Critical proximal obstruction of LAD, transient intraventricular conduction disturbances, and Brugada ECG pattern: BrS?

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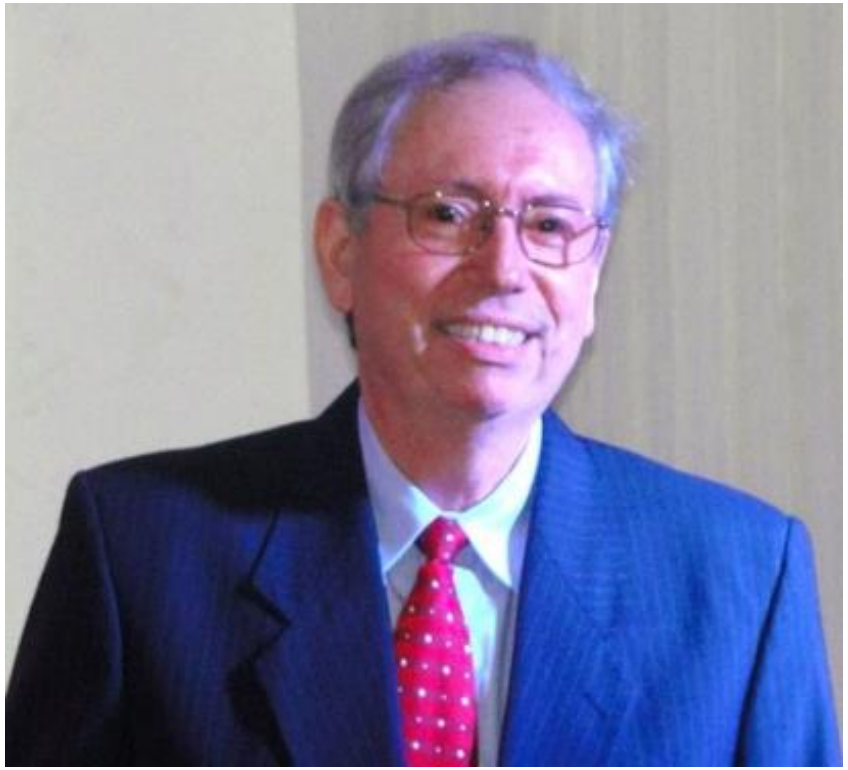


Raimundo **Barbosa-Barros**, MD
Chief of the Coronary Center of the Hospital de Messejana Dr. Carlos Alberto Studart Gomes. Fortaleza – CE- Brazil

Case report

Male Asian patient, 58 years old, mesomorph biotype, reported that approximately 72 hours ago he had presented several episodes of constrictive precordial pain, each of short duration (≤ 10 minutes) that ceased with rest, and some of them radiated to the mandible and the inner part of the left upper arm until the wrist. The patient also reported cold sweating and nausea triggered by usual activities such as walking or driving a car.

Personal pathological antecedents: hypertension diagnosed five years ago, but well controlled. Familial type II dyslipidemic controlled by in regular use of 10 mg of rosuvastatin daily, with recent normalized lipid laboratory exams. Smoker since adolescence that stopped two years ago.

Family history: A brother and a first-degree cousin (29 and 35 years old respectively) from father's side both had sudden death during nighttime sleep. In both, a molecular autopsy was performed that revealed a mutation in the SCN5A gene. Genetic screening has not been performed yet in the rest of the family.

Physical examination: Nothing noteworthy.

An electrocardiogram was performed on admission (April 18, 2017 at 10:00 AM). in the emergency room (Figure 1) and the next day another (Figure 2). Only the second one placing the electrodes in high right precordial leads (V_{1H}, V_{2H} and V_{3H})

Transthoracic Two-dimensional Doppler Echocardiogram: Normal

Multi-slice Coronary computed tomography (MSCT) examination for evaluation of acute chest pain: showed a proximal critical obstruction of the left anterior descending artery (LAD) before the first perforator branch(S_1) (Figure 3).

Questions:

1. What is the diagnosis of ECG-1?
2. What is the diagnosis of ECG-2?
3. Do VCGs help us? Why?
4. What is the clinical-electrocardiographic diagnosis?
5. Mark the differences between both tracings and explain their mechanisms.

Figure 1 - ECG-1 (April 18, 2017 at 10:00 AM)

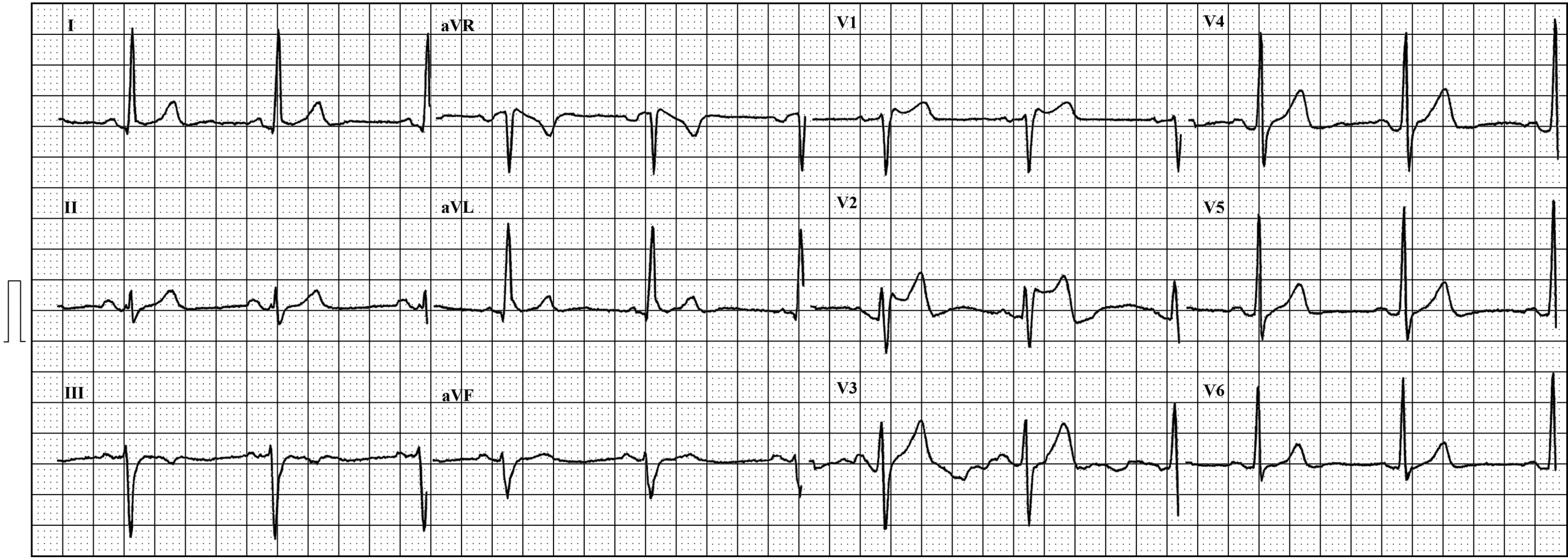


Figure 2 - ECG-2 (April 19, 2017 at 9:10 AM)

