Case report

Female, 57 years old, married, white, clerk, natural from Fortaleza, Ceará, Brazil (stopped working 15 years ago)

She was hospitalized with picture of congestive heart failure (CHF).

Main complaint "heart grown" about five years ago.

Family history: One of the five siblings died by “cardiomegaly". Negative epidemiology for Chagas disease.

Pathological personal history:

She was smoker from 16 to 28 years old.

Systemic hypertension since 2012 in irregular treatment.

In 2014 presented the first major CHF that led to hospitalization for 15 days. At that time, rheumatoid arthritis was diagnosed with mild joint aggression. After two years she stopped the medication on her own and presented a new CHF causing a second hospitalization.

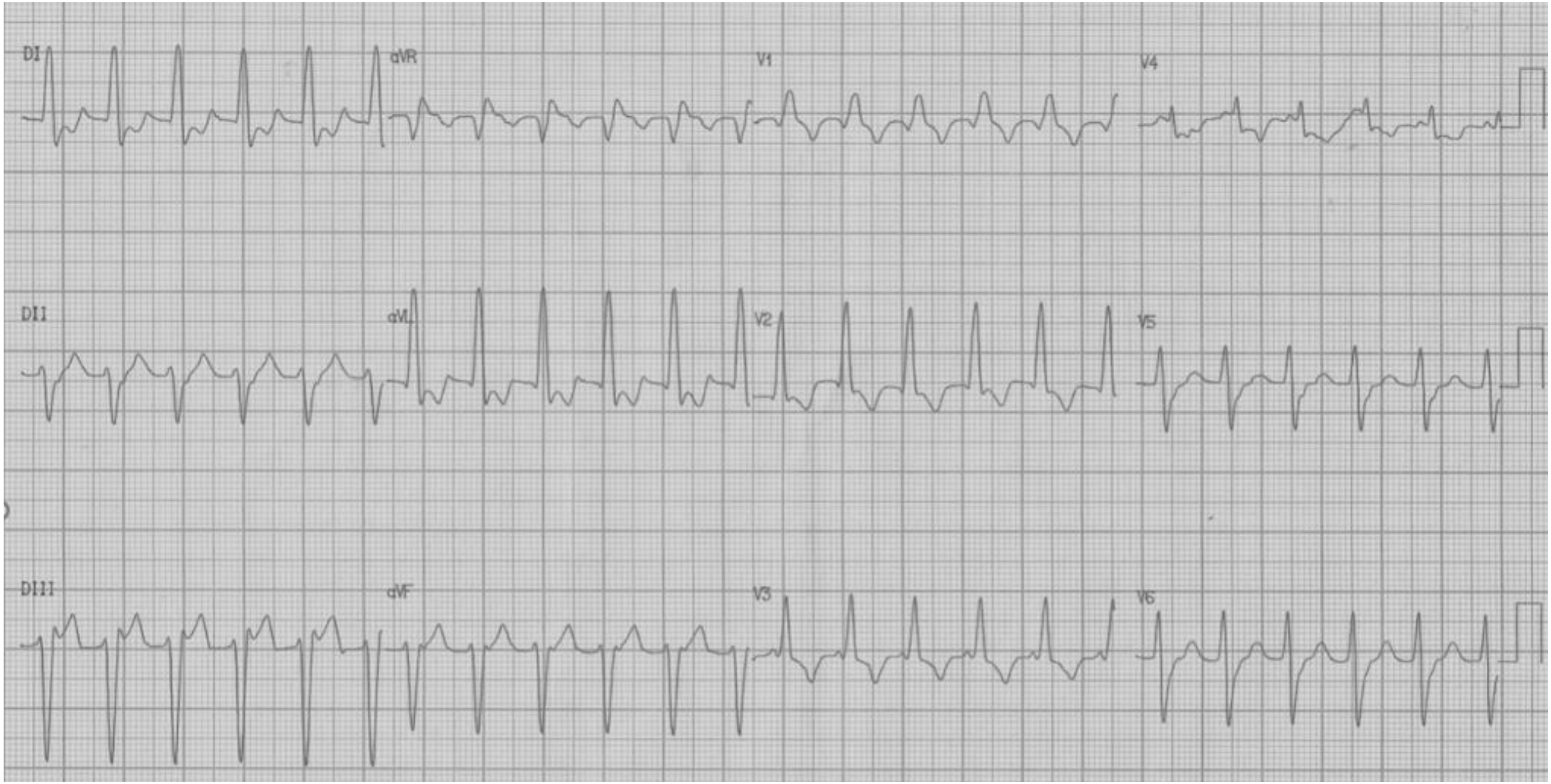
In April 2019 a sudden cardiac arrest was reversed. The event was interpreted as a resulting from not confirmed "acute myocardial infarction". Cardiac catheterization shows normal coronary arteries and left ventricular dilatation.

Transthoracic echocardiogram: significant increase of the left ventricle with eccentric modality. Left ventricular mass = 290g (increased), LV diameters = 62/52, LVEF = 32%.

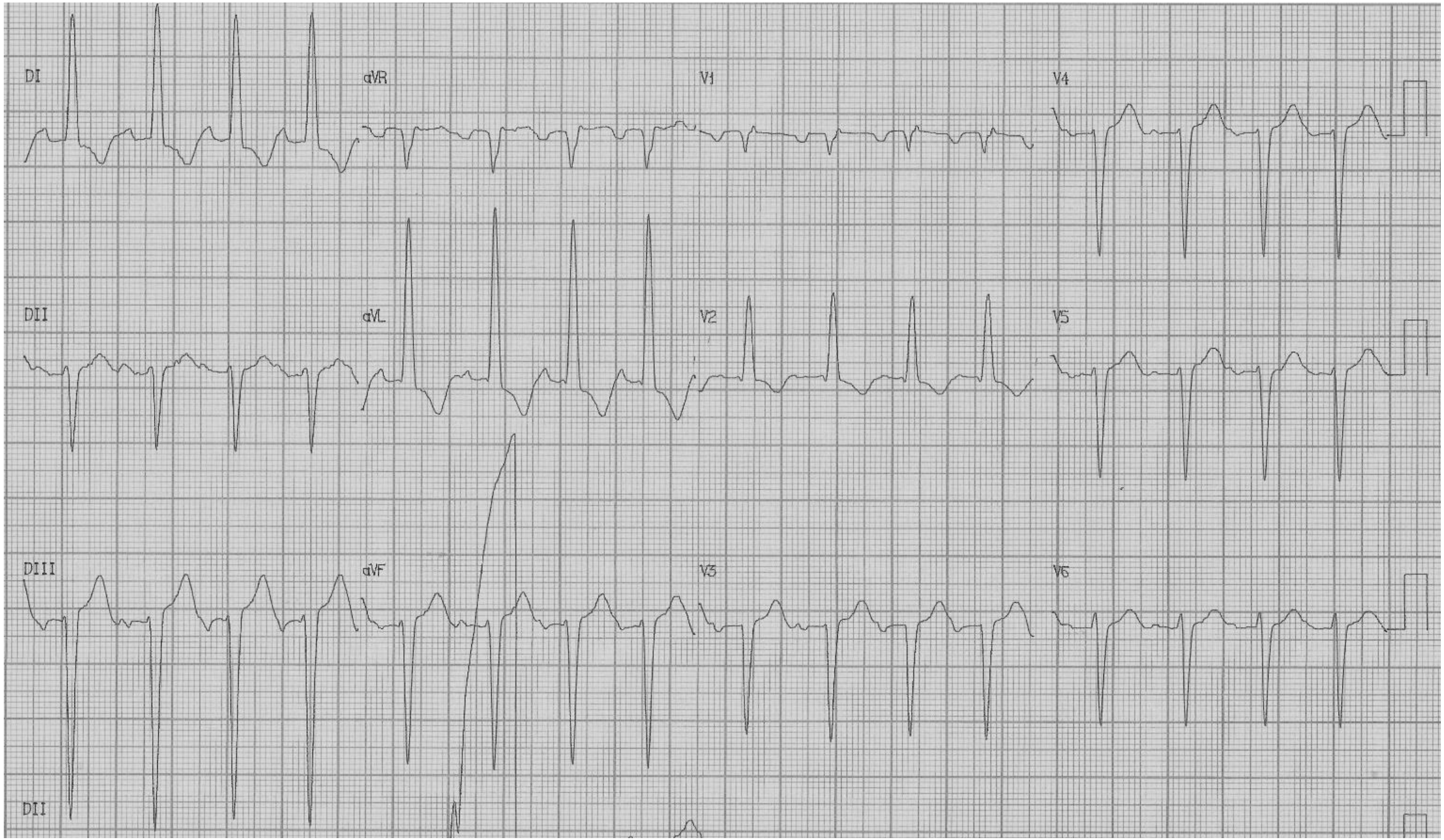
Questions:

1. Which is the diagnosis of this wide QRS complex tachycardia of tracing A or 1?
2. Why is the width of ECG B different from ECG C?
3. What is the electrocardiographic diagnosis of each of the three tracings?

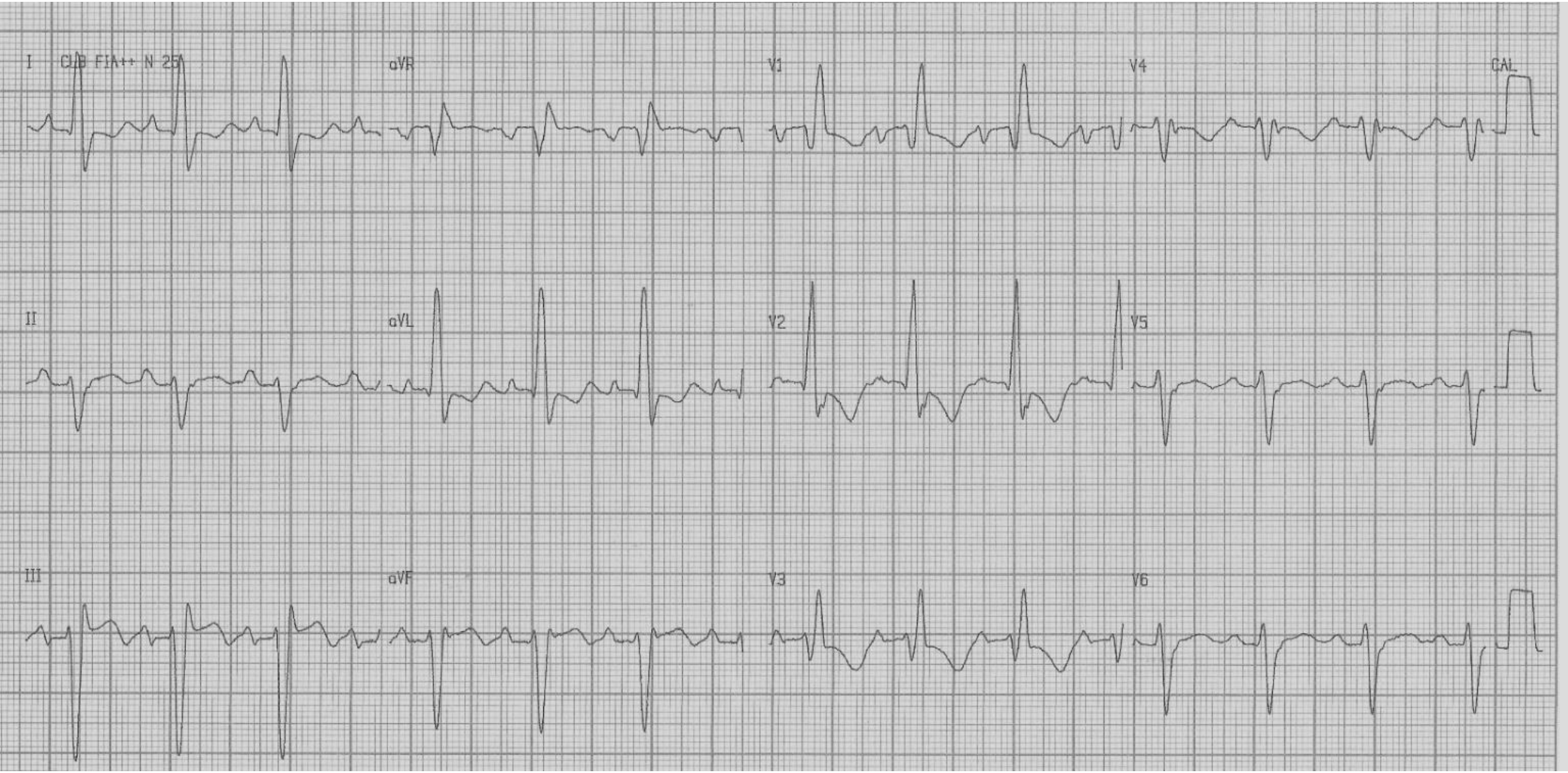
Tracing A or 1



Tracing B or 2



Tracing C or 3



Dear Andrés,
My interpretation may be somewhat limited based on the size of the complexes on my phone.
I believe the first EKG shows a left ventricular tachycardia originating somewhere near the apical portion of the left ventricle. Retrograde P waves are easily seen in most leads.
The second EKG shows sinus tachycardia with left atrial enlargement, left ventricular hypertrophy and left anterior fascicular block. Lead V2 seems inappropriate as it interrupts the progression of the QRS complexes.
The EKG-C is very interesting. There is also sinus tachycardia as in the previous EKG but the QRS is now widened with right bundle branch block, left anterior fascicular block, and prominent anterior forces suggesting left septal fascicular block.
I would be most interested in your thoughts on these EKGs.
I am currently enjoying my retirement, but still find time to teach EKG interpretation to our residents at the University of Utah.
I am sending best wishes to you and your family. I look forward to continue participating in your wonderful EKG offerings.

Frank Yanowitz MD
Professor of Medicine (Retired)
University of Utah School of Medicine

Very tough ECG'S.

General comments :

I will only discuss the ECG's regardless of the clinical story.

The ECG calibration is standard especially the amplitude with 1 cm scale identical for all 3 tracings (I am saying that since one may believe – as it looks – that the scale of the QRS amplitude during tachycardia is half the one on the 2 other tracings).

Tracing A: Tachycardia 150/min, QRS with a pattern of RBBB, LAFB , and also maybe LSFB (prominent R in V2).

Mechanism of the tachycardia very difficult to ascertain: a) atrial tachycardia; b) AVRT. In any case since the P is obviously negative in V1, this cannot be an AT or AVRT involving a left AT or a retrogradely conducting left lateral accessory pathway. The P seem negative after the QRS in leads I and aVL but also negative in V1; are we dealing with a retrogradely conducting right anteroseptal accessory pathway.

Tracing B: Sinus rhythm 100/min with PR=0.20s; QRS with a pattern of left anterior fascicular block and left septal fascicular septal. No RBBB

Tracing C: Sinus rhythm 80/min with PR=0.16s QRS with CRBBB plus left anterior fascicular block and left septal fascicular block.

Unsolved issues:

In addition to the mechanism of the tachycardia

I do not understand the marked reduced voltage of the QRS during tachycardia as compared to sinus rhythm.

I do not understand why CRBBB is present in **Tracing C** (sinus 80/min) but not in **Tracing B** (sinus 100/min).

In summary – A Lot of incertitude's with these tracings.

I would certainly be happy to hear about my colleague's interpretation;

Please point-to-point comments.

Thanks

Prof Bernard Belhassen

Department of Cardiology, Tel Aviv Medical Center and

Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel.

Electronic address: bblhass@tasmc.health.gov.il

SVT due to Concealed left lateral accessory pathway, plus left anterior fascicular block and variable RBBB
Poor r progression.

Josep Brugada

Most likely bundle branch reentry with antegrade conduction over left bundle. VT not excluded.

Prof. Em. Dr. Pedro Brugada.
Scientific Director, Cardiovascular Division.
Free University of Brussels (UZ Brussel) VUB.
CEO Medical Centre Prof. Brugada, Aalst, Belgium.
Director, Arrhythmia Unit, Hospiten Estepona, Spain.

Tracings 2 and 3 show sinus rhythm Incomplete and complete RBBB 1 degree AV block , LAFB , biatrial abnormality and LVH

Tracing 1 shows similar QRS pattern with short RP SVT the P wave is negative in Lead 1 hence either LA focal tachycardia or left sided pathway

Atrial morphology doesn’t look like AVNRT.

Look forward to your impression

Melvin M. **Scheiman MD**

A little known fact about the Master of masters...Mel is actually his middle name. His real first name is Mahaim. He is truly one of the greats in cardiac electrophysiology, and his humility, kindness and knowledge will forever pave the way in this field.
Jonathan C. Hsu, MD,

Spanish

Hola Andrés
Presenta una taquicardia supraventricular con diferentes grados de aberrancia de las fibras medioseptales de acuerdo a la frecuencia por lo que impresiona una taquicardia auricular ectopica.
En el 3er ECG ritmo sinusal y continúa con bloqueo del fasciculo anteroseptal y signos de crescimiento atrial derecho. Con fuerças anteriores prominentes.
No descartaría una miocardiopatia por taquicardia asociada a hipertensión pulmonar o embolia pulmonar aguda.
Un abrazo
Dr. Martín Ibarrola
Centro Cardiovascular Bella Vista, Buenos Aires, Argentina.

English
Hello Andres
It presents an ectopic atria tachycardia with different degrees of aberrancy on the left septal fibers of the LBB according to the frequency, thus affecting this ectopic atrial tachycardia.
In the 3rd ECG sinus rhythm and continues with anteroseptal fascicular block and signs of right atrial enlargement. With prominent anterior forces.
I would not rule out a myocardiopathy for tachycardia associated with pulmonary hypertension or acute pulmonary embo
A hug
Martin Ibarrola MD Grupo HTA en la Mujer, SAC - FASE, FAHA, FESC
Bella Vista Cardiovascular Center, Buenos Aires, Argentina.

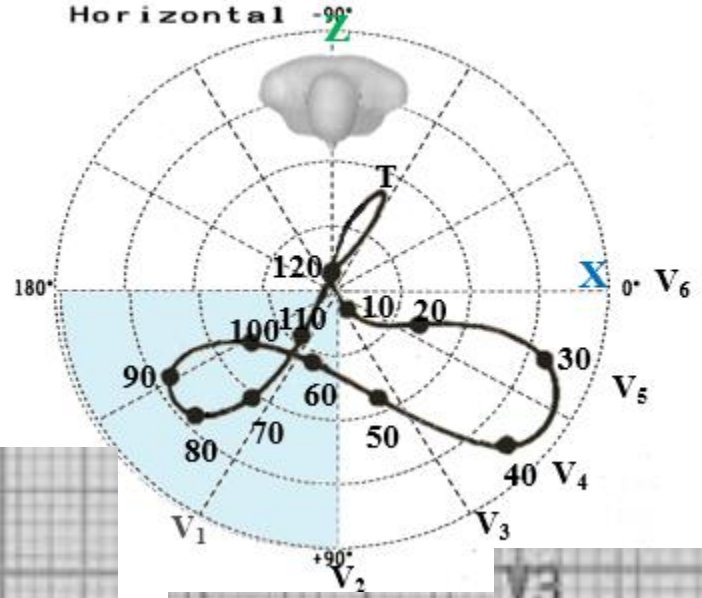
Hi friend: It is not easy, but I risk it:

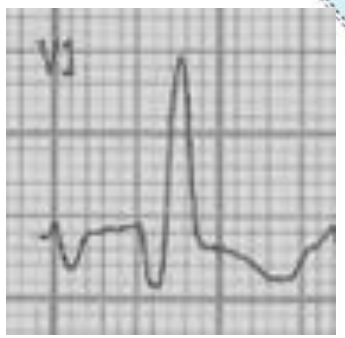
Answer 1: Sustained wide supraventricular tachycardias with aberrancy, HR 145bpm. atrial tachycardia with aberrant preexisting LAFB associated with prominent anterior QRS forces(PAF) that evoke fibrosis / necrosis on inferolateral wall of the left ventricle

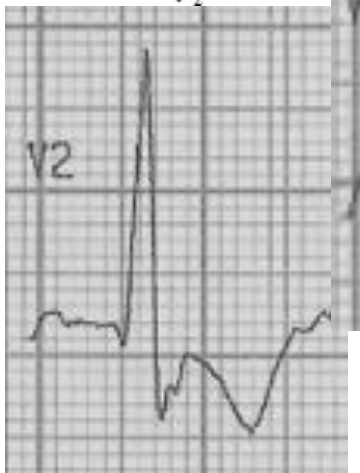
Answer 2: ECG 2-B differs from ECG 3-C by association with LAFB, righ end conduction delay (RECD) or RBBB Kennedy type 3(see figure below), persisting PAF.

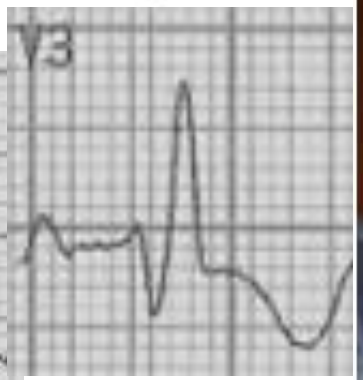
Answer 3: ECGs evoke non-chagasic and not ischemic dilated cardiomyopathy probably de caused infiltrative (intra o extracelular) predminantly in the laterobasal LV wall.

Juan José **Sirena MD**
Santiago del Estero Argentina
Institute of Cardiology of Santiago del Estero









ECG1=taquicardia com padrão de BRD/BDAS/BDAM com morfologia semelhante ao ECG C-3 em ritmo sinusal com um detalhe: quando o ECG durante a taquicardia é mais estreito que durante o ritmo sinusal segundo Dr Stevenson sugere TV, neste caso seria fascicular
Diag diferencial TSV com aberrância: TAatrial/TSV por via acessória
ECG2=BDAS&BDAM,HVE,SAE,BIA-A,
ECG3+ BRD+ BDAS+ BDAM ,BIA-A,SAE,HVE

English

ECG A-1 = Tachycardia with RBBB / LAFB / LSFB pattern with ECG3-like morphology in sinus rhythm with one detail: when the ECG during tachycardia is narrower than during sinus rhythm according to Dr Stevenson suggests VT, in this case it would be fascicular Differential Diag TSV with aberration: atrial tachycardia / supraventricular tachycardia with by accessory pathway ECG B-2 = LAFB& LSFB, LAE, LVH , BIA-A, ECG3-C: LAE+ LVH + RBBB + LAFB + LSFB

Dr Stevenson wrote: The 12-lead ECG of the tachycardia is important.

1. A RBBB–like configuration, with a dominant R wave in V1
2. Superior and rightward-directed frontal plane axis (indicates initial depolarization from the inferolateral LV)
3. A monophasic R wave in aVR,
4. Notching of the QRS can also be a sign of scar.

Delayed gadolinium enhancement in the CMR is consistent with scar, which can be the substrate for VT

These features are specific for VT.

1. Stevenson WG. My approach to ventricular tachycardia. Trends Cardiovasc Med. 2015 Aug;25(6):565-6. doi:10.1016/j.tcm.2014.11.016.)

