Taquicardia de QRS largo em paciente com extrema hiperpotassemia

Wide Complex QRS Tachycardia (WCQRST) in a patient with Extreme hyperkalemia

Português

Trata-se de um homem de 62 anos, portador de doença arterial coronariana, diabetes Mellitus tipo 2, hipertensão arterial sistêmica e coronariano: História prévia de infarto agudo com colocação de stent e posterior revascularização cirúrgica.
Deu entrada com queixa de tonturas há 4 dias com sudorese e palidez (sem síncope). Faz uso regular de anlodipino, AAS, sinvastatina, monocordil e insulina. PA=170/90; RCR FC 130mbm bulhas hipofonéticas. K^+ 9,3 mEq/L; Ureia > 150mg/dL; creatinina = 4,1 mg/dL).
ECG-1 que revela taquicardia sustentada de QRS largo
Ecocardiograma =contratilidade normal; FE= 52%.
ECG-2 realizado imediatamente após a reversão.
O ECG3 de base após as medidas e hemodiálise.
Pergunta:
Quais são os diagnósticos eletrocardiográficos dos 3 ECGs e por quê?

English

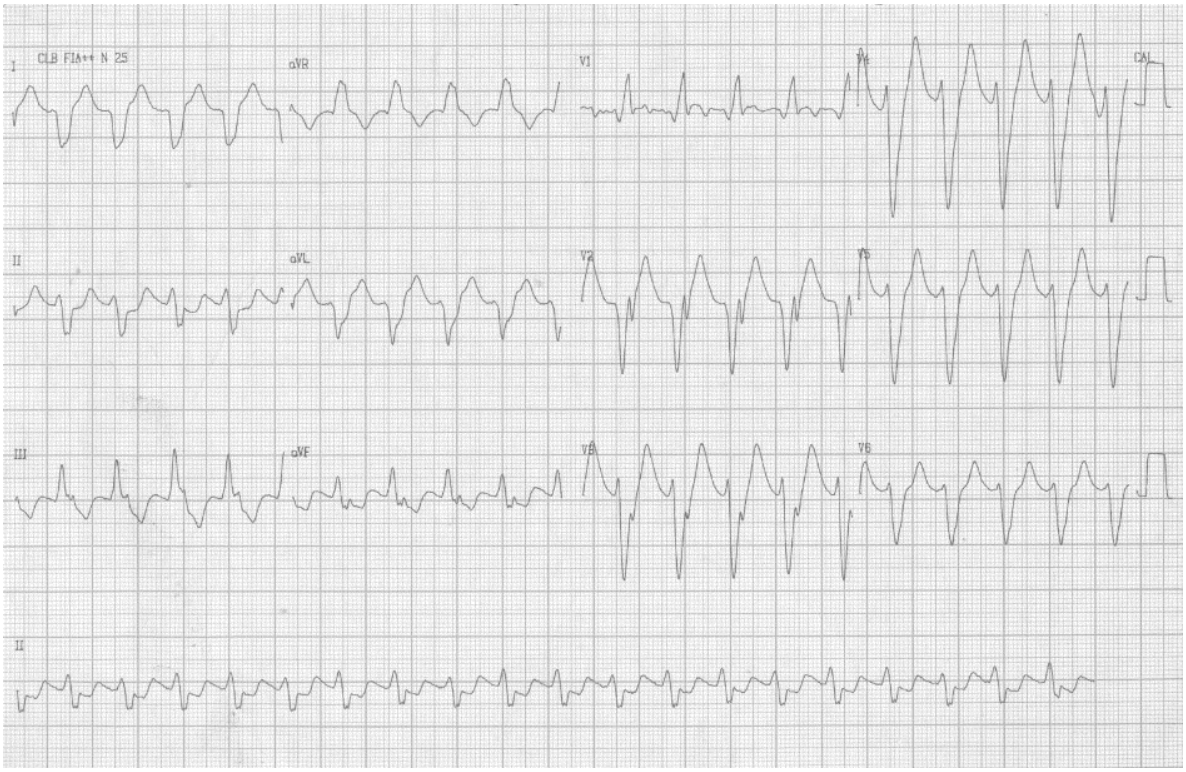
It is a Caucasian man of 62 years old, with coronary artery disease, type 2 diabetes mellitus and hypertension long time ago. He has previous history of acute myocardial infarction treated during emergency with stent placement and subsequent surgical revascularization.
He was admitted with complaints of dizziness for 4 days, sweating and pallor (without syncope). He is using regularly amlodipine, aspirin, simvastatin, monocordil and insulin. BP = 170/90 mmHg; RCR = 130 bpm; hypophonetic sounds. The serum potassium level was very high (K + 9.3 mEq /l). Additionally, uremic picture was present (Blood Urea Nitrogen (BUN > 100mg/dl and Creatinine = 4,1mg/dl.).
ECG-1 revealing sustained complex wide QRS tachycardia
Echocardiogram = normal contractility; FE = 52%.
ECG-2 performed immediately after reversion.
ECG-3: Basal ECG after dialytic treatment.

Question:

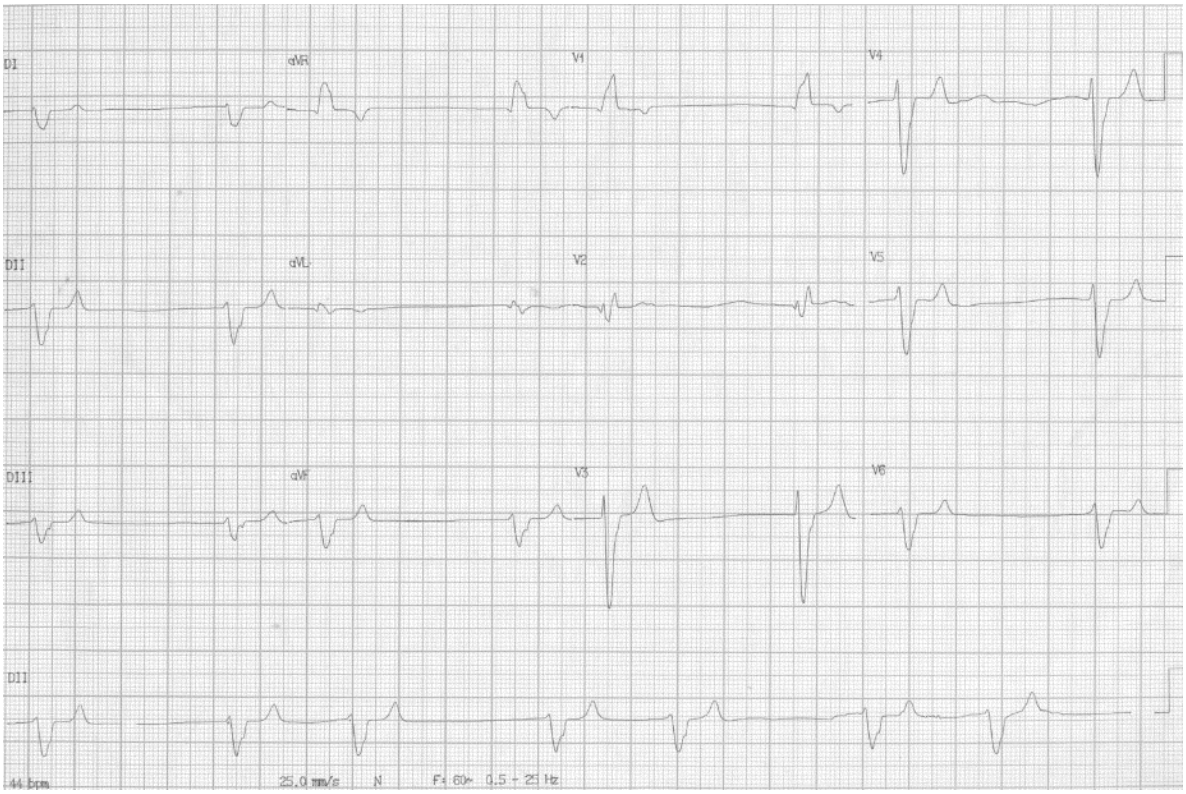
Which are the ECG diagnosis of the three ECGs and why?

