Sustained wide QRS complex tachycardia: A challenge in the emergency room

Case Presentation: A 56-year-old woman with known coronary artery disease (CAD) and past history of myocardial infarction presented to the emergency department with palpitations followed by an episode of syncope.

The initial electrocardiogram (ECG), illustrated in Figure 1, revealed a sustained wide QRS tachycardia (heart rate ≥100 bpm and QRS duration ≥120 ms) with a pattern of monophasic right bundle branch block (RBBB). The ECG immediately after electrical cardioversion, illustrated in Figure 2, revealed sinus rhythm with a probable antero-apical infarct scar, residual ST segment elevation, and low voltage QRS fragmentation in leads V4-V5, suggesting a left ventricular (LV) aneurysm. A previous ECG taken one year earlier (Figure 3) showed a similar pattern.

An echocardiogram confirmed the presence of antero-apical LV aneurysm with fibrosis and apical dyskinesia (Figure 4). A previous cardiac catheterization showed a complete left anterior descending (LAD) occlusion in the absence of reperfusion therapy.
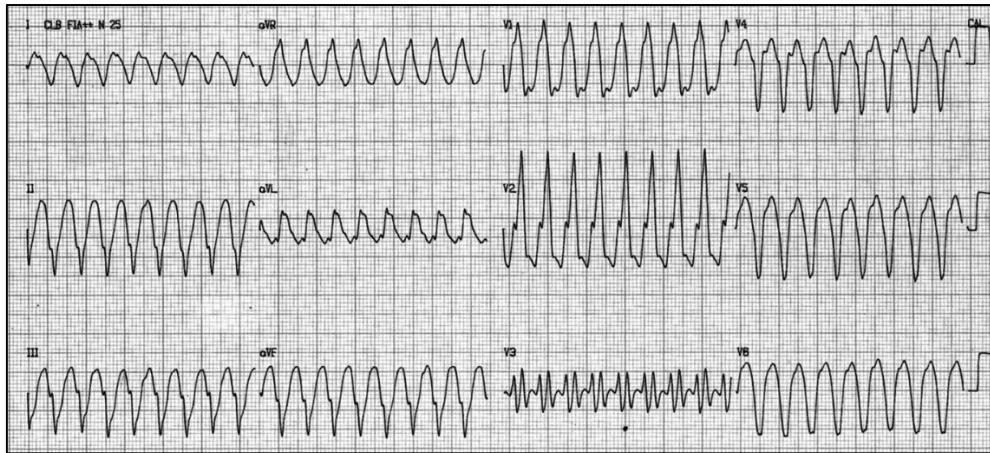
Figure 1 - *Sustained wide QRS tachycardia; heart rate of 214 bpm, RBBB pattern with monophasic R in V1; predominantly negative QRS complexes in the inferior and lateral precordial leads, and pure R in aVR and aVL. This QRS morphology suggests ventricular tachycardia (VT) with a probable focus in the apical region of the left ventricle.*
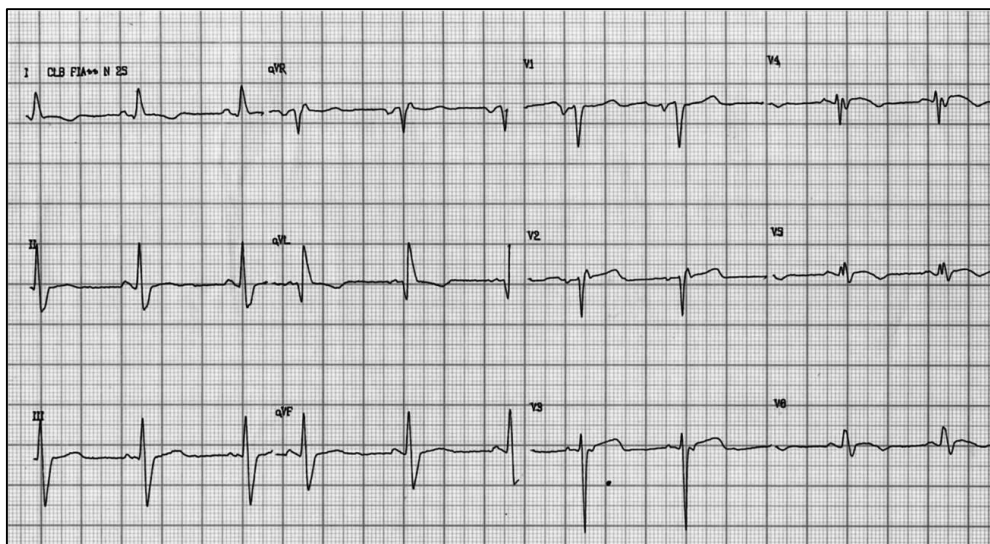

Figure 2 - *ECG after cardioversion showing sinus rhythm; SÂQRS -40°, qR pattern in I and aVL, SIII >SII suggesting left anterior fascicular block. Slight ST elevation is present in aVL and I; old electrically inactive antero-apical area is manifested by low voltage QRS in V4-V6) and fragmented QRS (f-QRS) in V4-V5. f-QRS is defined as additional notches within the QRS complex. In patients with CAD f-QRS is associated with myocardial scar that can be detected with single-photon emission computed tomography. f-QRS is a predictor of mortality and arrhythmic events in patients with reduced left ventricular function.*
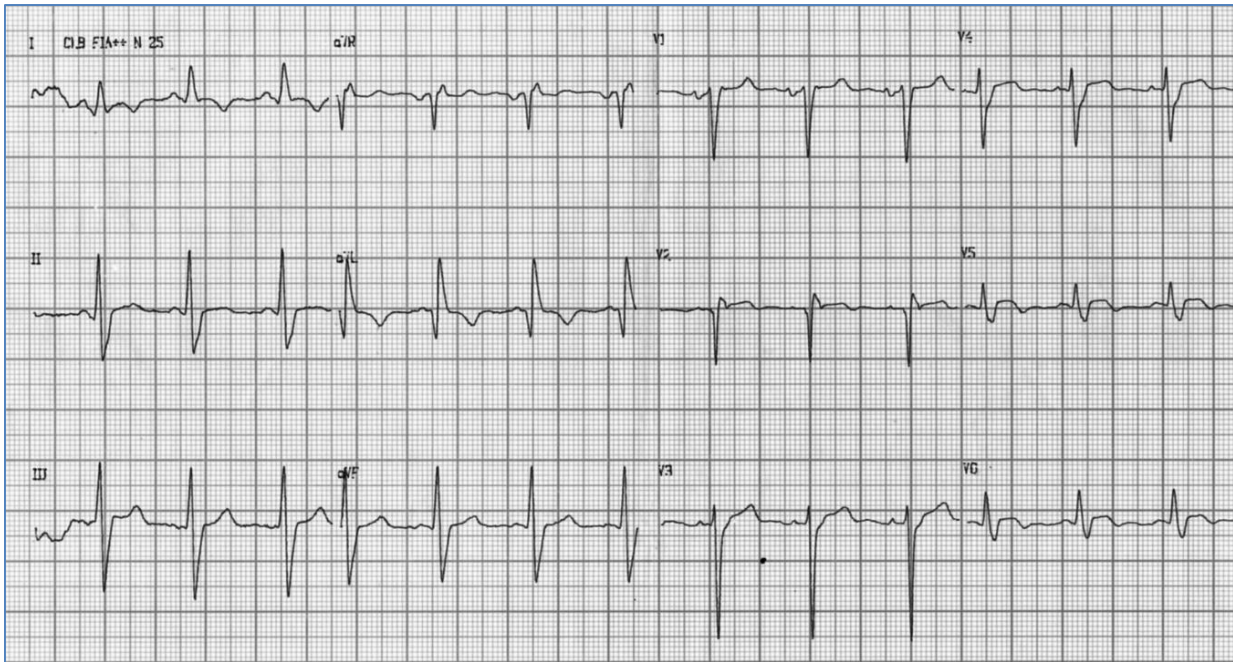
Figure 3 - ECG from the same patient made one year ago, displaying a similar pattern to the current alterations in Figure 2.
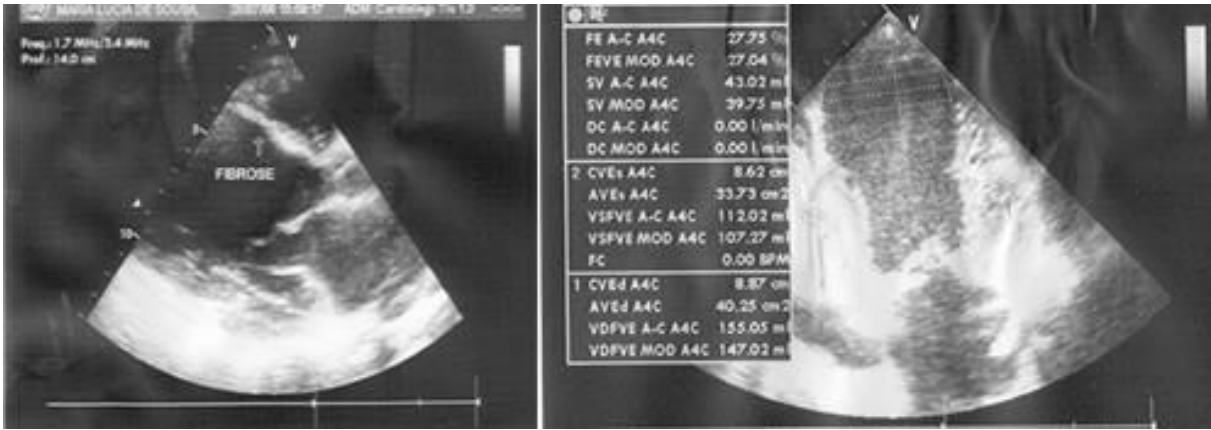

Figure 4 - Echocardiogram showing anteroapical LV aneurysm.

Case Discussion

The diagnosis of sustained wide QRS tachycardia (defined by heart rate ≥100 bpm, and QRSd ≥120 ms) remains a challenge. The differentiation between supraventricular tachycardia with aberration (SVT-A) and ventricular tachycardia (VT) has important therapeutic and prognostic implications.

Conceptual definitions

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

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.

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. SVT-A due to bundle branch block (BBB) (15% to 30% of the cases).

        i. Pre-existing or fixed BBB.

        ii. Functional or tachycardia-dependent BBB.

    b. SVT using an accessory preexcitation pathway for antegrade 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 atrioventricular (AV) junction resulting in a wide QRS complex tachycardia. This SVT entity is known as AV reentrant tachycardia (AVRT).

    c. SVT with nonspecific intraventricular (IV) conduction delays caused by drugs, electrolyte abnormalities or hypothermia that result in QRS prolongation.

    d. VT (1): 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.

    e. Paced ventricular rhythms at tachycardia rates.

II)   Markedly or grossly irregular wide QRS tachycardia.

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

b. AF with rapid heart rate response and typical BBB.

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

d. Atrial flutter with BBB.

Important clues from the history and physical examination

History: VT is the most common cause of wide QRS tachycardias, consisting of 80% of all cases (2). A history of previous myocardial infarction, angina, or congestive heart failure has a high positive predictive value of 95% (3) for the diagnosis of VT.

Physical examination: The ECG presence of AV dissociation is highly predictive of the diagnosis of ventricular tachycardia. The following physical findings are suggestive of AV dissociation:

I) Irregular large 'A' waves in the jugular venous pulse, called 'cannon A waves', indicate atrial contractions occurring at times when the AV valves are closed due to the dissociated ventricular contractions (4).

II) Variations in the intensity of the first heart sound (S1). Since S1 is due to the closing of AV valves, in the presence of AV dissociation the position of the valve leaflets varies on a beat by beat basis causing variations in the intensity of S1. Three other ECG conditions including third-degree AV block, second-degree AV block (Type I or Wenckebach), and AF, can also vary the intensity of S1.

III) Beat by beat variations in systolic blood pressure unrelated to breathing caused by variable LV filling and resulting in changing the appearance of Korotkoff sounds. These modifications occur because the time lapse between atrial and ventricular contraction is different with each cycle, resulting in variable ventricular filling.