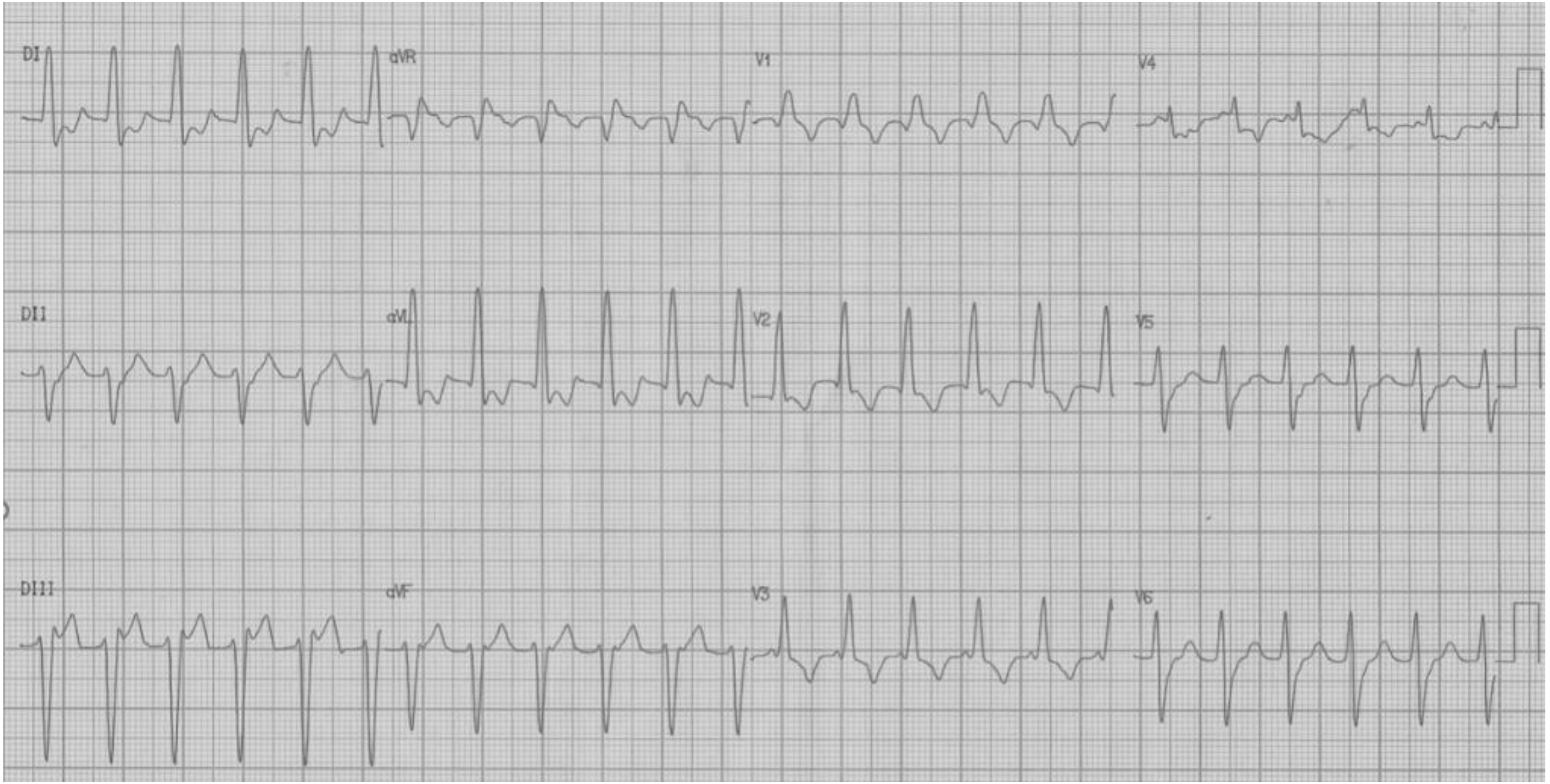
Raimundo Barbosa Barros MD Nick name “The fox”

Final comments by Andrés Ricardo Pérez-Riera MD PhD

Design of Studies and Scientific Writing Laboratory in the ABC Medicine Faculty, Santo André, São Paulo, Brazil.

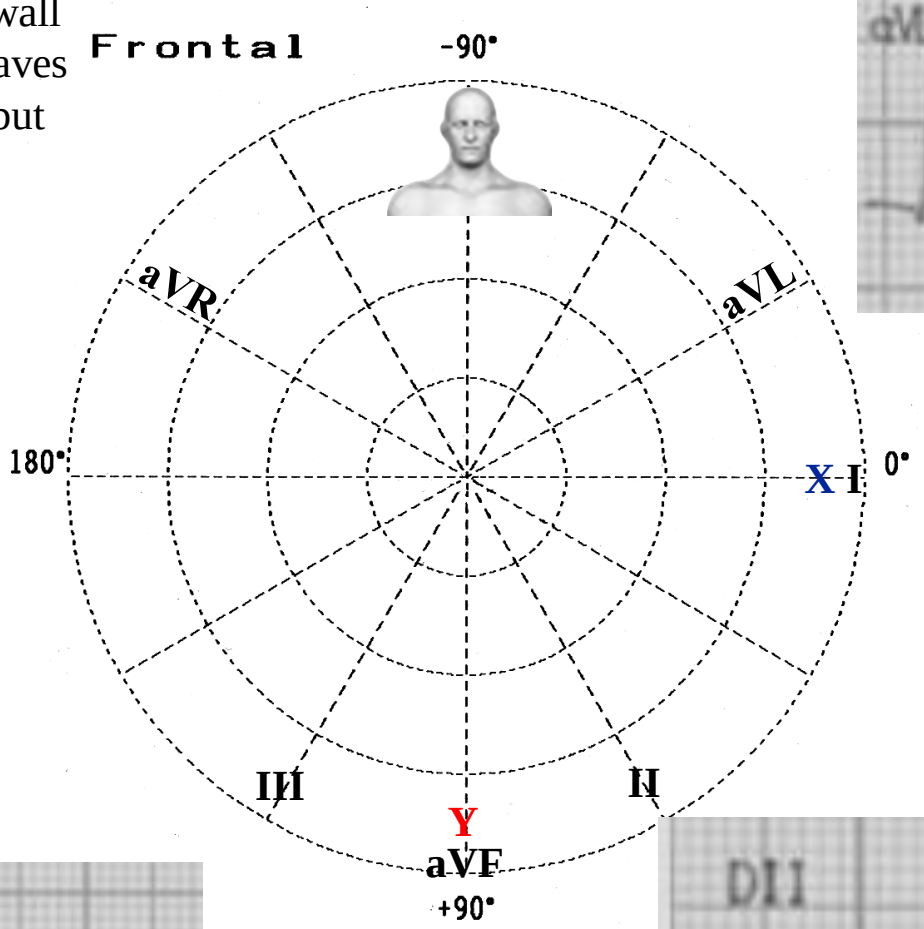
My cardiology site of scientific interests
https://ekgvcg.wordpress.com/

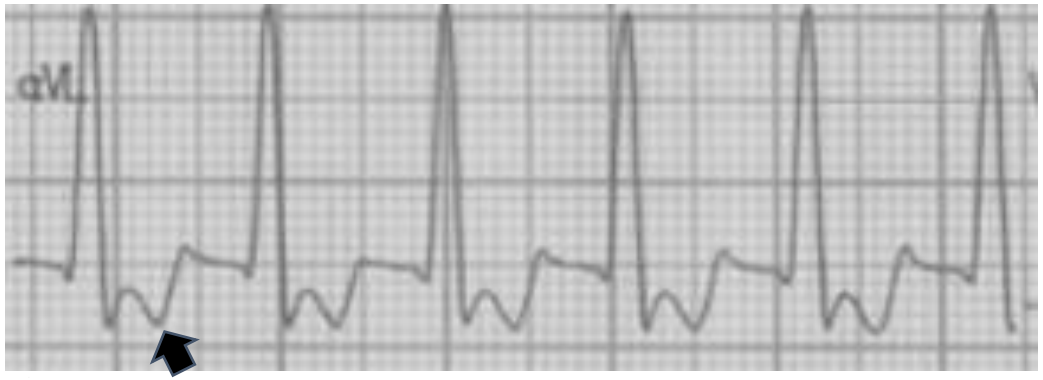
ECG-1-A

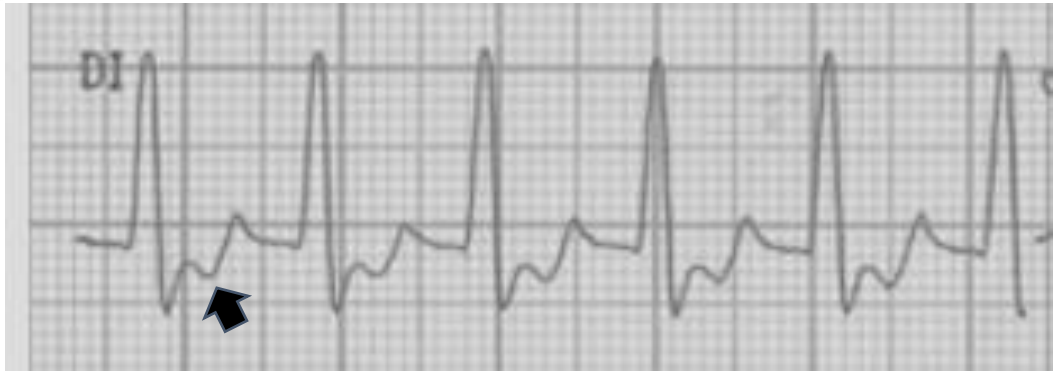


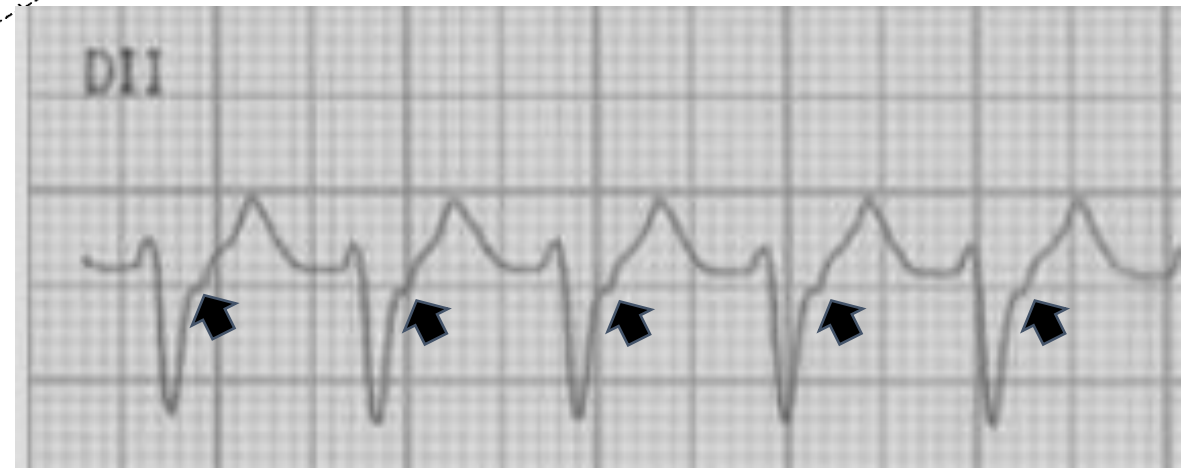
Wide QRS sustained tachycardia, HR 150bpm, RBBB–like configuration with LAFB pattern, negative P waves are seen in I, aVL, leads suggesting atrial tachycardia most likely originating from the left side: **focal left atrial tachycardia.** Probably with focus on lateral wall of the left atrium: negative P waves in the lateral leads and lead II, but positive in leads III and V1. Please see in the next slide……

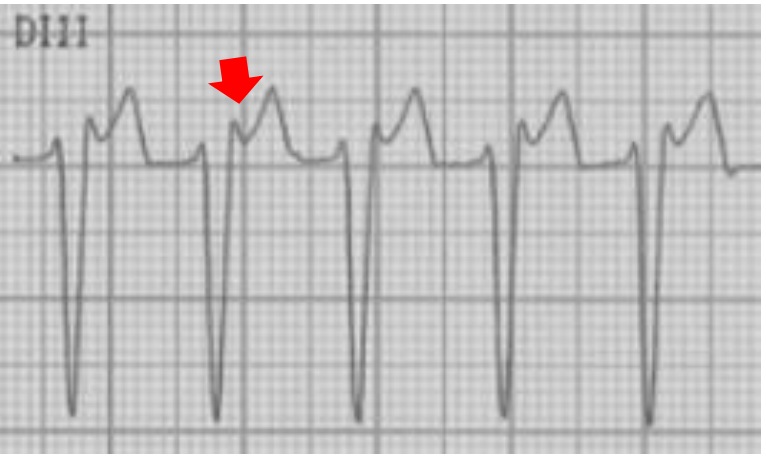
Focal left atrial tachycardia.
Probably with focus on lateral wall of the left atrium: negative P waves in the lateral leads and lead II, but positive in leads III and V1.









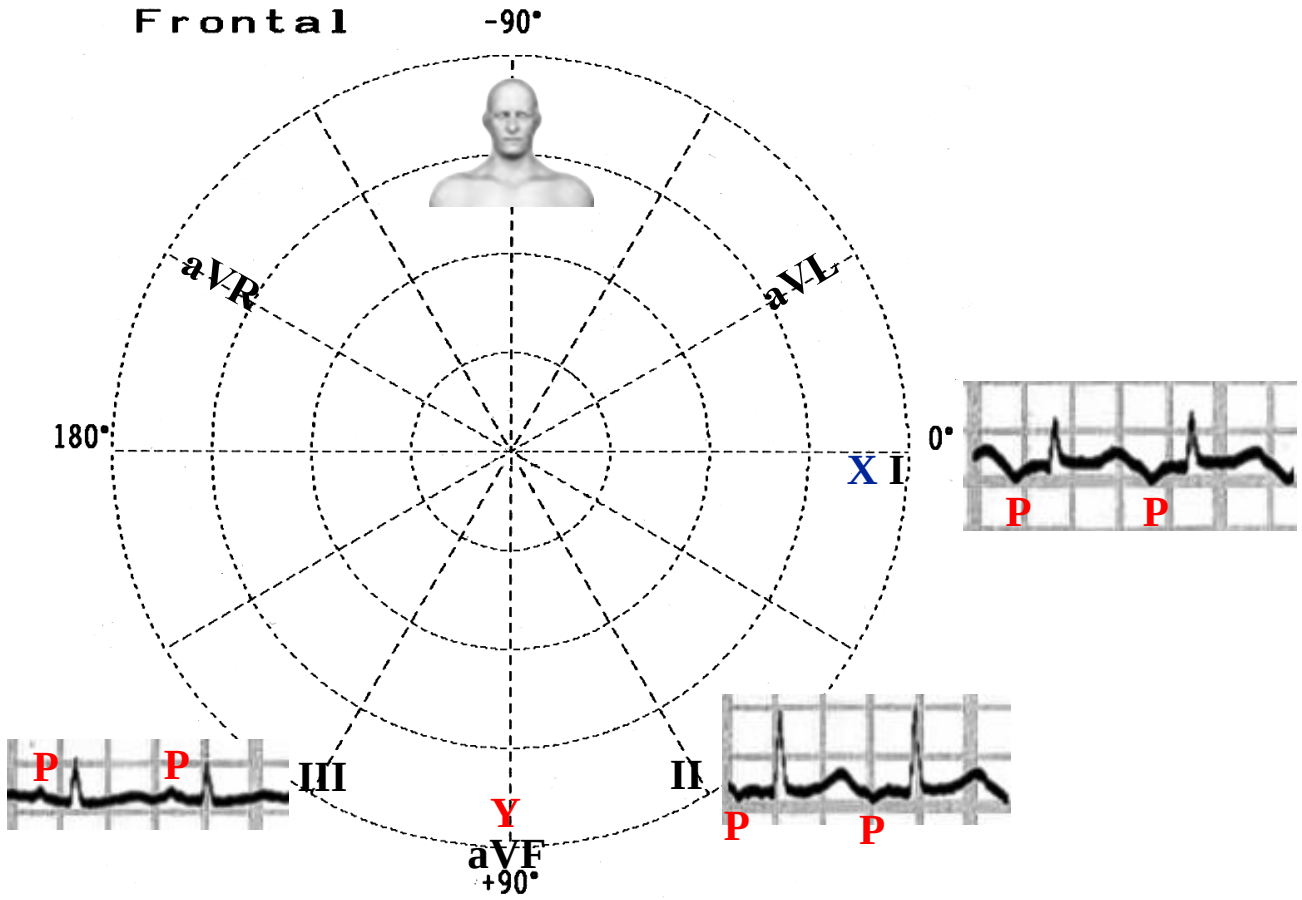


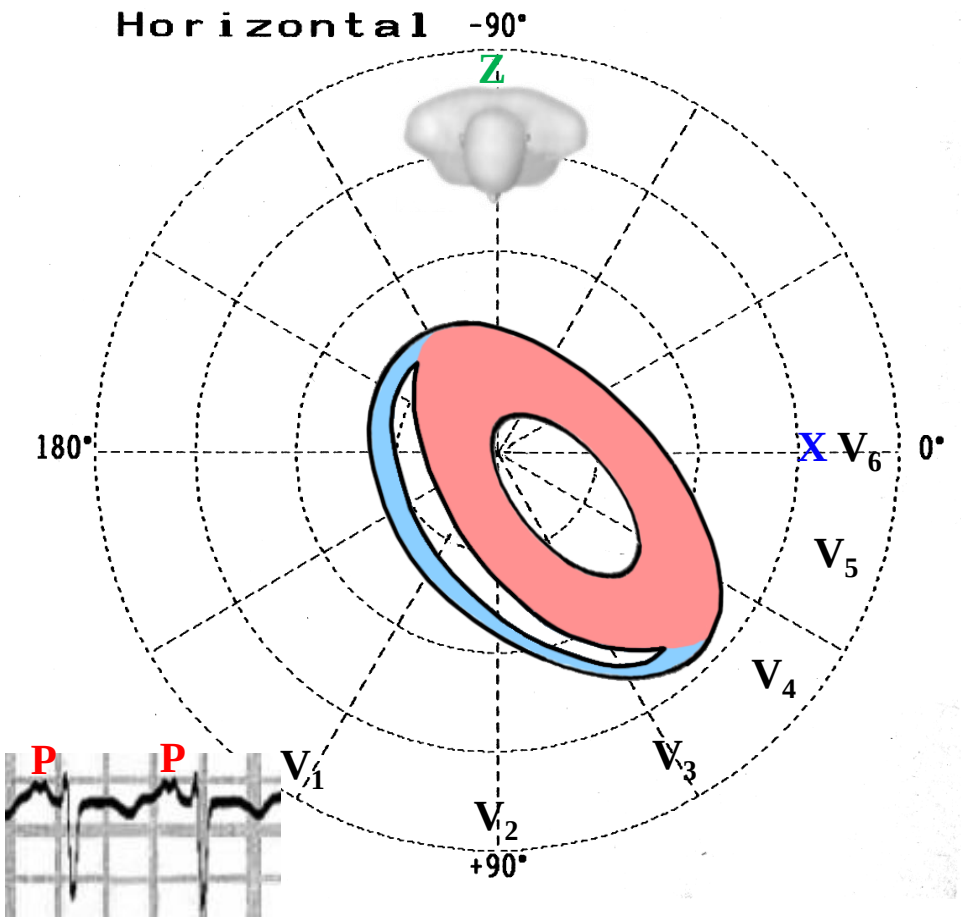
In 2001, the Joint Expert Group from the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (now called the Heart Rhythm Society) classified regular ATs according to electrophysiologic mechanisms and anatomy (**Saoudi N 2001**).

The Joint Expert Group defined focal AT as "being characterized by atrial activation starting rhythmically at a small area (focus) from where it spreads centrifugally. Frequent locations of such foci are the crista terminalis and the pulmonary veins" This definition indicates that the arrhythmia arises from an area that is smaller than would be required for classical macroreentry, which conventionally is considered to be a circuit greater than 2 cm in diameter. When a focus is located high in the crista terminalis, the atrial activation sequence will not be very different from that during sinus rhythm or inappropriate sinus tachycardia. Only sharp changes in rate with minor, but significant changes in origin of activation detected by endocardial mapping permit diagnosis (**Kalman JM1998)**. Multiple atrial tachycardia foci have been described, and can be a cause of recurrence after surgical or RFCA (**Garson A, 1990**). Available information suggests that focal activity can be due to automaticity, triggered activity (afterpotentials) or reentry (**Chen SA, 1994**). Atrial tachycardia cycle length is usually 250 ms. However, it can be as short as 200 ms. Over a prolonged period of observation (minutes to hours), atrial tachycardia cycle length can exhibit important variations. A progressive rate increase at tachycardia onset (warm up) (**Goldreyer BN, 1973**) and/or a progressive rate decrease before tachycardia termination (cool down) are suggestive of an automatic mechanism. Rate can increase during exercise (**Davis J, 1986**). Typically, adrenergic stimulation can accelerate the rate of focal discharge. Relatively small reentry circuits may resemble focal atrial tachycardia, especially if a limited number of endocardial recordings are collected.

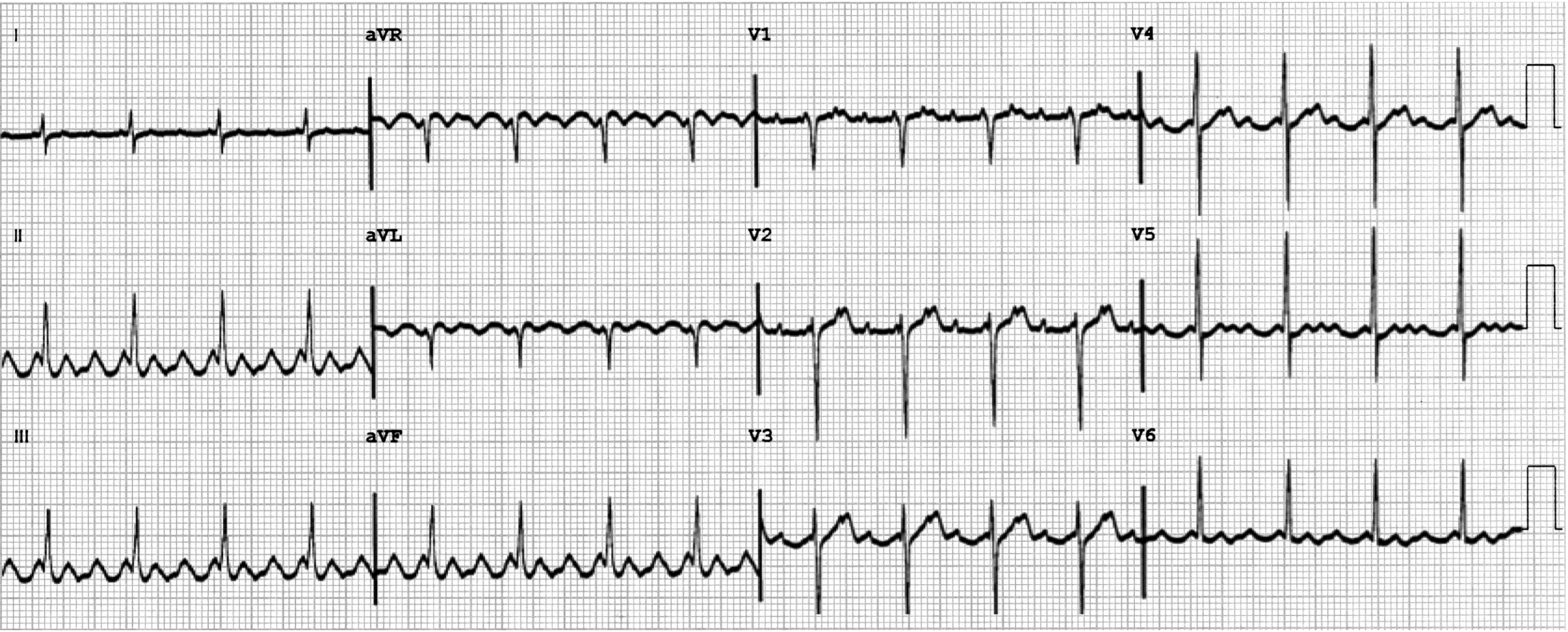
ECG pattern

Typically there are discrete P waves at rates 130–240 bpm, but possibly as low as 100 bpm or as high as 300 bpm (**Schamroth L. 1971**). There is a clearly defined isoelectric baseline between P waves in all leads (See next slide). P wave morphology will depend on focus location, and it can be used to approximately localize it before EPS (**Tang CW, 1995**). The multilead body surface potential recording can be used to help localize the site of origin of the tachycardia (**Tang CW, 1995**). During EPS, ventricular pacing may help by removing QRS complexes which are superimposed on atrial activity. Adenosine infusion to provide transient increased atrioventricular block can be used to obtain a clear view of the P wave, assuming that the tachycardia does not terminate. In the presence of rapid rates and/or intraatrial conduction disturbances, P waves can be very broad, and there may be no isoelectric baseline (Slide 14). In these cases, the ECG will show a pseudo atrial flutter pattern (continuous undulation without isoelectric baseline).



