Middle age type II diabetic woman with history of syncope of repetition by instable regular wide monomorphic sustained QRS complex tachycardia (WCT)

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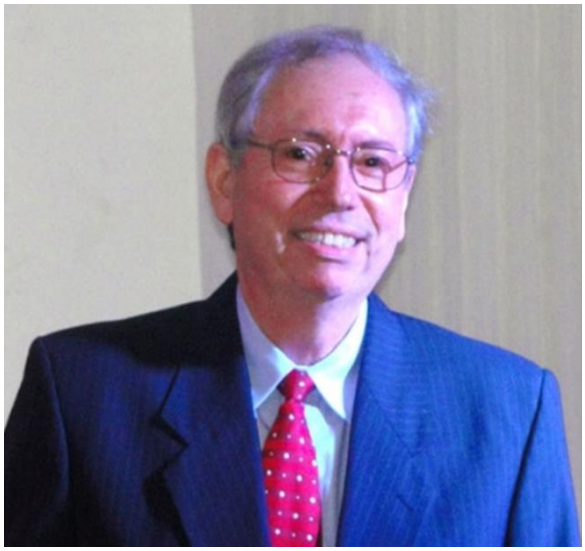


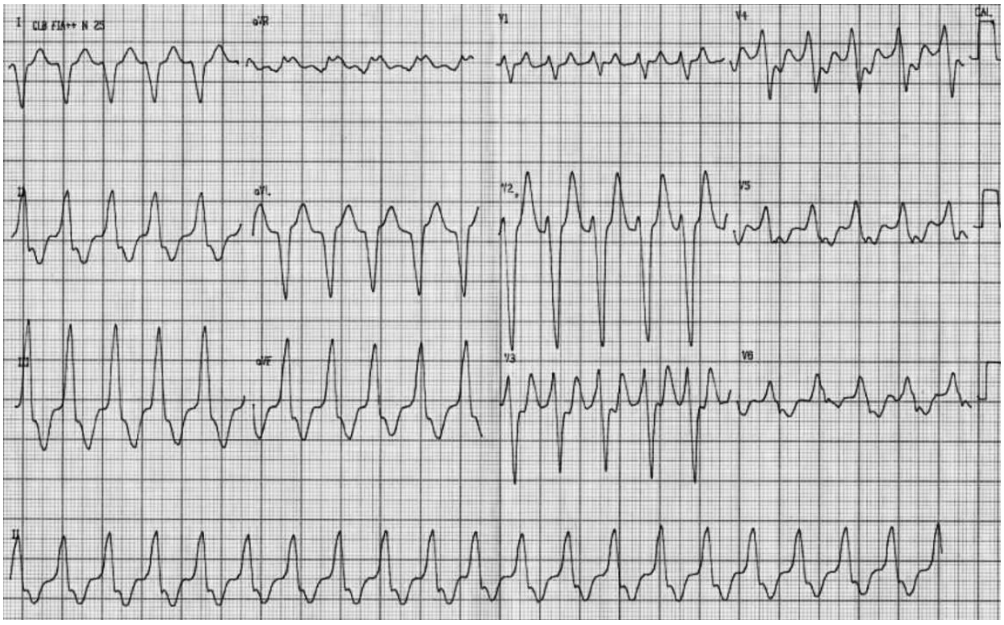
Clinical data

43-year-old patient, female, admitted into the emergency room with symptoms of syncope preceded by tachycardic palpitations. She is diabetic, using metformin and amiodarone. In 2008 she was admitted into the hospital due to arrhythmia investigation. In 2011 she went to the Municipal Hospital with episode of syncope. For 5 years, she suffered occasional palpitations.

Questions

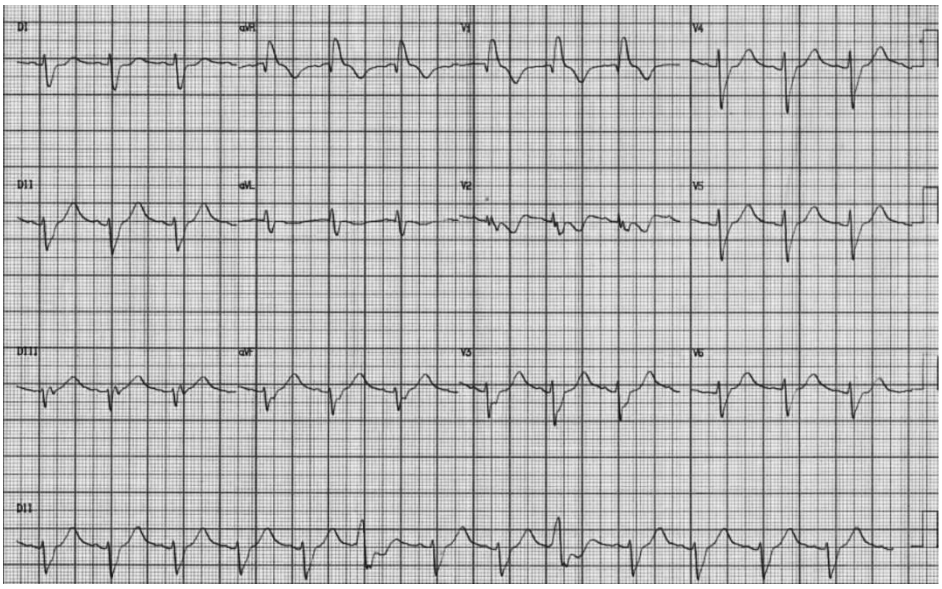
1. Which is the ECG-1 diagnosis? (wide QRS complex tachycardia) and where is the QRS axis located?
2. Which is the ECG-2 diagnosis?
3. Which is the ECG/VCG diagnosis?
4. Which is the possible clinical diagnosis?

ECG-1



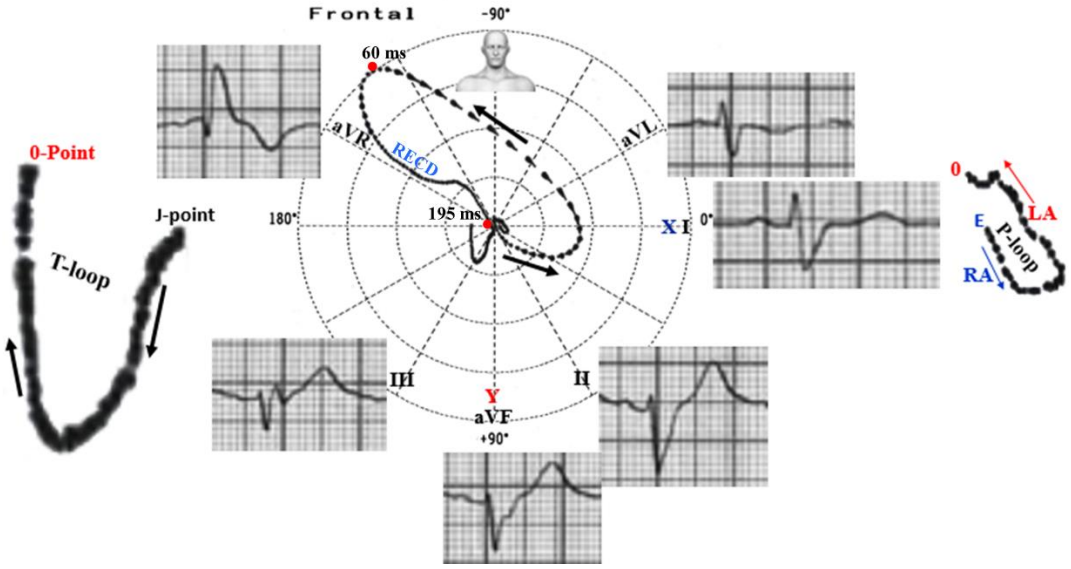
ECG diagnosis:

ECG-2

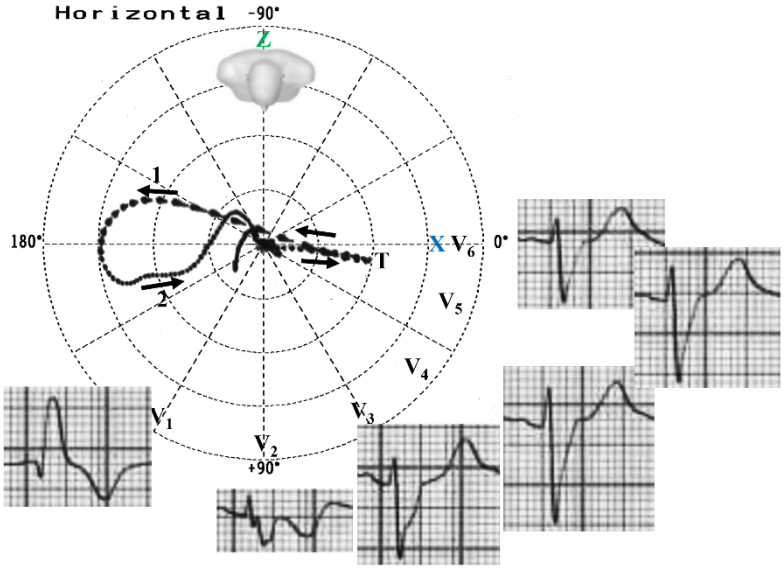


ECG diagnosis:

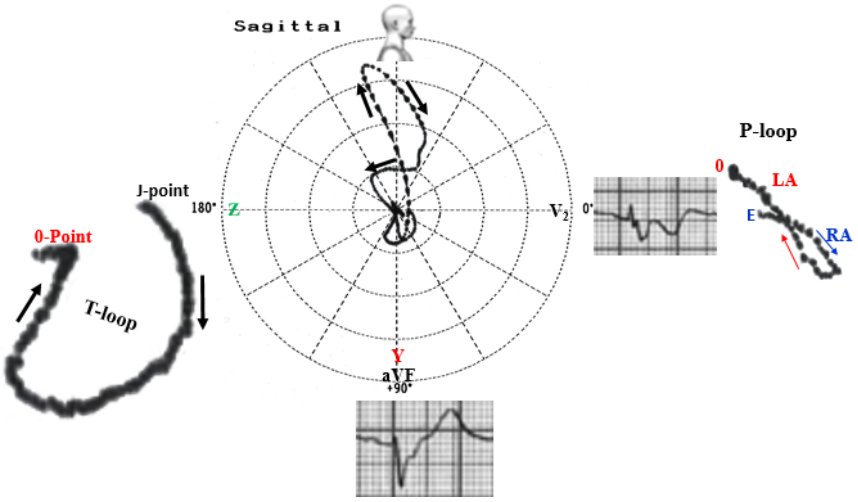
ECG-2/VCG correlation in the frontal plane



ECG-2/VCG correlation in the horizontal plane



ECG/VCG correlation in the right sagittal plane



Diagnosis:

Colleagues opinions

The WCT is VT in origin based on the extreme RAD,Verecky criteria and Brugada criteria in V4.The QRS doesn't show a RBBB pattern as seen in sinus. It looks like there is 1:1 VA conduction. The location is at the lateral basal area of the LV and raises the possibility of origin from the antero-lateral Papillary muscle, left Anterior fascicle,atypical BBRVT or from myocardial source (ie prior MI scar or Chagas disease.

The sinus rhythm tracing shows a RBBB and LAFB pattern note the qR in V1 suggesting an antero septal scar.The bifascicular block pattern is also compatible with Chagasic myopathy,Sarcoid,prior MI.Is there evidence for a skeletal myopathy?,family history of cardiac disease?

Needs thorough evaluation including Echo,Cardiac MRI and EP study.

Spanish

La taquicardia de QRS ancho es una TV. Me fundamento en el extremo desvío derecho del eje, y la presencia de los criterios de Verecky y de Brugada en V4. El QRS no muestra el patrón de BRD como se ve en trazado en ritmo sinusal. Parece que tiene conducción VA 1:1. El foco se encuentra en la zona basal lateral del VI y plantea la posibilidad de origen del músculo papilar anterolateral de la mitral, el fascículo antero-superior izquierdo, BBRVT atípicos o de una fuente de miocárdica (Ej cicatriz infarto de miocardio previo o enfermedad de Chagas).

El trazado en ritmo sinusal muestra patrón de BRD + BDASI (bloqueo bifascicular). El qR en V1sugiera fibrosis septal anterior. También es compatible con miocardiopatía chagásica, sarcoidosis, o infarto previo.

¿Hay pruebas de miopatía esquelética? Hay historia familiar de enfermedad cardiaca? Hay necesidad de evaluar con ECO, CMR e EEF

Melvin Scheinman

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Português

Caros colegas Raimundo e Andrés: O caso é realmente interessante. Vamos às respostas:
ECG 1: Taquicardia Ventricular Monomórfica originada no miocárdio ventricular direito (morfologia de BRE), dissociação A-V, QRS em aVR sem q, sendo totalmente positivo (critério do húngaro Verekai e no plano frontal, critérios de Brugada). Localização do ÂQRS: +130° e para trás. Não me surpreenderia se tratasse de um caso inusitado da síndrome de Brugada, para explicar as síncopes. Só que o que se pode ver, sugere isquemia miocárdica como causa das alterações encontradas.
ECG 2 – Ritmo sinusal, BRD com BDAS e infarto ântero-septal antigo (presença de onda q em V1). Localização do ÂQRS: - 130° e para frente. VCG: BRD tipo Grishman, BDASRE, infarto ântero-septal. Note-se que a alça de QRS não retorna ao ponto de origem, o que sugere isquemia miocárdica. Diagnóstico final miocardiopatia isquêmica, com infarto ântero-septal antigo.
Um grande abraço à dupla dinâmica.

Da República de Curitiba, Brasil Prof. Hélio Germiniani.

English
Dear colleagues Raimundo and Andrés: The case is really interesting. Let's answer:
ECG 1: Monomorphic Ventricular Tachycardia originated in the right ventricular myocardium. (LBBB Morphology, AV dissociation, QRS in aVR initial without q, being totally positive (criterion of the Hungarian Verekai and in the frontal plane, criteria of Brugada). QRS axis at +130° in the frontal plane and to back in HP and RSP plane. It would be surprising if this is an unusual case of Brugada syndrome, which would explain syncope repetition, but what can be seen suggests myocardial ischemia as the cause of the alterations found.

ECG 2: Sinus rhythm, right bundle branch block (RBBB) with left anterofascicular block (LAFB) and anteroseptal MI (by the presence of initial q-wave in V1). QRS axis at - 130° forward.
VCG: Grishman type CRBBB + LAFB + anteroseptal MI.
It should be noted that the QRS loop does not return to the point of origin, which suggests myocardial ischemia.
Final diagnosis: ischemic cardiomyopathy, with anteroseptal MI.
A big hug to the dynamic duet.
From the Republic of Curitiba, Brazil
Prof. Hélio Germiniani MD PhD

Estimado Potro:
En el primer ECG durante el evento de taquicardia con QRS aberrante el eje eléctrico se ubica cercano a los 120º. El diagnóstico de la taquicardia ventricular monomorfa sostenida no observo disociación AV, si observo una deflexión negativa al final del QRS en II lo que levanta la sospecha de conducción VA retrograda en la TV. Presenta inicio R-nadir S >100 ms en derivaciones precordiales y por algoritmo plano Frontal (aVR)(Vereckei) tiene presencia de onda R inicial >40 ms, notch en la rama descendente de aVL y Vi/Vf ≤1. El foco en pared lateral baja.
En el segundo ECG ritmo sinusal, PR normal, bloqueo del fascículo posterior izquierdo y Bloqueo intraventricular compatible con un BRD atípico. Con signos de sobrecarga del VD en V1 y V2 y retraso final del QRS con onda S profunda y empastada. Fragmentación del QRS en cara inferior y V2. Se observan EV aisladas en II con morfología de Rs.
La etiología no impresiona por isquemia miocárdica sino una miocardiopatía de base con mayor compromiso del VD, Por lo que sospecho de miocardiopatía no compactada o displasia arritmogénica del VD con compromiso del VI.
Un cordial saludo
Dr. Martín Ibarrola
FAHA, FESC, FASE
English
Dear Friends:

At the first ECG during the tachycardia event wide QRS, the electrical axis is located close to 120º. The diagnosis is sustained monomorphic ventricular tachycardia. I do not see AV dissociation. There is a negative deflection at the end of the QRS in II which raises the suspicion of retrograde VA conduction. It presents the initial R-nadir S >100 ms in precordial shunts and by frontal algorithm (aVR) (Vereckei) has an initial R wave> 40 ms, notch in the descending ramp of aVL and Vi / Vf ratio ≤1. The focus is located on the low lateral wall.

ECG-2: sinus rhythm, normal PR, left anterior fascicular block and intraventricular block compatible with atypical RBBB, with signs suggesting RV overload in V1 and V2 and final delay of QRS in left leads with deep and broad S waves.

There is fragmentation of the QRS on the inferior leads and on V2.

Isolated PVCs are observed in IBD with Rs morphology.

The etiology does not impress me as being ischemic but a cardiomyopathy with greater RV involvement, so I suspect of non-compacted cardiomyopathy or RV arrhythmogenic dysplasia with LV involvement.

Kind regards

Dr. Martín Ibarrola FAHA, FESC, PHASE

ECG-1: Wide complex tachycardia with right axis deviation and incomplete LBBB. The tachycardia morphology is different from ECG 2 that suggests VT. In general, if an ECG shows RBBB, a second rhythm cannot be SVT due to different morphology, i.e. LBBB. Otherwise, it will have bilateral block. I am not sure if there is retrograde P wave or VA dissociation. In summary, my first guess would be VT. Please do let me know the final diagnosis.

ECG-2: Sinus tachycardia. RBBB, LAD. PVCs in long lead II with post-PVC pause. P-waves are clearly seen. This excludes atrial tachycardia. Mild QRS fractionation in lead V_2.

