
Selected Topics: Cardiology Commentary

ELECTROCARDIOGRAPHIC MANIFESTATIONS: ELECTROLYTE ABNORMALITIES

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□ **Abstract**—Because myocyte depolarization and repolarization depend on intra- and extracellular shifts in ion gradients, abnormal serum electrolyte levels can have profound effects on cardiac conduction and the electrocardiogram (EKG). Changes in extracellular potassium, calcium, and magnesium levels can change myocyte membrane potential gradients and alter the cardiac action potential. These changes can result in incidental findings on the 12-lead EKG or precipitate potentially life-threatening dysrhythmias. We will review the major electrocardiographic findings associated with abnormalities of the major cationic contributors to cardiac conduction—potassium, calcium and magnesium. © 2004 Elsevier Inc.

□ **Keywords**—electrolyte abnormality; electrocardiogram; EKG; dysrhythmias; cardiac conduction; cations; potassium; calcium; magnesium

INTRODUCTION

Cardiac electrical activity is mediated by ionic shifts across cellular membranes. Aberrations in normal electrolyte homeostasis are often associated with alterations in cardiac conduction. These may be limited to clinically insignificant changes in the surface electrocardiogram (EKG) or result in life-threatening dysrhythmias. Al-

though the laboratory is indispensable in making definitive diagnoses of fluid, acid-base, and electrolyte disturbances, the bedside EKG can often provide immediate insight and prompt life-saving care.

The clinical syndromes that create hypo- and hyperemic concentrations of potassium, calcium and magnesium are associated with the most common and clinically important disturbances of cardiac rhythm related to electrolyte abnormality. Although discussed individually, it is important to remember that there is a dynamic physiologic interrelationship to electrolyte homeostasis and that aberration in one “compartment” may have impact on another.

CASE REPORTS

Case 1

A 34-year-old man presented to the Emergency Department (ED) complaining of generalized, progressive weakness and shortness of breath over the past 4 days. The patient’s shortness of breath had progressed from dyspnea on exertion to dyspnea at rest over the same time. He also complained of lightheadedness, nausea, and occasional vomiting, but denied chest pain, palpitations, or abdominal pain. The patient admitted to recent

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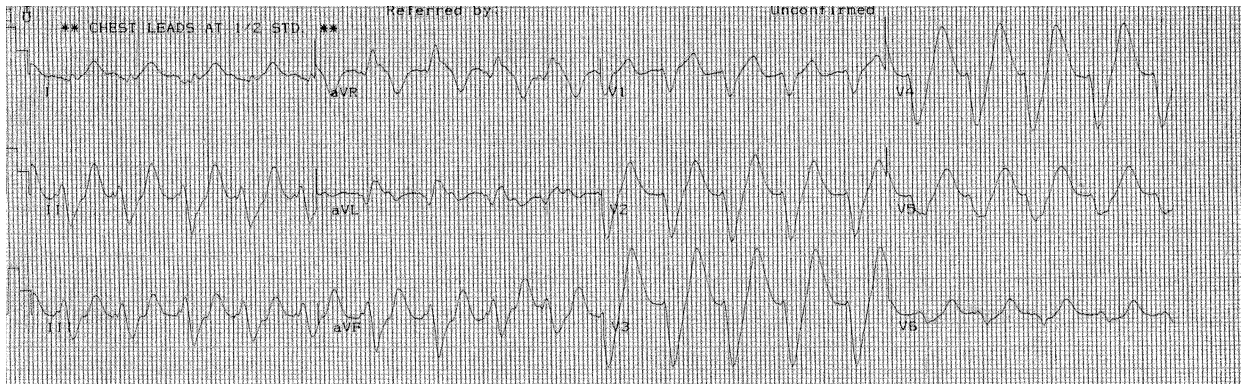


Figure 1. Wide complex tachycardia consistent with a “sine-wave” appearance on 12-lead EKG in patient with serum potassium level of 8.1 mg/dL.

methamphetamine use, but denied prior cardiac history, hospitalizations, or surgery.

