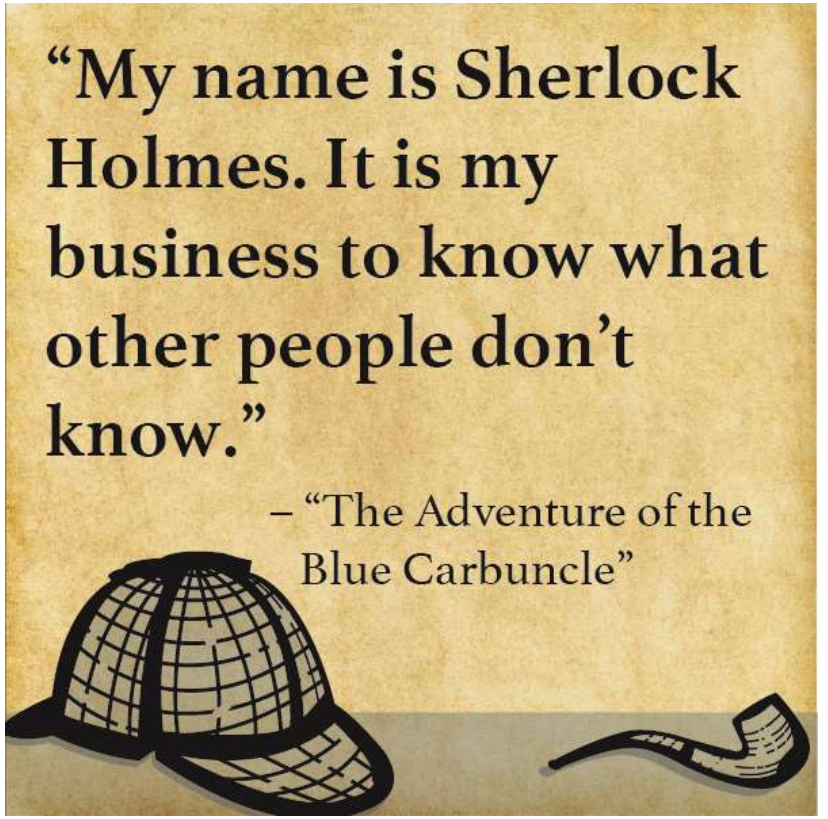
A monotonous electrovectorcardiographic pattern gives us the diagnostic clue

I do not understand anything about ECG/VCG....



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https://ekgvcg.wordpress.com

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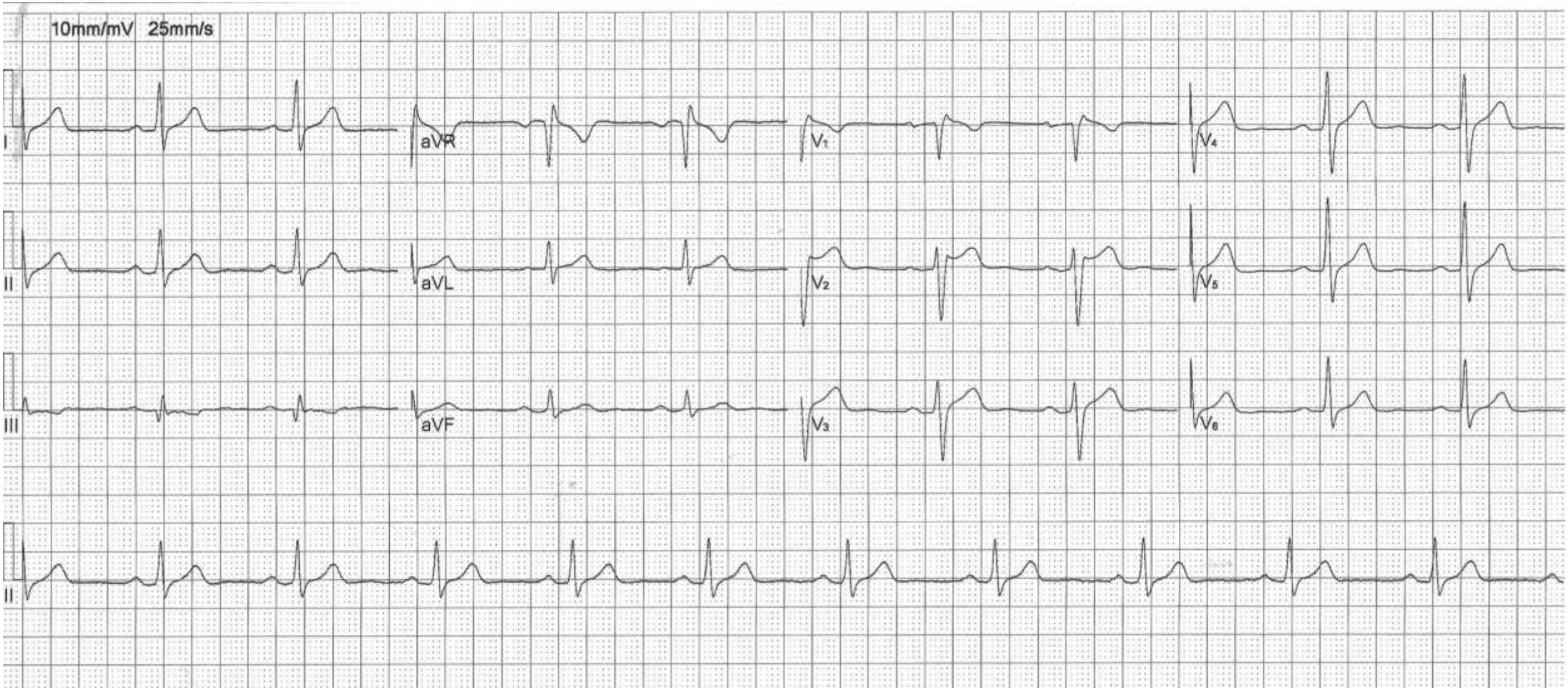
Relato de caso

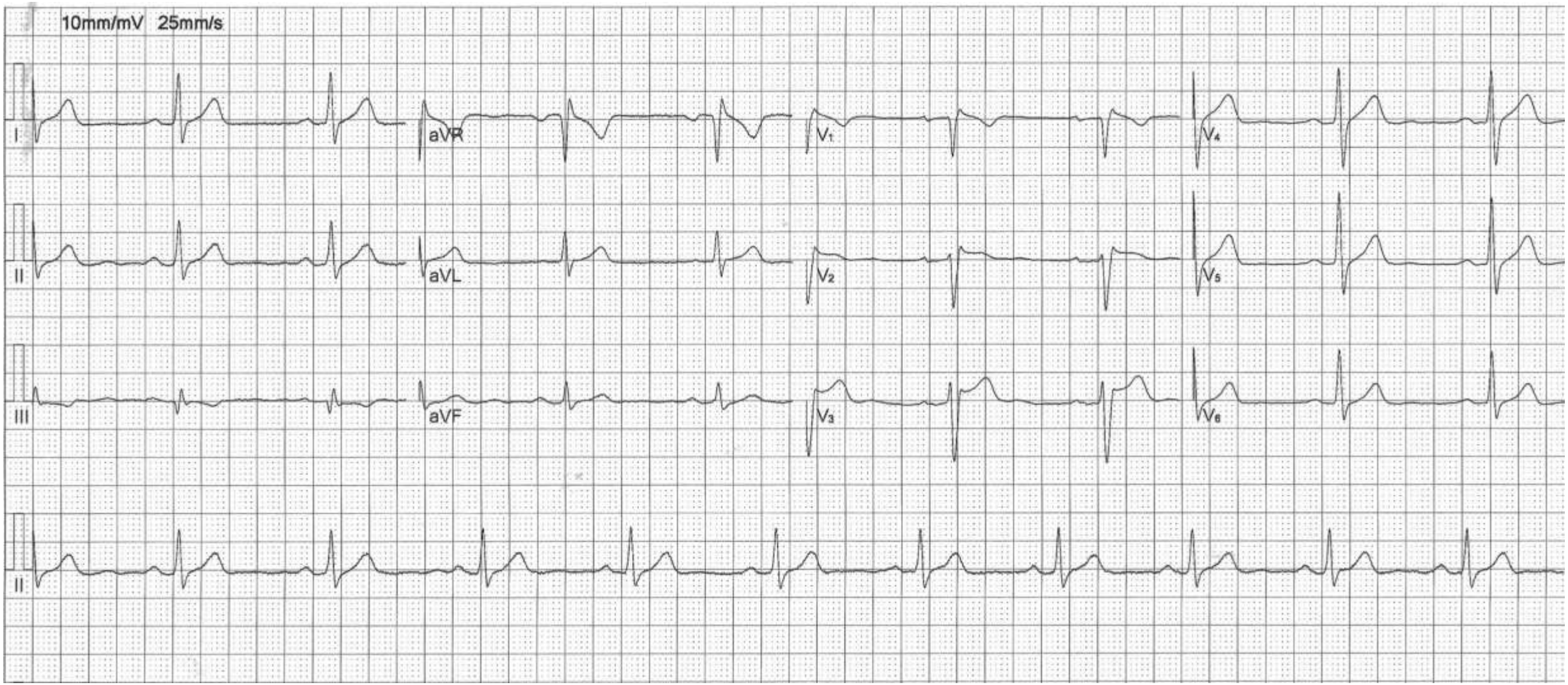
Masculino, asiático, 55 anos, hipertenso em uso de losartana.
Vários síncopes, todos em repouso ou durante o sono & dor precordial atípica.
Relato de um episódio de taquicardia ventricular polimórfica em repouso com reversão após cardioversão (não temos a documentação gráfica)
Foi inicialmente conduzido pela equipe de emergência como uma síndrome coronariana aguda.
Exame físico normal
Eco normal coronariografia normal troponina normal.
História familiar: um irmão faleceu enquanto dormia aos 55 anos. Quais os elementos vetorcardiográficos para o diagnóstico? (Europace. 2012 Jun;14(6):889-97).

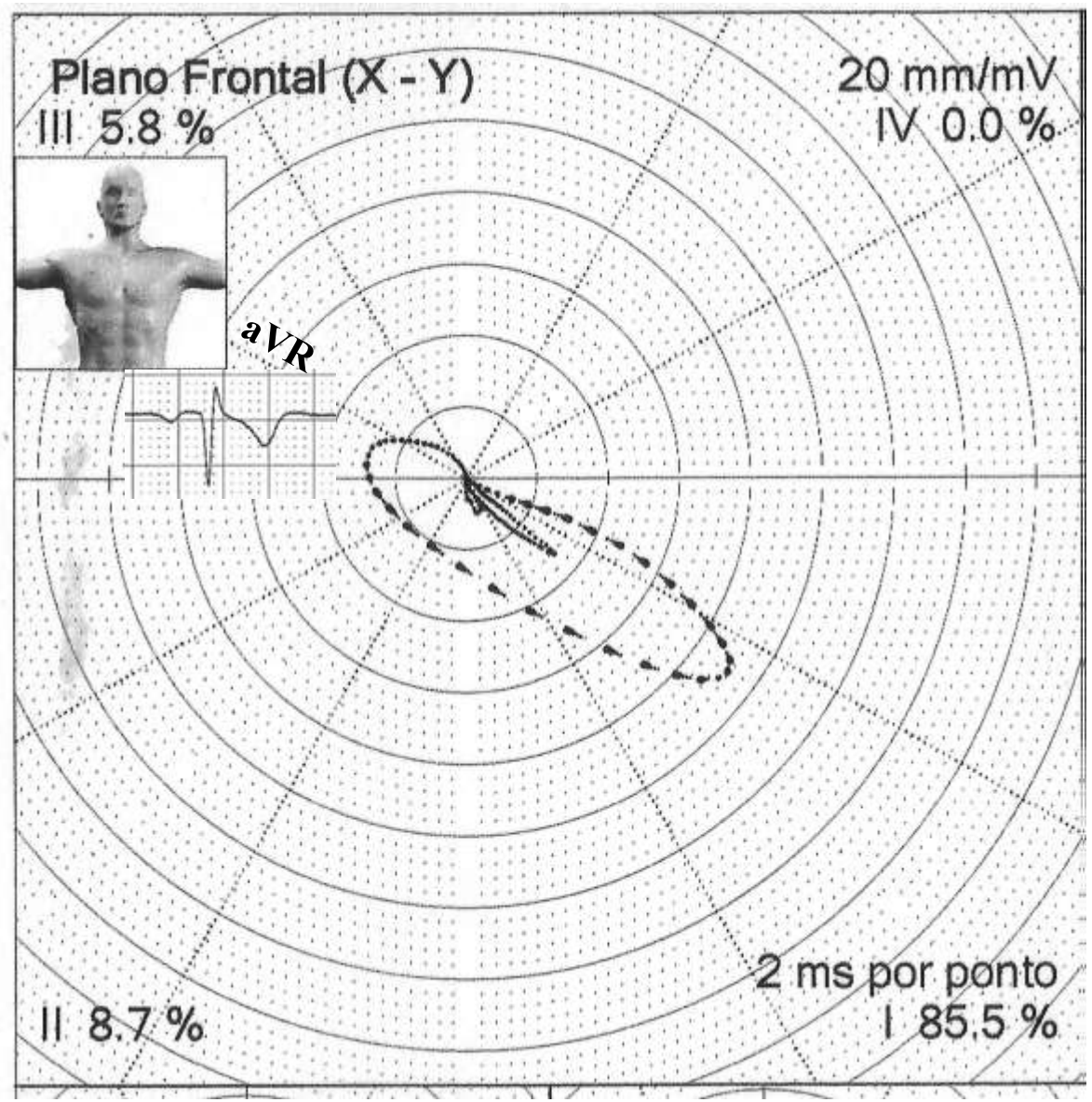
Case report

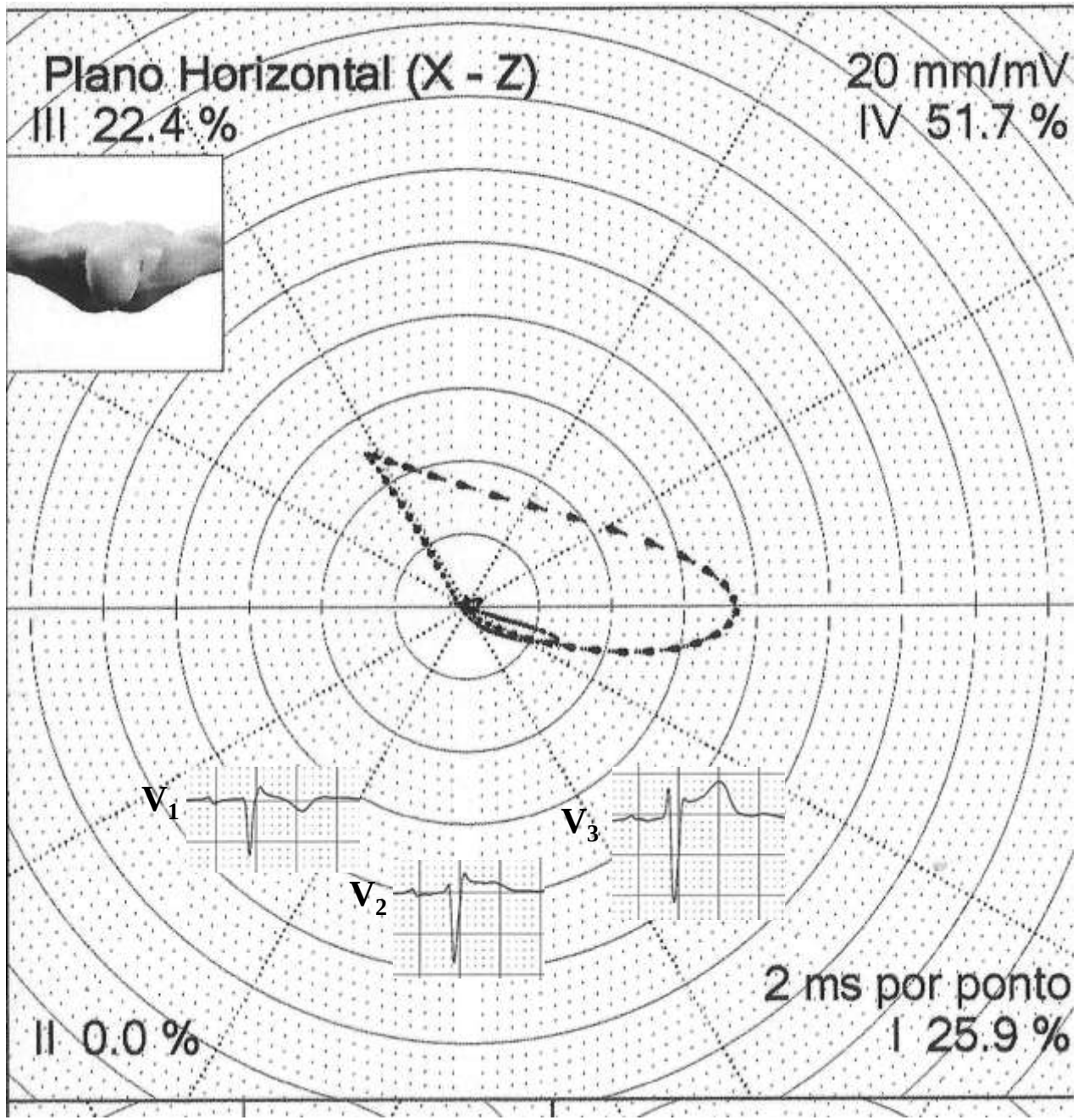
Male, Asian, 55 years old, hypertensive on the use of losartan.
Several syncopes, all at rest or during sleep & atypical precordial pain.
Report an episode of polymorphic ventricular tachycardia at rest with reversion after cardioversion (we do not have the graphic documentation)
It was initially conducted by the emergency team as an acute coronary syndrome.
Normal physical examination
Normal echo, coronary angiography, and troponin level.
Family history: a brother died while sleeping at age 55. What are the vectorcardiographic elements for diagnosis? (Europace. 2012 Jun;14(6):889-97).

