ELECTROCARDIOGRAPHIC CHALLENGES IN PATIENTS WITH POTASSIUM ABNORMALITIES

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Abstract. *Potassium, the most abundant intracellular cation, is critically important for many physiologic processes, including maintenance of cellular membrane potential, homeostasis of cell volume, and transmission of action potentials in nerve cells. Therefore, potassium is necessary for the normal functioning of the muscles, heart, and nerves. A great number of metabolic disorders could be associated with potassium abnormalities.*

Experimental and human studies have demonstrated that serum potassium imbalance is followed by progressively severe electrophysiological derangements in cardiac impulse generation and conduction. These electrophysiological changes are reflected in the electrocardiogram (ECG) manifestations, with specification that the ECG tracing reflects more faithfully the potassium extracellular concentration.

Though laboratory tests are "the gold standard" for the diagnosis of potassium abnormalities, they have the disadvantage of delivering the information with delay. Thus, the electrocardiogram (ECG) becomes a very useful instrument for diagnosis.

Keywords: cation, hyperkalemia, hypokalemia, electrocardiogram (ECG), repolarization.

1. Introduction

Potassium (K) is the most abundant intracellular cation. Almost all (98%) potassium in the body is found inside the cells (intracellular) and only about 2% occurs in the fluids outside of the cells (extracellular). The concentration of potassium is often expressed in units of milliequivalents per liter (mEq/l), rather than in units of millimolarity (mM). Both units mean the same thing when applied to concentrations of potassium ions. Normally plasma potassium concentration ranges from: 3.5 – 5.0 mEq/l.

The contribution of potassium to resting membrane potential is related to this ratio of intracellular to extracellular potassium.

The mild to moderate changes in plasma potassium value are relatively poor associated with ECG tracing (10 - 30%), while the severe abnormalities have more specific ECG expression (90%).

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The specific ECG changes caused by electrolyte imbalance are usually reversible and their development and regression follow a predictable course.

Hyperkalemia

Hyperkalemia is a common electrolyte disorder with potentially life-threatening consequences. Hyperkalemia is often silent, could occur suddenly, and leads to cardiac arrhythmias and potentially to death [1].

In the general outpatient population, the incidence is relatively low and not well reported. In hospitalized patients, incidence ranges from 1% to 10%, with a mortality rate of 1 per 1000 patients [2, 3].

A value of plasma potassium between: 5.5 - 6.5 mEq/l reflects mild hyperkalemia, moderate hyperkalemia for a range of 6.5 - 8 mEq/l and severe hyperkalemia over 8 mEq/l.

The incidence of hyperkalemia in the general population has been reported in less than 5% of people. The most common causes are potassium shift from the intracellular to the extracellular space, impaired excretion due to renal failure, or medications, with most patients having multiple etiologies. Risk factors include advanced age, significant prematurity, and the presence of renal failure, diabetes mellitus, and heart failure. Additionally, one series documented an increased incidence of hyperkalemia with cancer and gastrointestinal disease [4]. Polypharmacy, particularly the use of potassium supplements and potassium-sparing diuretics, in patients with underlying renal insufficiency contributed to hyperkalemia in almost one half of the cases.

Because hyperkalemia can lead to life-threatening cardiac arrhythmias, prompt recognition and diagnosis are crucial. Patients with severe hyperkalemia could present with generalized weakness, paralysis, arrhythmias, or sudden cardiac arrest.

The most prominent effect of hyperkalemia is on the myocardium. The generation of a resting membrane potential is crucial for cardiac myocyte contraction. Movement of potassium into the intracellular space via the sodium-potassium adenosine triphosphatase pump is responsible for the – 90 mV resting membrane potential. As the extracellular potassium concentration increases, the concentration gradient across the myocyte cell membrane decreases, eventually leading to a slowing of myocardial functioning [5].

