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# Circulation

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## ECC Guidelines

## Part 8: Advanced Challenges in Resuscitation

### Section 1: Life-Threatening Electrolyte Abnormalities

#### ▶ Introduction

Electrolyte abnormalities are commonly associated with cardiovascular emergencies. These abnormalities may cause or contribute to cardiac arrest and may hinder resuscitative efforts. It is important to identify clinical situations in which electrolyte problems may be expected. In some cases therapy for life-threatening electrolyte disorders should be initiated even before laboratory results become available.

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#### ▶ Potassium

The magnitude of the potassium gradient across cell membranes determines excitability of nerve and muscle cells, including the myocardium. Minor changes in serum potassium concentration can have major effects on cardiac rhythm and function. Of all the electrolytes, rapid changes in potassium concentration can cause the most immediate life-threatening consequences.

Evaluation of serum potassium must consider the effects of changes in serum pH. When serum pH falls, serum potassium rises because potassium shifts from the cellular to the vascular space. When serum pH rises, serum potassium falls because potassium shifts intracellularly. *In general, serum  $K^+$  decreases by approximately 0.3 mEq/L for every 0.1 U increase in pH above normal.* Effects of pH changes on serum potassium should be anticipated during evaluation and therapy for hyperkalemia or hypokalemia. Correction of an alkalotic pH will produce an increase in

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serum potassium even without administration of additional potassium. If serum potassium is "normal" in the face of acidosis, a fall in serum potassium should be anticipated when the acidosis is corrected, and potassium administration should be planned.

### **Hyperkalemia**

Hyperkalemia is defined as serum potassium concentration above the normal range of 3.5 to 5.0 mEq/L. Hyperkalemia is most frequently caused by increased  $K^+$  release from cells or by impaired excretion by the kidneys (see Table 1<sup>1</sup>). The most common clinical presentation of severe hyperkalemia involves patients with end-stage renal failure. These patients may present with severe weakness or arrhythmias.

**View this table:** **Table 1.** Causes of Hyperkalemia  
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Medications may also contribute to development of hyperkalemia, particularly in the presence of impaired renal function. Not surprisingly, potassium supplements commonly prescribed to prevent hypokalemia may lead to potassium overload. Potassium-sparing diuretics such as spironolactone, triamterene, and amiloride are well-recognized causes of hyperkalemia. Use of angiotensin-converting enzyme (ACE) inhibitors (eg, captopril) can also lead to elevation of serum potassium, particularly when combined with oral potassium supplements. Nonsteroidal anti-inflammatory medicines (eg, ibuprofen) can cause hyperkalemia through direct effects on the kidney. Identification of potential causes of hyperkalemia will contribute to rapid identification and treatment of patients who may be experiencing hyperkalemic cardiac arrhythmias.<sup>1 2 3</sup>

Changes in pH inversely affect serum potassium. Acidosis (low pH) leads to an extracellular shift of potassium, thus raising serum potassium. Conversely, high pH (alkalosis) shifts potassium back into the cell, lowering serum potassium.

Physical symptoms of hyperkalemia include ECG changes, weakness, ascending paralysis, and respiratory failure. ECG changes suggestive of hyperkalemia include

- Peaked T waves (tenting)
- Flattened P waves
- Prolonged PR interval (first-degree heart block)
- Widened QRS complex
- Deepened S waves and merging of S and T waves
- Idioventricular rhythm
- Sine-wave formation
- VF and cardiac arrest

Tenting of T waves is one of the prominent early ECG changes. If untreated, hyperkalemia causes progressive heart dysfunction, leading to sine waves and finally to asystole. Aggressive therapy should begin as soon as possible to improve outcome.

### Treatment of Hyperkalemia

Treatment of hyperkalemia depends on level of severity and the patient's clinical condition:

- *Mild elevation* (5 to 6 mEq/L): Remove potassium from the body
  1. Diuretics—furosemide 1 mg/kg IV slowly
  2. Resins—Kayexalate 15 to 30 g in 50 to 100 mL of 20% sorbitol either orally or by retention enema (50 g of Kayexalate)
  3. Dialysis—peritoneal or hemodialysis
- *Moderate elevation* (6 to 7 mEq/L): Also shift potassium intracellularly by using
  1. Sodium bicarbonate—50 mEq IV over 5 minutes
  2. Glucose plus insulin—mix 50 g glucose and 10 U regular insulin and give IV over 15 to 30 minutes
  3. Nebulized albuterol 10 to 20 mg nebulized over 15 minutes
- *Severe elevation* (>7 mEq/L with toxic ECG changes)
  1. Calcium chloride—10% 5 to 10 mL IV over 2 to 5 minutes to antagonize the toxic effects of potassium at the myocardial cell membrane (lowers risk of ventricular fibrillation [VF]).
  2. Sodium bicarbonate—50 mEq IV over 5 minutes (may be less effective for patients with end-stage renal disease)<sup>4 5 6</sup>
  3. Glucose plus insulin—mix 50 g glucose and 10 U regular insulin and give IV over 15 to 30 minutes
  4. Nebulized albuterol—10 to 20 mg nebulized over 15 minutes
  5. Diuresis (furosemide—40 to 80 mg IV)
  6. Kayexalate enema
  7. Dialysis

### Hypokalemia

Hypokalemia is defined as a serum potassium level <3.5 mEq/L. As with hyperkalemia, nerves and muscles (including the heart) are most affected by hypokalemia, particularly if the patient has other, preexisting disease (such as coronary artery disease).

Hypokalemia results from one or more of the following: decreased dietary intake, shift into cells, or increased net loss from the body. The most common causes of low serum potassium include gastrointestinal loss (diarrhea, laxatives), renal loss (hyperaldosteronism, potassium-losing diuretics, carbenicillin, sodium penicillin, amphotericin B), intracellular shift (alkalosis or a rise

in pH), and malnutrition. Symptoms of hypokalemia include weakness, fatigue, paralysis, respiratory difficulty, muscle breakdown (rhabdomyolysis), constipation, paralytic ileus, and leg cramps.

Hypokalemia is suggested by changes in the ECG, including

- U waves
- T-wave flattening
- ST-segment changes
- Arrhythmias (especially if the patient is taking digoxin)
- Pulseless electrical activity (PEA) or asystole

Hypokalemia exacerbates digitalis toxicity. Thus, hypokalemia should be avoided or treated promptly in patients receiving digitalis derivatives.

#### **Treatment of Hypokalemia**

The treatment of hypokalemia includes minimizing further potassium loss and giving potassium replacement. IV administration of potassium is indicated when arrhythmias are present or hypokalemia is severe ( $K^+ < 2.5$  mEq/L).

Acute potassium administration may be empirical in emergent conditions. When indicated, maximum IV  $K^+$  replacement should be 10 to 20 mEq/h with continuous ECG monitoring during infusion. Central or peripheral IV sites may be used. A more concentrated solution of potassium may be infused if a central line is used, but the catheter tip should not extend into the right atrium.

If cardiac arrest from hypokalemia is imminent (ie, malignant ventricular arrhythmias), rapid replacement of potassium is required. Give an initial infusion of 2 mEq/min, followed by another 10 mEq IV over 5 to 10 minutes. *In the patient's chart, document that rapid infusion is intentional in response to life-threatening hypokalemia.* Once the patient is stabilized, reduce the infusion to continue potassium replacement more gradually.

Estimates of total body deficit of potassium range from 150 to 400 mEq for every 1-mEq decrease in serum potassium. The lower range of the estimate would be appropriate for an elderly woman with low muscle mass and the higher range for a young, muscular man. Gradual correction of hypokalemia is preferable to rapid correction unless the patient is clinically unstable.

## **Sodium**

Sodium is the major positively charged ion in the extracellular space and the major intravascular ion that influences serum osmolality. An acute increase in

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serum sodium will produce an acute increase in serum osmolality; an acute decrease in serum sodium will produce an acute fall in serum osmolality.

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Under normal conditions sodium concentration and osmolality equilibrate across the vascular membrane. *Acute* changes in serum sodium will produce acute free water shifts into and out of the vascular space until osmolality equilibrates in these compartments. An acute fall in serum sodium and an acute fluid shift into the interstitial space may cause cerebral edema.<sup>7 8</sup> An acute rise in serum sodium will produce an acute shift of free water from the interstitial to the vascular space. Rapid correction of hyponatremia has been associated with development of pontine myelinolysis and cerebral bleeding.<sup>9 10 11</sup> For these reasons, monitor neurological function closely in the patient with hypernatremia or hyponatremia and during correction of these conditions. Whenever possible, correct serum sodium slowly, carefully controlling the absolute magnitude of change in serum sodium over 48 hours and avoiding overcorrection.<sup>12 13</sup>

### **Hypernatremia**

Hypernatremia is defined as a serum sodium concentration above the normal range of 135 to 145 mEq/L. Hypernatremia may be caused by a primary Na<sup>+</sup> gain or excess water loss. A common cause of hypernatremia is free water loss in excess of sodium loss, such as that which occurs with diabetes insipidus or hypernatremic dehydration.