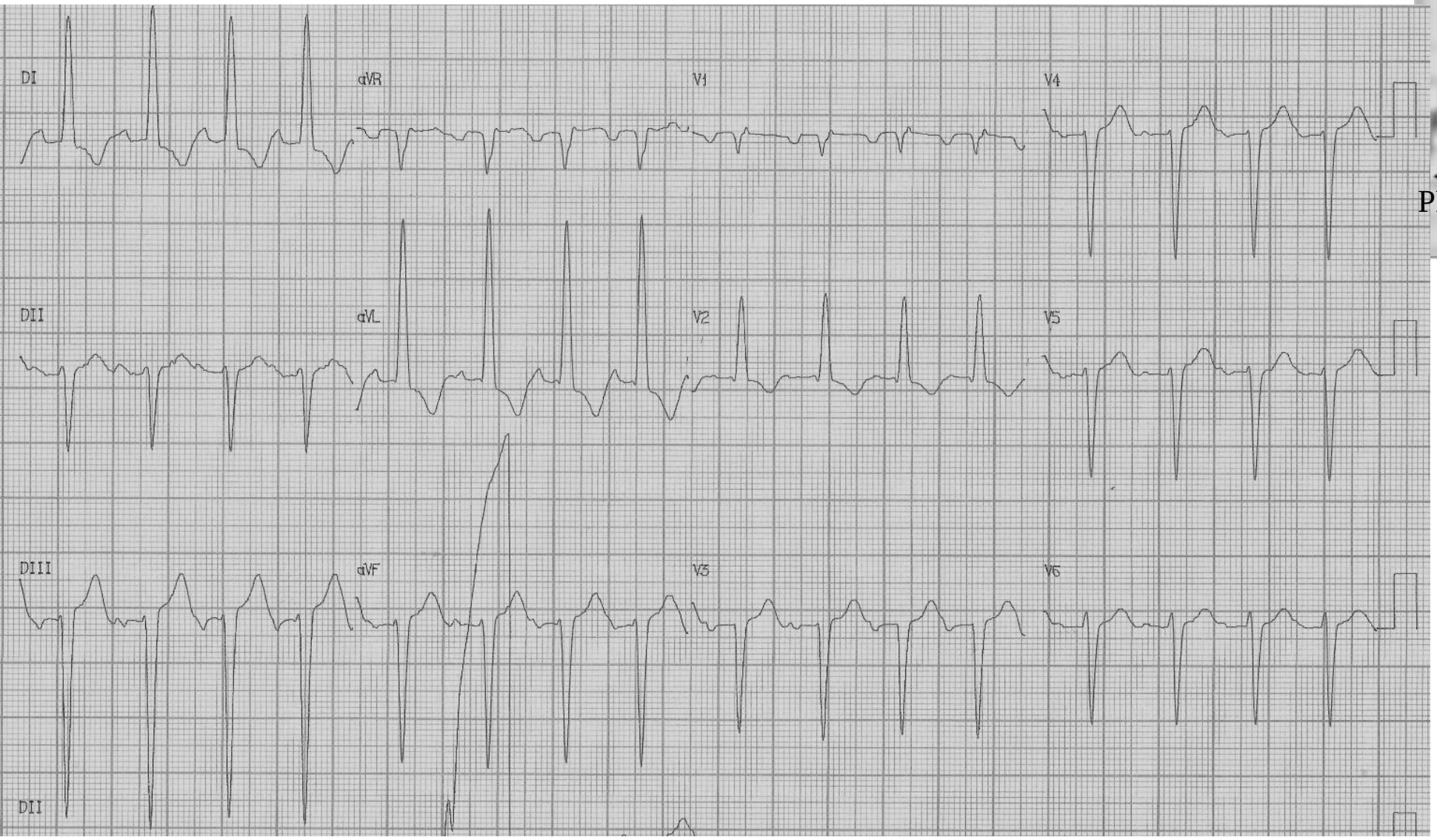


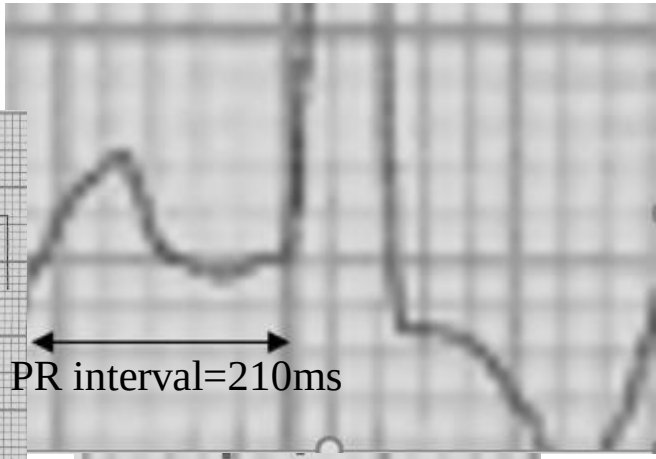
16-year-old girl, focal left atrial tachycardia with a cycle length of 480 ms originates in the lateral wall of the left atrium. P waves are negative in the lateral leads and lead II, but positive in leads III and V1.

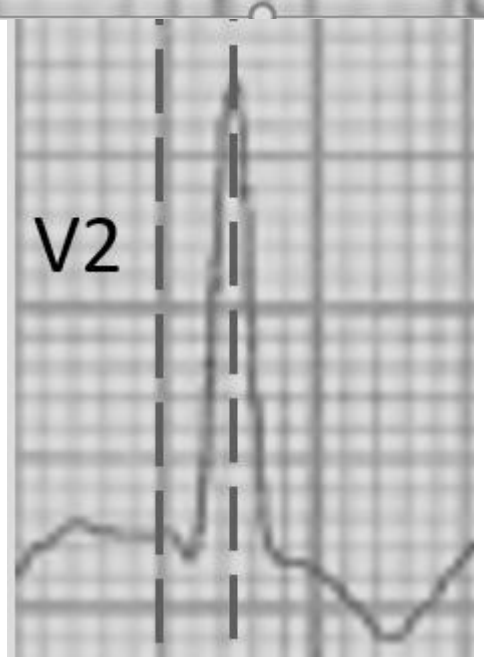


Focal atrial tachycardia presenting with an pseudo atrial flutter pattern. In this example of atrial tachycardia, the impulse originates in a left atrial focus, but due to the rapid rate of discharge (240 ms) and possible associated intra-atrial conduction disturbances, exhibits a pseudo atrial flutter pattern in the 12-lead ECG.

ECG-2-B



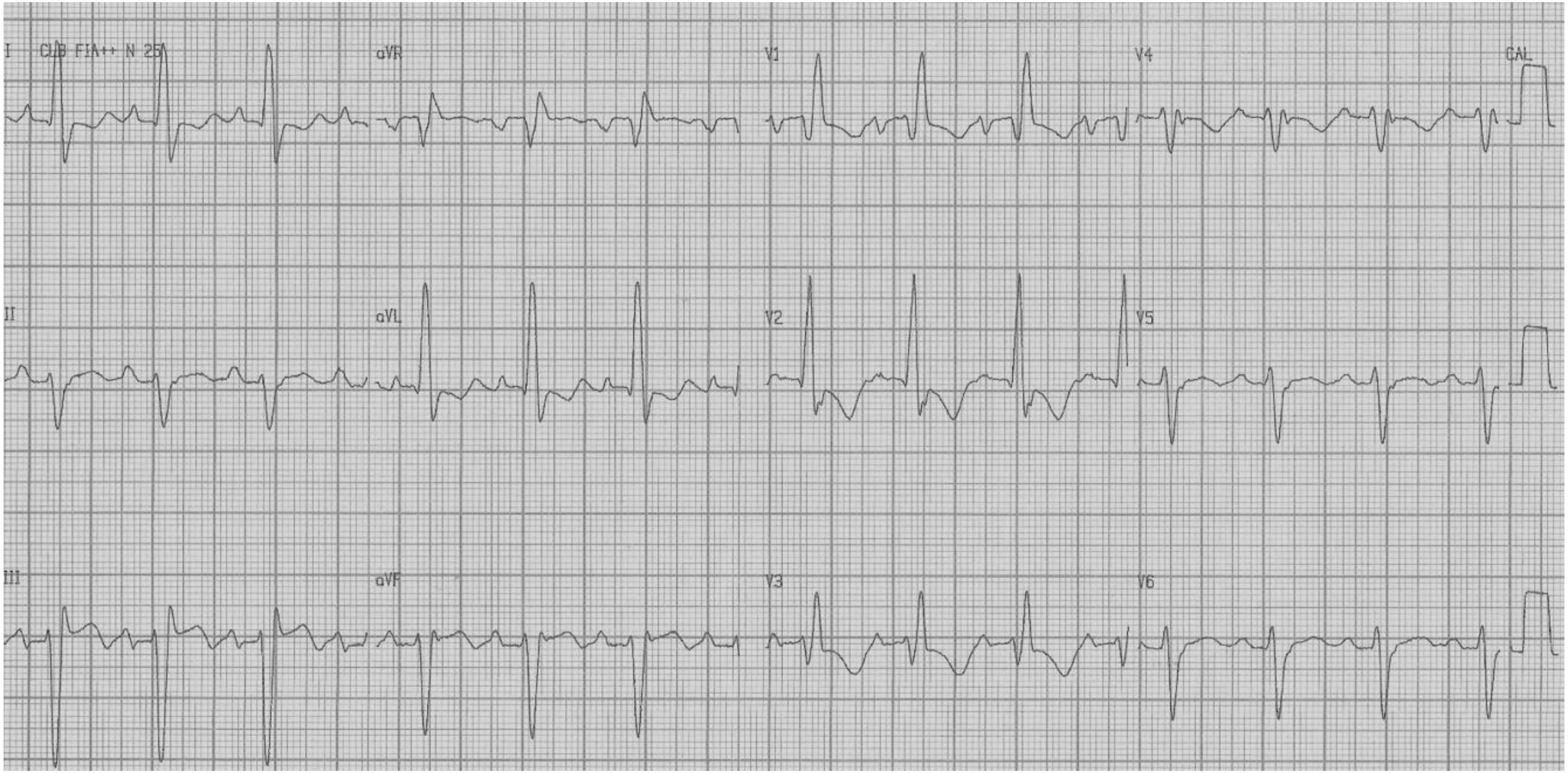




Prolonged R-wave peak time

Heart rate 107bpm: Sinus tachycardia, P-duration 120ms: LAE, PR interval=210ms: first degree AV block, LVH, LAFB Rosembaum type IV (SIII ≥ 15mm) + atypical LSFB: qR pattern in V2, prolonged R-wave peak time in V2 (>35 ms), absence of initial q wave in the left lateral leads I, aVL, V5-V6 consequence of absence of the first septal vector dependent of LSF, left bifascicular block: LAFB+LSFB.

ECG-3-C



Sinus rhythm, HR 87bpm, left atrial enlargement, left ventricular hypertrophy, Rosembaum type left anterior fascicular block IV (SIII>15mm), Right bundle branch block, prominent anterior QRS forces suggesting left septal fascicular block because QRS duration is wider related the second ECG and with initial q wave in V1-V2. BB question: I do not understand why CRBBB is present in **Tracing C** (sinus 80/min) but not in **Tracing B** (sinus 100/min). **Answer BB question:** because in trace B-2 we have only LAFB+LSFB on the other hand in tracing C-3 we have **RBBB + LSFB+LAFB.**

Important clues from the history and physical examination for the differential diagnosis between VT and SVT-A

History: VT is the most common cause of wide QRS tachycardias, consisting of 80% of all cases (**Akhtar M 1988**). A history of previous myocardial infarction, angina, or congestive heart failure has a high positive predictive value of 95% (**Baerman JM 1987**) for the diagnosis of VT. Advanced heart disease (e.g., coronary heart disease) statistically favors VT

Physical examination: The following physical findings are suggestive of AV dissociation:

I) Irregular large ‘a’ waves in the jugular venous(JV) pulse, called ‘cannon A waves’, indicate atrial contractions occurring at times when the tricuspid atrioventricular valve is still closed due to the dissociated ventricular contractions (**Colman AL 1966**). Cannon 'a' waves in the jugular venous pulse suggests VT with AV dissociation. With AV dissociation these giant a-waves occur irregularly.

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 vary 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 atrial fibrillation, can also vary the intensity of S1.

III) Beat by beat variations in systolic blood pressure unrelated to breathing caused by variable left ventricular 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 tachycardias are grouped as stable or unstable. Important indicators of instability include:
Hypotension, Syncope, Precordial chest discomfort and 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.

Wide complex tachycardia: VT versus SVT with aberrance (SVT-A)

During wide complex tachycardia (HR> 100/min, QRS ≥ 100 ms) the differentiation between VT from SVT-A origin of the arrhythmia is important to guide therapy. Several algorithms have been developed to aid in this differentiation. It is important to keep in mind that a good estimate of VT *versus* SVT can be made based on the clinical vignette: Older patient with previous myocardial infarction = most likely VT. On the other hand, younger patient with known paroxysmal tachycardias and who is hemodynamically stable = most like SVT.

- Wide QRS tachycardia (WQRST): A name given to any ECG arrhythmic event with HR ≥ 100 bpm and QRSd ≥ 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 HR of ≥ 100 bpm originating proximal to the His bundle bifurcation.

Possible causes of Wide Complex QRS Tachycardia

- Ventricular Tachycardia VT: > 80% of cases
- SVT with aberration (SVT-A) right or left bundle aberration conduction
- SVT with preexcitation (WPW-S): accessory pathway conduction. Preexcited SVT: 1% a 5% cases.
  - Atrial fibrillation with one or multiple Accessory Pathway.
  - 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).
- SVT with baseline preexisting BBB. Due to abnormal muscle-to- muscle spread of impulse.
- SVT with hyperkalemia other electrolytes disturbances or drugs such as IA and IC antiarhythmic.
- 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.

Differential diagnosis of supraventricular tachycardia (SVT) from ventricular tachycardia (VT) is of paramount importance for appropriate patient management. Several diagnostic algorithms for discrimination of VT and SVT based on surface 12 lead electrocardiogram (ECG) analysis have been proposed. Following established diagnosis of VT, a specific origination tachycardia site can be supposed according to QRS complex characteristics. Electrocardiographic diagnosis and localization of cardiac tachyarrhythmia origin are sometimes challenging. Several algorithms for differentiation of SVT from VT have been proposed. ECG characteristics of ectopic QRS complexes have also been characterized in order to define their origin in the heart ventricles. Based on surface ECG analysis it is possible to perform relatively accurate differential diagnosis between SVT and VT. Contemporary algorithms of VT discrimination based on ECG analysis, and characteristics which help to understand an origination/exit site of VT or PVC. The term "tachycardia" indicates heart rate≥100 beats per minute (**Blomström-Lundqvist C 2003**). A tachycardia can be classified as wide-complex (QRS ≥120 ms) and narrow-complex (QRS < 120 ms). SVT has its origin in the atria or in the atrioventricular (AV) node while VT is originated in the ventricular myocardium or in the conduction system below the AV node. Wide complex tachycardia (WCT) can be VT (80%), SVT (15%) with right or left bundle branch aberration (either atrial flutter, focal atrial tachycardia, AV nodal reciprocating tachycardia, reciprocating tachycardia utilizing an accessory pathway) (**Katritsis DG 2017**). When a narrow QRS tachycardia is seen on ECG, it is usually SVT. In rare cases VT originating from the conduction system (fascicular tachycardia, interfascicular tachycardia) can be characterized by QRS ≤ 120 ms (**Cohen HC 1972**). The main challenge is encountered when a tachycardia is regular, with wide QRS, and no AV dissociation can be identified. Discrimination of WCT has an important role due to its clinical significance (**Lebedev DS 1998**).

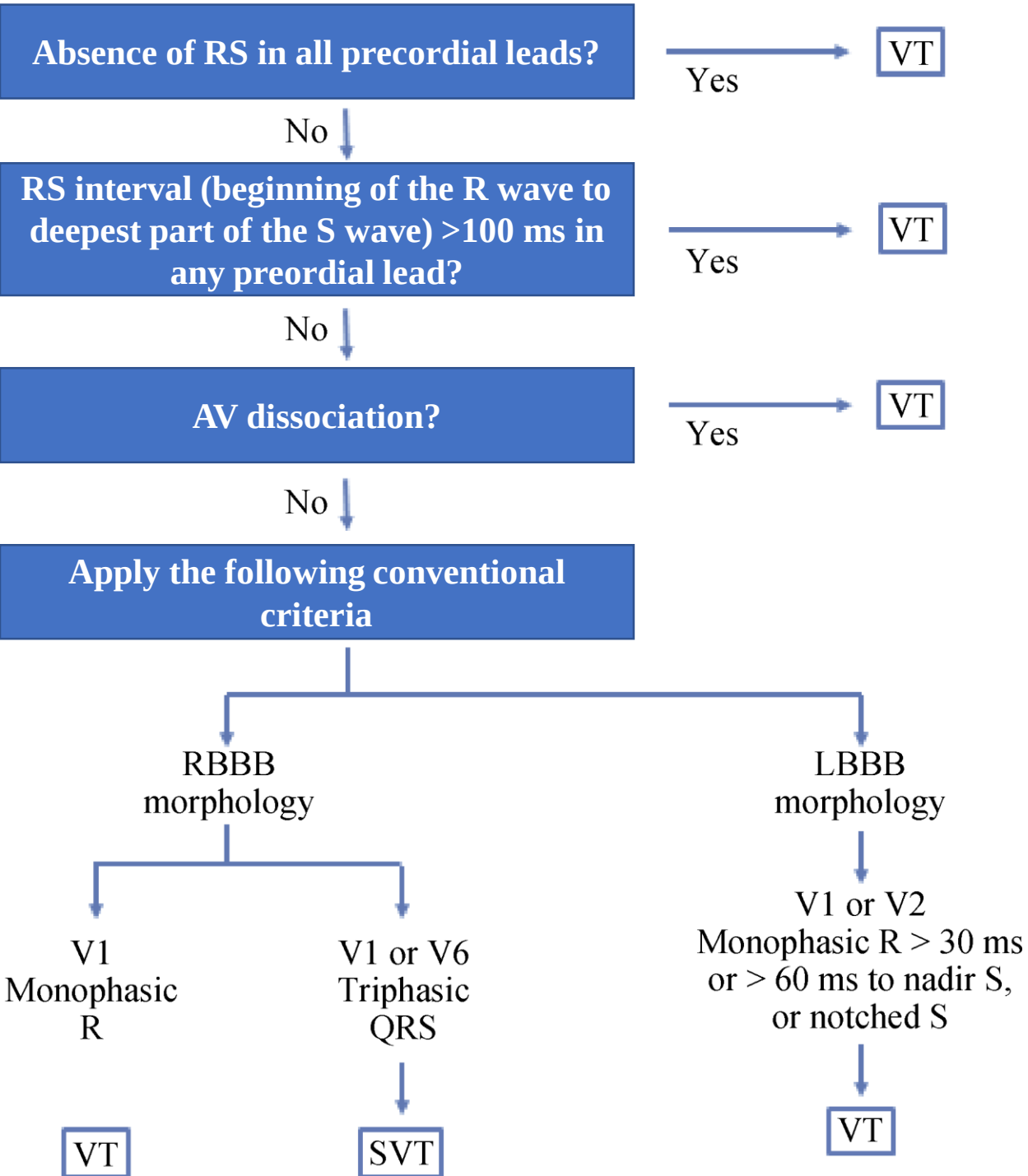
Brugada algorithm

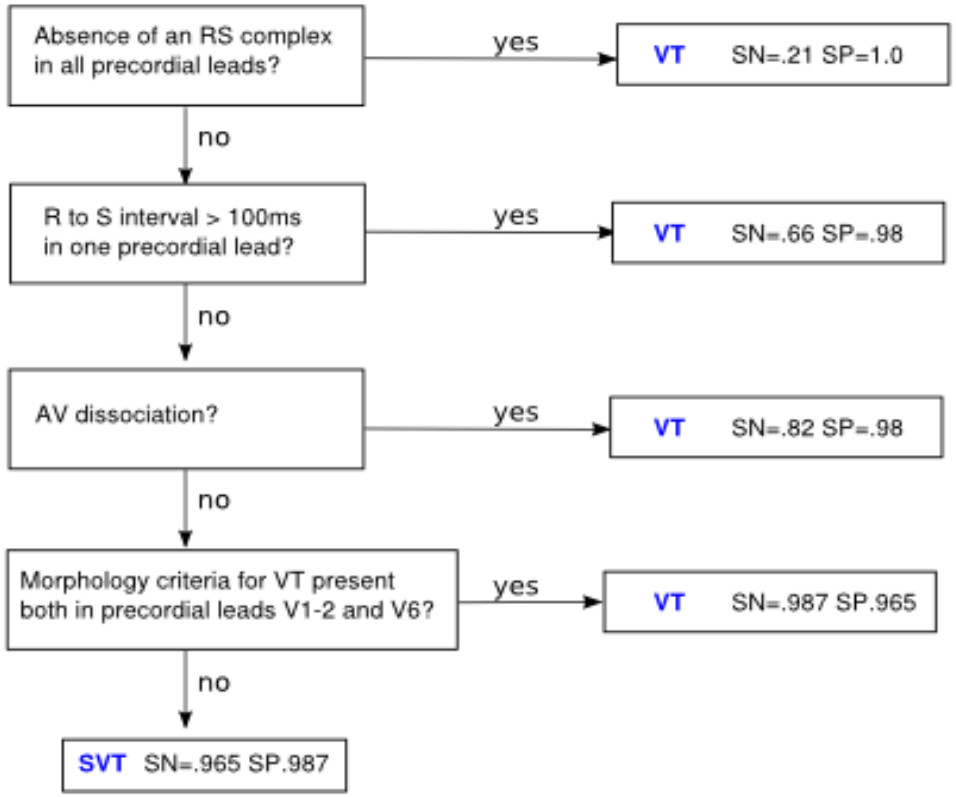
Professor Pedro Brugada et al presented systemic analysis of numerous ECGs and proposed an algorithm based on 4 steps that could relatively accurate diagnose a VT (See algorithm in the next slide):

1. Absence of an RS complex in all precordial leads;
2. An RS complex in any precordial lead with an RS interval >100 ms;
3. AV dissociation; and
4. Morphological criteria for VT present in both leads V1 and V6 (Figure 2).

In this study, a total of 554 ECGs with WCTs were analyzed by two independent observers that reported 384 VT and 170 SVT, VT was diagnosed when any criterion was present. In the case that the first step was absent, the rest of the criteria were evaluated, and if any of them was not present, SVT with aberrant conduction was diagnosed by exclusion.

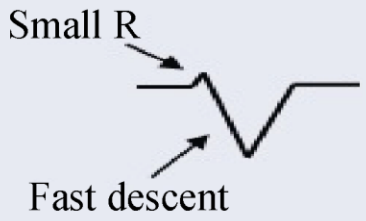
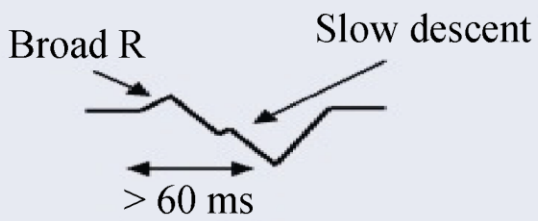
The sensitivity of these four steps criteria for the diagnosis of VT was 98.7%, and the specificity was 96.5% (**Brugada P 1991**).



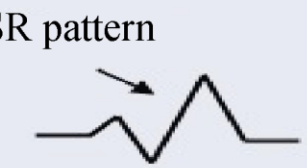
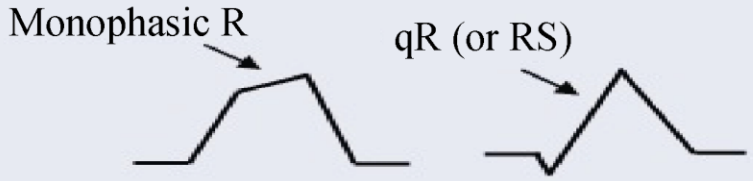
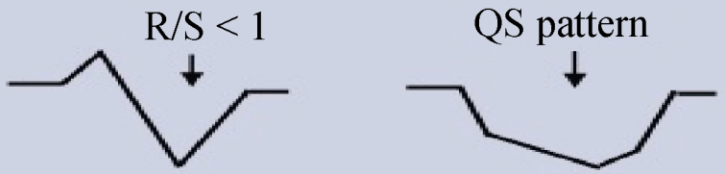


Brugada algorithm for the differential diagnosis of WCTs
With modifications from Brugada, *et al.*(**Brugada P 1991**) AV: atrioventricular; LBBB: left bundle branch block; RBBB: right bundle branch block; SVT: supraventricular tachycardia; VT: ventricular tachycardia; WCTs: wide complex tachycardias.