Raimundo **Barbosa-Barros &** Andrés Ricardo **Pérez-Riera**

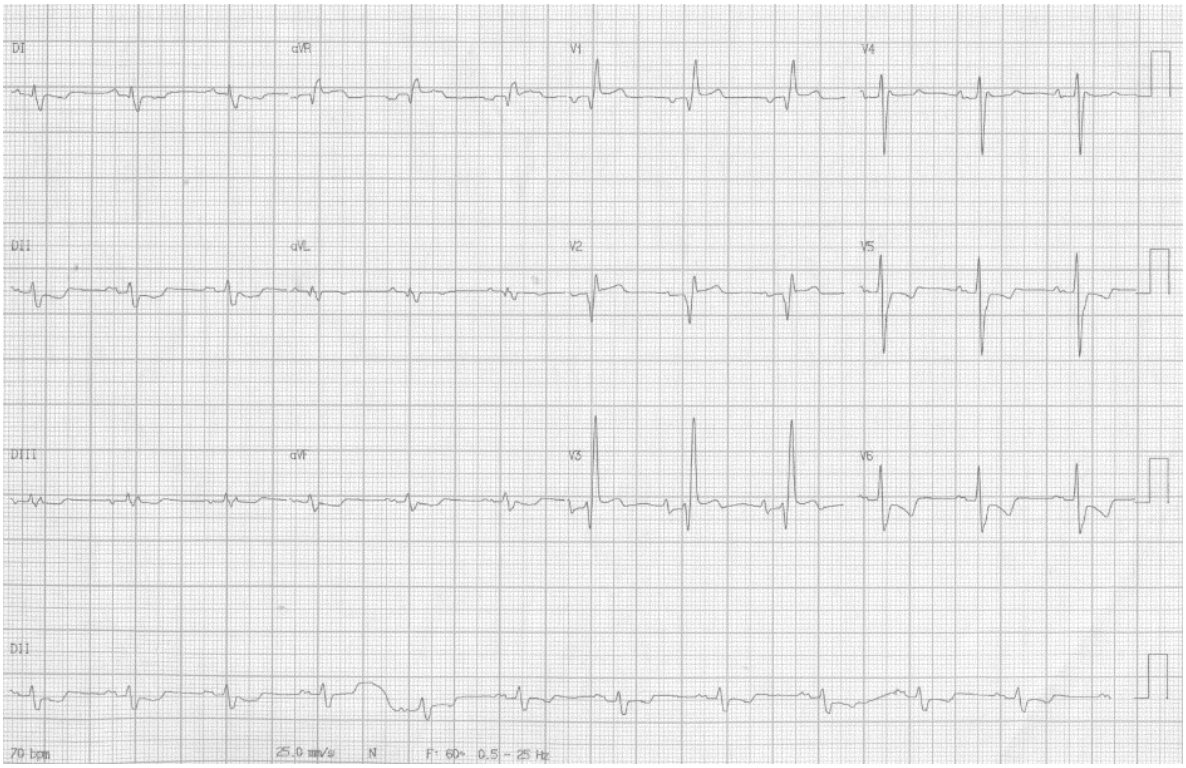
ECG-1: realizado durante o evento / preformed during the event



ECG-2: Realizado imediatamente após a reversão / immediately after reversion



ECG-3: ECG de base após sessão de hemodiálise / Basal ECG after dialitic therapy



Colleague's opinions

Dear Andrés

ECG-1 Supraventricular tachycardia with broad QRS complexes. Tall T waves.

ECG-2- Brady arrhythmia with escape rhythm, right bundle branch block pattern and also tall T waves.

ECG-3 Ischemic T waves from V4 to V6.

Possible renal insufficiency because high potassium levels, elevated urea and creatinine. After dialysis ECG features disappear.

Regards

Eduardo Quiñones

Spanish

Estimado Andres:

1er ECG: Taquicardia supra ventr de QRS ancho, ondas T altas.
2do Ecg: Bradicardia con ritmo de escape, BCRD, onda T sigue alta
3er ECG:Onda T se aplana y se muestra como isquemica en V4 a V6 (por antecedentes coronarios)
Los electrolitos están elevados (K), la creatinina y la urea.
No esta agregada una insuficiencia renal?, ya que el paciente es DBT, coronario e HTA. Al realizar la diálisis el ECG tiende a normalizar su ritmo.
Saludos Eduardo Quiñones

The initial ECG looks like VT from anterolateral LV site.

The 2nd ECG shows a bigeminal rhythm which looks vey similar to ECG in sinus rhythm.the group beating may be due to Wenkebach conduction from junctional focus with QRS widening due to hyperkalemia. Alternatively, this may be sinoventricular conduction (also due to severe hyperkalemia) with S-A atrial exit block.

ECG #3 shows an acute basal injury current compatible with a proximal LAD lesion (note ST elevation in aVR and V1).Alternatively, ST elevation has been described in hyperkalemia as a dialyzable current of injury so we need to know the K level at time of ECG #3

Melvin Scheinman MD

Spanish

El primer ECG-1 parece una TV con foco de origen en la pared anterolateral del VI.

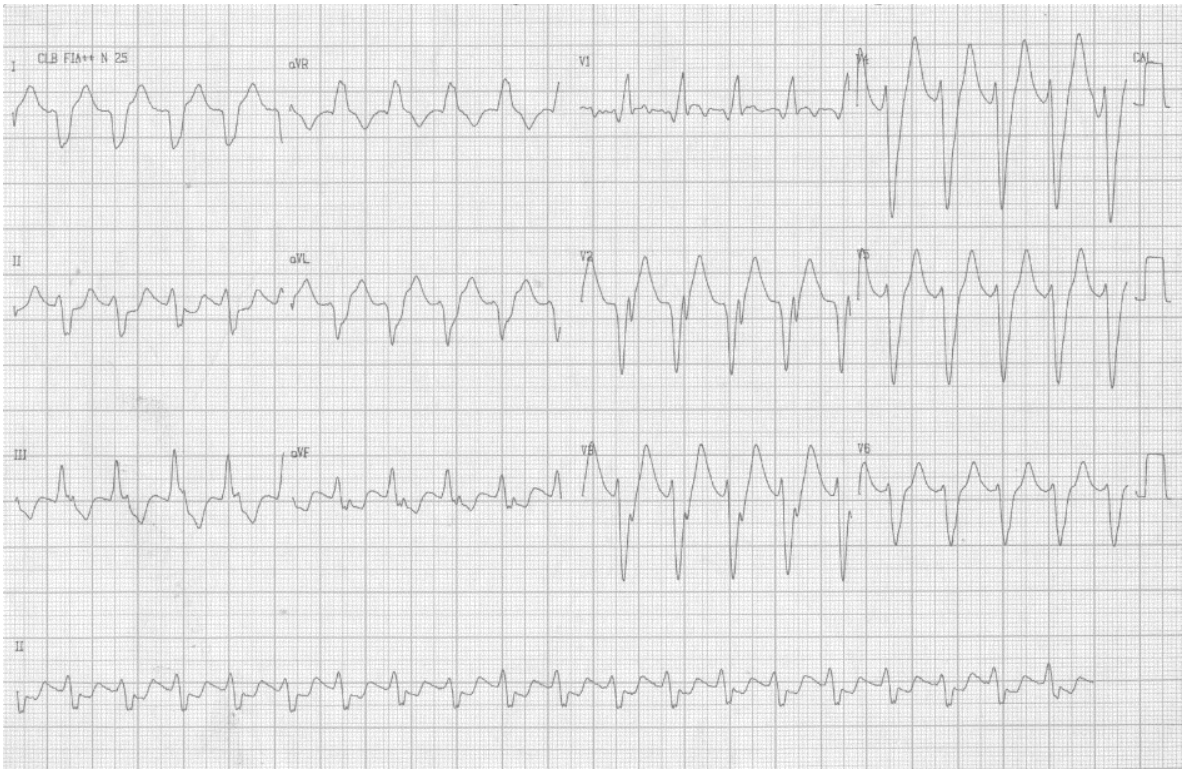
El segundo ECG-2 muestra un ritmo bigeminado el cual parece un ritmo sinusa. Los latidos agrupados pueden ser causados por conducción tipo Wenckebach desde un foco de la unión con QRS anchos por la hiperkalemia, Alternativamente, puede ser un ritmo sinoventricular (también consecuencia de la hiperkalemia) con bloqueo de salida SA,

El ECG-3 muestra una corriente de lesión compatible con obstrucción de LAD (note ST elevado en aVR y V1) Alternativamente , la elevación del ST puede ser consecuencia de hiperkalemia com corriente dializable de injuria , entonces necesitamos saber el nivel de K+ en el momento de la realización del ECG-3.