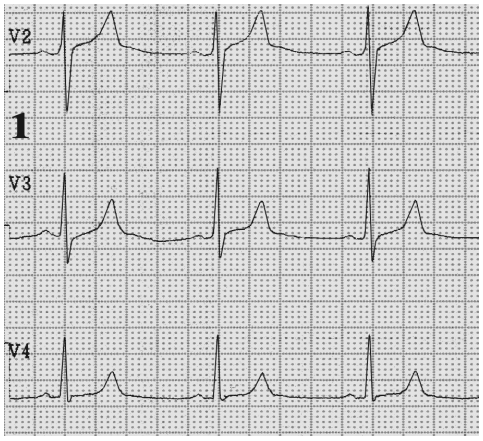
Electrocardiographic (ECG) findings could provide the first evidence of hyperkalemia, but there are studies which show that a correct ECG diagnosis can usually be made when plasma potassium concentrations exceed 6.7 mEq/l [6]. In contrast, Tarail [7] found that patients with renal insufficiency did not consistently have ECG changes typical of hyperkalemia until the serum potassium concentration exceeded 7.6 mEq/l. It has been postulated that levels of potassium

greater than 8 mEq/L are almost always associated with the classic ECG manifestations [8].

The relationship between the degree of hyperkalemia and the ECG changes, however, is variable, and in rare cases of severe hyperkalemia the ECG may even be normal or near normal. Briefly, in mild hyperkalemia T waves changes occur (because of the acceleration of terminal repolarization), than in mild-to-moderate where PR interval is frequently prolonged and QRS complex is wide. In clinical practice it was noticed that ECG changes for mild and moderate hyperkalemia are known, while severe hyperkalemia is often source of error.

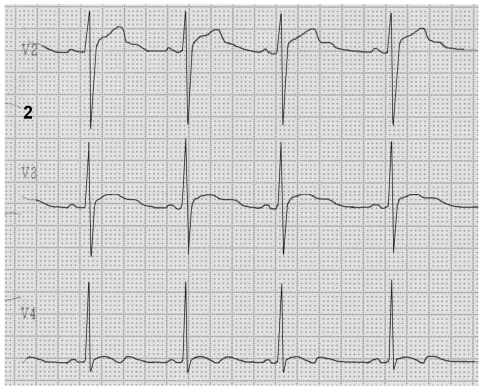
Further, we present some ECG aspects related to plasmatic potassium (Kp) concentration.

When the plasma K concentration exceeds approximately 5,5 mEq/l, the T waves become tall, symmetric, narrow and peaked, tented as if pinched from above because of the faster repolarization of the cardiac action potential; usually they are best seen in leads II, III, V2 and V4 **(Case I)**. The P waves and QRS complexes are normal. The QT interval is shortened at this stage, associated with decreased action potential duration.



Case I.
Electrocardiograms of a 57-year-old male with dilated cardiomyopathy treated with spironolactone.

(1) Plasma K concentration (Kp) is 6.1 mEq/l. There is a regular rhythm at a rate of 60 beats/min, T waves are tall, narrow and pointed in leads V2 - V4.

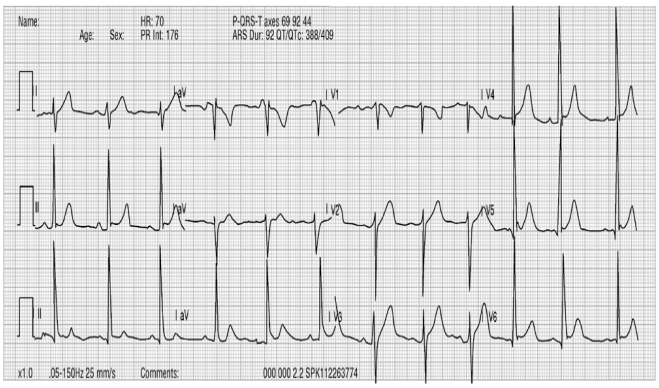


(2) After treatment, Kp is 4.2 mEq/l. Note that T waves are lower in leads V2 - V3, and biphasic in lead V4;
U wave is seen too.

Hyperkalemia cannot be diagnosed with certainty on the basis of T wave changes alone. Braun et al. found that the characteristic tall, steep, narrow and pointed T waves were present in only 22 percent of patients with hyperkalemia [9] and so, it is necessary to make the difference with hyperacute ischemic changes (where T waves are symmetric, broad-based, not tented, not pointed; the QT interval tends to be long) or a normal variant (where T waves are asymmetric and not narrow, frequently associated with sinus bradycardia) - **Case II**.

Progressive extracellular hyperkalemia reduces atrial and ventricular resting membrane potentials, thereby inactivating sodium channels, which decreases *V*max and conduction velocity.