Dr. Mohammad Shenasa, MD

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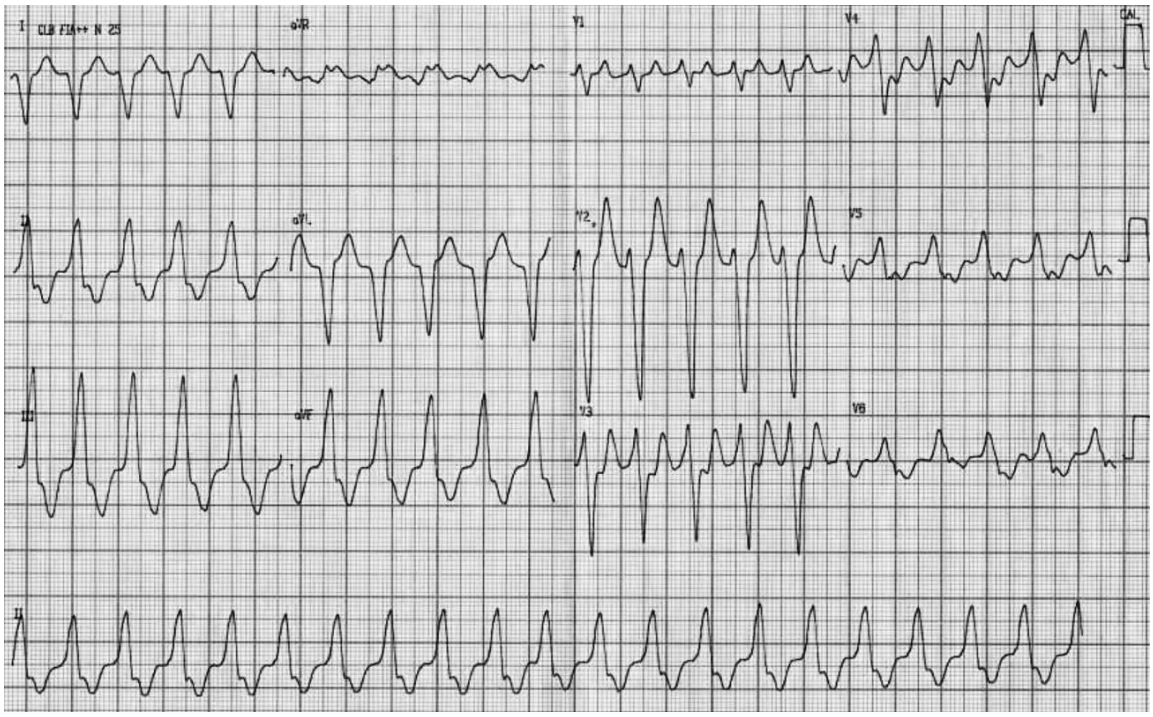
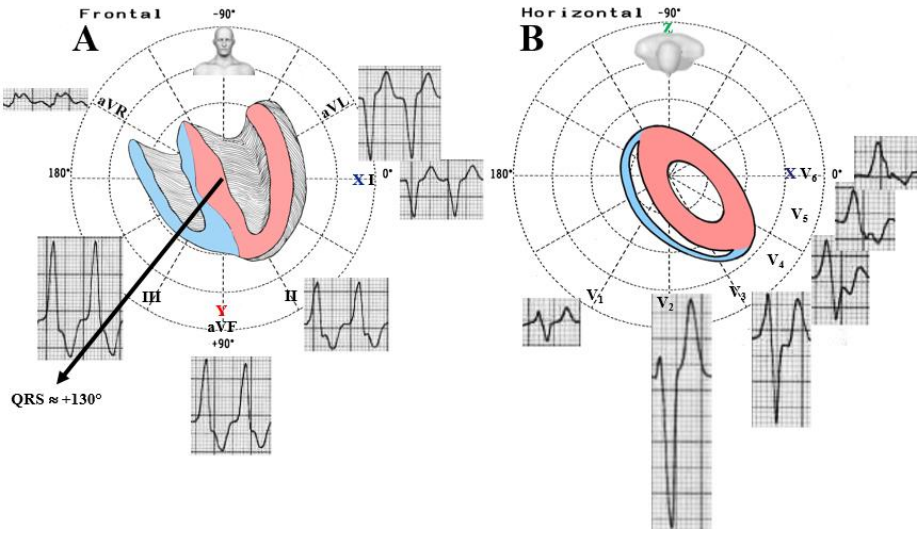
Final comments by Andrés Ricardo Pérez-Riera & Raimundo Barbosa-Barros

ECG-1



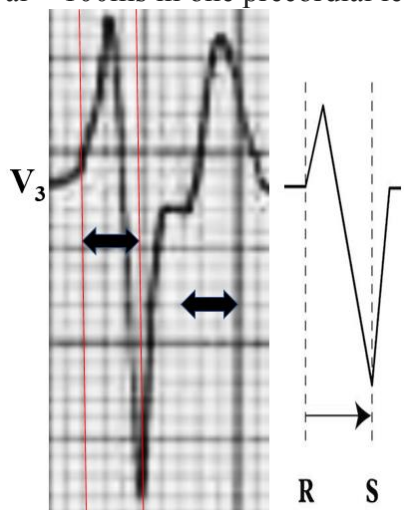
ECG-1 diagnosis: Regular monomorphic wide QRS tachycardia, HR 214bpm, QRS axis +130°, QRS duration =160ms, QRS morphology LBBB-like with negative QRS in aVL. In this case, the QRS axis is at +130°, indicating that the origin focus is in the right ventricular outflow tract (RVOT). In such cases; the QRS has a QS morphology in lead I. QS in I is found in two-thirds of cases of VT originated in the right ventricle. RS interval =120ms in the precordial leads (>100ms = VT), presence of an initial R wave in aVR is highly suggestive of VT (**Vereckei 2008**). 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.



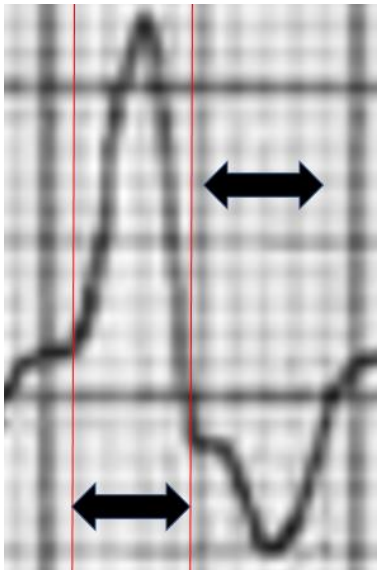
A: ECG-1/VCG correlation in the FP: QRS axis ≈ +130°; **B)** ECG-1/VCG correlation in the HP: LBBB-like morphology, the presence of an inferior axis in a WQRST with a LBBB pattern suggests VT originating the right ventricular outflow tract (RVOT) (**Wellens**

1978), RS interval =120ms in the precordial leads (>100ms = VT). Kindwall uses a value of 70ms. A delay from the onset of QRS until the S wave nadir ≥70 ms (**Kindwall 1988**).

RS interval > 100ms in one precordial lead =VT



RS interval 120ms

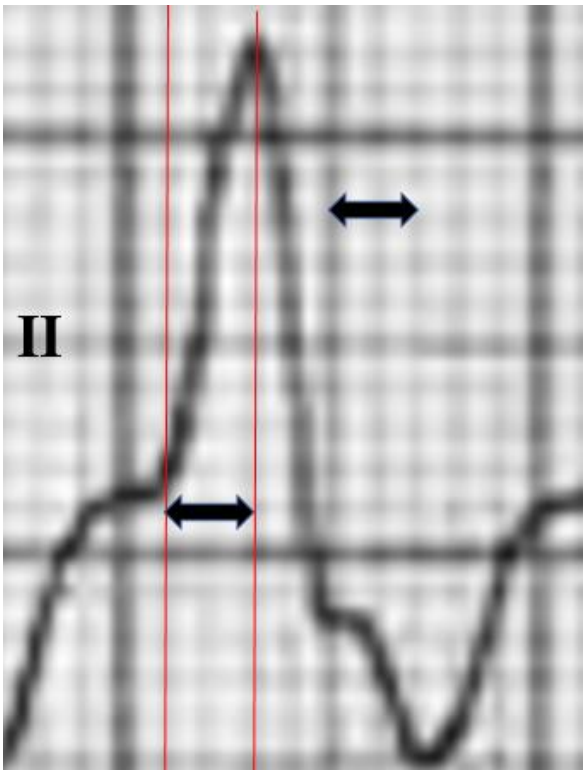


QRS duration = 160ms

Comments: In general, QRS durations tends to be greater in VT than in SVT-A. QRS duration >140 ms with RBBB pattern or ≥160 ms with LBBB pattern suggests VT (**Wellens 1978**). VTs originated 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:

1. Previous BBB in elderly patients with fibrosis of the conduction system and ventricular myocardium;
2. AV conduction through an accessory pathway;
3. During use of class IC antiarrhythmic agents (flecainide, propafenone) (**Wellens 2001**).

Ultra-simple Pava (Pava 2010) criterion or R Wave to Peak Time (RWPT). From QRS onset (R Wave) to the peak of R in lead II ≥ 50ms. Sensitivity: 60%, Specificity: 82%.