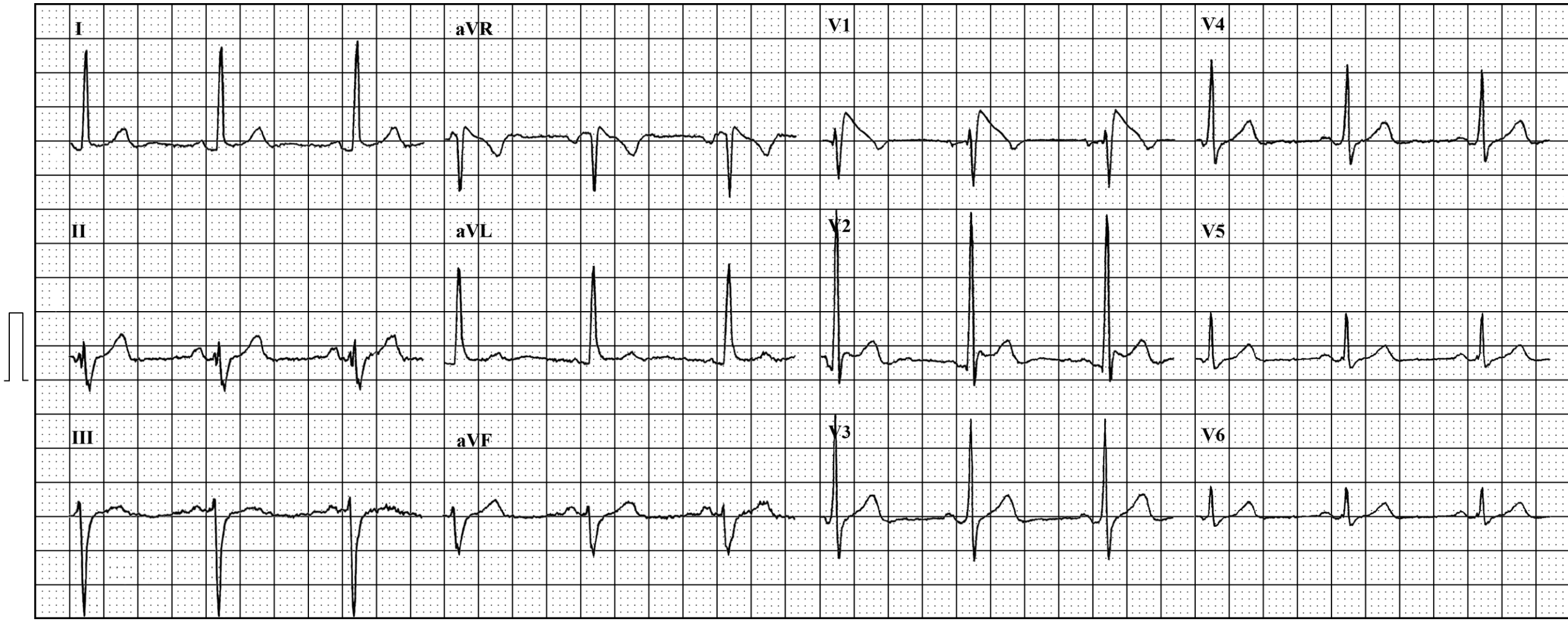
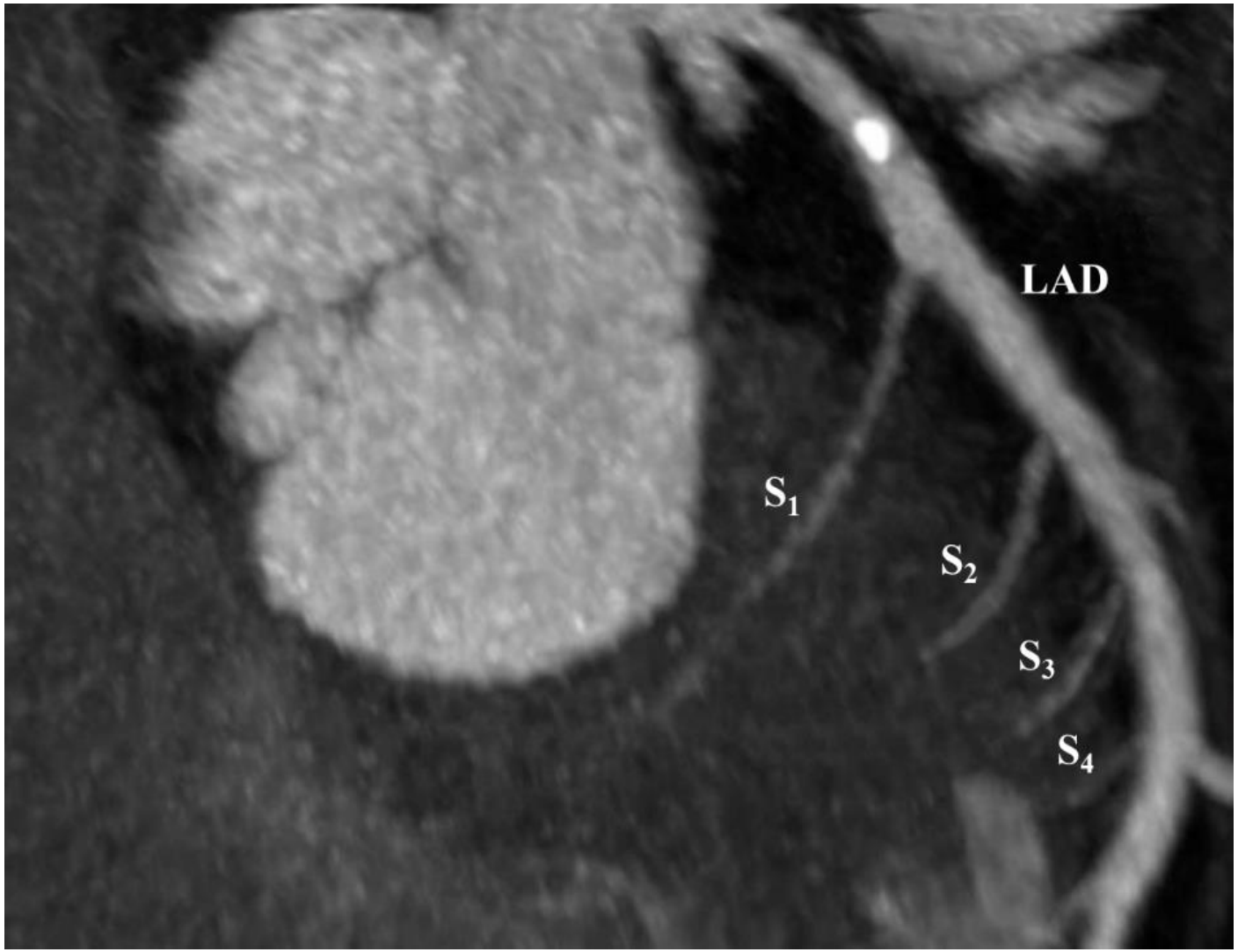
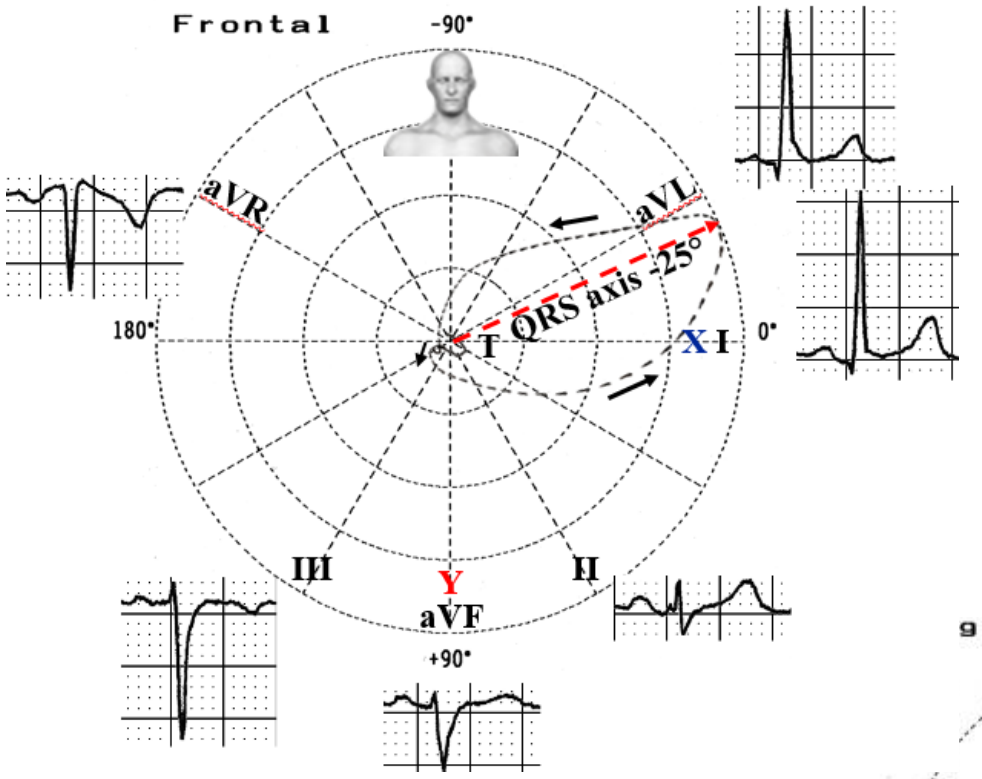