Hypernatremia produces a free water shift from the interstitial to the vascular space. Hypernatremia also causes water to shift out of cells, leading to decreased intracellular volume. In the brain, decreased nerve cell volume can cause neurological symptoms, including altered mental status, weakness, irritability, focal neurological deficits, and even coma or seizures.

The hypernatremic patient usually complains of excessive thirst. The severity of symptoms depends on how acute and how great the increase in serum sodium is. If the sodium rises quickly or to a very high level, signs and symptoms will be more severe.

#### **Treatment of Hypernatremia**

To treat hypernatremia, it is important to stop ongoing water losses (by treating the underlying cause) while correcting the water deficit. In hypovolemic patients the extracellular fluid (ECF) volume must be restored with normal saline.

The quantity of water needed to correct hypernatremia can be calculated by the following equation: Water deficit

$$= \frac{\text{plasma Na}^+ \text{ concentration} - 140}{140} \times \text{total body water.}$$

Total body water is approximately 50% of lean body weight in men and 40% in women. For example, if a 70-kg man had a serum Na<sup>+</sup> level of 160 mmol/L, the estimated free water deficit would be

$$\frac{160-140}{140} \times (0.5 \times 70) = 5 \text{ L.}$$

Once the free water deficit is calculated, administer fluid to lower serum sodium at a rate of 0.5 to 1.0 mEq/h with a decrease of no more than 12 mmol in the first 24 hours. Total correction should be achieved over 48 to 72 hours. The method of replacement of free water depends on the patient's clinical status. For stable, asymptomatic patients, replacement of fluid by mouth or through a nasogastric tube is effective and safe. If this is not possible or if the patient's clinical status demands more aggressive treatment, 5% dextrose in half-normal saline may be given IV. Check the patient's serum sodium and neurological function frequently to avoid overly rapid correction.

### **Hyponatremia**

Hyponatremia is defined as a serum sodium concentration below the normal range of 135 to 145 mEq/L. It is caused by an excess of water relative to sodium. Most cases of hyponatremia are caused by reduced renal excretion of water with continued water intake. Impairment of renal water excretion may be due to

- Use of thiazide diuretics
- Renal failure
- ECF depletion (eg, vomiting with continued water intake)
- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- Edematous states (congestive heart failure, cirrhosis with ascites, etc)
- Hypothyroidism
- Adrenal insufficiency
- "Tea and toast diet" or excessive beer drinking (diminished solute intake)

Most cases of hyponatremia are associated with low serum osmolality (so-called hypo-osmolar hyponatremia). The one common exception to this is in uncontrolled diabetes, in which hyperglycemia leads to a hyperosmolar state, whereas serum sodium is below normal (hyperosmolar hyponatremia).

Hyponatremia is usually asymptomatic unless it is acute or severe (<120 mEq/L). An abrupt fall in serum sodium produces a free water shift from the vascular to the interstitial space that can cause cerebral edema. In this case the patient may present with nausea, vomiting, headache, irritability, lethargy, seizures, coma, or even death.

SIADH is an important cause of potentially life-threatening hyponatremia. It can occur in a wide variety of clinical situations. SIADH can complicate a variety of conditions common to ACLS patients, including trauma, increased intracranial pressure, cancer, and respiratory failure.

#### **Treatment of Hyponatremia**

Treatment of hyponatremia involves administration of sodium and elimination of intravascular free water. If SIADH is present, the treatment is strict restriction of fluid intake to 50% to 66% of

maintenance fluids.

Correction of asymptomatic hyponatremia should be gradual: usually an increase in  $\text{Na}^+$  of 0.5 mEq/L per hour to a maximum change of 10 to 15 mEq/L in the first 24 hours. Rapid correction of hyponatremia can cause *pontine myelinolysis*, a lethal disorder thought to be caused by rapid fluid shifts in the brain.<sup>9 10 11</sup>

If the patient demonstrates neurological compromise, urgent administration of 3% saline IV at a rate of 1 mEq/L per hour is necessary to correct hyponatremia until neurological symptoms are controlled. Thereafter, continued correction should be at a rate of 0.5 mEq/L per hour to raise serum sodium.