The reversion of WQRST with vagal maneuvers or drugs like adenosine strongly suggests the diagnosis of SVT-A, although fascicular VT may also convert with these interventions. The criterion of hemodynamic stability is not useful in the differential diagnosis of WQRST since functional cardiac reserve is the main determinant of hemodynamic stability during the tachycardia. In a doubtful case, IV verapamil, beta blocker or digoxin should not be used since they may cause hypotension or cardiac arrest especially when heart rates are above 200 bpm. When the diagnosis is uncertain in a hemodynamically stable patient procainamide is useful in non-ischemic WQRST, and this drug also reduces the conduction velocity in accessory pathways in patients with antidromic AVRT.

From the hemodynamic point of view, wide QRS tachycardia are grouped as stable or unstable. Important indicators of instability include: hypotension, syncope, precordial chest discomfort, acute heart failure

Patients exhibiting one or more indicators of instability should undergo immediate synchronized cardioversion starting with an energy level of 100 J (monophasic) or 70 J (biphasic) with increasing energy levels as necessary.

Electrocardiographic criteria for the differential diagnosis of wide QRS complex tachycardia

A 12-lead ECG is the primary tool for differentiating the origin of a WQRST. Since 1978 most criteria are based on the presence or absence of AV dissociation, QRS duration and morphology, and frontal plane QRS axis.

Importance of atrioventricular dissociation

P waves may be difficult to detect during a WQRST. Nevertheless, in the presence of slower runs of VT, the detection of dissociated P waves, if present, confirms the ventricular origin of the

tachycardia (Figure 5). In addition, the presence of capture and/or fusion beats is also indicative of AV dissociation and VT. Capture beats are recognized by narrow QRS complexes preceded by P waves similar to those in sinus rhythm. Fusion beats (aka Dressler beats) are hybrids with intermediate QRS morphology between the pure sinus beats and beats of ventricular origin. The fusion indicates the merger of two wave fronts, one from the ventricular focus and one from the activation of the sinus beat (Figure 6). AV dissociation has a specificity of virtually 100% for the diagnosis of VT; however, sensitivity is low, ranging from 20 to 50% because many cases of ventricular have retrograde atrial capture or 'VA' association (5-7). Rarely VA association may have a Wenckebach type of second-degree retrograde block rather than 1:1 VA conduction. It is also important to remember that the fusion beats are not pathognomonic of VT since it may also be seen in:

- AF in the setting of accessory pathways. In this setting fusions and captures are due to the presence of two or more antegrade pathways into the ventricles.
- End-diastolic premature ventricular contractions.
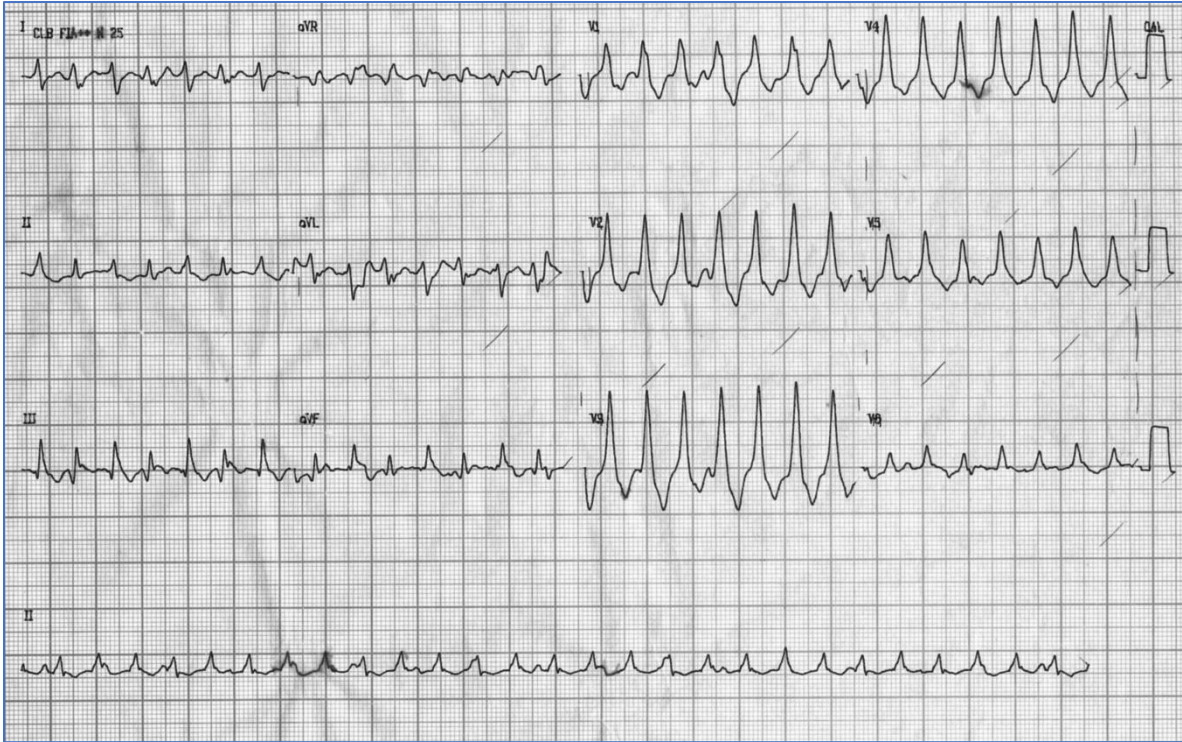- Ventricular parasystole.

Figure 5 - *Wide QRS tachycardia clearly showing the presence of AV dissociation, mainly in leads V1-V3 and the rhythm lead II.*
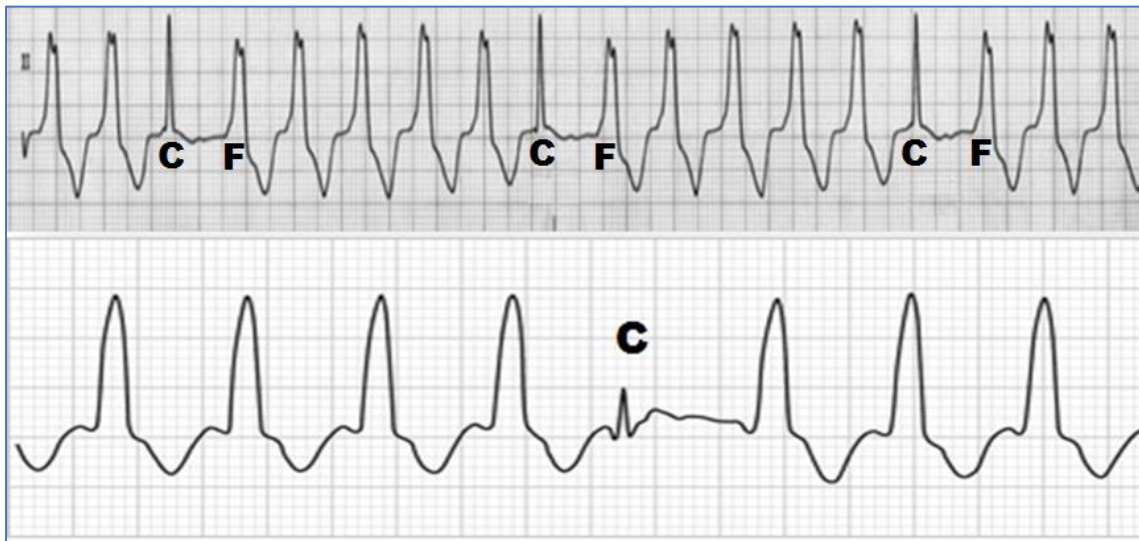

Figure 6 - *In the upper tracing there is wide QRS tachycardia with fusions (F) and captures (C). Note that the fusion beat occurs after the capture beat. The lower tracing shows a single beat of capture corresponding to the fifth beat.*

QRS complex duration

In general, QRS durations tends to be greater in VT than in SVT-A. A QRS duration >140 ms with RBBB pattern or >160 ms with left bundle branch block (LBBB) pattern suggests VT (8). VTs originating in or near the ventricular septum, however, may present with shorter QRS durations. On the other hand, SVT may have QRS durations >140 ms with RBBB and >160 ms with LBBB in the following situations: (i) previous BBB in elderly patients with fibrosis of the conduction system and ventricular myocardium; (ii) AV conduction through an accessory pathway; (iii) during use of class IC antiarrhythmic agents (flecainide, propafenone) (1).

Axis of QRS complex in the frontal plane

A QRS axis in the right upper quadrant between -90° and ±180° strongly indicates VT (northwest axis). When VT originates in the apical region of the ventricles, the direction of ventricular depolarization is upwards or superior resulting in negative QRS complexes in the inferior leads. When VT originates in the basal region of the ventricles, QRS direction is downwards or inferior with positive QRS complexes in the inferior leads (8). The presence of an inferior axis in a WQRST with a LBBB pattern suggests VT originating the right ventricular outflow tract (RVOT) (8) (Figure 7).
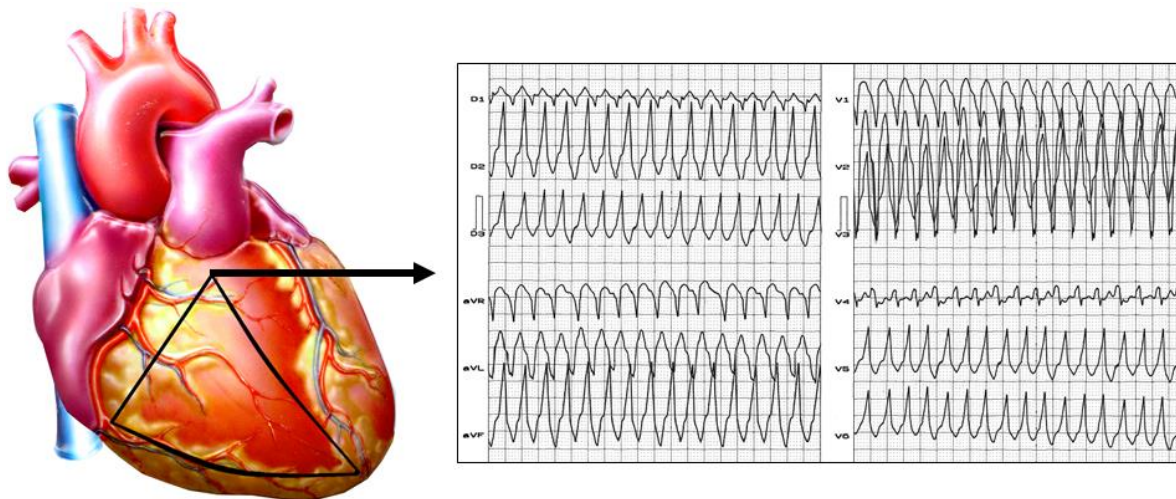
Figure 7 - *Sustained monomorphic ventricular tachycardia (SMVT) with LBBB pattern and inferior frontal plane axis; note the positive QRS complexes in the inferior leads and the negative QRS in aVL and aVR. In this case, SÂQRS is located to the right of +90°, between +90° and +120°, indicating that the origin is in the RVOT. In such cases the QRS has a QS morphology in lead I.*

Morphology of the QRS complex

One of the most important features in the differential diagnosis of WQRST is the morphology of the QRS in various ECG leads. The following are important clues to the correct diagnosis.

A combination of a LBBB-like pattern with the SÂQRS shifted to the right suggests a ventricular origin or VT. The presence of a LBBB-like morphology with negative QRS complexes in lead I is found in two-thirds of cases of VT originating in the right ventricle.

The presence of an initial R wave in aVR is highly suggestive of VT (9).