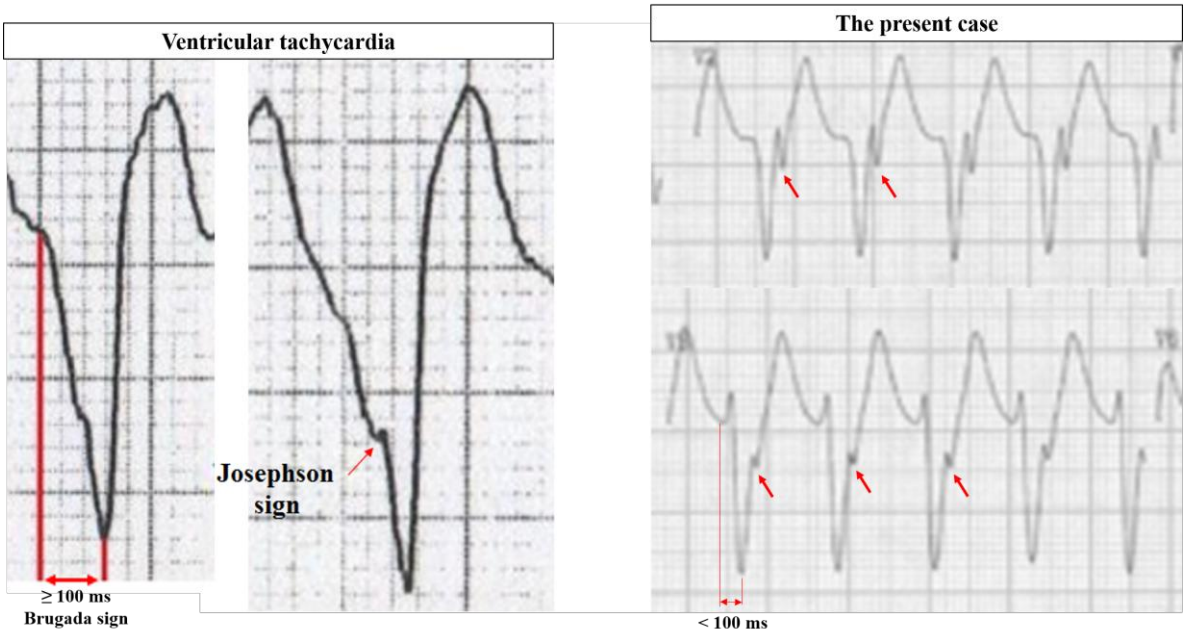
Melvin Scheinman MD

Final Analysis/Comment by Raimundo Barbosa-Barros & Andrés Ricardo Pérez-Riera

ECG-1: realizado durante o evento / preformed during the event



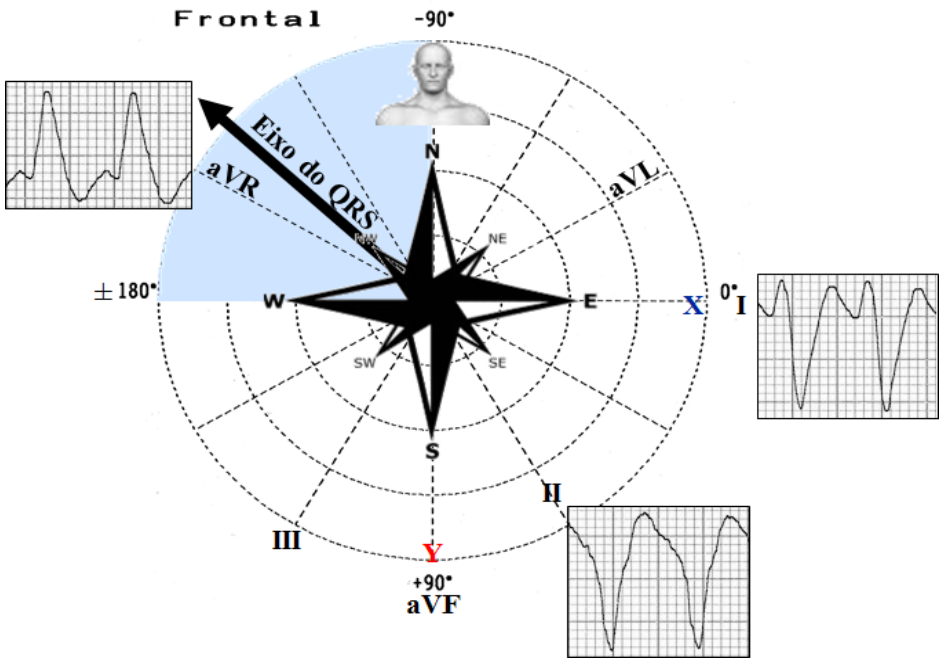
ECG diagnosis: Sustained Wide QRS Complex Tachycardia (WQRST), HR = 125bpm, QRS axis + 150° (it is located on right inferior quadrant: positive QRS only in III and aVR), QRS duration = 161 ms (significantly broad).
Precordial leads show QR pattern in V1, QS in V2 and rS from V3 to V6. Notch on ascending ramp of S wave in V2-V3. This is not the Josephson sign, because it is located on descending ramp of S wave (see next figure)
This pattern is similar to ECG-3, performed in sinus rhythm after dialytic therapy. Peaked T waves with narrow base from V2 to V5.
Conclusion: SVT with nonspecific IV conduction delays caused by electrolyte abnormalities (hyperkalemia) that resulted in Wide QRS Complex Tachycardia.



Notch on the downward slope of the QS or rS near the nadir = TV.	**It is not Josephson sign of VT because notch in the ascending ramp of rS**

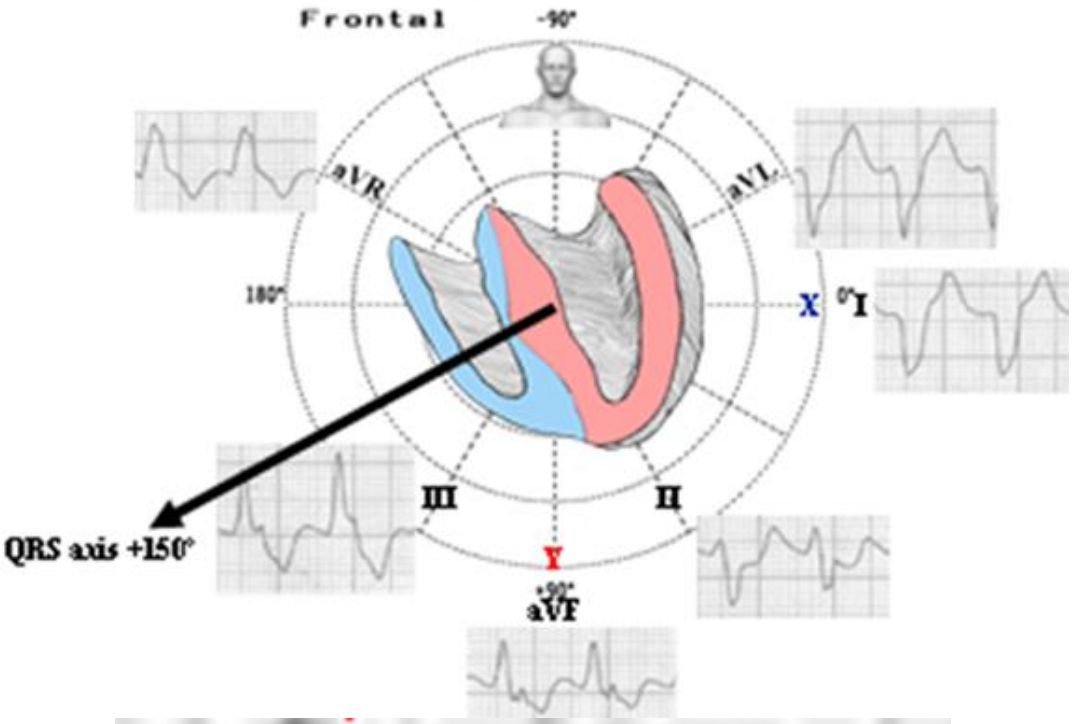
Observation: Notch in the ascending ramp of S wave of V_3-V_4. It is seen more often with anterior MI associated with LBBB (anterior more often than inferior) (**Kindwall 1986; Cabrera 1953.**). Rapid descending ramp of S-waves.

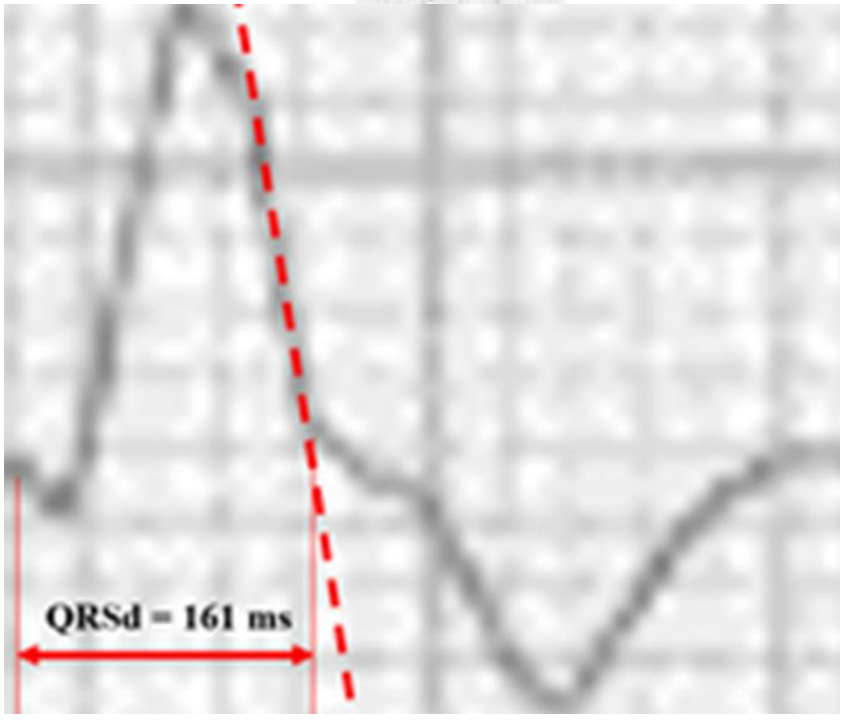
In the present case, the QRS axis is not located in Norwest quadrant (right superior). This location is indicative of VT



