oral rehydration salts) during intense physical activity.⁶,⁸,⁹,¹²,¹³ This is commonly referred to as “water intoxication” or “exercise-associated hyponatremia.”⁶,⁸,⁹,¹²,¹³ In addition to vague symptoms common to electrolyte imbalances, presentations specifically concerning for severe hyponatremia include seizures, presenting initially or in the course of management.⁶,⁸ Conversely, some patients may be asymptomatic.⁶ Monitoring for hyponatremia should be considered in burn patients due to loss of extracellular sodium caused by increased cellular permeability. This should be managed as per Joint Trauma System (JTS) clinical practice guidelines (CPG), however they are not mentioned in JTS CPGs for prolonged care.¹⁴

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Acute hyponatremia should be suspected in most military settings, with a treatment focus on free water restriction and sodium replacement. Hyponatremia in the alert patient can be treated with oral replacement therapy in the form of hypotonic IV fluids such as dextrose 5% (containing 0mEq/L of Na⁺), 0.45% NaCl (77mEq/L Na⁺), or LR (130mEq/L Na⁺) for likely hypovolemia.⁵,¹² Free water via nasogastric tube may be considered in the unconscious or intubated patient. Correction rates should be deliberate and controlled with serial lab draws, but no consensus on frequency exists, requiring telemedicine consult in prolonged field care.

Hypernatremia (Excess Sodium)

Hypernatremia (Na⁺ > 145mEq/L) describes a hyper-osmolar state with excess sodium in the ECF. Hypernatremia can be classified as hypervolemic (excessive sodium retention without commensurate water retention), isovolemic (loss of free water), or hypervolemic (decreased fluid volume from ICF to ECF without excretion of sodium). In military patients, hypernatremia could stem from excessive water loss in acute illness (fever, diarrhea), or low free water intake (due to nonavailability or poor hydration).²,⁴,¹²,¹¹ Isolated high sodium intake occurs less frequently.⁵ Additionally, environmental heat can trigger excessive water loss relative to sodium, to include thermal burn injuries.¹²,¹³

Hypernatremia symptoms may present vaguely, but more suggestive symptoms include excessive thirst and/or excessive urination (polyuria).²,²² Neurologic symptoms occur as brain cells shrink secondary to intracellular fluid shifts, which can trigger cerebral vascular rupture and intracranial hemorrhage, resulting in severe headache syndromes.²,¹ Hypovolemic hypernatremia present with extreme thirst complaints and an overall “dry” appearance, including abnormal skin turgor.²,²⁴ Conversely, patients may be completely asymptomatic.²,²⁵

Severity classifications are not universal, although concentrations greater than 160mEq/L generally establishes a “severe” diagnosis.⁷ Treatment of hypernatremia balances symptom severity with laboratory values and rates of correction. While overall volume replacement is generally the goal of treatment, correction should be pursued over a 24- to 48-hour period to avoid cerebral edema from rapid water movement into dehydrated brain cells.³,⁵ Despite variation in correction rates, most agree serum sodium should not be lowered more than 8–12mEq/L within a 24-hour period.²,³,⁵,¹¹,²² Alert patients can be encouraged to drink free water, while those unable to take oral fluids can be treated with hypertonic IV fluids such as dextrose 5% (containing 0mEq/L of Na⁺), 0.45% NaCl (77mEq/L Na⁺), or LR (130mEq/L Na⁺) for likely hypovolemia.⁵,¹² Free water via nasogastric tube may be considered in the unconscious or intubated patient. Correction rates should be deliberate and controlled with serial lab draws, but no consensus on frequency exists, requiring telemedicine consult in prolonged field care.