The presence of monophasic or biphasic QRS complexes (R, qR, QR or RS) in the right precordial leads in wide QRS tachycardias with a RBBB pattern favor the diagnosis of VT. In the cases of biphasic or notched upright QRS complexes in V1 with the initial R wave > the subsequent peak or R' wave strongly indicates VT (rabbit ear sign) (10, 11). The V6 lead also provides significant information during the tachycardia with a RBBB pattern; complexes of the Rs, QS, qR type or an R:S ratio <1, indicates ventricular origin (8) (Figure 8).
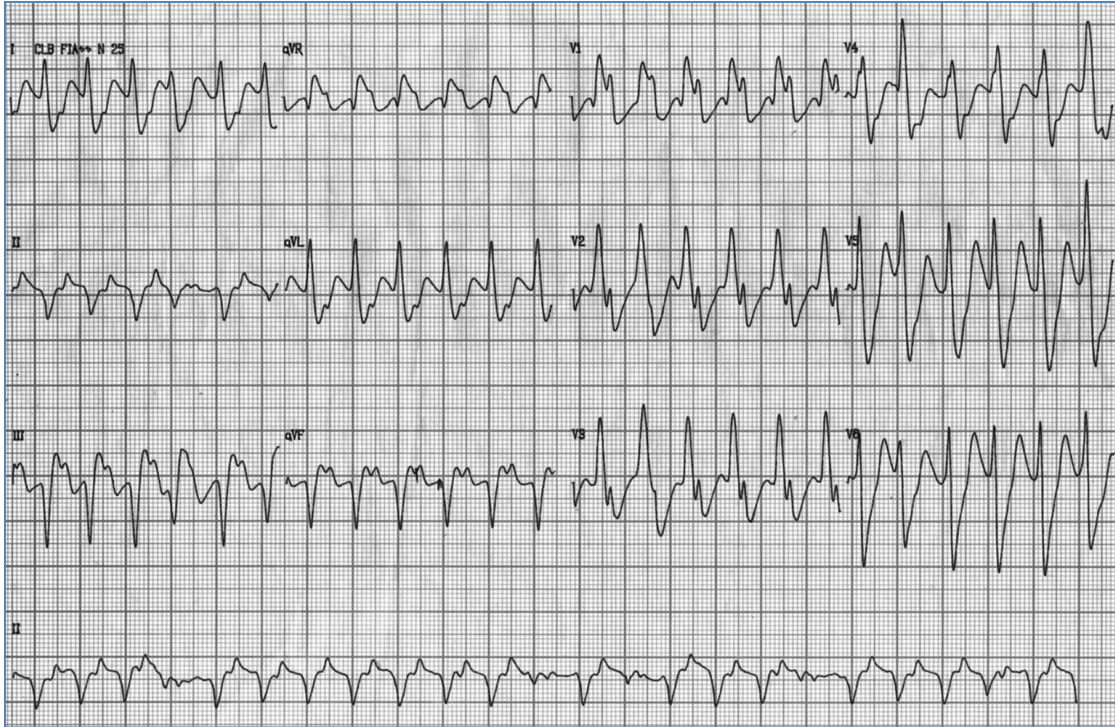
Figure 8 - *Wide QRS tachycardia with a RBBB pattern; note initial R wave > R' wave in V1, R/S ratio in V6 <1, and presence of hybrid fusion beats in the long lead II rhythm strip (all these are findings that suggest VT).*

Figure 9 illustrates two cases of WQRST with a RBBB pattern in leads V1-V3. Important 'rabbit-ear' features that differentiate ventricular tachycardia from SVT with RBBB aberrancy are shown. In the cases of wide QRS tachycardia with a LBBB pattern, the following features suggest VT (Figure 10):

✓ Initial r wave in V1 ≥40 ms (present in 87.7% of the cases of VT, and only 13.3% of the cases of SVT-A). This is sometimes called the 'fat little r-wave' sign.

✓ Notch or slurring in the descending slope of S wave in V1-V2. A notch close to the S wave nadir is called the Josephson sign.

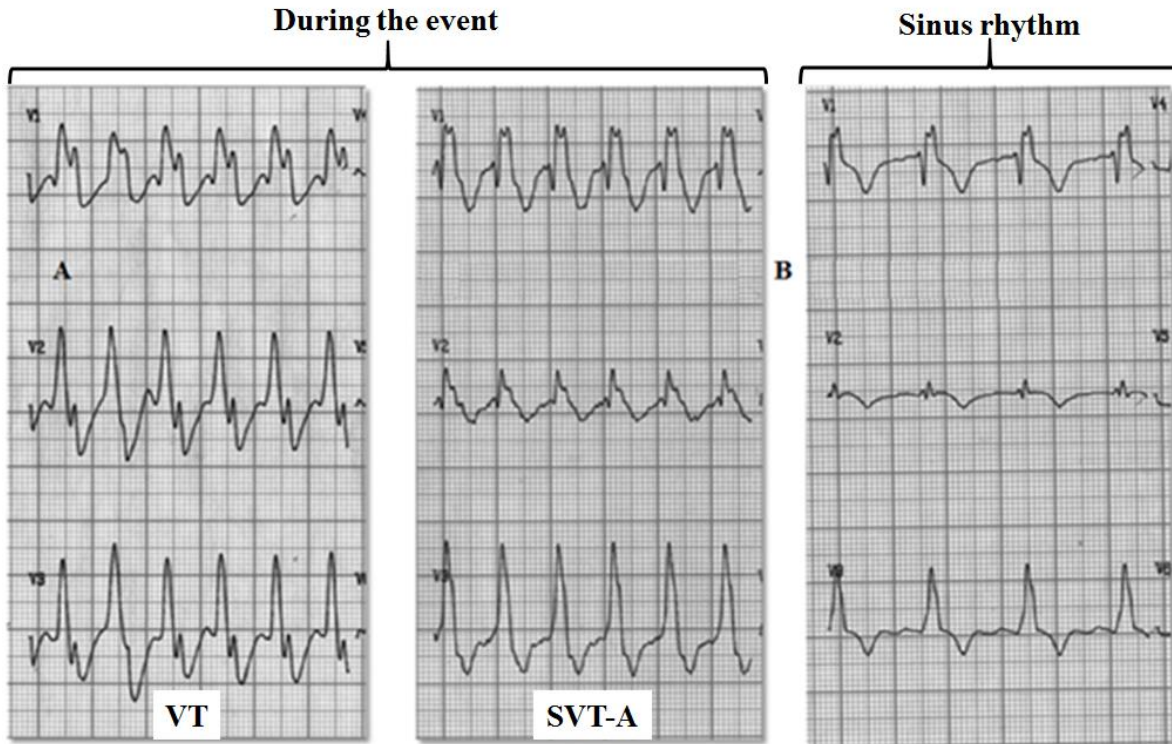✓ A delay from the onset of QRS until the S wave nadir ≥70 ms (12).

✓ Any Q wave in V6.

Figure 9 - *Two cases of wide QRS tachycardia with RBBB pattern in leads V1-V3. A) VT with R>R' in V1; B) (left panel) SVT with classic RBBB pattern; (right panel) same patient in sinus rhythm; note the rSR' pattern with R'>R.*
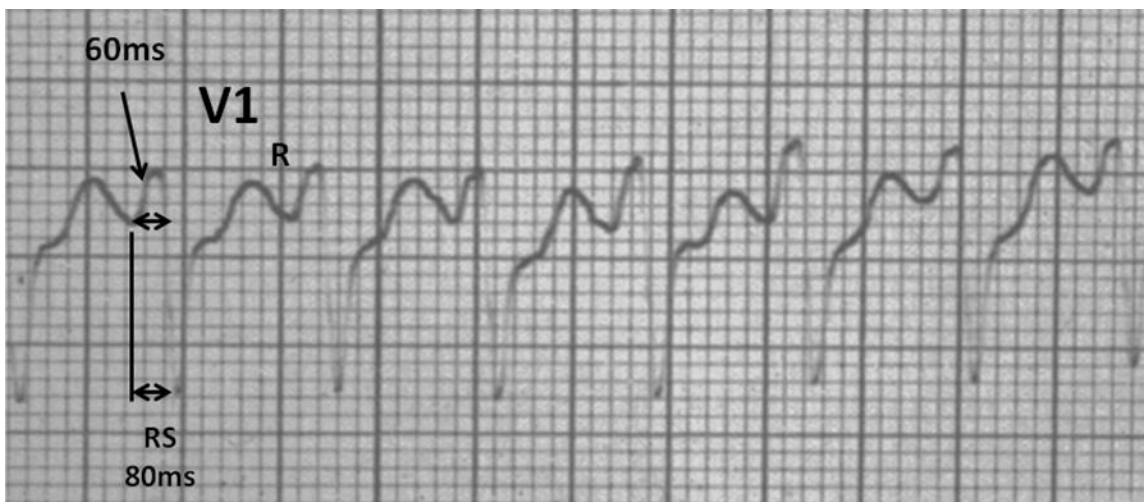

Figure 10 - *A V1 rhythm of wide QRS tachycardia with a LBBB pattern where a fat initial r wave is observed with duration >40 ms; note that the interval from QRS onset to S wave nadir is >60 ms. Both features strongly suggest VT.*

During SVT-A with a LBBB pattern lead V1 has no initial r-wave or a 'thin' r-wave of small amplitude, a rapid descending phase of S wave, and a short interval between the QRS onset and the nadir of the S wave (Figure 11).

Figure 12 shows differences between RBBB and LBBB patterns in V1 and V6.
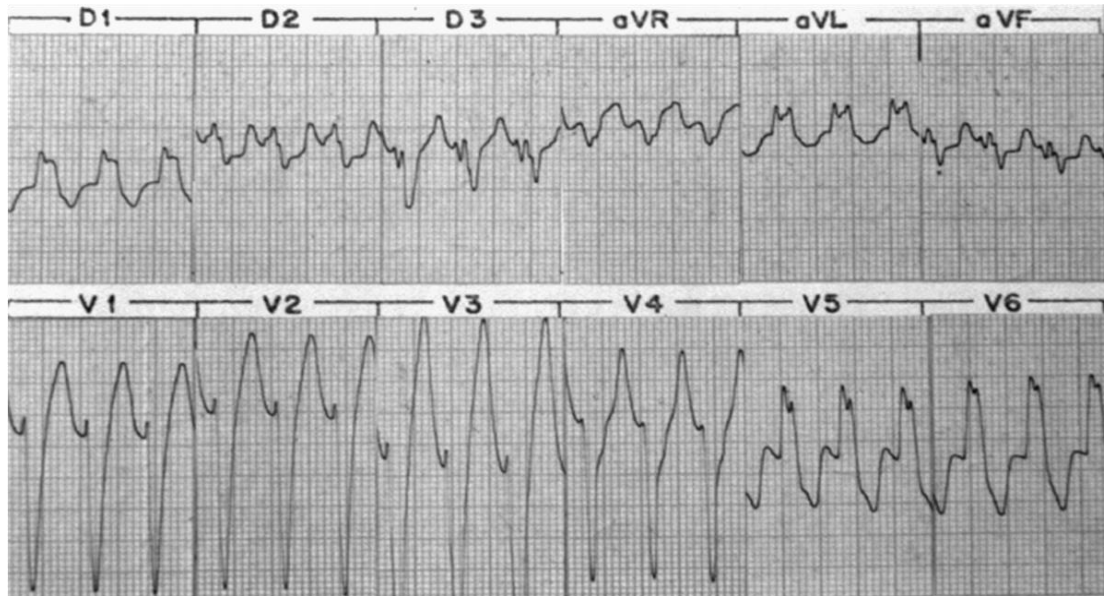

Figure 11 - Wide QRS tachycardia with typical complete LBBB morphology; note the small, thin initial r-wave in lead V1 with a rapid descending S wave. These findings suggest SVT with LBBB.
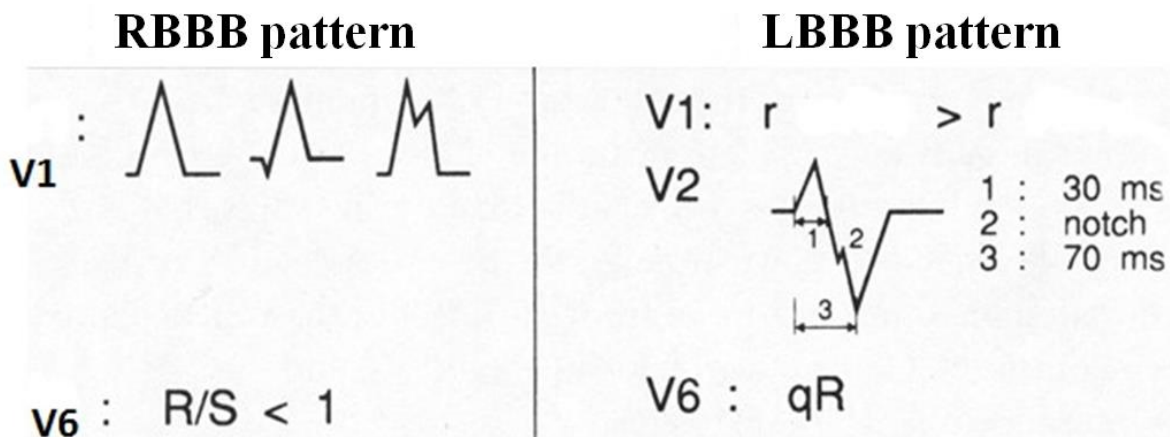

Figure 12 - Morphological features in leads V1 and V6 that strongly suggest a wide QRS tachycardia is ventricular in origin and not SVT-A.