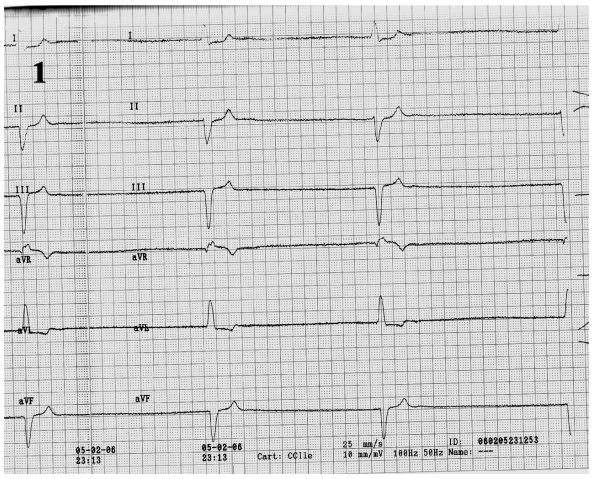
When the plasma K concentration exceeds 7 mEq/l the QRS complex begins to widen, P wave amplitude decreases, and the duration of the P wave increases because of the slower conduction in the atria. The PR interval prolongation can occur, followed sometimes by second or third – degree AV block.



Case II.
Electrocardiogram of a 30-year-old male, sportsman, complai-ning of pricking chest pain. Plasma K concentration is normal. There is a regular sinus rhythm at a rate of 70 beats/min and signs of early repolarization.

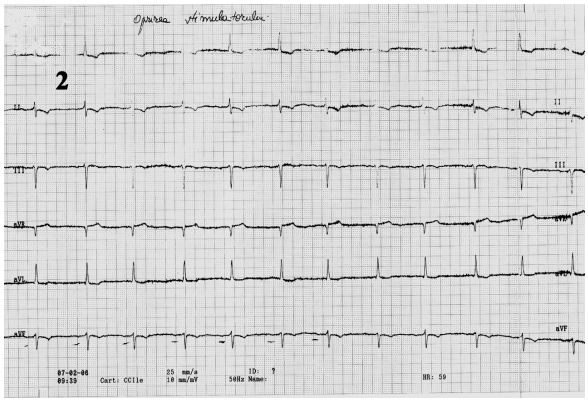
Characteristically, the uniformly wide QRS complex due to hyperkalemia differs from the ECG pattern of bundle branch block or preexcitation because widening affects both the initial and terminal portions of the QRS complex. The wide S wave in the left precordial leads sometimes helps differentiate the pattern of hyperkalemia from that of typical left bundle branch block (LBBB), whereas the wide initial portion of the QRS complex may help differentiate the pattern of hyperkalemia from that of typical right bundle branch block (RBBB) .

When the plasma K concentration exceeds 8 mEq/l, the P wave frequently becomes invisible (**Case III**).



Case III.
Electrocardiograms of a 65-year-old woman with hypertension, diabetes mellitus and heart failure:

(1) Kp is 8.1 mEq/l; the rhythm is regular, at a rate of 21 beats/min, P waves are absent; the QRS duration is 160 ms.

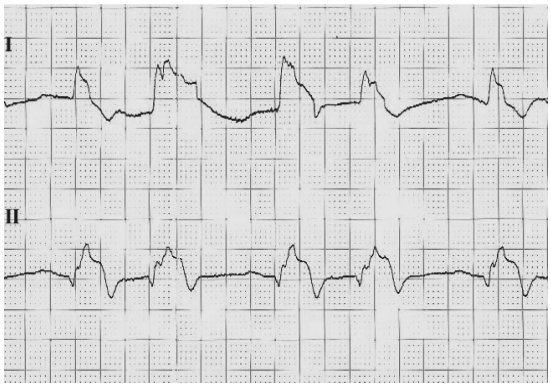


(2) After treatment of hyperkalemia (Kp is 4.83 mEq/l) there is a sinus rhythm at a rate of 64 beats/min,
T wave is inverted in leads I, II, III, aVF – maybe "memory T waves".