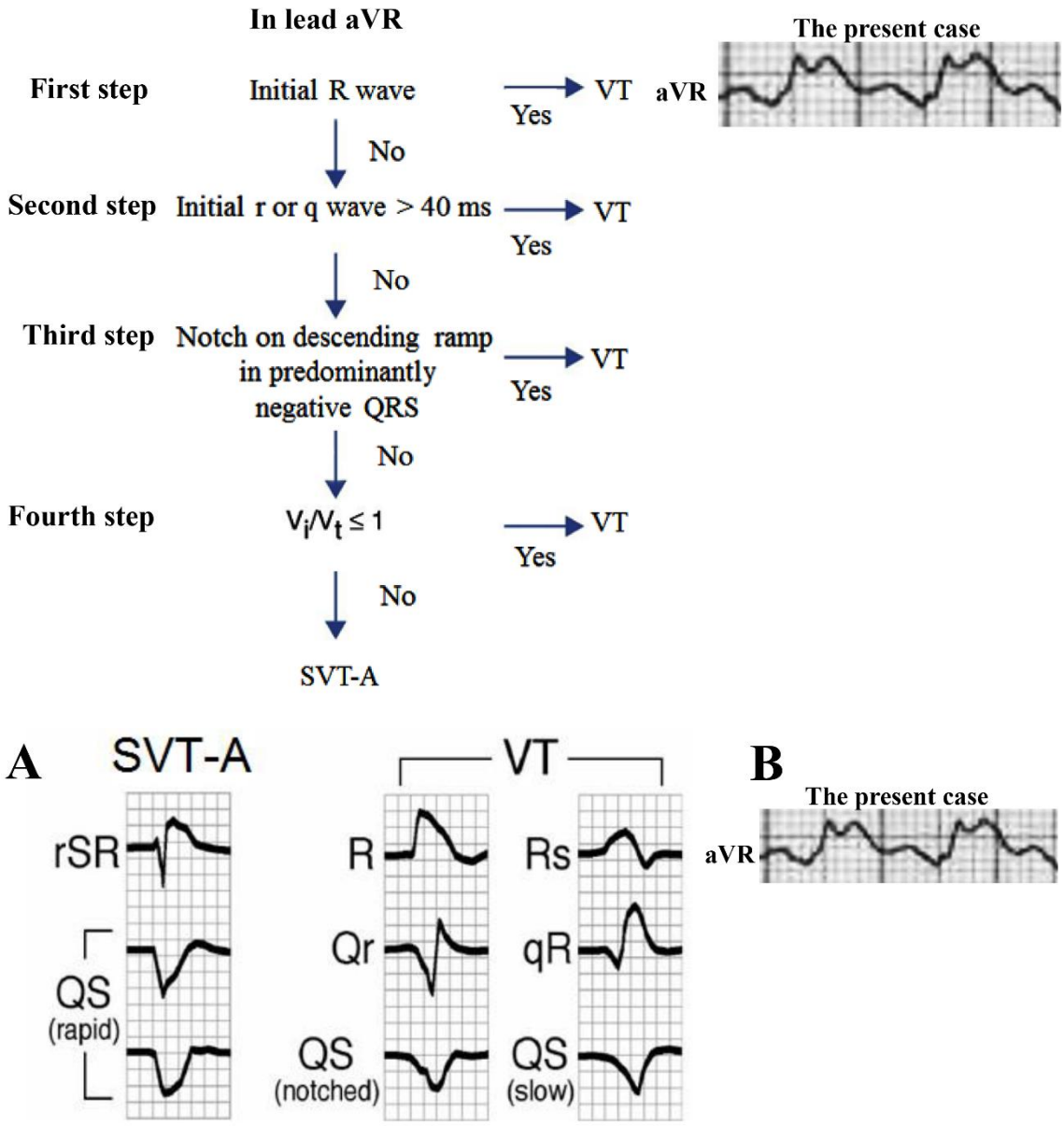


II R Wave to Peak Time (RWPT) ≥50 ms (in this case = 80ms) = VT

New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia by Vereckei

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

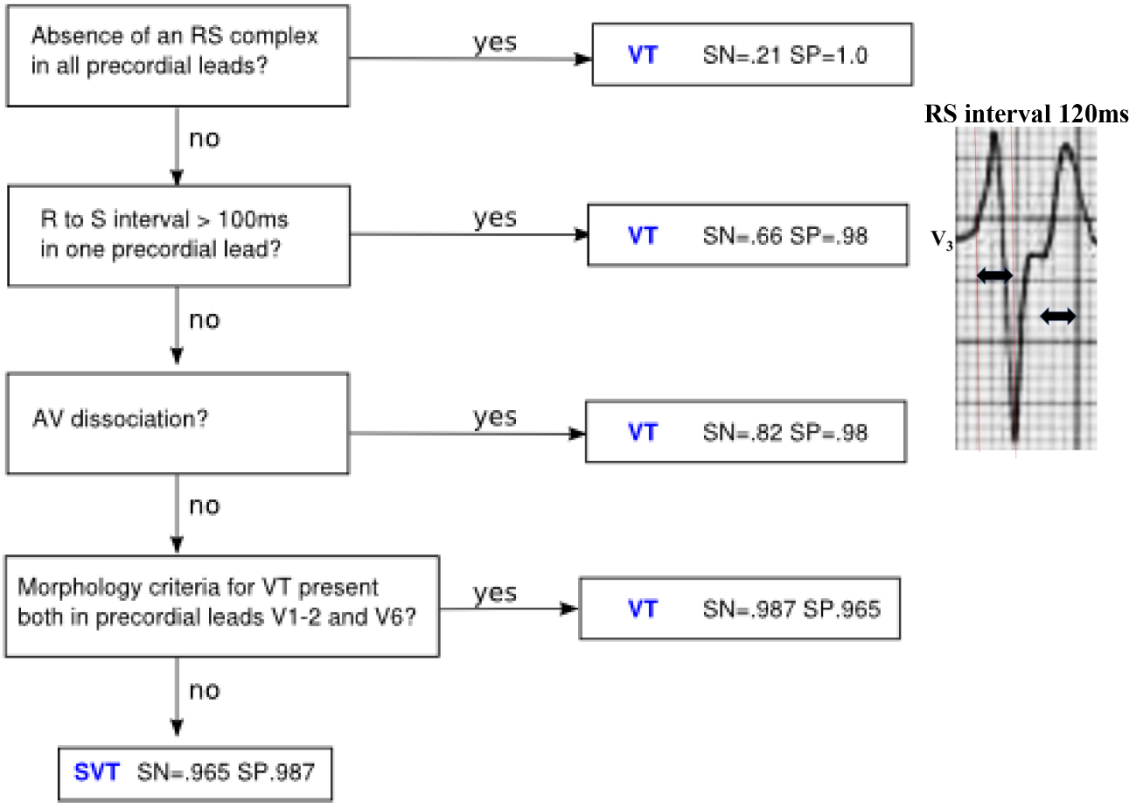
- The exclusive analysis of a single lead (aVR) for the differential diagnosis of wide QRS tachycardias;
- VT is classified in two main groups: A) VT with origin in the inferior and apical regions of the ventricle having an initial R wave in aVR; B) VT with origin in other regions without an initial 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.



A) Representative examples of the most common lead (aVR). ECG patterns taken from real tracings recorded from patients with wide QRS complex tachycardias due to ventricular tachycardia (VT) and supraventricular tachycardia (SVT) superimposed on a grid (small box 40 ms). Descriptions are given to the left of each QRS complex. Patterns seen in VT cases are shown in the left; SVT examples are at right. "Notched," "slow," and "rapid" refer to the type of descent of the initial portion of the QRS complex from onset to nadir. **B)** The present case.

Brugada algorithm

Brugada algorithm (**Brugada 1991**) is the most commonly used algorithm having a potential sensitivity (SN) of 99%; and specificity (SP) of 97%.



Observation: In the second step, we observe clear VT criterion.

Miller and Das practical approach to evaluate the WCT for the most specific criteria in some sequence. When one such criterion is met the diagnosis is made, as follow:

Step 1: AV relationship (if dissociated, VT is diagnosed; if not, proceed to next step)

Step 2: Rightward superior axis (if present, VT is diagnosed; if not, proceed to next step)

Step 3: V_1/V_2 ratio (if greater than 1, SVT-A is diagnosed; if not, proceed to next step)

Step 4: Precordial RS pattern (if absent, VT is diagnosed; if not, proceed to next step)

Step 5: Precordial RS pattern (if R is present an interval greater than 10 ms, VT is diagnosed; if not, proceed to next step)

Step 6: In LBB type WCT, R wave less than 30 ms or R onset to S nadir less than 60 ms in V1 (is present SVT-A is diagnosed)

This process continues in like manner for remaining, less specific criteria (**Miller 2009**).