Interval between the onset of QRS complex and the nadir of S wave in the precordial leads

An RS interval ≥100 ms in one or more precordial leads is highly suggestive of VT (13), although it may also occur during SVT with AV conduction through an accessory pathway, during treatment with antiarrhythmic drugs that slow interventricular conduction (mainly flecainide), and in SVT with pre-existing BBB (especially LBBB).

Concordant QRS complexes pattern

Concordance of the QRS complexes in the precordial leads means that the QRS maintains the same direction (all upwards or all downwards) from V1 to V6. Negative concordance, i.e. QRS complexes of entirely negative polarity in V1-V6, suggests VT of apical left ventricular origin. Positive concordance (QRS complexes of entirely positive polarity in V1-V6) is characteristic of VT originating in the postero-basal region of the LV (Figure 13). The differential diagnosis should also consider SVT with AV conduction through an accessory pathway (Figures 14 and 15) which has a similar ECG pattern (1).
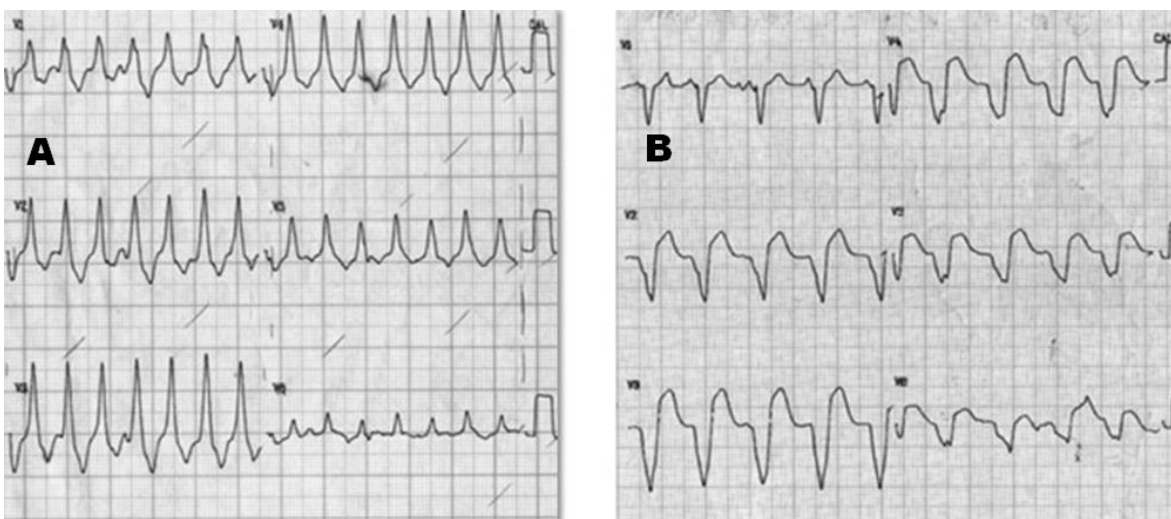

Figure 13 - Two tracings of wide QRS tachycardia from V1-V6 suggesting ventricular origin with (A) positive concordance and (B) negative concordance.
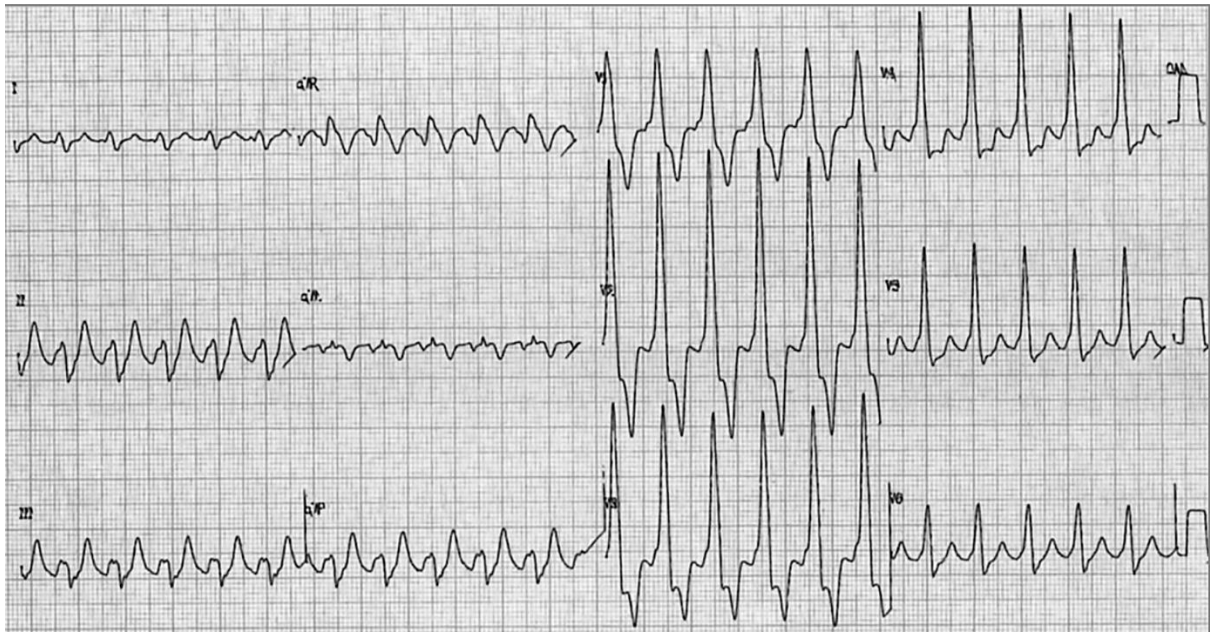
Figure 14 - *Positive concordance in the precordial leads during wide QRS. tachycardia*
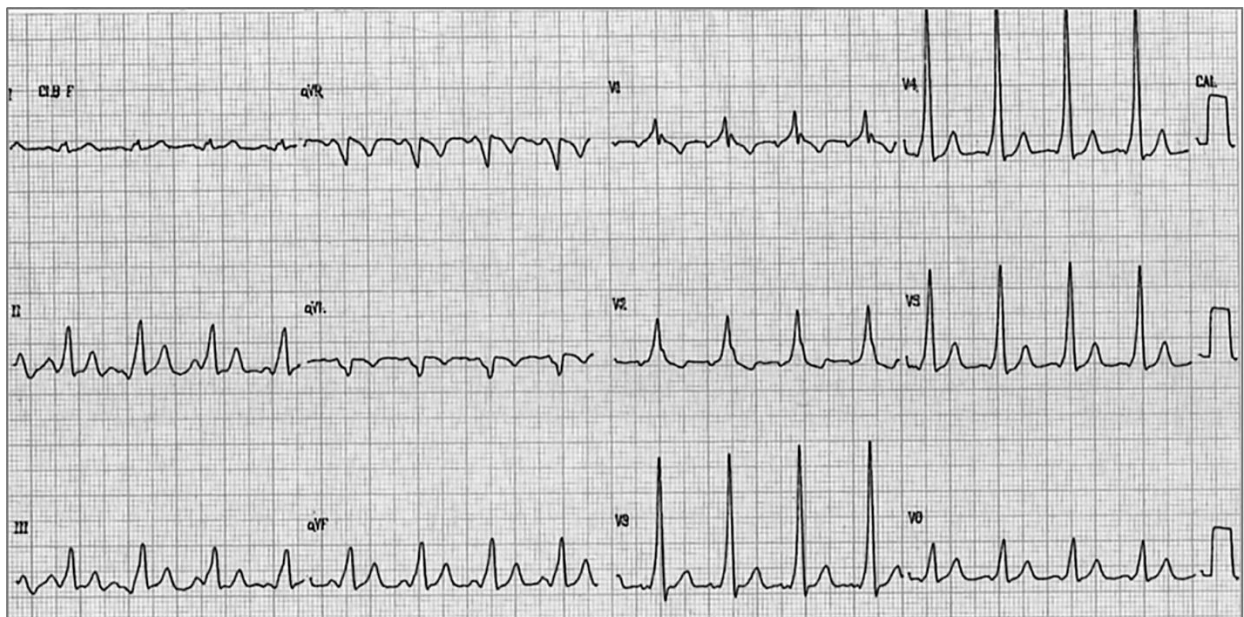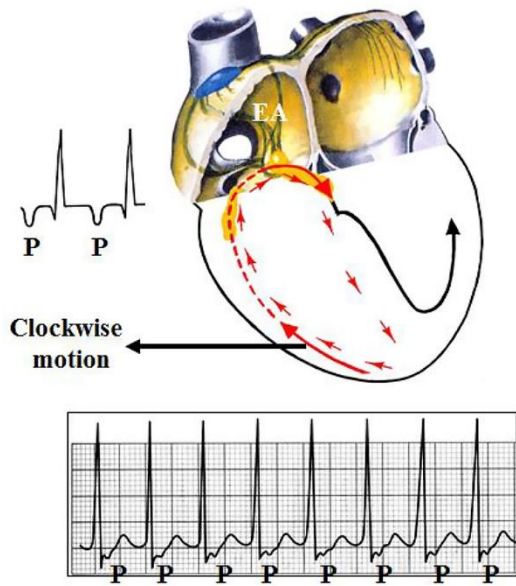

Figure 15 - *ECG from the same patient in Figure 14, after reversion to sinus rhythm, which clearly shows ventricular pre-excitation pattern (WPW). QRS morphology is similar to that observed during the tachycardia event, indicating that the stimulus activates the ventricle in the anterograde direction through an accessory pathway (Figure 16B).*
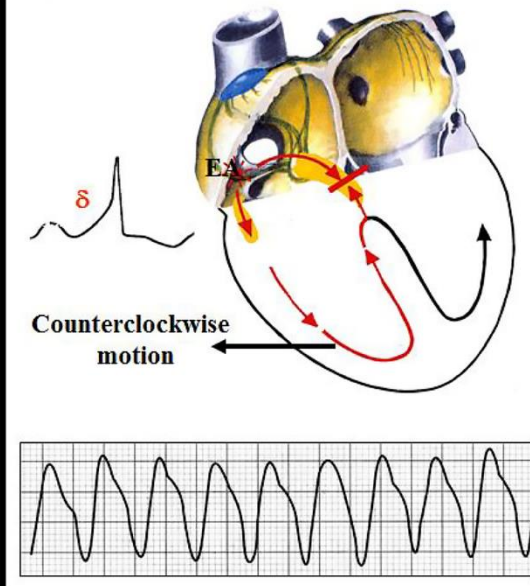
Figure 16 – Paroxysmal supraventricular tachycardia by reciprocal macro-reentry or reentrant in WPW. A) Narrow QRS, clockwise macro-reentry motion. It uses a normal pathway in anterograde fashion and/or anomalous bundle in retrograde fashion. B) Wide QRS, counterclockwise macro-reentry motion. It uses a normal pathway in retrograde fashion and/or anomalous bundle in anterograde fashion.