A regular rhythm in the absence of P waves has been attributed to sinoventricular conduction via atrionodal tracts in the presence of atrial (SA) block [10, 11]. Also, a regular rhythm in the absence of P waves can be caused by displacement of the pacemaker into the atrioventricular (AV) junction or the Purkinje fibers, but precise localization of the pacemaker in patients with absent P waves is usually not possible.

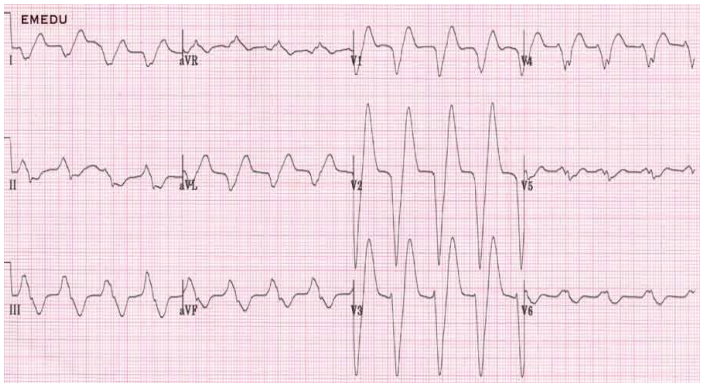
Sometimes in patients with advanced hyperkalemia the ST segment deviates appreciably from baseline and simulates the "acute injury" pattern, which resembles the pattern of acute myocardial ischemia (**Case IV**). Deviation of the ST segment or a monophasic pattern can be readily produced by topical application of K on the ventricular surface or an intracoronary KCl injection [12].

Case IV.



Electrocardiogram of a 68-year-old man with hypertension, diabetes mellitus and chronic renal failure, hospitalized with acute pulmonary edema. The plasma K concentration is 10.7 mEq/l. The rhythm is irregular at a rate of 52 beats/min; the QRS complex is wide; there is marked diffuse ST segment elevation in leads I, II

This type of ST abnormality in patients with hyperkalemia rapidly disappears accompanying the regression of potassium concentration with hemodialysis. The ST-segment deviation probably is caused by nonhomogenous depolarization in different portions of the myocardium. According to this hypothesis, a voltage gradient is created between normal myocardial cells and those depolarized by potassium, resulting in current flow between these regions. Since dialysis rapidly normalizes the ST-segment elevation, it is also known as the *dialyzable current of injury* [13] (Case V).



Case V.

Electrocardiogram of 83-year-old male with hypertension, hospitalized for increased fatigue. The serum potassium level is 9.3 mEq/l. The tracing shows the absence of P waves and wide QRS complexes, with bizarre aspect. After hemodialysis the ECG trace chanced to normal

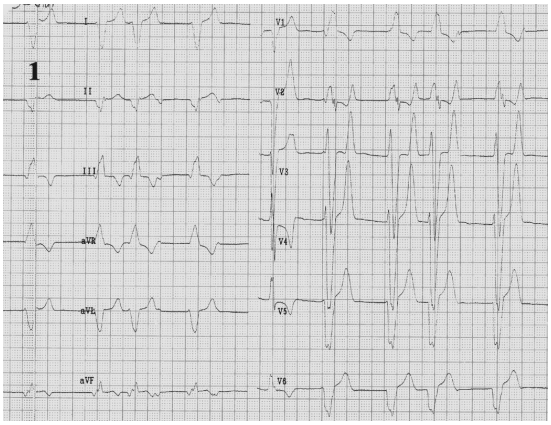
When the plasma K concentration exceeds about 10 mEq/l, the ventricular rhythm may become irregular owing to the simultaneous activity of several escape pacemakers in the depressed myocardium. The combination of an irregular rhythm and an absent P wave may simulate atrial fibrillation. In patients with preexisting atrial fibrillation and hyperkalemia, the ventricular rate is usually slow.

An increase in the plasma K concentration to above 12 - 14 mEq/l causes the final changes: a sine-wave pattern, in which the widened QRS complex merges with the T wave. This is followed by ventricular asystole or ventricular fibrillation. The latter may or may not be preceded by acceleration of the ventricular rate [14]. The danger in the majority of hyperkalemia cases is cardiac dysrhythmia. Although

there are many previous reports addressing this threatening problem and associated therapeutic maneuvers, there have not been many previous reports citing the fatal concentration of hyperkalemia irrespective of the causes. However, it is uniformly accepted that a K^+ concentration greater than 10 mEq/l is fatal unless urgent treatment is instituted.