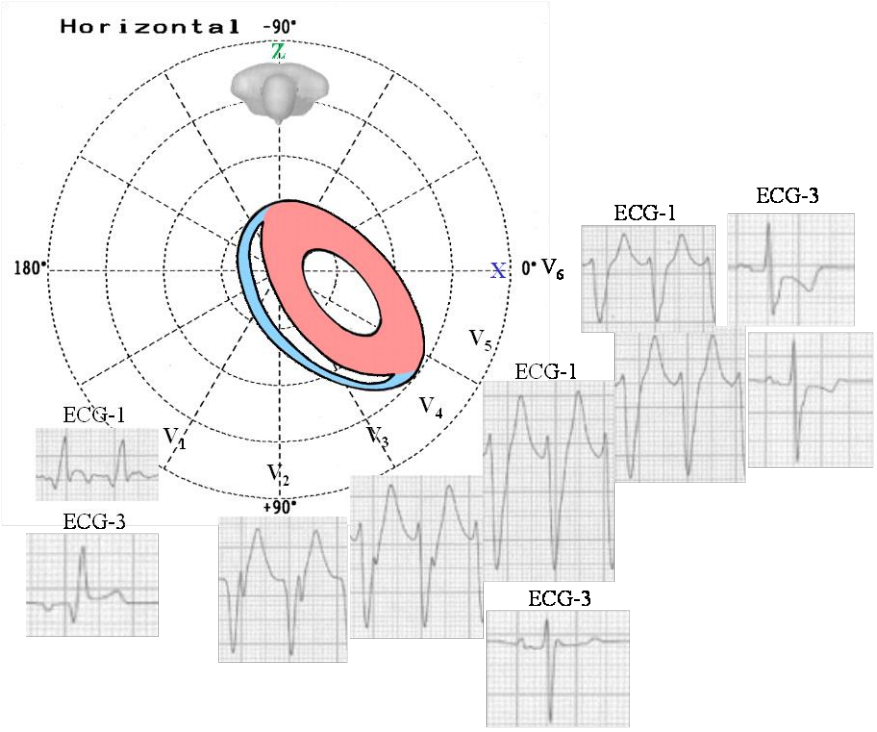
Extreme right QRS axis deviation (Between - 90° and ±180. “Norwest axis” or “no man's land”) in the presence of wide QRS tachycardia is indicative of VT. In these cases, QRS complexes are predominantly negatives in I and II and positive in aVR. Lead a VR is sometimes called **no man's land** or the orphan lead because it stands alone and does not view any single surface of the heart as directly as other lead systems. The **ECG** complexes in lead a VR are usually negative.

The QRS axis is located in inferior right quadrant, consequently it is not characteristic of VT





Very broad QRS duration is suggestive of nonspecific IV conduction delays caused by electrolyte abnormalities (hyperkalemia) that resulted in Wide QRS Complex Tachycardia.



The ECG-1(during the event) and the ECG-3(in sinus rhythm after dialytic therapy) have similar patterns.

Conceptual definitions

Wide QRS tachycardia (WQRST): A name given to any ECG arrhythmic event with heart rates ≥100 bpm and QRS duration ≥120 ms.

Ventricular tachycardia (VT): A wide QRS tachycardia with at least ≥ 3 consecutive QRS complexes with a heart rate of ≥100 bpm originating below the His bundle, i.e. in the ventricular chambers.

Supraventricular tachycardia with aberration (SVT-A): ≥ 3 consecutive wide QRS complexes with a heart rate of ≥100 bpm originating proximal to the His bundle bifurcation. In the presence of a WQRST the following arrhythmias should be considered in the differential diagnosis.

I) Regular or minimally irregular wide QRS complex tachycardia.

- **a.** Supraventricular tachycardia with aberration (SVT-A) due to bundle branch block (15% to 30% of the cases).
  - **i.** Pre-existing or fixed bundle branch block.
  - **ii.** Functional or tachycardia-dependent bundle branch block. (SVT with functional aberrancy)

b. SVT using an accessory pre-excitation pathway for anterograde conduction with a macro-reentry circuit (pre-excited SVT, 1-5% of the cases). The supraventricular impulse reaches the ventricles through the anomalous pathway and returns retrogradely to the atria through the normal AV junction resulting in a wide QRS complex tachycardia. This SVT entity is known as atrioventricular reentrant tachycardia (AVRT).

c. Preexcited SVT using an atriofascicular or nodofascicular accessory pathway as anterograde limb of the circuit, the retrograde limb is usually the normal His-Purkinje system, but may be a second accessory pathway

d. SVT with a bystander atriofascicular or nodofascicular pathway(**Vereckei 2014**)

e. SVT with nonspecific IV conduction delays caused by drugs, electrolyte abnormalities or hypothermia that result in QRS prolongation (the present case).

f. Bundle branch reentrant VT

g. Ventricular tachycardia (VT) (**Wellens 1978**): 80% of the cases and 95% of the cases in patients with structural heart disease. The onset of VT may be markedly irregular during the first 30 sec.

h. Pacemaker-related WCT

i. Artifacts

II) Markedly or grossly irregular wide QRS tachycardia.

a. Pre-excited atrial fibrillation (AF) with anterograde conduction through an accessory pathway.

b. AF with rapid heart rate response and typical bundle branch block.

c. Pre-excited atrial flutter with anterograde conduction through the accessory pathway.

d. Atrial flutter with bundle branch block.

Summary of differential diagnosis of Wide Complex Tachycardia (WCT)

Broad Classification	Specific Arrhythmias/ Considerations
Supraventricular tachycardia with aberrant ventricular conduction(SVT-A)	**SVT with either preexisting BBB or tachycardia-related aberrancy**
Pre-excited tachycardia (in patients with WPW)	Antidromic tachycardia Atrial fibrillation with preexcitation Any other SVT with preexcitation
Ventricular tachycardia	Monomorphic VT Polymorphic VT
Toxic and metabolic derangement	Acidemia Electrolyte abnormalities • Hyperkalemia • Hypomagnesemia Drug toxicity/poisoning • Class IC anti-arrhythmic drugs • Tricyclic antidepressants • Lithium (**Francis 2004**)
Pacemaker-related WCT	Runaway pacemaker Sensor-mediated WCT Atrial-tracking-mediated WCT Endless loop tachycardia
Artifact	Need 12-lead ECG to distinguish artifact from a true WCT

Comments

Important teaching points can be found in this case. First, this patient's history is diabetes mellitus type 2, hypertension with renal failure (uremia) and severe hyperkalemia.

This ECG-1 is suggestive of hyperkalemia because slightly peaked T-waves, widening of the QRS complexes and rightward QRS axis (positive QRS complexes only in III and aVR and large QS-wave in left leads I and aVL), right bundle branch block with QR pattern in V1. Hyperkalemia is known to produce almost any finding on the ECG (**Mattu 2000**), which is

one of the reasons that I often refer to hyperkalemia as the great imitator or "the syphilis of electrocardiography". The physician in this case, however, did not notice the slightly peaked T-waves. Patients with chronic renal failure often "tolerate" much higher serum potassium levels than patients with normal renal function. A serum potassium level of 7.3 mEq/L in a normal patient would likely produce a typical abnormal ECG. Yet, in this patient produced more subtle abnormalities in T-waves with 9.3mEq/L.

The second key teaching point in this case is less well-known: the diagnosis of VT should be avoided in patients with heart rates < 130 bpm. In this case, looked like VT, a rate of 125 beat/min virtually excludes this diagnosis. The only exception would be in a patient already being treated with ventricular antidysrhythmics such as amiodarone, in which VT might occur at slower rates. In contrast, there are a few classic conditions that are notorious for mimicking VT but with slower rates: **hyperkalemia, sodium-channel blocking medication toxicities (e.g., tricyclic antidepressants, cocaine (Levis 2005) and post-myocardial infarction (MI) reperfusion arrhythmias** are the most notable. Common teaching in medicine is that when faced with a wide-complex regular tachycardia, the treating physician should always treat the patient for VT. While this teaching is generally true, the use of traditional "VT medications," such as lidocaine, procainamide or amiodarone, for any of the mimics noted above can actually be deadly. Recall that lidocaine and procainamide are Class I antiarrhythmics — sodium channel blockers. Even amiodarone, which is primarily a Class III antiarrhythmic, does have Class I effects as well. The use of Class I antiarrhythmics for patients with hyperkalemia (which itself is known to poison the sodium channels (**McLean 2000**) or sodium-channel blocker toxicity can produce such pronounced sodium-channel blockade that asystole may result Yet another category of WCTs are those associated with toxic and metabolic derangement. Classic examples in this category of WCTs include anti-arrhythmic toxicity, tricyclic antidepressant overdose, and severe hyperkalemia. Even though the specific etiology can be quite varied, the underlying pathology relates to "poisoning" of the His-Purkinje System through their effect on the ion channels that are responsible for the action potential of the His-Purkinje System. With many of the toxic and metabolic abnormalities, the effects are more generalized. They can also affect the action potential of the sinoatrial (SA) node, atria, atrioventricular (AV) node, and the ventricular myocardium. The net result is a wide QRS complex tachycardia. This is an important category to