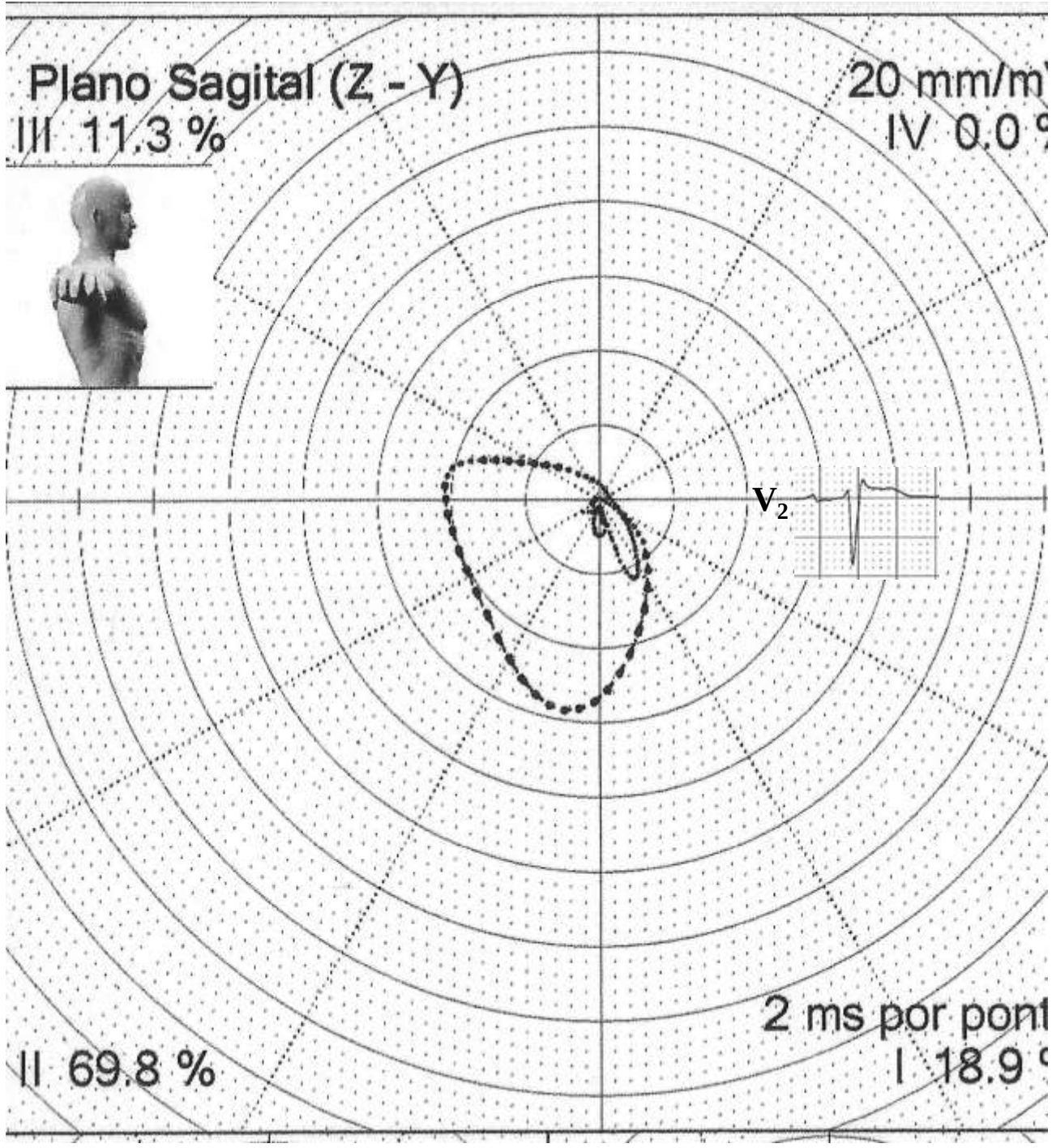

10mm/mV 25mm/s
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

HIGH PRECORDIAL LEADS




Plano Frontal (X - Y)
20 mm/mV
III 5.8 %
IV 0.0 %
aVR
2 ms por ponto
II 8.7 %
I 85.5 %


Plano Horizontal (X - Z)
III 22.4 %
20 mm/mV
IV 51.7 %
V1
V2
V3
2 ms por ponto
II 0.0 %
I 25.9 %


Plano Sagital (Z - Y)
III 11.3 %
20 mm/m
IV 0.0 %
V2
2 ms por pont
II 69.8 %
I 18.9 %

Colleagues opinions

Dear Andrés and Raimundo,

The first electrocardiogram shows sinus rhythm, normal PR, QRS duration, and QT interval.
The ST segment and T waves in leads V1 and V2 are suspicious for type 2 Brugada pattern. The high precordial leads (ECG 2) show a more obvious Brugada pattern, with a history of cardiac arrest and polymorphic VT. My first diagnosis would be Brugada syndrome. Although the family history is non-specific in Brugada syndrome, his brother has passed away of sudden cardiac death.
The other diagnosis could be long QT syndrome with intermittent normal QT, which has been occasionally reported in the history of nocturnal syncope and cardiac arrest, which has been reported in normal phenotype and genotype QT syndrome.
In summary, my first diagnosis would be Brugada. Thank you for sharing.

Very best,

Mohammad Shenasa MD, FACC, FESC, FAHA, FHRS,
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San Jose, CA 95128
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408-286-2922 (Fax)
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Dear Andres: I do recognize that I have no idea what should be the additional value of vectocardiography in patients with Brugada syndrome.
I am anxious to learn.
These MARANOS are absolutely incredible @@@
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

Buenas noches estimados Maestros!
Se trata de un varón , de 55 años, asiático, con historia de síncopes, en reposo, mientras dormía, y por lo menos un episodio recuperado (TV polimorfa) por CVE. Además antecedente de un hermano fallecido por MS en circunstancias similares, y con un patrón electrocardiográfico de Brugada tipo 1, en ambos registros, estándar y derivaciones accesorias altas.
Es un Síndrome de Brugada.
Ritmo sinusal, bradicárdico, onda P aumentada (110 mseg) , intervaloPR 220-240, bloqueo 1° grado.
Punto J elevado > 2 mm en V1-V2 y en "altas" también en V3 acompañado de onda T negativa con ST convexo. TdP cercano a los 90 Mseg.
Signo de aVR (+) R > 3 mm. (Peor pronóstico). No veo fQRS. Eje eléctrico normal.
En VCG con bucle terminando en cuadrante superior derecho en plano frontal, por R en aVR y RFCD en TSVD. Patrón Rs en DI y aVL, SII > SIII , bloqueo fascículo superior derecho
Juan Manzardo MD Mendoza Argentina

English
Good evening dear Masters!
It is a male, 55 years old, Asian, with a history of repetitive syncopes, at rest, while he slept, and at least one episode recovered (polymorphic ventricular tachycardia) by CVE.
Additionally, his brother had sudden cardiac death in similar circumstances, and with an electrocardiographic pattern of Brugada type 1, in both registers, standard and high accessory leads.
Conclusion: It's a Brugada Syndrome.
Sinus rhythm, HR bradycardic, increased P wave duration (>110 ms), prolonged PR interval (220-240) , 1st degree block. J point raised> 2 mm in V1-V2 and in "high right leads" also in V3 followed by negative T wave with ST segment convex to the top ST.
Presence of aVR Sign R wave > 3 mm. (Worst prognosis). Normal electric axis.
In VCG with loop ending in upper right quadrant in frontal plane, by R in aVR and RFCD in TSVD. Rs pattern in DI and aVL, SII> SIII, superior or subpulmonary fascicle.

It is a very interesting case. Brugada is related to the genetic background, so I have a question: is there also any relation to the anatomical characteristics of the heart? According the VCG I would consider biventricular hypertrophy with dominant right ventricular hypertrophy. If the dimensions of the heart are within normal limits, it is even more interesting.

Best regards,

Ljuba

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"The East Coast of Florida" where several ISCE meetings were held. Painted by Lyuba Bacharova, the senior scientist from Bratsislava, Slovakia. Her main research interests are left ventricular hypertrophy and changes in QRS complex morphology due to altered electrical properties of mycardium. She is an active member of ISE and ISCE, the President of ISE (1997-1999), and the Secretary of ISE (2005-2014)., Together with Galen S. Wagner she initiated the change in the cover page design of the *Journal of Electrocardiology*, and now the readers can enjoy paintings related to Elctrocardiology.

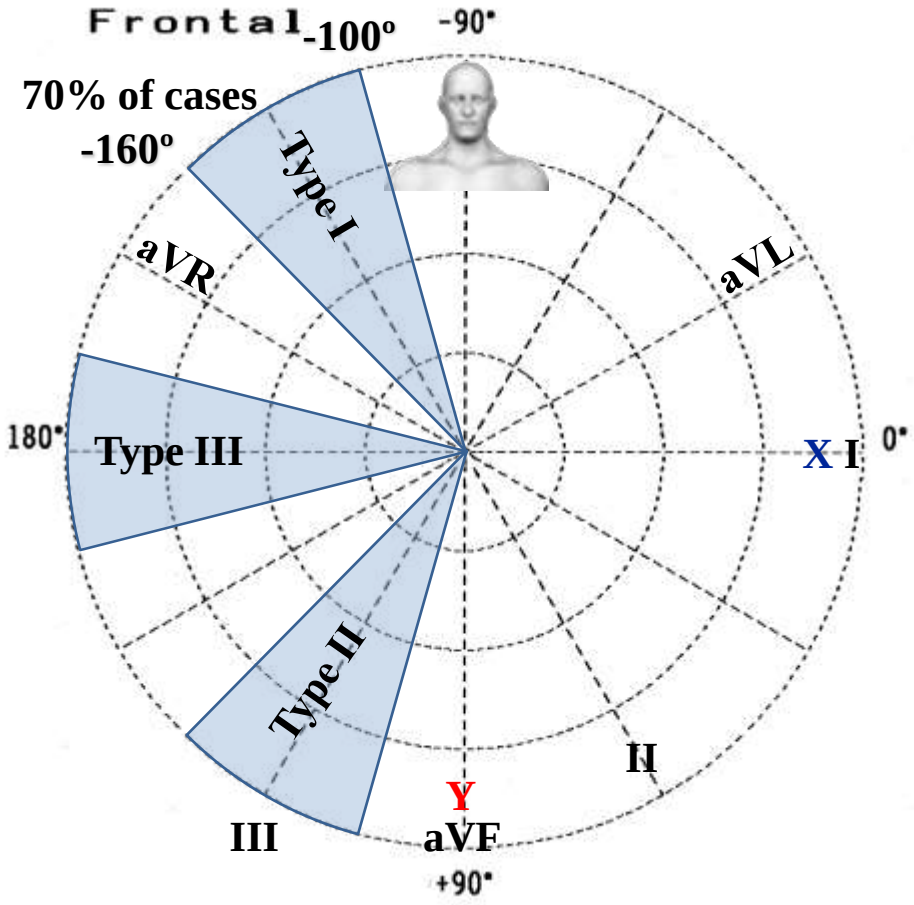
Answer to Dr. Ljuba Bacharova:

Dear Ljuba: This Brugada´s patient has a typical pattern of Right End Conduction Delays (**RECD**) zonal, fascicular, parietal, peripheral, distal or Purkinje right superior ventricular blocks. These electrovectorcardiographic changes, are secondary to physiological delay or to true dromotropic disturbances in the territory of one of the three fascicles of the right bundle branch, in isolation in the RV free wall. To speak about blocking it is necessary the presence of dromotropic disorder or slowing of ventricular activation process because in its absence can not call it so properly. These blocks cause localized or regional delay on basal portion of RV on its free wall. Zonal right ventricular blocks correspond to block of the superoanterior division of the right bundle on RV free wall (on RVOT) or inferoposterior zone (on RVIT) of the right free wall ventricle.

Others denominations Search: **Parietal focal blocks (Masini 1952; Alzamora-Castro 1953; Rossi 1954; Noseda 1963); Right focal blocks; Peripheral branch block of the right bundle; Peripheral blocks of the right branch; Right peripheral fascicular blocks (Pastore 1983); Right peripheral blocks; Distal right bundle block; Divisional blocks of the right branch; Fascicular block of the His bundle; Delayed activation of the wall of the Right Ventricle.** Electrocardiogrfically are characterized by QRS duration is < 120ms, frequent absence of evident final broad r' in V1 (**Uhley 1961**). Blocks of the superoanterior division of the right bundle on RV free wall or block of the superoanterior zone produce prolongation of R-wave peak time on aVR, V3R and eventually in V1 (VAT = 50 ms, maximum normal ≤40 ms), additionally, frequently SI-SII-SIII pattern indistinguishable from the positional SI-SII-SII or the one produced by right ventricular enlargement (**Bayes de Luna 1987**), QRS axis on superior quadrants (above and between ± 180º and 0º). Concomitantly, there are slurred of S waves in leads to “face” the opposing regions (**de Micheli 2009**). This is the block observed constantly in Brugada syndrome (**Pérez-Riera 2012**).

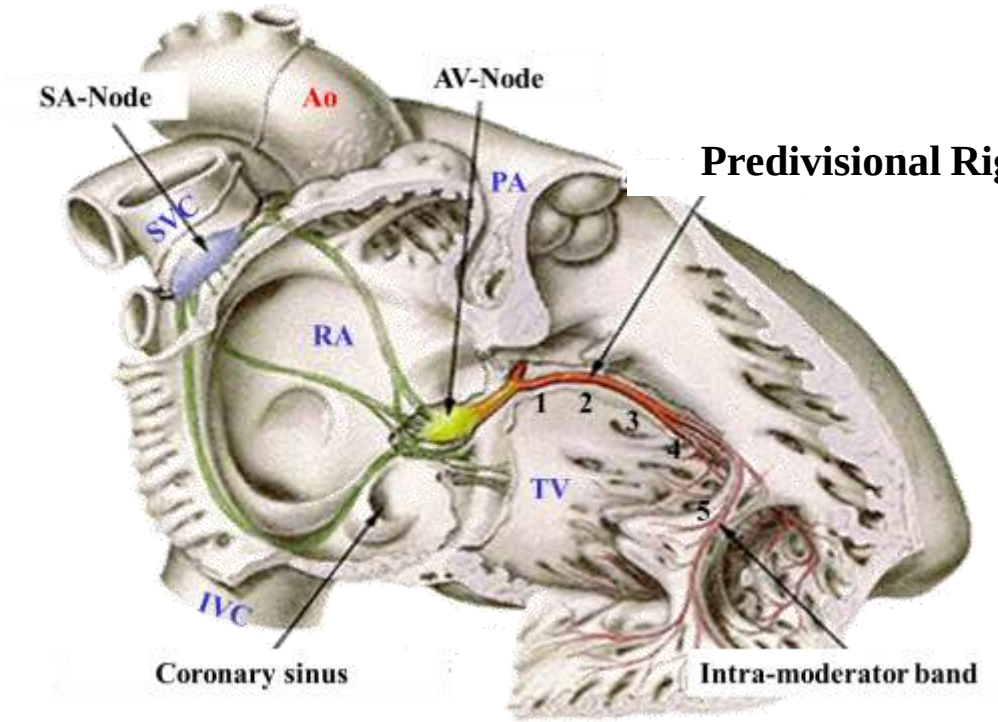
Experimentally, it has been demonstrated that these blocks result from a peripheral delay of the stimulus localized in a certain right ventricular zone on RV free wall**.** (**Bayes de Luna 1982**).

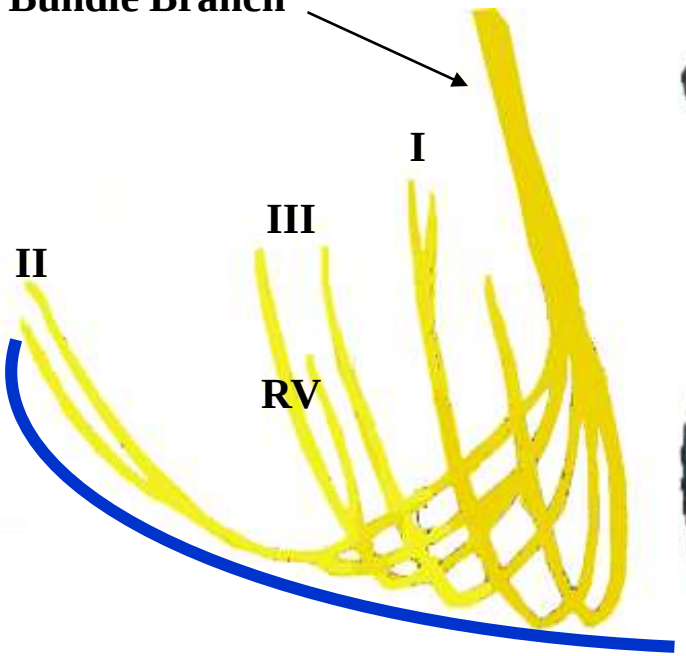
Frontal Plane: Location of End Conduction Delay (RECD) in the 3 types

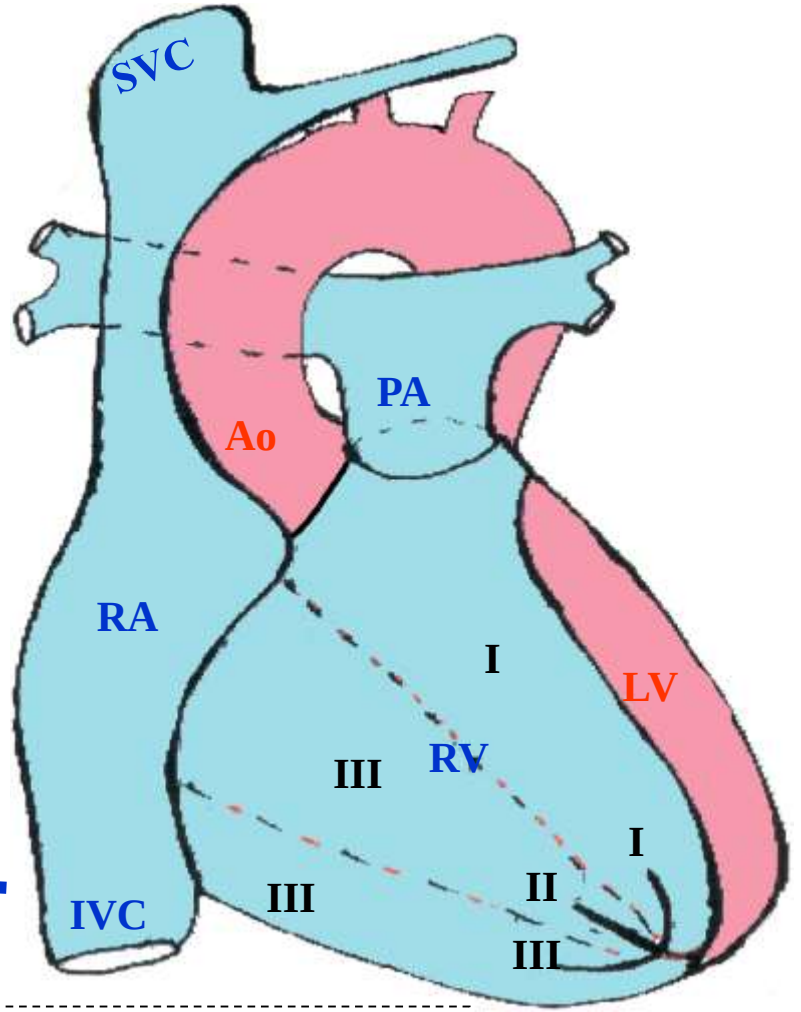


Classification of **RECD,** taking into account the location of the end delay of the QRS loop in the frontal plane in Type I, II and III. New proposal of VCG classification.