Ultimate correction of serum sodium requires calculation of the sodium deficit. The following formula may be used:  $\text{Na}^+$  deficit = (desired  $[\text{Na}^+]$  - current  $[\text{Na}^+]$ )  $\times$  0.6\*  $\times$  body wt (kg) (\*Use 0.6 for men, 0.5 for women.)

Once the deficit is estimated, determine the volume of 3% saline (513 mEq  $\text{Na}^+$ /L) necessary to correct the deficit (divide the deficit by 513 mEq/L). Plan to increase the sodium by 1 mEq/L per hour over 4 hours. Check serum sodium frequently and monitor neurological status closely.

## ► Magnesium

Magnesium is the fourth most common mineral in the human body, but it is also the most frequently overlooked clinically. One third of extracellular magnesium is bound to serum albumin. Therefore, serum magnesium levels are not reliable predictors of total body magnesium stores. Magnesium is required for the action of many important enzymes and hormones. It is necessary for the movement of sodium, potassium, and calcium into and out of cells. In fact, when a patient is hypomagnesemic, it is impossible to correct intracellular potassium deficiency. Magnesium is also important in stabilizing excitable membranes and useful for atrial and ventricular arrhythmias.<sup>14</sup>

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### Hypermagnesemia

Hypermagnesemia is defined as a serum magnesium concentration above the normal range of 1.3 to 2.2 mEq/L. Magnesium balance is influenced by many of the same regulatory systems that control calcium balance. In addition, magnesium balance is influenced by diseases and factors that control serum potassium. As a result, magnesium balance is closely tied to both calcium and potassium balance.

The most common cause of hypermagnesemia is renal failure. Hypermagnesemia may also be iatrogenic (caused by overuse of magnesium) or caused by a perforated viscus with continued intake of food and use of laxatives/antacids containing magnesium (an important cause in the

elderly).

Neurological symptoms of hypermagnesemia include muscular weakness, paralysis, ataxia, drowsiness, and confusion. Gastrointestinal symptoms include nausea and vomiting. Moderate hypermagnesemia can produce vasodilation, and severe hypermagnesemia can produce hypotension. Extremely high serum magnesium levels may produce a depressed level of consciousness, bradycardia, hypoventilation, and cardiorespiratory arrest.<sup>14</sup>

ECG changes of hypermagnesemia include

- Increased PR and QT intervals
- Increased QRS duration
- Variable decrease in P-wave voltage
- Variable degree of T-wave peaking
- Complete AV block, asystole

#### **Treatment of Hypermagnesemia**

Hypermagnesemia is treated by antagonizing magnesium with calcium, removing magnesium from serum, and eliminating sources of ongoing magnesium intake. Cardiorespiratory support may be needed until magnesium levels are reduced. Administration of calcium chloride (5 to 10 mEq IV) will often correct lethal arrhythmias. This dose may be repeated if needed.

Dialysis is the treatment of choice for treatment of hypermagnesemia. Until that can be done, if renal function is normal and cardiovascular function adequate, IV saline diuresis (IV normal saline and furosemide [1 mEq/kg]) can be used to hasten elimination of magnesium from the body. However, this diuresis can also increase calcium excretion; the development of hypocalcemia will make signs and symptoms of hypermagnesemia worse. While treatment continues, the patient may require cardiorespiratory support.

#### **Hypomagnesemia**

Hypomagnesemia is far more common clinically than hypermagnesemia. Defined as a serum magnesium concentration below the normal range of 1.3 to 2.2 mEq/L, hypomagnesemia usually results from decreased absorption or increased loss, either from the kidneys or intestines (diarrhea). Alterations in parathyroid hormone and certain medications (eg, pentamidine, diuretics, alcohol) can also induce hypomagnesemia. Lactating women are at higher risk of developing hypomagnesemia.<sup>15</sup>

The various causes of hypomagnesemia are listed in Table 2<sup>15</sup>.