distinguish from the others because the usual management strategies for SVT or VT are less efficacious in this setting unless the underlying toxic and metabolic abnormalities are treated. Any anti-arrhythmic drug has the potential to be proarrhythmic, particularly the class IC agents which are potent sodium channel blockers (**Roden 2003**). Overdose or toxicity with class IC agents (e.g., propafenone or flecainide) can cause severe conduction system dysfunction, malignant ventricular arrhythmias, electromechanical dissociation, and asystole (**Koppel 1990**). Flecainide overdose has been reported to result in a mortality rate of 8% compared with other drug overdoses in general that have a mortality rate of less than 1%. Other anti-arrhythmic agents, like class IA and III drugs (e.g., quinidine, sotalol, dofetilide, etc.), tend to be proarrhythmic by their QT interval prolonging effects, which can put patients are risk for torsades de pointes (TdP). This risk of QT interval prolongation is not restricted to anti-arrhythmic drugs, but can happen with a whole host of other commonly used medications (**Al-Khatib 2003**). Tricyclic antidepressant (TCA) toxicity is well known to cause numerous cardiovascular complications including hypotension and wide complex tachycardias (**Thanacoody 2005**). Toxicity is worsened by acidemia, hypotension, and hyperthermia (**Bradberry 2005**). The spectrum of TCA-related cardiac dysrhythmias ranges from sinus tachycardia with a wide QRS complex to ventricular tachycardia and ventricular fibrillation (**Harrigan 1999**). Distinguishing sinus tachycardia with a wide QRS from VT can be difficult in this setting. The presence of an anticholinergic toxidrome supports the diagnosis of TCA overdose and should be sought. TCA toxicity is also supported by the presence of deep S waves in lead I and prominent R waves in lead aVR—which indicates a far rightward deviation of the terminal 40 ms of the QRS complex. This finding is not only suggestive of TCA cardiotoxicity (sodium channel blocking agents in general) but also predictive of dysrhythmia (**Liebelt 1995**). Many other psychiatric drugs have also been reported to cause a WCT (e.g., lithium toxicity) (**Francis 2004**). Among electrolyte abnormalities, hyperkalemia is a common source of severe conduction abnormalities. As serum concentrations of potassium rise, persistent membrane depolarization impairs sodium channel activity, resulting in a wide QRS complex arrhythmia that can simulate VT and, if unabated, can result in VF and asystole. Because hyperkalemia causes slowing of atrioventricular and intraventricular conduction, the wide QRS complex arrhythmias that result from hyperkalemia are rarely faster than 140 bpm, usually have extremely wide and

bizarre QRS morphologies, and do not demonstrate any rapid deflections within the QRS complex (**Dittrich 1986; Mattu 2000; Fisch 1973; Dananberg 1999**).

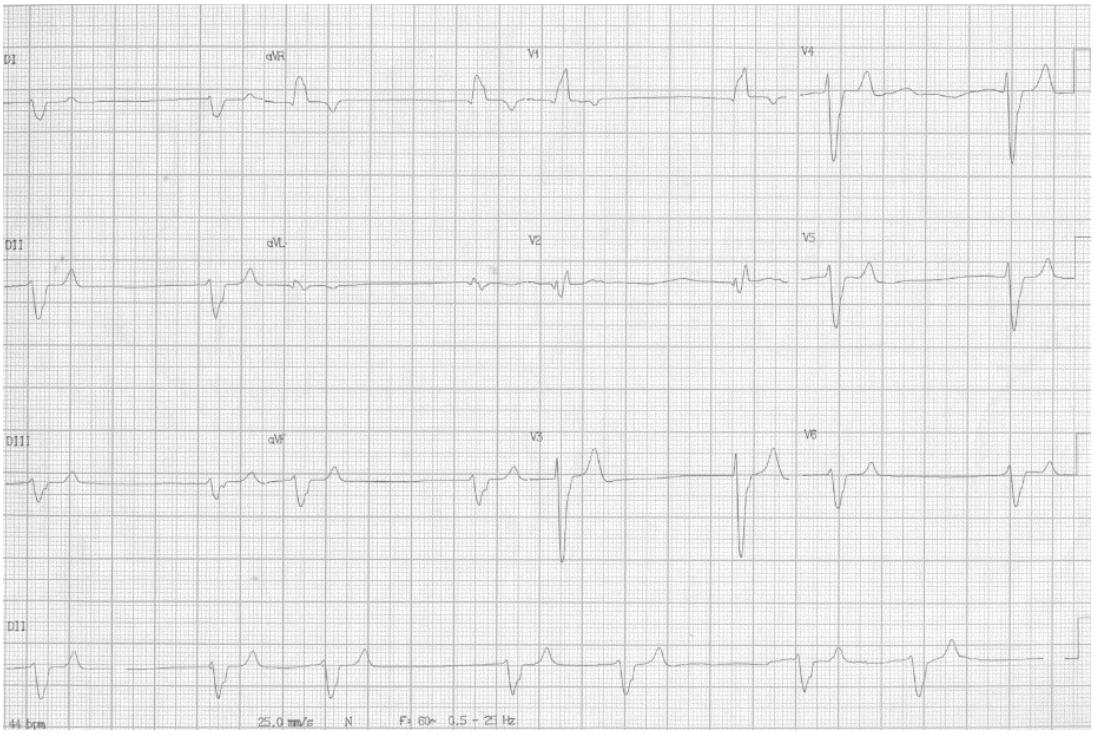
When a rhythm looks like VT but the rate is too slow consider:

- **Hyperkalemia**
- **Sodium-channel blocker toxicity**
- **Pos-MI repefusion arrhythmia** recorded arrhythmias following different revascularization procedures in acute ST elevation MI may not always indicate vessel patency and reperfusion. Ongoing vascular occlusion and ischemia may lead to various arrhythmias which may not be distinguished from reperfusion arrhythmias. The most frequently observed arrhytmias that are defined as reperfusion arrhytmias are ventricular premature contractions, sustained or nonsustained episodes of VT, **Accelerated Idioventricular Rhythm(AIVR)**, AF, and VF. These arrhythmias are thought to be indicators of successful reperfusion. However, in some studies it was mentioned that these arrhytmias may be due to ongoing myocardial cell damage and ischemia (**Heper 2008**). The presence of AIVR combined with normalization of ST segments was demonstrated to indicate successful reperfusion in patients treated with thrombolytics and there was no requirement for emergency coronary angiography and rescue PCI in this group of patients (**Chikadakis 2001**). Post-MI reperfusion arrhythmias often take the form of a regular, wide-QRS complex dysrhythmia with a rate of 90-120. The rhythm is often mistaken for VT. Treatment with “standard” VT medications is well-known to induce asystole (**Kastor 1998**). Proper treatment of this reperfusion arrhythmia is simple observation; the rhythm is usually transient, lasting no more than a few minutes, and it is not hemodynamically-compromising. In these settings, the use of Type I antiarrhytmics or amiodarone can be deadly. Patients with chronic renal failure can “tolerate” greater levels of hyperkalemia than normal patents withotuh pronounced ECG abnormalities. Consider the presence of hyperkalema in all patients with chronic renal failure that present to de ED with any systemic complain Rapid evaluation of the patient’s electrolytes, especially the serum potassium and magnesium level, can be very useful. Electrolyte abnormalities cannot only trigger ventricular arrhythmias, but can also complicate management by decreasing the success rate of cardioversion/defibrillation.

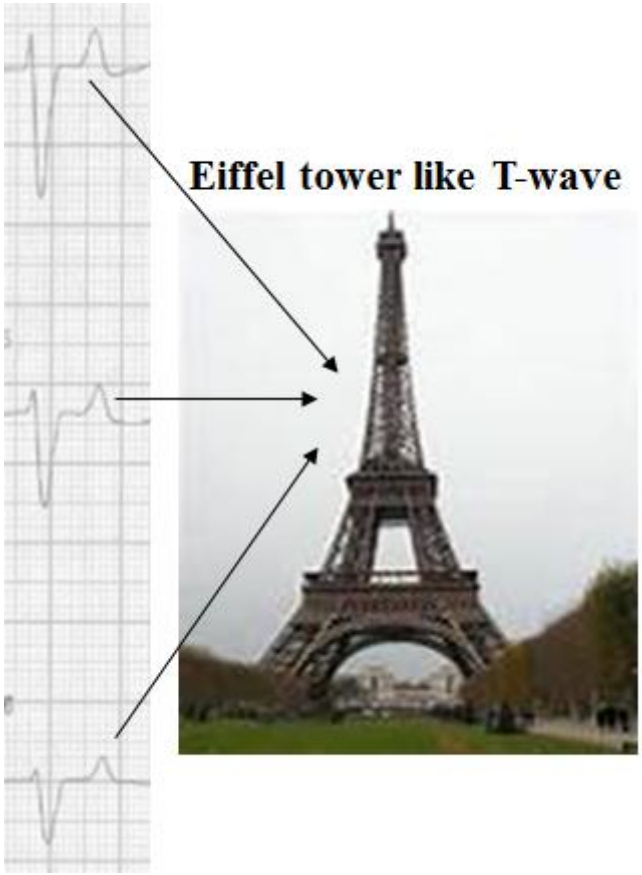
Other laboratory studies that might assist in the diagnosis of WCT are troponin and BNP assays. Because of the urgency involved, laboratory studies that are not available within a 10- to 15-minute turn-around time are generally not that useful in the initial evaluation and management of WCT. Therefore, a venous or arterial blood gas with electrolytes might be the most expedient method of measuring pH and electrolytes (i.e., the potassium) in most institutions.

Hyperkalemia is defined as a serum potassium concentration > 5.5 mEq/L in adults; the range in infants and children is age-dependent. Levels higher than 7 mEq/L can lead to significant hemodynamic and neurologic consequences, whereas levels exceeding 8.5 mEq/L such us the present case can cause respiratory paralysis or cardiac arrest and can quickly be fatal.