On physical examination, vital signs were notable for a pulse of 152 beats/min, blood pressure of 142/68 mm Hg, and respiratory rate of 24 breaths/min. Pulse oximetry was 95% oxygen saturation on room air. The patient was in moderate distress. His chest examination was clear to auscultation and cardiac examination was notable for a regular tachycardia with a grade 2/6 systolic murmur. The abdomen was soft and nontender, and neurologic examination was notable for mild diffuse weakness. An EKG was obtained, revealing a wide complex tachycardia of unclear etiology (Figure 1). Initial treatment included oxygen, intravenous fluids, and bolus intravenous lidocaine without change. After an arterial blood gas revealed a significant metabolic acidosis (pH 7.08, pO_2 217, pCO_2 22), sodium bicarbonate was administered. A repeat EKG 15 min later demonstrated slowing of the heart rate, narrowing of the QRS width, and the presence of “peaked” T waves primarily in the precordial leads consistent with hyperkalemia (Figure 2). The suspicion of hyperkalemia was confirmed by a

serum potassium level of 8.1 mEq/L.

The patient was diagnosed with amphetamine-induced acute renal failure (creatinine 20.1 mEq/L) and was stabilized with calcium chloride, additional bicarbonate, insulin/glucose therapy, followed by hemodialysis. A repeat EKG obtained the day after admission revealed a return to sinus rhythm with normal QRS interval and resolution of the peaked T-waves (Figure 3). The patient was subsequently discharged in good condition after a 5-day hospitalization.

Case 2

A 24-year-old woman with a history of type 1 renal tubular acidosis presented to the ED complaining of 2 days of headache, generalized weakness, and diffuse muscle cramping. The patient also noted the onset of nausea and vomiting over the last day. She denied any chest pain, palpitations, shortness of breath, respiratory difficulty, cough, fevers, chills, diarrhea, or urinary complaints. She had run out of her usual medications, includ-

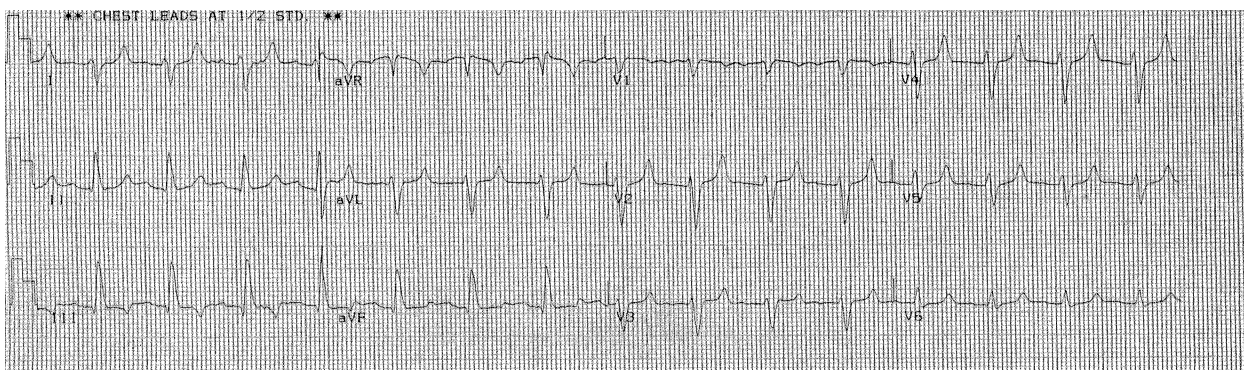


Figure 2. Repeat 12-lead EKG in patient with hyperkalemia early after initiation of treatment. Note the “tent” symmetric T waves most prominent in leads V2–V5.