Potassium

Potassium (K⁺) is the most abundant intracellular cation, with over 75% of body stores found within skeletal muscle.
Hypokalemia (Low Potassium)

Hypokalemia ($K^+ < 3.5\text{mEq/L}$) commonly results from impaired renal regulation and retention of potassium, with much rarer instances of insufficient intake.\textsuperscript{27,29–31} Given pervasive hypertension diagnoses, and treatment to include the use of diuretics (such as hydrochlorothiazide), a brief medical history is important in suspected or diagnosed hypokalemia. However, in an otherwise healthy servicemember, more acute losses through vomiting and/or diarrhea are the most common causes of hypokalemia.\textsuperscript{27,30} Additionally, hypokalemia has been documented in military cases of environmental hypothermia and hyperthermia, the latter both with and without significant exertion.\textsuperscript{32–34}

Hypokalemia is often asymptomatic, but beyond vague electrolyte derangement symptoms, patients may present with numbness, tingling, and palpitations, with decreased deep tendon reflexes on exam.\textsuperscript{30,35,36} If electrocardiogram (ECG) is used, hypokalemia can manifest with decreased or flattened T waves and the growth of a subsequent U wave.\textsuperscript{27,30,35,37} As severity progresses, sinus bradycardia, ventricular tachycardia or fibrillation and torsades de pointes can develop.\textsuperscript{30,35,37,38} Cardiac monitoring should be used to monitor treatment effects when an abnormal ECG is present. Additionally, all patients with hypokalemia should be evaluated for magnesium levels, if available, as refractory hypokalemia can result from hypomagnesemia.\textsuperscript{30}

Hypokalemia treatments are dependent on level of severity, with a focus on identifying and treating underlying causes as well as direct electrolyte replacement. Severe hypokalemia can be defined as a serum level less than $2.5\text{mEq/L}$; however, ECG or other abnormal exam findings supersede laboratory values in making the diagnosis.\textsuperscript{30,36} Conversely, nonsevere hypokalemia can then be defined in patients with potassium above $2.5\text{mEq/L}$ without symptoms or ECG findings.

In stable, alert patients, oral potassium supplementation is preferred, although caution is warranted for gastric irritation and possible ulceration.\textsuperscript{29} Low dosages (20–40mEq tablets) should be diluted in other oral fluids to reduce this risk.\textsuperscript{30} IV potassium replenishment in more severe or otherwise unstable patients is given as a “piggyback” fluid with saline or LR due to its caustic nature on vasculature, but should not be given in dextrose-containing fluids as this can prolong hypokalemia due to triggered endogenous insulino release.\textsuperscript{30} For every 1mEq/L deficit, approximately 200–400mEq of potassium is required for correction.\textsuperscript{27,29,30} In non-severe hypokalemia, oral potassium tablets or IV doses of 10–20mEq/L can be given hourly, with no more than four doses in a 24-hour period.\textsuperscript{30} More severe cases require repeat up to 40mEq/L administered three to four times a day.\textsuperscript{27,29,30} Replacement with IV concentrations greater than 40mEq/L generally requires central venous access due to its vesicant effect in smaller vessels which can cause loss of IV access.\textsuperscript{29} Serum levels can be rechecked every 2–4 hours, although there is no firm consensus on frequency. Resuscitation should proceed slowly to avoid overcorrection and risk of rebound hyperkalemia (below).\textsuperscript{30,36}

Hyperkalemia (High Potassium)

Hyperkalemia ($K^+ > 5\text{mEq/L}$) has numerous causes, most commonly from pseudohyperkalemia, or a false elevation due to cell lysis during blood collection.\textsuperscript{30,40} Despite this, findings of hyperkalemia should not be lightly dismissed. Rhabdomyolysis is the most common cause of hyperkalemia in the setting of extreme exertion. This diagnosis is associated with diffuse muscle aches, as well as dark urine (myoglobinuria). Military trauma patients who have sustained battlefield injuries such as crush and burn injuries, should be evaluated for hyperkalemia, as high levels of ICF potassium are released into the ECF during tissue destruction.\textsuperscript{30,41,42} Specifically, prolonged field care of trauma patients are at risk of acute kidney injury (AKI) which may worsen hyperkalemia.\textsuperscript{41}

Hyperkalemia assessment focuses on cardiac manifestations with complaints of symptomatic palpitations, best evaluated through ECG and cardiac monitoring. Although not always present, peaked T waves are pathognomonic for hyperkalemia; however, ECG manifestations may also show PR interval prolongation, loss of p waves, and widening QR S as severity progresses.\textsuperscript{30,35,41,43} Conversely, ECGs may appear largely unremarkable despite the presence of significant hyperkalemia.\textsuperscript{35,42}

Regardless of mild ($K^+ = 5.5–6.5\text{mEq/L}$) or moderate-severe ($K^+ > 6.5\text{mEq/L}$) hyperkalemia classification, the initial evaluation of hyperkalemia in the mild or asymptomatic atraumatic patient should include consideration of repeat sampling to confirm initial findings and exclude pseudohyperkalemia.\textsuperscript{30,40} Once this is done, treatment should be initiated with concerns for short progression of severity and possibly fatal dysrhythmia manifestations from cardiac effects.\textsuperscript{29,30,42} A largely two-prong approach for treatment focuses on cellular membrane stabilization and transcellular shifting therapies (from ECF to ICF).