**View this table:** **Table 2.** Causes of Hypomagnesemia  
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The principal signs of hypomagnesemia are neurological, although interesting research has tied together the neurological and cardiac effects of magnesium.<sup>16</sup> Hypomagnesemia interferes with the effects of parathyroid hormone, resulting in hypocalcemia. It may also cause hypokalemia. Symptoms of low serum magnesium include muscular tremors and fasciculations, ocular nystagmus, tetany, and altered mentation. Other possible symptoms include ataxia, vertigo, seizures, and dysphagia. A number of ECG abnormalities occur with low magnesium levels, including

- Prolonged QT and PR intervals
- ST-segment depression
- T-wave inversion
- Flattening or inversion of precordial P waves
- Widening of QRS
- Torsades de pointes
- Treatment-resistant VF (and other arrhythmias)
- Worsening of digitalis toxicity<sup>17</sup>

#### Treatment of Hypomagnesemia

Treatment of hypomagnesemia depends on its severity and the patient's clinical status. For severe or symptomatic hypomagnesemia, administer 1 to 2 g IV MgSO<sub>4</sub> over 15 minutes. If torsades de pointes are present, administer 2 g of MgSO<sub>4</sub> over 1 to 2 minutes. If seizures are present, administer 2 g IV MgSO<sub>4</sub> over 10 minutes. Calcium gluconate administration (1 g) is usually appropriate because most patients with hypomagnesemia are also hypocalcemic.<sup>18</sup>

*Replace magnesium cautiously in patients with renal insufficiency because there is a real danger of causing life-threatening hypermagnesemia.*

## ► Calcium

Calcium is the most abundant mineral in the body. It is essential for bone strength and neuromuscular function and plays a major role in myocardial contraction. Half of all calcium in the ECF is bound to albumin; the other half is in the biologically active, ionized form. The ionized form is most active.

The serum ionized calcium level must be evaluated in light of serum pH and serum albumin. The concentration of ionized calcium is pH-dependent. Alkalosis increases the binding of calcium to albumin and thus reduces ionized calcium. Conversely, the development of acidosis will produce an increase in the ionized calcium level.

Total serum calcium is dependent on serum albumin concentration. Serum calcium changes in the same direction as a change in albumin (adjust total serum calcium by 0.8 mg/dL for every 1 g/dL

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change in serum albumin). Although total serum albumin is directly related to total serum calcium, the ionized calcium is inversely related to serum albumin. The lower the serum albumin, the higher the ionized calcium. In the presence of hypoalbuminemia, although total calcium level may be low, the ionized calcium level may be normal.

Calcium antagonizes the effects of both potassium and magnesium at the cell membrane. Therefore, it is extremely useful for treating the effects of hyperkalemia and hypermagnesemia.

Calcium concentration is normally closely regulated by PTH and vitamin D. When such control fails, a wide variety of clinical problems occur.

### **Hypercalcemia**

Hypercalcemia is defined as a serum calcium concentration above the normal range of 8.5 to 10.5 mEq/L (or an elevation in ionized calcium above 4.2 to 4.8 mg/dL). Primary hyperparathyroidism and malignancy account for >90% of reported cases.<sup>19</sup> In these and most forms of hypercalcemia, calcium release from the bone and intestines is increased, and renal clearance may be compromised.

Symptoms of hypercalcemia usually develop when the total serum calcium concentration reaches or exceeds 12 to 15 mg/dL. Neurological symptoms include depression, weakness, fatigue, and confusion at lower levels. At higher levels patients may exhibit hallucinations, disorientation, hypotonicity, and coma. Hypercalcemia interferes with renal concentration of urine, causing dehydration to develop.

Cardiovascular symptoms of elevated calcium levels are variable. Myocardial contractility may initially increase until the calcium level reaches 15 to 20 mg/dL. Above this level myocardial depression occurs. Automaticity is decreased and ventricular systole is shortened. Arrhythmias occur because the refractory period is shortened. Digitalis toxicity is worsened. Hypertension is common. In addition, many patients with hypercalcemia develop hypokalemia; these conditions both contribute to cardiac arrhythmias.<sup>20</sup>

ECG changes of hypercalcemia include

- Shortened QT interval (usually when  $\text{Ca}^{2+}$  is >13 mg/dL)
- Prolonged PR and QRS intervals
- Increased QRS voltage
- T-wave flattening and widening
- Notching of QRS
- AV block: progresses to complete heart block, then to cardiac arrest when serum calcium is >15 to 20 mg/dL

Gastrointestinal symptoms of hypercalcemia include dysphagia, constipation, peptic ulcers, and pancreatitis. Effects on the kidney include diminished ability to concentrate urine; diuresis,

leading to loss of sodium, potassium, magnesium, and phosphate; and a vicious circle of calcium reabsorption that further worsens hypercalcemia.