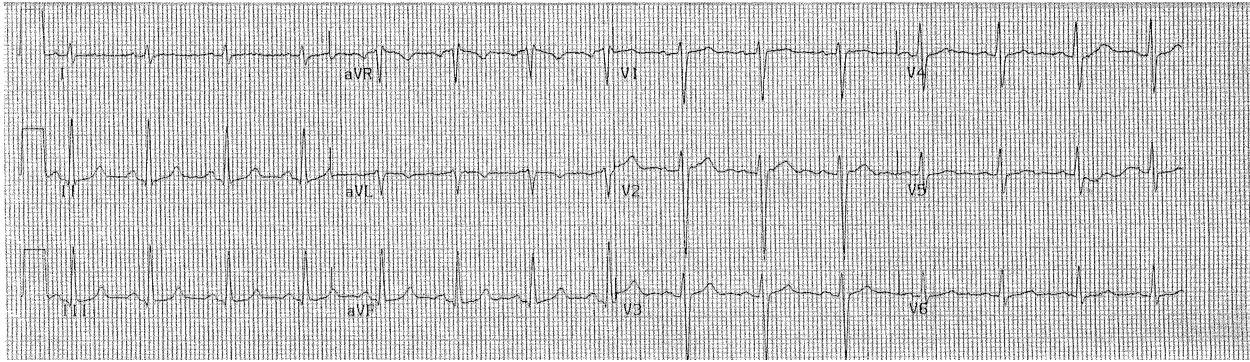


Figure 3. Follow-up 12-lead EKG in patient with hyperkalemia 1 day after treatment. Note the resolution in the T wave abnormalities seen in Figure 2.

ing potassium supplementation, 4 days prior. Her temperature was 36.2°C (97.1°F), pulse 71 beats/min, blood pressure 98/68 mm Hg, and respiratory rate 18 breaths/min. Her examination revealed a well and non-toxic appearing young woman in no acute distress. The pulmonary and cardiac examinations were unremarkable. On neurologic examination, she demonstrated intact sensation and motor strength, but had a slow gait.

An initial 12-lead EKG was obtained (Figure 4) that demonstrated a U wave deflection after the T wave, best seen in the precordial leads V3–V6. Serum potassium was 2.1 mEq/L and the patient was admitted for aggressive intravenous and oral potassium repletion. During the first day of admission, the patient's serum potassium level dropped to as low as 1.9 mEq/L. A repeat EKG demonstrated further flattening of the T wave and increase in U wave size such that it masked the end of the T wave and beginning of the P wave (Figure 5). The patient eventually developed so-called “giant” U waves nearly completely masking the T wave, especially in the precordial leads (Figure 6). With aggressive electrolyte correction, the patient's serum potassium normalized over the next 48 h and the EKG abnormalities resolved.

Case 3

A 55-year-old man with a history of bladder cancer presented to the ED complaining of weakness and constipation over the past week. He had recently completed a course of radiation therapy for his cancer. He denied palpitations, lightheadedness, chest pain, and shortness of breath. He complained of mild intermittent abdominal pain associated with his diffuse weakness. On examination, his pulse was 98 beats/min, blood pressure 138/72 mm Hg, respiratory rate 16 breaths/min, and he was afebrile. The chest and cardiac examinations were unremarkable. The patient had some mild diffuse abdominal tenderness to palpation, but no rebound tenderness or guarding. His neurologic examination revealed mild diffuse weakness, but no other focal findings.

A 12-lead EKG was obtained demonstrating a sinus rhythm with an abnormally short QT interval (Figure 7). When corrected, the QTc interval measured 0.37 s. Serum electrolyte levels confirmed the diagnosis of hypercalcemia with a calcium level of 14.7 mg/dL. The patient's elevated calcium was attributed to his cancer and he was admitted for therapy. He received intravenous

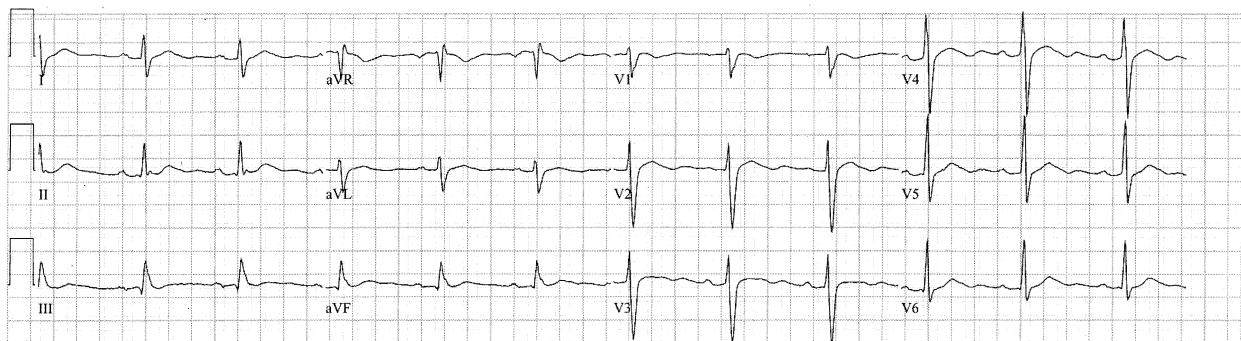


Figure 4. 12-lead EKG in patient with type 1 RTA presenting with weakness. Note the prominent U wave, best seen in the precordial leads (particularly lead V4) consistent with the diagnosis of hypokalemia.

