Taquicardia de Complejos QRS Anchos en hombre mayor

Wide Complex QRS Tachycardia in senior man

Case from Dr. Luciano Pereira, Ciudad del Este, Paraguay

Español: Reporte de un caso

Hombre caucasiano de 76 años, dio ingreso a la sala de urgencias en un hospital del interior del Departamento Alto Paraná, Paraguay. Refería que desde hacia una media hora presentaba palpitaciones rápidas y regulares acompañadas de opresión torácica. El médico que lo recibió siguió el laudo de interpretación del trazado informado automáticamente por la máquina.

Antecedentes personales patológicos: diabético tipo 2 e hipertenso, en tratamiento regular con glimepirida/metformina y enalapril. El paciente presentó Troponina cualitativa positiva y el ecocardiograma transtorácico reveló hipocinesia ínfero-septal. Sin embargo, la cinecoronariografia (CCG) no mostró lesiones coronarias significativas.

Fue adicionado atorvastatina 40 mg, clopidogrel, ácido acetilsalicílico y ansiolítico.

ECG-1 realizado en la admisión durante el evento.

ECG-2 realizado a los dos días de haber ocurrido la reversión del episodio.

Pregunta:

• ¿Cuál es el diagnóstico ECG de esta taquicardia de QRS ancho?

Dr. Luciano Pereira, Ciudad del Este, Paraguay

English: Case report

Caucasian man 76 years, was admitted to the emergency room at the regional hospital (Department of Alto Parana, Paraguay). He complained of fast, regular palpitations with chest tightness started half an hour before. The emergency doctor followed the automatically report of the device layout.

Medical history: antecedent of type 2 diabetes mellitus and hypertension for long time ago. He is in regular treatment with glimepiride / metformin and enalapril maleate. The patient had positive troponin qualitative and transthoracic echocardiogram showed inferiorseptal hypokinesis. However, coronary angiography showed no significant coronary obstructions.

It was added 40 mg atorvastatin, clopidogrel, aspirin and anxiolytic agent.

ECG-1 performed on admission during the event.

ECG-2 conducted two days of the reversal episode occurred.

Question:

What is the ECG diagnosis of this wide complex QRS tachycardia?

ECG1 - Name: WM; Age: 76 y/o; Ethnic group: Caucasian; Weight: 81 kg; Height: 1.68 m; Body surface: 1.91 m.



ECG-2 performed 48 hours after the event



Collegue's opinions

Spanish: La taquicardia de complejo ancho de morfologia BCRI tiene la misma morfologia que el complejo ventricular en sinusal. Creo que se trató de una taquicardia supraventricular (flutter o TA). Juan Sebastián

Wide QRS tachycardia has the same LBBB morphology of sinus rhythm, consequently I think that it is SVT-A flutter or atrial tachycardia.

The Wide complex tachycardia is quite similar to that recorded in Sinus rhythm. Possibilities include SVT-A of diverse etiology especially Atrial tachycardia (a little too rapid for flutter). In addition, one must exclude bundle to bundle reentry as described by Akhtar (1) in this setting since the latter may be cured with simple ablation of the Right bundle Branch. An invasive EP study is indicated. Melvin Scheinman

1. Akhtar M, Gilbert C, Wolf FG, Schmidt DH. Reentry within the His-Purkinje system. Elucidation of reentrant circuit using right bundle branch and His bundle recordings. Circulation. 1978 Aug;58(2):295-304.



Spanish: Caso del Dr Luciano Pereira segun mi opinion es una taquicardia atrial con conducción anterógrada 1/1 en presencia de un bloqueo troncular de rama izquierda

Samuel Sclarovsky M.D. Israel

Dr Luciano Pereira's case report: in my opinion it is an atrial tachycardia with 1:1 anterograde conduction in the presence of troncular Left Bundle Branch Block.



Spanish: Taquicardia de QRS ancho con morfología de BRE con presencia de disociación AV en la derivación aVF (latidos de fusión) sugestivo de TV. Además la duración del QRS durante taquicardia es menor que ritmo sinusal (este hallazgo también habla a favor de la TV). El ritmo sinusal muestra un patrón de crecimiento de cámaras izquierdas + BRE. Creo que debemos pensar en TV por reentrada rama - rama.

Saludos

Raimundo Barbosa Barros Fortaleza Ceará Brasil

English: Wide complex QRS tachycardia with left bundle branch block morphology, because the presence of AV dissociation in lead aVF (fusion or Dressler beats) suggestive of TV. In addition, the QRS duration during the event is lesser than QRS duration during the sinus rhythm (this finding also suggest TV). Sinus rhythm shows enlargement of the left chambers + left bundle branch block. I think we should think in TV by branch to branch reentry.