Presence of QR morphology

Complexes of the QR type, except in lead aVR, during wide QRS tachycardia (Figure 17), and in similar leads during sinus rhythm (Figure 18) suggest scar from previous infarction. This is present in nearly 40% of post-AMI VT (5, 6, 14, 15).
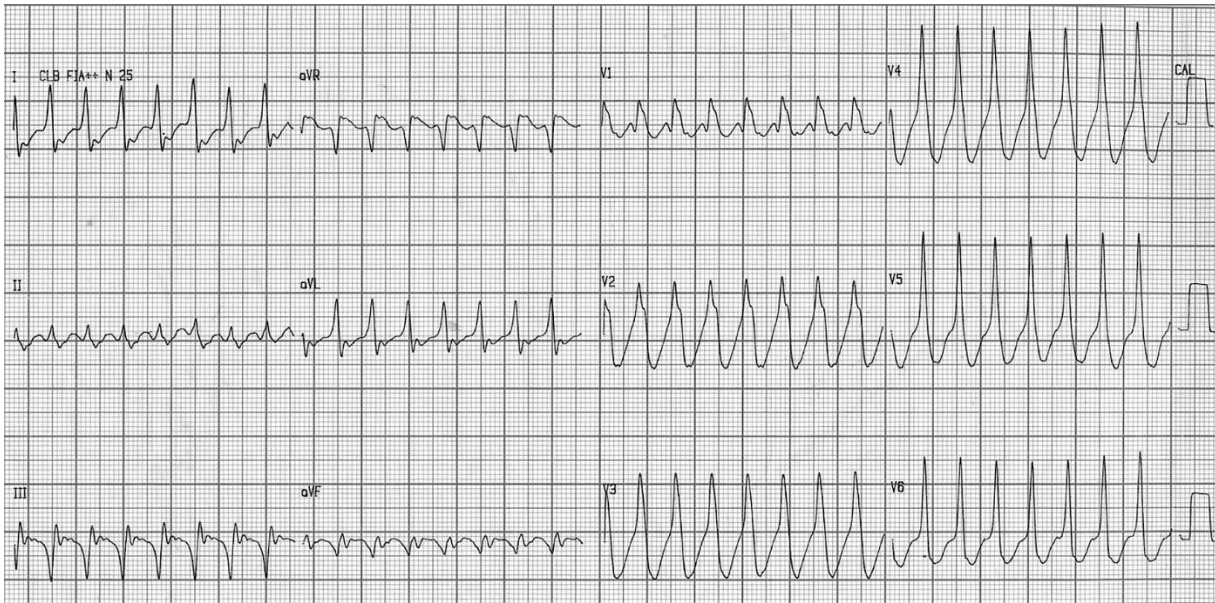
Figure 17 - Wide QRS tachycardia with QR pattern in leads III and aVF suggesting previous inferior wall scar.
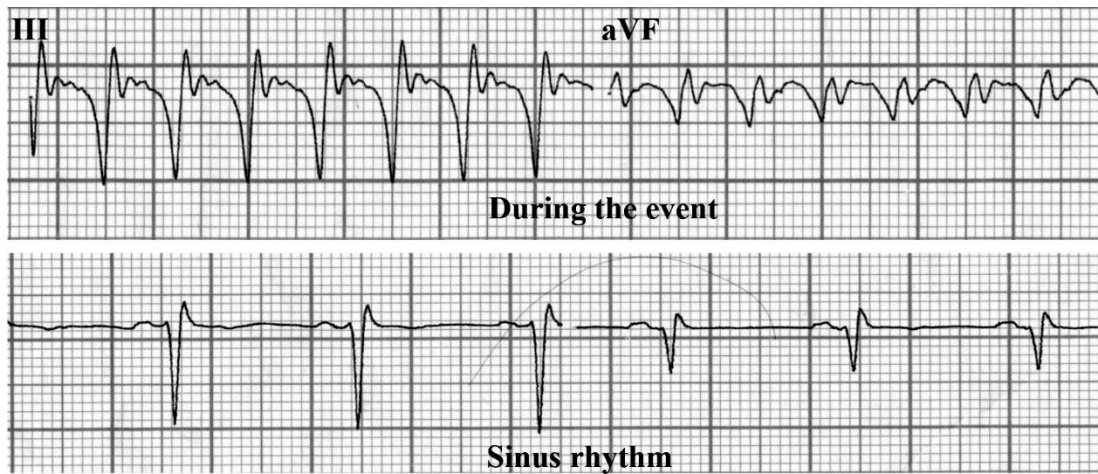
Figure 18 - The same patient above after restoration of sinus rhythm (SR), confirming the presence of an old inferior wall infarct. During sinus rhythm the same QR morphology observed during ventricular tachycardia is present.

Significance of electrocardiogram during sinus rhythm

The ECG during sinus rhythm may show abnormalities such as pre-existing BBB, ventricular pre-excitation, or myocardial infarction scar which help in the differential diagnosis of WQRST. In

addition, the presence of less wide QRS complexes during a tachycardia than in sinus rhythm suggests VT originating near or within the interventricular septum resulting in a more synchronized or simultaneous activation of the ventricles (1) (Figures 19, 20, 21).

Figure 19 - VT with less wide QRS complexes and a pattern of RBBB with axis shifted to the left and AV dissociation suggesting a fascicular VT.

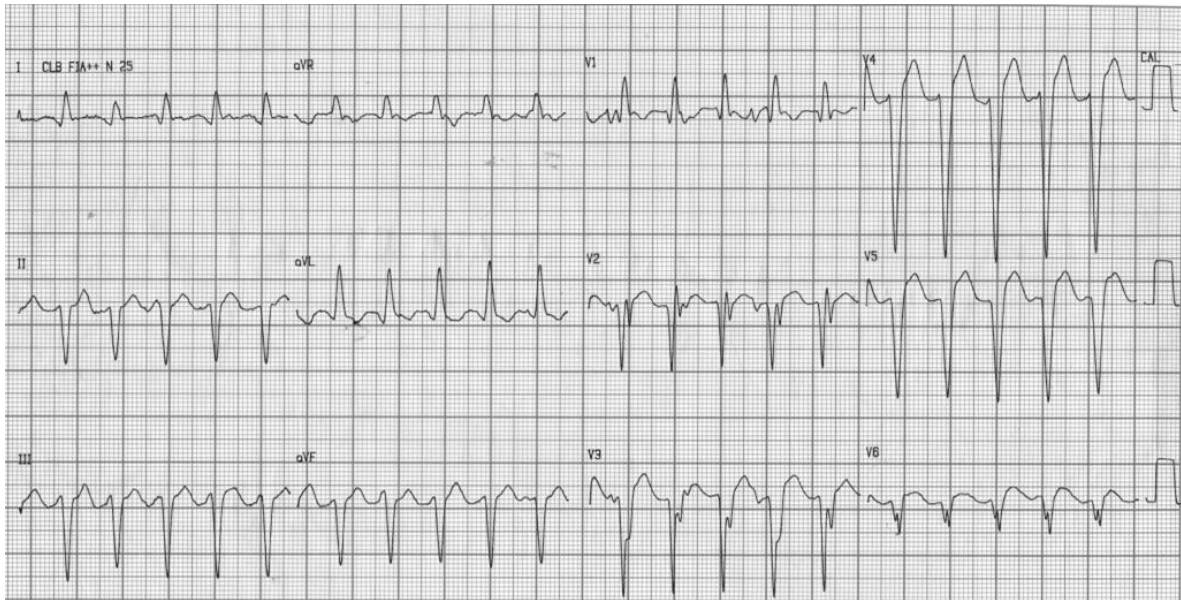*Figure 20 - ECG from the same patient in Figure 19 during sinus rhythm with wider QRS complexes (LBBB) compared to those during ventricular tachycardia.*
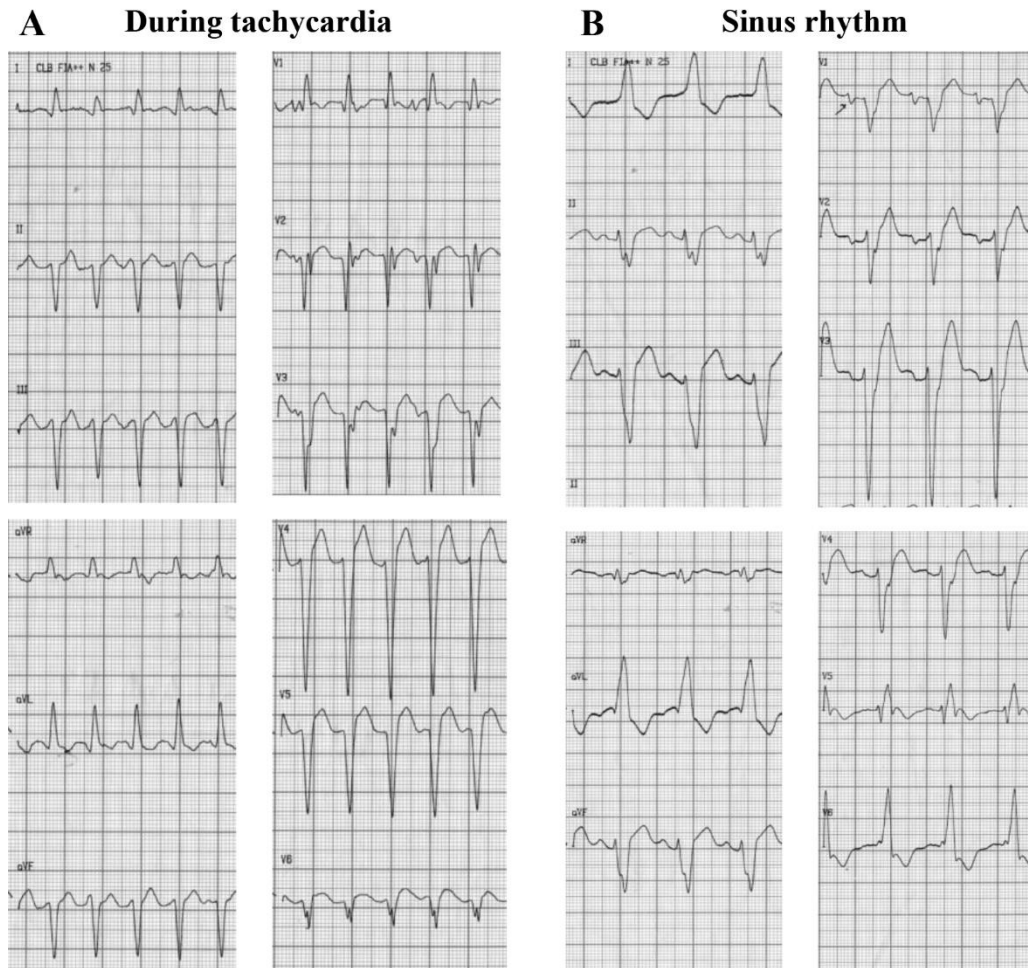
Figure 21 - Narrow QRS (A) and wide QRS (B)

In spite of criteria previously proposed for the differential diagnosis of wide QRS tachycardia, the incidence of errors in the interpretation of these arrhythmias remains high, with serious prognostic and therapeutic implications (16, 17). A systematic sequential analysis of the ECG using algorithms with pre-established and validated criteria improves the accuracy in the diagnosis of WQRST.

Electrocardiographic algorithms for the differential diagnosis of wide QRS tachycardia

Several ECG algorithms were developed to differentiate wide QRS tachycardia. Most presented a very good development in the population on which they were based. A recent review tested five

commonly used algorithms in a control population, and discovered that every one of them performed only relatively well in differentiating VT from SVT (66-77% accuracy) (18). The five algorithms used were: 48%.

I. Brugada algorithm (13). This is the most commonly used algorithm having a potential sensitivity (Se) of 99%; and specificity (Sp) of 97%.

II. Ultra-simple Brugada criterion or RW to peak time (RWPT); i.e. the duration from the onset of QRS to the peak of R in lead II (19): Se 60%, Sp 82%.

III. Vereckei aVR algorithm (20): Se 87.1% and Sp 48%.

IV. New Vereckei algorithm (9).

V. Griffith algorithm (21): Se 94.2% and Sp 39.8%.

VI. The algorithm using a Bayesian approach (22) estimates a score based on 19 electrocardiographic patterns: Se 89% and Sp 52%.

I. Brugada algorithm (Figure 22): Brugada et al (13) proposed 4 sequential criteria for the differential diagnosis of wide QRS tachycardias:

- Absence of complexes of the RS type in all precordial leads (Figure 23);

- R to S interval in any precordial lead with RS complex >100 ms;

- AV dissociation;

- Morphological criteria present in both V1-V2 and V6.