Calcium, generally given in the form of calcium gluconate (1g ampule via slow IV push), aids cardiac membrane stabilization and should be administered as a temporizing measure in the presence of ECG changes.\textsuperscript{30,41,44} Alternatively, 1g of calcium chloride can be given via IV push or diluted in a 50mL minibag and given over 10 minutes. The use of calcium gluconate to treat trauma-induced coagulopathy may make this intervention readily available, although calcium chloride can be given (via central line access only given its caustic nature and risk of tissue necrosis).\textsuperscript{30,41} Medic should be mindful that calcium supplementation is strictly for membrane stabilization, and will not significantly lower serum potassium levels.\textsuperscript{30,45} Calcium administration can be repeated after 5 minutes if there is no significant ECG changes.\textsuperscript{30,45}

Concurrent treatment to shift excess potassium from the ECF to ICF should be considered for continued patient management. These include the use of β-agonist medications, such as albuterol (20mg in 4mL of saline nebulized over 10 minutes), given to alert patients with positive airway control.\textsuperscript{30,41,44} Insulin (10–20 units IV) is highly recommended to drive potassium back into the ICF, but should be given concurrently with one ampule of $D_8W$ per 10U of regular insulin) to prevent...
hypocalcemia. Sodium bicarbonate (one 50mEq ampule IV push) may be considered, but is generally reserved for patients with concomitant acidosis (military literature specifically advises against its use unless blood pH is less than 7.2). Especially in cases of suspected rhabdomyolysis, IV fluids should be given often and early to replenish fluid losses and dilute ECF potassium. The diuretic furosemide may be considered to aid renal excretion, but this should be balanced against suspected renal injury, as abnormally high doses may be required to attain effect. Although unlikely to be available in remote settings, medics may consider the use of potassium removing oral agents such as sodium polystyrene sulfonate (better known as Kayexalate), although some caution may be warranted given a few extremely rare case reports of bowel necrosis.

Potassium reevaluation frequency is scattered but can be considered at 1- to 4-hour intervals or longer depending on severity and evaluation capacity. Continuous cardiac monitoring should be utilized until potassium levels return to normal ranges, with repeat ECGs performed with subsequent laboratory evaluation.

**Calcium and Hypocalcemia**

Calcium (Ca^{2+}) helps regulate cellular membrane stability, especially important in cardiac muscle. Prehospital point of care testing generally measures calcium not bound to proteins, known as ionized calcium (iCa). In military medicine, calcium loss is primarily concerning in traumatic hemorrhage given its coagulation cascade prominence and the “lethal diamond” resulting when combined with hypothermia, acidosis, and coagulopathy that demonstrates increased patient mortality. Definitions of hypocalcemia vary (generally iCa < 4.5mg/dL or < 1.2 mmol/L), but severity focuses beyond laboratory cut-offs to include symptomatic progression, cardiac findings, and hemorrhage implications. The transfusion of chelated blood products risks citrate toxicity and worsening hypocalcemia in hemorrhage. Acute evaluation in atraumatic patients may reveal perioral paresthesias, muscle cramping, Chvostek’s sign (facial muscle twitching with facial nerve tapping), and Trouseau’s sign (involuntary wrist flexion when blood pressure cuff is inflated 20mmHg above systolic blood pressure for 3 minutes). Hypocalcemia-induced prolonged cardiac cell repolarization manifests as QT prolongation on ECG, which can progress to lethal torsades de pointes if untreated.

Treatmen of hypocalcemia in the hemorrhagic shock patient should be initiated immediately with blood product resuscitation. JTS guidelines advise for initial dosing of 1g of calcium (as 30mL of 10% calcium gluconate or 10mL of 10% calcium chloride) with initial transfusion, and an additional gram after every 4 units of blood products are given. Although calcium monitoring is recommended, no specified frequency is recommended in military literature. Additional measurements may be considered with each calcium dose, specifically with every 4 units of blood products given, and after any cardiac changes on monitoring, in order to maintain an iCa > 1.2 mmol/L.