Greetings

Raimundo Barbosa Barros Fortaleza M.D. Ceará Brazil



Spanish

Estimados amigos Dr. Luciano y Maestro Potro: Pienso que el amigo Barbosa describió con mucha propriedad los hallagos curiosos del ECG e veo dissociacion tambien em V2 e V3. El eco con una acinesia inferobasal apunta al foco de origem del foco de la TV e asi lá excitacion de los ventriculos es similar a la excitacion en ritmo sinusal con bloqueo de lá rama izquierda pues son próximas.

Recuerdo que el ECG 1º del libro de los hermanos Brugadas "Nuestros mas queridos electrocardiogramas" - Similar no significa idéntico, in honor a David Ross.

Adail Paixão Almeida Vitória da Conquista Bahia Brasil

Dear friends Dr. Luciano and master Potro: I think that our friend Raimundo Barbosa described appropriately the ECG features. I also observe dissociation phenomena in V2-V3. Additionally, the echo with inferobasal akinesis signalizes the focus of the VT and thus the activation of the ventricles during the event is similar to the activation in ECG-2 with sinus rhythm and left bundle branch block because they are close to each other.

I remember that in the first page of the book of the Brugada brothers entitled "**Our beloved electrocardiograms**" – Similar does not mean identical, in honor of David Ross.



Spanish: Estimado Andrés: Presenta una taquicardia regular de 240 por minuto con bloqueo completo de rama izquierda con S empastada en V4. Diagnóstico presuntivo: aleteo auricular con conducción AV 1.1.

En el ECG-2 posterior se evidencia ritmo sinusal con frecuencia cardiaca de 60 latidos por min con signos de crecimiento auricular izquierdo. BCRI con eje eléctrico desviado a la izquierda y fragmentación del QRS en cara inferolateral.

Pienso en una miocardiopatía primero descartar enfermedad de Chagas y de ser negativa, miocardiopatía diabética.

Un cordial saludo

Martín Ibarrola Buenos Aires Argentina.

English

Dear Andrés: Presents a regular broad QRS tachycardia of 240 bpm and complete left bundle branch block-like pattern, with broad S wave in V4. **Presumptive diagnosis**: atrial flutter with AV conduction 1.1.

ECG-2 further evidenced sinus rhythm with heart rate of 60 beats per minute with signs of left atrial enlargement. LBBB with electrical axis shifted to the left and QRS fragmentation in inferolateral.

I think of a first rule out Chagasic cardiomyopathy and be negative disease, diabetic cardiomyopathy. Best regards

Martin Ibarrola Buenos Aires MD Centro Cardiovascular Bella Vista, Buenos Aires, Argentina,



Dear Andres,

The second ECG has a gain of 20 mm/mV and the first the usual 10 mm/mV.

The QRS during tachycardia is similar to that recorded in sinus rhythm. Some difference between them may be due to lead position and/or rate-related QRS changes.

My first impression was supraventricular tachycardia in the presence of left bundle branch block or ventricular tachycardia due to bundle branch reentry.

However, the width of the QRS during tachycardia is less than during sinus rhythm (140-160 msec versus 200 msec). The only possibility in this situation is ventricular tachycardia (BBR-VT still possible).

Thank you,

Mario D. Gonzalez, M.D. Professor of Medicine Director, Clinical Electrophysiology Penn State Heart & Vascular Institute Milton S. Hershey Medical Center Penn State University 500 University Drive Hershey, PA 17033 (717) 531-3907 Fax (717) 531-4077 mgonzalez@hmc.psu.edu



Estimados Andrés y Luciano Mi opinión: En principio conociendo el ECG post reversión ((BCRI mas una sobrecarga auricular izquierda) deberíamos pensar primero en como diferenciar una TV con TSV con trastorno de conducción preexistente (en este caso). Teniendo en cuenta el contexto clínico del paciente: edad mayor de 35 años , diabético tipo 2 una taquicardia con QRS ancho con compromiso hemodinámico debería inducirnos hacia una TV, no obstante, si utilizamos los criterios de Wellens QRS con patrón de bloqueo de rama izquierda con QRS < 160 ms. No veo honestamente disociación atrio-ventricular, ni fusión ni captura. Si aplicamos el algoritmo de Vereckei no existe R inicial en aVR El algoritmo de Brugada muestra un rS < 100 ms y el criterio de Pava es negativo para TV (distancia desde el comienzo del QRS al pico de R en DII < 50 ms). Todo esto orienta al diagnóstico de una TSV con bloqueo de rama prexistente