Tachycardia with a LBBB-like QRS

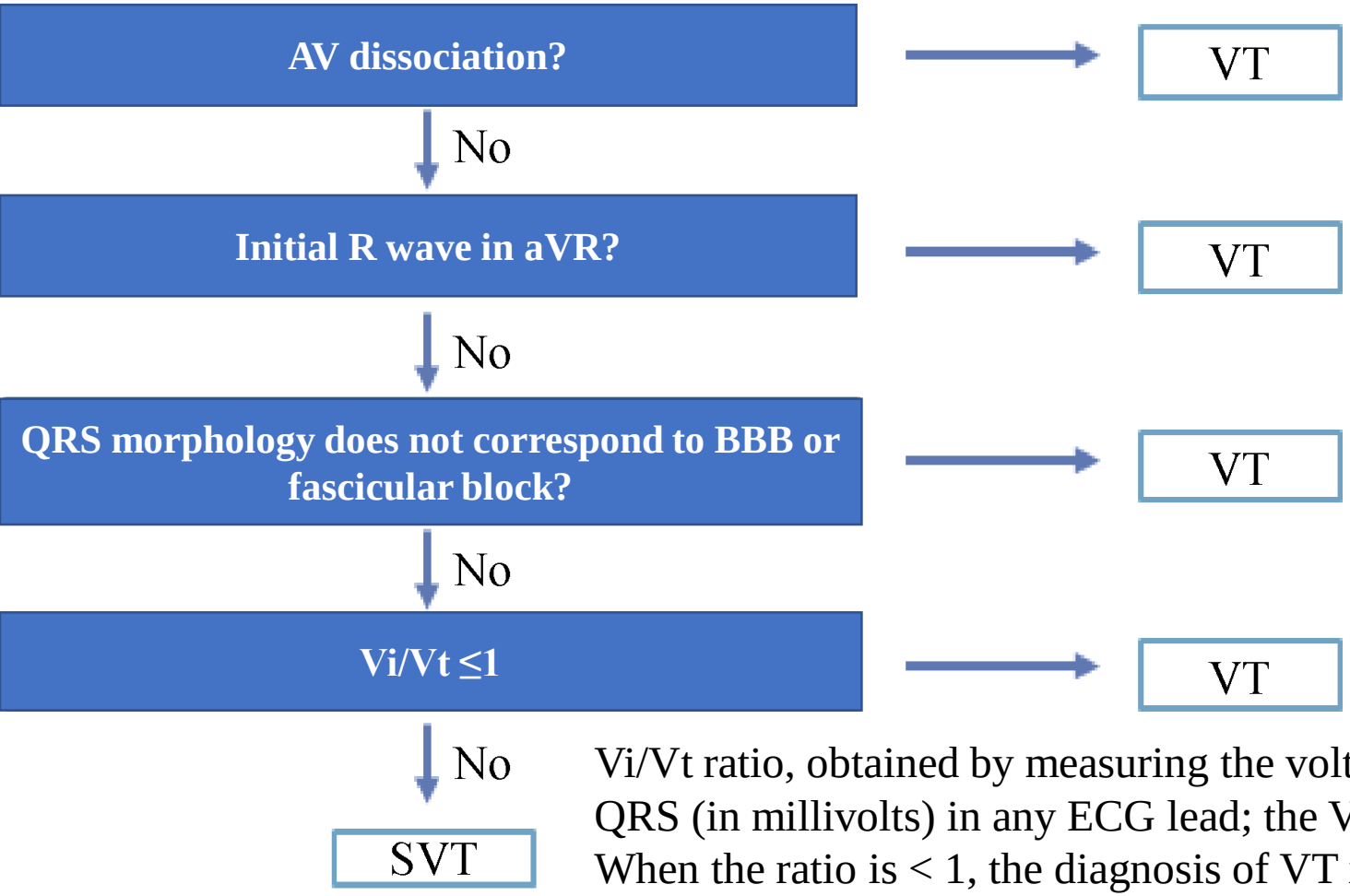
Lead		SVT	VT
Lead V_1 or V_2			
R > 30 m sec.	VT		
> 60 m sec to nadir of S	VT		
Notched S	SVT		
			
Lead V_6			
QR or RS	VT		
Monophasic R	SVT		

Tachycardia with a RBBB-like QRS

Lead		SVT	VT
Lead V_1			
Monophasic R	VT		
QR or RS	VT		
Triphasic	SVT		
			
Lead V_6			
R to S ratio < 1	VT		
QR or RS	VT		
Monophasic R	VT		
Triphasic	SVT		
R to S ratio > 1	SVT		
			

Morphological criteria for diagnosis of VTs used by Brugada, *et al*. (**Brugada P 1991**). With modifications from **Eckardt L et al.2006**,
LBBB: left bundle branch block; RBBB: right bundle branch block; SVT: supraventricular tachycardia; VT: ventricular tachycardia.

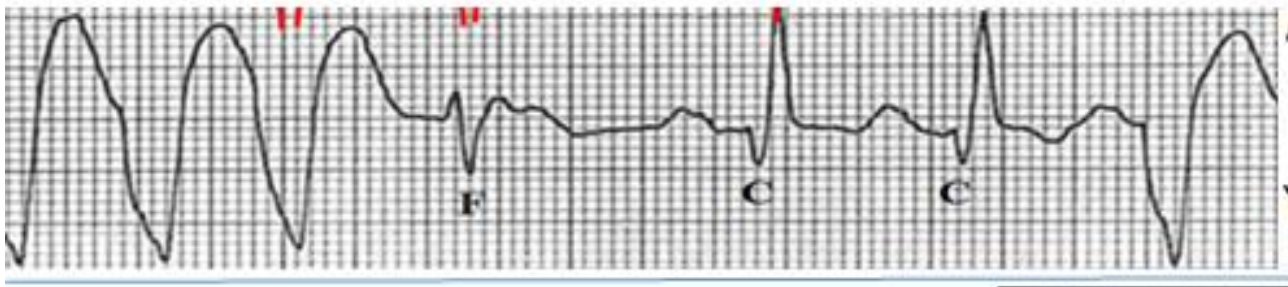
Vereckei algorithm Vereckei, et al. (**Vereckei A 2007**) proposed a new simplified algorithm. The algorithm comprised the following four steps



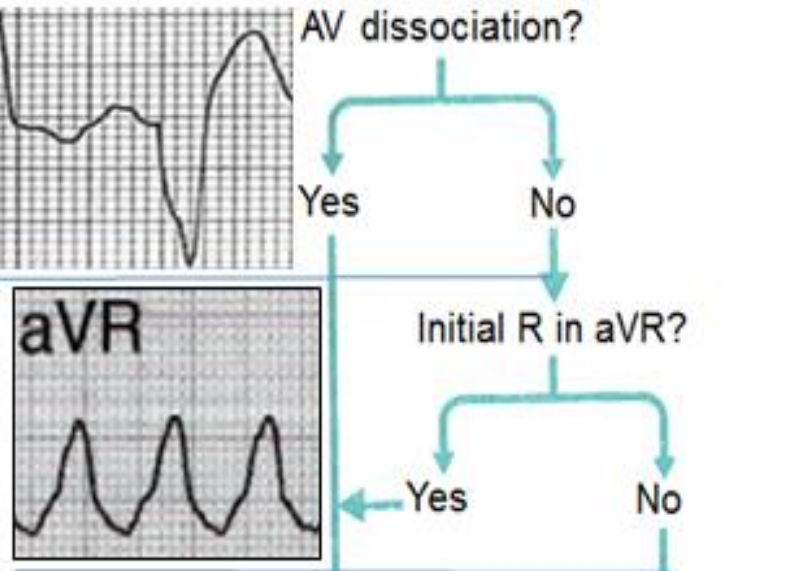
If any criterion is present the analysis is terminated and VT is diagnosed, if no criteria are found SVT is diagnosed. Vereckei, et al. (**Vereckei A 2007**) reported sensitivity 95.7% for VT diagnosis and specificity 72.4%, even though limitations were present in this algorithm unable to recognize certain WCT (bundle branch re-entry VT, fascicular VT, and SVT involving an atriofascicular accessory pathway are associated with typical BBB pattern indistinguishable from that associated with SVT with functional aberrancy or pre-existent BBB, unless A-V dissociation is present).

Vi/Vt ratio, obtained by measuring the voltage of the initial 40 ms (Vi) and the terminal 40 ms of a QRS (in millivolts) in any ECG lead; the Vi/Vt ratio represents ventricular activation velocity. When the ratio is < 1, the diagnosis of VT is made. If the Vi/Vt is > 1, the diagnosis is SVT

Vereckei algorithm for the differential diagnosis of WCT. AV: atrioventricular; BBB: bundle branch block; SVT: supraventricular tachycardia; VT: ventricular tachycardia; WCT: wide complex tachycardia.

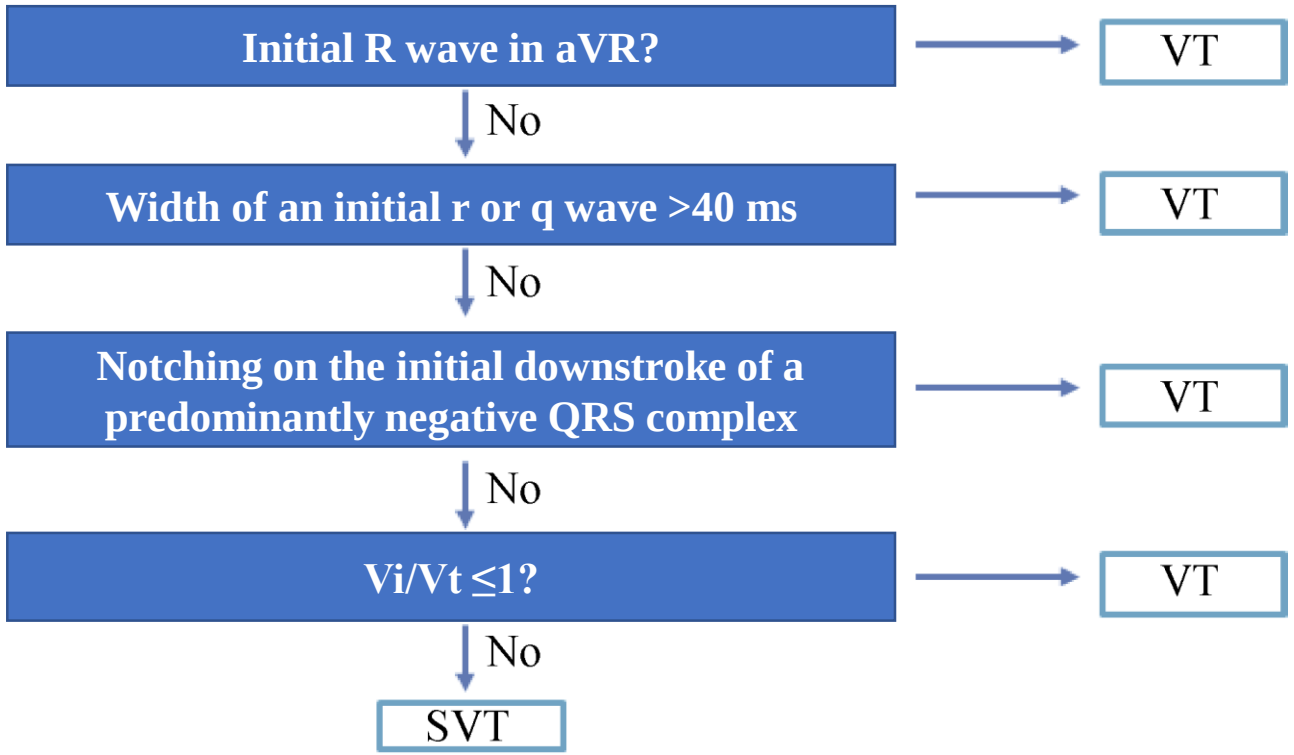


F: fusion beat, C: Capture beat

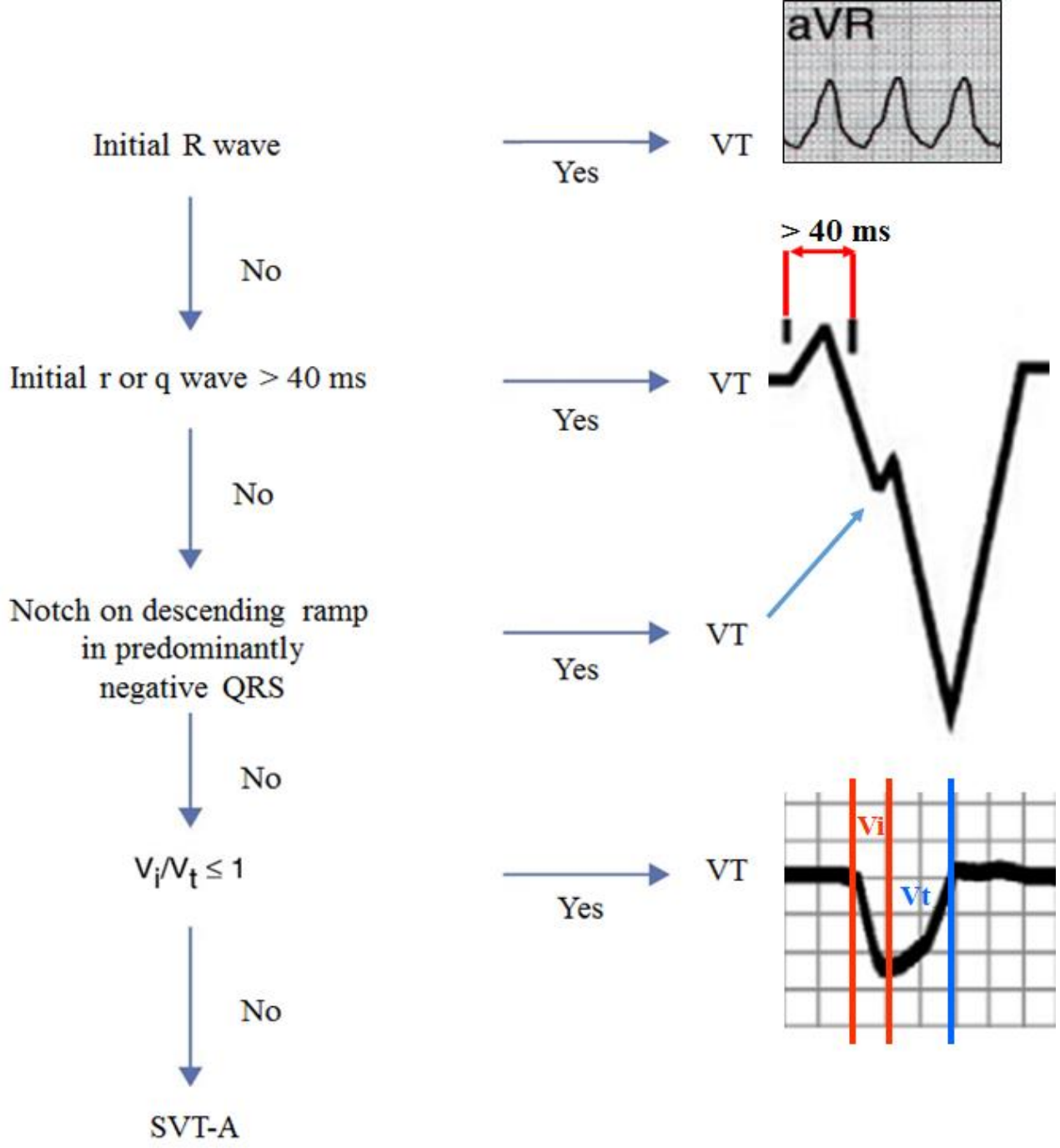


Vi/Vt ratio, obtained by measuring the voltage of the initial 40 ms (Vi) and the terminal 40 ms of a QRS (in millivolts) in any ECG lead; the Vi/Vt ratio represents ventricular activation velocity. When the ratio is < 1, the diagnosis of VT is made. If the Vi/Vt is > 1, the diagnosis is SVT

New aVR lead algorithm. In order to further simplify VT diagnosis, Vereckei, et al. (**Vereckei A 2008**) proposed another algorithm based on the direction and velocity of initial and terminal ventricular activation in the aVR lead. The QRS morphological criteria were completely eliminated and only the aVR lead was evaluated, this diagnostic method was also based on four steps (Figure below): (1) initial R-wave in aVR; (2) initial r- or q-wave with width >40 ms; (3) notching on the descending limb of a negative onset, predominantly negative QRS complex; and (4) Vi/Vt ratio, obtained by measuring the voltage of the initial 40 ms (Vi) and the terminal 40 ms of a QRS (in millivolts) in the aVR lead. When the ratio is < 1, the diagnosis of VT is made. If the Vi/Vt is > 1, the diagnosis is SVT (Figure below). If any criterion is present the analysis is terminated and VT is diagnosed if no criteria are found SVT is diagnosed. Vereckei, *et al*. (**Vereckei A 2008**) reported sensitivity 96.5% and specificity 75% for VT diagnosis, and suggested that this single lead algorithm had the advantage of being a suitable tool for emergency situations in which a quick diagnosis is required, even though the required time for its evaluation was not compared with the previous algorithms. Factors that may influence the Vi/Vt ratio like anteroseptal myocardial infarction, scar at a late activated ventricular site, fascicular VT, and VT exit site close to the His-Purkinje system were described as limitations. Another limitation was the limited number of VTs occurring in the absence of structural heart disease that can be confused with SVT, these VTs present a narrower QRS complex compared with QRS complexes in patients with CAD and can be confused with SVT when other algorithms are applied.



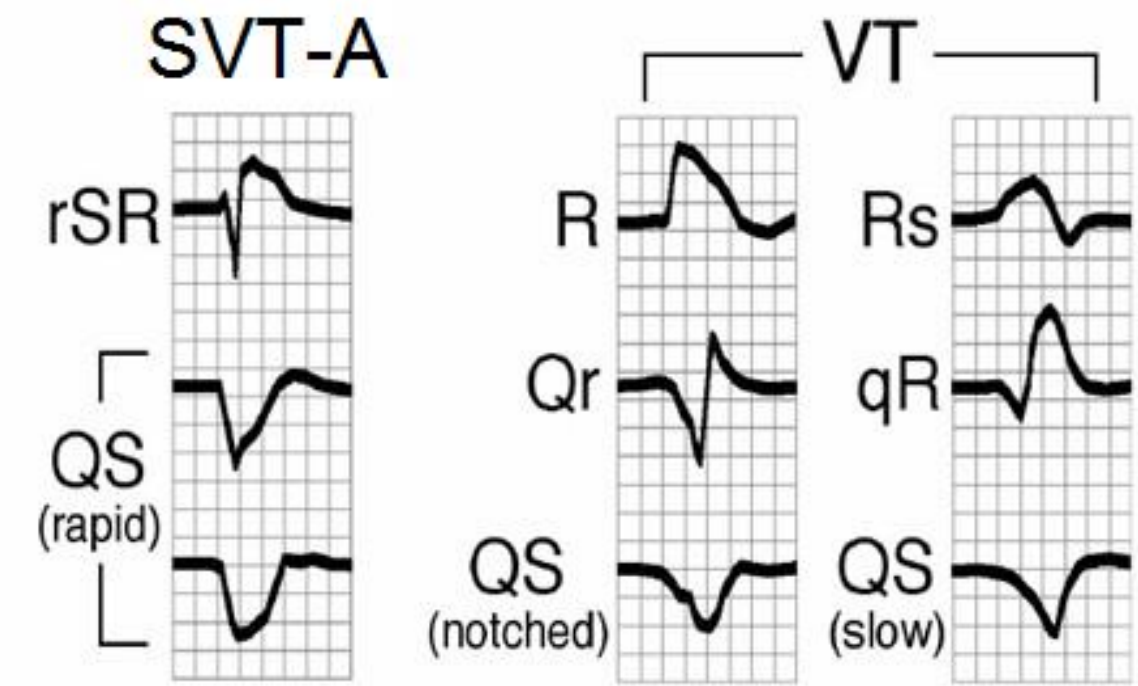
Vereckei algorithm for the differential diagnosis of WQRS tachycardia based on the aVR lead. With modifications from Vereckei, *et al*. (**Vereckei A 2008**)
SVT: supraventricular tachycardia; VT: ventricular tachycardia.



The new 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.

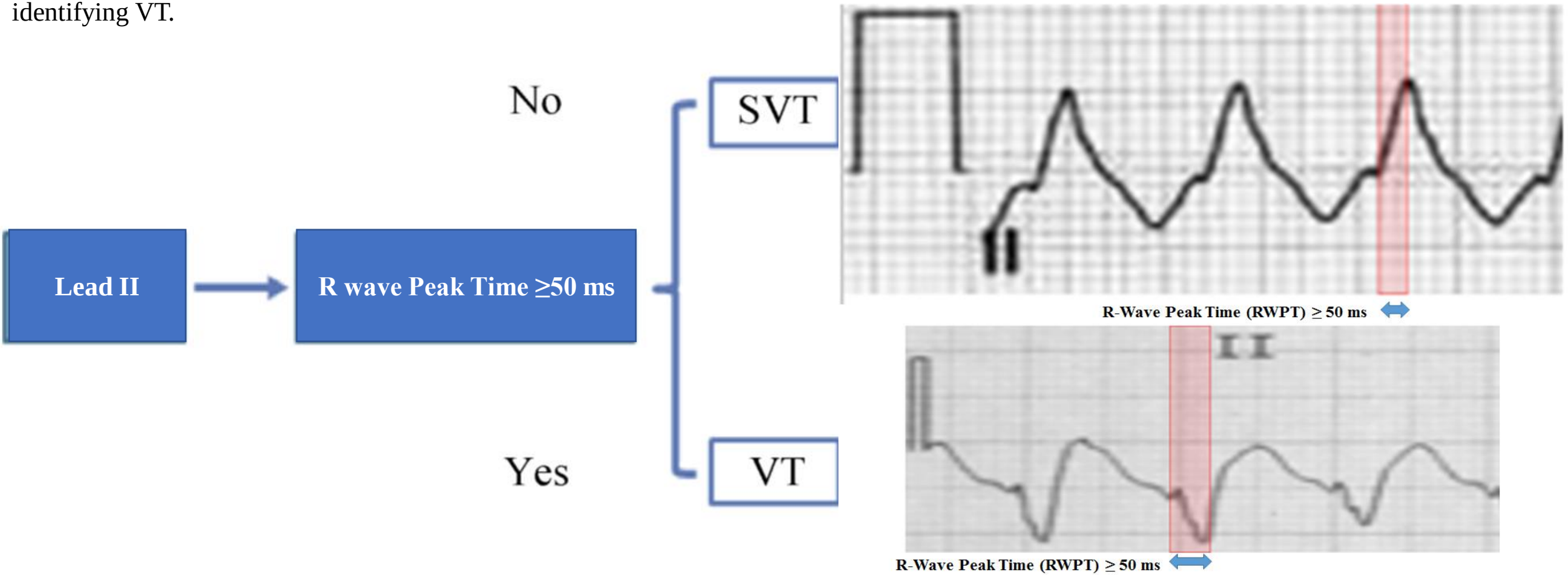
The newer Vereckei algorithm also has of a sequence of four steps but only uses a single lead (aVR) for the analysis