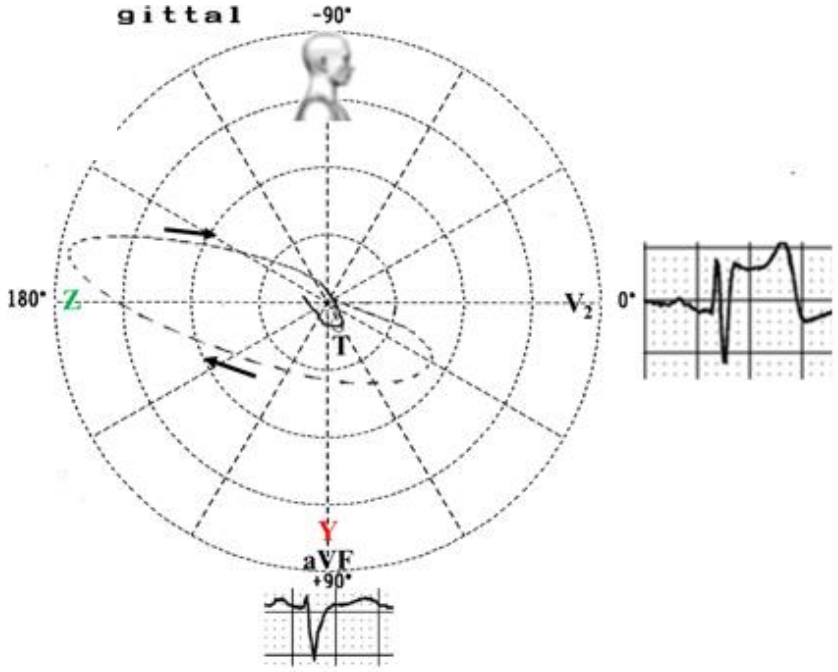
Figure 3 Multi-Slice Coronary Computed Tomography (MSCT) examination for evaluation of acute chest pain

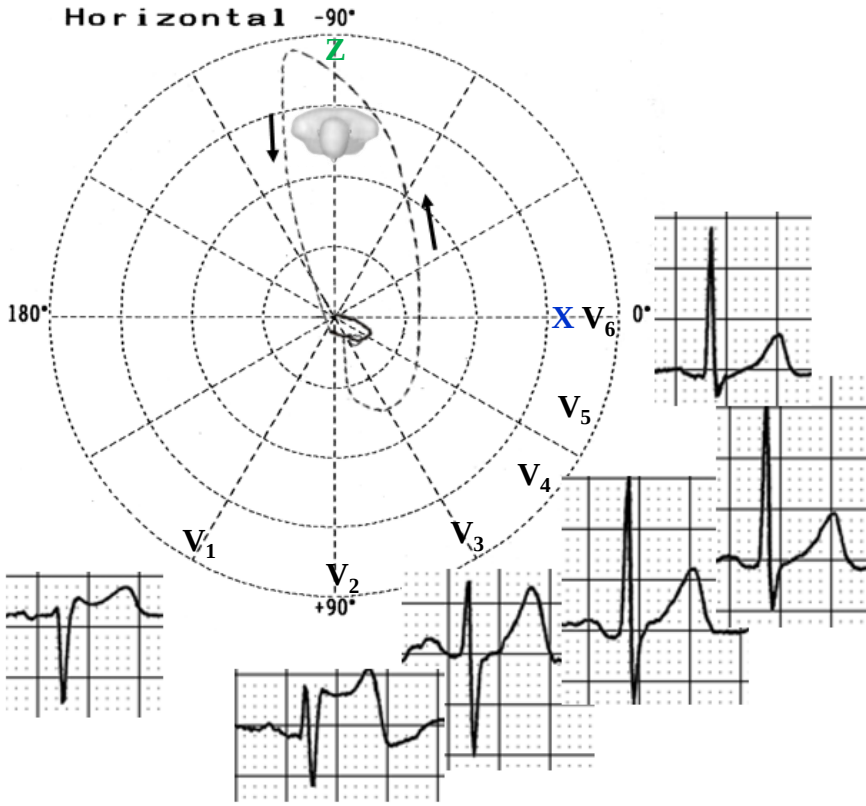


Atherosclerotic plaque with critical obstruction located in the first third of **LAD** before the first anterior septal perforator branch (S_1); **S_2:** second Anterior Septal Perforator Branch; **S_3:** third Anterior Septal Perforator Branch; **S_4:** fourth Anterior Septal Perforator Branch; **LAD**: Left Anterior Descending Artery.