#### **Treatment of Hypercalcemia**

If hypercalcemia is due to malignancy, careful consideration of the patient's prognosis and wishes is needed. If the patient is in the last stages of death, hypercalcemia need not be treated. In all other cases, however, treatment should be rapid and aggressive.

Treatment for hypercalcemia is required if the patient is symptomatic (typically a concentration of approximately 12 mg/dL). Treatment is instituted at a level >15 mg/dL regardless of symptoms. Immediate therapy is directed at promoting calcium excretion in the urine. This is accomplished in patients with adequate cardiovascular and renal function with infusion of 0.9% saline at 300 to 500 mL/h until any fluid deficit is replaced and diuresis occurs (urine output  $\geq$ 200 to 300 mL/h). Once adequate rehydration has occurred, the saline infusion rate is reduced to 100 to 200 mL/h. This diuresis will further reduce serum potassium and magnesium concentrations, which may increase the arrhythmogenic potential of the hypercalcemia. Thus, potassium and magnesium concentrations should be closely monitored and maintained.

Hemodialysis is the treatment of choice to rapidly decrease serum calcium in patients with heart failure or renal insufficiency.<sup>21</sup> Chelating agents may be used for extreme conditions (eg, 50 mmol PO<sub>4</sub> over 8 to 12 hours or EDTA 10 to 50 mg/kg over 4 hours).

Use of furosemide (1 mg/kg IV) during treatment of hypercalcemia is controversial. In the presence of heart failure, furosemide administration is required, but it can actually foster reuptake of calcium from bone, thus worsening hypercalcemia. Calcium may also be lowered by drugs that reduce bone resorption (eg, calcitonin, glucocorticoids). A discussion of this therapy is beyond the scope of these guidelines.

#### **Hypocalcemia**

Hypocalcemia is defined as a serum calcium concentration below the normal range of 8.5 to 10.5 mg/dL (or an ionized calcium below the range of 4.2 to 4.8 mg/dL). Hypocalcemia may develop with toxic shock syndrome, abnormalities in serum magnesium, and tumor lysis syndrome (rapid cell turnover with resultant hyperkalemia, hyperphosphatemia, and hypocalcemia). Calcium exchange is dependent on concentrations of potassium and magnesium, so treatment depends on replacing all 3 electrolytes.

Symptoms of hypocalcemia usually occur when ionized levels fall below 2.5 mg/dL. Symptoms include paraesthesias of the extremities and face, followed by muscle cramps, carpopedal spasm, stridor, tetany, and seizures. Hypocalcemic patients demonstrate hyperreflexia and positive Chvostek and Trousseau signs. Cardiac symptoms include decreased contractility and heart failure. ECG changes of hypocalcemia include

- QT-interval prolongation

- Terminal T-wave inversion
- Heart blocks
- Ventricular fibrillation

Hypocalcemia can exacerbate digitalis toxicity.

#### Treatment of Hypocalcemia

Treatment of hypocalcemia requires administration of calcium. Treat acute, symptomatic hypocalcemia with 10% calcium gluconate, 90 to 180 mg of elemental calcium IV over 10 minutes. Follow this with an IV drip of 540 to 720 mg of elemental calcium in 500 to 1000 mL D<sub>5</sub>W at 0.5 to 2.0 mg/kg per hour (10 to 15 mg/kg). Measure serum calcium every 4 to 6 hours.

Aim to maintain the total serum calcium concentration between 7 and 9 mg/dL. Abnormalities in magnesium, potassium, and pH must be corrected simultaneously. Note that untreated hypomagnesemia will make hypocalcemia refractory to therapy.

## ▶ Summary

Electrolyte abnormalities are among the most common reasons for patients to develop cardiac arrhythmias. Of all the electrolyte abnormalities, hyperkalemia is most rapidly fatal. A high degree of clinical suspicion and aggressive treatment of underlying electrolyte abnormalities can prevent many patients from progressing to cardiac arrest.

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*Circulation*. 2000;102(suppl I):I-217–I-222.