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 (13).
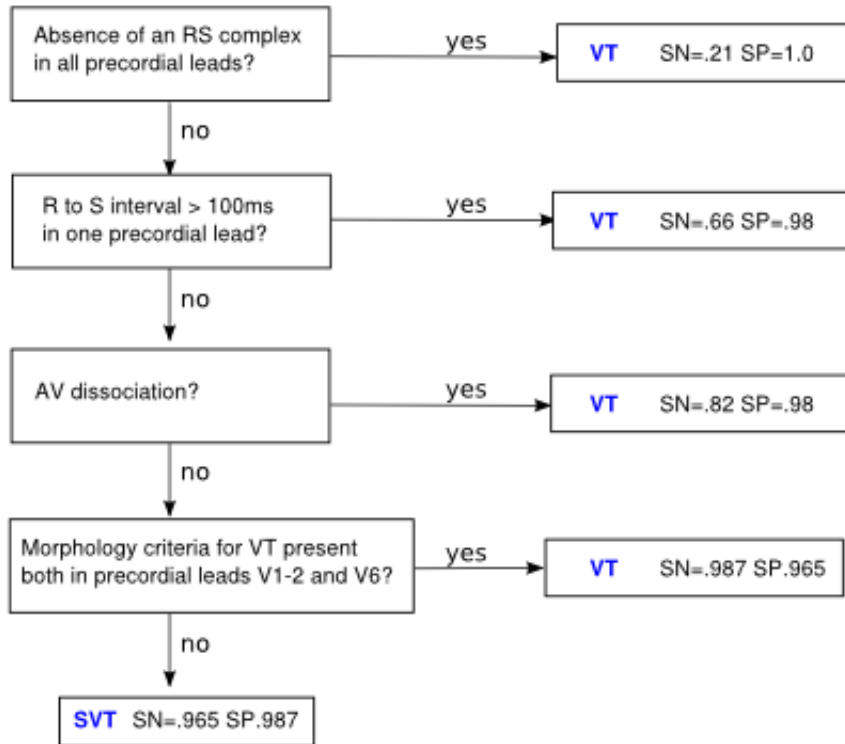
Figure 22 - *Brugada algorithm for the differential diagnosis of tachycardia with wide QRS, with the respective sensitivity and specificity rates of every criterion.*
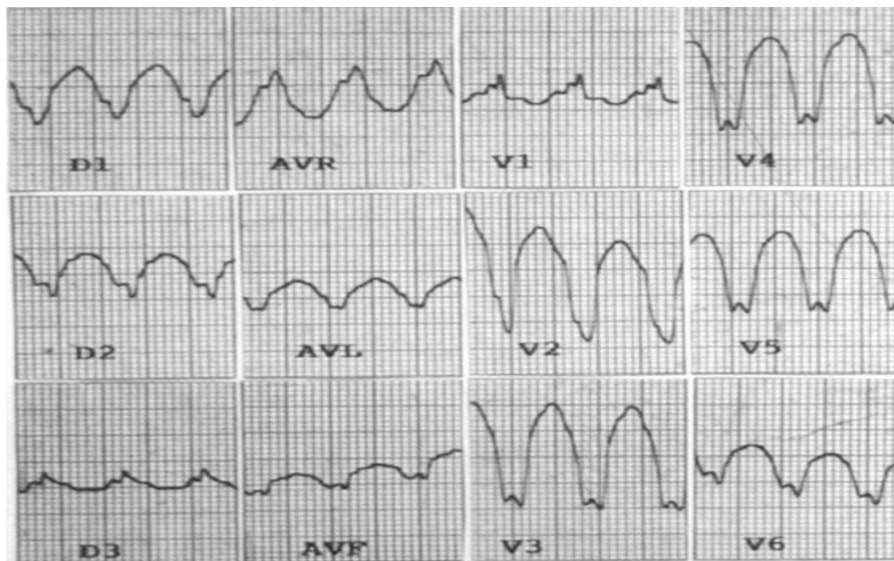

Figure 23 - *Tachycardia with wide QRS showing absence of complexes of the RS type in the precordial leads (first step of Brugada algorithm).*

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

- Patients using antiarrhythmic drugs were not included.

- The authors did not comment if patients with previous BBB, idiopathic VT and pre-excited tachycardia were included in the group studied.

- 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;

- The 4th step of the algorithm keeps the morphological criteria, which are hard to apply in clinical practice.

In addition, other authors found lower rates of sensitivity and specificity (6, 23, 24) than those initially reported by Brugada.

II. Ultra-simple Brugada criterion or RW to peak time (RWPT); i.e. the existing duration from QRS onset (R Wave) to the peak of R in lead II (19). Se 60%, Sp 82%: This criterion uses only lead II (Figure 24). This lead was chosen because it is easily obtained and by the fact of being present in most ECG strips made in the emergency room.

R-Wave Peak Time (RWPT) ≥ 50 ms

I I

R-Wave Peak Time (RWPT) ≥ 50 ms
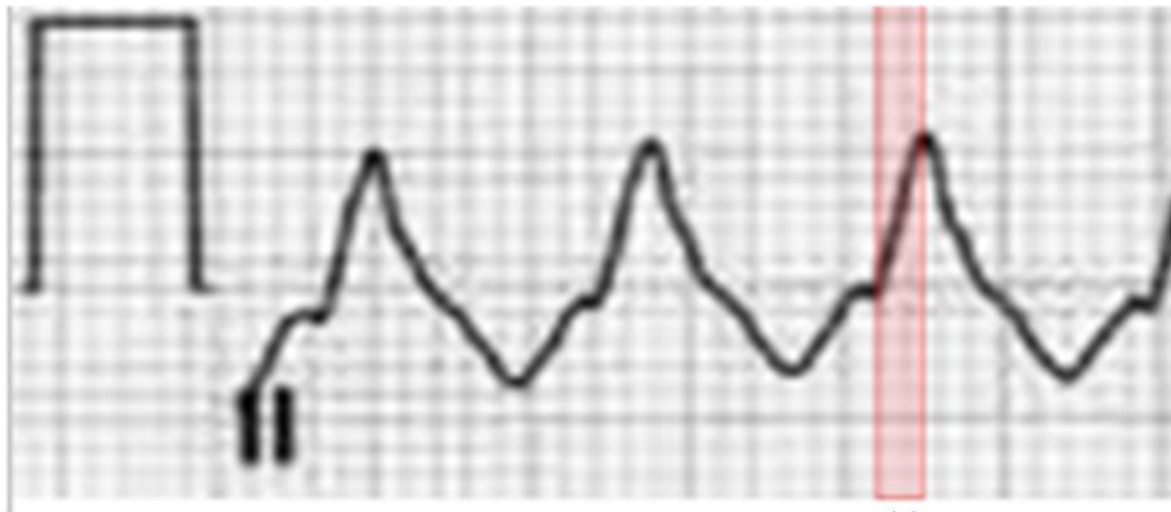
Figure 24 - *Ultra-simple Brugada criterion.*

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 new 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 ≥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.

III. Vereckei aVR algorithm. Se 87.1% and Sp 48%.

In 2007 Vereckei et al (20) proposed a new algorithm for the differential algorithm of wide QRS tachycardia (Figure 25).

***Figure 25** - Vereckei algorithm, published in 2007, for the differential diagnosis of wide QRS tachycardia. F = fusion beat; C = capture beat.*
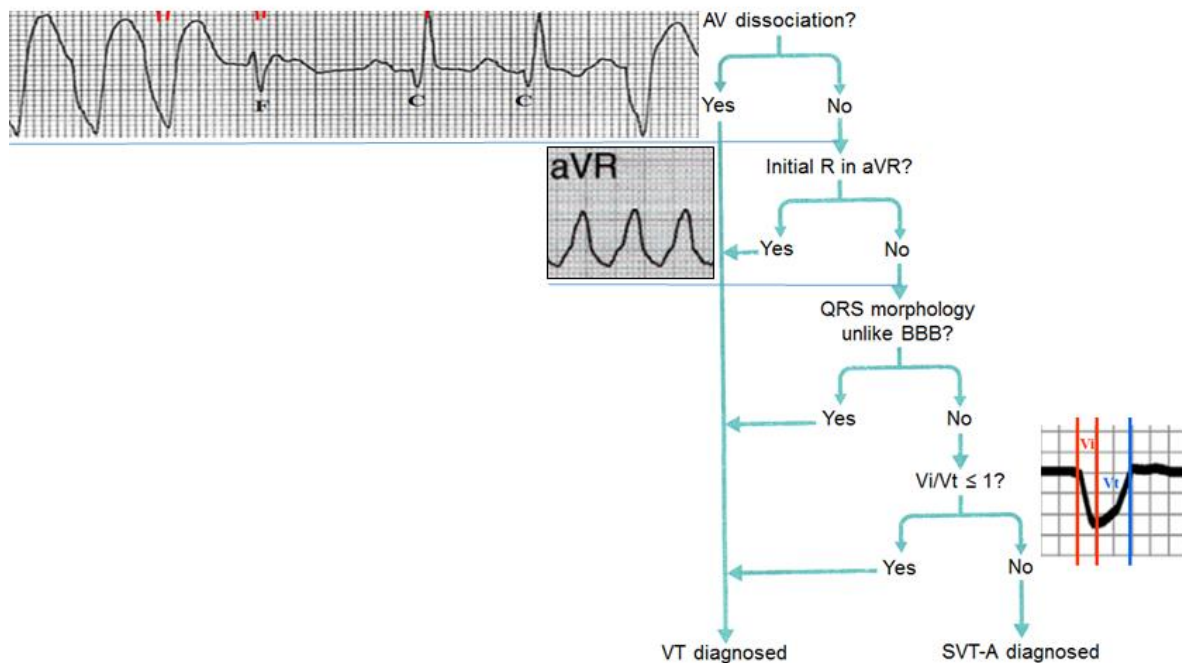
The algorithm is also based on four steps:

- If AV dissociation is present (F and/or C beats), 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 (20).

The v_i/v_t ratio (Figure 26) 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 ventricular focus and a value >1 suggests supraventricular focus.
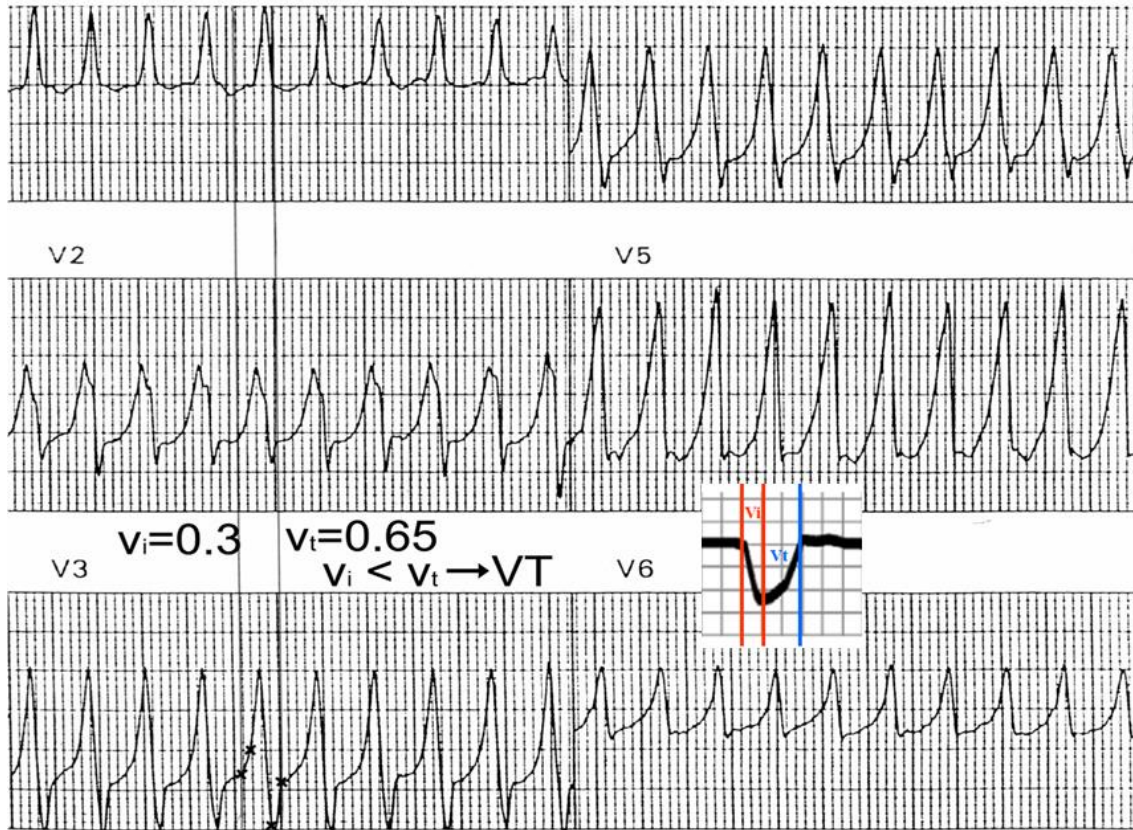
Figure 26 - *Application of the v_i/v_t criterion. The figure shows a wide QRS tachycardia with v_i measured in a lead where the biphasic QRS has easily identified QRS onset and end. Vertical lines indicate the onset and the end of the chosen QRS and small stars indicate the initial and terminal 40 ms of QRS. During the initial 40 ms of QRS the impulse was shifted vertically 0.3 mV, and thus $v_i = 0.3$. During the final 40 ms the impulse shifted vertically 0.65 mV, so $v_t = 0.65$ mV. Thus, the v_i/v_t ratio is <1 and suggests the diagnosis is VT (20).*

Rationale for lead aVR

During SVT-A with BBB the initial ventricular activation sequence moves away from lead aVR resulting in an initial negative QRS complex in aVR (9).