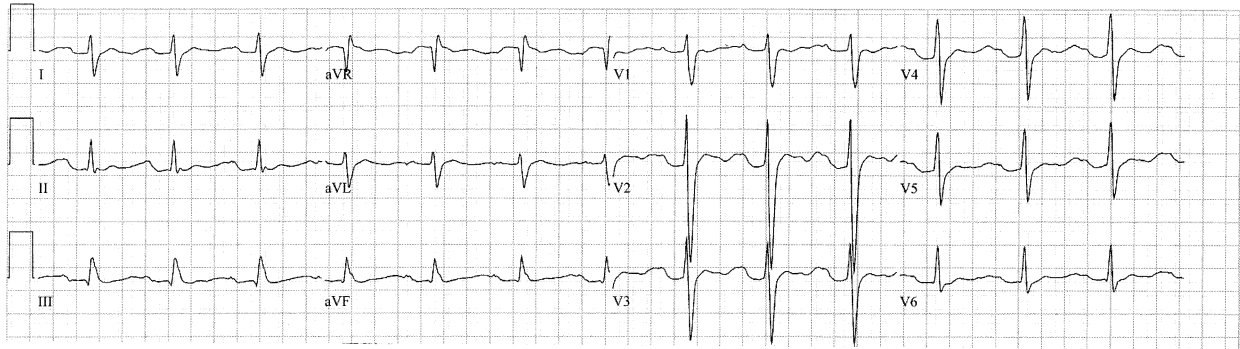


Figure 5. 12-lead EKG in patient with worsening hypokalemia. Note the flattening of the T waves and increasing prominence of the U waves.

fluid hydration, as well as furosemide to increase renal calcium loss and etidronate to reduce bone mineral loss. After 3 days of hospitalization, the patient's serum calcium level was 9.2 mg/dL and his QTc interval normalized on repeat EKG.

DISCUSSION

The most common electrolyte abnormalities affecting the EKG include disorders of potassium, calcium and magnesium (Table 1). Electrolyte imbalances affect the depolarization and repolarization phases of the cardiac cycle action potential by altering potentials across cardiac myocyte cellular membranes. Although we will discuss each electrolyte and its associated abnormalities separately, it is important to remember that combined disorders do occur and there is a dynamic physiologic interaction between these cations.

Potassium

Potassium is predominantly an intracellular cation with only 2% of the total body potassium in the extracellular

space. Although the mechanism of potassium regulation between spaces is complex, there are two main transport processes. An active transport process involves the Na^+/K^+ -ATPase pump, insulin, beta-adrenergic agents, and mineral corticosteroids; and passive transport results from alterations in the pH and extracellular cellular fluid osmolality. Homeostatic serum potassium concentration is maintained by the terminal nephron segments of the kidney. Factors that lead to alterations in serum potassium regulation include renal failure and medications such as non-steroidal anti-inflammatory agents, angiotensin-converting enzyme inhibitors, diuretics, and digoxin (1,2).

Potassium plays an important role in maintaining the electrical potential across the cellular membrane, as well as in depolarization and repolarization of the myocytes. Alterations in serum potassium levels can have dramatic effects on cardiac cell conduction and may lead to electrocardiographic (EKG) changes. Although in some patients EKG changes do not accompany serum potassium abnormalities, the electrocardiogram is a useful screening tool for gauging the severity of the serum potassium