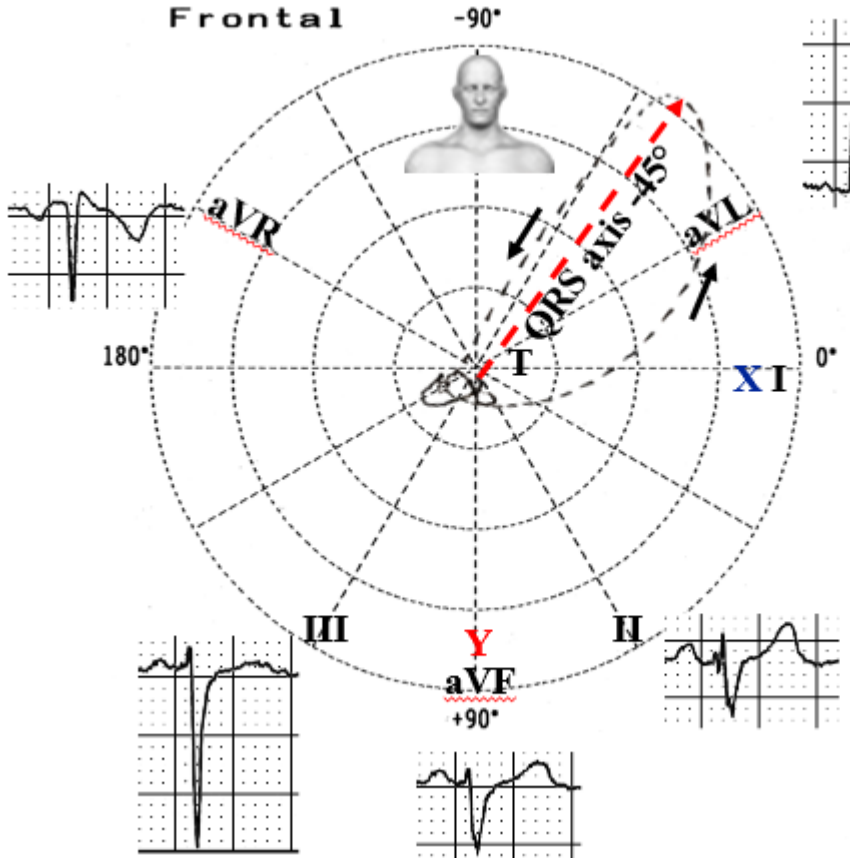
ECG-1/VCG-1 correlation in the three planes

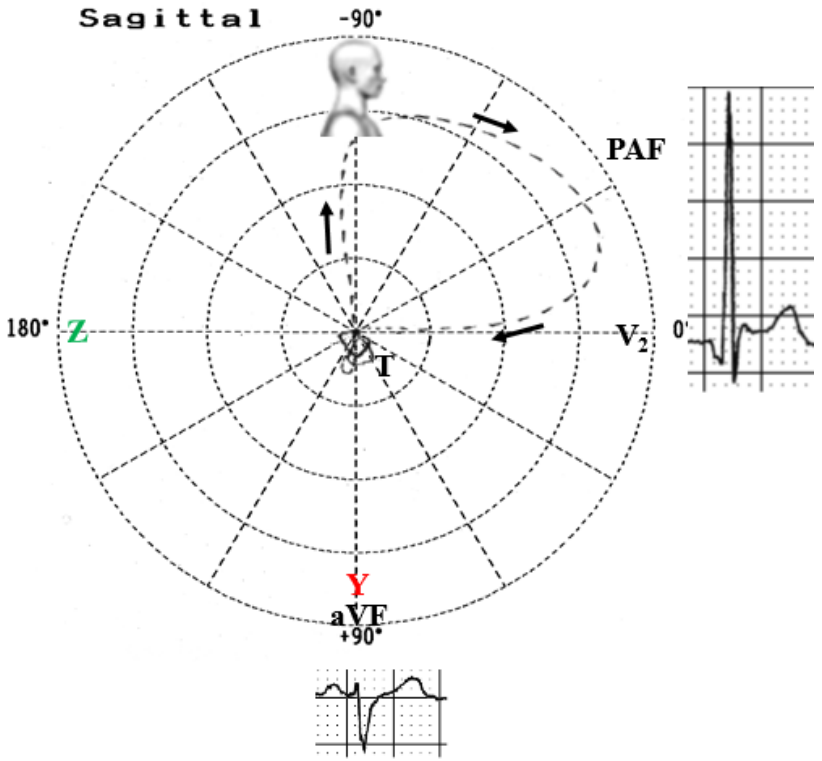


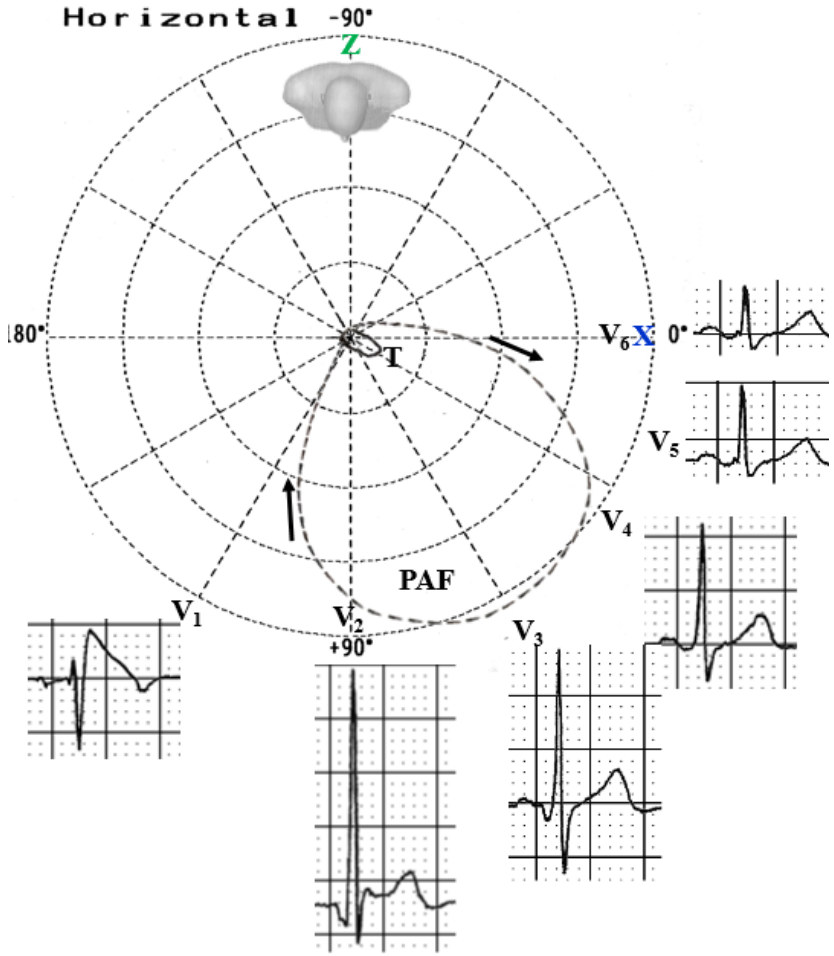




ECG-2/VCG-2 correlation in the three planes







PAF: prominent QRS anterior forces

Colleagues opinions

Very interesting

The ECGs are suggestive of Brugada ECG pattern

Some patients with Brugada syndrome present with atypical chest pain.

I am thinking that Brugada syndrome is more complex than other channelopathies

CT angio could be an incidental finding

Do not know much about vectocardiographic pattern

looking forward for the right answer

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Dear Andrés and Raimundo – certainly a rare case! It is difficult to interpret, but I will try.

1. Possible proximal LAD occlusion in a patient with type 1 Brugada ECG pattern, left axis deviation
2. Brugada 1 pattern, septal fascicular block , left anterior fascicular block
3. Helps in the diagnosis of intraventricular block
4. Coronary spasm associated with proximal LAD occlusion, Brugada ECG
5. ECG 1: ischemia involved, ECG 2: no ischemia. The placement of the electrodes affect the recording.

Best regards

Kjell Nikus MD PhD.