Pregunto a Raimundo: Como explicaría una TV con reentrada rama a rama en presencia de BRI ? Las taquicardias ventriculares rama a rama cursan con frecuencias cardiacas muy rápidas (en torno de 300 latidos por minuto) y ocurren en el contexto casi siempre de una miocardiopatía dilatada o\isquémica. No vemos con frecuencia BCRI en la miocardiopatía chagásica, creo que no es el caso de este paciente En síntesis mi opinión es que se trata de una TSV con trastorno de conducción prexistente pudiendo ser una taquicardia auricular a 240 lpm en un paciente con sobrecarga auricular izquierda consecuencia de una presión diastólica final del ventrículo izquierdo elevada disparó una TSV Abrazos

Juan José Sirena Santiago del Estero Argentina.

Dear Andrés and Luciano: My opinion: In principle post reversal knowing the ECG ((LBBB plus a left atrial overload) should think first about how to differentiate a VT from SVT with preexisting conduction disorder (in this case) Given the clinical context: age over 35 years, type 2 diabetes with wide QRS tachycardia and hemodynamic compromise should lead us to a TV, however, if we use the criteria of Wellens QRS pattern LBBB-like pattern with QRS <160 ms. Honestly, I do not see AV dissociation, (fusion or capture) If we apply the Vereckei 'S algorithm does not exist initial R in aVR Brugada algorithm shows an RS <100 ms. Additionally, Pava's criterion is negative (distance from the . beginning of the QRS to R peak in DII <50 ms). This guides the diagnosis of SVT with preexisting BBB.

I ask to Raimundo: How to explained a BBB-VT reentry in the presence of LBBB pattern?. In BBB-VT the HR is very rapid (around 300 bpm) and occur mostly in the context of non-ischemic cardiomyopathy.

We do not see often in Chagas cardiomyopathy with LBBB, I think is not the case of this patient.

In summary my opinion is that this is an SVT with preexisting conduction disorder that can be an atrial tachycardia at 240 bpm in a patient with atrial left overload consequence of a high end-diastolic left ventricular pressure fired a TSV?

Hugs

Juan José Sirena Santiago del Estero Argentina MD.

Spanish La TV por reentrada rama a rama, como sugirió Raimundo, es muy frecuente en el BCRI y cardiopatía no isquémica. De hecho, es el sustrato mas frecuente, justamente porque propicia las condiciones necesarias para la existencia del circuito. Además, el evento es similares al ECG de base, dado que ambos utilizan parte del His y las ramas, para su existencia. De hecho, las variaciones del hh precediendo al rr son parte del criterio diagnóstico.

Es claramente uno de los diagnósticos diferenciales en este caso.

Saludos AB

Branch to branch reentry-VT, as suggested Raimundo, is very common in LBBB and non-ischemic heart disease. In fact, it is the most common substrate, precisely because it fosters the conditions for the existence of the circuit. In addition, the event has similar pattern to the basic ECG, since both use of the His and branches, for their existence. In fact, variations preceding the RR and HH are part of the diagnostic criteria. It is clearly one of the differential diagnoses in this case.

Greetings

Adrian Baranchuk, M.D. FACC FRCPC

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Final conclusions By Andrés Ricardo Pérez-Riera



Andrés Ricardo Pérez Riera, M.D.Ph.D.

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HR = 187 bpm, QRS duration = 162 ms, QRS axis = -15° is the rule of QRS axis in typical LBBB-like morphology





Analysis of QRS configuration in leads V1 and V6 is a keystone in distinguishing the origin of wide QRS complex tachycardia: diagnostic criteria rely upon the assumption that aberration is due to a functional Bundle Branch Block(BBB), whereas ectopy derives from a totally abnormal activation of the ventricles. Aberration, thus, results in a "typical" BBB morphology, whereas ectopy is expressed by an "atypical" BBB.

Specific criteria, based on analysis of leads V1 and V6, have been developed to distinguish the two conditions from each other. The criteria based on QRS configuration, however, suffer from limitations since unexpected complicating factors, such as a previous myocardial infarction, can result in an "atypical" form of BBB even in the presence of supraventricular tachycardia.

With Complete LBBB-like pattern: rS or QS wave in V1 and V2 with nadir of S wave <70 ms and pure monophasic R wave in V6 suggest SVT-A (**The Griffith algorithm (Griffith 1994): Sensitivity 94.2%, Specificity 39.8%.**)

Typical QRS morphology LBBB-like, because negative QRS on right precordial leads and pure R wave in V6. This pattern suggests SVT-A. In cases of VT, the LBBB is atypical.