Distribution of the three fascicles of the right branch of the His bundle in the RV free wall (Lev 1964-1968; Mahaim 1931; Lenegre 1958)

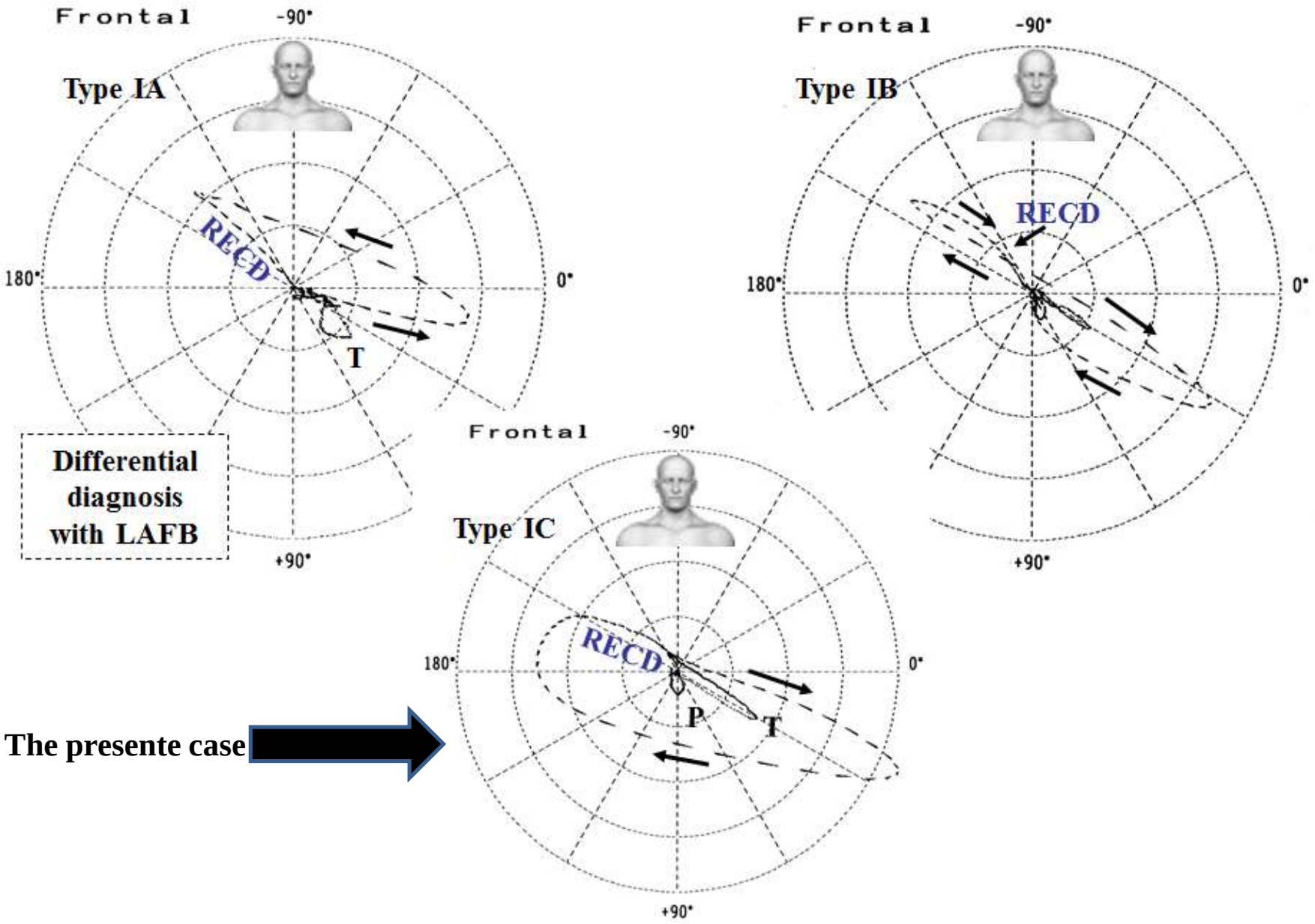






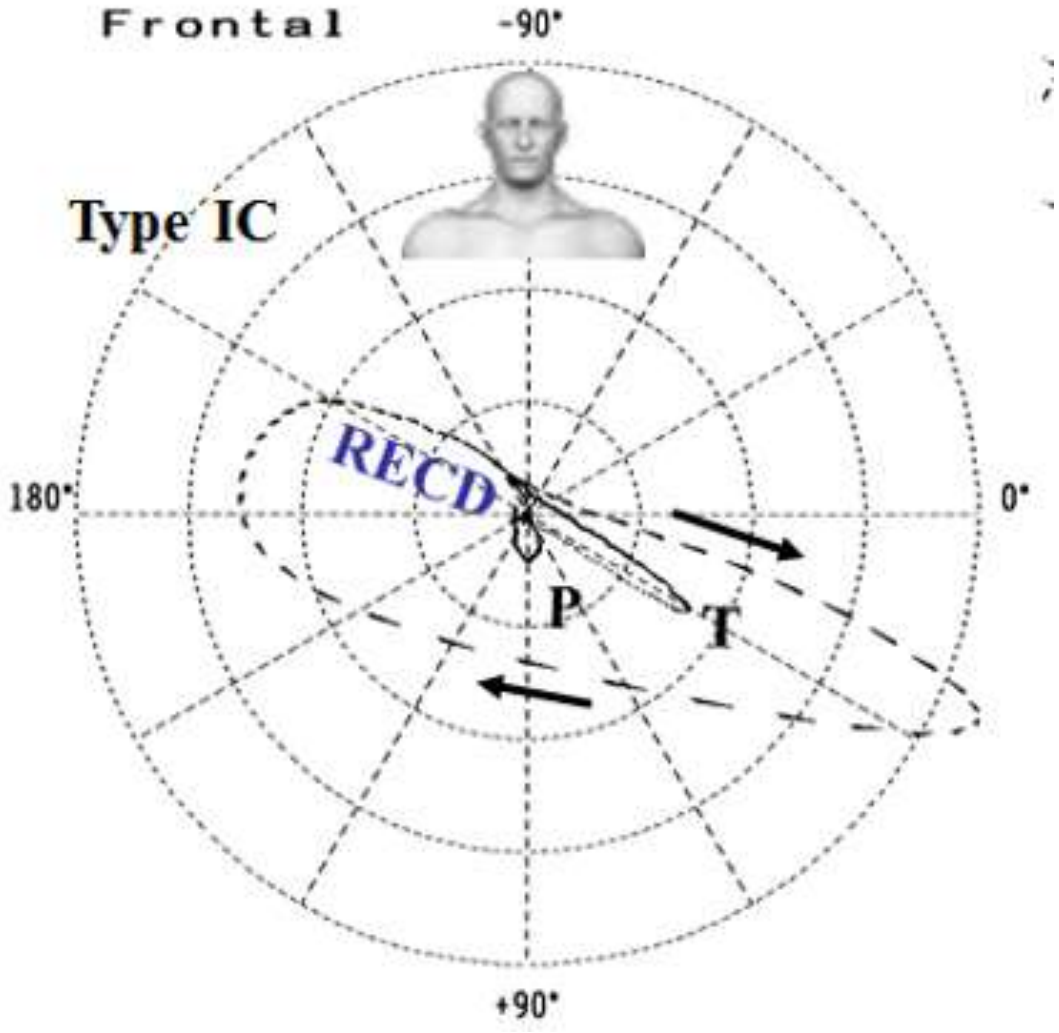
I - Territory of superior or subpulmonary fascicle
II – Territory of inferior or posteroinferior fascicle
III – Territory of middle fascicle

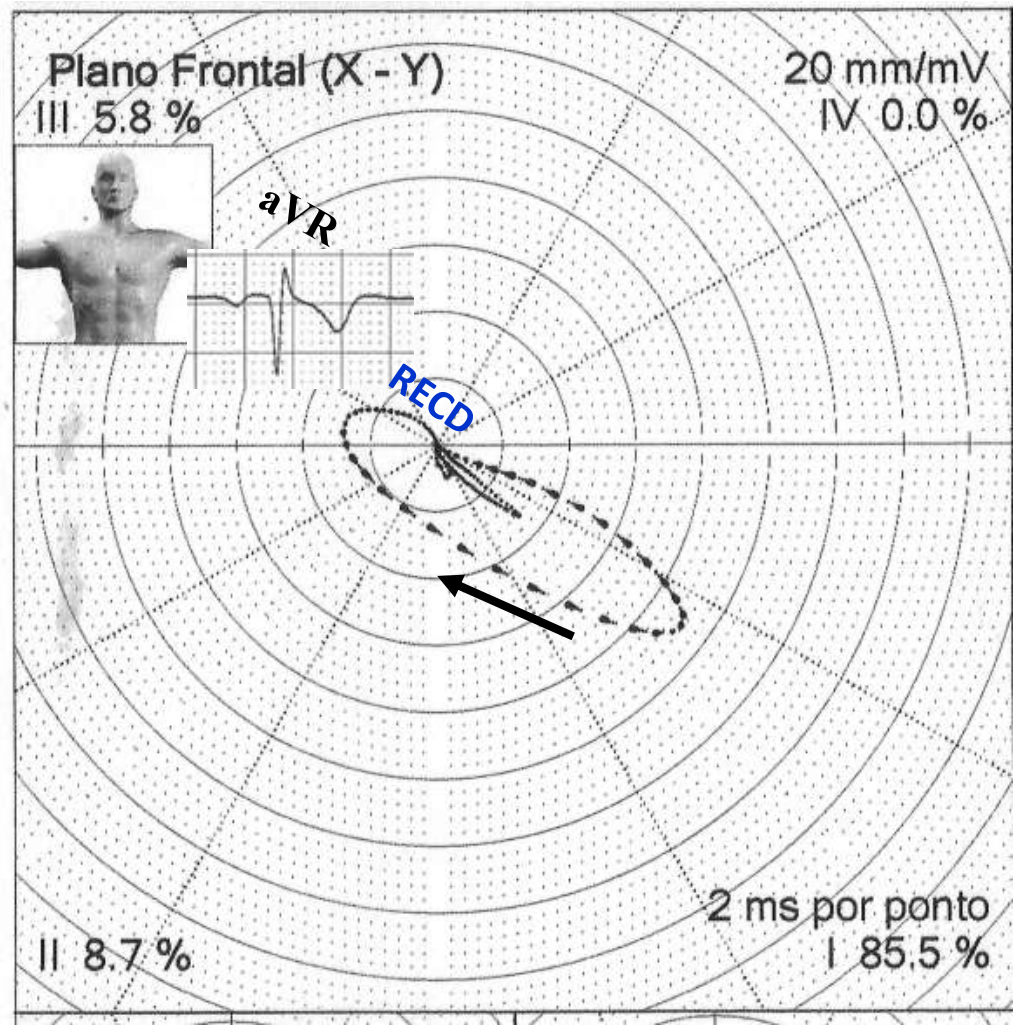
VCG Type I RECD variants according to QRS rotation on FP



Vectorcardiographic loop in the frontal plane of the three subtypes of Type I. It is clear that only type IA may be confused with LAFB.

The present case: RECD Type IC

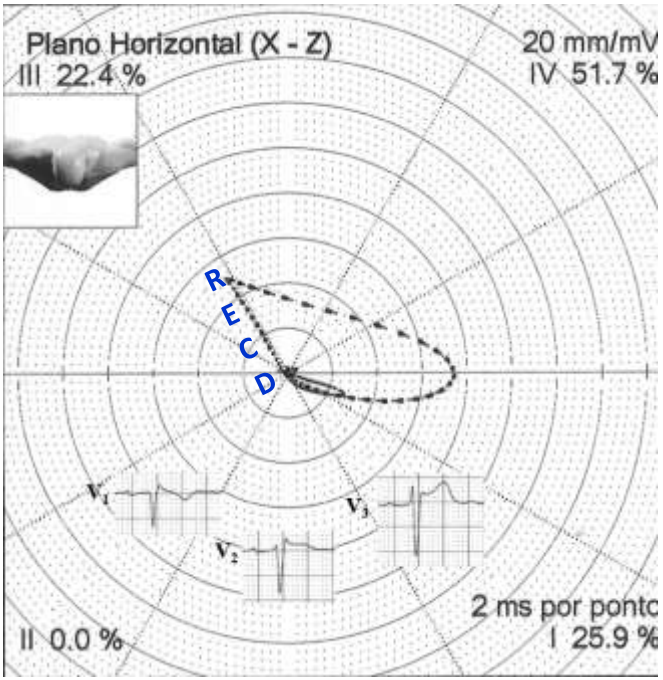




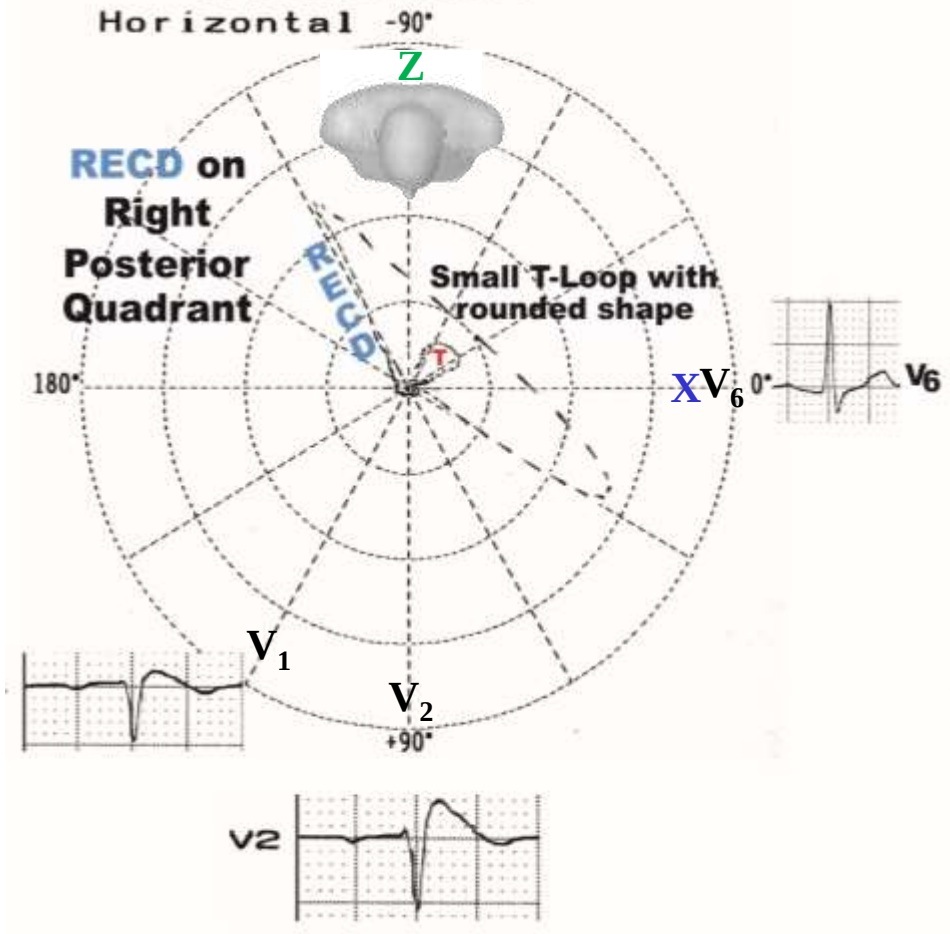
VCG characteristic in the Frontal Plane: SÂQRS not deviated or deviated to the right; pointed aspect of QRS loop; QRS loop of clockwise rotation; **RECD** in the right superior quadrant; frequent SI-SII-SIII; frequent broad R wave of aVR