Tampere, Finland

Spanish

Buen día estimados Andrés y Raimundo! Hermoso caso para leer vuestra devolución y seguir aprendiendo. Hombre de origen asiático con típico angor, cortejo neurovegetativo y FRCV positivos para enfermedad coronaria. Con historia familiar de 2 varones (hermano y primo paterno) con MS antes de los 45 años y estudio genético con mutación de SCN5A. Pero sin historia de palpitaciones paroxísticas, síncope, TV/TVP documentada, respiración agónica. El BrS es más frecuente en varones 8:1 en relación a mujeres, dominante y diferente penetrancia.
En ECG #1 ritmo sinusal 60 lpm con PR normal y patrón de BrS tipo 2.
En ECG #2 con precordiales altas patrón de BrS tipo 1, con eje desviado a la izquierda por HASI y BDAM, esta última por oclusión de la DA antes de las septales, única rama del sistema hisiano irrigada 100% por ramas perforantes septales de DA. Ambos tienen Ángulo beta > 58° y base del triángulo > a 160 mseg.
Diagnóstico diferencial:
1) BrS verdadero congénito
2) BrS adquirido
3) Fenocopia.
En el vectocardiograma se observa la progresión del asa del QRS hacia izquierda y hacia adelante (HASI + BDAM).
La isquemia puede inducir fenocopia o desenmascarar un BrS latente, adquirido.
Observaría la evolución de los ECG, (la normalización descartaría fenocopia), haría pruebas de provocación con drogas bloqueantes de canales de sodio (ajmalina, flecainida, etc) y estudios genéticos en busca de mutaciones.
Saludos cordiales
Dr Juan Carlos **Manzzardo MD Mendoza Argentina**

English

Good morning Dear Andrés and Raimundo Beautiful case to read your answer and continue learning. Asian man with typical angor, neurovegetative cortege and positive cardiovascular risk factors for CAD. Positive family history of 2 males (paternal brother and cousin) with MS before age 45 yo and genetic study with mutation of SCN5A. But no history of paroxysmal palpitations, syncope, documented TV / DVT, agonized breathing. The BrS is more frequent in men 8: 1

In ECG # 1 sinus rhythm 60 bpm with normal PR and type 2 Brugada ECG pattern.

In ECG # 2 with high right precordial leads type 1 BrP , left axis deviated by LAFB and LSFB the latter by occlusion of the DA before the first septal perforator. This fascicles is irrigate in 100% of cases bal LAD. Both ECGs have broad β- angle(> 58 °) and triangle base > 160 msec.

Differential diagnosis:

1) True hereditary BrS

2)Adquired BrS

3) Phenocopy.

In the VCG, the QRS loop of the HP is located predominanty tiin anterior left quadranat and in the FP has extreme left axis deviation (LSFB + LAFB) is observed.

Ischemia may induce phenocopy or unmask a latent, acquired BrS.

I would observe the evolution of ECGs, (normalization would rule out phenocopia), provocative tests with sodium channel blockers (ajmaline, flecainide, etc.) and genetic screening .

Best regards

Dr Juan Carlos Manzzardo MD Mendoza Argentina

Estimados Potro, Raimundo e colegas:
Interessantíssimo e raro caso. Inicialmente acho que o paciente tem duas patologias:
1. Padrão de Brugada típico espontâneo no ECG.
2. Aterosclerose clínica homem 58 anos em quadro agudo angina severa de manifestação tardia pelo diagnóstico de hiperlipidemia familiar cujo curso clínico habitualmente costuma ser mais grave e precoce.
Quanto aos questionamentos:

1. ECG 1: Síndrome coronária com isquemia grau 3 (Sclarovsky & Birbaum) em V1 e V2. Parede ântero-septal e onda q embrionária em I e aVL com elevação de 1 MV nestas derivações.
2. ECG 2: Padrão espontâneo típico de Brugada tipo I nas derivações direitas altas. Diminuição da elevação em V1 e V2. Desaparecimento de Q e elevação em I e aVL. Forças anteriores proeminentes. RV2 = 2,3MV sugerindo bloqueio divisional septal esquerdo e bloqueio divisional antero-superior esquerdo (SÂQRS no plano frontal = 45º).
3. O VCG ajuda porque comparativamente no plano horizontal a alça do QRS desloca-se para frente e a esquerda, com rotação horária e no sagital direito a alça QRS está predominantemente localizada no quadrante antero-superior com o mesmo sentido.
4. Diagnóstico clínico eletrocardiográfico: Síndrome coronária aguda, síndrome de Brugada e padrões tipo 1 e 2 associado a BDAS + BDAM.
5. Alterações eletrocardiográficas produzidas por isquemia aguda e Brugada detectada pela reperfusão (espasmos coronários). Trombo semioclusivo?

Abraços
Adail

Dear Andrés, Raimundo and colleagues:

Very interesting and exceptional case. Initially I think that the patient has clinically two entities: BrS + CAD.

1. Typical spontaneous Brugada pattern in the ECG.

2. Clinical atherosclerosis man 58 years in acute severe angina of late manifestation by the diagnosis of familial hyperlipidemia whose clinical course usually is more severe and precocious.

As for the questions:

1. ECG 1: CAD with ischemia grade 3 (Sclarovsky & Birbaum) in V1 and V2. Anteroseptal wall with embryonic initial q wave in I and aVL with elevation of 1 mV in these leads.
2. ECG 2: Typical spontaneous type I Brugada pattern in the right precordial leads with high placement. Elevation decreased in V1 and V2. Q disappears and elevation in I and aVL. Prominent anterior forces. RV2 = 2.3mV suggesting left septal fascicular block (LSFB) and left anterior fascicular block (LAFB). The last one because SÂQRS in the frontal plane = -45º.
3. VCG helps because comparatively in the horizontal plane the QRS loop shifts to the left anterior quadrant clockwise rotation and in the sagittal plane goes to the antero-superior quadrant with the same rotation.
4. Clinical-Electrocardiographic diagnosis: CAD with critical proximal obstruction of LAD + LAFB + LSFB (left bifascicular block).
5. Electrocardiographic changes produced by acute ischemia and Brugada are detected by reperfusion (coronary spasms). Semiocclusive thrombus?

Hugs

Adail

Spanish

La presentación es de un paciente masculino con antecedentes de angor de esfuerzo con episodios de corta duración que comienzan 3 días previos a su internación, con factores de riesgo coronario y antecedentes familiares de muerte súbita por mutación en el gen SCN5A. Por angiografía coronaria computarizada multicorte se detecta obstrucción significativa proximal de la descendente anterior.
El primer ECG muestra un ritmo sinusal, con una FC de 75 por minuto. El primer vector se ubica hacia la derecha abajo y adelante, probablemente con rotación horaria en su comienzo en el plano frontal y luego antihoraria (ver FIG1), lo cual podría corresponder a una fibrosis inferior con HBAI (onda R empastada en DII y aVF). Las derivaciones precordiales muestran un supradesnivel del punto J y ST en V1 y V2, difícil de diferenciar entre una patente tipo II de Brugada y/o una cardiopatía isquémica, ya que las ondas T tienden a ser picudas y simétricas en cara anterior extensa.

El segundo ECG las derivaciones precordiales altas muestran una patente que impresiona como el de un patrón tipo I de Brugada. No entiendo porque en las derivaciones frontales aumenta tanto la onda S de DII y DIII; y la de aVF permanece de la misma magnitud (debería aumentar en forma proporcional a DII y DIII); DI presenta un infradesnivel del PR por lo tanto no se si la falta de onda Q es verdadera o no.
Un VCG hubiese aclarado posiblemente lo que ocurre.