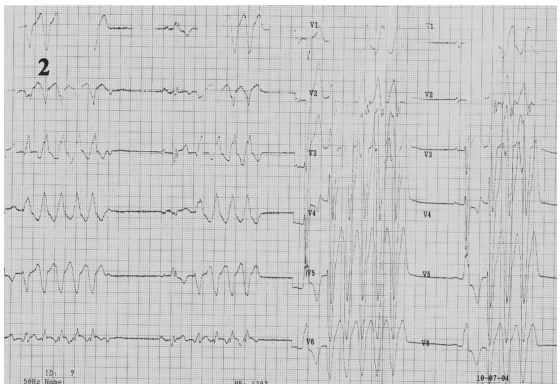
However, even severe hyperkalemia can be associated with atypical or non-diagnostic ECG findings [15]. These include arrhythmias, depression or elevation of ST segment, decrease in the height of the R wave with the development of deep S waves, and QRS axis shift to the left or right, bundle branch blocks, and sino-atrial exit blocks. Some case reports in the literature describe patients with severe hyperkalemia who present with a normal ECG result. Left ventricular hypertrophy, intraventricular conduction defects, and myocardial ischemia all mask ECG manifestations of hyperkalemia. Acidosis, hypoxia, hyponatremia, and hypocalcemia may increase myocardial sensitivity to hyperkalemia, whereas hypernatremia and hypercalcemia may minimize or eliminate the effect of hyperkalemia on the heart. Aslam et al. showed an inverse correlation between serum calcium and the height of the T wave and postulated that the lack of ECG changes of hyperkalemia could partly be due to fluctuations in serum calcium concentration [16].

Sometimes, in the same patient it can be noted a pleiade of ECG changes (**Case VI**).

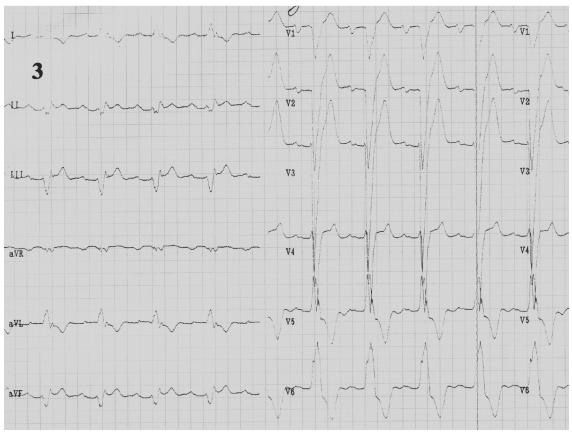


Case VI .
Electrocardiograms of a 63-year-old, with dilated cardiomyopathy, LBBB, chronic renal insufficiency, treated with sotalol, is admitted with palpitations.

(1) The plasma K concentration is 6.5 mEq/l. The rhythm is irregular at a rate of 64 beats/min; P waves are absent; QRS complex is wide and its duration is 180 ms; T waves are tall, symmetric and narrow.



(2) Two hours later the ECG monitoring records non - sustained ventricular tachycardia.



(3) The treatment with sotalol is stopped and after treatment of hyperkalemia the ECG tracing is: sinus rhythm, at a rate of 72 beats/min, the PR interval is 140 ms and LBBB.

No evidence exists indicating at which serum potassium value life-saving therapies should be administered. Calcium infusion, the first step in emergency management, stabilizes cardiac myocyte membranes. Because of the unpredictable nature of cardiac arrhythmias, calcium infusion should be administered if any ECG change suggests hyperkalemia. Patients without ECG changes but who are at high risk for developing arrhythmias (eg, those with rapidly increasing potassium levels or coexisting electrolyte disorders) might benefit from prophylactic administration of calcium. If ECG changes are present, administration of *intravenous calcium* should normalize the ECG patterns. *Insulin* is a well-established therapy that rapidly decreases serum potassium concentrations by inducing intracellular shift.