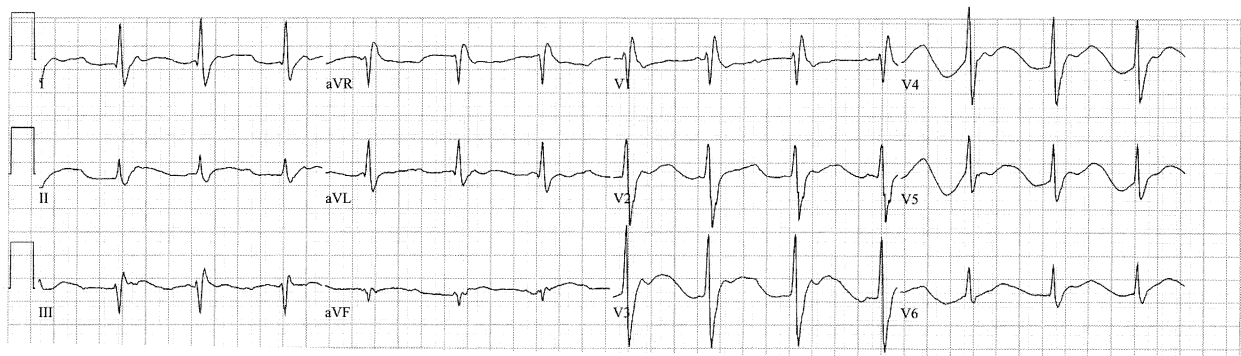


Figure 6. 12-lead EKG in same patient demonstrating so-called "giant" U waves, particularly in V3-V5 where the U wave masks the preceding T wave and after P wave.

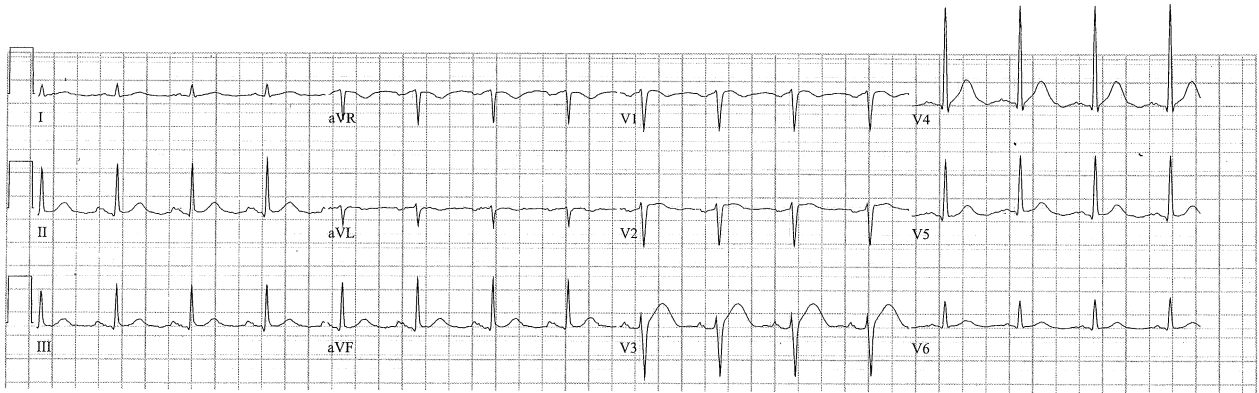


Figure 7. Patient with history of bladder cancer and marked hypercalcemia demonstrating an abnormally shortened QTc interval of 0.37 s in 12-lead EKG.

abnormality and the urgency of therapeutic intervention (3,4).

Hypokalemia

Hypokalemia is the result of renal loss, gastrointestinal losses, extracellular to intracellular shift, or inadequate potassium intake (1). Due to the high prevalence of patients on medications that can result in hypokalemia,

the diagnosis is relatively common. In addition to common medications, conditions such as diarrhea and vomiting may lead to low potassium levels. It can also accompany other metabolic abnormalities such as hypomagnesemia.

As serum potassium levels decline, the transmembrane potassium gradient is decreased. The effect on the cell membrane is an elevation in the resting membrane potential and a prolongation of the action potential, particularly phase 3 repolarization and refractory periods (5).

The earliest EKG change associated with hypokalemia is a decrease in the T wave amplitude. As potassium levels decline further, ST segment depression and actual T wave inversions can be seen. The PR interval can be prolonged and there can be an increase in the amplitude of the P wave. With even lower serum potassium levels, the classic EKG change associated with hypokalemia is the development of U waves. The U wave is described as a positive deflection after the T-wave that is often best seen in the mid-precordial leads, such as V2 and V3 (Figures 4, 5). These changes have been reported in almost 80% of patients with potassium levels < 2.7 mEq/L (6). With extreme hypokalemia, giant U waves may often mask the smaller preceding T waves or after P waves (Figure 6) (7).