ECG/VCG correlation in the horizontal plane

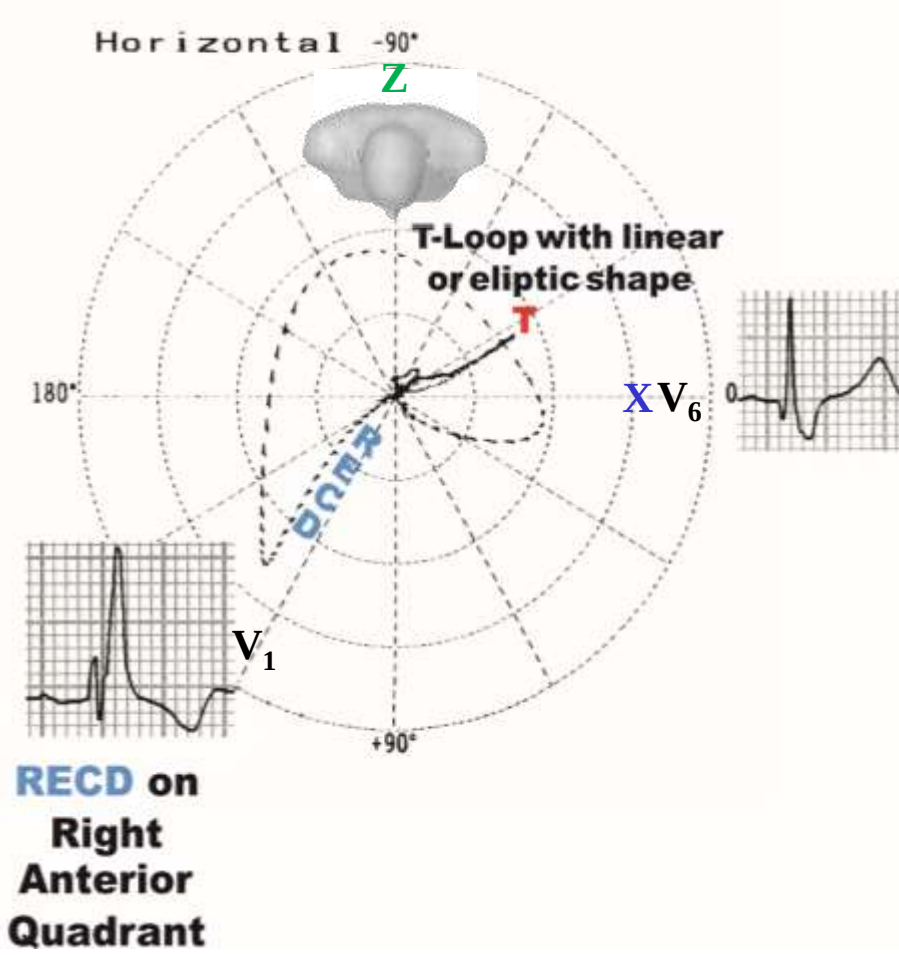
The present case HP



Type 1 Brugada pattern



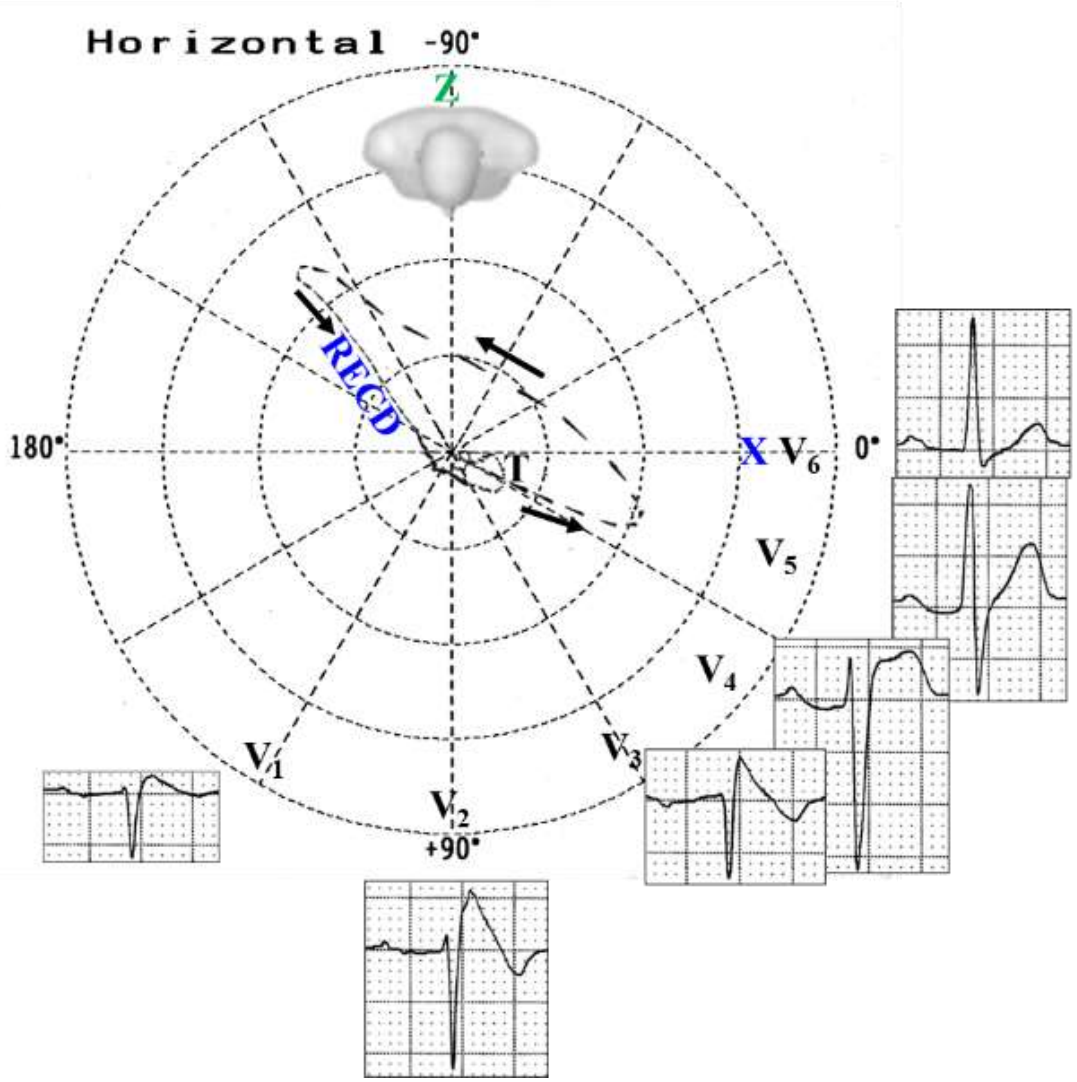
Truly RBBB pattern

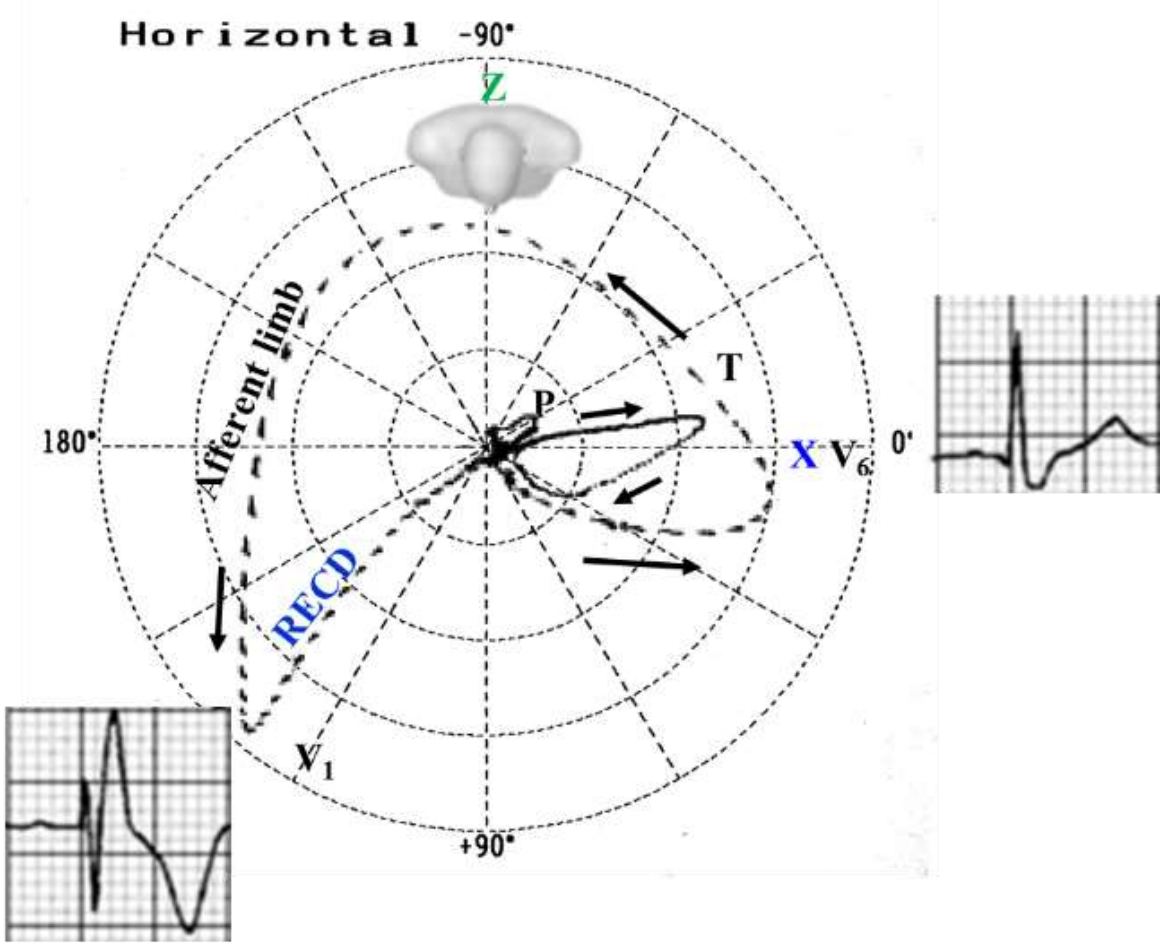


RECD on right posterior quadrant

(Pérez-Riera 2012)

Differential diagnosis between QRS loop of type 1 BrS and CRBBB in the Horizontal Plane





RECD located in the right posterior quadrant	RECD (terminal appendix). Located in the right anterior quadrant
The beginning (0 point) and the end (J point) of the QRS loop are not coincident	Points 0 and J matching
Rounded, small and symmetrical T-loop with CCW rotation.	Asymmetrical, elongated T-loop with CW rotation and directed to left.

Vectocardiographic classification of RECD according to QRS loop in the FP

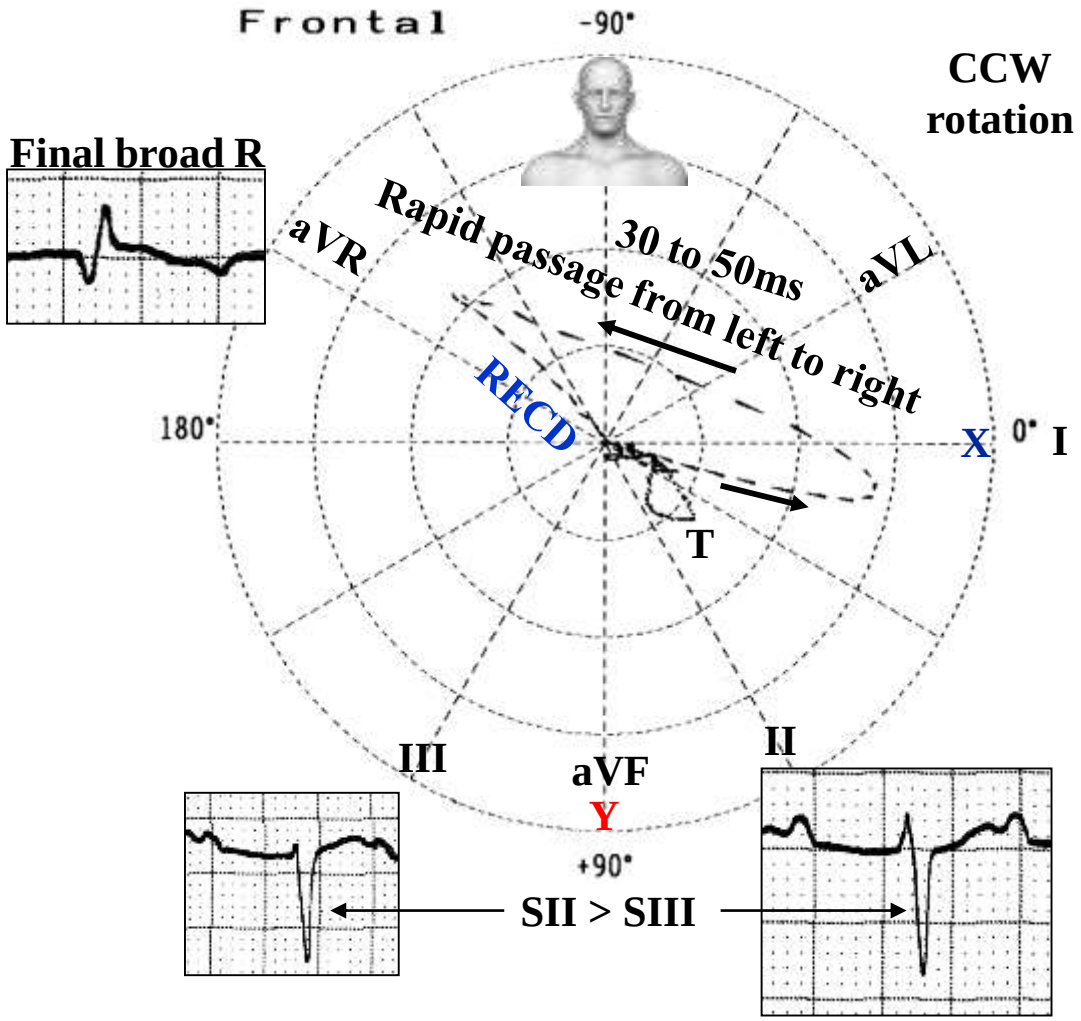
I. Type I: RECD Other names: right superior, anterosuperior or subpulmonary fascicular block. Characterized by presenting end conduction delay (**RECD**), located in the right superior quadrant of the FP, corresponding to the territory of the superior or subpulmonary fascicle of the right branch, in the site of the RV outflow tract between -100° and 160°. The location of the delay justifies the recording of prominent R waves with a certain delay in the lead that faces the RV outflow tract: aVR. Additionally, notched in the apex of the R wave, which prolongation of ventricular activation time, R peak time or intrinsicoid deflection (≥ 50 ms) circunscript only in the right unipolar lead that explore the right ventricular outflow tract affected region (aVR) (**de Micheli 1987**). The superior infundibular subpulmonary region of the RV is the last one to activate, generating a final basal vector (basal vector 3d), heading upward and to the right between -100º and -160º in the FP. This is the most frequent variant of **RECD** (70% of all of our cases). By the aspect and rotation of the QRS loop in the FP, we propose to divide the RECD Type I into: RECD type IA; RECD type IB and RECD type IC(the present case).

II. Type II: **RECD** located in the right inferior quadrant, in the territory of the inferior fascicle of the right branch;

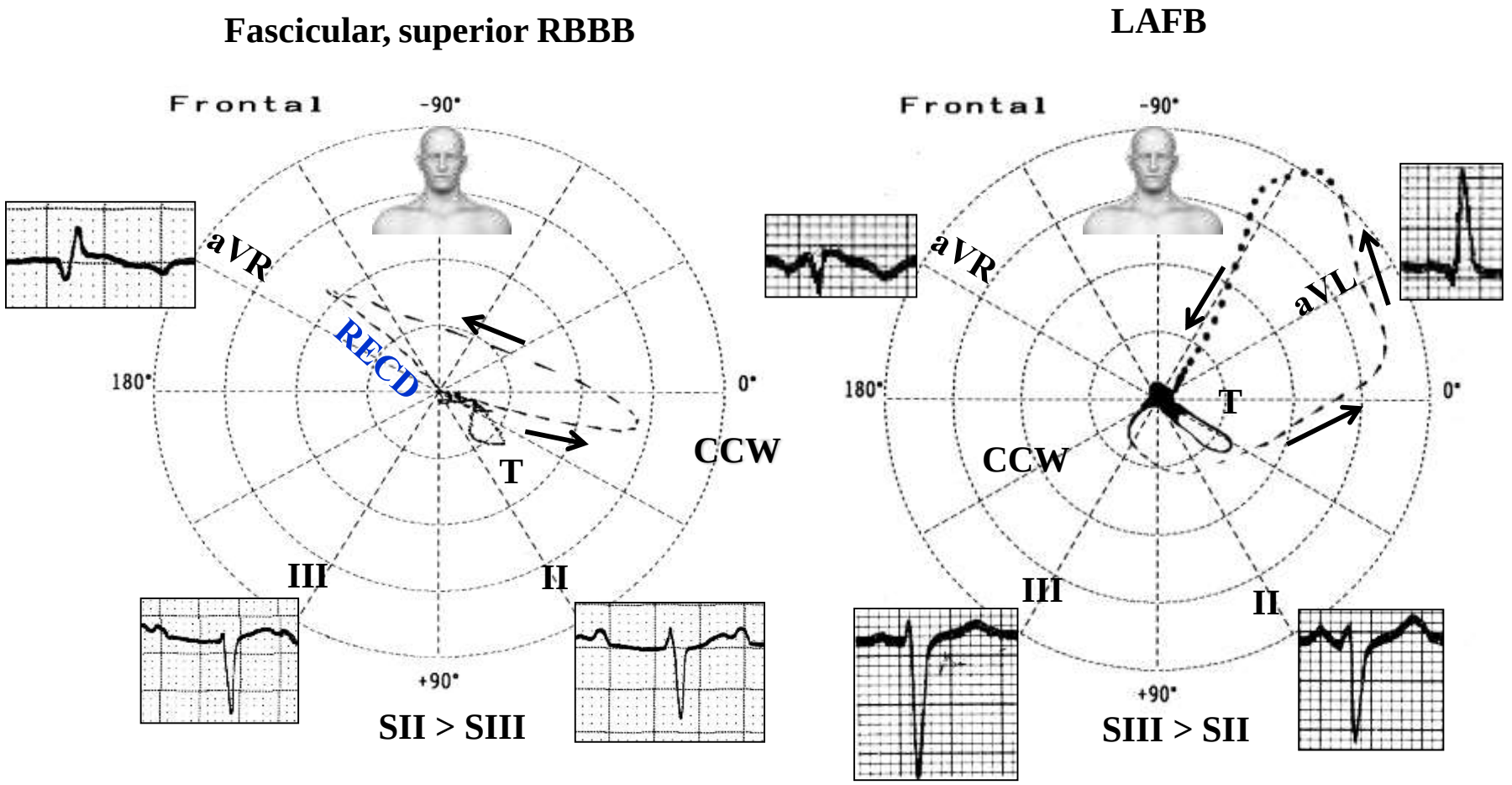
III. Type III: **RECD** located on the right portion of the 0±180° line, corresponding to the territory of distribution of the middle fascicle of the right branch;