Possible causes of Wide Complex QRS Tachycardia

- I. Ventricular Tachycardia VT: >80% of cases. In presence of coronary artery disease: 95%.
- **II.** SVT with aberrancy (SVT-A) right or left aberrancy conduction. Sometimes intermittent (e.g. rate-related aberrancy)

III. SVT with preexcitation (WPW-S): accessory pathway conduction. Preexcited SVT: 1% a 5% cases.

- Atrial fibrillation with Accessory Pathway.
- Atrial fibrillation with multiple accessory Pathways
- Atrial flutter with an Accessory pathway.
- Antidromic Circus Movement Tachycardia (CMT): multiple accessory Pathways in 50% of cases.
- Broad QRS Paroxismal Supraventricular Tachycardia Using Nodoventricular fibers (Mahaim fibers).
- IV. SVT with baseline preexisting BBB. Due to abnormal muscle-to- muscle spread of impulse.
- V. SVT with hyperkalemia other electrolytes disturbances or drugs such as IA and IC antiarrhythmic and tricyclic antidepressants.
- **VI. Ventricular pacing rhythm:** presence of a pacing device on physical examination is a strong clue to pacing as the cause of the WCT. When the pace is in the apex the QRS pattern is LBBB with superior axis.

VII. Hypothermia

The pre-existing Left bundle branch block detection during sinus rhythm with the same morphology of the event suggest supraventricular origin (TSV-A): 1) Atrial tachycardia?; 2) Flutter?; or 3) Sustained bundle branch reentrant (BBR) tachycardia ? In this case, the last possibility 3 is improbable. Why? Because, in the BBR-VT the heart rate is very fast, often > 200bpm, on the other hand, in this case is lesser;. Additionally, patients typically present with syncope or sudden death because of VT with fast rates frequently above 200 bpm. In this case we did not have sensory commitment or hemodynamic compromise. Finally, in BBR-VT there are frequent advanced structural heart disease. On the other hand in this case the heart is near normal ***. We do not have dilated cardiomyopathy, both ischemic and non-ischemic, present in the majority of these cases. Such as in this case the QRS morphology during the event is a typical bundle branch block pattern, usually LBBB, and may be identical to that in sinus rhythm. Prolonged H-V interval in sinus rhythm is found in the majority of patients with BBR-VT, although some patients may have the H-V interval within normal limits. We agree with professor Melvin that the diagnosis of BBR-VT is based on EPS findings and pacing maneuvers that prove participation of the His-Purkinje system in its mechanism. RFCA of a bundle branch can cure BBR-VT and is currently regarded as the first line therapy. The technique of choice is ablation of the right bundle branch. The reported incidence of clinically significant conduction system impairment requiring implantation of a permanent pacemaker varies from 0% to 30%. Long-term outcome depends on the underlying cardiac disease. Patients with poor systolic LVEF are at risk of SCD or death from progressive CHF despite successful BBR VT ablation and should be considered for an ICD.

*** In the present case, transthoracic echo showed inferiorseptal hypokinesis. However, coronary angiography showed no significant coronary obstructions. The specific echo abnormality demonstrated in LBBB is a very dynamic posterior motion of the interventricular septum occurring within 0.04 seconds of the onset of the QRS and preceding the anterior motion of the posterior LV wall during ventricular ejection. This type of septal motion is not seen in the other forms of abnormal septal motion and appears to be specific for LBBB. The abnormal septal motion found in these patients with LBBB is interesting from several points of view. First of all, the pattern of the early systolic motion seems to be specific for this abnormality since it was not found in other patients with abnormal septal motion. Thus paradoxical septal motion due to LBBB probably can be distinguished from patients with right ventricular volume overload and from patients with CAD. This finding also supports the concept that the pattern of electrical depolarization will influence the mechanical aspects of ventricular systole. In LBBB the interventricular septum has been found to depolarize from right to left rather than the usual left to right depolarization that is found in both normal patients and those with RBBB. The fact that the interventricular septum depolarizes from the opposite direction with LBBB is a very likely explanation for the peculiar early systolic motion seen in the interventricular septum (Dillon 1974).