## ▶ References

1. Jackson MA, Lodwick R, Hutchinson SG. Hyperkalaemic cardiac arrest successfully treated with peritoneal dialysis [see comments]. *BMJ*. 1996;312:1289–1290. [\[Full Text\]](#)
2. Voelckel W, Kroesen G. Unexpected return of cardiac action after termination of cardiopulmonary resuscitation. *Resuscitation*. 1996;32:27–29. [\[Medline\]](#)
3. Niemann JT, Cairns CB. Hyperkalemia and ionized hypocalcemia during cardiac arrest and resuscitation: possible culprits for postcountershock arrhythmias? *Ann Emerg Med*. 1999;34:1–7. [\[Medline\]](#)
4. Lin JL, Lim PS, Leu ML, Huang CC. Outcomes of severe hyperkalemia in cardiopulmonary resuscitation with concomitant hemodialysis. *Intensive Care Med*. 1994;20:287–290. [\[Medline\]](#)

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5. Allon M. Hyperkalemia in end-stage renal disease: mechanisms and management [editorial]. *J Am Soc Nephrol*. 1995;6:1134–1142. [\[Abstract\]](#)
6. Allon M, Shanklin N. Effect of bicarbonate administration on plasma potassium in dialysis patients: interactions with insulin and albuterol. *Am J Kidney Dis*. 1996;28:508–514. [\[Abstract\]](#)
7. Adroque HJ, Madias NE. Aiding fluid prescription for the dysnatremias. *Intensive Care Med*. 1997;23:309–316. [\[Medline\]](#)
8. Fraser CL, Arieff AI. Epidemiology, pathophysiology, and management of hyponatremic encephalopathy. *Am J Med*. 1997;102:67–77. [\[Medline\]](#)
9. Lauren R, Karp BI. Myelinolysis after correction of hyponatremia [see comments]. *Ann Intern Med*. 1997;126:57–62. [\[Medline\]](#)
10. Gross P, Reimann D, Neidel J, Doke C, Prospert F, Decaux G, Verbalis J, Schrier RW. The treatment of severe hyponatremia. *Kidney Int Suppl*. 1998;64:S6–S11. [\[Medline\]](#)
11. Soupart A, Decaux G. Therapeutic recommendations for management of severe hyponatremia: current concepts on pathogenesis and prevention of neurologic complications. *Clin Nephrol*. 1996;46:149–169. [\[Medline\]](#)
12. Brunner JE, Redmond JM, Haggard AM, Kruger DF, Elias SB. Central pontine myelinolysis and pontine lesions after rapid correction of hyponatremia: a prospective magnetic resonance imaging study. *Ann Neurol*. 1990;27:61–66. [\[Medline\]](#)
13. Ayus JC, Krothapalli RK, Arieff AI. Treatment of symptomatic hyponatremia and its relation to brain damage: a prospective study. *N Engl J Med*. 1987;317:1190–1195. [\[Medline\]](#)
14. Higham PD, Adams PC, Murray A, Campbell RW. Plasma potassium, serum magnesium and ventricular fibrillation: a prospective study. *Q J Med*. 1993;86:609–617. [\[Medline\]](#)
15. Navarro-Gonzalez JF. Magnesium in dialysis patients: serum levels and clinical implications. *Clin Nephrol*. 1998;49:373–378. [\[Medline\]](#)
16. Fiset C, Kargacin ME, Kondo CS, Lester WM, Duff HJ. Hypomagnesemia: characterization of a model of sudden cardiac death. *J Am Coll Cardiol*. 1996;27:1771–1776. [\[Medline\]](#)
17. Leier CV, Dei Cas L, Metra M. Clinical relevance and management of the major electrolyte abnormalities in congestive heart failure: hyponatremia, hypokalemia, and hypomagnesemia. *Am Heart J*. 1994;128:564–574. [\[Medline\]](#)
18. al-Ghamdi SM, Cameron EC, Sutton RA. Magnesium deficiency: pathophysiologic and clinical overview [see comments]. *Am J Kidney Dis*. 1994;24:737–752. [\[Abstract\]](#)
19. Barri YM, Knochel JP. Hypercalcemia and electrolyte disturbances in malignancy. *Hematol Oncol Clin North Am*. 1996;10:775–790. [\[Medline\]](#)
20. Aldinger KA, Samaan NA. Hypokalemia with hypercalcemia: prevalence and significance in treatment. *Ann Intern Med*. 1977;87:571–573. [\[Medline\]](#)
21. Edelson GW, Kleerekoper M. Hypercalcemic crisis. *Med Clin North Am*. 1995;79:79–92. [\[Medline\]](#)

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