IV. Type IV: **RECD** located in the line of – 90° (neither to the right nor the left). Does it exist?

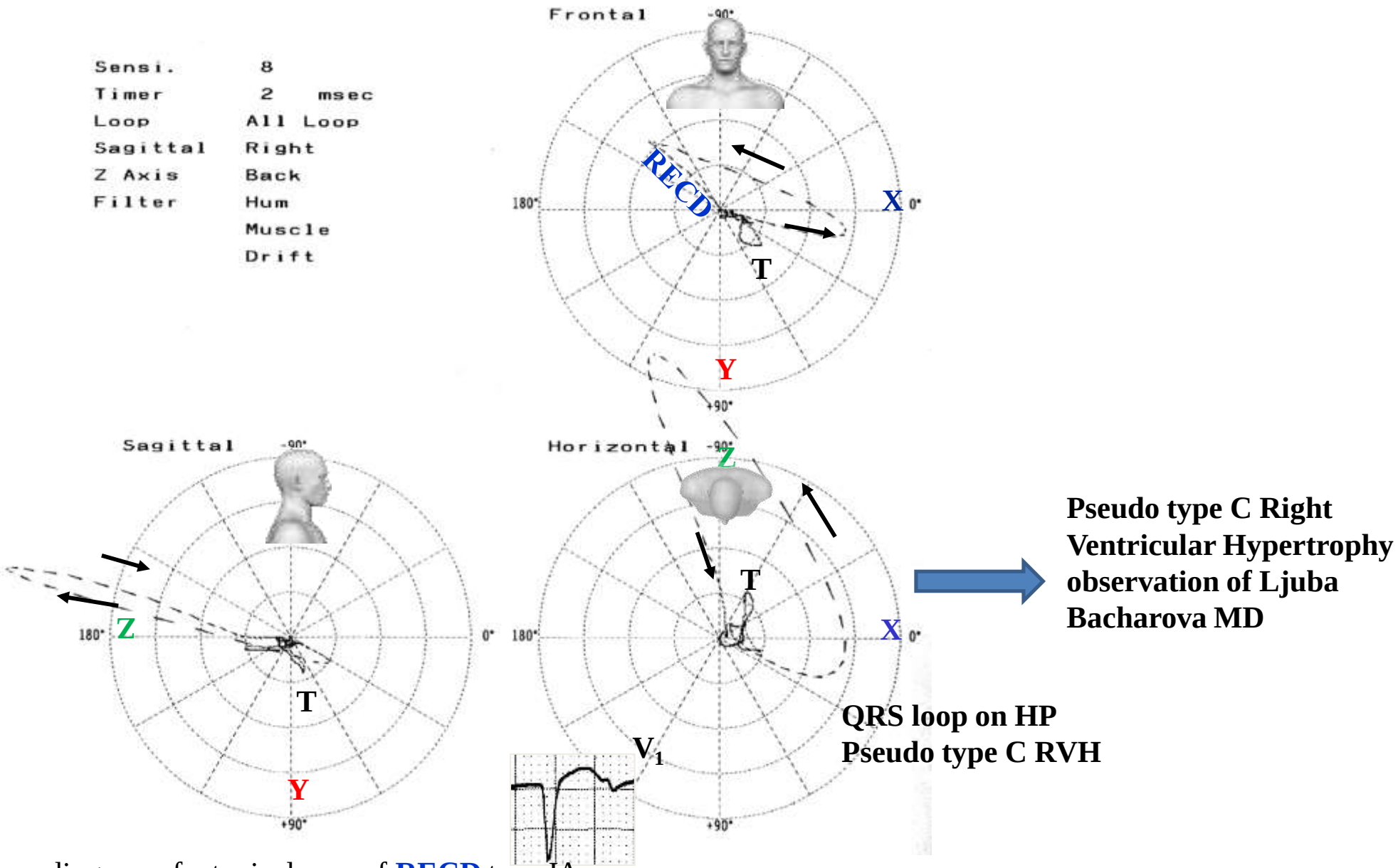
Typical case of RECD type IA showing ECG/VCG correlation in the Frontal Plane



ECG/VCG correlation between RECD type IA and LAFB

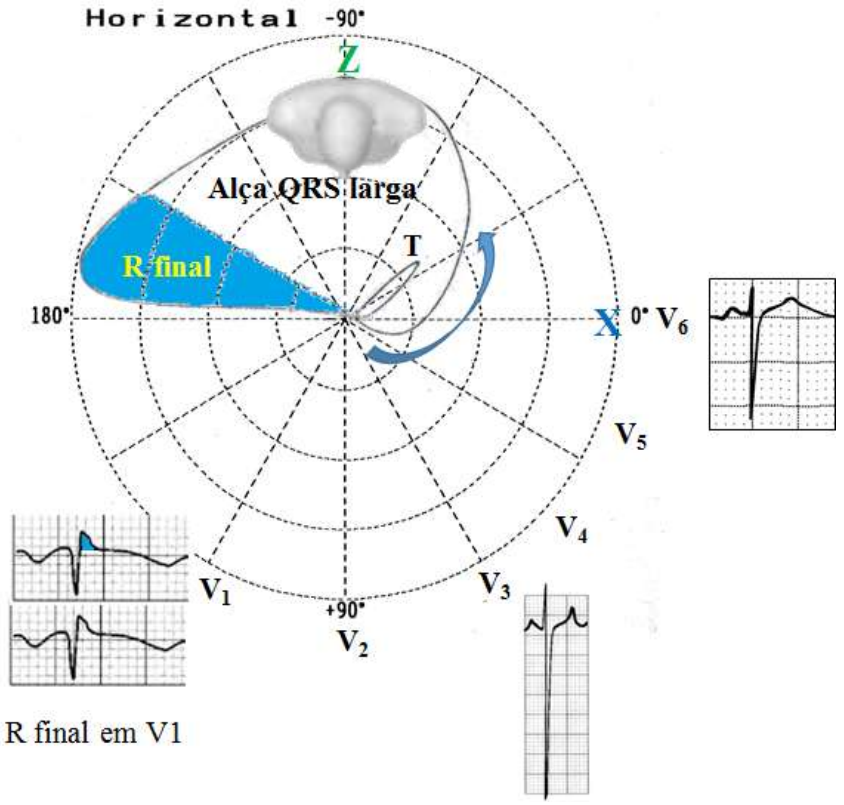


Name: MPC; **Sex:** Fem.; **Age:** 75 yo.; **Race:** mulatto; **Weight:** 44Kg; **Height:** 1.48 m; **Biotypo:** athletic; **Date:** 23/08/2017; **Medication in use:** Lisinopril 20 mg + Adalat Oros 30 mg + Asa 100 mg 2X + Betamethyldigoxin 1X .

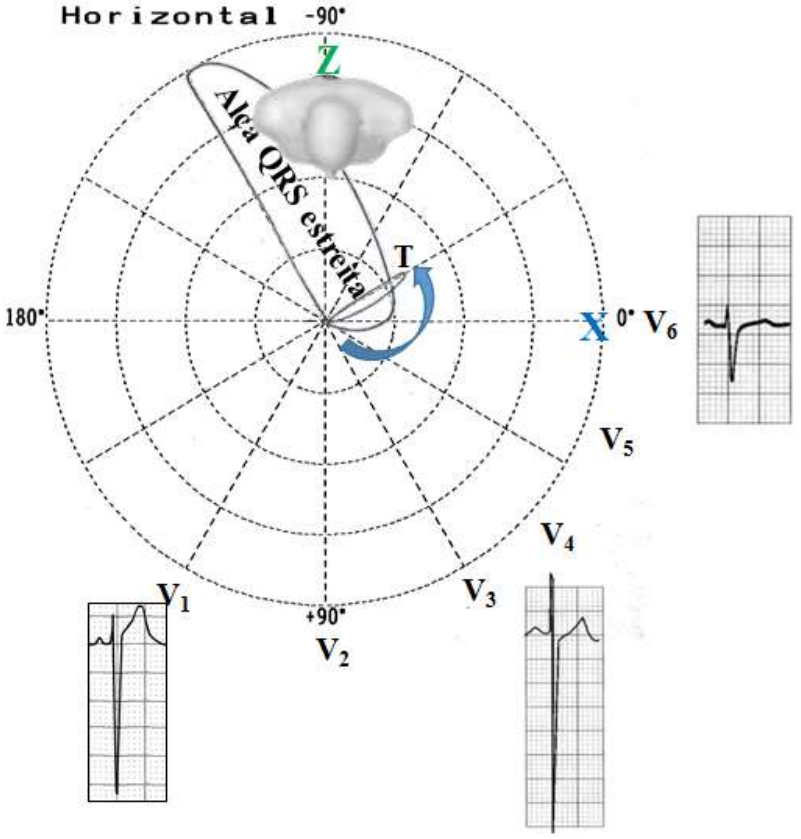


Electro-vectocardiogram of a typical case of **RECD** type IA.

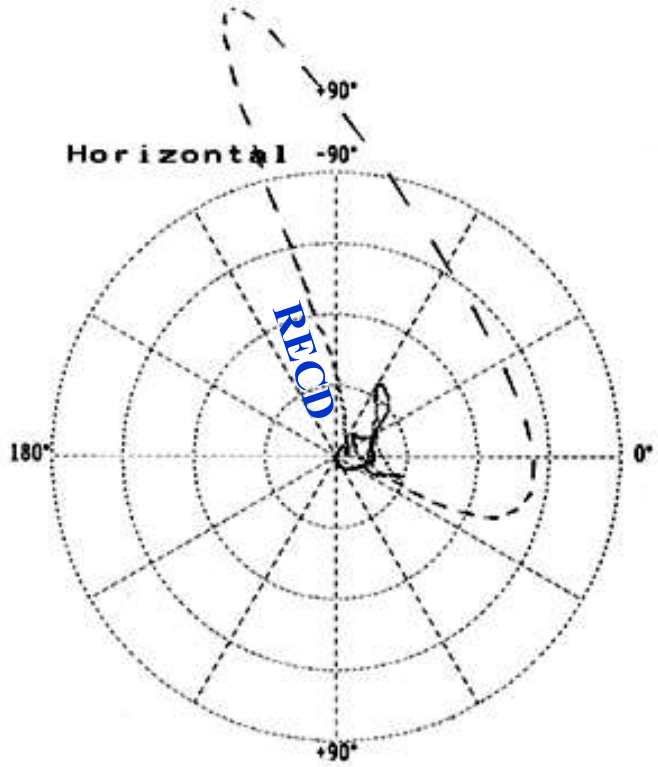
VCG in type C right ventricular hypertrophy



R final em V1

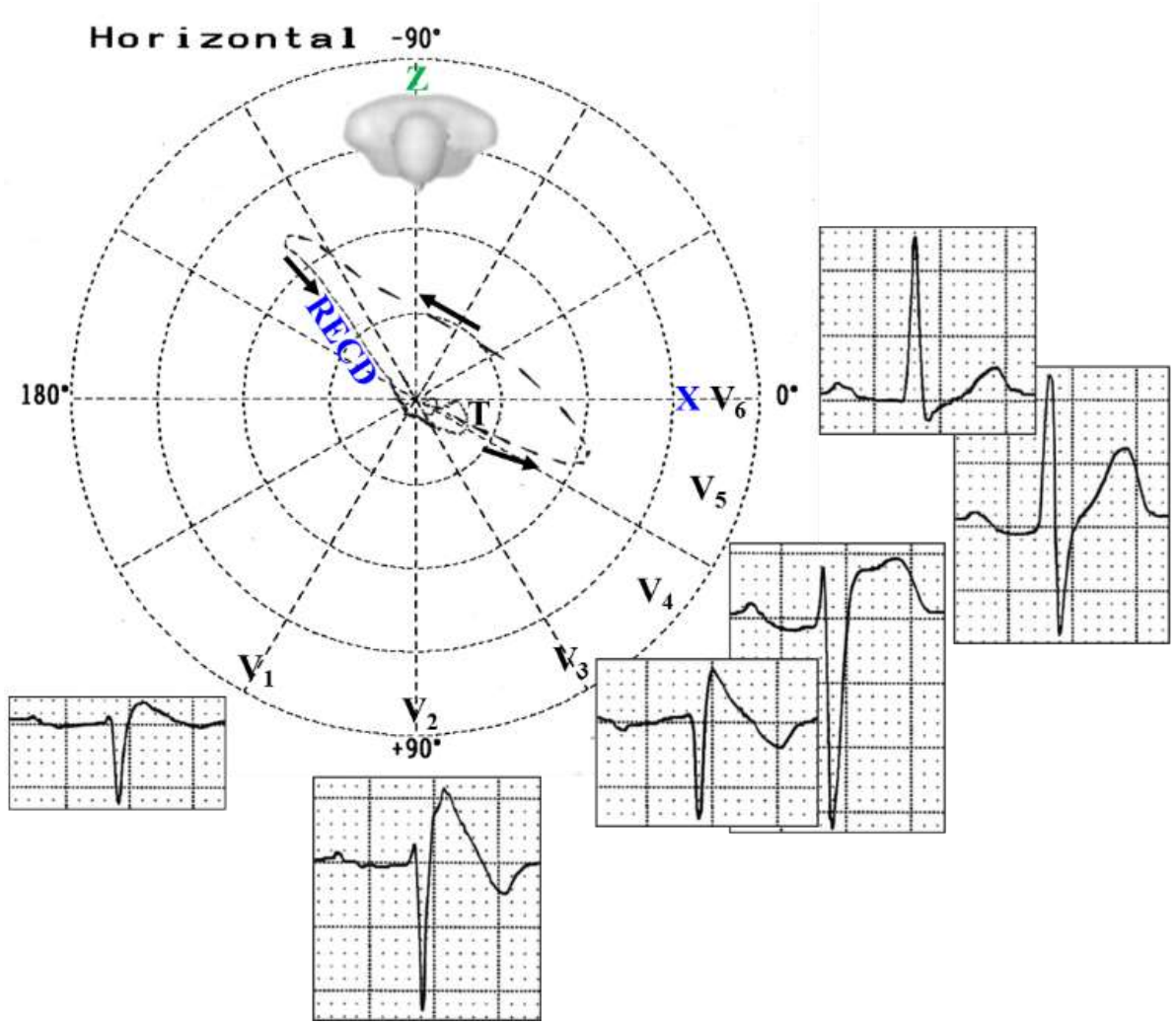


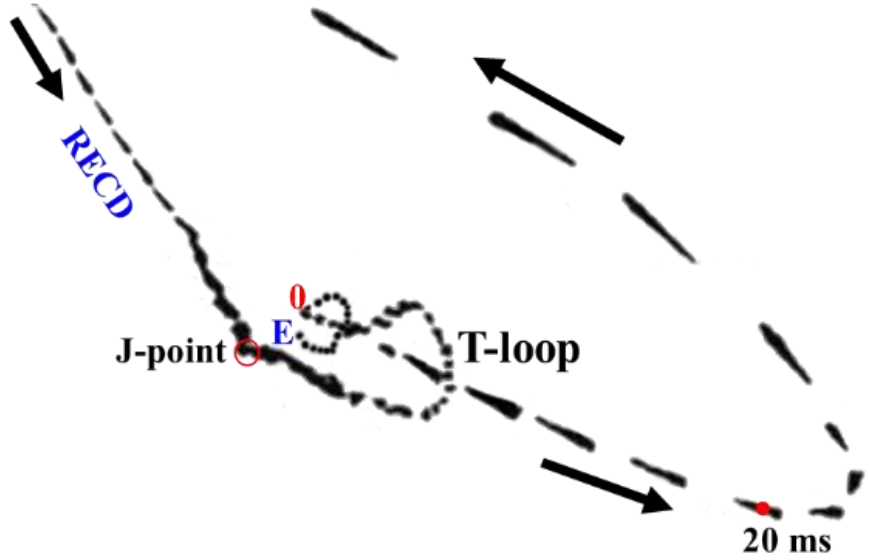
VCG in right fascicular block



Type C right ventricular hypertrophy morphology in horizontal plane (HP) has similar shape of the right divisional blocks.

Classical features of the loops in the horizontal plane in type 1 ECG/VCG BrS





Vector from the initial 10 to 20 ms heading to the front and left, counterclockwise rotation, rapid passage from left to right, >20% of the QRS loop area located in the right posterior quadrant and right end conduction delay (RECD) back and at the right (depolarization abnormality). QRS loop end (J point) distant from the initial point 0, indicating a significant J point and ST segment elevation visible in the right precordial leads.
Small and rounded T loop, with limbs of slow and symmetric inscription (repolarization abnormality).

Clinical significance of RECD

Its clinical significance and interest lies in the fact that:

1) They may be confused with left fascicular blocks: Left Anterior Fascicular Block (LAFB) and Left Posterior Fascicular Block (LPFB);
2) They may be confused with myocardial infarctions (pseudo electrically inactive areas) both in the anterior and the inferior walls.
3) They may represent the electro-vectocardiographic pattern of Brugada syndrome and one subpopulation of Arrhythmogenic Right Ventricular Dysplasia (ARVD/C).

From 100 consecutive cases of our series, 15 presented diagnostic doubt by ECG with MI. This caused us to request a VCG to clarify this issue.