When administered intravenously, or by a nebulizer or metered-dose inhaler, *beta-agonists* decrease plasma potassium levels. *Cation exchange resins* bind potassium in the gastrointestinal tract and enhance fecal elimination. It is important to correct metabolic acidosis with *sodium bicarbonate*. *Hemodialysis* can rapidly remove large amounts of potassium and is the treatment of choice for

patients with life-threatening hyperkalemia that is refractory to medical management.

Lethal hyperkalemia results predominantly from renal failure and occasionally from an error in the amount of K administrated intravenously. The effect of intravenously administrated K depends on the rate of administration rather than the absolute amount of K given [4, 17].

A particular aspect is about giving much attention to factitious hyperkalemia (part of the differential diagnosis) which occurs when the laboratory potassium value is higher than the actual plasma potassium value. The most common cause is lysis of red blood cells due to specimen handling or collection errors. Hematological abnormalities, such as leukocytosis, thrombocytosis, and polycythemia, can also cause factitious hyperkalemia by increasing cell fragility [18]. When faced with an elevated potassium value of uncertain significance, the physician should consider the patient's risk factors for hyperkalemia. A history of renal disease, obstructive uropathy, clinical features of weakness or myopathy, and use of medications that increase potassium (eg, angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs], aldosterone antagonists, nonsteroidal anti-inflammatory drugs [NSAIDs], potassium supplements, trimethoprim) should prompt concern. To help differentiate a factitious from a true value, the potassium level should be retested, with care taken to ensure minimal trauma, optimal storage conditions, and rapid analysis. If the patient is at considerable risk for hyperkalemia, an ECG test is warranted. To assess factitious hyperkalemia due to increased cell fragility, additional samples of serum and plasma potassium should be taken using a heparinized tube. A discrepancy of more than 0.3mEq/l will secure the diagnosis [19].

Hypokalemia

Hypokalemia, defined as a serum potassium concentration less than 3.5 mEq/l, is a common and potentially serious electrolyte disorder.

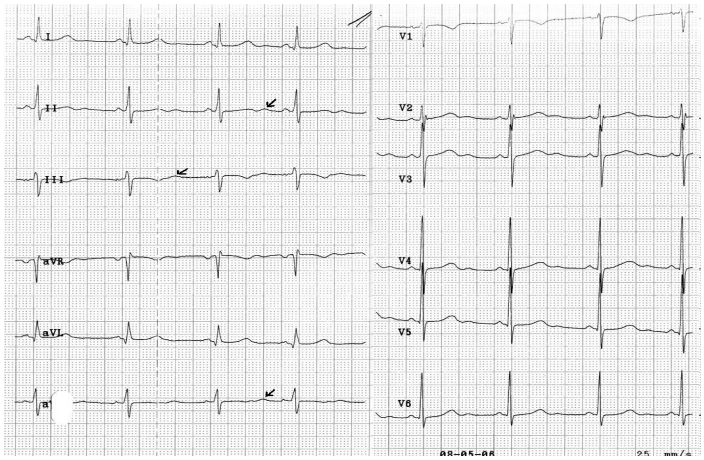
The estimated incidence of hypokalemia in hospitalized patients is 20%, while the incidence of severe hypokalemia, defined as < 3.0 mEq/l, is approximately 5% [20]. Low serum (or plasma) concentrations of potassium may occur in up to 40% of outpatients treated with thiazide diuretics. Of elderly patients, 5% demonstrate potassium levels lower than 3 mEq/l.

Causes of hypokalaemia can be divided in either true potassium depletion, mostly caused by renal or gastrointestinal losses, and a shift of potassium from the extracellular into the intracellular compartment. The electrophysiological changes associated with hypokalemia, in contrast, include hyperpolarization of myocardial cell membranes and increased action potential duration.

Because the duration of mechanical systole does not change during hypokalemia, one can best describe the pattern of hypokalemia as a gradual shift of the major repolarization wave from systole into diastole.

In most circumstances, mild hypokalemia (plasma [K+] 3.0 to 3.5 mEq/l) causes no symptoms. The major disturbances seen with more severe potassium deficiency result from changes in cardiovascular, neuromuscular, and renal function. Cardiac toxicity may be manifested by serious arrhythmias, which occur, as we said, because hyperpolarization of the myocardial cell membrane leads to a prolonged refractory period and increased susceptibility to reentrant arrhythmias.