Our dear friend Dr. Raimundo Barbosa Barros observed fusion beats in aVF lead. I am not sure about this diagnosis because QRS modifications are minimal, non anticipated and could be consequence of respiratory movement. A Dressler beat is a beat that occurs during VT and is also known as a fusion beat. This occurs when sinus node activity (P wave) or supraventricular begins to conduct through the normal conduction pathway during an episode of VT. The fusion beat result from activation via two different pathways: the normal His-Purkinje system and the ventricular reentrant circuit. There is a P wave before the QRS complex. The resulting QRS is a "hybrid" beat an intermediate complex between the pure sinus beat and the pure ectopic beat and that of the ventricular morphology from the VT. Fusion beat interrupts the VT producing an hybrid complex result of the fusion of two impulse from the VT and the supraventricular focus one impulse from the VT and the other from a supraventricular focus. Dressler beats strongly support the diagnosis of ventricular tachycardia by interruption of it.

In bellow strip we show a VT with fusion and capture beats. The heart rate during the fusion and capture beats occur at lower heart rate related the 7 first beat, which is not observed in the upper strip of the present case.





Our dear friend Dr. Adail Paixão Almeida observed also dissociation phenomena in V2-V3. Sincerely I do not see fusion or capture beats. Additionaly, in the present case we do not observe the Brugada($RS \ge 100ms$) and/or Josephosn's signs(notched on descending ramp of S wave.) In this case there are many doubts therefore becomes necessary to apply existing algorithms in the literature for the differential diagnosis of wide complex QRS tachycardia. The differential diagnosis of wide QRS tachycardias has important short and long term implications in the therapeutic management and prognosis. The ECG is still the primary tool to establish a specific diagnosis. In spite of proper use of various diagnostic algorithms, in approximately 10% of cases the diagnosis is not made. In cases of uncertainty, an acute therapeutic approach is advisable focused on the diagnosis and treatment of VT, since most wide QRS tachycardias have a ventricular origin. Appropriate referral for electrophysiology studies can then be used to establish the mechanism of the arrhythmia and make relevant treatment decisions.

Main algorithms for differential diagnosis of the Wide Complex QRS Tachycardia

- I. The Brugada algorithm (Brugada P 1991)
- II. The Griffith algorithm (Griffith 1994)
- III. The ACC algorithm (Blomström-Lundqvist C 2003)
- IV. The Vereckei algorithm (Vereckei 2007)
- V. The New Vereckei's algorithm (Vereckei 2008)
- VI. The Pava algorithm or ultrasimple Brugada criterion: RW to peak Time (RWPT) (Pava 2010)
- VII. The Miller algorithm practical approach (Miller 2009)

VIII. The new easy criteria using the bipolar I-II and the precordial unipolar V1 and V6 or "Rodrigues dos Santos Neto-

Scanavacca algorithm" (data not yet published)

I. The Brugada algorithm (Brugada P 1991)

This is the most commonly used algorithm having a potential sensitivity (Se) of 99%; and specificity (Sp) of 97%.

Brugada et al proposed 4 sequential criteria for the differential diagnosis of wide QRS tachycardia. (see next slide):

- 1. Absence of complexes of the RS type in all precordial leads;
- 2. R to S interval in any precordial lead with RS complex >100 ms;
- 3. AV dissociation;
- 4. Morphological criteria present in both V1-2 and V6 (Table 1).

Using these 4 steps the authors reported up to a sensitivity of 99% and specificity of 97% based on an analysis of 554 tachycardias. This approach

can significantly reduce the number of mistaken diagnoses.



First step of the Brugada algorithm



Wide QRS Tachycardia showing absence of complexes of the RS type in the precordial leads (first step of Brugada algorithm).

Second step of the Brugada algorithm: R to S interval \geq 100 ms



Absence of Brugada signal: distance from the beginning of QRS to nadir of S wave ≥ 100 ms. In this case, < 80 ms. This is the criteria that suggest SVT-A. An interval > 100 ms from the beginning of the QRS complex to the nadir of S wave in any precordial lead is diagnosis of VT.

Third step of the Brugada algorithm: the presence of dissociation



F = Fusion beat: occurs when a supraventricular and a ventricular impulse coincide to produce a hybrid complex. The fusion beats are of intermediate width and morphology to the supraventricular and ventricular complexes.

C = Capture beat: occur when the sinoatrial node transiently 'captures' the ventricles, in the midst of AV dissociation, to produce a QRS complex of normal duration.

Fourth step of the Brugada algorithm: morphology in V1 and V6



Brugada algorithm limitations

In spite of the innovation and significance of the Brugada algorithm the following limitations have been noted:

- I. Patients using antiarrhythmic drugs were not included
- II. The authors did not comment if patients with previous BBB, idiopathic VT and pre-excited tachycardia were included in the group studied
- III. Brugada mentions that its algorithm presents a greater sensitivity and specificity than other traditional criteria, but did not compare the diagnostic accuracy of the algorithm with other reported criteria
- IV. The 4th step of the algorithm keeps the morphological criteria, which are hard to apply in clinical practice
- V. In addition other authors found lower rates of sensitivity and specificity (Miller 2004; Drew 1995; Alberca 1997) than those initially reported by Brugada.