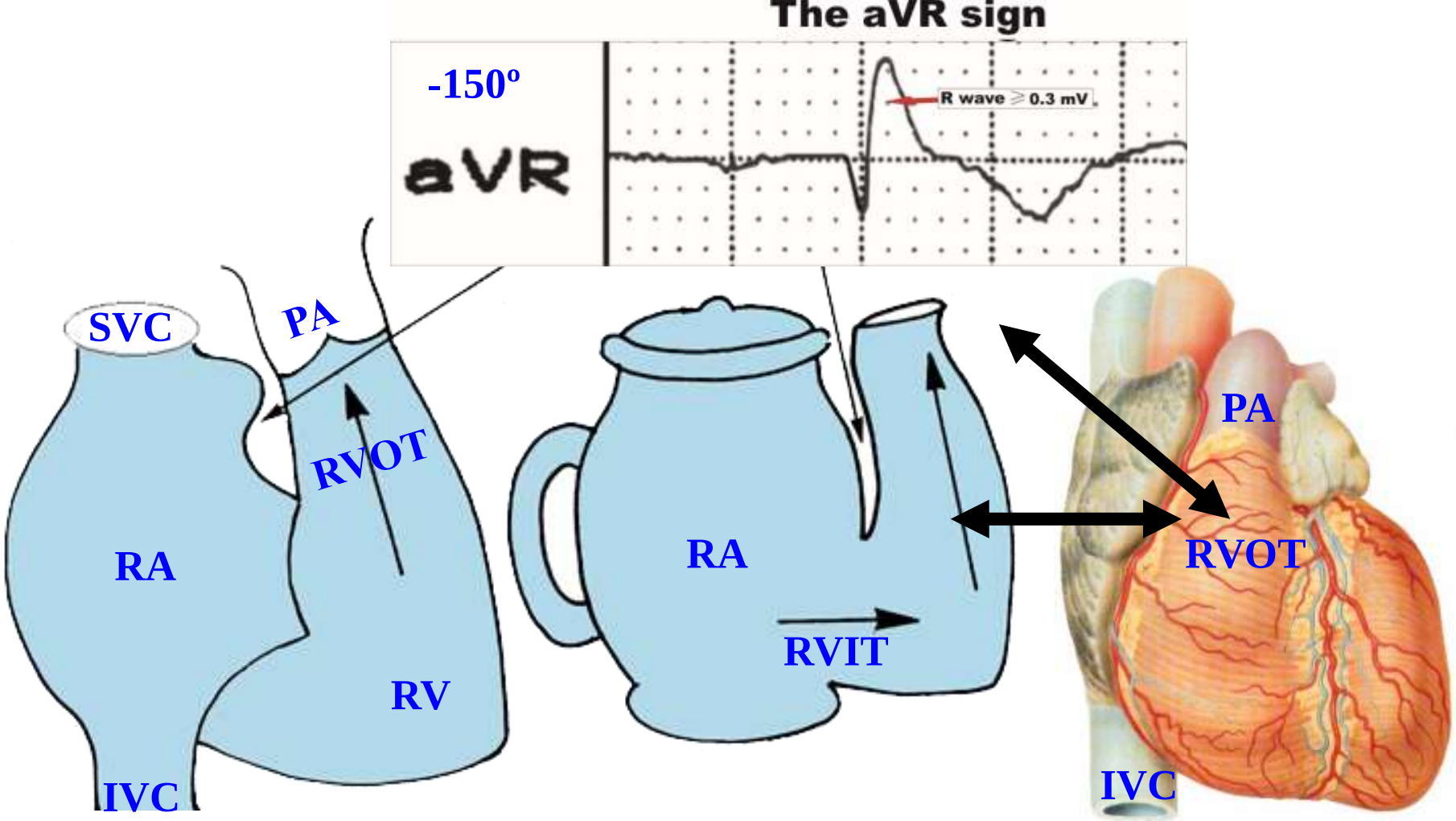
From these, 12 raised the suspicion of inferior MI electrically inactive area (IEIA) and 3 anterior or septal MI.

From these 12 that presented diagnostic doubt with inferior electrically inactive area, 11 were diagnosed as RECD Type IA and 1 as RECD Type III subtype A.

Differential diagnosis between RECD type IA and LAFB

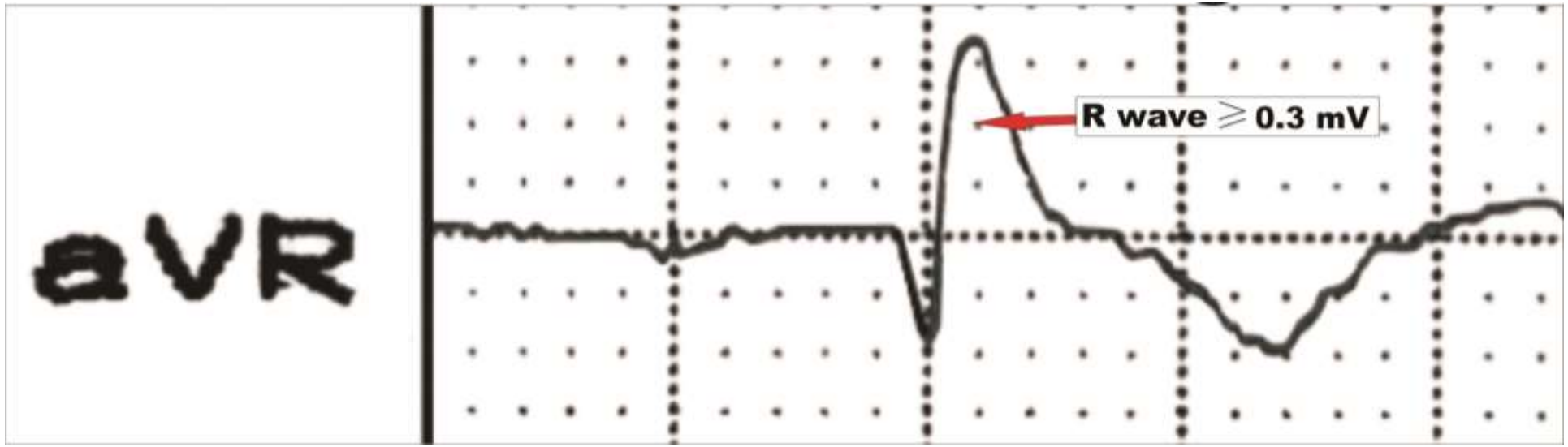
	RECD type IA	LAFB
Depth of S wave in II and III	SII > SIII (inconstant)	SIII > SII (inconstant)
I and aVL	Rs	qR
Prominent and broad R wave in aVR	Present and characteristic: QR or qR.	Absent: Qr or QS.
Vector of initial 10 to 20 ms	Downward and to the left (inconstant)	Downward and to the right. (inconstant) ≅+120°
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.	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.

Criteria for differential diagnosis between **RECD** type IA and LAFB.



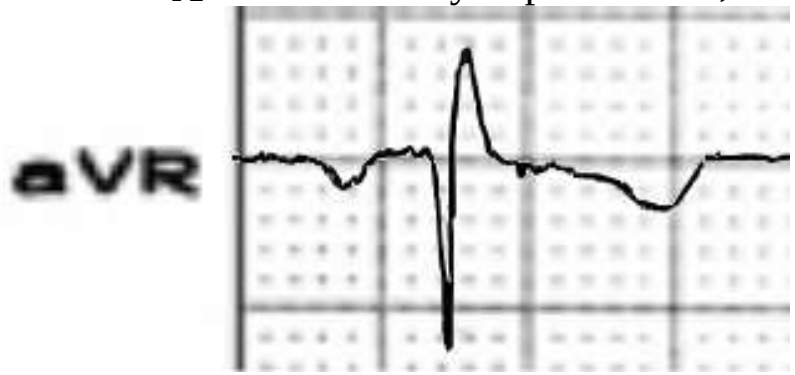
The BrS affects predominantly the right ventricle in the right ventricle outflow tract (RVOT) epicardium (**Doi 2010**). The larger part of clinical evidence supports the presence of right end conduction delay (**RECD**) as part of the process of BrS pathophysiology in the RVOT, as a consequence of structural abnormalities in the heart as part of BrS (**Coronel 2005**). On the other hand, in the concealed forms of arrhythmogenic right ventricular cardiomypathy/dysplasia (ARVC/D), the RECD pattern can also be observed showing type-1 ECG pattern. This pattern was shown many years ago by Guy Fontaine et al (**Hayashi 2010**).

The aVR sign

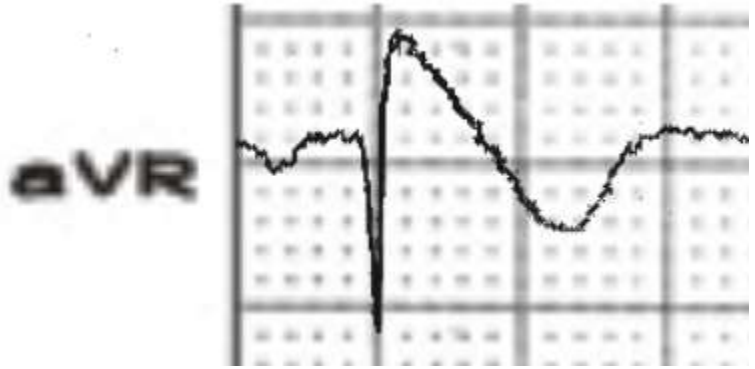


aVR sign: final R wave of aVR lead >3 mm

A **Date:** Friday September 30, 2011.



B **Date:** Tuesday October 4, 2011.

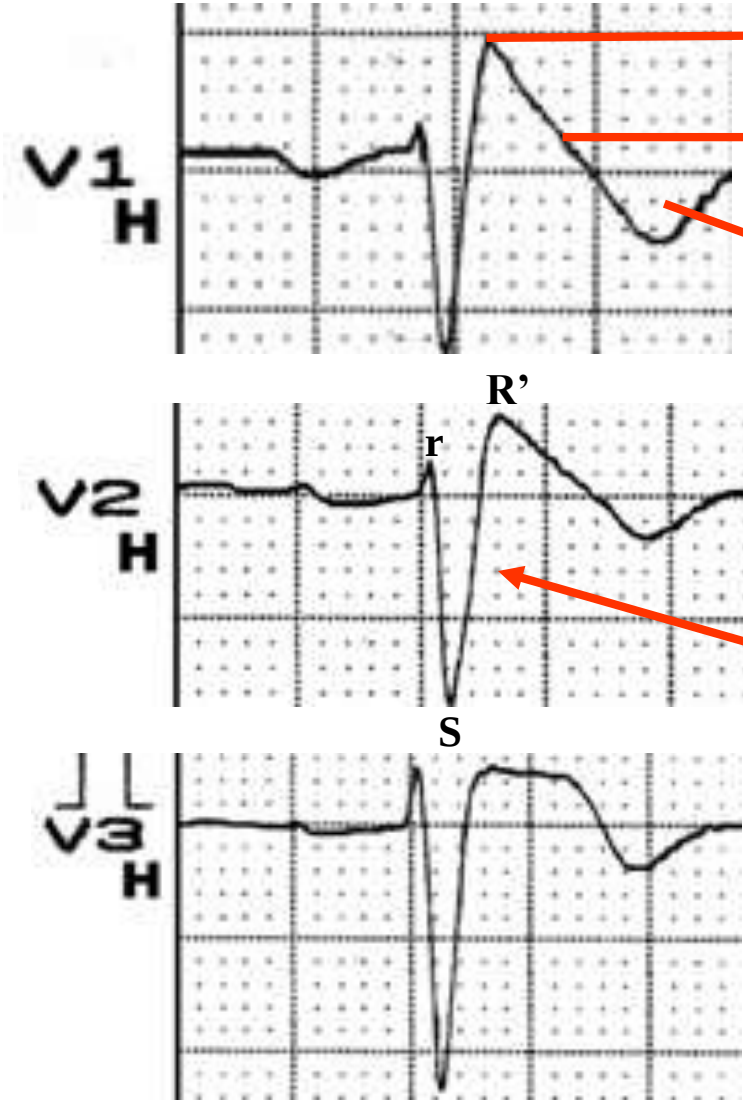


Terminal broad R wave of the QRS complex in lead aVR (Babai Bigi 2007)

The aVR sign: Presence of prominent final R wave on aVR lead; R wave ≥3 mm or R/q ≥0.75 in lead aVR (aVR sign). Slow conduction at the RVOT may contribute to the induction of VF by PVS.

Value of ECG and VCG in the diagnosis and prognosis of the Brugada syndrome

Signature ECG-change



High take-off: it is not the end of the QRS complex

STSE rectilinear oblique and downward. Impossible to determine the J point location

Negative symmetrical T-wave

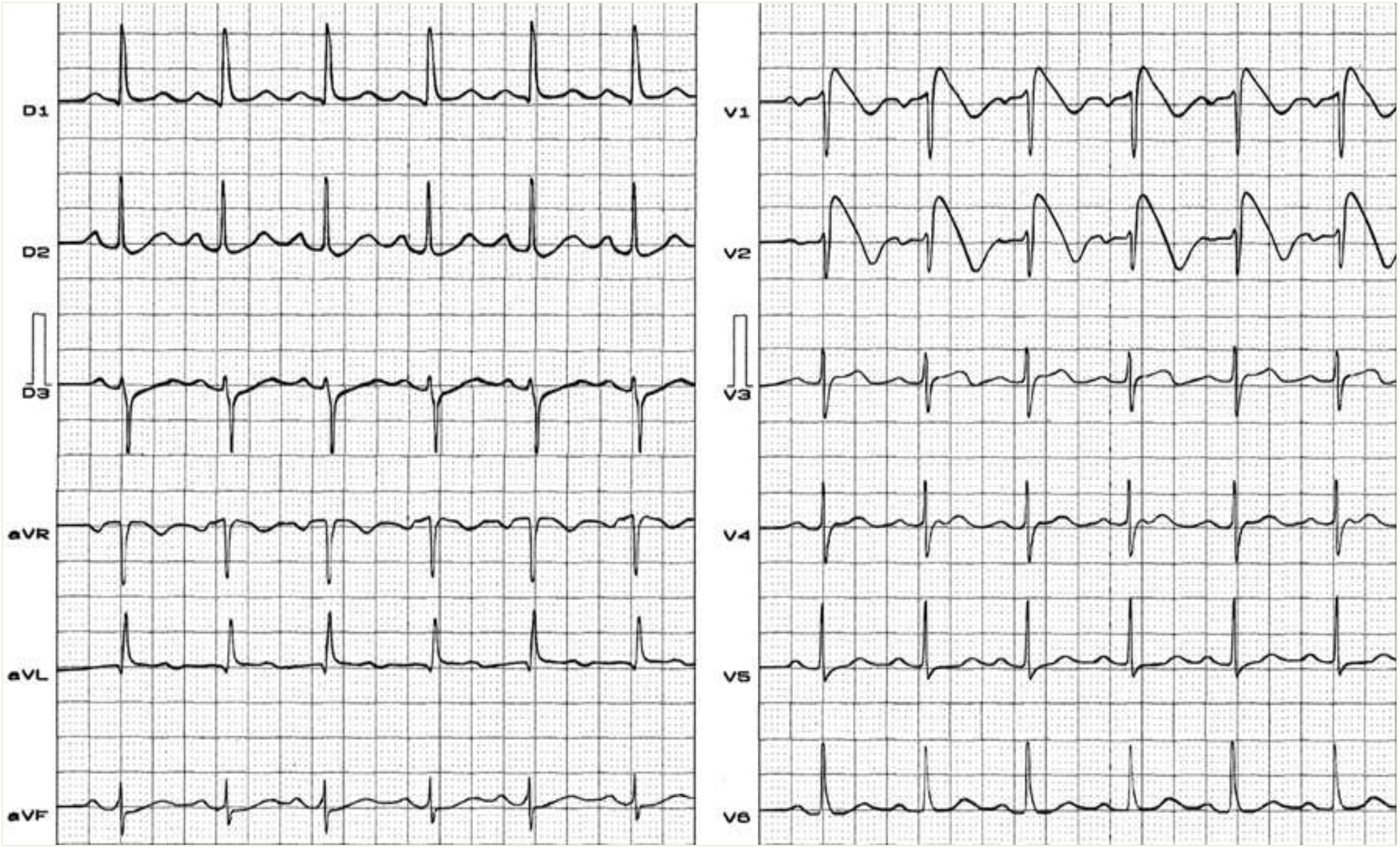
Type 1 ECG Brugada patten

Triphasic QRS complex with broad final R' wave rSR'

Truly Right Bundle Branch Block?

Type 1 Brugada pattern: J point and ST segment elevation ≥2 mm, with upper convexity or descending oblique rectilinear followed by negative T wave in the right precordial leads (V_1-V_2 or from V_1 through V_3) and/or high right precordial leads V_{1H}, V_{2H} and V_{3H}.