The ECG diagnosis of hypokalemia is usually based on abnormalities of the ST segment, T wave, and U wave. The major ECG manifestations are ST depression with flattened T waves and increased U wave prominence (**Case VII**). The U waves can exceed the amplitude of T waves.



Case VII.
Electrocardiogram of a 19-year - old woman with, treated with indapamide.
The plasma K concentration is 2.4 mEq/l. Sinus rhythms is present at a rate 62 beats/min, the amplitude of T wave is decreased in all precordial leads and U wave is present. Note: T wave inversion in III with U wave upright (arrows).

In an attempt to evaluate the pattern of hypokalemia quantitatively, Surawicz et al. considered the following three ECG features:

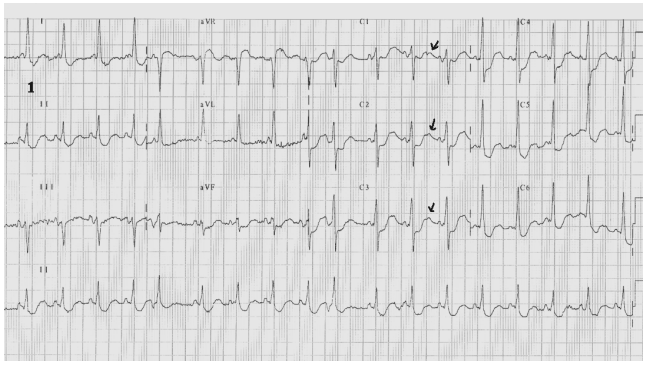
(1) depression of the ST segment of ≥ 0.5 mm;

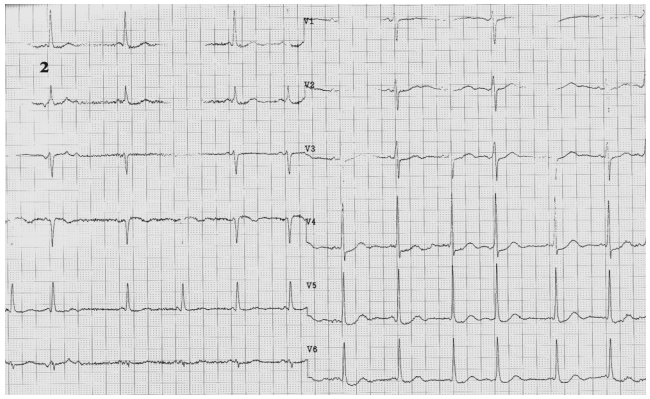
(2) U wave amplitude > 1 mm;

(3) U wave amplitude greater than the T wave amplitude in the same lead [2].

The ECG was considered to be ***"typical of hypokalemia"*** if three or more of above feature were present in two leads; it was considered ***"compatible with hypokalemia***" if two of the above features or one related to the U wave were present. In clinical practice, when the plasma K concentration was < 2.7 mEq/l the ECG was "typical" in the 78 percent and "compatible" in 11 percent of all patients. When the plasma K concentration was 2.7 - 3.0 mEq/l the ECG was "typical" in 35 percent and "compatible with hypokalemia" in 35 percent of patients.

β2 - agonist therapy in patients with chronic obstructive pulmonary diseases (COPD) was associated with significant increases in heart rate and reductions in potassium concentrations, which are known to be common systemic effects of β-adrenergic stimulation. In patients with obstructive airway disease, serum potassium levels could be decreased further with the use of corticosteroids and diuretics, and the cardiac effects of hypokalemia could be aggravated by underlying hypoxemia. (**Case VIII**).





Case VIII

Electrocardiograms of a 55-year-old male with hypertension and chronic obstructive pulmonary disease (COPD), hospitalized for one episode of lipothymia.

(1) The plasma K concentration is 1.85 mEq/l. The sinus rhythm is present at a rate of 120 beats/min, diffuse ST segment depression and T and U waves (arrow) are fused, the U wave amplitude exceeding the T wave amplitude.

(2) After treatment of hypokalemia: the reduction of ST segment depression and low amplitude of the U wave.