ECG-2: Imediatamente após a reversão / Immediately after reversion

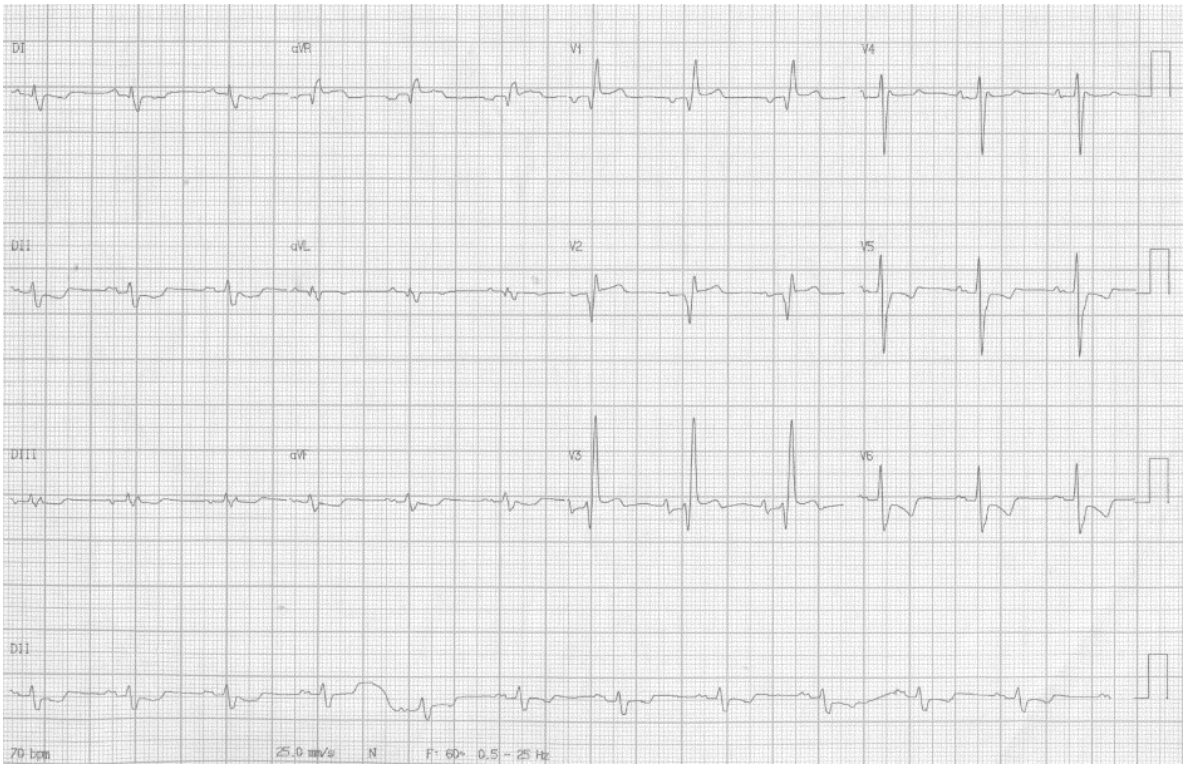


ECG diagnoses: irregular sinoventricular thythm or sinoventricular conduction in atrial standstill. with 2:1 pattern sequence. This rhythm implies preserved sinus node function with conduction of impulses to the A-V junction without generalized atrial excitation. The absence of P wave is characteristic. The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded). Impulse propagation in such cases is presumably via specialized internodal tracts. bradiarrhythmia with peaked T waves (**Ross 1976**). Upright peaked T-waves (see next figure).



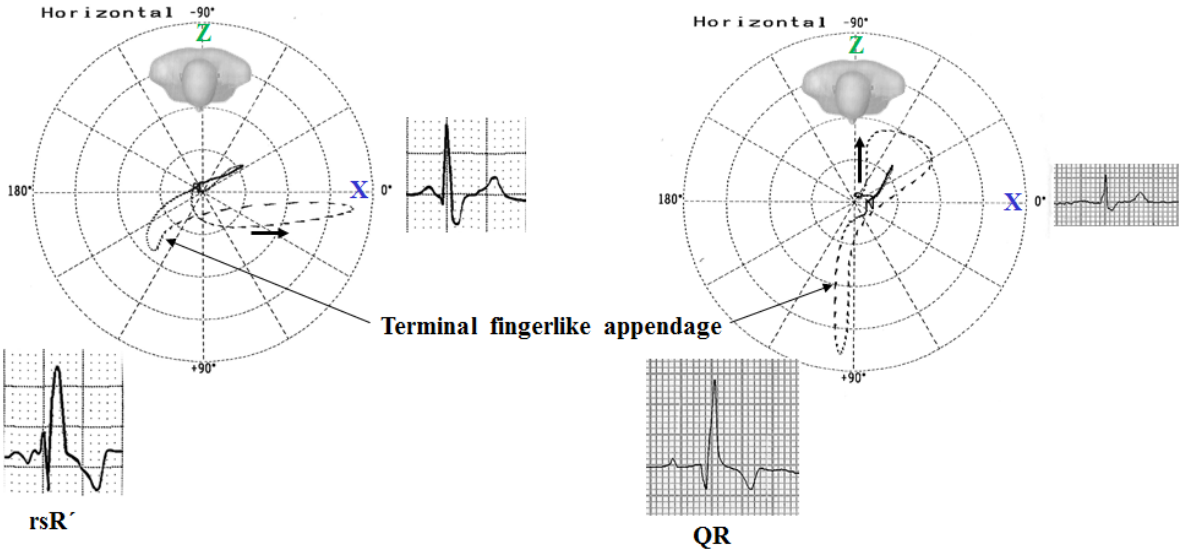
Tall, peaked/pointed or sharp apex, symmetric and narrow base T waves called tended T-wave or Eiffel tower T-wave. Hyperacute T waves are the earliest-described electrocardiographic sign of acute ischemia, preceding ST-segment elevation. The principle entity to exclude is hyperkalemia-this T-wave morphology may be confused with the hyperacute T wave of early transmural myocardial infarction. The hyperacute T wave includes both transmural acute myocardial infarction and hyperkalemia as well as early repolarization, left ventricular hypertrophy, and acute myopericarditis (**Levis 2015**). (**Brady 2000**)(**Morris 2002**). Additionally, in acute coronary syndromes, patients with diabetes such as this case have significantly higher serum potassium concentrations and do not exhibit the early dip seen in non-diabetics. This may reflect sympathetic nerve dysfunction that commonly complicates diabetes (**Foo 2003**).

ECG-3: ECG de base após sessão de hemodiálise / Basal ECG after dialysis therapy



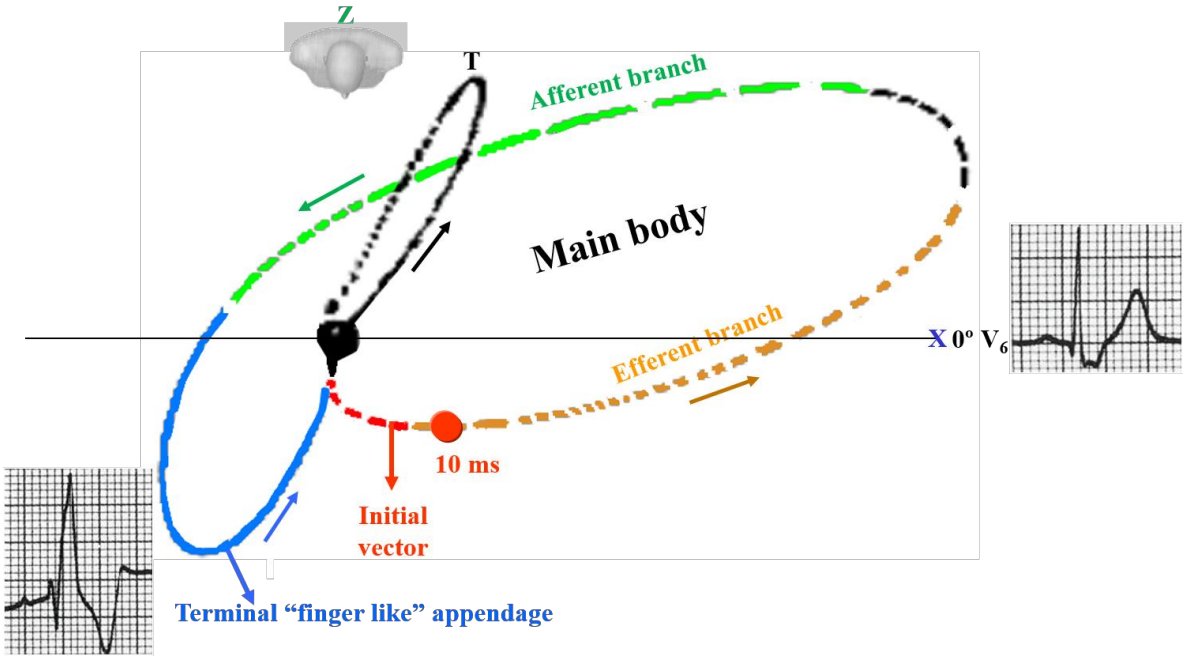
ECG diagnosis: sinus rhythm, low QRS voltage only in frontal plane leads (no wave exceeds 5 mm, 1 large square or 5 small squares, vertically) in the leads of the Frontal Plane). Determination of QRS axis is hard, QRS duration 160ms (equal QRS duration than ECG-1), qR or Qr pattern the right-side precordial leads from V1-V2. In this case, it is a diagnostic signs of septal myocardial infarction in association with RBBB: There is loss of the initial R wave in these leads However, the QRS patterns are modified by the presence of RBBB, so that in addition to the abnormal Q waves tall late R waves appear in these leads: complete right bundle branch block associated to septal myocardial infarction. (See possible causes of qR pattern bellow). In cases where the MI is more extensive, abnormal Q waves may also appear in leads aVL and I and the left precordial leads. In this case, negative T waves are observed in lateral precordial leads from V4 to V6.
Electrocardiogram demonstrating RBBB septal MI and absence of peaked T waves after hemodialysis and normalization of serum potassium.
Observation: Coexisting RBBB and anterior MI are individually recognizable in the VCG and ECG because the electrical effects of the two conditions appear at different times in the QRS interval. The vector loop of RBBB, therefore, can be divided into an initial portion representing the activation of the LV and a terminal portion representing activation of the RV. Since most infarctions involve the LV and produce changes during the initial portion of the QRS complex, their recognition is not hampered. The orientation and configuration of the first portion of the QRS loop are affected only by myocardial infarction, while the right bundle branch block did not interfere with this portion of the loop. However, the terminal deflection of the QRS loop is typical of right bundle branch block (**Doucet 1965**).