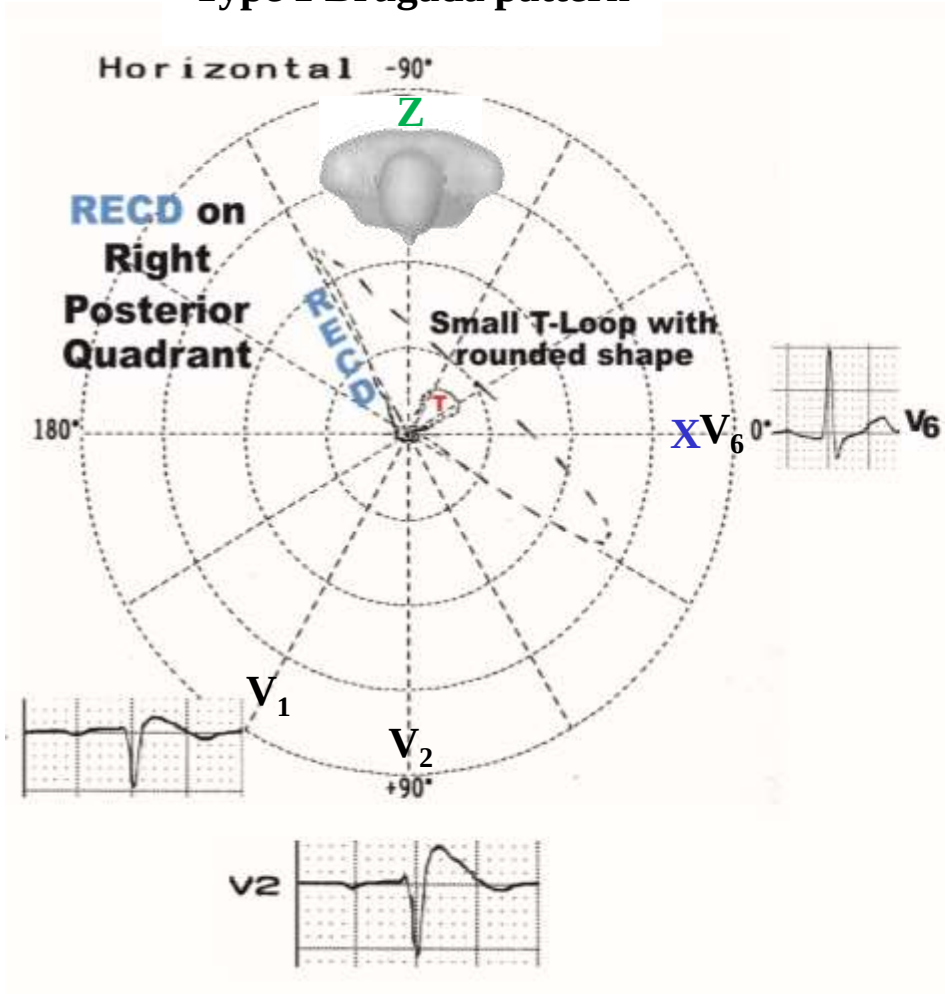
Name: SJC; **Sex**: M; **Age**: 32 y/o; **Ethnic group**: Caucasian; **Weight**: 82 Kg; **Height**: 1.76 m



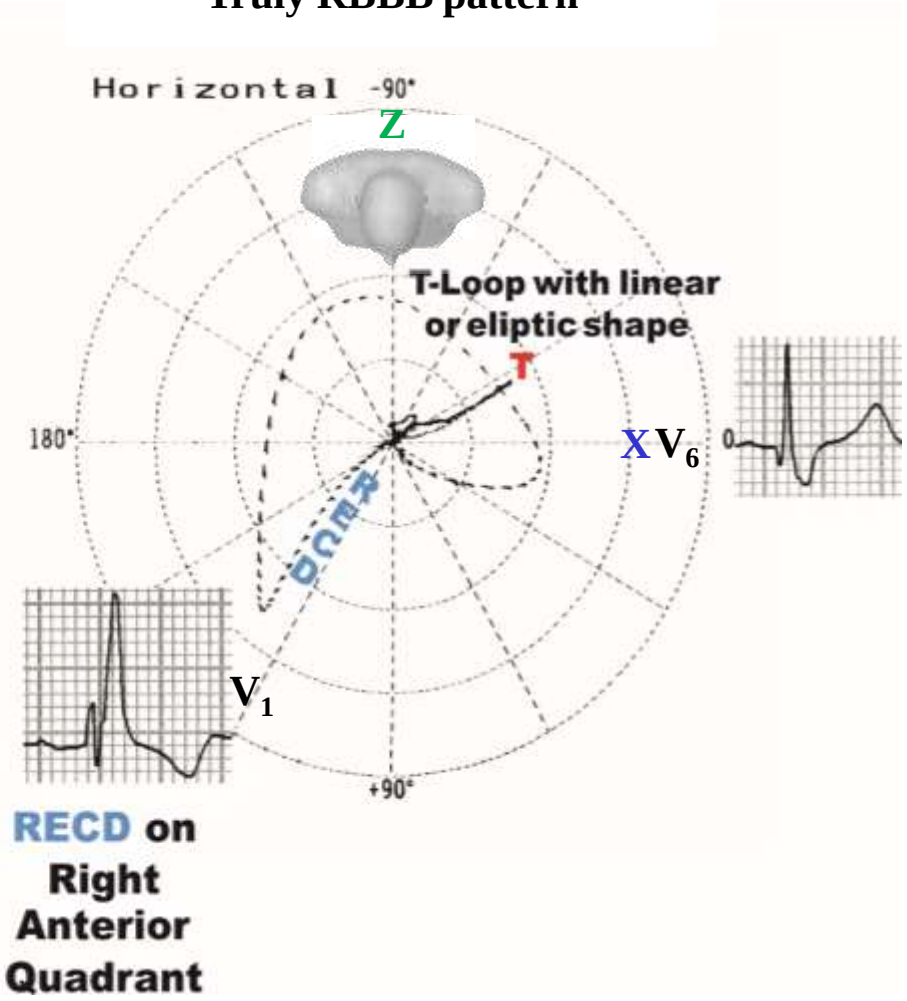
Clinical diagnosis: Symptomatic young man with Brugada syndrome. **ECG diagnosis**: Type 1 Brugada pattern without RBBB.

ECG/VCG correlation in the horizontal plane

Type 1 Brugada pattern



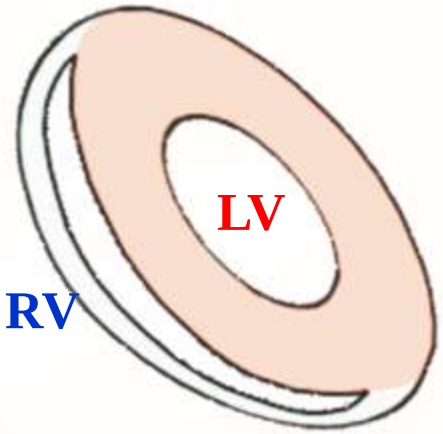
Truly RBBB pattern



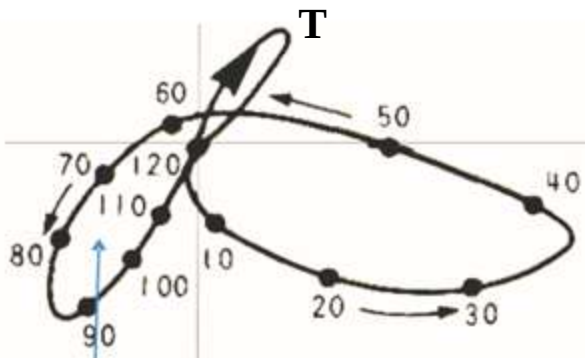
RECD on right posterior quadrant

(Pérez-Riera 2012)

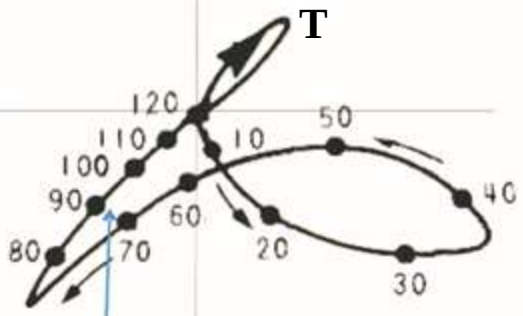
Typical VCG in RBBB HP



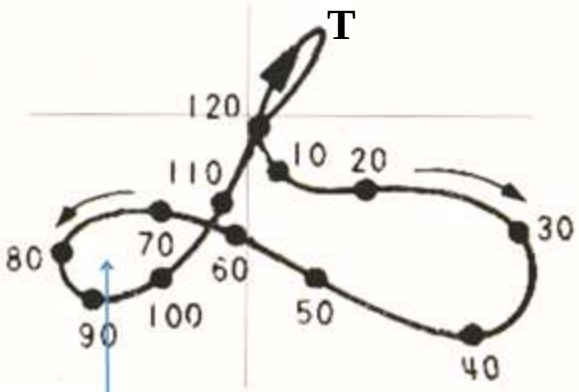
Grishman type or type I



Cabrera type or type II

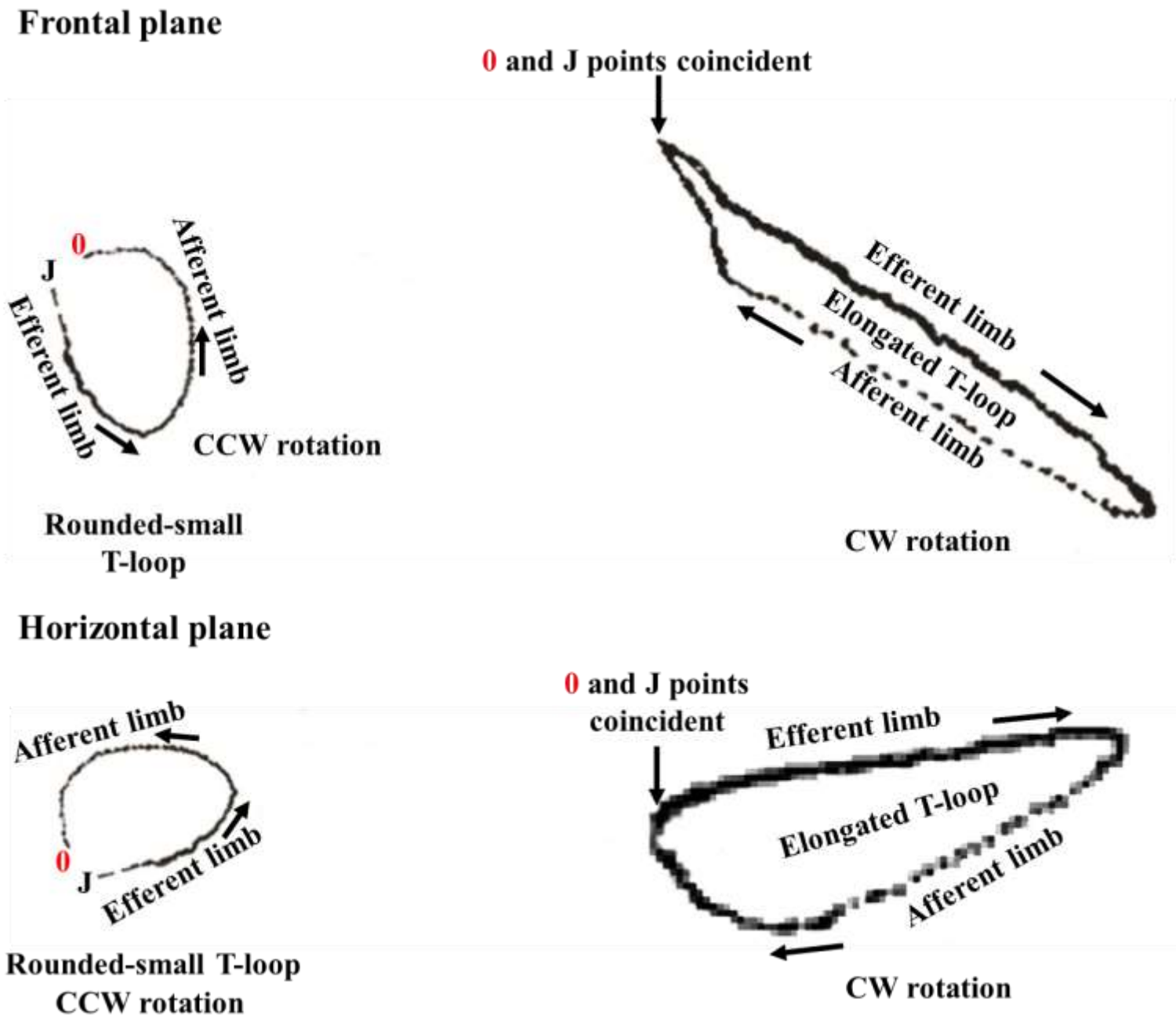


Type III or C



"Finger-like" terminal appendix

Differential characteristics between T loops of type 1 ECG Brugada pattern and RBBB



Numerous manuscripts have been published using different methods with the aim of clarifying the substrate and pathophysiological mechanism underlying BrS, such as depolarization, conduction abnormalities or conduction delay versus repolarization abnormality, for the purpose of prevention of VT/VF events (epicardial RFCA) and elimination of Brugada ECG pattern with epicardial substrate RFCA.

The methods used can be non-invasive, minimally invasive, invasive, post-mortem necropsy with molecular pathological study, and in vivo open thoracotomy RFCA experiments in animals.

Noninvasive: Body Surface (**Eckardt 2002**), tissue Doppler echocardiography (**Tukkie 2004**), vectorcardiogram, signal-averaged electrocardiogram (SAECG), and non-invasive epicardial and endocardial mapping (**Rudic 2016**).

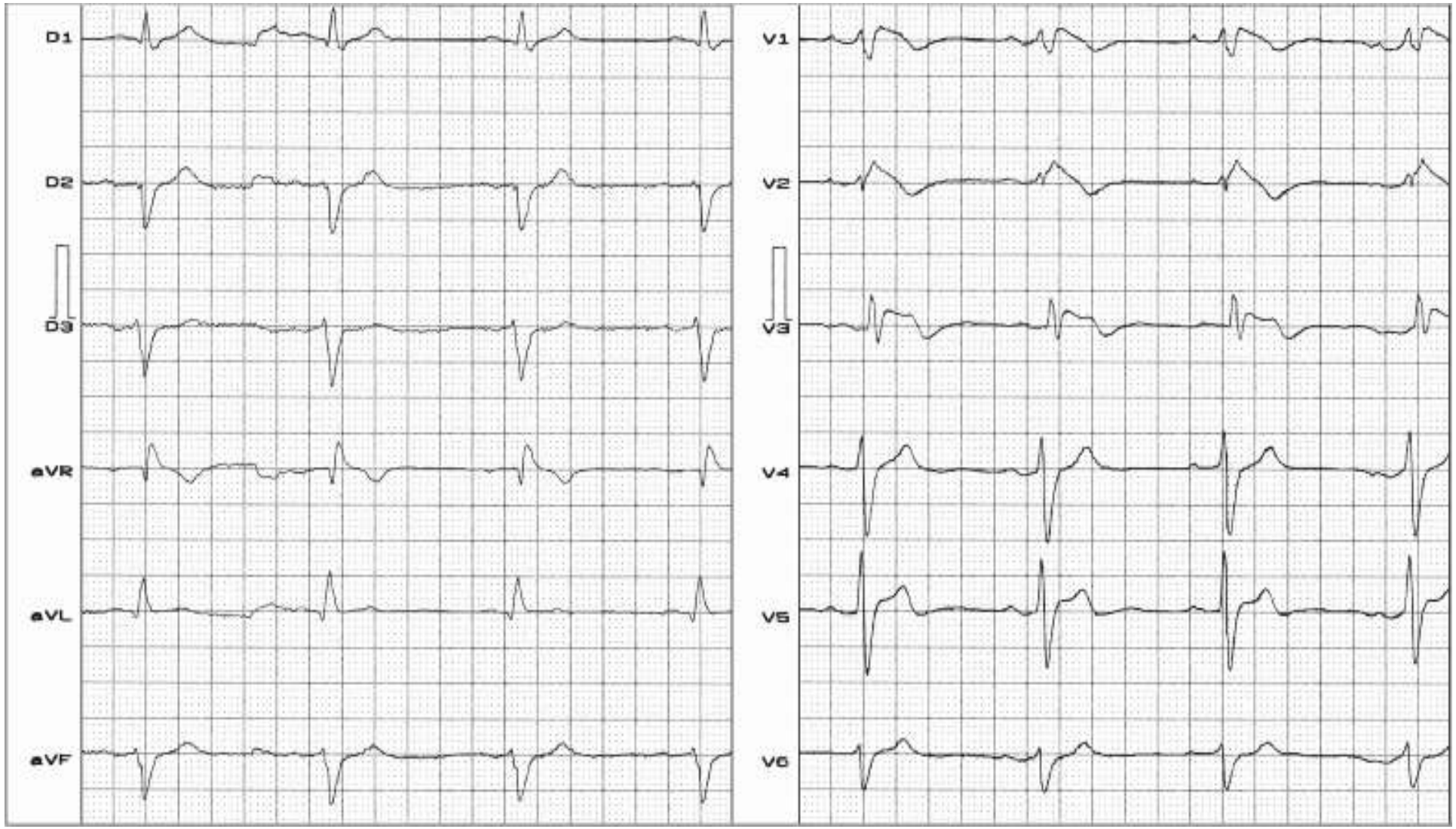
Minimally invasive: (the epicardial electrogram of the RVOT directly, introducing an electrical guidewire into the conus branch of the right coronary artery before and after class IC antiarrhythmic drug administration with SAECG (**Nagase 2002**), electrophysiology study, mapping, RFCA over the anterior RVOT epicardium, endocardial mapping and focal RFCA for ventricular fibrillation prevention (**Haïssaguerre 2003**), RV endocardial electroanatomic mapping and stimulation studies. With these methods, Postema et al observed wide and fractionated electrograms at the RV endocardium. Type 1 ECG Brugada pattern patients display additional dromotropic disturbances during sinus rhythm and premature stimulation along with abnormal transversal conduction velocity restitution caused by abnormal active membrane processes and electric coupling. The authors concluded that BrS is not solely attributable to abnormal electrophysiological properties but requires the conspiring effects of conduction slowing and tissue discontinuities (**Postema 2008**). The underlying electrophysiological mechanism in patients with BrS is dromotropic disturbance in the epicardium of the RVOT. RFCA over this abnormal area results in the normalization of type 1 ECG Brugada pattern and prevents events, both during electrophysiological studies as well as spontaneous recurrent Vt/VF episodes (**Nademanee 2011**). Using combined epicardial and endocardial electroanatomical mapping, Cortez-Dias et al performed extensive epicardial RFCA of RVOT which eliminated the fractionated electrograms, led to the disappearance of the type 1 ECG Brugada pattern six weeks after RFCA and despite discontinuation of quinidine, no further events occurred during follow-up (**Cortez-Dias 2014**).

Biopsy: it demonstrates that BrS is associated with epicardial surface and interstitial fibrosis and reduced gap junction expression in the RVOT. Additionally, BrS is also associated with increased collagen throughout the heart. Abnormal myocardial structure and conduction abnormalities are therefore responsible for BrS (**Nademanee 2015**).

Post mortem: patients with explanted heart studied electrophysiologically and histopathologically showed dromotropic disturbance based on interstitial fibrosis, but not transmural repolarization differences (repolarization abnormality) (**Coronel 2005**). The post mortem necropsies were conducted with molecular pathological study screened using PCR and direct Sanger sequencing methods (**Huang 2017**).

With experimental models: Morita et al studied a canine model using an arterially perfused canine right ventricle preparation with pinacidil (5 microM) and pilsicainide (5 microM) with which they induced a model of BrS. They then applied RFCA to the earliest activation site of premature ventricular complexes (PVCs) in the epicardium or endocardium of the RV. They observed that the epicardium may be a more effective application site than the endocardium in eliminating VT in this BrS model (**Morita 2009**).