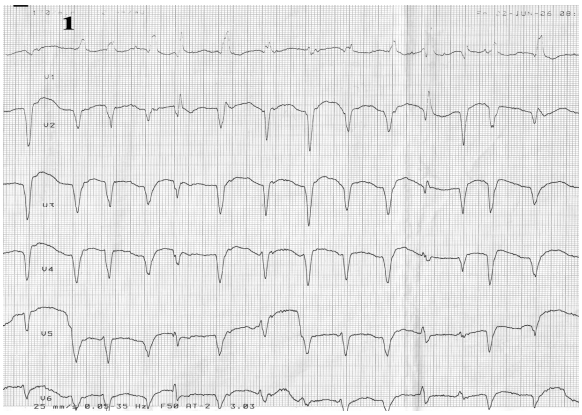
In the presence of a left ventricular hypertrophy pattern, the U wave amplitude is frequently increased (as part of the overall amplitude increases). Digitalis usually causes a more distinct separation of the T wave from the U wave than hypokalemia, because digitalis shortens the QT interval. The U wave amplitude is also increased during bradycardia.

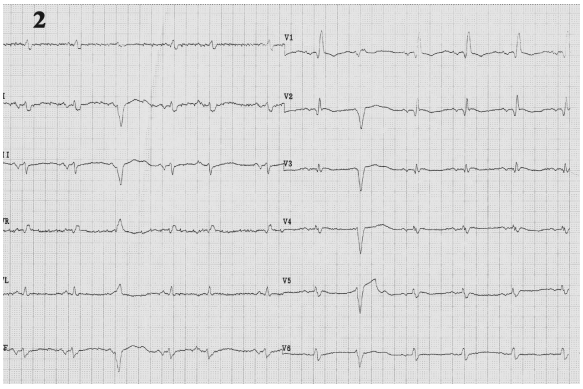
When hypokalemia is advanced, both the amplitude and duration of the QRS interval are increased. The QRS complex is widened diffusely. The increased duration of the QRS is the result of widening without a change in shape, which suggests that it is caused by slower intraventricular conduction without changes in

the depolarization sequence. The amplitude and duration of the P wave in hypokalemia is usually increased, and the PR interval is often slightly or moderately prolonged.

The hypokalemia promotes the appearance of supraventricular and ventricular ectopic complexes. Similar to digitalis, hypokalemia increases sensitivity to vagal stimulation.

In patients with severe hypokalemia, serious ventricular tachyarrhytmias including ventricular tachycardia, torsade de pointes, and ventricular fibrillation have been reported in the absence of heart disease or digital therapy (**Case IX**).





Case IX.
Electrocardiograms of a 68-year-old male with hypertension, diabetes mellitus, and prior inferior myocardial infarction hospitalized after two weeks from an acute anterior myocardial infarction with episodes of non-sustained ventricular tachycardia.

(1) The plasma K concentration is 1,3 mEq/l. The ECG tracing shows polymorphic non-sustained ventricular tachycardia.

(2) After treatment of hypokalemia ventricular tachycardia is no longer present.

Hypokalemia is frequently present in patients with acute myocardial infarction [21, 22] or after resuscitation from out-of-hospital ventricular fibrillation possibly due to treatment with thiazide diuretics or administration of sodium bicarbonate during resuscitation.

In treating hypokalemia, the first step is to identify and stop ongoing losses of potassium; repletion of potassium losses is the second step. The next steps are: monitor for toxicity of hypokalemia and determine the underlying cause to treat and prevent further episodes

The potassium deficit can be treated with oral or intravenous potassium suppletion and/or a potassium-sparing diuretic. Unfortunately, supplemental potassium administration is also the most common cause of severe hyperkalemia in patients who are hospitalized, and this risk must be kept in mind when one is initiating treatment. The risk is greatest with the administration of intravenous potassium, which should be avoided if possible. When potassium is given intravenously, the rate should be no more than 20 mmol per hour, and the patient's cardiac rhythm should be monitored. Oral potassium is safer, because potassium enters the circulation more slowly.

In conclusion, though frequently included in secondary chapters ("varia", "miscellaneous", etc), the potassium abnormalities are a very important subject. The specific electrocardiographic changes caused by imbalance are attributed to the effect of the altered concentration of ions on the transmembrane potentials of cardiac cells. The accuracy of the ECG diagnosis improves when the interpreter is alert to the possibility of an electrolyte imbalance, when control tracing are available for comparison and when the patient is followed with serial tracings.

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