The EKG correlates of hypokalemia can be confused with myocardial ischemia. In addition, it can be difficult to differentiate a U wave from a peaked T wave that is present in hyperkalemia. In patients with hyperkalemia, the peaked T wave usually has a narrow base and the QRS may or may not be widened.

As a result of the increased duration of the action potential and refractory period, patients with hypokalemia are at increased risk for certain dysrhythmias (5). The prolongation of the QT, or more correctly QTU interval, can precipitate torsades de pointes or ventricular

Table 1. Electrocardiographic Findings Associated with Disturbances in Serum Electrolyte Levels

Electrolyte Abnormality	Electrocardiographic Findings
Hypokalemia	Decreased T wave amplitude T wave inversion ST segment depression Prominent U Wave Prolongation of QT(U) Interval Ventricular tachycardia Torsades de pointes
Hyperkalemia	
Mild	Large amplitude T waves "Peaked" or "tenting" T waves
Moderate	PR interval prolongation Decreased P wave amplitude, disappearance QRS complex widening Conduction blocks with escape beats
Severe	Sine-wave pattern Ventricular fibrillation Asystole
Hypocalcemia	Prolongation of QTc interval Ventricular dysrhythmias Torsades de pointes
Hypercalcemia	Shortening of QTc interval Bradycardias
Hypo/ Hypermagnesemia	No unique electrocardiographic abnormalities, but often associated with calcium abnormalities

tachycardia. A pseudo-prolonged QT interval may be seen, which is actually the QU interval with an absent T wave. It has been reported that patients with hypokalemia are at increased risk of ventricular fibrillation (8). Treatment of hypokalemia focuses on parenteral and oral potassium supplementation, as well as identification and treatment of the source of the electrolyte abnormality.

Hyperkalemia

Studies validate a good correlation with hyperkalemia and EKG changes. The earliest EKG abnormalities have been reported in patients with serum potassium levels as low as 5.5 mEq/L (3). In addition, there is a predictable EKG progression as the serum potassium becomes more elevated.

With the increased extracellular concentration of potassium, transmembrane permeability is increased, causing an influx of potassium into the cells. There is alteration of the transmembrane potential gradient, a decrease in magnitude of the resting potential, and a decrease in velocity of phase 0 of the action potential. The potassium influx causes a shortening of the action potential and results in delayed conduction between the myocytes (3). Ultimately, these changes produce a slowing of conduction. These conduction abnormalities result in a number of electrocardiographic changes that are present in hyperkalemia. The earliest EKG correlate is T wave tenting, classically described as symmetrically narrow or peaked, though the deflection is often wide and of large amplitude (Figure 2) (9). In addition, the inverted T waves associated with left ventricular hypertrophy can pseudonormalize (i.e., flip upright) with hyperkalemia (10). These T wave changes occur as a result of the acceleration of the terminal phase of repolarization and are most prominently seen in the precordial leads. This is often seen when potassium levels exceed 5.5 mEq/L.

With higher levels of serum potassium, cardiac conduction between myocytes is suppressed. Reduction in atrial and ventricular transmembrane potential causes an inactivation of the sodium channel, decreasing the cellular action potential. Atrial tissue is more sensitive to these changes earlier and, as a result, P wave flattening and PR interval prolongation may be seen before QRS interval prolongation. These changes generally occur when potassium levels exceed 6.5 mEq/L (5). As the serum potassium level increases further, there is eventual loss of the P wave, attributable to the increased sensitivity of the atrial myocytes to elevated potassium levels.

As the serum level continues to rise to levels above two times normal, there is suppression of sinoatrial and atrioventricular conduction, resulting in sinoatrial and atrioventricular blocks, often with escape beats. Other

blocks including intraventricular conduction delay, bundle branch block, and fascicular blocks have been reported. Interestingly, bypass tracts are more sensitive to delayed conduction from potassium elevation, which can result in the normalization of the EKG and loss of the delta wave in patients with Wolff-Parkinson-White syndrome

With extremely high serum potassium levels, a markedly prolonged and wide QRS complex can fuse with the T wave, producing a slurred, "sine-wave" appearance on the EKG (Figure 1). This finding is a pre-terminal event unless treatment is initiated immediately. The fatal event is either asystole, as there is complete block in ventricular conduction, or ventricular fibrillation (5).