In the population studied by Vereckei, this new algorithm had a better accuracy than the Brugada algorithm (20) 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 (20). 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 (25). 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 (9).

IV. **New algorithm by Vereckei:** In 2008, Vereckei et al (9) published a new algorithm that was based on the direction and velocity of the initial and terminal portions of ventricular activation (Figure 27). In spite of not having any new fundamental criterion, it is based on three new concepts:

- The exclusive analysis of a single lead (aVR) for the differential diagnosis of wide QRS tachycardias (Figure 28);

- VT is classified in two main groups: A) VT with origin in the inferior and apical regions of the ventricle having an initial R wave in aVR; B) 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.

- Removal of AV dissociation and morphological criteria used in previous algorithms and traditional criteria.
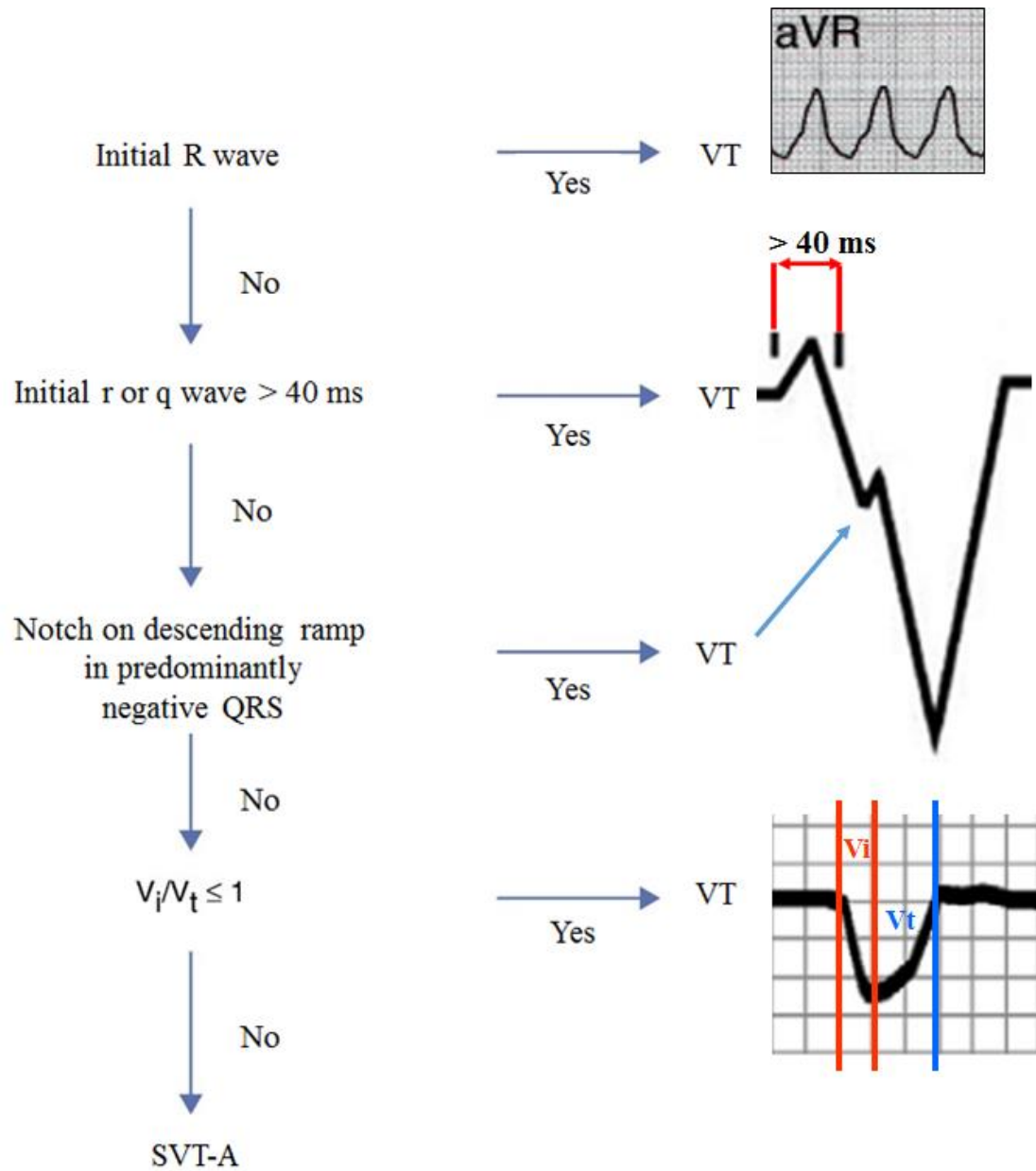
Figure 27 - Algorithm proposed by Vereckei to differentiate wide QRS tachycardias based on the aVR lead.

The criterion of AV dissociation, in spite of 100% specificity, is not very sensitive because identifying dissociated atrial activity in fast wide QRS tachycardias 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 (Figure 29).

Figure 28 - *Wide QRS tachycardia with initial R wave in aVR (first step of Vereckei algorithm) suggesting VT.*

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Figure 29 - *Morphological criteria used in the Vereckei algorithm for the differential diagnosis of wide QRS tachycardia.*

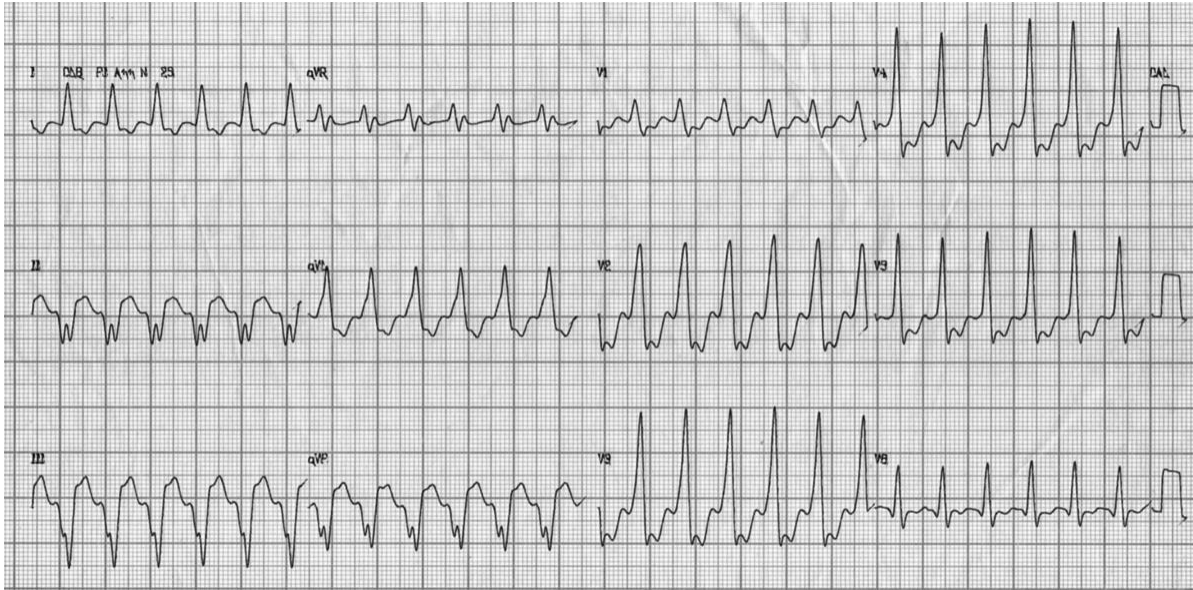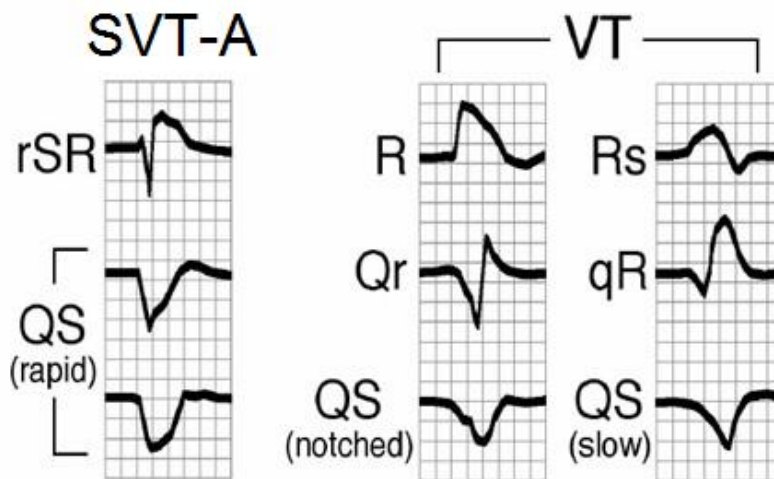The accuracy of the new Vereckei algorithm was superior to the Brugada algorithm not by showing a statistically significant difference compared to its earlier version, but having a greater sensitivity and specificity in the diagnosis of SVT compared to the Brugada criteria.

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.

V. Griffith algorithm. Se 94.2%, Sp 39.8%: The algorithm of Griffith (21) reverses the diagnostic strategy: SVT-A is diagnosed when the ECG findings correspond to typical LBBB or RBBB:

- Complete LBBB: rS or QS wave in V1 and V2 with nadir of S wave <70 ms and pure monophasic R wave in V6 (Figure 30).
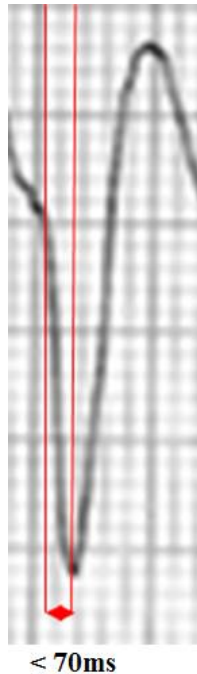
Figure 30 - *CRBBB: triphasic pattern RSR' in V1 and RS in V6, with height of R wave greater than S wave depth.*

VI. The Bayesian approach (22) estimates a score based on 19 ECG patterns: Se 89% and Sp 52%.

Finally, a practical approach in the diagnosis of wide QRS tachycardia is the use of the following six successive steps proposed by Miller (6):

- **First step:** Determine the atrioventricular ratio. In the presence of AV dissociation, the diagnosis is VT. If not, got to Step 2.

- **Second step:** QRS axis in the FP in the right superior quadrant (northwest quadrant axis). When present, it indicates VT. When absent, go to Step 3.

- **Third step:** Vi/Vt ratio when > than 1, SVT-A is diagnosed; if not, continue to the Step 4.

- **Fourth step:** Absence of RS pattern in the precordial leads indicates VT. If not, go to Step 5.

- **Fifth step:** RS interval in the precordial leads >100 ms indicates VT. If not, continue to Step 6.

- **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.