II. The Griffith algorithm: Sensitivity 94.2%, Specificity 39.8%.

The algorithm of Griffith (Griffith 1994) reverses the diagnostic strategy: supraventricular tachycardia (SVT-A) is diagnosed when the ECG findings correspond to typical left or right bundle branch block:

• With Complete LBBB-like pattern: rS or QS wave in V1 and V2 with nadir of S wave <70 ms and pure monophasic R wave in V6.





• CRBBB: triphasic pattern RSR' in V1 and RS in V6, with height of R wave greater than S wave depth.

III. The ACC algorithm (Blomström-Lundqvist C 2003)



IV. The Vereckei algorithm (Vereckei 2007): Sensitivity: 87.1% and Specificity: 48%.



During SVT-A with BBB the initial ventricular activation sequence moves away from lead aVR resulting in an initial negative QRS complex in aVR (Vereckei 2008). In the population studied by Vereckei, this algorithm had a better accuracy than the Brugada algorithm (Vereckei 2007) likely due to a low accuracy of the 4th Brugada criterion when compared to the 4th criterion of Vereckei. The Vereckei algorithm has some of the same limitations found in the Brugada approach; i.e., it is unable to differentiate certain wide QRS tachycardias uncluding VT using branch to branch reentry, fascicular VT, and SVT using antegrade accessory pathways, unless AV dissociation is present during VT (Vereckei 2007). Another limitation is due to the fact that an initial R wave in aVR may also be seen in patients with left anterior fascicular block and myocardial infarction (**Dendi 2007**). Moreover, the v_i/v_t ratio could be altered in other conditions such as anteroseptal myocardial infarction, fascicular VT, and VT near the His-Purkinje system (Vereckei 2008).

V. The new Vereckei algorithm using only lead aVR for differential diagnosis of Wide QRS Complex Tachycardia

In 2008, Vereckei et al (Vereckei 2008) published a new algorithm that was based on the direction and velocity of the initial and terminal portions of ventricular activation (Fig. 27). In spite of not having any new fundamental criterion, it is based on three new concepts:

- I. The exclusive analysis of a single lead (aVR) for the differential diagnosis of wide QRS tachycardia;
- II. VT is classified in two main groups:
 - I. VT with origin in the inferior and apical regions of the ventricle having an initial R wave in aVR;
 - II. VT with origin in other regions without an initial R wave in aVR but with slow velocity of the initial phase of QRS in contrast with SVT that has a rapid initial velocity
- III. Removal of AV dissociation and morphological criteria used in previous algorithms and traditional criteria.



Vereckei, A et al Heart Rhythm 5:89-98, 2008

The accuracy of the new Vereckei algorithm was superior to the Brugada algorithm not by showing a statistically significant difference compared to its earlier version, but having a greater sensitivity and specificity in the diagnosis of SVT compared to the Brugada criteria. This advantage in relation to Brugada criteria is due to the superiority of two criteria - initial R wave in aVR and v_i/v_t ratio. Another advantage is practicality, since the new algorithm by Vereckei was faster than the previous Brugada algorithm.





The limitation of both the Vereckei algorithms and Brugada's, is the inability to differentiate pre-excited SVT from VT, except when there is an initial R wave in aVR. In fact in 20 cases of pre-excited SVT none presented an initial R wave. Another limitation of this algorithm is the small number of patients selected with VT without structural heart disease.

Algorithm proposed by Vereckei to differentiate wide QRS tachycardia based on the aVR lead



The criterion of AV dissociation, in spite of 100% specificity, is not very sensitive because identifying dissociated atrial activity in fast wide QRS tachycardia is difficult.

In the study by Vereckei the finding of AV dissociation did not affect the accuracy of the test when compared to the four-step algorithm.

The newer Vereckei algorithm also has of a sequence of four steps but only uses a single lead (aVR) for the analysis The Vereckei aVR algorithm is also based on four steps:

- If AV dissociation is present, the diagnosis of VT is made and the analysis is over;
- If there is an initial R in aVR, the diagnosis of VT is made; if not the following next step is made;
- If the QRS morphology of the tachycardia does not correspond to typical morphology of BBB or fascicular blocks, the diagnosis of VT is made and the analysis is stopped;
- Finally, when the ratio between initial activation velocity and the terminal activation velocity (v_i/v_t) is ≤ 1 , the diagnosis of VT is made and in the case of a v_i/v_t ratio being >1, the diagnosis of SVT-A is made.