Although the above EKG progression is descriptive of the classic presentation of hyperkalemia, these changes do not always accompany elevated serum potassium levels. Metabolic alterations such as alkalosis, hypernatremia, or hypercalcemia can antagonize the transmembrane effects of hyperkalemia and result in the blunting of the EKG changes associated with elevated potassium levels (11).

Treatment of life-threatening hyperkalemia focuses on blocking the effects on myocyte transmembrane potential and cardiac conduction, as well as decreasing extracellular potassium levels. Response to therapy is often prompt with visualization noted on the EKG or electrocardiographic monitor. Calcium can effectively block the effect of extracellular potassium elevation on cardiac myocytes within minutes of administration by restoring a more appropriate electrical gradient across the cellular membrane. Calcium is usually administered in the form of calcium chloride or gluconate, though calcium chloride delivers a much larger amount of calcium per unit volume. However, calcium may induce toxic dysrhythmias such as asystole in digitalis toxic patients. Sodium bicarbonate, magnesium, beta-2 adrenergic agonists, and the combination of glucose and insulin all drive potassium intracellularly and lower the extracellular serum potassium level. Finally, excess body potassium can be removed with the use of sodium polystyrene sulfonate or dialysis.

Calcium

The human body has a nearly inexhaustible reservoir of calcium (Ca^{++}) stored as hydroxyapatite in skeletal bone. Of a total of 1 to 2 kg of adult total body Ca^{++} , only 1 gram of calcium is present in plasma. Approximately half is protein bound primarily to albumin. A small fraction is complexed with phosphate, bicarbonate, citrate and lactate, with the remainder being ionized free calcium. Extracellular Ca^{++} concentrations are tightly

controlled through a complex homeostatic mechanism mediated primarily by parathyroid hormone and modulated through effector cells in kidney, bone and intestine. The gradient between intracellular and extracellular Ca^{++} is 1000- to 10,000-fold, making rapid transmembrane shifts through "gated" channels possible. Ca^{++} is important in a myriad of regulatory mechanisms, skeletal muscle contraction, control of enzymatic reactions, and is a key ion in myocyte electrical activity and myocardial contraction (12).

Hypocalcemia

Hypocalcemia is classically seen with functional parathyroid hormone deficiency, either as absolute hormone deficiency (primary hypoparathyroidism), post-parathyroidectomy, or related to a pseudo-hypoparathyroid syndrome. Other causes of hypocalcemia include vitamin D deficiency, congenital disorders of calcium metabolism, chronic renal failure, acute pancreatitis, rhabdomyolysis, and sepsis. Hypocalcemia is commonly seen in critically ill patients, with a reported incidence of as high as 50% (13). Furthermore, hypocalcemia is often associated with hypomagnesemia. Neuromuscular irritability is the cardinal feature of hypocalcemia, with carpal-pedal spasm being the classical physical sign that may progress to frank tetany, laryngospasm or tonic-clonic seizure activity.

The primary electrocardiographic manifestation of hypocalcemia is lengthening of the QTc interval. Hypocalcemia prolongs phase 2 of the action potential with the impact modulated by the rate of change of serum calcium concentration and function of the myocyte calcium channels. Prolongation of the QTc interval is associated with early after-repolarizations and triggered dysrhythmias. Torsades de pointes potentially can be triggered by hypocalcemia but is much less common than with hypokalemia or hypomagnesemia.

Whereas electrocardiographic conduction abnormalities are common, serious hypocalcemia-induced dysrhythmias such as heart block and ventricular dysrhythmias are infrequent (13). The development of dysrhythmias is often associated with other co-morbidities such as structural heart disease, ischemia, or in association with drug therapy (e.g., digitalis, catecholamines). Severe symptoms and life-threatening dysrhythmias mandate immediate treatment with parenteral calcium salts. In addition, associated electrolyte abnormalities, including hypomagnesemia, phosphate abnormalities and acidemia may need to be corrected. Vitamin D and oral calcium supplementation can be administered for chronic hypocalcemia.