Uncomplicated RBBB **RBBB associated with septal or anteroseptal MI**



	Uncomplicated RBBB	RBBB associated with anterior MI
Initial 40 ms deflection	It is recorder to right and anteriorly	It is directed to back
QRS pattern on right precordial leads	Triphasic rSR'	Biphasic QR or qR
Final 60 to 980 QRS forces	Located on right anterior quadrant	Located on right anterior quadrant
Terminal VCG forces of QRS loop in HP	Terminal finger-like appendage on right anterior quadrant which is recorder slowly	Terminal finger-like appendage on right anterior quadrant which is recorder slowly

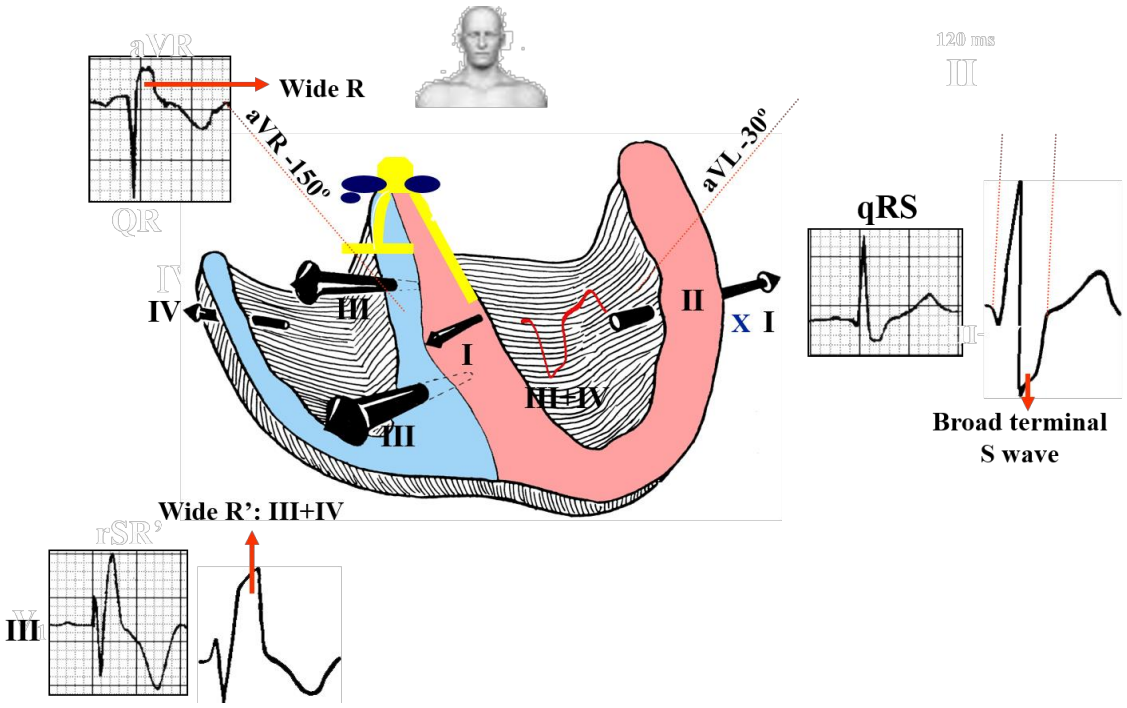
ECG/VCG criteria of uncomplicated complete RBBB in the HP



QRS loop in the HP formed by: initial vector, efferent branch, afferent branch, main body and terminal appendage with delay.

The terminal late forces of the horizontal plane are directed to the right and anteriorly with characteristic terminal "finger like" appendage of the QRS loop.

Vectorial representation of ventricular activation in uncomplicated complete RBBB



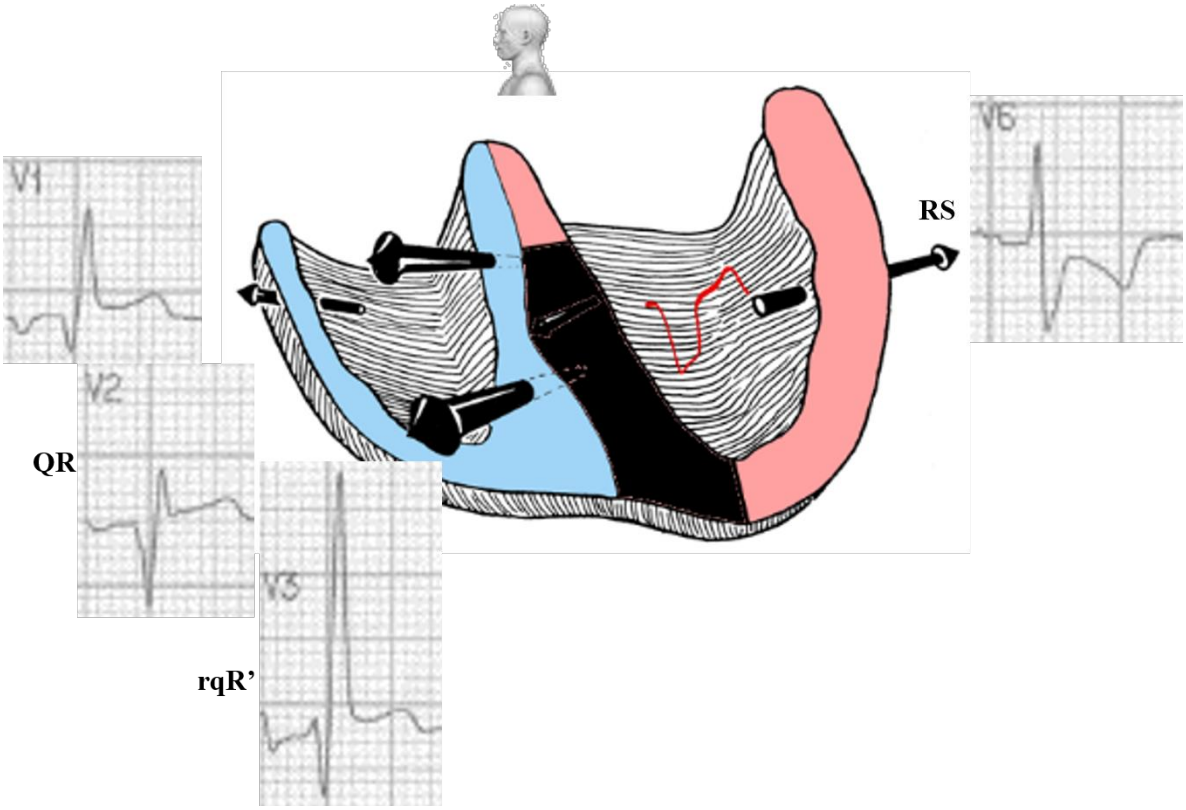
I. Initial activation of middle third of left septal surface and activation of muscle mass of left septum and of apico-anterior LV free wall;

II. Activation of anterolateral wall of LV free wall from endo to epicardium;

III. Activation of basal LV wall; continued left-to-right septal activaton and apicoanterior activation of RV; Completion of septal activation (slow trans-septal vectors) and continued activation of RV free wall;

IV. Activation of basal wall of RV (RVOT) and/or septum.

Complicated complete RBBB with septal MI



Biphasic QR or qR pattern in V1-V2, absence of initial vector.

Possible causes of QR/qR pattern in right precordial leads
Severe right ventricular hypertrophy (**Gandhi 1962**) (supra-systemic intraventricular pressure inside right ventricle)>
• Right Atrial Enlargement: qR pattern in V_1 may be an indirect sign of RAE (**Sodi-Pallares1952).**
• Complete RBBB complicated with anterior Myocardial Infarction (**Rudiakov 1964**).
• Ebstein's anomaly: bizarre and low voltage RBBB with initial q wave (**Kumar 1971**).
• Congenitally Corrected Transposition: Secondary to inversion of septal activation, RAE, by progressive tricuspid regurgitation that occurs with age and associated with deterioration of RV function (**Warnes 2006; Ruttenberg 1966**).
• Endomyocardiofibrosis (**Tobias 1992**).
• MI or ischemia / injury associated with LSFB. S-T elevation and increase in R-wave voltage, "giant R waves", and concomitant shift of the frontal QRS axis toward the locus of injury is also displayed (**David 1982**; Deanfield 1983; Schick 1980; **Feldman 1986; Hassapoyannes 1991; Madias 1993; Moffa 1996; Moffa 1997; Uchida 2006**).