Table 1 summarizes 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 (Table 2)	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
History of infarction, angina, CHF, cardiomyopathy, history of correction of congenital heart disease, family history of SCD: suggestive	Strongly suggestive	No

of HCM, ARVD/C, long QT syndrome and Brugada syndrome		
History of paroxysmal tachycardias responsive to vagal maneuvers or adenosine.	No	Characteristic
Previous ECGs with short PR (<120 ms), wide QRS and delta wave.	No	It indicates pre-excitation as cause.
Previous ECG with bundle branch block pattern identical to the pattern of the event	No	Characteristic
End of event with vagal maneuvers or adenosine	Rare	Yes.
QRS duration	>140 ms if RBBB pattern; >160 ms of LBBB pattern	< 140 or < 160 ms
SÂQRS in the frontal plane	Suggestive when SÂQRS is in the northwest quadrant between –90º and ± 180º	No
QRS Pattern in V1	In the presence of LBBB pattern, initial r >40 ms and rS interval greater than 70 ms is suggestive.	Initial narrow r, and clean s, with no notches if LBBB and triphasic pattern if RBBB

	Biphasic or monophasic pattern if RBBB. When biphasic in V1 R' > R (rabbit ear sign) (Figure 29) (10)	
QRS Pattern in V6	rS, Qrs, QS, QR or monophasic R. If the pattern was RS R<S.	qRs, Rs or RS with R>S
The distance from the onset of QRS up to the nadir of S >100 ms (Brugada sign) (Figure 30)	If present, it is diagnostic.	Lower
Notch near the nadir of the S wave (sign of Josephson) (Figure 31)	Characteristic	Absent
QRS complexes of the R or Rs type	Diagnostic	No
Initial q or r wave with duration >40 ms in aVR (qR or rS)	Diagnostic	No
Pattern matching in precordial leads	Strongly suggestive.	No
Presence of fusion beats	Strongly suggestive.	No
Presence of capture beats	Strongly suggestive.	No
Second-degree ventricular-atrial block	Characteristic when present: QRS/P ratio;	No

	however, with a greater number of QRS than P.	
Pattern of LBBB with axis in the right upper quadrant	Nearly always VT.	No
Ratio of duration between the initial and final part of QRS ≤ 1 (26)	Suggestive.	>1

Table 1 – *CHF: congestive heart failure; SCD: sudden cardiac death; HCM: hypertrophic cardiomyopathy; ARVD/C: arrhythmogenic right ventricular dysplasia/cardiomyopathy; LBBB: left bundle branch block; RBBB: right bundle branch block.*
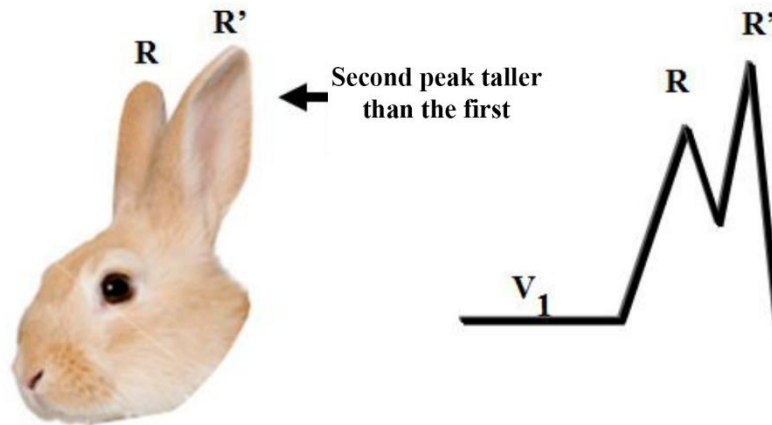
Figure 31 – *R' > R = Sign of rabbit ears.*

Figure 32 illustrates an example of Brugada's sign for VT
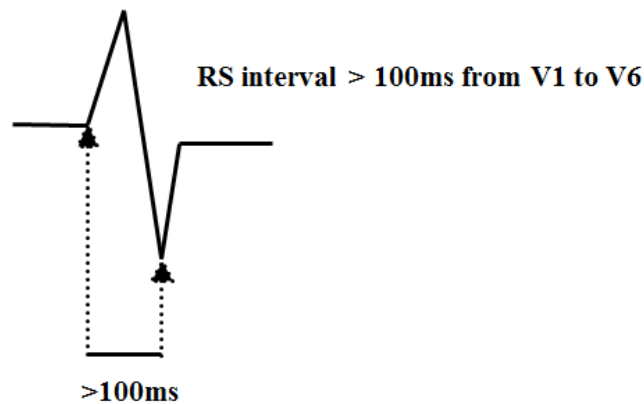
Figure 32 – *Brugada's sign is highly specific for VT, however not much sensitive.*

Josephson's sign

> 100 ms
Brugada's sign

Figure 33 -

VT with structural heart disease	VT without structural heart disease
CAD: in this scenario scar tissue from previous AMI develops. This leads to uneven conduction of the electrical impulse in the heart resulting in a reentry circuit for developing VT.	Idiopathic ventricular tachycardia.

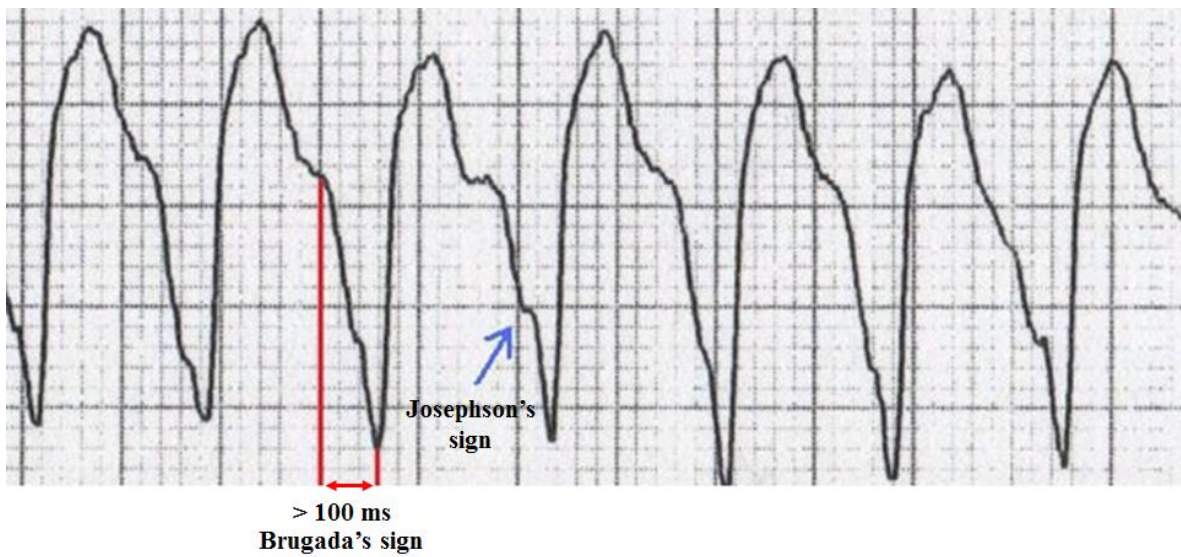|	
Systemic diseases that affect the myocardium including: sarcoidosis, systemic lupus, hemochromatosis, rheumatoid arthritis and others.	Idiopathic ventricular fibrillation.
Dilated cardiomyopathy, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, and chronic Chagasic cardiomyopathy.	Channelopathies.
Mitral valve prolapse syndrome.	Early repolarization syndrome.
Post-operative of congenital heart diseases surgery. Example: tetralogy of Fallot.	Electrolytic alterations: hypokalemia, hyperkalemia, hypocalcemia, hypomagnesemia.
Heart valve disease.	Effects of drugs that prolong the QT interval causing torsade de pointes.
Ventricular hypertrophy of any etiology.	Use of illegal drugs as cocaine, methamphetamines, etc.
	Increased sympathetic tone
	Digitalis intoxication: bidirectional tachycardia.

Table 2 – Comparison of VT with or without structural heart disease

III)   Markedly or grossly irregular wide QRS tachycardia.

Figure 34 illustrates the admitting ECG in a 9-year-old boy presenting to the emergency room following an episode of syncope. He did not have clinical evidence of structural heart disease. There were no physical signs of AV dissociation such as Cannon "a" waves in the jugular pulse, variations in intensity of S1, or beat by beat variations in systolic blood pressure. The physical exam revealed a very rapid irregular heart rate between 160 and 300 bpm (average: 230 bpm).
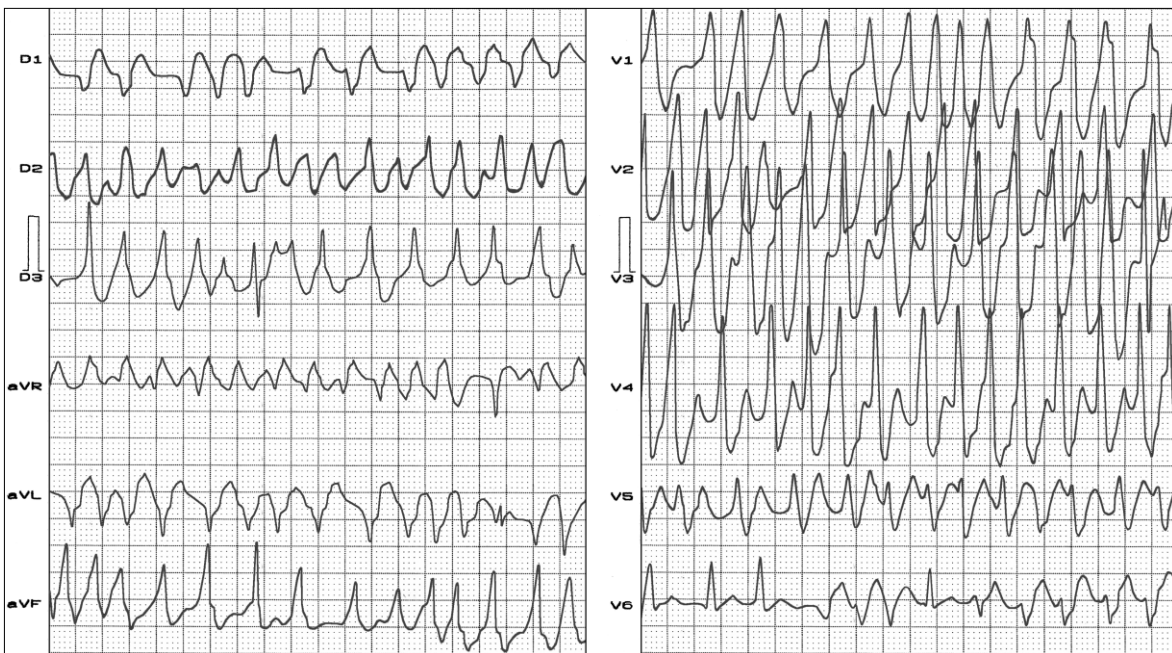
Figure 34 - Very irregular and fast wide QRS tachycardia. In the English language this is known as FIB (Fast, Irregular, and Broad). The tracing corresponds to pre-excited AF with a left lateral accessory pathway. The so-called concertina effect or Öhnell accordion phenomenon is observed and caused by progressive greater or smaller amount of ventricular activation by the anomalous bundle and manifesting QRS complexes with greater or lesser degrees of fusion. HR irregularity (between 160 and 300 bpm) is due to changes in the refractory period of the action potential of the anomalous bundle. The wide QRS complex is due to the anterograde ventricular activation through

the anomalous pathway, and QRS duration variations indicate variable anomalous ventricular activation. Capture and/or fusion beats are visible. V1 shows a monophasic pattern. These three elements may be misinterpreted as VT.

A shorter RR interval indicates the short anterograde refractory period of the anomalous pathway that may increase the risk of sudden cardiac death (27, 28). From the morphological aspects of the QRS complexes the arrhythmia is very suggestive of VT; however, the great irregularity of the rhythm suggests atrial fibrillation.

The differential diagnosis with AF with bundle branch block is easily made by high HR in this case (29). Pre-excited AF is a very serious arrhythmia with increased risk of sudden cardiac death, and it requires therapeutic electrical cardioversion.

Summary

The differential diagnosis of wide QRS tachycardia 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 tachycardia 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.

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