Hypercalcemia

Hypercalcemia is the cardinal feature of hyperparathyroidism. It is typically chronic, mild and well tolerated. Severe hypercalcemia with serum levels above 14mg/dL can be precipitated in these patients by dehydration from gastrointestinal losses, diuretic therapy, or ingestion of large amounts of calcium salts. The most common clinical presentation of hypercalcemia is in patients with metastatic non-parathyroid cancers. Accelerated bone resorption dramatically increases the load of filtered calcium and renal sodium reabsorption becomes impaired, creating a cascade of volume depletion and worsening hypercalcemia. Symptoms of hypercalcemia can be relatively vague, including fatigue, lethargy, motor weakness, anorexia, nausea, constipation and abdominal pain.

The effect of hypercalcemia on the electrocardiogram is the opposite of hypocalcemia with the hallmark of abnormal shortening of the QTc interval (Figure 7). Clinically significant rhythm disturbances associated with hypercalcemia are rare because elevation of extracellular calcium is generally not associated with triggered dysrhythmias. Cardiac conduction abnormalities may occur, with bradydysrhythmias being the most common (14).

Therapy for hypercalcemia is generally instituted based on clinical signs more than absolute serum levels, although empiric therapy is often started at levels of 14mg/dL even in the asymptomatic patient. In patients with hypoalbuminemia, measured serum calcium levels may mask significant elevations in free ionized extracellular calcium. The mainstays of treatment are intravenous volume repletion and bisphosphonate agents that inhibit osteoclastic bone resorption. The use of loop diuretics to promote calciuresis is often recommended but is problematic in the hypovolemic patient. Dialysis may be required in severe cases.

Magnesium

Magnesium, a primarily intracellular cation, participates in hundreds of enzymatic reactions and in essential hormonal regulation. It is important in the maintenance of cellular ionic balance with the modulation of sodium, potassium and calcium. The average adult contains about 24 g of magnesium, with only 1% found in the extracellular space. Ongoing dietary intake is required to maintain normal levels (15).

The historical medical indication for magnesium therapy has been in the management of the eclamptic syndromes of pregnancy. Magnesium also has a role in the acute management of wide-complex tachydysrhythmias where therapy may be lifesaving. There is a burgeoning,

though inconclusive literature related to other potential therapeutic uses in asthma, myocardial infarction, and acute cerebral ischemia (16).

Hypomagnesemia is the most common clinically encountered aberration of magnesium homeostasis and is caused by decreased intake, increased losses, or altered intracellular-extracellular distribution. Unlike other electrolyte disturbances, there is no classical syndrome and measurement of serum levels do not correlate well with clinical manifestations. Hypomagnesemia is associated with far-reaching adversity across the physiologic spectrum, including central nervous system (CNS) effects (seizures, mental status changes), cardiovascular effects (dysrhythmias, vasospasm), endocrine effects (hypokalemia, hypocalcemia), and muscle effect (bronchospasm, muscle weakness). Hypermagnesemia is primarily seen in patients with renal failure who ingest large amounts of magnesium salts and is usually well tolerated. Symptomatic hypermagnesemia is most often iatrogenic in origin, and is associated with errors in intravenous dosing. Severe symptoms include CNS depression, areflexia, respiratory failure, and rarely, cardiac arrest.

There are no unique electrocardiographic effects that can be attributed specifically to either hypomagnesemia or hypermagnesemia. It is well known that both supraventricular and ventricular dysrhythmias can be potentiated if not directly caused by hypomagnesemia. Magnesium has been shown to be efficacious in the management of atrial tachydysrhythmias although there is no correlation between rhythm conversion and plasma concentration. Magnesium sulfate is considered first-line therapy by the American Heart Association ACLS guidelines for torsades de pointe (17).

CONCLUSIONS

Intracellular and extracellular cations such as potassium, calcium and magnesium critically affect the cardiac ac-

tion potential and myocyte depolarization and repolarization. Changes in extracellular levels of these electrolytes can have profound effects on cardiac conduction. Electrocardiographic findings associated with these disturbances can provide clues to the diagnosis, as well as guide therapeutic interventions.

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