Glucose

Glucose is the body’s primary energy source, generally obtained from carbohydrate breakdown or hepatic gluconeogenesis. It additionally functions as a vital cotransporter for cellular electrolyte movement. Blood glucose levels are primarily regulated through a balance of insulin, facilitating cellular storage, and glucagon, which mobilizes stored energy for conversion to glucose (gluconeogenesis). Both hormones are produced by the pancreas. Normal glucose levels generally range between 70mg/dL and 99mg/dL in a fasted state and less than 140mg/dL in a non-fasted state, but random levels can generally be as high as 200mg/dL without concerns. Dysregulation of blood glucose primarily results from diabetes mellitus, occurring from an outright lack of insulin (type 1) or insulin receptor resistance (type 2). As diabetic patients can require specialty care and may quickly deteriorate in the setting of trauma or disease, the diagnosis is considered a bar to deployment status and by regulation results in submission for medical discharge. However, the remote medic should not therefore assume all military patients do not have these conditions, as both can present after entering active service, and can be present in co-located civilian counterparts.

**Hypoglycemia (Low Blood Glucose)**

The most common cause of hypoglycemia is overdose of diabetes controlling medications. While recent strenuous activity, dietary restrictions, and starvation may suggest the diagnosis, this information may be unavailable on initial evaluation. Hypoglycemia (blood glucose < 70mg/dL) can manifest with diaphoresis, palpitations, and behavioral and neurocognitive changes (given the brain’s high glucose consumption relative to other body systems). While 70mg/dL is often the cut-off for diagnosis, more severe symptoms generally present with levels below 55mg/dL. Immediate treatment depends largely on patient cognition. If able, oral supplementation with 15–20g of simple carbohydrates is recommended via an apple juice box or 2 tablespoons of cake frosting or table honey. Resourceful medics can likewise use small amounts of candy or other carbohydrate-rich beverages. Patients unable to tolerate oral supplementation (severely altered or impaired) should be treated with IV glucose. While 1 ampule of dextrose 50% (D50W) is traditionally recommended, administration should be done only via 18g or larger angiocatheter, with caution for risk of extravasated tissue necrosis with if extravasation occurs, as well as for rebound hypoglycemia. Lesser complication concerns exist for IV D10W, though a greater volume is required to have a significant effect.

Hyperglycemia (random blood glucose > 200mg/dL) may manifest with excessive thirst, frequent urination or less specific symptoms. This can occur secondary to insulin dysregulation (undiagnosed or improperly treated diabetes mellitus) or stress release of the hormone cortisol as occurs secondary to infection. Evaluation includes search for underlying cause, with further evaluation (including venous blood gas and lactate, if available) to exclude diabetic ketoacidosis and hyperosmolar hyperglycemia state. The use of IV fluids may help initially, but persistent hyperglycemia generally requires short-acting insulin (0.1mg/kg given subcutaneously or intravenously). Particular care should be made to evaluate for potassium levels prior to insulin administration, as insulin can shift potassium and cause hypokalemia (described above). Labs should be repeated every 30–120 minutes, depending on presentation, and additionally within 20–30 minutes after completion of interventions. Medics should note that subsequent blood sugar levels below 200mg/dL are a relative goal, and further evaluation and management should include specialty consultation.

**Creatinine and Blood Urea Nitrogen**

Though not common to all point of care testing, evaluation for overall renal dysfunction can help explain electrolyte...
derangements, and if available, should be pursued. Renal processing of metabolic wastes depends on the proper function of the glomerular filtration rate (GFR), a measurement not available with most point of care testing. However, serum creatinine (Cr) serves as an effective marker of kidney function, helping to establish a diagnosis of AKI, with assistance from blood urea nitrogen (BUN) levels. A byproduct of natural muscle catabolism, creatinine is continuously produced and found in a normal range of 0.6–1.2mg/dL. The constant clearance of circulatory wastes (with low and stable levels) signifies healthy renal function, while elevated levels establish AKI. Military literature varies on AKI incidence, with 13–34% of mild to moderate battlefield casualties, but no significant trends for disease and non-battle injuries. Additionally, rhabdomyolysis and crush injuries can cause significant AKI through traumatic cellular destruction.