El primer VCG (FIG 3) está superpuesto el plano frontal con el horizontal. El primer vector en el plano frontal se dirige hacia la derecha y en el horizontal se orienta a la izquierda: Ambos planos comparten el eje de X, por lo tanto la proyección en un plano de los vectores sobre el mismo debe ser la misma. El vector R del plano frontal (línea azul) queda fuera del bucle en el plano horizontal; no coincide. Además la rotación inicial que se visualiza en el ECG en el plano frontal del asa del QRS no se observa en el VCG presentado.
He tratado de esquematizar porque esto no es un VCG en las FIG 4. En la FIG5 trato de explicar como debería inscribirse, si esto fuese así, el ECG en el segundo VCG en el plano horizontal: los complejos QRS deberían ser QR en V1 con una onda q decreciente hacia V3 y la onda s recién debería aparecer en la derivación V6

.

Afectuosamente Isabel Konopka

English

The presentation is of a male patient with a history of exertional angor with short-term episodes beginning 3 days prior to hospitalization, with coronary risk factors and family history of sudden death due to mutation in the SCN5A gene. Multislice computerized coronary angiography shows significant proximal obstruction of the anterior descending.? The first ECG shows a sinus rhythm, with a HR of 75 per minute. The first vector is located to the right down and forward, probably with hourly rotation at its beginning in the frontal plane and then counterclockwise (see FIG1), which may correspond to a lower fibrosis with HBAI (R wave packed in DII and aVF) . The precordial leads show an elevation of the point J and ST in V1 and V2, difficult to differentiate between a Brugada type II patent and / or ischemic heart disease, since T waves tend to be bearded and symmetrical in an extensive anterior face.

• The second ECG the high precordial leads show an impressive patent like that of a Brugada type I pattern. I do not understand why in the frontal leads increases both the S wave of DII and DIII; And that of aVF remains of the same magnitude (should increase proportionally to DII and DIII); DI has a lack of PR so I do not know if the lack of Q wave is true or not. A VCG would have clarified possibly what happens.

• The first VCG (FIG 3) is superimposed on the front plane with the horizontal plane. The first vector in the frontal plane is directed to the right and in the horizontal is oriented to the left: Both planes share the axis of X, therefore the projection in a plane of the vectors on it must be the same. The vector R of the front plane (blue line) is outside the loop in the horizontal plane; Does not match. In addition, the initial rotation displayed on the ECG in the frontal plane of the QRS loop is not seen in the presented VCG. I have tried to schematize because this is not a VCG in FIG 4. In FIG 5 I try to explain how it should The ECG should be recorded in the second VCG in the horizontal plane: QRS complexes should be QR in V1 with a decreasing q wave towards V3 and the s wave should only appear in lead V6

Affectionately Isabel Konopka

Spanish

Hola amigos del foro:
Caso interesante , como todos los que enviais.
Mis comentarios:
- Patrón de Brugada tipo 1 que se magnifica al colocar los electrodos V1-V3 mas altos (antecedentes familiares de MS)
- En un paciente que refiere dolor torácico. Posibilidades:
a) Cardiopatia isquémica con arteria no ocluida (angina) en un paciente con S. de Brugada
b) Fenocopia de Brugada en una paciente con espasmo sobre una lesión proximal de la descendente anterior (la tomografia muestra una "calcificación" proximal a S1 pero no quiere decir que la oclusión sea crítica)
c) STEMI por oclusión proximal de la DA que simula un patrón de Brugada (fenocopia). No lo creo.
- Criterios de hipertrofia en el plano frontal
- Hemibloqueo de la subdivisión anterior
- No puedo opinar sobre VCG porque en nuestro medio no se utiliza

Saludos

Miquel **Fiol-Sala, MD, PhD**
Director Científico del Instituto de Investigación
Sanitaria de Palma (IdISBa)

English

Hello friends of the forum: Interesting case, like all that send to us. My comments:

- Type 1 Brugada pattern that is magnified by placing the highest V1-V3 electrodes (family history of MS)
- In a patient with Chest pain. Possibilities:

A) Ischemic cardiomyopathy with non-occluded artery (angina) in a patient with Brugada syndrome

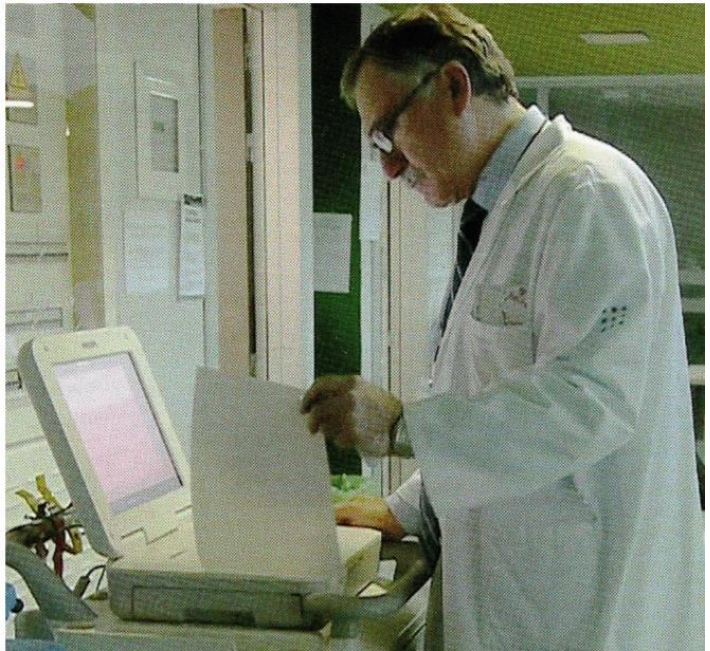
B) Brugada's phenocopy in a patient with spasm on a proximal lesion of the anterior descending (CT scan shows a "calcification" proximal to first perforator branch (S) but does not mean that the occlusion is critical

C) STEMI by proximal occlusion of the LDA that simulates a Brugada pattern (phenocopy). I do not think so.

- Criteria of left ventricular hypertrophy in the frontal plane
- Left Anterior Fascicular Block
- I can not comment on VCG because in our hospital it is not used

Regards

Miquel **Fiol-Sala,** MD, PhD nickname: “The ambassador of the Balearic Islands”
Scientific Director of the Research Institute Sanitary of Palma (IdISBa) Palma de Majorca Balearic Islands