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

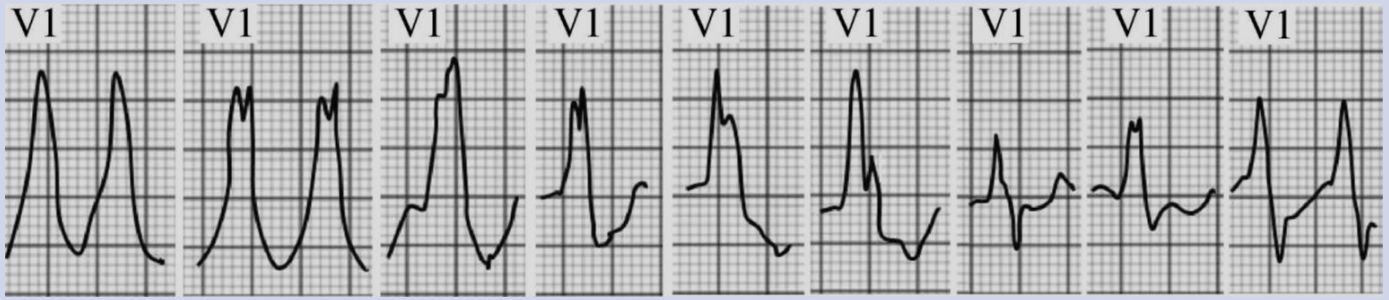
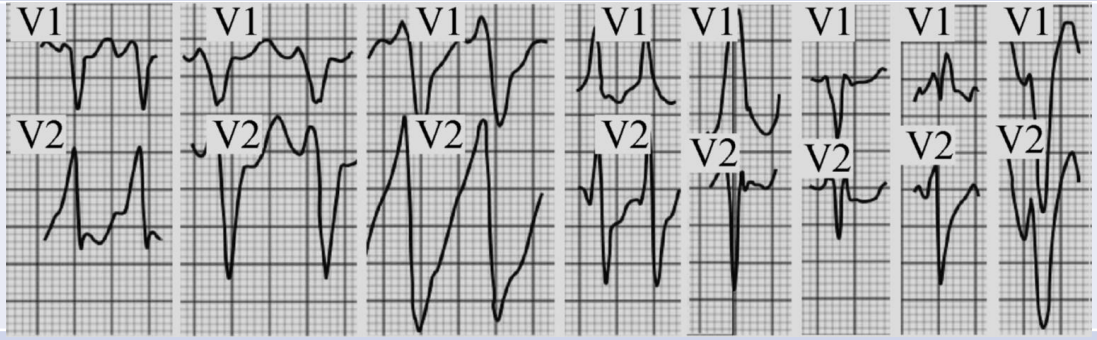
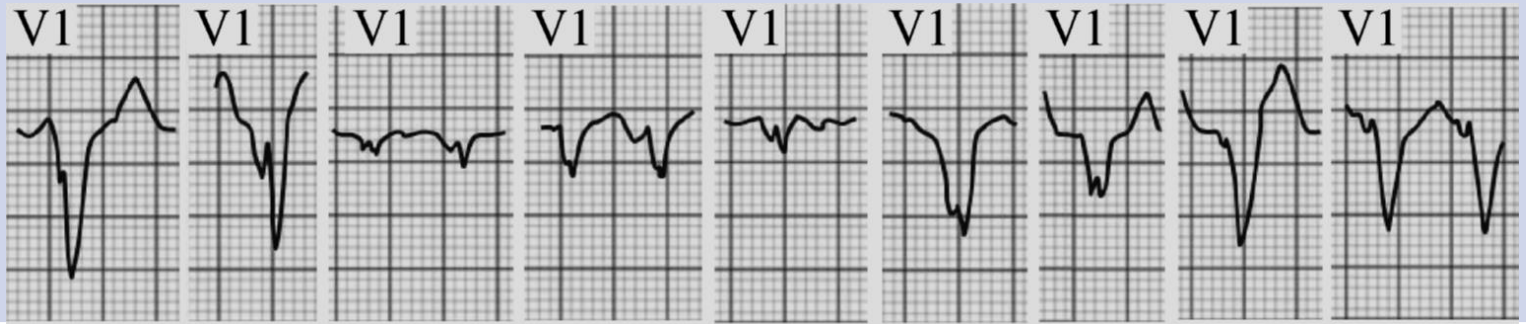
R-Wave Peak Time (RWPT) ≥ 50 ms in lead II, as a single criterion for VT-Pava's criterion

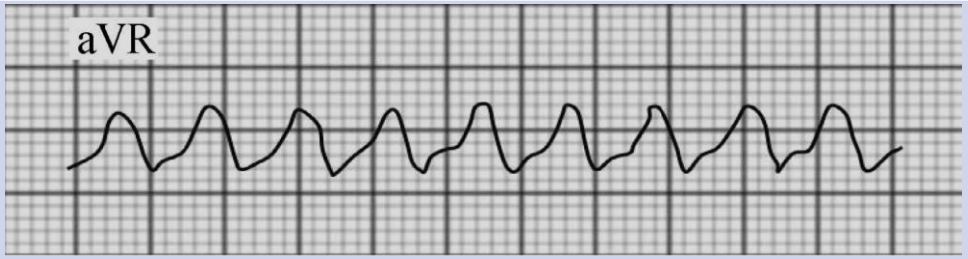
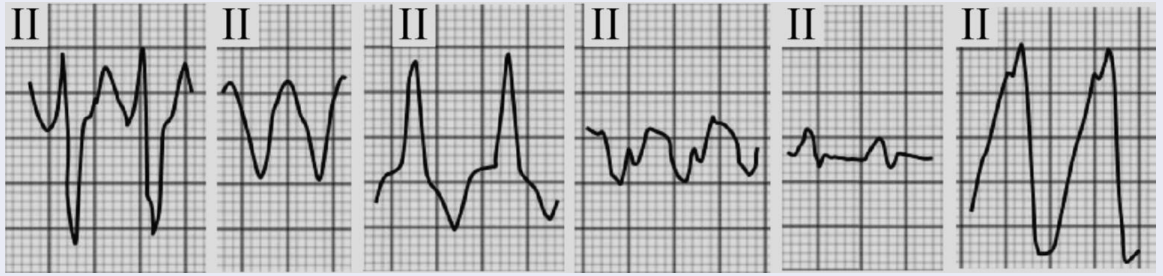
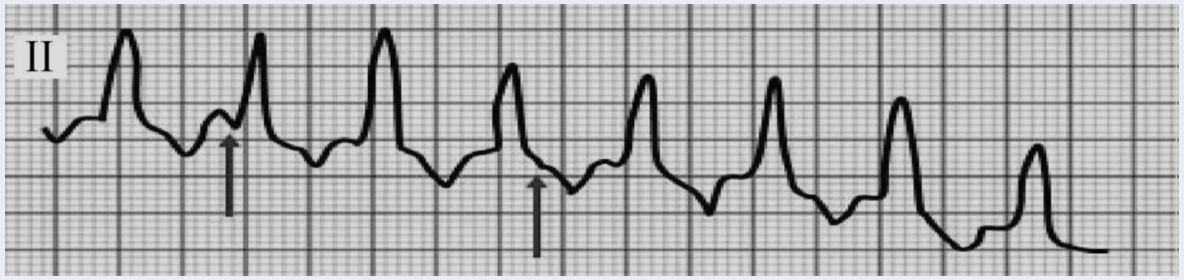
Pava, et al. proposed a VT criterion based on the analysis of the lead II (Figure bellow), considering that this lead is usually present on ECGs registered during acute clinical situations. The criterion is applied by measuring the interval from the QRS onset to the peak of the R-wave. In this study, R-wave peak time (RWPT) of ≥ 50 ms in lead II had a sensitivity of 93%, specificity of 99% and positive predictive value of 98% in identifying VT.



Measurement of the RWPT in lead II. RWPT measured from the "isoelectric" line to the point of first change in polarity, was >50 ms (80 ms), with modifications from Pava, *et al.* (**Pava LF 2010**). RWPT: R-wave peak time; SVT: supraventricular tachycardia; VT: ventricular tachycardia.This criterion has not been compared with the previously described algorithms, the EPS was used as a gold standard to determine if VT or SVT was present. Difficult determination of QRS onset during fast VT is a limitation of this method, this method still waits for its validation by other researchers (**Pava LF 2010**).

VT score-criteria for the diagnosis of VT. 2.5 VT score algorithm Jastrzebski et al propose a VT score algorithm for the differentiation between VT and SVT. This novel score was based on seven characteristics and showed high specificity for VT diagnosis (**Jastrzebski M 2016**). Points attributable to each characteristic are summarized, and the resultant number represents a total score. A VT score ≥ 1 is considered diagnostic for VT. A VT score = 0 indicates SVT diagnosis. A VT score ≥ 3 indicates VT with the highest specificity, but low sensitivity (Table bellow) (**Jastrzebski M 2016**).

Criteria	ECG examples	Point
1. Initial R wave in V1 Criterion proposed by **Sandler 1965**)		1
2. Initial r > 40 ms in V1 or V2 Criterion proposed by Swanick, *et al*.[12], and later validated by Kindwall **Kindwall KE et al 1988**)		1
3. Notched S in V1 Criterion proposed by **(Kindwall KE, 1988**).		1

Criteria	ECG examples	Point
4. Initial R wave in aVR	 Criterion proposed by Vereckei , *et al*.[8]	
5. Lead II RWPT ≥ 50 ms	 Criterion proposed by (**Pava LF 2010**)	
6. Lack of RS complex in leads V1-V6	If the QRS complexes from V1-V6 have the following configurations --- > QS, R, qR, Qr, rSR′, Rsr′, this criterion is fulfilled. Criterion proposed by Brugada, *et al*.[5]	
7. AV dissociation		

Comparison of VT algorithms diagnostic value

Comparison of the VT score with other ECG-based methods was performed (Table bellow). Chai and collaborators performed a comparison of Brugada, Vereckei and other algorithms. Thirteen studies were included in this metanalysis, and 1918 ECGs were analyzed. The Brugada algorithm showed the highest pooled sensitivity 0.92 and moderate pooled specificity 0.71 for discrimination SVT from VT (**Chai Q 2018**), as compared with other algorithms. The VT score and lead II RWPT criterion were not included in this meta-analysis. Table 5 summarizes the sensitivity and specificity of the algorithms reported by each author.

In the European Heart Rhythm Association (EHRA) consensus document on the management of supraventricular arrhythmias and 2015 ACC/AHA/HRS Guidelines for the management of adult patients with supraventricular tachycardia the following algorithms and criteria were advised for VT discrimination: the Brugada, Vereckei (aVR algorithm) and lead II (Pava's) criterion (**Page RL 2016**).

Comparison of the VT score with other ECG-based methods.

Parameter	VT score ≥ 1	Brugada algorithm	aVR algorithm	Lead II RWPT
Accuracy	83.1%	80.4%	73.4%	70.1%
Sensitivity	96.6%	91.0%	79.9%	62.0%
Specificity	63.4%	60.4%	61.2%	85.3%

With modifications from Jastrzebski, *et al*.(**Jastrzebski M 2016**) RWPT: wave peak time; VT: ventricular tachycardia.

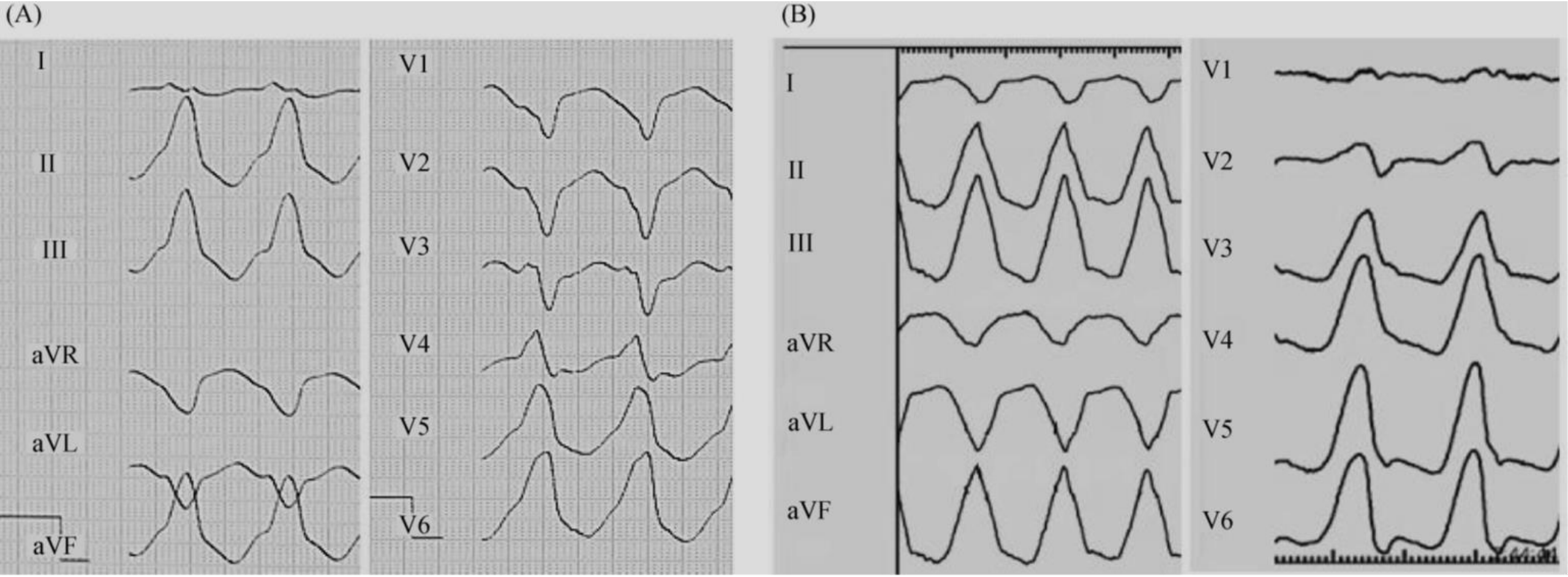
3 Specific VT/PVC origin Discrimination of VT from SVT is the first step in the direction of tachyarrhythmia treatment. The site of origin (SoO) of VT needs to be defined in certain cases. This is of paramount importance for interventional procedures, such as RFCA. The other important clinical aspect is the definition whether VT origination site is consistent with ventricular lesion site (for instance, post-myocardial infarction scar, or abnormal findings detected by visualization methods-magnetic resonance imaging, echocardiography, etc.).

Outflow tract ventricular tachycardia

Most commonly, ventricular arrhythmias (VA) in patients without structural heart disease are originated in the right ventricular outflow tracts (RVOT) and left ventricular outflow tracts (LVOT) (**Hutchinson MD 2013**). Outflow tract arrhythmias are characterized by inferior axis, positive QRS complexes in II, III and aVF (Figure next slide) (**Scanavacca M 2015**). Not infrequently, distinguishing RVOT from LVOT based on surface ECG analysis. challenging. Some features that can help in differential diagnosis (See Table on next slide).

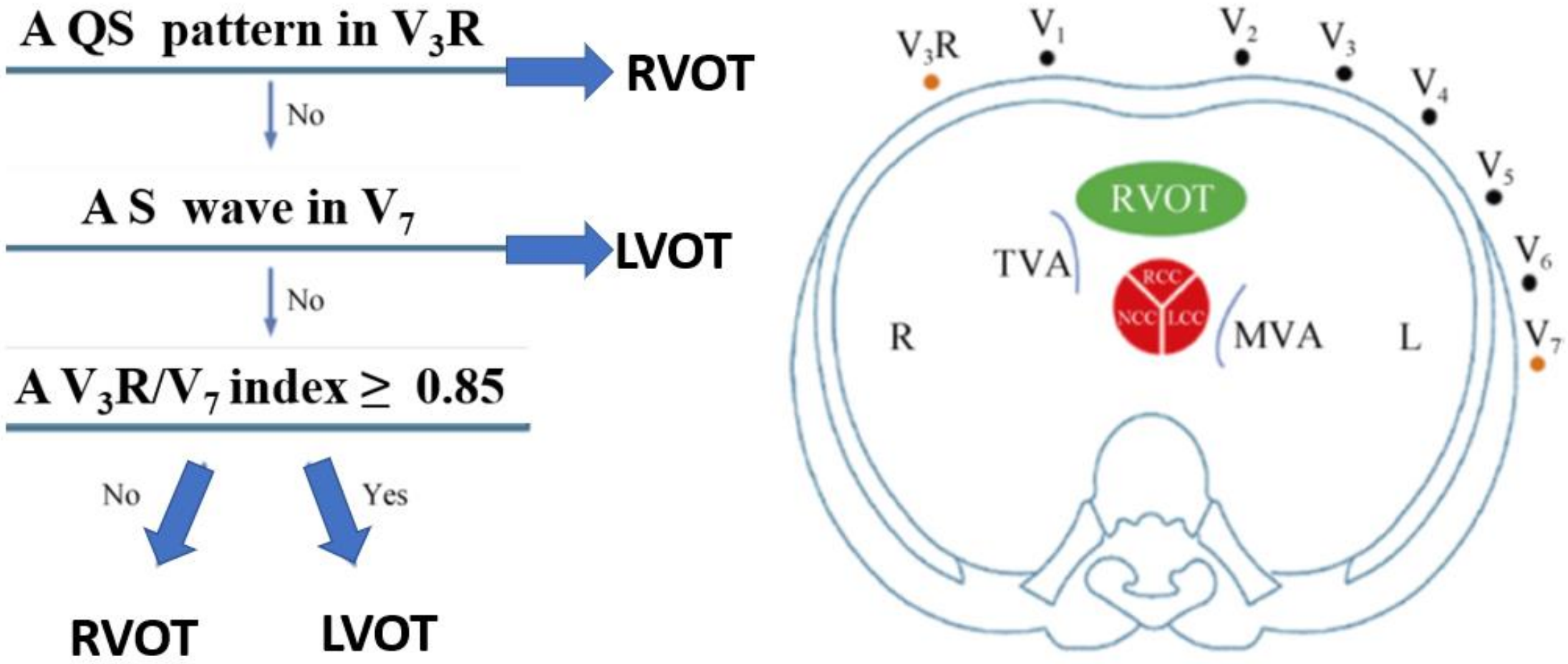
ECG features VA arising from RVOT and LVOT

Location of VA	BBB	Axis	Precordial transition	I	V1	V6	Other features
RVOT							
Anteroseptal	LBBB	Inferior	≤V3	rS	rS	R	Negative, isoelectric, or multiphasic I
Posterior, free wall	LBBB	Inferior	≤V3	R	rS	R	Positive I, broad late notched inferior leads
LVOT							
Supravalvular							
LCC	LBBB	Inferior	≤V2	rS	rS, RS	R	QS or RS in lead I; notched M or W in V1
RCC	LBBB	Inferior	≤V3	R	rS, RS	R	Broad R in V2
LCC/RCC junction	LBBB	Inferior	V3	R/Rsr’	qrS	R	Notched on the downward deflection or W pattern in V1
Infravalvular							
AMC	RBBB	Inferior	Positive concordance	R/Rs	qR	R	No S in V6
Septal-para-Hisian	LBBB	Left inferior	Early	Rs	QS, Qr	Rs	QS amplitude lead II/III ratio >1
Anterior interventricular great cardiac vein junction	LBBB	Inferior	Early	rS	rS, QS	R	Precordial pattern break, MDI >0.55



Common characteristics found in outflow tract ventricular tachycardia (inferior axis deviation, positive QRS complexes in II, III and aVF) (A): RVOT tachycardia; (B): LVOT tachycardia. LVOT: left ventricular outflow tract; RVOT: right ventricular outflow tracts.

Cheng et al evaluated the right precordial and posterior leads with calculation of the V_3R/V_7 index (defined as R-wave amplitude in lead V_3R divided by that in lead V7 when measured in ectopic QRS). ECG leads V5 and V6 are placed into positions V_3R and V_7 (Figure bellow). If QS pattern is present in lead V3R, the origin of VAs is RVOT, then if an S-wave is observed in lead V7, the origin of VA is LVOT. If the QRS morphology does not show these characteristics, then the V_3R / V_7 index is measured. Index < 0.85 indicates RVOT origin, whereas index ≥ 0.85 predicts LVOT origin. The V_3R / V_7 index ≥ 0.85 predicted an LVOT origin with 87% sensitivity and 96% specificity. The major limitations of this study were the small sample size, lack of validation in larger populations, and even if the origin of VA was defined by the successful ablation sites, it may not represent the true origin, but a site which is anatomically close enough to affect the focus (**Cheng D 2018**).



Criteria for differentiating LVOT from RVOT. LVOT: left ventricular outflow tract; RVOT: right ventricular outflow tract.

A practical approach for the diagnosis of wide QRS tachycardia is the use of the following six successive steps proposed by Miller (**Miller JM 2006**):

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

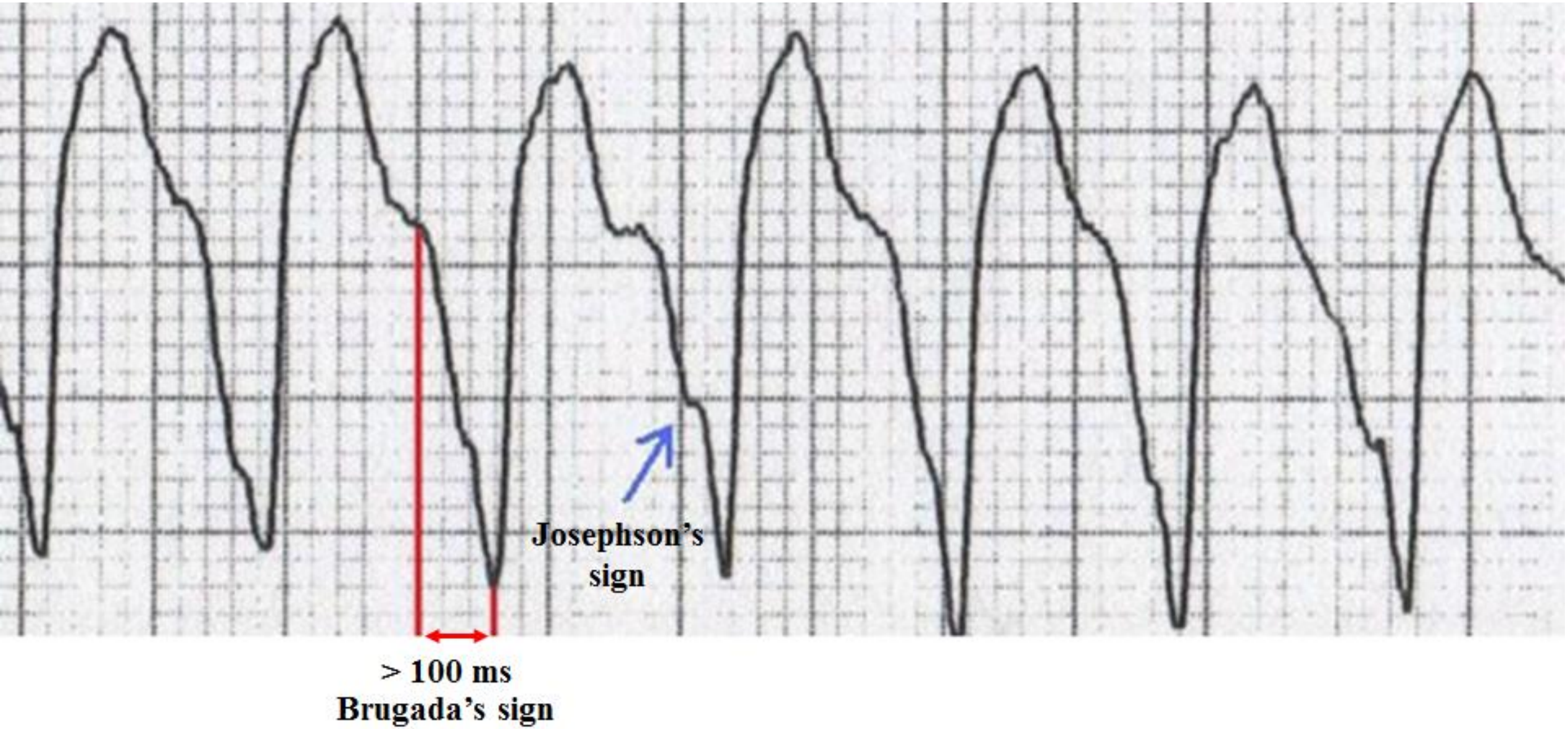
- **Second step:** QRS axis in the FP in the right superior quadrant (northwest quadrant axis). When present, it indicates VT. When absent, go to Step 3.
- **Third step:** Vi/Vt ratio when > than 1, SVT-A is diagnosed; if not, continue to the Step 4.
- **Fourth step:** Absence of RS pattern in the precordial 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 that 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 3)	Atria and/or AV junction
Presence of cannon A waves in the jugular veinous 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, correction of congenital heart disease, family history of SCD: suggestive of HCM, ARVD/C, long QT syndrome and Brugada syndrome, Early repol.Syndrome, IVF,	Strongly suggestive	No
Focus and etiologies	Bundle branches, Purkinje or ventricular muscle. The causes of VT may be with or without structural heart disease (Table 3)	Atria and/or AV junction
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