This new algorithm presented two new concepts, in relation to those existing previously:

v_i/v_t ratio

During wide QRS tachycardia of supraventricular origin (SVT-A), the initial activation of ventricular muscle should be rapid through the Purkinge network but with QRS widening and conduction delay occurring in the mid-to-terminal portions of the QRS. Thus, during SVT-A or fixed RBBB, the conduction velocity of initial ventricular activation should be faster than the terminal ventricular activation. On the contrary, during VT there is initial slow ventricular activation until the impulse reaches the His-Purkinje system, after which the rest of the ventricle will be quickly activated (Vereckei 2007).

The v_i/v_t ratio is measured as the voltage variation in the ECG tracing during the initial 40 ms (Vi) and the terminal 40 ms (Vt) of the same QRS complex. A ratio ≤ 1 suggests a VT and a value >1 suggests supraventricular focus.



VI. The Miller practical approach in the diagnosis of Wide QRS Tachycardia (Miller 2009)

This algorithm uses of the following six successive steps proposed by Miller

- First step: Determine the atrioventricular ratio. In the presence of AV dissociation, the diagnosis is VT. If not, got to second step.
- Second step: QRS axis in the FP in the right superior quadrant (northwest quadrant axis). When present, it indicates VT. When absent, go to third step.
- **Third step:** Vi/Vt ratio when > than 1, SVT-A is diagnosed; if not, continue to the fourth Step.
- Fourth step: Absence of RS pattern in the precordial leads indicates VT. If not, go to the fifth Step.
- **Fifth step:** RS interval in the precordial leads >100 ms indicates VT. If not, continue to sixth Step.
- Sixth step: in the case of a tachycardia with LBBB-like morphology, an initial r < 30 ms or an interval from QRS onset to the nadir of S in V1 <60 ms indicates SVT-A.

VII. The Pava criterion, ultra simple Brugada criterion or RW to peak Time (RWPT) (Pava 2010)

This criterion uses only lead II. This lead was chosen because it is easily obtained and by the fact of being present in most ECG strips made in the ER.



R-Wave Peak Time (RWPT). In this case RWPT = 85 ms

RWPT \geq 50 ms = VT

The rationale of this criterion is based on the slower initial conduction velocity in the ventricular muscle tissue, compared to the much faster His-Purkinje conduction system and, as such, differentiating a ventricular focus from a supraventricular origin. The application of this criterion is made by measuring the time interval from the QRS onset to the apex of the R wave; this corresponds to the so-called ventricular activation time, R peak time or intrinsicoid deflection regardless of QRS complex polarity.

In this study a value \geq 50 ms in lead II had a sensitivity of 93%, specificity of 99%, a positive predictive value of 98% in the diagnosis of VT. However, the study did not compare this new criterion with those existing previously and requires future validation.



In Pava criterion, when the QRS complex is negative in II (type rS or QS) should perform the measurement from the beginning of QRS complex until the nadir of the S QS wave, such us the present example. In the present case the R-Wave Peak Time (RWPT) is > 50ms = VT

VIII. The new easy criteria using the bipolar I-II and the precordial unipolar V1 and V6

Observation: I propose the eponym *Rodrigues dos Santos Neto-Scanavacca algorithm*, because these researches were the first to validate this simple algorithm. This criteria showed good accuracy in the differential diagnose of wide QRS tachycardias, and can be used as an alternative to Brugada algorithm by non expert physisians.(data not yet published)

First step - The 4 leads (I, II, V1 and V6) have predominantly negative polarity? If yes: VT; If no, go to the second step.



First step - The 4 leads (I, II, V1 and V6) have predominantly negative polarity? If yes: VT; If no, go to the second step



Second step – At least three of the four leads have predominantly negative polarity on their QRS? If yes: VT; If no, go to the third step. The answer is No.

Third step - At least two of the four leads have predominantly negative polarity on their QRS (including I or V6)? If yes: VT; If no, SVT-A. The answer is yes.

Conclusion: This new algorithm shows to us that this is a VT.

This algorithm has great advantage over other ones because the physician does not need to be an expert on Electrocardiology, and make measurements, because it consists only in observing if the QRS complexes are predominantly negative in bipolar I and II and precordial unipolar V1 and V6. Using this algorithm does not need memorizing values like the other ones. Dr. Francisco Rodrigues dos Santos Neto and Maurício Ibrahim Scanavacca evaluated 120 VT or SVT-A ECGs using two assessment methods: the Brugada and the present algorithm. The authors concluded that the new ECG algorithm showed good accuracy in the differential diagnosis of wide QRS tachycardia and can be used by non-expert physicians.