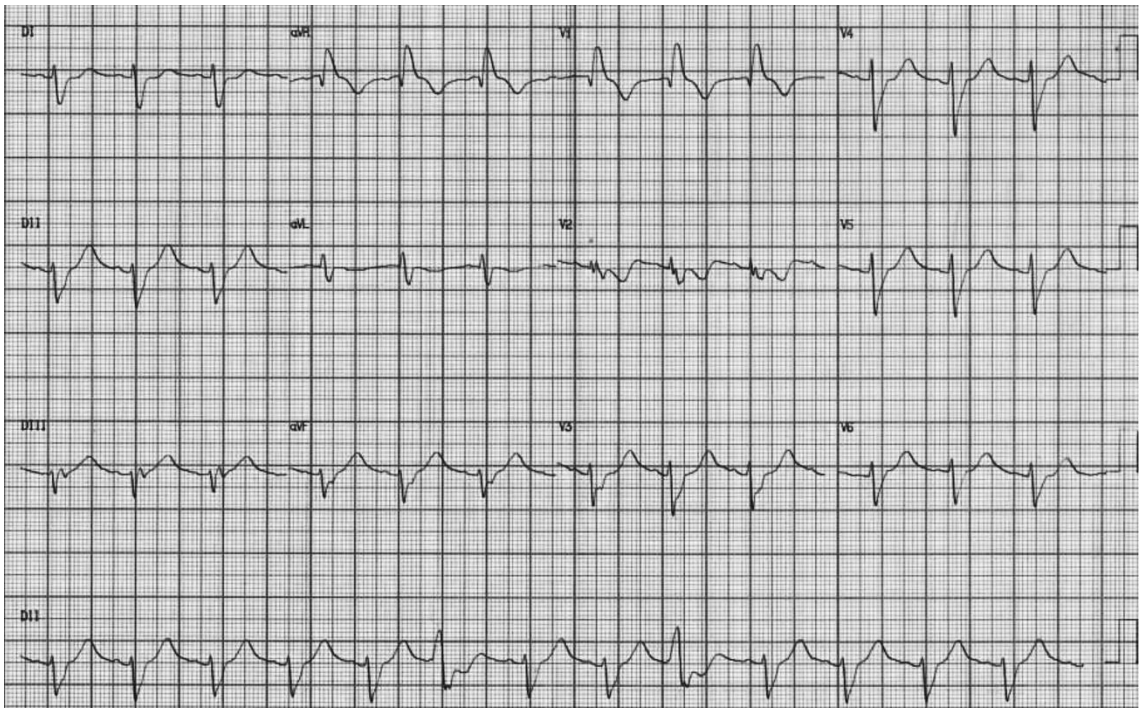
The table below summarizes the main differences between VT and SVT-A.

	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 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 of HCM, ARVD/C, long QT syndrome and Brugada syndrome	Strongly suggestive	No
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 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
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) (Fig. 30)	If present, it is diagnostic.	Lower.

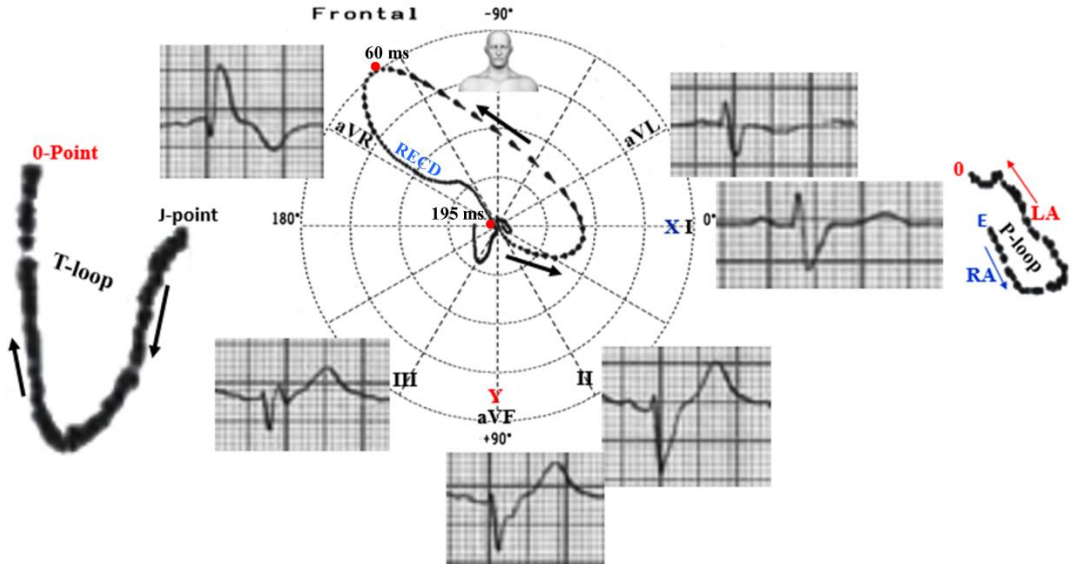
Notch near the nadir of the S wave (sign of Josephson) (Figure 31)	Characteristic	Absent.
QRS complexes of the R or Rs type	Diagnostic	No
Initial q or r wave with duration >40 ms in aVR (qR or rS)	Diagnostic	No
Pattern matching in precordial leads	Strongly suggestive.	No
Presence of fusion beats	Strongly suggestive.	No
Presence of capture beats	Strongly suggestive.	No
Second-degree ventricular-atrial block	Characteristic when present: QRS/P ratio; 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 2009)	Suggestive.	>1

ECG-2



ECG-2 diagnosis: see comments ECG/VCG diagnosis

ECG-2/VCG correlation in the frontal plane



Very prolonged Right End Conduction Delay (**RECD = 135 ms!!** from 60 ms to 195 ms), QRS morphology compatible with **RECD** Type IA of our classification (see explanation in the next table/figures): very wide QRS duration = 195 ms, QRS loop predominantly located in the right superior quadrant (QRS axis ≈ -140° with counterclockwise rotation (CCW) and very prolonged **RECD** located in the right superior quadrant near aVR (-150°). Very similar pattern to LAFB (see the differential diagnosis between **RECD** type IA and LAFB in the table/figures below). We will explain what means **RECD** type IA in sequence.

Differential diagnosis between RECD type IA and LAFB

	RECD Type IA	LAFB
Depth of S wave in II and III	SII > SIII (inconstant)	SIII > SII (inconstant)
I and aVL	Rs	Qr
Prominent and broad R wave in aVR	Present and characteristic: QR or qR.	Absent: Qr or QS.
Vector of initial 10 to 20 ms	Downward and to the left (inconstant)	Downward and to the right (inconstant)
Rapid passage from left to right	Yes	No
RECD	In the right superior quadrant.	With or without delay, above and to the left.
Triphasic pattern in V_1 or V_1 and V_2.	Very frequent.	Possible. Final R' wave or r' wave of V_2 is greater than in V_3R and V_4R, indicating that the final forces are heading predominantly to the left.

Electrovectorcardiographic characterization of Type IA (the present case)

I. Initial 10 to 20 ms vector directed downward and leftward (differential diagnosis with LAFB: directed to right and downward);
II. SÂQRS with extreme deviation in the right superior quadrant;
III. QRS loop of counterclockwise rotation in the frontal plane;
IV. Rapid passage from left to right of the QRS loop;

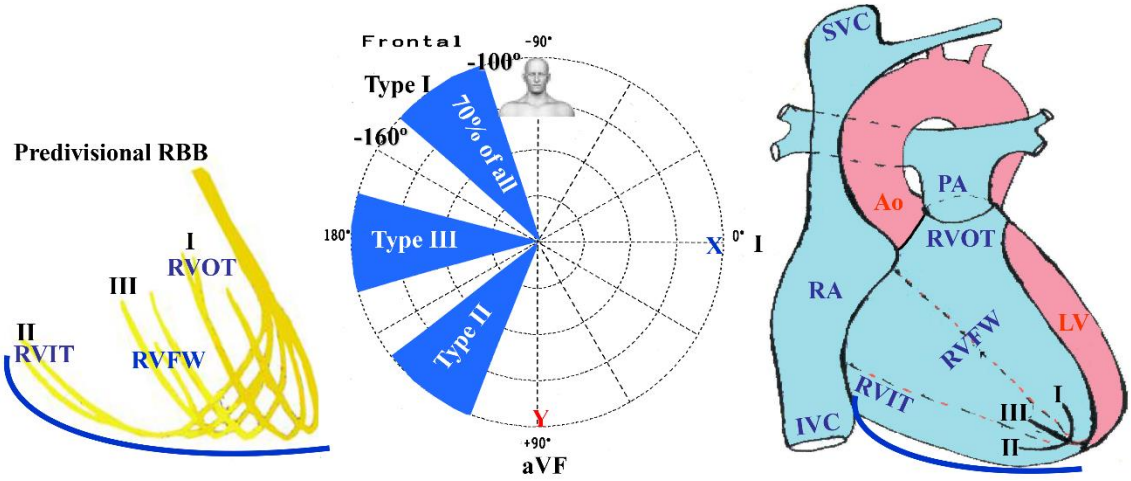
V. **RECD** ≥30 ms (≥15 dashes) located in the right superior quadrant between –100º and –160º; or greater in this case: **RECD** is enormous!!!!!: 135 ms!!!