	VT	SVT-A
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 FP	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. Biphasic or monophasic pattern if RBBB. When biphasic in V1 R' > R (rabbit ear sign) (Figure 29) (Gozensky 1974)	Initial narrow r, and clean s, with no notches if LBBB and triphasic pattern if RBBB
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 FP	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. Biphasic or monophasic pattern if RBBB. When biphasic in V1 R' > R (rabbit ear sign) (**Gozensky 1974**)	Initial narrow r, and clean s, with no notches if LBBB and triphasic pattern if RBBB
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
Distance from the onset of QRS up to the nadir of S >100 ms (Brugada sign)	If present, it is diagnostic.	Lower

	VT	SVT-A
Notch near the nadir of the S wave (Josephson´s sign)	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; however, with a greater number of QRS than P.	No
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 (**Oreto G 2009**)	Suggestive.	>1

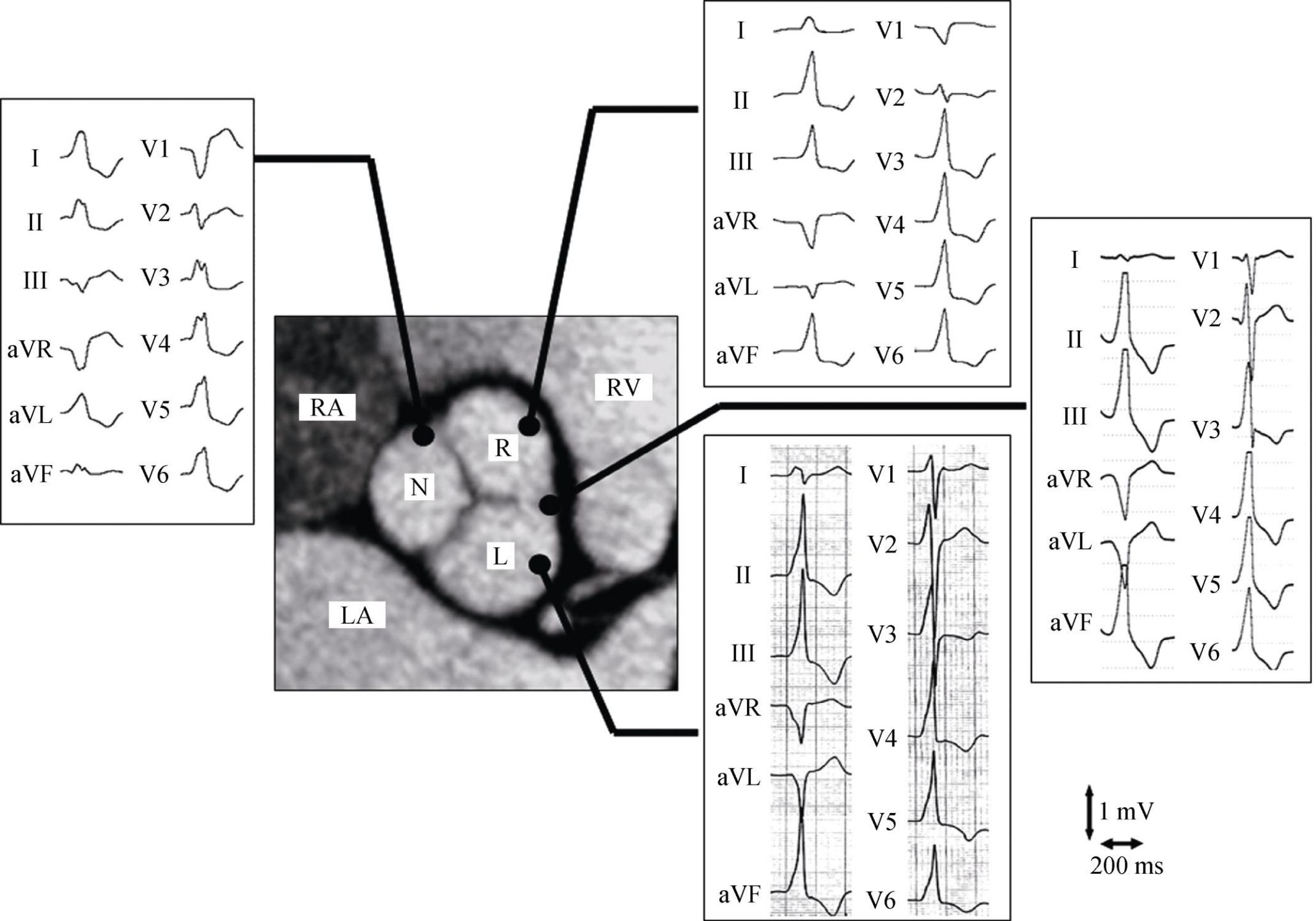

Josephson's sign
> 100 ms
Brugada's sign

Ventricular arrhythmias originating from the aortic root

Yamada, et al. reported that VT arising from the aortic root are more commonly originated from the left coronary cusp (LCC), as compared with the right coronary cusp (RCC), non-coronary cusp (NCC) and at the junction between the LCC and RCC (L-RCC). Surface ECG is a useful tool that allows differentiating the site of origin (Figure 10), such us, an R-wave in lead aVL rules out origin in the LCC, RCC, or L-RCC. Right bundle branch block (RBBB) and inferior axis indicate a VT arising from the LCC. To distinguish between the most common origins (LCC and RCC), the R-wave amplitude ratio in leads II and III might be a practical differential element. A III/II ratio > 0.9 determines LCC origin (**Yamada T 2008**).

Normal heart idiopathic monomorphic VT from aortic sinus cusp (ASC) or aortic sinus of Valsalva has the following ECG pattern: Right bundle branch block-like pattern, QRS inferior axis, Early precordial transition with Rs or R in V_2 or V_3 , rS pattern in lead I, if VT arise from the left sinus, Notched R wave in lead I if VT arise from the noncoronary sinus and the indexes of R-wave duration and R/S-wave amplitude are significantly lower in VTs originate from the RVOT than in group VT from aortic sinus cusp (ASC) (**Ouyang F 2002**). Normal heart idiopathic monomorphic VT from aortic cusp or aortic sinus of Valsalva with left bundle branch block, inferior axis and early precordial transition can be ablated in the majority of patients from either the left or the noncoronary aortic sinus of Valsalva (**Kanagaratnam L 2001**).

In young patients, with symptomatic normal heart idiopathic monomorphic ventricular tachycardia originating from the left aortic sinus cusp, RFCA is safe and effective . RMVT originating from the left ventricular outflow tract has specific ECG characteristics, and can be successfully and safely cured using RFCA directed at the left coronary cusp (**Tang CW 2004; Strobel JS 2002**).



Typical ECGs for VT originating from the aortic root. With permission from Yamada T, *et al.*[20] L: left coronary cusp; LA: left atrium; N: non-coronary cusp; R: right coronary cusp; RA: right atrium; RV: right ventricle; VT: ventricular tachycardia.

Fascicular VT/PVC, idiopathic left VT (ILVT)verapamil-sensitive intrafascicular reentrant VT

Definition

VT is the commonest form of idiopathic VT usually seen in individuals (predominantly in young males) without apparent structural heart disease, usually presents as paroxysmal palpitations, with right bundle branch block pattern. The QRS axis depends on which fascicle is involved in the re-entry. Extreme left axis deviation is noted with left posterior fascicular tachycardia (> 90% of cases) and right axis deviation with left anterior fascicular tachycardia. Characteristically the QRS duration is a borderline-broad complex or relatively narrow QRS complex, usually between 120ms and 140ms and RS interval between 60 to 80ms occurring. It is an important cardiac arrhythmia with specific electrocardiographic features and therapeutic options.

Synonymous verapamil-sensitive fascicular tachycardia, idiopathic left ventricular tachycardia (IVLT), Belhassen VT and borderline-broad complex tachycardia.

Historical Background

May 1972: Cohen et al (**Cohen 1972**). First description: VT with a relative narrow QRS complexes (≤ 140ms).

1979: Zipes et al (**Zipes DP 1979**) reported three patients with VT characterized by QRS width of 120 to 140 ms, RBBB-Like pattern and extreme left-axis deviation. These authors described the characteristic triad:

- Induction with atrial pacing
- RBBB-like pattern with extreme left axis deviation
- Without structural heart disease

1981: Belhassen et al (**Belhassen B 1981**) were the first to report on the characteristic termination of this VT with intravenous verapamil, hence accounting for the terms Belhassen VT and verapamil-responsive VT to describe the condition.

Epidemiological Aspects

Age: Usually observed between 15 and 40 year.

Gender: Male predominance (70% of cases). Verapamil sensitive fascicular VT has 3:1 male predominance (**Nakagawa 2002**).

It is characterized by a RBBB pattern and which involves altered Purkinje fibers on the LV septal wall (**Della Rocca 2018**). Left posterior fascicular VT is present with left superior axis and RS complex in V5 and V6 and RBBB. Left anterior fascicular VT shows RBBB morphology with right axis deviation. The upper septal fascicular VT is rare and exhibits a narrow QRS complex with normal or right axis deviation. Left posterior fascicular VT is the most common compared to other fascicular VTs (**Prystowsky 2012; Lerman 1997; Kapa 2017**), the ECG features of fascicular VT are summarized in Table bellow

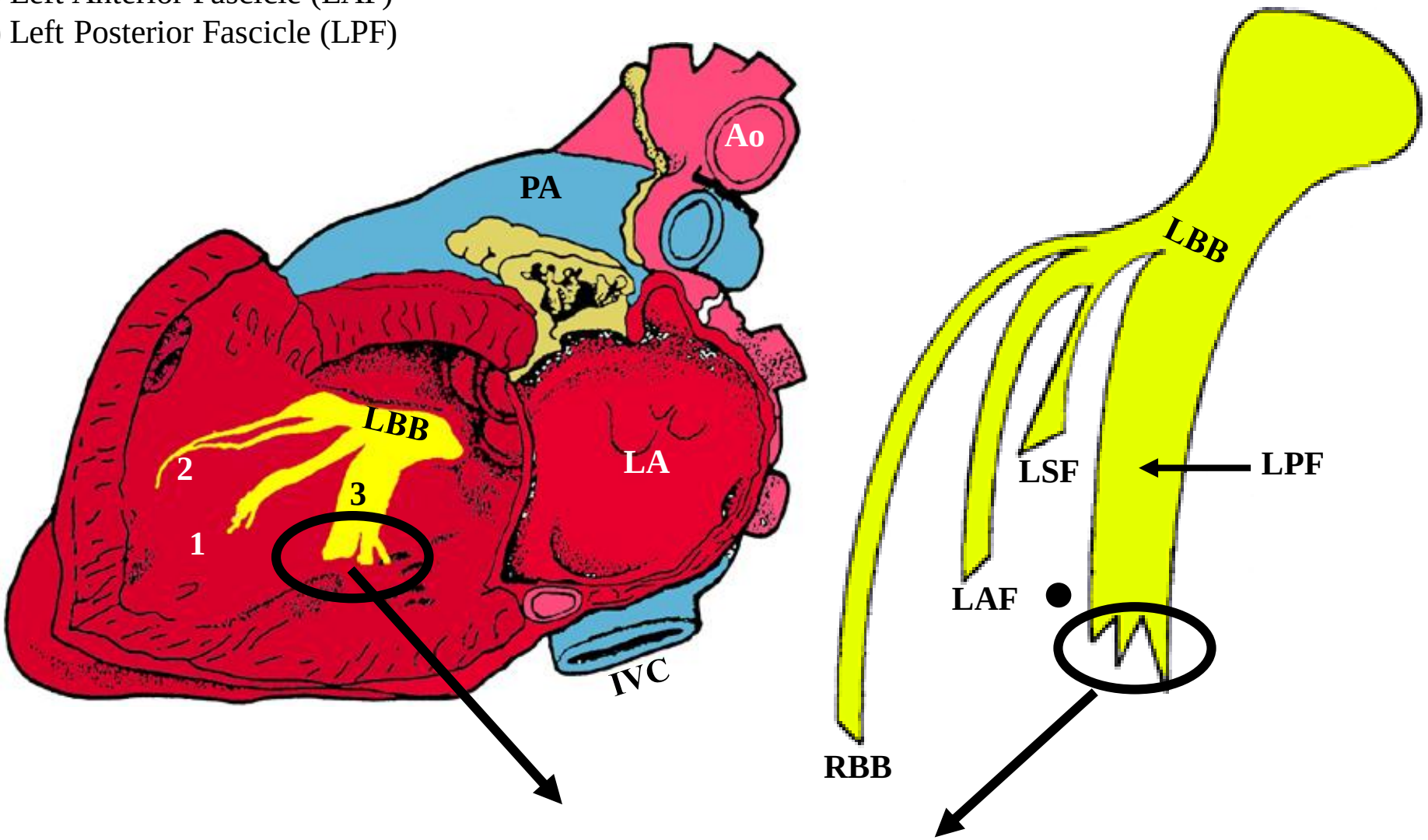
Table Different types of fascicular tachycardias.

Type of tachycardia	QRS width, ms	QRS pattern	QRS axis
Left posterior fascicular VT (most frequent) 92%	< 120-140	RBBB	Left
Left anterior fascicular VT 7%	< 120-140	RBBB	Right
Upper septal fascicular <1%	120	RBBB or normal	Normal

With modifications from (**Kapa S 2017**) RBBB: right bundle branch block.

Interfascicular Macro-Reentry Mechanism

1) Left Septal Fascicle (LSF)
2) Left Anterior Fascicle (LAF)
3) Left Posterior Fascicle (LPF)



Classical origin area: region of the posteroinferior interventricular septum

.

Verapamil-sensitive VT encompasses a heterogeneous group of tachycardias that may result from multiple cellular electrophysiologic mechanisms (German 1983).

- Intrafascicular macro re-entrant tachycardia (main and constant).
- cAMP-mediated triggered activity.
- Propranolol sensitive.

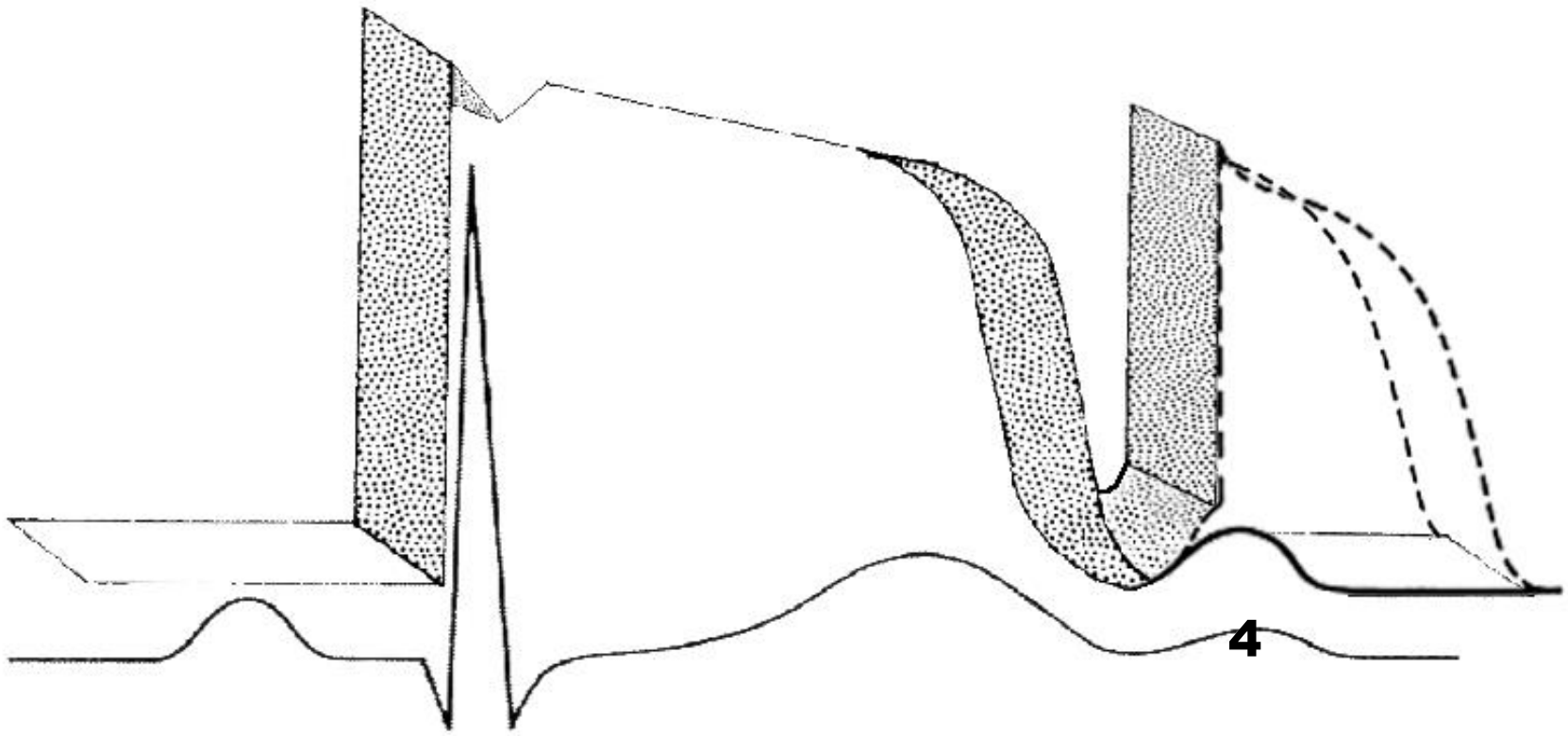
Manifestations

- Palpitations;
- Dizzines;
- Presyncope;
- Syncope.
- Sudden cardiac death was reported only in one case (Ramprakash 2008).

Events Trigger:

- At rest
- Catecholamine stimulation: exercise or post exercise and isoproterenol infusion

By delayed after depolarization cAMP-mediated triggered activity



Concept: they are oscillations of the membrane potential that occur after having completed phase 3 of AP or in phase 4. When they reach the limit, they trigger a new AP. They are observed in high rates (tachycardia-dependent). Their mechanism is caused by the opening of the $I_{NS.}$ channel, sensitive to intracellular Ca^{2+} concentration.

Michowitz, et al. demonstrated that left posterior fascicular VT (LPF-VT) is frequently misdiagnosed as SVT-A with aberrant RBBB and left anterior fascicular block (LAFB), and proposed a novel diagnostic method based on four variables for distinguishing LPF-VTs The authors reported sensitivity and specificity of 82.1% and 78.3%. It was stated that patients with 3 of 4 positive criteria had a high probability of LPF-VT, in contrast to patients with ≤ 1 positive criterion, in which RBBB and LAFB was always present. Left posterior ventricular tachycardia (LPVT) using the left posterior fascicle (LPF) can be easily mistaken for supraventricular tachycardia (SVT-A) with right bundle branch block (RBBB) and left anterior fascicular block (LAFB), and distinguishing these dromotrophic disturbances via ECG analysis is essential for appropriate management. 4 parameters supporting the diagnosis of LPFVT 1)Atypical right bundle branch block–like V1 morphology, ***(no rsR', or R larger than R' in V1***); 2) Positive QRS in aVR, 3) V6 R/S ratio ≤1, and 4) QRS ≤140 ms **(Michowitz Y, 2017**).

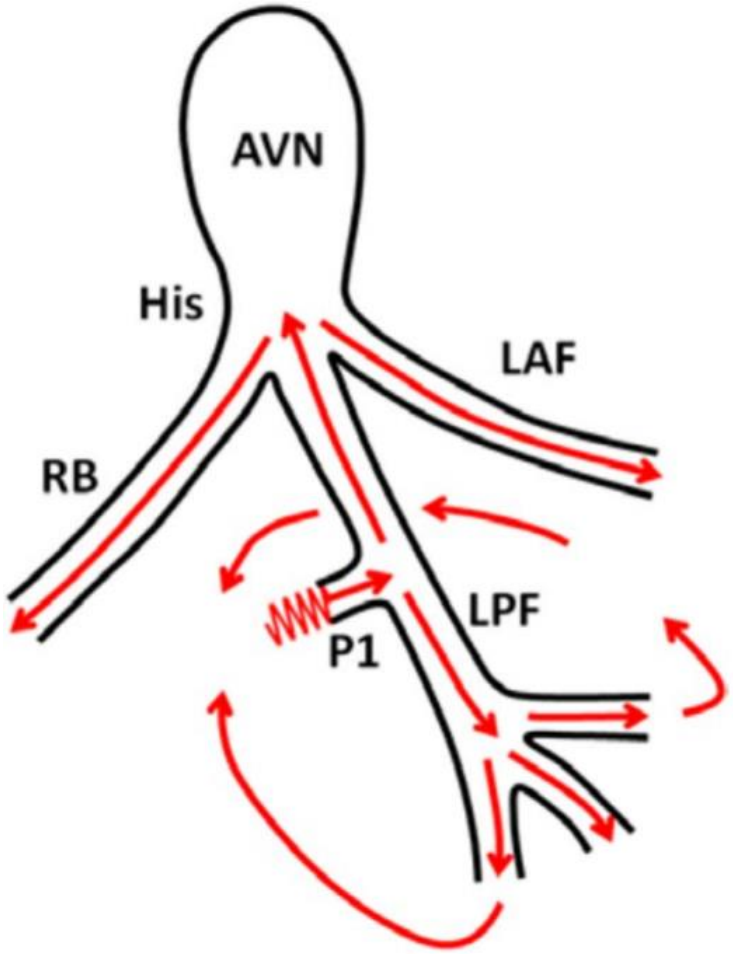
How does depolarization of the ventricles actually differ between LPF-VT and SVT with RBBB and LAFB?

Should we expect uniform surface ECG properties for either entity?

In the case of LPF-VT, earliest activation of the LV typically arises from the LPF, but contribution of the LAF, LSF, and even the RBB are not necessarily excluded. Liu et al (**Liu 2016**) postulated a macroreentrant circuit comprising a decremental P1 fiber that connects to the LPF at its distal portion and serves as the antegrade limb, a portion of the LPF, septal ventricular myocardium that serves as the retrograde limb, and a zone of slow conduction between the myocardium and the proximal portion of P1. They found that the site of merged P1 and P2 (the latter representing the LPF could be predicted by the V-H interval during tachycardia—the longer the V-H interval, the longer the P1 segment along the LV septum and the more distal the location of the earliest P2. In such a model where bystander retrograde conduction can continue along the portion of the LPF proximal to the connection of P1, varying degrees of ventricular depolarization and slight surface ECG fusion from antegrade LAF, LSF , and RBB conduction during tachycardia are feasible. Such fusion might even be expected to generate a slightly narrower QRS than might otherwise be seen with pure LPF activation, a concept that may be further supported by the demonstration of less common but relatively narrow QRS nonreentrant VT arising from the LPF (**Talib 2016**).

Earlier activation of the lateral LV via the LAF could also contribute to positivity in aVR. Conversely, in SVT with RBBB and LAFB, our natural inclination is to assume complete conduction block in the RBB and LAF, with ventricular depolarization initiated entirely via antegrade conduction of the LPF and LSF. However, it is possible that some residual slow conduction in the fascicles remains. Analogous pseudoblock is often discovered when treating bundle branch reentrant VT—even in many patients with presumed complete antegrade LBBB, elimination of RBB conduction via ablation induces a RBBB ECG pattern rather than complete atrioventricular block. It stands to reason, therefore, that depolarization of the ventricle (and the resulting surface ECG pattern) with RBBB and LAFB can vary based on the degree of residual slow conduction in either the RBB or the LAF and how the differential conduction properties of the fascicles may vary based on heart rate. Anatomic variability in the morphology of the left fascicles, particularly with regard to site of origin of a LSF, may further contribute to variability in ventricular depolarization (**Demoulin 1972**). These factors may help explain some of the nonuniformity of surface ECG findings between patients. Li et al adding another valuable tool to our armamentarium for correctly distinguishing LPF-VT from SVT with RBBB and LAFB, and particularly for its applicability in clinical practice—the criteria specified are simple, easy to remember, nonambiguous, and not dependent on identifying dissociated atrial activity.

Schematic diagram of the left posterior fascicular ventricular tachycardia (LPF-VT) reentry circuit



What is known

I. Left posterior fascicular ventricular tachycardia (LPF-VT) originates from the left ventricular conduction system and reentry is the mechanism.

II. During LPF-VT, a diastolic potential (P1) can be recorded in some patients, and is involved in the reentry circuit in these patients.

What the Liu´s study adds

I. The mechanism of LPF-VT is macro-reentry involving the ventricular myocardium, a part of the left posterior fascicle, a slow conduction zone, and a P1 fiber (in the cases with recorded P1).