Name: MK; **Age:** 38 y/o; **Gender:** Male; **Ethnic Group:** Asian; **Weight:** 68 kg; **Height:** 1.70 m



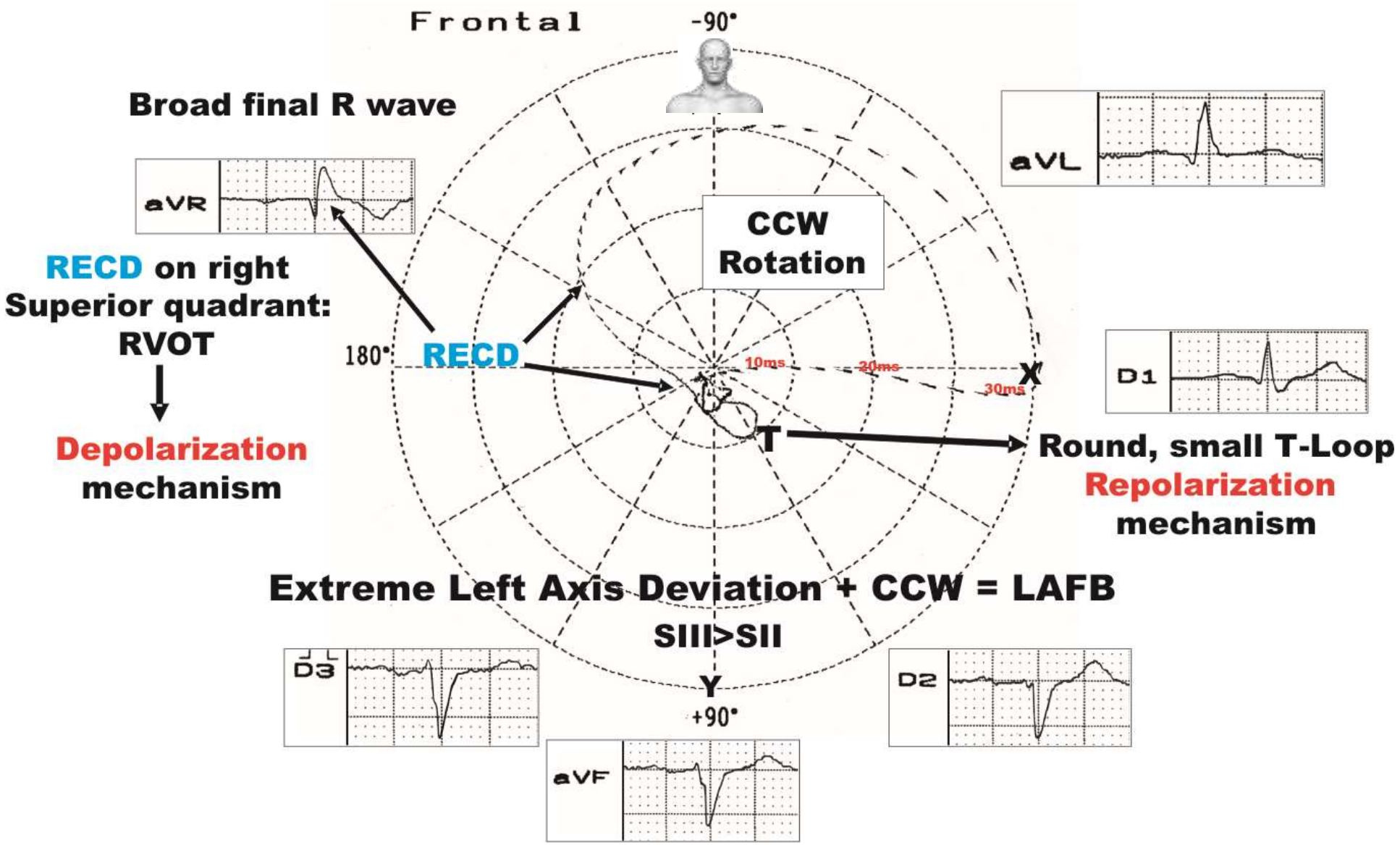
Clinical diagnosis: Syncope. Positive familiar background of sudden death in young (≤35 y/o) first-degree relative. Genetic research performed: negative.

ECG diagnosis: Sinus bradychardia (HR <60 bpm), Brugada type 1 ECG pattern, prolonged QRS duration, aVR signal: final R wave of aVR lead >3 mm, fQRS in V1-V2.

Extreme left axis deviation: Left anterior fascicular block? or Right superior fascicular block?

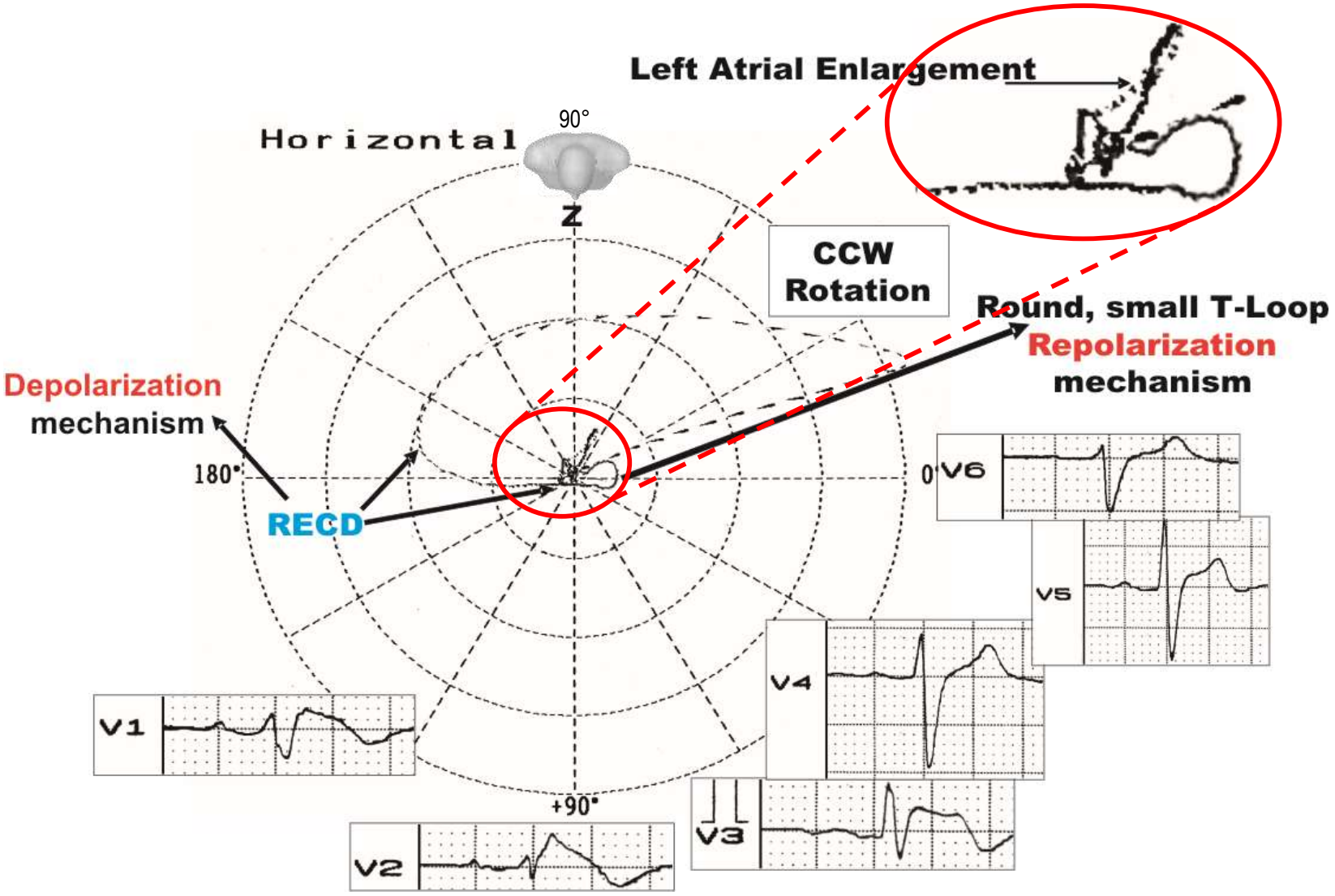
ECG/VCG correlation in the frontal plane

Name: MK; **Age:** 38 y/o; **Gender:** Male; **Ethnic Group:** Asian; **Weight:** 68 kg; **Height:** 1,70 m



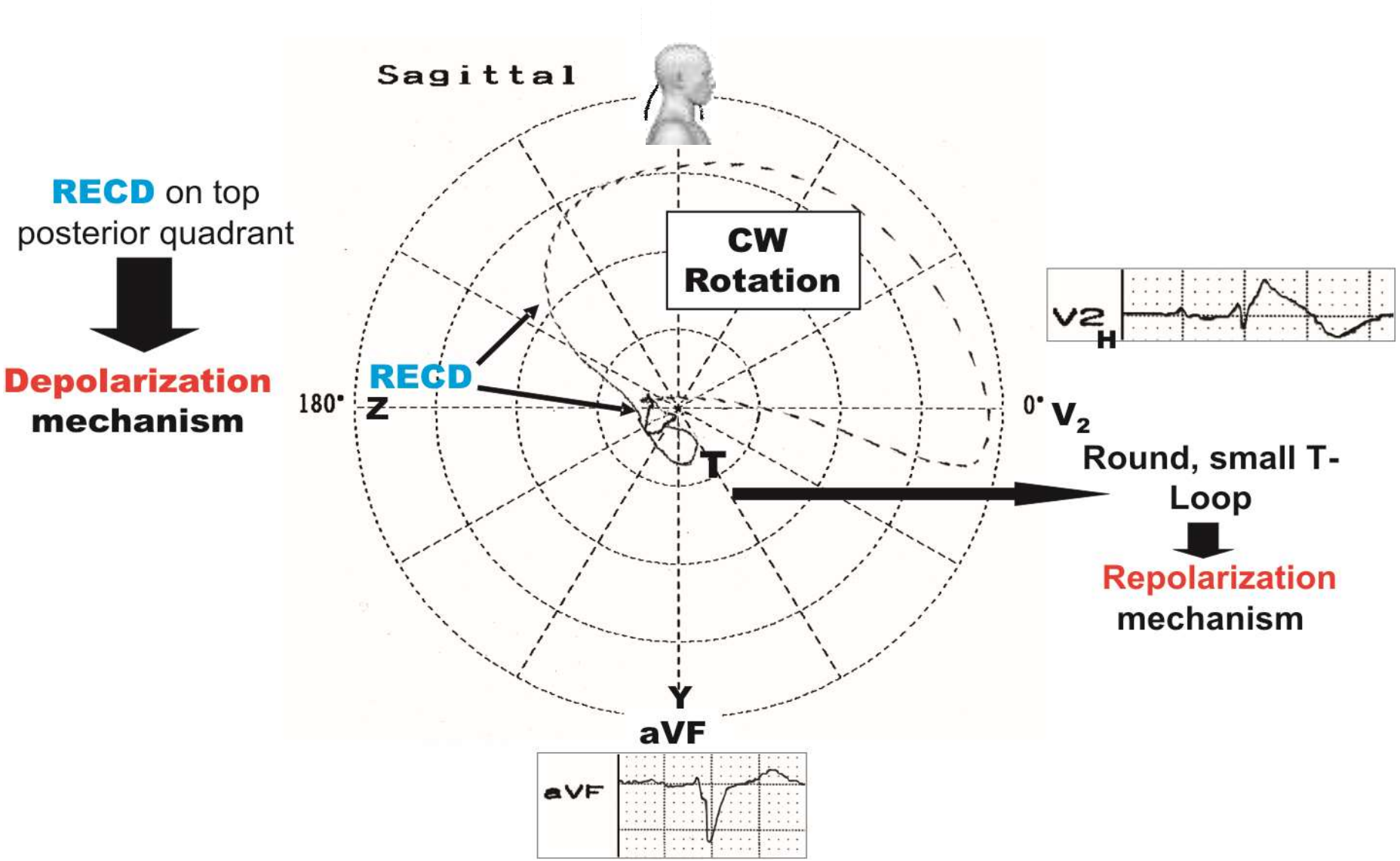
ECG/VCG correlation horizontal plane

Name: MK; **Age:** 38 y/o; **Gender:** Male; **Ethnic Group:** Asian; **Weight:** 68 kg; **Height:** 1.70 m.



ECG/VCG correlation in the right sagittal plane

Name: MK; **Age:** 38 y/o; **Gender:** Male; **Ethnic Group:** Asian; **Weight:** 68 kg; **Height:** 1.70 m



References

1. Alzamora-Castro V, Abugattas R, Rubio C, et al. Parietal focal block; an experimental and electrocardiographic study. Circulation. 1953;7(1):108-15.
2. Babai Bigi MA, Aslani A, Shahrzad S. aVR sign as a risk factor for life-threatening arrhythmic events in patients with Brugada syndrome. Heart Rhythm. 2007;4(8):1009-12.
3. Bayés de Luna A , Cosin J, Carrió J, et al. Right Ventricular peripheric blocks: Diagnostic Problem. In Masoni A, Alboni P, eds. Cardiac Electrophysiology Today. Academic Press, London; New York, 1982, 401.
4. Bayés de Luna A, Carrió I, Subirana MT, et al. Electrophysiological mechanisms of the SI SII SIII electrocardiographic morphology. J Electrocardiol. 1987;20(1):38-44.
5. Coronel R, Casini S, Koopmann TT, et al. Right ventricular fibrosis and conduction delay in a patient with clinical signs of Brugada syndrome: a combined electrophysiological, genetic, histopathologic, and computational study. Circulation. 2005;112(18):2769-77.
6. Cortez-Dias N, Placido R, Marta L, Bernardes A, Sobral S, Carpinteiro L, de Sousa J. Epicardial ablation for prevention of ventricular fibrillation in a patient with Brugada syndrome. Rev Port Cardiol. 2014;33(5):305.e301–307.
7. de Micheli A, Medrano GA. [Disorders of intraventricular conduction]. Arch Inst Cardiol Mex. 1987 May-Jun;57(3):247-58.
8. de Micheli A, Medrano GA, Iturralde-Torres P. Uncomplicated and complicated myocardial peripheral blocks. Arch Cardiol Mex. 2009;79 Suppl 2:3-12.
9. Doi A, Takagi M, Maeda K, Tatsumi H, Shimeno K, Yoshiyama M. Conduction delay in right ventricle as a marker for identifying high-risk patients with Brugada syndrome. J Cardiovasc Electrophysiol. 2010;21(6):688-96.
10. Eckardt L, Bruns HJ, Paul M, et al. Body surface area of ST elevation and the presence of late potentials correlate to the inducibility of ventricular tachyarrhythmias in Brugada syndrome. J Cardiovasc Electrophysiol. 2002;13(8):742-9.
11. Haïssaguerre M, Extramiana F, Hocini M, et al. Mapping and ablation of ventricular fibrillation associated with long-QT and Brugada syndrome. Circulation 2003;108(8):925-8.
12. Hayashi H, Sumiyoshi M, Yasuda M, et al. Prevalence of the Brugada-type electrocardiogram and incidence of Brugada syndrome in patients with sick sinus syndrome. Circ J. 2010;74(2):271-7.
13. Huang L, Tang S, Chen Y, et al. Molecular pathological study on LRRC10 in sudden unexplained nocturnal death syndrome in the Chinese Han population. Int J Legal Med. 2017;131(3):621-8.

14. Lenègre J. Contribution a l'Etude des Blocs de Branche. Paris, JB Bailliere, 1958.
15. Lev M. The normal anatomy of the conduction system in man and its pathology in atrioventricular block. Ann N Y Acad Sci. 1964;111:817-29.
16. Lev M. The conduction system. In: Gould S. Pathology of the heart and blood vessels. Springfield, III. Ed. Charles C Thomas; 1968. pp. 180.
17. Mahaim I. Les maladies organiques du faisceau de His-Tawara. Paris. Ed. Masson & Cie; 1931. pp. 31.
18. Masini V, Testoni F, Farulla A. Focal blocks of the right intraventricular conduction: morphology, pathogenesis and clinical significance. Cardiologia. 1952;21(3):171-87.
19. Morita M, Zipes DP, Morita ST, Lopshire JC, Wu J. Epicardial ablation eliminates ventricular arrhythmias in an experimental model of Brugada syndrome. Heart Rhythm 2009;6(5):665-71.
20. Nagase S, Kusano KF, Morita H, et al. Longer repolarization in the epicardium at the right ventricular outflow tract causes type 1 electrocardiogram in patients with Brugada syndrome. J Am Coll Cardiol. 2008;51(12):1154-61.
21. Nademanee K, Veerakul G, Chandanamattha P, et al. Prevention of ventricular fibrillation episodes in Brugada syndrome by catheter ablation over the anterior right ventricular outflow tract epicardium. Circulation 2011;123(12):1270-9.
22. Nademanee K, Raju H, de Noronha SV, et al. Fibrosis, Connexin-43, and Conduction Abnormalities in the Brugada Syndrome. J Am Coll Cardiol. 2015;66(18):1976-86.
23. Noseda V, Santi A, Aguggini G, et al. Experimental focal block in the sheep and the dog. An electrocardiographic and histological study. Cardiologia. 1963;42:243-55.
24. Pastore CA, Moffa PJ, Spiritus MO, et al. Fascicular blocks of the right branch. Standardization of vectorelectrocardiographic findings. Arq Bras Cardiol. 1983;41(3):161-6.
25. Peréz-Riera AR, Ferreira Filho C, de Abreu LC, et al. Do patients with electrocardiographic Brugada type 1 pattern have associated right bundle branch block? A comparative vectorcardiographic study. Europace. 2012;14(6):889-97.
26. Postema PG, van Dessel PF, de Bakker JM, et al. Slow and discontinuous conduction conspire in Brugada syndrome: a right ventricular mapping and stimulation study. Circ Arrhythm Electrophysiol. 2008;1(5):379-86.
27. Rossi P. Atypical electrocardiographic aspects of focal blocks of the right intraventricular conduction. Cuore Circ. 1954;38(1):16-22
28. Rudic B, Chaykovskaya M, Tsyganov A, et al. Simultaneous Non-Invasive Epicardial and Endocardial Mapping in Patients With Brugada Syndrome: New Insights Into Arrhythmia Mechanisms. J Am Heart Assoc. 2016;5(11). pii: e004095.
29. Tukkie R, Sogaard P, Vleugels J, de Groot IK, Wilde AA, Tan HL. Delay in right ventricular activation contributes to Brugada syndrome. Circulation. 2004;109(10):1272-7.

30. Uhley HN, Rivkin L. ECG patterns following interruption of main and peripheral branches of the canine right bundle Am J Cardiol. 1961;7:810-6.