VI. QRS complexes predominantly negative in inferior leads: prominent S wave in these leads;

VII. SII>SIII: useful for the differential diagnosis with LAFB;

VIII. R wave of aVR: prominent and/or broad. aVR of the qR or QR type, with R being frequently broad.

VCG types of RECD in the Right Ventricular Free Wall (RVFW)



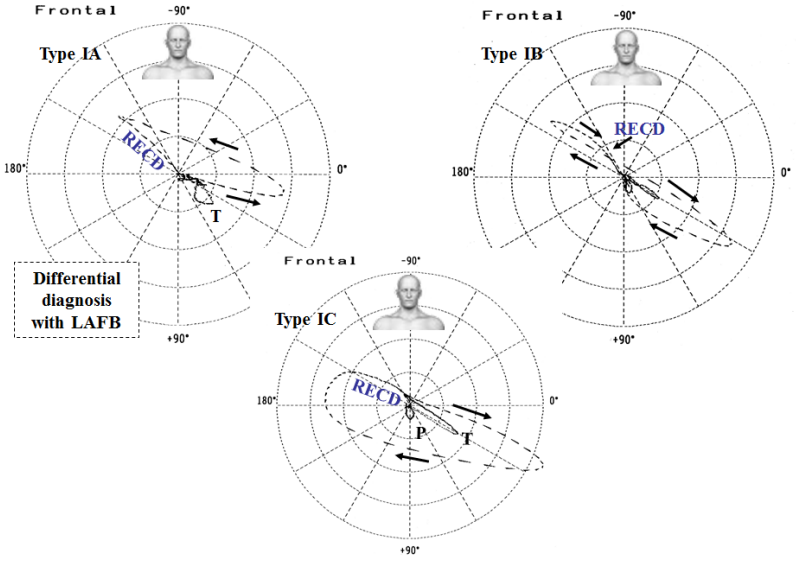
Type I – RECD in the territory of superior or subpulmonary contingent **(RVOT)** of fibers of RBB in the Right Ventricle Free Wall **(RVFW).** This variant corresponds to the present case.

Type II – RECD in the territory of inferior or posteroinferior contingents **(RVIT)** of fibers of RBB in the Right Ventricle Free Wall **(RVFW)**

Type III – RECD in the territory of middle contingents of fibers of RBB in the Right Ventricle Free Wall **(RVFW).**

VCG Type I RECD variants according to QRS rotation in the FP: Types IA, IB and IC

1) RECD Types IA, B and C

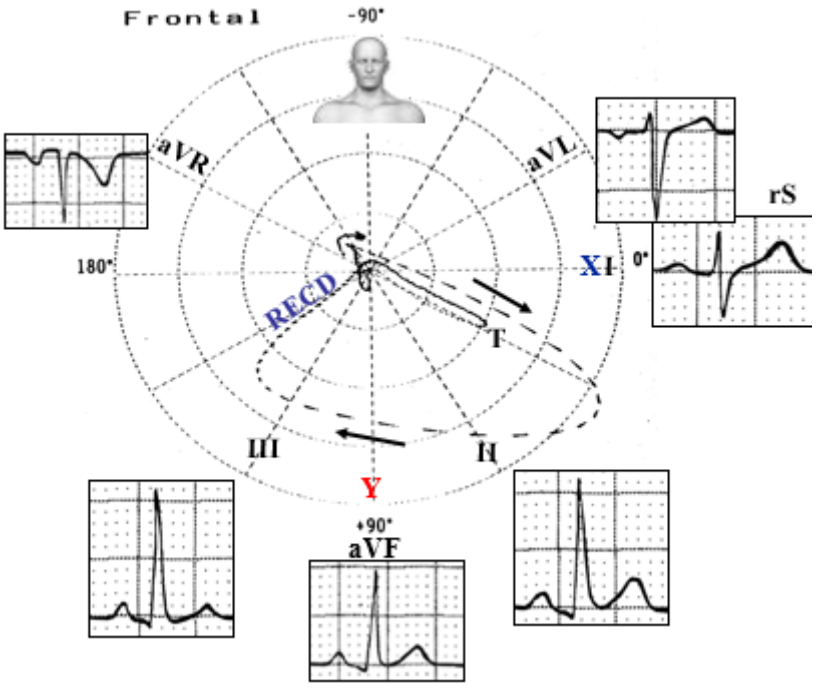


RECD type I are characterized by presenting right end conduction delay **(RECD)**, located in the right superior quadrant, corresponding to the territory Right Ventricular Outflow Tract (RVOT) between -100° and -160° in the FP. The location of the delay justifies the recording of prominent R waves with a certain delay in the lead that faces the RVOT: aVR. Additionally, notched in the apex of the R wave, which prolongation of ventricular activation time or R-wave peak time deflection (≥ 50 ms) circumscript only in the right unipolar lead that explores the RVOT affected region (aVR) (**de Micheli 1987;2009**). The superior infundibular subpulmonary region of the RV is the last one to activate, generating a final basal vector (basal vector 4d), heading upward and to the right between -100º and -160º in the FP. This is the most frequent variant of **RECD** (70% of all our cases). By the aspect and rotation of the QRS loop in the FP, we propose to divide the **RECD** types I into three variants:

- **RECD** type IA: CCWR of the QRS loop, QRS axis with superior shift and differential diagnosis with LAFB
- **RECD** type IB: QRS loop with in eight rotation
- **RECD** type IC. QRS loop with clock wise rotation of the QRS loop

2) RECD Type II

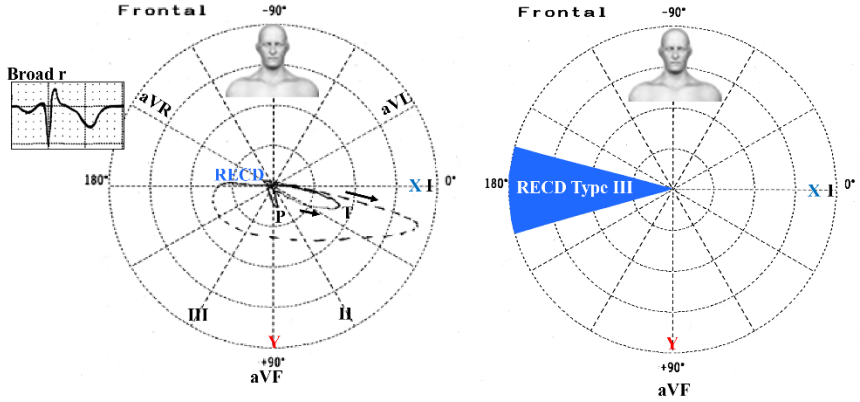
Characterized by presenting **RECD** located in the right inferior quadrant in the territory of the Right Ventricular Inflow Tract (RVIT)**.** The differential diagnosis occurs with left posterior fascicular block (LPFB). Many of the cases described in literature as LPFB are, the way we see it, **RECD** Type II, and since their electro-VCG differences are very subtle, the diagnosis must always be clinical-electro-vectorcardiographic.



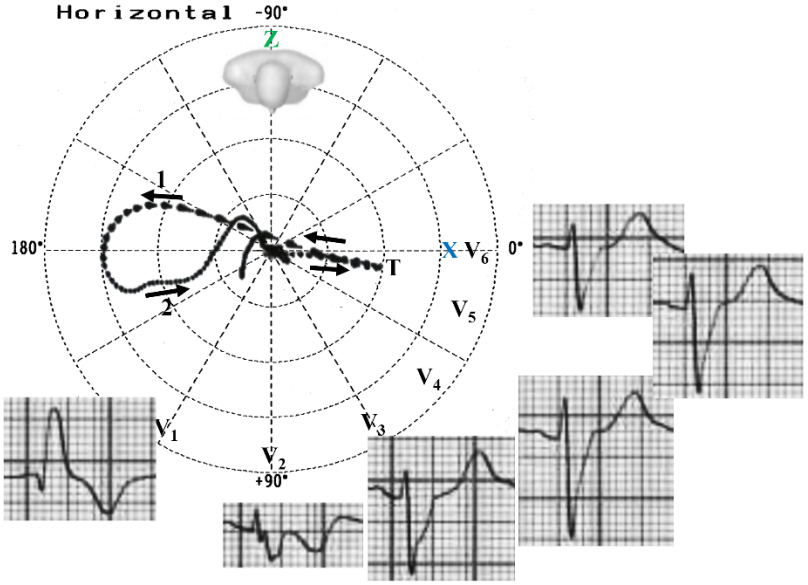
RECD type II: RECD located in the right inferior quadrant in the territory of the Right Ventricular Inflow Tract (RVIT)**.** SÂQRS: +95º. SI-RII-RIII pattern (RIII < 15 mm). I and aVL: rS. II and III: qR. The descending ramp of R wave is slightly slow. It may present differential diagnosis with LPFB.

3) RECD type III

Characterized by **RECD** located very close to right extreme of orthogonal X line (±180°).