II. P1 potentials may not be recorded in some patients for several reasons.

III. The HV interval during LPF-VT is a useful marker guide mapping and ablation of these arrhythmias.

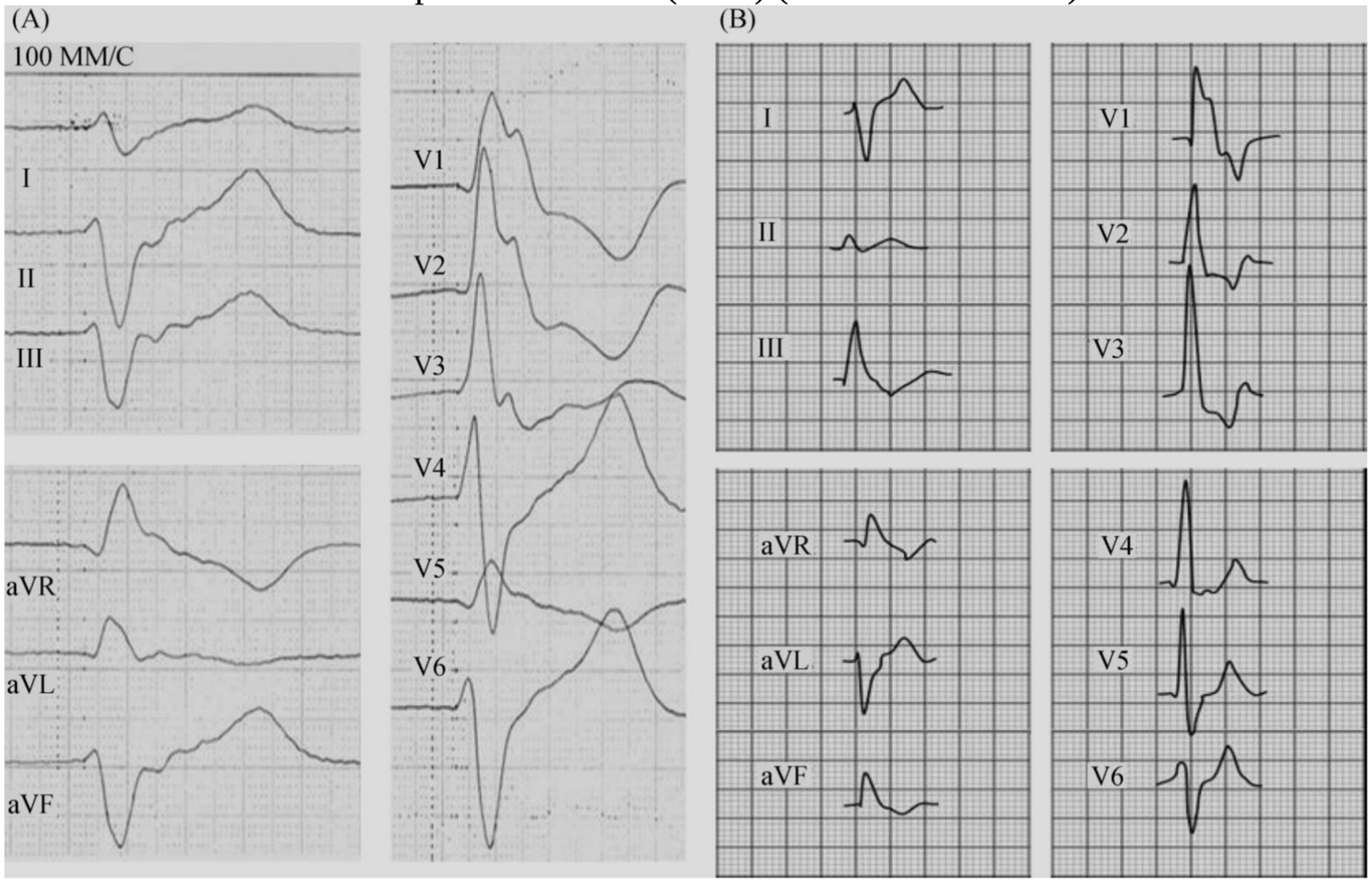
IV. The LPF-VT macroreentrant loop involves the ventricular myocardium, a part of the LPF, a slow conduction zone, and in certain cases, a specially conducting P1 fiber. The HV interval during LPF-VT correlates with multiple electrophysiological measures and is a useful marker for identification of the optimal ablation site.

AVN indicates atrioventricular node; LAF, left anterior fascicle; LPF, left posterior fascicle; and RB, right bundle branch.

From (**Liu Q 2016**),

Papillary muscle (PM) VT/PVC

VA originated in the left anterolateral PM exhibit an RBBB pattern with right inferior axis (**Yamada T 2009**), compared with VA arising from the left inferoseptal PM have RBBB pattern with right or left superior axis (Figure below) (**Doppalapudi H 2008**). VA arising from the right ventricular papillary muscles (RVPM) where analyzed by Crawford and coworkers, these arrhythmias can be originated from the posterior or anterior RVPM showing a superior axis with late R-wave transition (> V4), though septal right ventricular PM arrhythmias usually present an inferior axis with an earlier R-wave transition in the precordial leads (< V4) (**Crawford T 2010**).

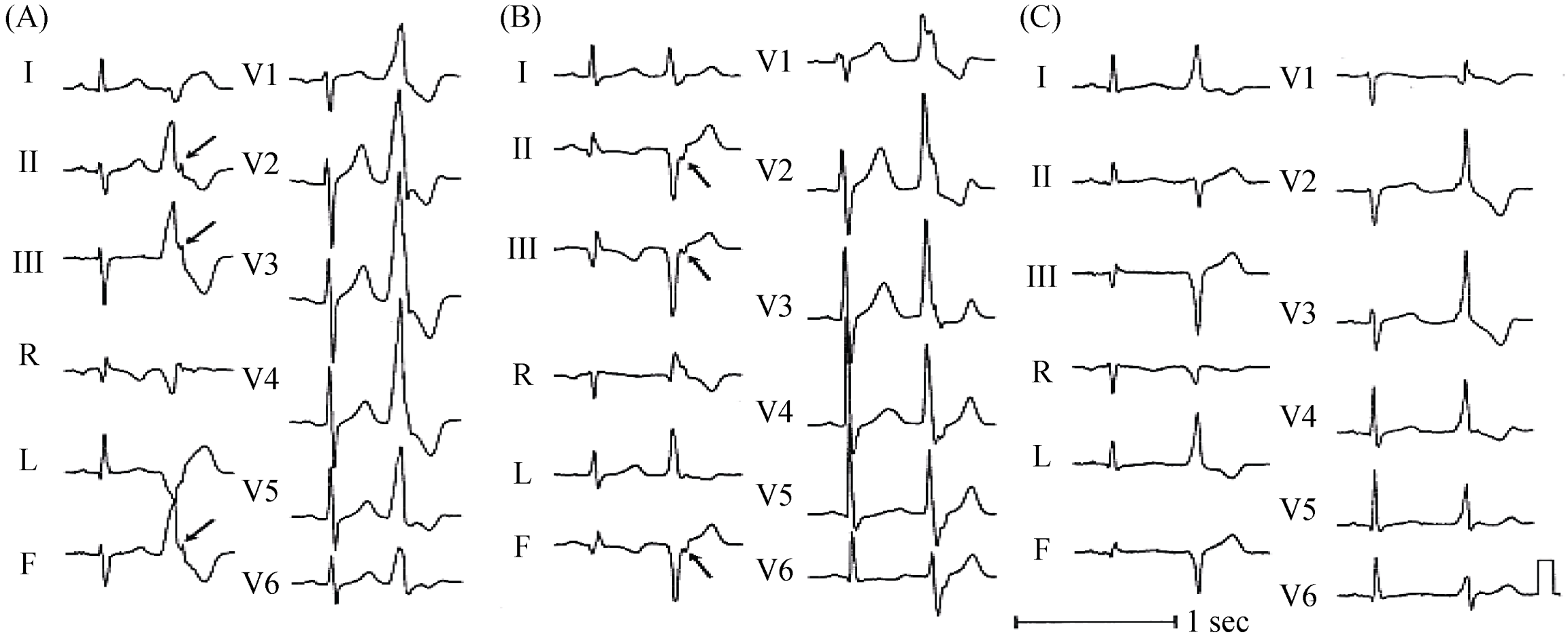


VT from the inferoseptal papillary muscle (A) and VT from the anteroseptal papillary muscle (B: schematic ECG representation). VT: ventricular tachycardia.

Differentiation of PM from fascicular and mitral annular ventricular arrhythmias

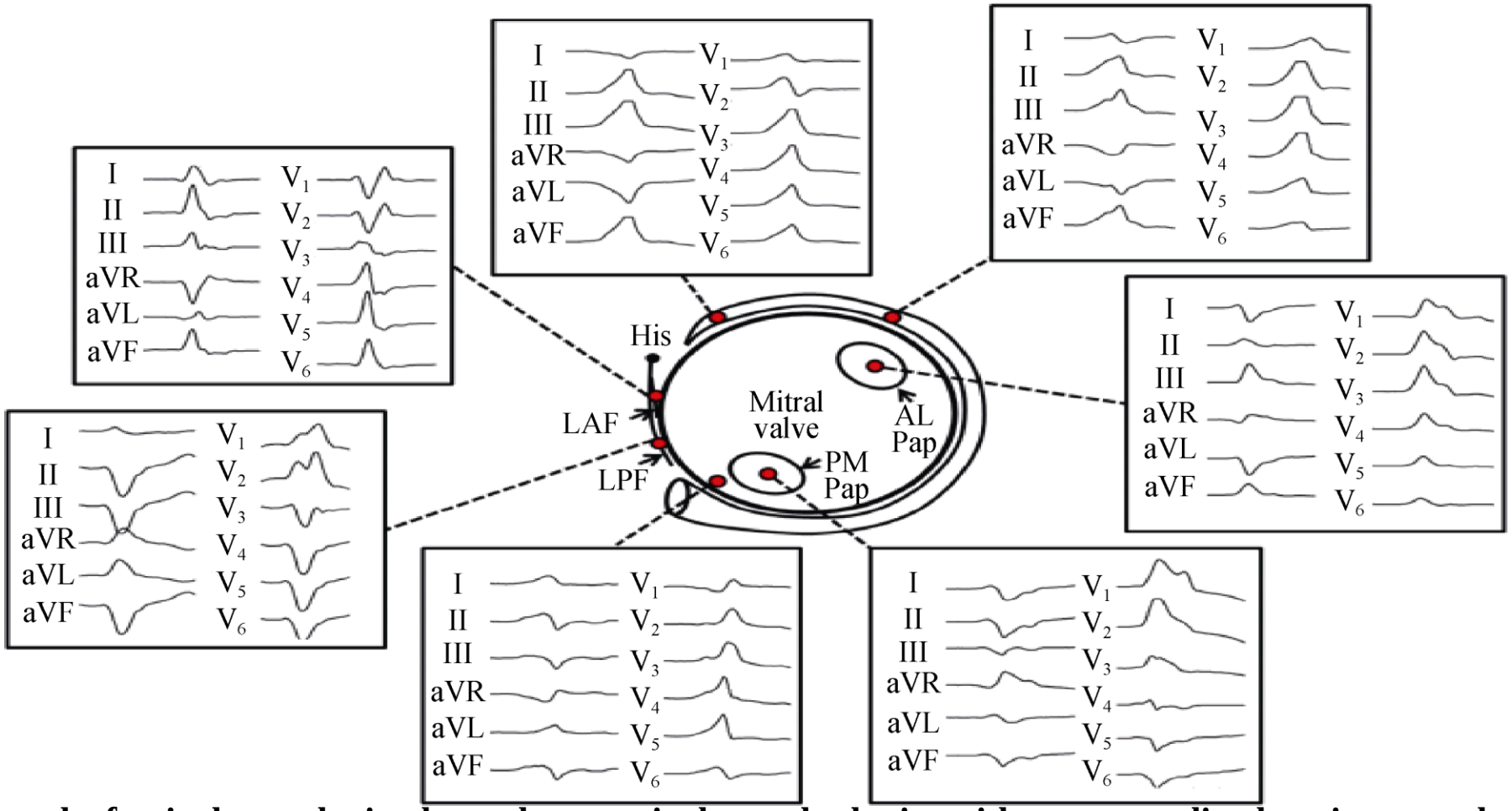
Ventricular arrhythmias can originate from PMs in structurally normal and diseased hearts. The PMs are the parts of the mitral valve (MV) and tricuspid valve apparatus (**Enriquez A 2017**). VA originating from the PMs, fascicles, and mitral annulus have RBBB pattern.

Tada, et al. (**Tada H 2005**) reported that mitral annulus (MA) VA most commonly originate from the anterolateral (58%), posteroseptal (31%) and posterior MA (11%). In this study, all patients with VA had right bundle branch block morphology, R or Rs pattern in leads V2-V6 and precordial transition in V1 or V2. The anterolateral MA SoO was associated with an inferior axis and negative polarity in leads I and aVL. A posteroseptal or posterior SoO was associated with a superior axis and positive polarity in leads I and aVL (Figure below).

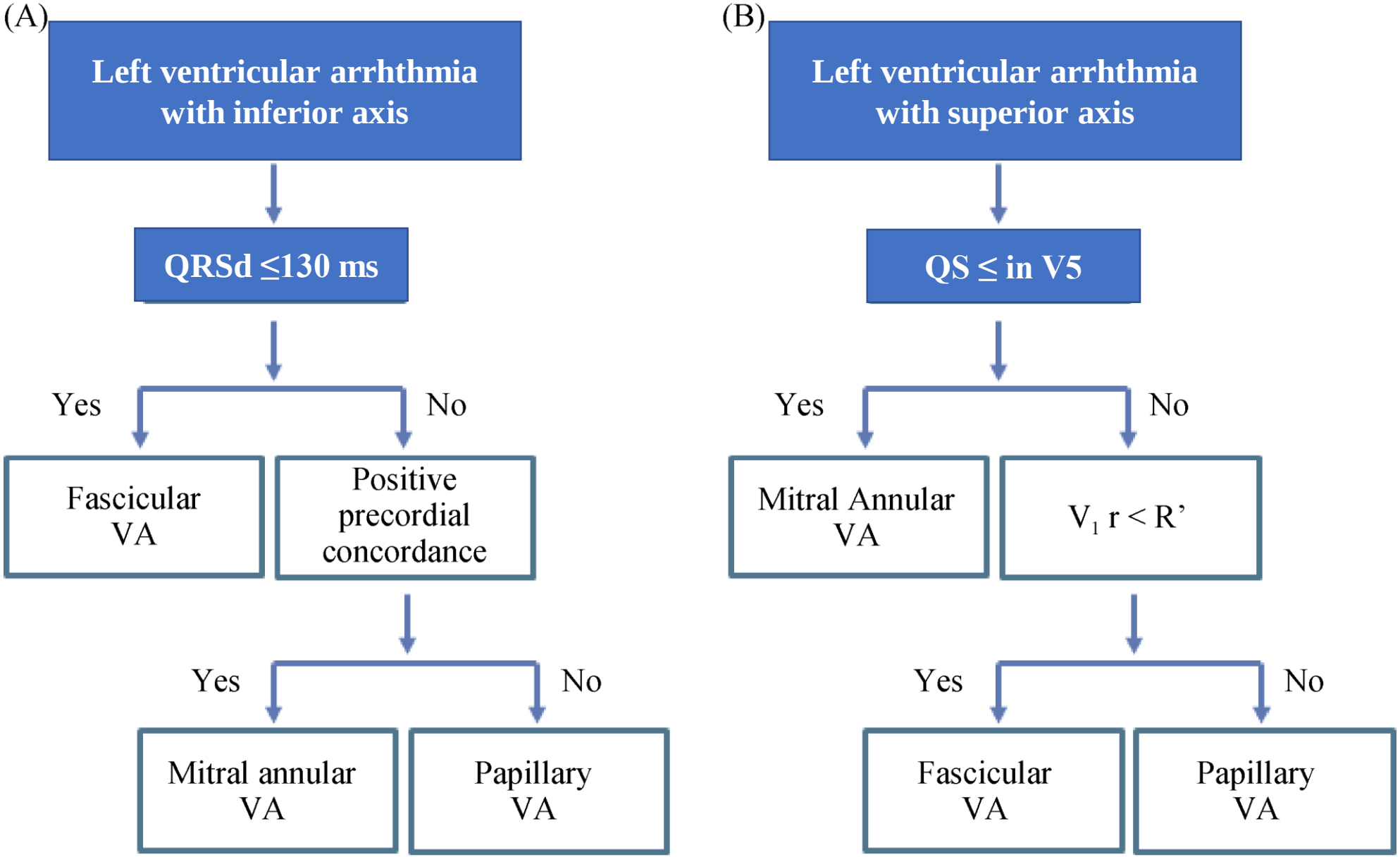


ECGs of PVCs originating from the anterolateral (A), posterior (B), and posteroseptal (C) portions of the mitral annulus. Arrows indicate "notching" of the late phase of the QRS complex in the inferior leads (**Tada H 2005**).

Differentiation of VT/PVC originating from these structures is important for management, since fascicular VT can be treated by calcium antagonists, ablation of VT from PM may require additional ultrasound visualization. Although ECG-based differentiation can be challenging, there are several useful characteristics that can be taken into account (Figure below) (**Al'Aref SJ 2015**). An algorithm for differentiation of fascicular, PM, and mitral annulus VT was proposed by Al'Aref, et al. (**Al'Aref SJ 2015**) and included the following features: QRS duration, precordial lead transition, and V1 QRS morphology (Figure next slide). This novel algorithm reported an acceptable accuracy rate for the diagnosis of papillary muscle VAs (83%), fascicular VAs (87%), and mitral annular VAs (89%).



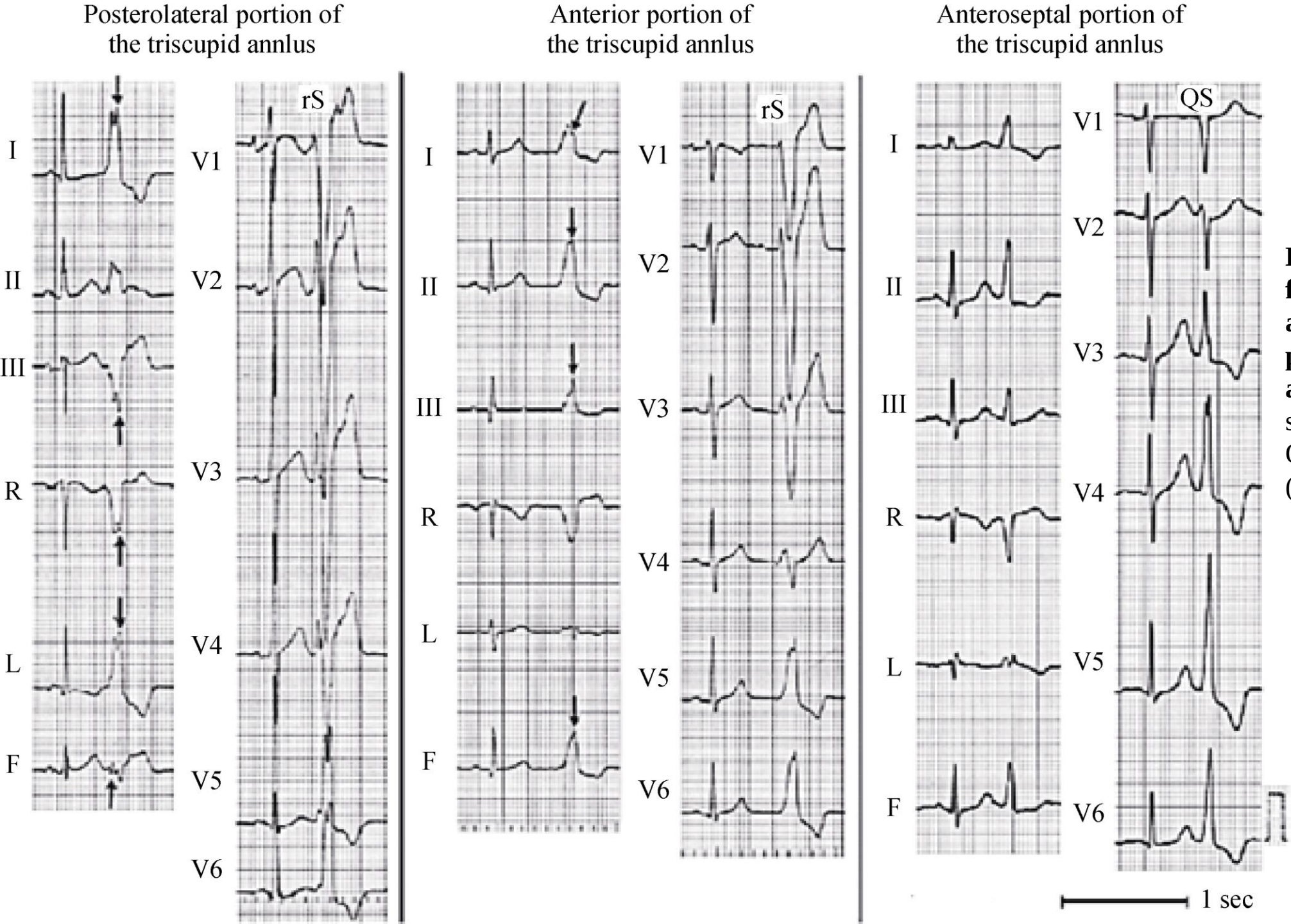
ECGs of papillary muscle, fascicular, and mitral annular ventricular arrhythmias with corresponding locations on schematic diagram. (**Al'Aref SJ 2015**). AL: anterolateral; LAF: left anterior fascicle; LPF: left posterior fascicular; Pap: papillary muscle; PM: posteromedial.



Algorithm for differentiation of focal left VA. (A): Flow chart shows algorithm for differentiation of inferior axis VA into papillary, fascicular, or mitral annular arrhythmia based on QRS duration and positive precordial concordance; (B): Flow chart shows algorithm for differentiation of superior axis VA into papillary, fascicular, or mitral annular arrhythmia based on QRS morphology in leads V1 and V5. VA: ventricular arrhythmia.

Ventricular arrhythmias originating from the tricuspid annulus

Although VAs originating from the tricuspid annulus are not infrequent, their prevalence is lower compared to other sites. Tada, et al. (**Tada H 2005**) in his study described that 8% (34 patients) of the total evaluated 454 patients exhibited a VT arising from the tricuspid annulus in which the preferential SoO was the septal portion, especially the anteroseptal portion, as well as it was described VT arising from the free wall. Many ECG features were found, in all patients QRS complexes during the VT had LBBB morphology, lead I had a R or r pattern, negative component in aVR (QS, qs, Qr, or qr pattern), positive component (r or R) in aVL. VT arising from the free wall portion of the annulus had a wider QRS (167 ± 21 ms) in comparison with VT arising from septal portion (143 ± 16 ms). The mid or late component of the notched QRS was more frequent in VT originated in the free wall than from the septal portion. In VT arising from the free wall the R-wave transition occurred after lead V3 and in VT arising from the septal portion of the annulus occurred in lead V3. V1-V3 Q-wave amplitude was higher in VT originated in the free wall portion than in the VT arising from the septal portion (Figure next slide) (**Tada T 2007**).