Elevated creatinine levels indicate AKI with varying severity generally on a three-tier system alongside urine output (UOP) findings (Tables 2 and 3). While UOP levels are diagnostically helpful, they are best used in cases of prolonged care and are difficult to quantify in the initial evaluation of the acutely presenting patient. The use of creatinine as a surrogate marker for GFR, and subsequent diagnosis of AKI, has been repeatedly trialed for diagnostic calculators, including the Acute Kidney Injury Network (AKIN) classification system for patients without prior kidney disease. As part of initial patient evaluation, medics should be aware of supplemental creatine is popular amongst military populations, and can result in artificially elevated serum levels. Table 3 AKI Staging

### TABLE 3 AKI Staging

<table>
<thead>
<tr>
<th>Stage</th>
<th>Serum Creatinine</th>
<th>Urine Output</th>
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<tbody>
<tr>
<td>1</td>
<td>Multiplier from baseline</td>
<td>Increase</td>
</tr>
<tr>
<td>2</td>
<td>Multiplier from baseline</td>
<td>Increase</td>
</tr>
<tr>
<td>3</td>
<td>Multiplier from baseline</td>
<td>Increase</td>
</tr>
</tbody>
</table>

During the course of initial evaluation, medics and telemedicine consultants may find it helpful to utilize a BUN to creatinine ratio model, which helps discriminate prerenal causes, such as dehydration, from those intrinsic to the kidney itself. Urea helps to regulate fluid and sodium levels in conjunction with the kidneys. Normal BUN levels range from 5–20mg/dL. A reduction in BUN excretion can stem from dehydration, increasing serum levels disproportionately above that of serum creatinine. Therefore, an elevated BUN/creatinine ratio greater than 20:1 is suggestive of prerenal azotemia, generally secondary to dehydration. However, medics should be aware that significant gastrointestinal bleeding (from peptic ulcer disease, cancer, or other causes) can increase BUN secondary to intestinal blood cell digestion. Additionally, other emergent causes of BUN elevation should be considered, including exogenous corticosteroid use (dexamethasone, prednisone, methylprednisolone), general muscle catabolism (recent weight loss), and increased protein intake (namely from red meats). A BUN/creatinine ratio less than 20:1 suggests intrinsic renal failure, which can result from a number of causes. These include acute tubular necrosis from ischemic injury or toxins, glomerulonephritis from infection complications (streptococcal pharyngitis) or idiopathic complications. Ischemic injury can result from decreased renal perfusion secondary to severe hypovolemia, especially prominent in cases of traumatic hemorrhage.

While there are numerous concerning causes for AKI described above, in the absence of trauma immediate concerns for remote medicine should focus on likely prerenal causes, primarily dehydration, and subsequent treatment with IV fluids. The liberal use of IV crystalloids for resuscitation can significantly improve subsequent laboratory findings and the overall clinical picture, and may begin with clinical suspicion of diagnosis or risk of AKI given non-hemorrhagic presentation (to include initial treatment of crush injuries), but vigilance for comprehensive patient evaluation and continued monitoring is important. Laboratory levels may temporarily improve after IV fluids, but medics must continue to consider and evaluate for other underlying causes with repeated laboratory checks every 2–4 hours to evaluate for fluid responsiveness in addition to consultation depending on suspected mechanism.

### Conclusion

With emergent evaluation in austere environments and the possibility of protracted remote care in prolonged field care scenarios, medics should be prepared to assess and treat common electrolyte derangements. The increasing availability of point-of-care testing devices for electrolytes necessitates concomitant increased knowledge of these pathologies and initial stabilization modalities.

### Conflicts of Interest

The authors have no conflicts of interest or relevant disclosures to report.

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None.

### Disclaimer

The views expressed herein are those of the authors and do not reflect the official policy or position of Madigan Army Medical Center, US Army Institute of Surgical Research, the US Army Medical Department, the US Army Office of the Surgeon General, the Department of the Army, or the Department of Defense of the US Government.
Author Contributions
AP and BMC conceived the review concept, wrote the first draft, and read, provided critical revisions, and approved the final manuscript.

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