Final comments

Final clinical diagnosis

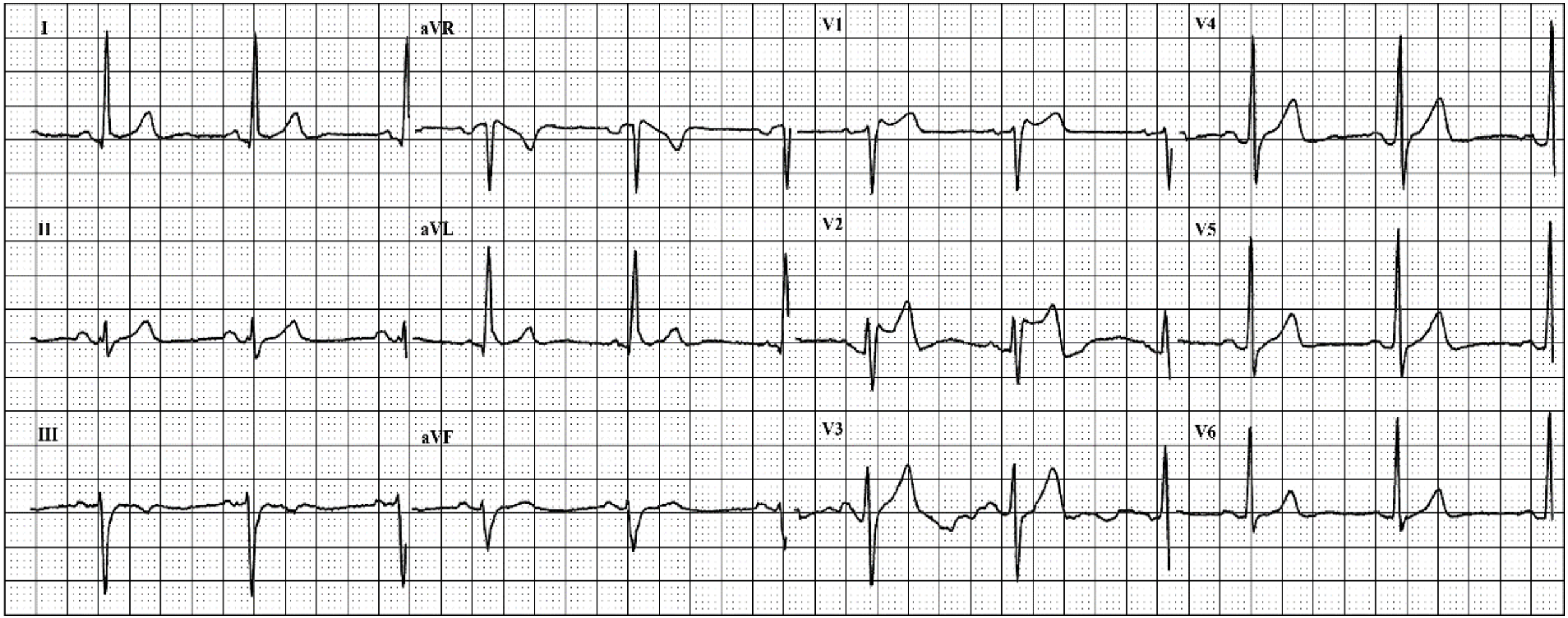
- CAD with proximal LAD artery obstruction before the first septal perforator branch.
- Brugada syndrome (strong familial background with positive SCN5A mutation in his brother and first degree cousin).

Final ECG diagnosis

- Type 1 and type 2 Brugada ECG pattern.
- Degree of LAFB: QRS axis in the frontal plane -25/-30°.
- Transient advanced or complete LAFB: QRS axis in the frontal plane -45°.
- Transient LSFB.

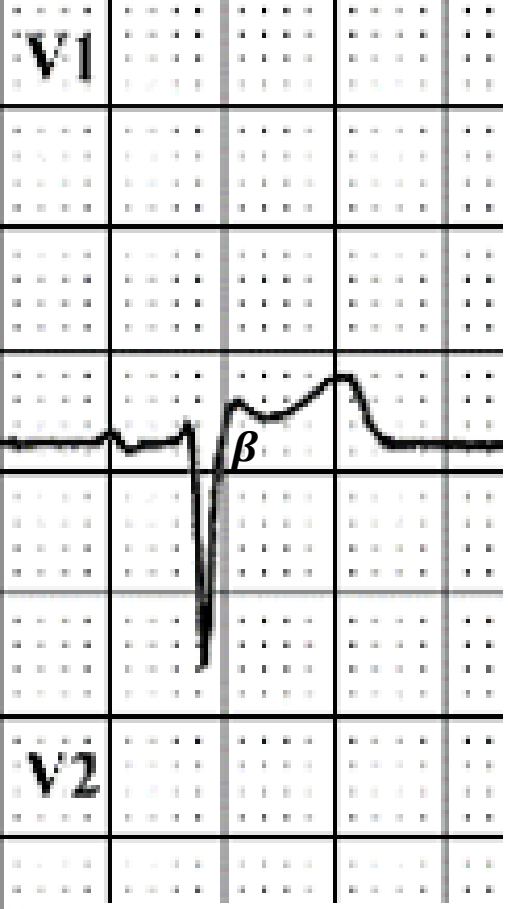
See explanation in the next slides.

Figure 1- ECG-1

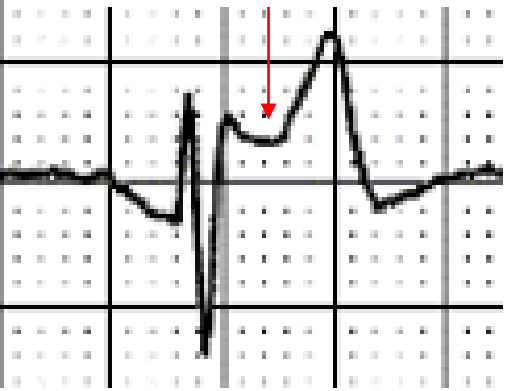


ECG-1 diagnosis: sinus rhythm, normal P-wave parameters, normal PR interval duration (148ms), prolonged QRS duration (124ms), QRS axis at -25°, QT-QTc 372-383 ms, and typical type 2 BrP: wide β-angle (angle formed by the ascending ramp of S the descending of r ' in V_1-V_2), ST segment with saddle-back appearance followed by plus-minus biphasic T wave.

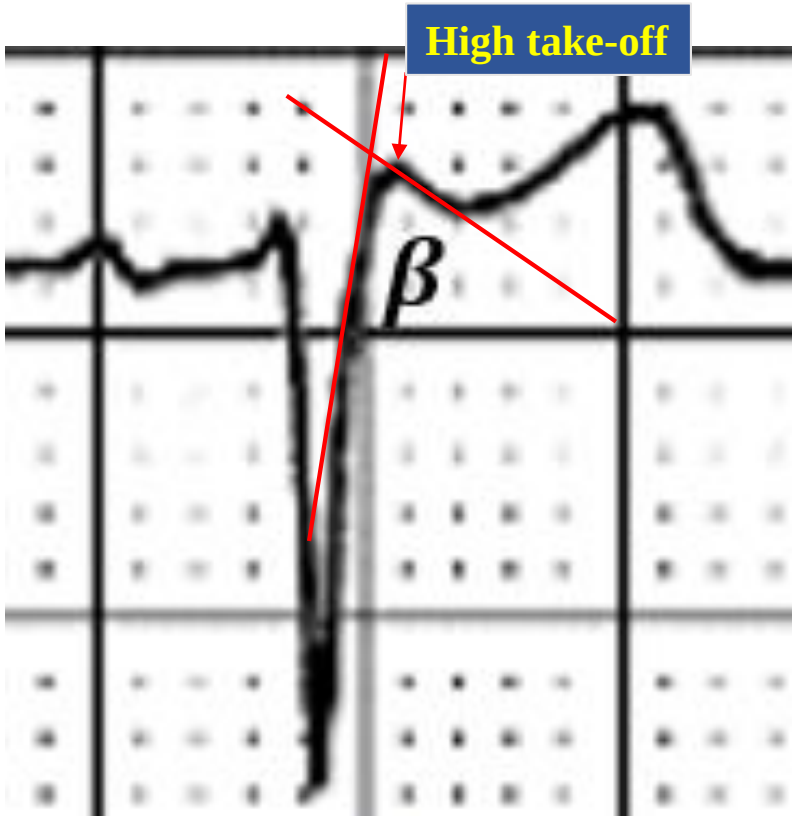
Wider β-angle related the “innocent” or ordinary incomplete right bundle branch block (IRBBB): This angle is formed by the ascending ramp of S and the descending of r' in V_1-V_2. In type 2 BrP the average value for the β-angle is 61.3 ± 25.7° in V1 for all cases, and 49.3 ± 23.1° in V2 for all cases (**Gottschalk 2016**). On the other hand, in “innocent” IRBBB it is narrow (**Chevallier 2011; Ohkubo 2011**). Additionally in type 2 Brugada pattern the high take-of is broader related ordinary IRBBB.