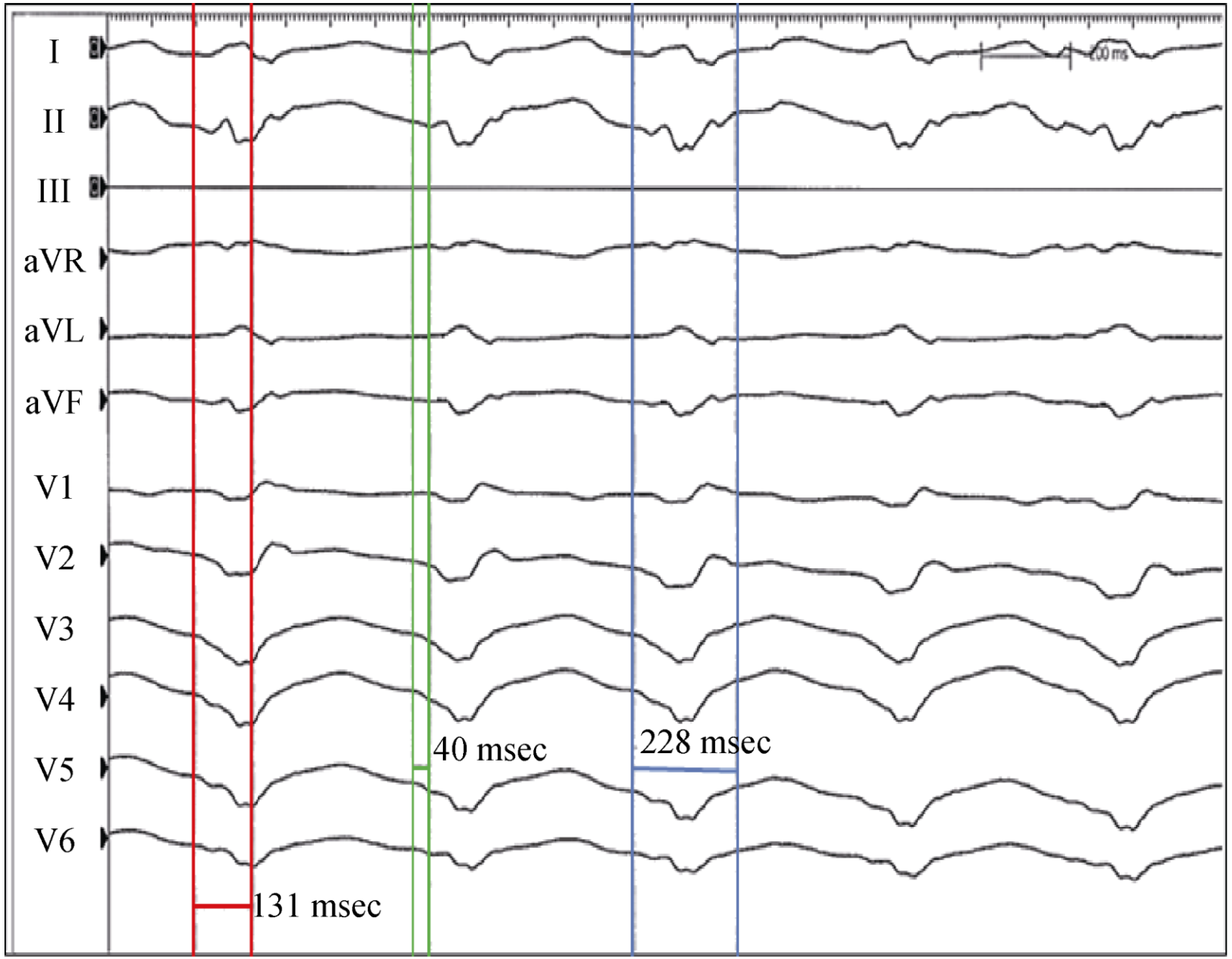
ECGs of PVCs originating from the posterolateral, anterior, and anteroseptal portions of the tricuspid annulus. Arrows indicate the second peak of the "notched" QRS complex in the limb leads (**Tada H 2007**).

Epicardial origin

About 15% of VTs have an epicardial origin (**Baman TS 2010**), not amenable to conventional endocardial RFCA. This is why electrophysiologists pay special attention to the pre-procedure screening of possible epicardial VT exit sites. Several attempts have been made to develop approaches for identification of ECG characteristics specific for epicardial VT origin. These criteria are summarized in Table below and Figure next slide. It should be acknowledged that these criteria are more specific for non-ischemic VTs, and in patients with postmyocardial infarction VT/PVC they are of less specificity (**Berruezo A 2004**). Once an epicardial SoO is suspected, an operator may consider that this site can be approached via cardiac venous system (through coronary sinus---for some left ventricular basal localizations) or via pericardial access. Pericardial access is performed surgically or via sybxyphoid or modified puncture (**Sosa E 1996; Simonova KA 2019**). Although transcutaneous pericardial puncture is less invasive, it can be associated with major life-threatening complications (**Koruth JS 2011**).

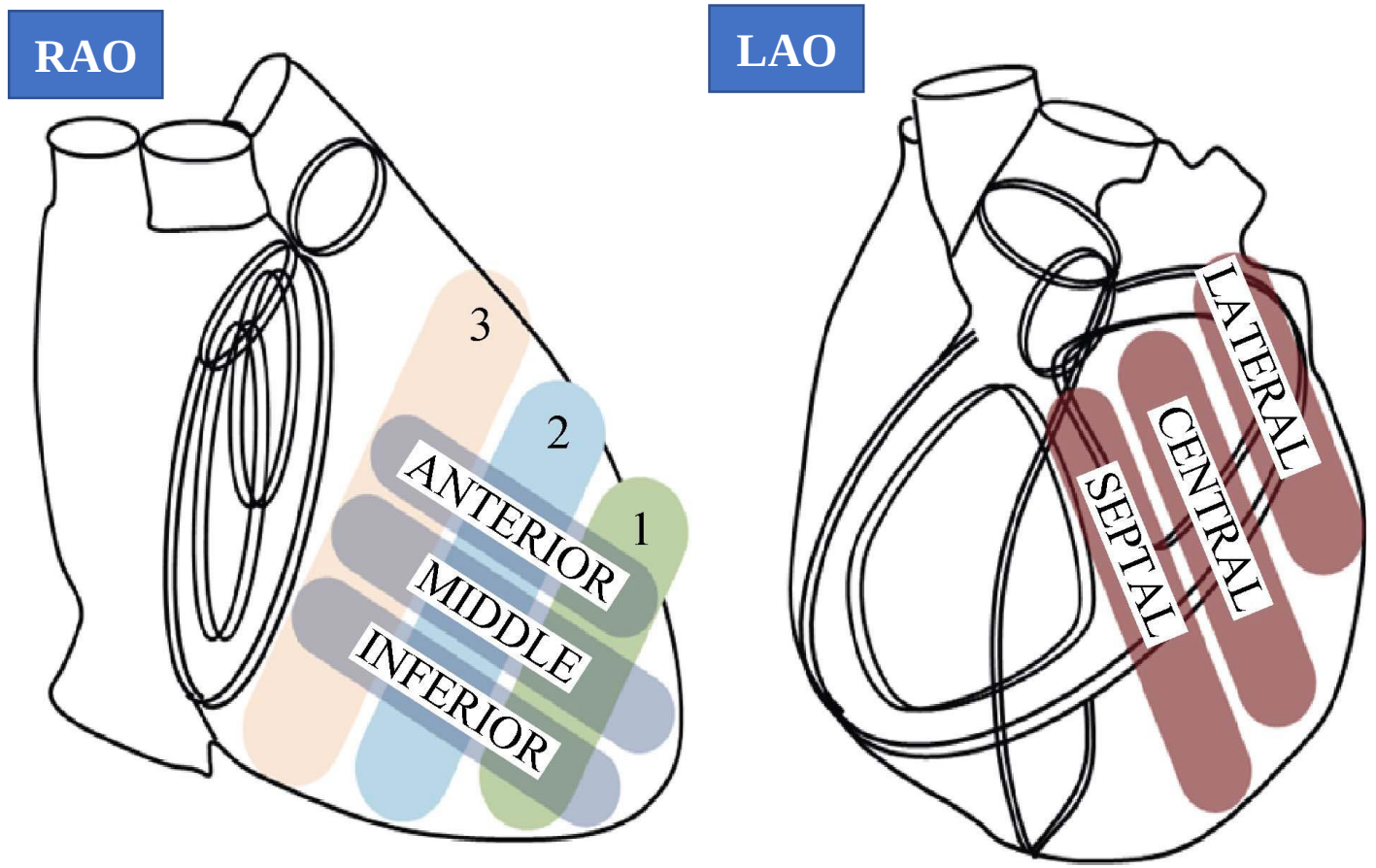
Electrocardiographic criteria proposed for the identification of epicardial VTs.

Author	Underlying heart disease	Limitations	ECG criteria
Berruezo, *et al.* (**Berruezo A 2004**).	CAD: 72% DCM: 28%	RBBB VT	Pseudodelta wave ≥ 34 ms Intrinsicoid deflection V2 ≥ 85 ms Shortest RS complex ≥ 121 ms
Daniels, *et al.* (**Daniels DV 2006**)	No SHD	Described for LVOT VT	Precordial maximum deflection index ≥ 0.55
Bazan, *et al.* (**Bazan V 2006**)	NICM	Absence of Q-wave in sinus rhythm	Q-wave in lead I for anterolateral epi VT Q-wave in inferior lead for inferior epi VT
Vallès, *et al.* (**Vallès E 2010**)			
Bazan, *et al.* .(**Bazan V 2006**)	CAD. DCM: , ARVC: , No SHD:	No tested in ARVC VTs. Absence of Q- wave in sinus rhythm	Q-wave in lead I / QS in lead V2 for anterior epi RV VT Q-wave in leads II, III, and aVF is inferior epi RV VT

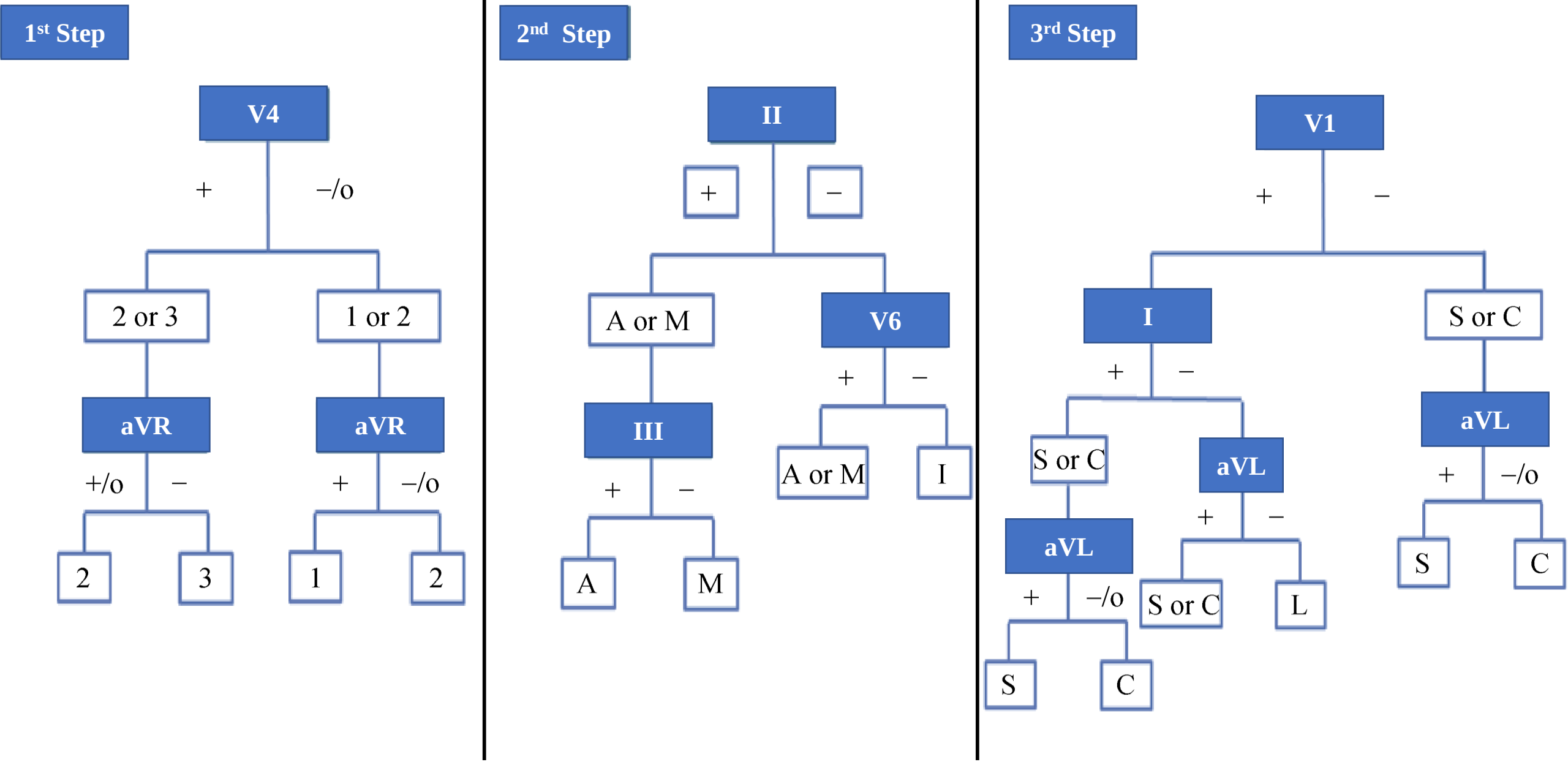


VT with epicardial origin. A pseudo δ wave ≥ 34 ms (measured from the earliest ventricular activation to the earliest fast deflection in any precordial lead), R wave peak time V2 ≥ 85 ms (defined as the interval measured from the earliest ventricular activation to the peak of QRS in V2), shortest RS complex ≥ 121 ms (defined as the interval measured from the earliest ventricular activation to the nadir of the first S wave in any precordial lead). VT: ventricular tachycardia.

VT in structural heart disease: (the present case!!!) SoO of VTs in patients without structural heart disease (SHD) can be defined by ECG features with high accuracy. Nevertheless, in patients with SHD, such as, MI, ICM, chronic Chagasic myocarditis, cardiac sarcoidosis, etc., determine a certain SoO can represent some difficulties due to the substitution of part of the myocardium with scar what causes changes in the QRS morphology. ECG features of patients with SHD and patients without SHD are differentiated by the presence of QRS complexes with low amplitude, prolonged duration, notches and a reentrant mechanism. In most cases of SHD, VT with RBBB or LBBB pattern arise from the LV (**Miller JM 2017**). Kuchar et al. analyzed ECG features in a group of 22 patients with prior MI in order to predict the origin of VA. Afterwards a three steps algorithm was elaborated and tested in 44 patients with SVT. The LV was divided into 3 different areas using two different fluoroscopy views, the right anterior oblique (RAO) at 30° and the left anterior oblique (LAO) at 60° (Figure next slide). This algorithm seems to be suitable for diagnosis of VT arising from anterior, inferior, septal and lateral sites offering a success rate of ≈82% to 90%. The impossibility for identifying VT arising from RV exits sites was described as a limitation (**Kuchar DL 1989**).



Schematic representation of anatomical areas in right anterior oblique (RAO) and left anterior oblique (LAO) views. LAO: left anterior oblique; RAO: right anterior oblique.



Kuchar et al's algorithms for the identification of exit sites of VT. A: anterior; C: central; I: inferior; L: lateral; M: middle; S: septal; VT: ventricular tachycardia; o: isoelectric; (+): positive; (-): negative.

Criteria for the diagnosis of fascicular blocks in the frontal plane

LAFB	LSFB	LPFB
Extreme shift of SÂQRS in the left superior quadrant (beyond 30° up to - 90°). Some authors accept between - 45° and – 90° (**Elizari MV 2007**)	Without manifestation in the FP	Frontal plane axis between +90 and 180 degree in adults
rS in II, III and aVF. If SÂQRS was in -30° II would be R = S		rS pattern in leads I and aVL
r III > r II (it indicates CCW rotation in the FP). The voltage of the r waves is 3 to 5 mm in average		qR pattern in III, aVF and II: Q wave is always present in III and may be small or absent in II or aVF
SIII > SII: this criterion differentiates it from ECD of the right branch and SI-SII-SIII syndrome, where SII > SIII		Notch in the descending limb of the R wave in III (middle-final notch)
qR pattern in I and aVL		RIII > RII: SÂQRS closer to +120º (III) than +60º (II), when closer to the latter, it would indicate an incomplete form of LPFB
Frequent notch of the descending limb of R wave in I and aVL (this sign would be present in 80% of the cases)		The q wave in III is always greater than the q wave in II and aVF. If there is association with inferior infarction, the Q wave > 40 ms
R-peak time in aVL ≥ 45 ms		QRSd <120 ms if isolated (without RBBB)
aVR always begins with q or Q wave: qR, QR or Qr. QS is rare		Ventricular activation time, R-wave peak time in aVF≥35 ms
Possible notch in R wave of aVR		
QRS duration <120 ms		

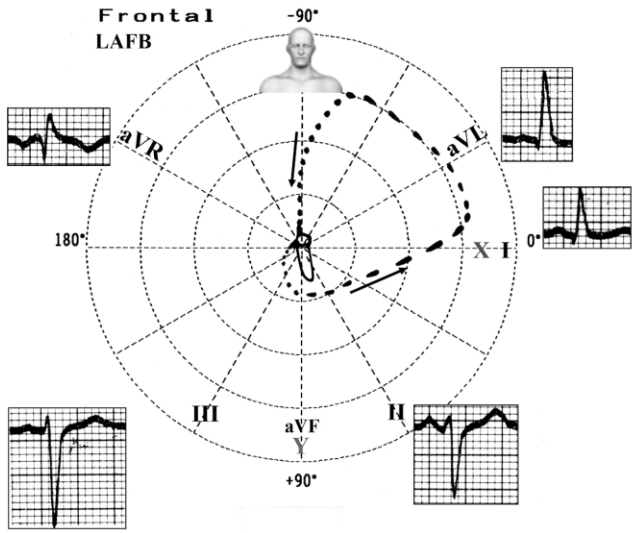
Criteria for the diagnosis of fascicular blocks in the horizontal plane

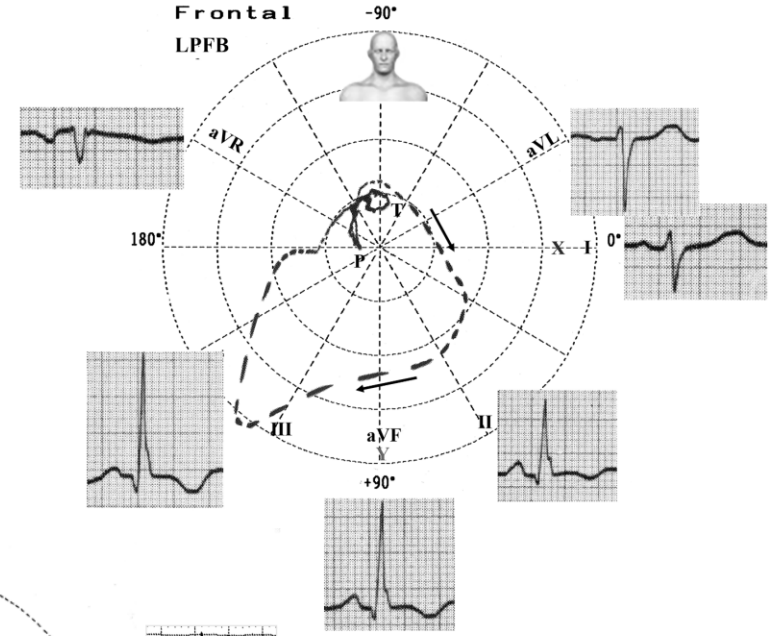
LAFB (HP) (Elizari MV 2007)	LSFB	LPFB
This plane is very little affected when compared to the frontal plane	Normal QRSd or with a minor increase (up to 110 ms). When associated with other fascicular or bundle blocks it could be ≥120 ms	V_1 and V_2: rS pattern, QS rarely
Possible dislocation to the left of the transition area: normally it is in V3 and V4. In LAFB it may be in V5 and V6	Increased ventricular activation time or RWPT in V1 and V2: ≥ 35 ms	S wave of V_2 -V_3 very deep by posterior dislocation and to the right of the final forces
Voltage decrease of R wave and concomitant increase in S wave depth in V5 and V6, as a consequence of the superior dislocation of the forces	R wave voltage of V1 ≥5 mm	Scant progression of growth of r wave in precordial leads: dislocation to the left of the transition area
Possible pattern of pseudo anterior MI with appearance of q wave in the right precordial leads, as a consequence of the initial forces heading below and back. In case of doubt, recording one intercostal space below removes the q wave from pseudo infarction	R/S ratio in V1 and V2 >2	V_5 and V_6: qRs or Rs patterns
	S wave depth in V1 <5 mm	Prolonged R-wave peak time of V_5 and V_6 (> 45 ms to 50 ms)
	Possible small (embryonic) q wave in V2 and V3 or V1 and V2	Disappearance of q wave in V5 and V6 when LPFB occurs
	R wave of V2 ≥15 mm	
	RS or Rs pattern in V2 and V3 (frequent rS in V1) with R wave "in crescendo" from V1 through V3 and decreasing from V5 to V6	

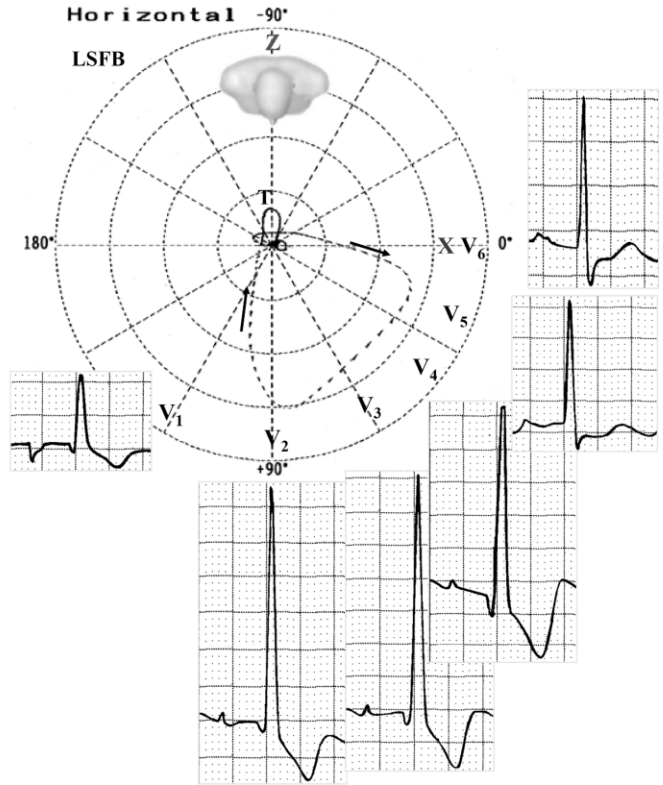
Criteria for the diagnosis of fascicular blocks in the horizontal plane

LAFB (HP)	LSFB	LPFB
	Absence of q wave in left precordial leads V_5, V_6 and I (by absence of vector 1_{AM}). One first needs to exclude ILBBB, CLBBB and WPW	
	Intermittent PAF during hyperacute phase of myocardial infarction **(Madias 1993),**) (**Pérez-Riera 2019**) or during an exercise stress test in patients with severe myocardial ischemia **(Moffa PJ 1997; Uchida 2006; Childers 1973)** and during early atrial extrastimuli with some degree of ventricular aberration **(Hoffman I 1976)**	
	Appearance of intermittent, rate-dependent q wave in V_1 and V_2 (**Pérez-Riera 2019**) (**Pérez-Riera 2019**), Associted with giant J wave (**Pérez-Riera AR 2018**)	

ECG/VCG correlation of LAFB, LPFB (frontal plane) and LSFB (horizontal plane)







Final Clinical Reflections

Exmples of conditions with increased LV mass and thick ventricular walls: *Cardiac amyloid, Fabry disease, Danon disease, Friedreich ataxia, cardiac oxalosis, mucopolysaccharidoses* and **hypertensive heart disease** such as this woman with positive history of systemic hypertension inadequately controlled. This conditon is characterized by symmetrical increase in LV wall thickness, mild LV dilation, normal EF, increased QRS complex, nonspecific ST-T-wave changes, No pattern, predominantly subendocardial, enlarged myocytes with enlarged or replicated nuclei(1) Additionally, this woman has **rheumatoid Arthritis (please see interrogatory)** diagnosed five years ago. This collagenopathy may cause several cardiovascular manifestations: pericarditis, cardiomyopathy/myocarditis, cardiac amyloidosis, coronary vasculitis, conduction disturbances, and others arrythmia and valve diseases, but also congestive heart failure and ischemic heart disease which are found more frequently and are associated with an increased mortality compared with the general population.

In the present case is very important to preform contrast-enhanced cardiac magnetic resonance(1)

1. Rudolph A, Abdel-Aty H, Bohl S, et al. Noninvasive detection of fibrosis applying contrast-enhanced cardiac magnetic resonance in different forms of left ventricular hypertrophy relation to remodeling. J Am Coll Cardiol 2009;53:284–91.

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