Summary of the main differences between VT and SVT-A

•		
	VT	SVT-A
Focus and etiologies	Bundle branches, Purkinje or ventricular muscle. The causes of VT may be with or without structural heart disease.	Atria and/or AV junction
Presence of cannon A waves in the jugular venous pulse	When present, it is diagnostic	No
Beat by beat variations in the intensity of the first heart sound,	Characteristic	No
Beat by beat variations of systolic blood pressure,	Characteristic	No
Focus	 Reciprocal supraventricular tachycardia. Reciprocal supraventricular tachycardia conducted by accessory bundle: WPW. Atrial flutter conducted by accessory pathway. 	It originates in the His bundle branches, in the Purkinje network or in the ventricular myocardial contractile cells.
History of MI, angina, CHF, cardiomyopathy, correction of congenital heart disease, family history of SCD: suggestive of HCM, ARVD/C, LQTS, SQTS, IFV, BrS, J wave syndromes	Strongly suggestive	No
History of paroxysmal tachycardia responsive to vagal maneuvers or adenosine.	No	Characteristic
Previous ECGs with short PR interval (<120 ms), wide QRS and delta wave.	No	It indicates pre-excitation as cause.

	VT	SVT-A
Previous ECG with bundle branch block	No	Characteristic
pattern identical to the pattern of the event		
End of event with vagal maneuvers or	Rare	Yes.
adenosine		
QRS duration	>140 ms if RBBB pattern; >160 ms of LBBB	RBBB-Like pattern < 140ms
	pattern	LBBB-Like pattern < 160 ms
SÂQRS in the FP	Suggestive when SÂQRS is in the northwest $auadrant$ between -90° and $\pm 180^{\circ}$	No
QRS Pattern in V1	In the presence of LBBB pattern, initial $r >40 \text{ ms}$	Initial narrow r, and clean s, with no notches if
	and rS interval greater than 70 ms is suggestive.	LBBB and triphasic pattern if RBBB
	Biphasic or monophasic pattern if RBBB. When biphasic in $V1 P' > P$ (rabbit car sign) (Figure in	
	nevt slide) (Cozonsky 1974)	
ORS Pattern in V6	rS Ors OS OR or monophasic R	aRs Rs or RS with R>S
	If the pattern was RS R <s.< th=""><th></th></s.<>	
The distance from the onset of QRS up to the nadir of S >100 ms (Brugada sign)	If present, it is diagnostic.	Lower.
Notch near the nadir of the S wave (sign of	Characteristic	Absent.
Josephson)		
QRS complexes of the R or QS type on	Diagnostic	No
precordial leads		
Initial q or r wave with duration >40 ms in	Diagnostic	No
aVR (qR or rS)		
Pattorn matching in procordial loads	Strong also as a active	No



LBBB-like pattern with QRS axis in the frontal plane located on right upper quadrant, northwest quadrant or "no man's land"

Frontal -90* ± 180° -X 0'I III *+90* aVF

Clinical causes of QRS axis between -90° and $\pm 180^{\circ}$:

- Emphysema
- Hyperkalemia
- Poisoning with tricyclic antidepressant
- Accidental misplacement of the limb lead electrodes
- Artificial pacemaker
- Divisional, right bundle branch block, right end conduction delay, right parietal blocks , *divisional*, *focal*, *partial*, *Purkinje*, and *zonal* right ventricular blocks.
- Ventricular ectopic beat
- Ostium atrioventricularis comunis
- Ventricular tachycardia

QRS complexes of the R or QS type on precordial leads: positive or negative concordance



Clinical diagnosis: patient with antecedent of anterior myocardial infarction.

Negative concordance: sustained monomorphic wide QRS tachycardia , HR = 167 bpm. All precordial leads are totally negative (QS) without RS complex. It is noted extreme left axis deviation (-80°). Atypical pattern of LBBB: Suggestive VT.

Note: The presence of negative concordance, either positive or negative, has 90% of specificity for VT. Unfortunately, the sensitivity is only 20%: 10% for negative and 10% for positive concordance.

Positive concordance: VT



Positive concordance: SVT with left accessory pathway (WPW)



Our conclusions

In the present case it is not possible to determinate if this patient had VT or SVT-A. It is absolutely necessary the electrophysiological study. This methodology is considered the gold standard for the diagnosis and treatment.

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