ECG/VCG correlation in the horizontal plane



ECG-2/VCG diagnosis:

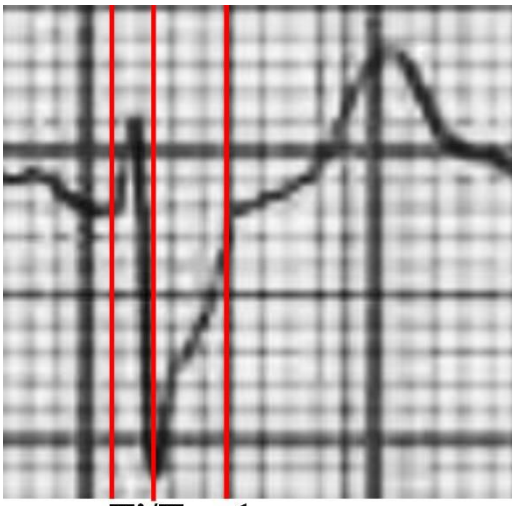
I. Efferent limb behind the orthogonal X lead (0/±180°): type I or Grishman type complete RBBB.

II. Afferent limb in the right anterior quadrant in front of X lead, with significant **RECD** (135 ms) typical of CRBBB.

III. QRS loop totally dislocated to the right quadrants.

IV. qR pattern in V1*. The pattern is caused by the disappearance of the first vector of the middle third of the left septum, Peñaloza.and Tanchesi vector (**Penaloza 1955**) or 1_{AM} vector (first anteromedial vector). The tumor occupies the middle third of the septum, eliminating the first vector of QRS.

V. Unlike a true anterolateral infarction, the delay is not in the initial part of the depolarization (Ti) but in the end of it (Te). The phenomenon can be observed in both ECG and VCG. In VCG, the final portion of QRS loop with **RECD** has 135 ms. And the initial portion only 60 ms.



Ti/Te <1

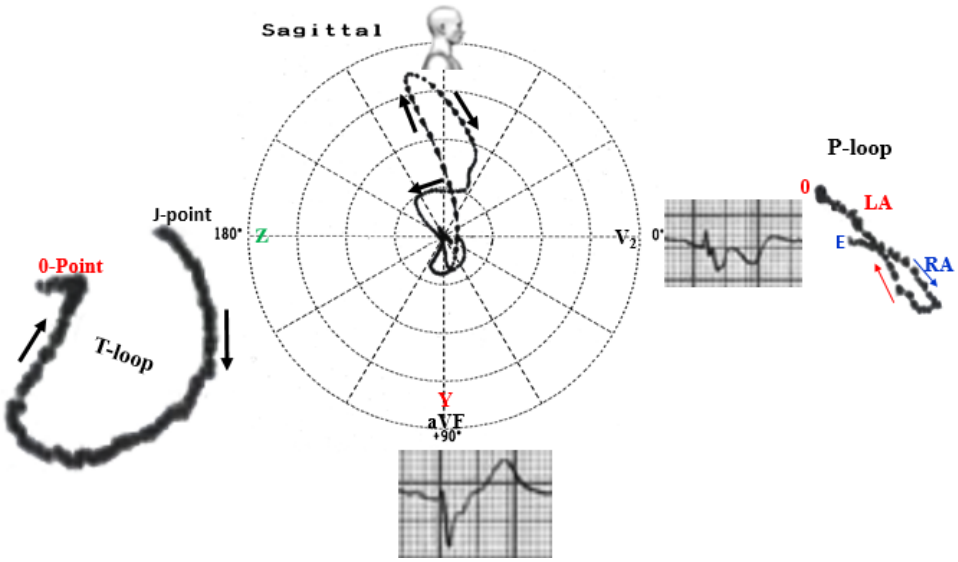
VI. rS from V1-V6.

* **Possible causes of qR pattern in V_1**

1. Systolic RVH with strain pattern: suprasystemic right intraventricular pressure. E.g.: PS with the form of a point or severe (see the previous ECG).
2. Important dilatation of the right atrium: E.g.: anomaly of Ebstein (**Lowe 1968**), tricuspid insufficiency. The volumetric increase of the RA gets it closer to the exploring electrode of V_1, registering negatively initially in this lead, because the electrode records the epicardial morphology of the RA.
3. RBBB associated to anterior myocardial infarction.
4. RBBB with isoelectric initial r wave in V_1
5. Situs inversus: ventricular inversion: inverted septal activation.
6. Pectus excavatum
7. Septal tumor (the tumor destroys the first vector when located in the middle third of the interventricular septum, justifying the qR pattern in V1).

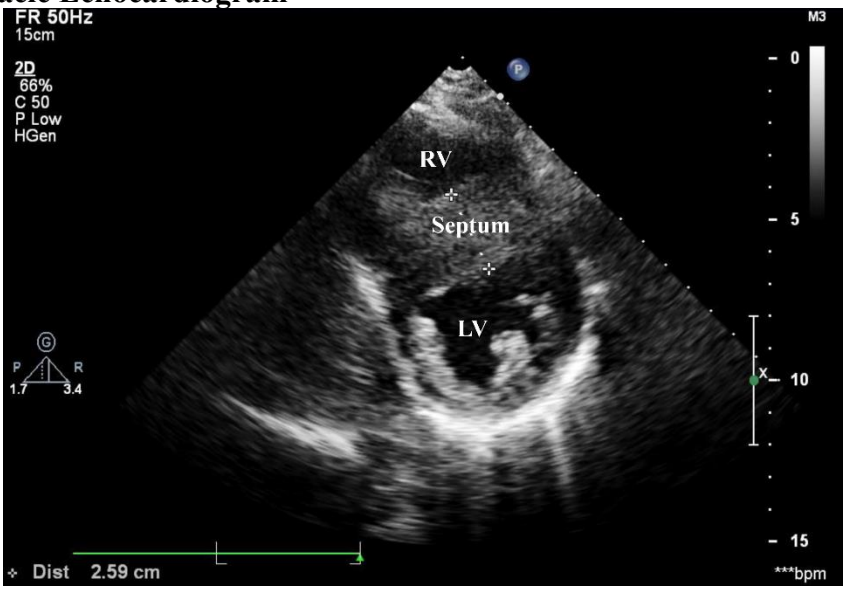
Conclusion: pseudo electrically anterolateral inactive area. **RECD** type 1A of our VCG classification (**RECD** in RVOT and CCWR in the FP).

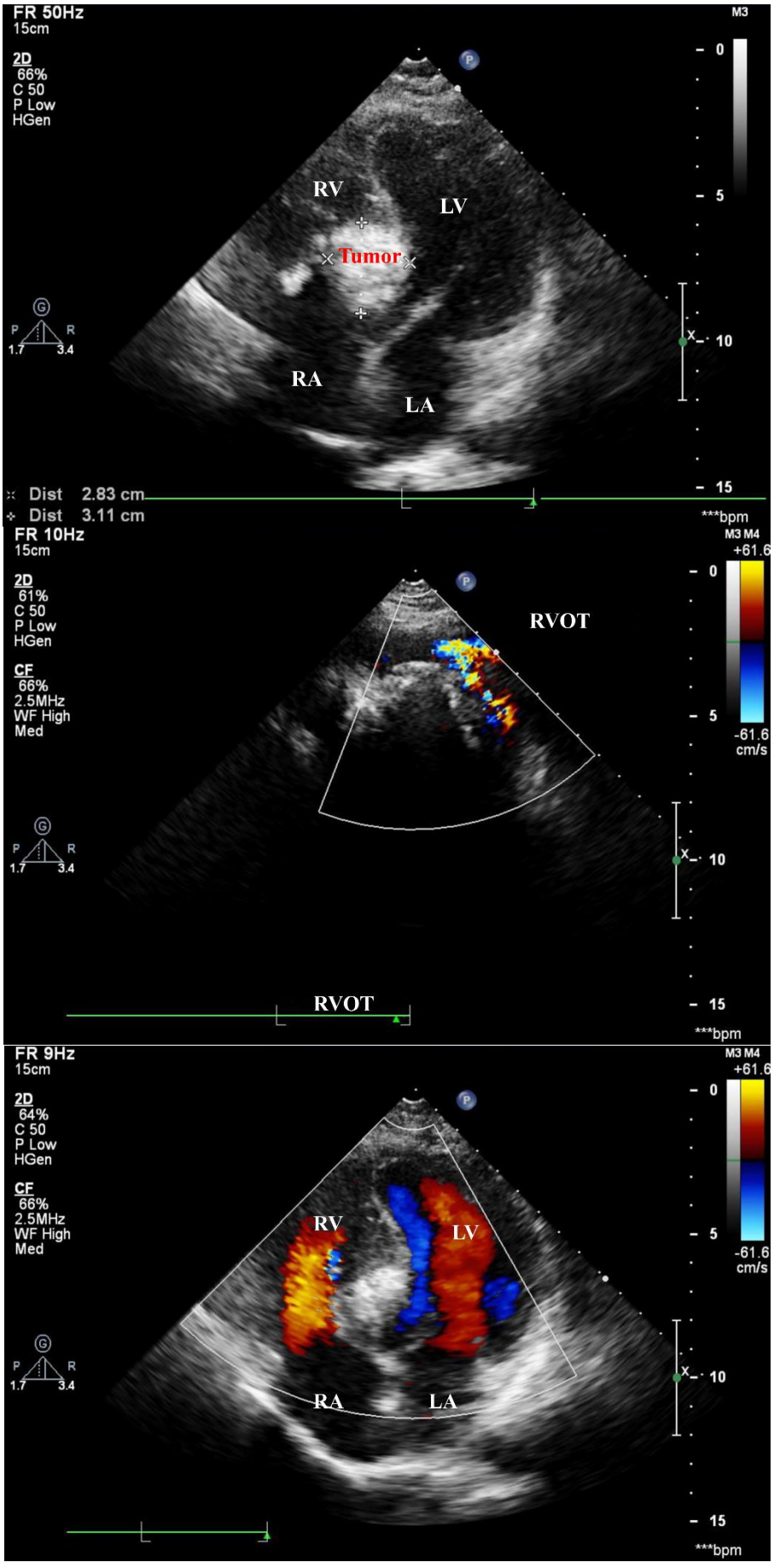
ECG/VCG correlation in the right sagittal plane



ECG-2/VCG diagnosis: QRS loop with rotation in eight and almost totally dislocated upward. End Conduction Delay (ECD) located in the anterosuperior and posterosuperior quadrants.
T-loop with rounded shape and similar slow speed in both efferent and afferent limbs.
J-point and 0 point are far from each other, concordant with ST segment depression ECG in the inferior leads.