Causes of hyperkalemia

Factitious hyperkalemia, artifactual hyperkalemia or pseudohyperkalemia: 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 (**Jaya 2013**). Centrifuging samples may help prevent false readings (**Gourlay 1997**). It is also seen in patients with thrombocytosis and in the rare patient with familial pseudohyperkalemia (**Sugimoto 2005**). Not well appreciated is its occasional occurrence in patients with extremely elevated white blood cell counts, particularly in patients with chronic lymphocytic leukemia (CLL).

Increased intake

- Potassium supplements
- Penicillin G potassium
- Nutritional supplements

Increased shift from intracellular space

- Cell destruction
- Massive hemolysis
- Tumor lysis syndrome
- Rhabdomyolysis
- Burns
- Trauma

Normal anion gap acidosis

Lack of insulin Diabetic

I. ketoacidosis
II. Starvation
III. Somatostatin

Hyperosmolality

Hyperkalemic periodic paralysis

Succinyl choline

β-Blockers

Digoxin intoxication Dried toad skin (Chan Su/Senso)

Intravenous amino acids

Impaired renal excretion

Decreased distal flow

- Decreased effective circulating volume
- Chronic or acute renal failure
- Nonsteroidal anti-inflammatory drugs

Hypoaldosteronism

Primary adrenal insufficiency

Medications and herbals

- Spironolactone
- Triamterene
- Amiloride
- Angiotensin-converting enzyme(ACE) inhibitors/ angiotensin receptor blockers(ARBs) When when these drug are initiated in patients with an estimated GFR of less than 60 mL/min, serum potassium levels should be checked within 1 week.
- Trimethoprim/pentamidine
- The simultaneous use of β-blockers and trimethoprim-sulfamethoxazole (TMP-SMX) (**Matthew 2010**)
- Cyclosporine/tacrolimus
- Heparin

Primary renin insufficiency

Pseudohypoaldosteronism

Distal renal tubular acidosis

Congenital adrenal hyperplasia

Interstitial renal disease

Unknown mechanism

- Alfalfa
- Dandelion
- Noni juice *ACE = angiotensin-converting

Serum K^+ level mEq/L	In healthy humans, serum K^+ levels are tightly controlled within the narrow range of 3.5 to 5.0 mEq/L(**Mc Donald 2003**)
Light hyperkalemia 5.5-6.5	T-waves become abnormally tall, peaked/pointed, symmetrical, with narrow base: “Eiffel tower T-waves” or in “desert tend T-waves”
Moderate hyperkalemia 6.5-7.0	P wave become broader and flatter (slow interatrial conduction): reduction in P wave amplitude, prolonged PR interval(first degree AV block R wave height decreases, QRS complexes become wider and ST segments have elevation in some leads and depression in others ST-segment deviation simulates “acute injury” pattern or “dialyzable injury current”. Brugada phenocopy
Severe hyperkalemia 7.0-7.5 It is called life threating hyperkalemia. It is commonly associated with acute renal failure	Further widening and distortion of QRS occurs Nonspecific intraventricular conduction pattern, prolonged QT interval, and premature ventricular beats become frequent
Extreme hyperkalemia >7.6	Absent P waves, frequent escape beats. Sinoventricular rhythm The combination of an irregular rhythm The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded). Absent P weave may simulate AF. atrioventricular block, very broad and bizarre QRS complexes. VT. VF or ventricular asystole with potassium concentration above 12 to 14 mEq/L

Summary of electrocardiographic features in hyperkalemia

1. Peaked T waves
2. Shortened QT intervals
3. Prolonged PR intervals
4. Reduction in the amplitude of P waves
5. Absent P waves(sinoventricular rhythm)
6. Nonspecific intraventricular conduction delay
7. Very broad and bizarre QRS complexes and 'sine-wave' ventricular rhythms.
8. Brugada phenocophy (**McIntyre 2011**; **Anselm 2014**)
9. Severe hyperkalemia eventually causes fatal arrhythmias such as ventricular fibrillation or asystole, leading to cardiac arrest

Action potential of rapid His-Purkinje system cardiac cells in hyperkalemia

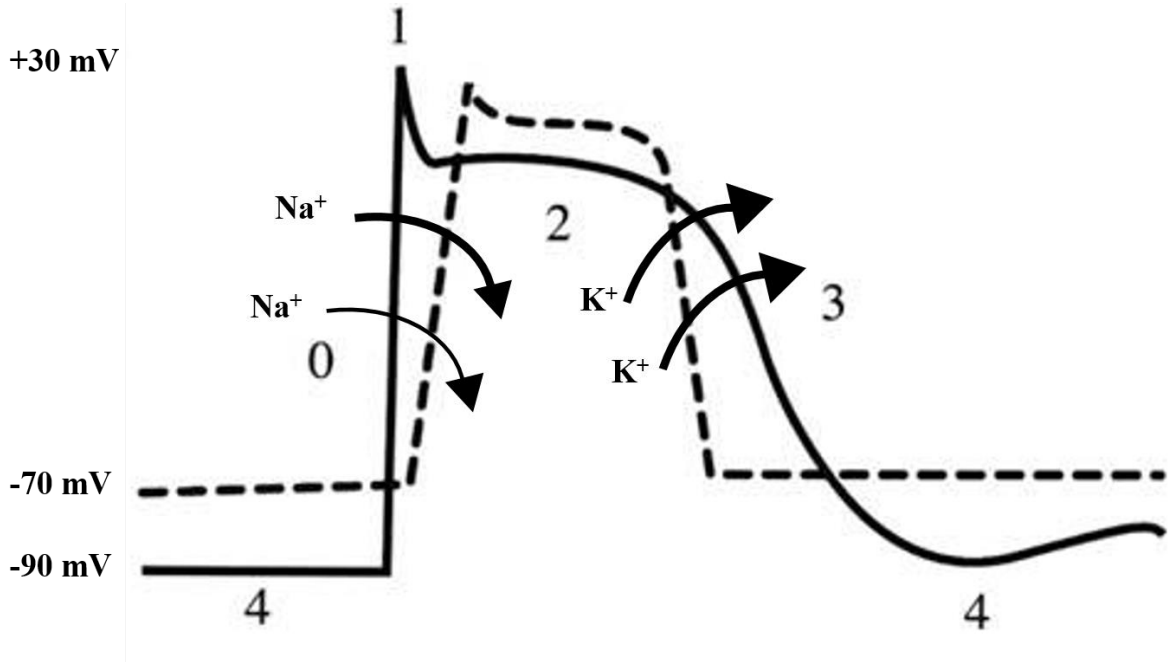


Illustration of a normal action potential (AP) (solid line) and the AP as seen in the setting of hyperkalemia (interrupted line). The phases of the AP are labeled on the normal AP. Note the decrease in both the resting membrane potential and the rate of phase 0 of the AP (V_{max}) seen in hyperkalemia. Phase 2 and 3 of the AP have a greater slope in the setting of hyperkalemia compared with the normal AP (**Parham 2006**).

Management of Hyperkalemia with ECG changes

The goal of treatment is to prevent cardiac arrhythmia, then lower serum potassium. The management of hyperkalemia can be summarized by the mnemonic C (See) BIG K DROP from (**Moses 2014**).

Mnemonic	What it means
C "See"	**C**alcium: Calcium gluconate (10%) 10 mL IV over 10 min. Calcium is a – cardiac stabilizer.
B	**B**eta agonists: Salbutamol 10 - 20 mg in 4 mL normal saline nebulized over 10 min OR - Bicarbonate: sodium bicarbonate 8.4% (50 mEq) 1 ampoule IV over 5 minutes (**Kamel 2003**) – Both of these agents cause temporary intracellular shift.
I	**I**nsulin: Short acting insulin 10 units IV push followed by ... (see next box!)
G	**G**lucose: D50W 1 ampoule IV over 5 minute given with insulin. Insulin causes– temporary intracellular shift and glucose is given to maintain blood glucose levels.
K	**K**ayexalate: Sodium polystyrene sulfonate 15-30 g in 15-30 mL (70% sorbitol) PO. (**Kamel 2003**.) Kayexalate may facilitate– gastrointestinal removal. This is mainly for CHRONIC renal failure. Not for use in the ACUTE phase.
D	Diuretics: Furosemide 40-80 mg IV push. This facilitates – renal removal.
ROP "Renal unit for dialysis Of Patient"	Dialysis achieves extracorporeal removal.

A Cochrane review concluded that, when ECG changes due to hyperkalemia are present, IV calcium is effective in preventing deterioration (**Mahoney 2009**). Thereafter, emergent therapies for lowering potassium levels are nebulized or inhaled salbutamol and/or IV insulin-and-glucose (**Mahoney 2009**). There is no evidence supporting the use of bicarbonate as monotherapy (**Mahoney 2009**). Existing evidence does not support the use of bicarbonate for inducing intracellular shift in treating acute hyperkalemia (**Kamel 2003**). Kayexalate is an exchange resin used to bind potassium in the intestine when given orally. The two concerns with Kayexalate are its ineffectiveness in lowering serum potassium and its potential toxicity. Dialysis is efficient, but usually takes about 2 hours to get started in a patient with no dialysis access.

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