Transthoracic Echocardiogram

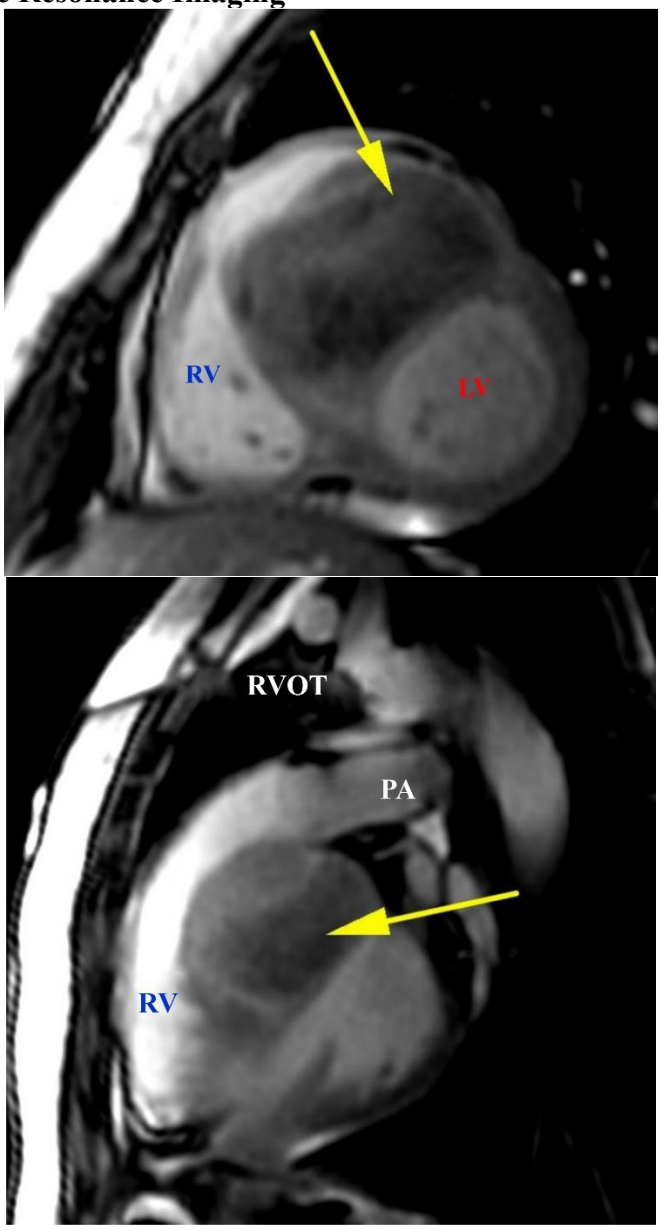



FR 50Hz
15cm
2D
66%
C 50
P Low
HGen
M3
RV
LV
Tumor
RA
LA
Dist 2.83 cm
Dist 3.11 cm
***bpm
FR 10Hz
15cm
2D
61%
C 50
P Low
HGen
CF
66%
2.5MHz
WF High
Med
M3 M4
+61.6
-61.6
cm/s
RVOT
RVOT
***bpm
FR 9Hz
15cm
2D
64%
C 50
P Low
HGen
CF
66%
2.5MHz
WF High
Med
M3 M4
+61.6
-61.6
cm/s
RV
LV
RA
LA
***bpm

Flow analysis by Doppler
Mitral valve regurgitant flow area compatible with mild insufficiency.
Flow analysis by mapping in color
Mitral valve regurgitant flow area compatible with mild insufficiency.
Conclusion

- Visceroatrial situs solitus. Levocardia, apex to the left.
- Normal-sized cardiac chambers.
- Discrete mitral regurgitation.
- Hyperechoic image, with rounded shape, 3.5x3.2 cm located in the mid-basal portion with the interventricular septum, extending into the RV inflow and outflow tracts.
- Systolic gradient in the right ventricular outflow tract of 23 mmHg.
- Preserved contractile function.

Cardiac Magnetic Resonance Imaging



Presence of large tumor mass (arrow) in the interventricular septum (mid-basal), 70 mm x 61 mm x 44 mm, with features compatible with fibroma.
Normal biventricular systolic function.
Obstruction in both ventricular outflow tracts (right and left).

CMRI
Indication: Primary cardiac tumor?
Technique:
Test performed in a patient in regular heart rhythm. Multiple acquisitions were made by the following techniques: double inversion recovery, triple inversion recovery and cine-FIESTA, in the coronal, axial, short-axis, long-axis, 4-chamber, and 2-chamber planes, with use of contrast (gadolinium).
Commentaries:

- Situs solitus with levocardia
- Normal systemic and pulmonary venous drainage, with no obstructions or dilatations observed.
- Atrioventricular and ventricular-arterial matching
- Preserved IAS and IVS
- Cardiac chambers with normal dimensions: LVDD 35 mm, LVSD 21 mm, IVS 6 mm, posterior wall 6 mm, RV 28 mm.
- Presence of large intramural tumor mass affecting the mid-basal portions of the interventricular septum, with a size of 58 mm x 54 mm x 41 mm, with signal similar to the myocardium and mild heterogeneity in images studied in T1, non-enhancing mass, presenting signal reduction in images studied in T2. These findings suggest diagnosis of Fibroma. There is no evidence of malignancy (limited mass, absence of satellite lesions, absence of pericardial effusion and lymphonodular compromise). The mass produces obstruction in both ventricles outflow tracts.
- Normal biventricular systolic function (LVEDV 96 ml, LVESV 28 ml, LVEF 71%, RVEDV 88 ml, RVESV 31 ml, RVEF 63%).
- Normal cardiac leaflets
- Normal pericardium
- Ascending aorta, arch and descending aorta of normal sizes.

Conclusions
Test compatible with primary cardiac tumor located in the interventricular septum (58 mm x 54 mm x 41 mm), causing outflow tracts obstruction in both ventricles, and with aspect suggesting fibroma.

Theoretical considerations
In general, primary tumors of the heart are rare. In autopsy studies, the overall prevalence ranges from 0.002% to 0.33%, with about 75-79% of these considered benign (**Ren 2016; Yin 2016**).
Although most tumors of the heart are benign, because of their malignant potential, the risks secondary to impaired cardiac function (eg, congestive heart failure, inflow/outflow tract obstruction, such as the present case), conduction system involvement, and/or peripheral embolism mandate prompt evaluation and definitive treatment.
It has been reported that patients with benign cardiac tumors are at increased risk of first ischemic stroke, particularly patients younger than 50 years (**Lai 2015**).

The most common primary cardiac tumor is a myxoma. Other less common neoplastic tissue types occur; each has distinguishing characteristics that often aid in accurate preoperative diagnosis or diagnosis prior to death. A definitive diagnosis is important because some cardiac tumors can be malignant or, more commonly, can represent metastasis from a distant primary tumor.

Fibromas most commonly involve the intraventricular septum or left ventricular free wall. Less than 10% of reported cases have atrial or great vessel involvement. Unlike myxomas, tumor embolization is uncommon. Tumor growth can displace or directly involve mitral and aortic valves and result in hemodynamically significant valvular stenosis or regurgitation.

Symptoms are typically secondary to adverse effects on normal left ventricular geometry, filling, and ejection. Additionally, arrhythmias, particularly sudden death and abnormal atrioventricular conduction, are common because of tumor disruption of the nodal or septal conduction tissue. Benign cardiac tumors can also present without symptoms as incidental findings found on cardiac studies investigating other pathology.

Benign cardiac tumors are extremely rare. Of all primary cardiac tumors, 75% are histologically benign. Fibromas (4-6%) occur less commonly.

Despite complete resection, patients are still at risk for sudden death due to damage to the conduction system. In the present case, we propose percutaneous alcohol embolization because both LVOT and RVOT are narrowed with clinical behavior that resembles obstructive hypertrophic cardiomyopathy.

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