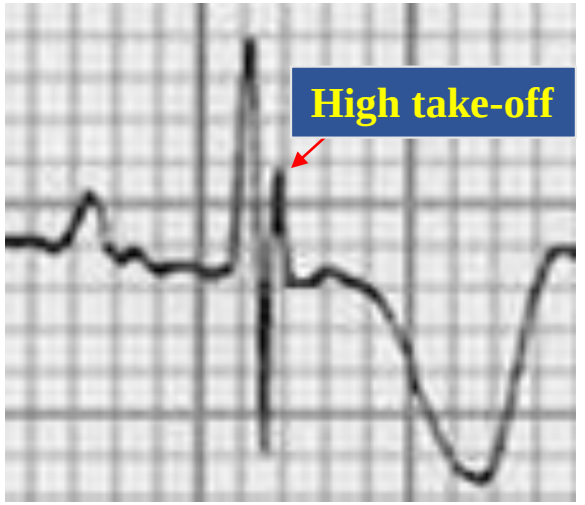
ST segment with saddleback appearance



Type 2 Brugada ECG pattern

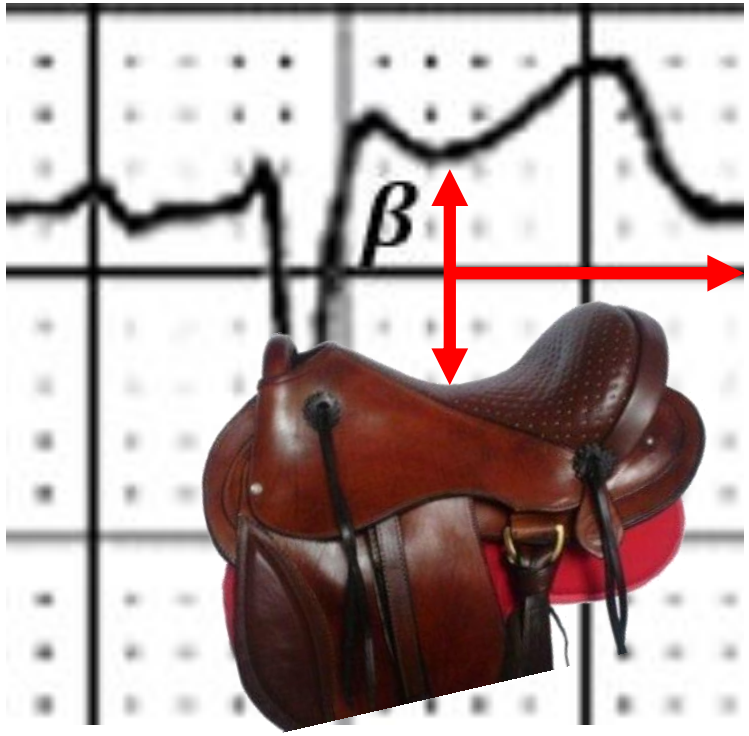


Ordinary innocent IRBBB



Type 2 Brugada pattern in V1-V2 ECG features

- High take-off that does not coincide with the J-point ≥ 2 mm.
- The descending arm of r' coincides with the beginning of ST segment.
- ST segment upslope is at least 0.5 mm.
- ST segment is followed by positive T wave in V2.
- The characteristics of the triangle formed by r' enable the different criteria to be defined that are useful for diagnosis: a) the duration of the base of the triangle formed by r' at 5 mm from the high take-off is greater than 3.5 mm, and b) the duration of QRS in type 2 Brugada syndrome is longer than in other cases with r' in V1, and there is a mismatch between V1 and V6.
- Duration of the base of the triangle at 0.5mV from r'- wave ≥160ms (4mm), duration of the base of the triangle at the isoelectric line ≥60ms (1.5mm) and β-angle ≥ 58° (**Pieritz 2016**).



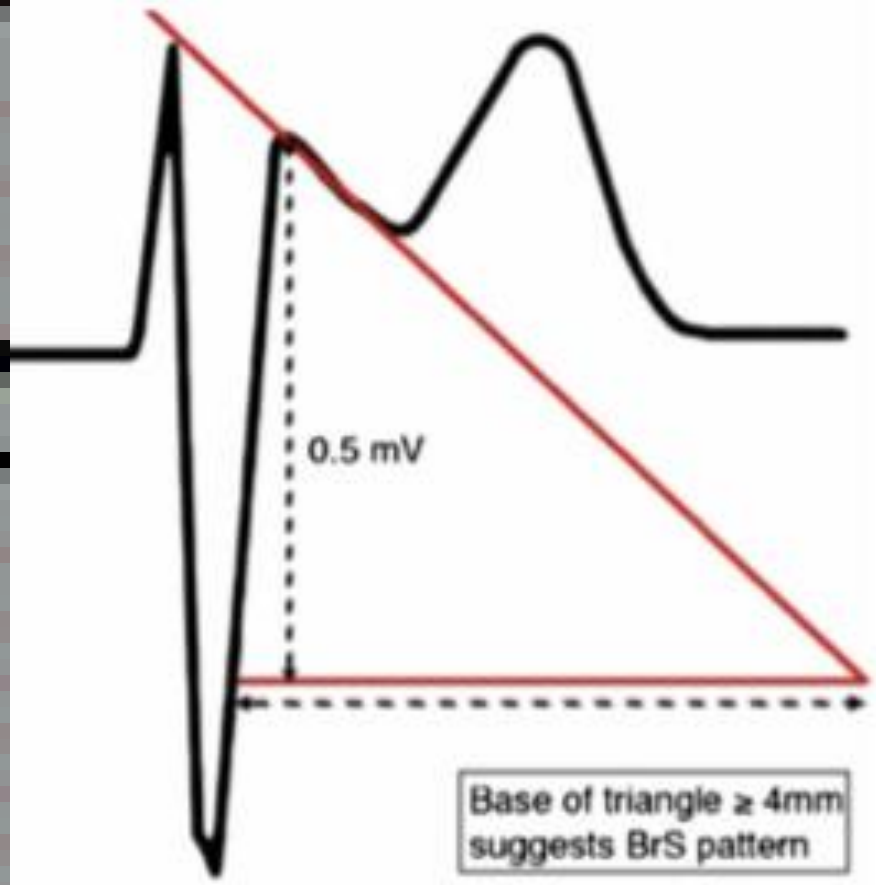
ST with saddleback appearance

High take-off

>60ms

5 mm

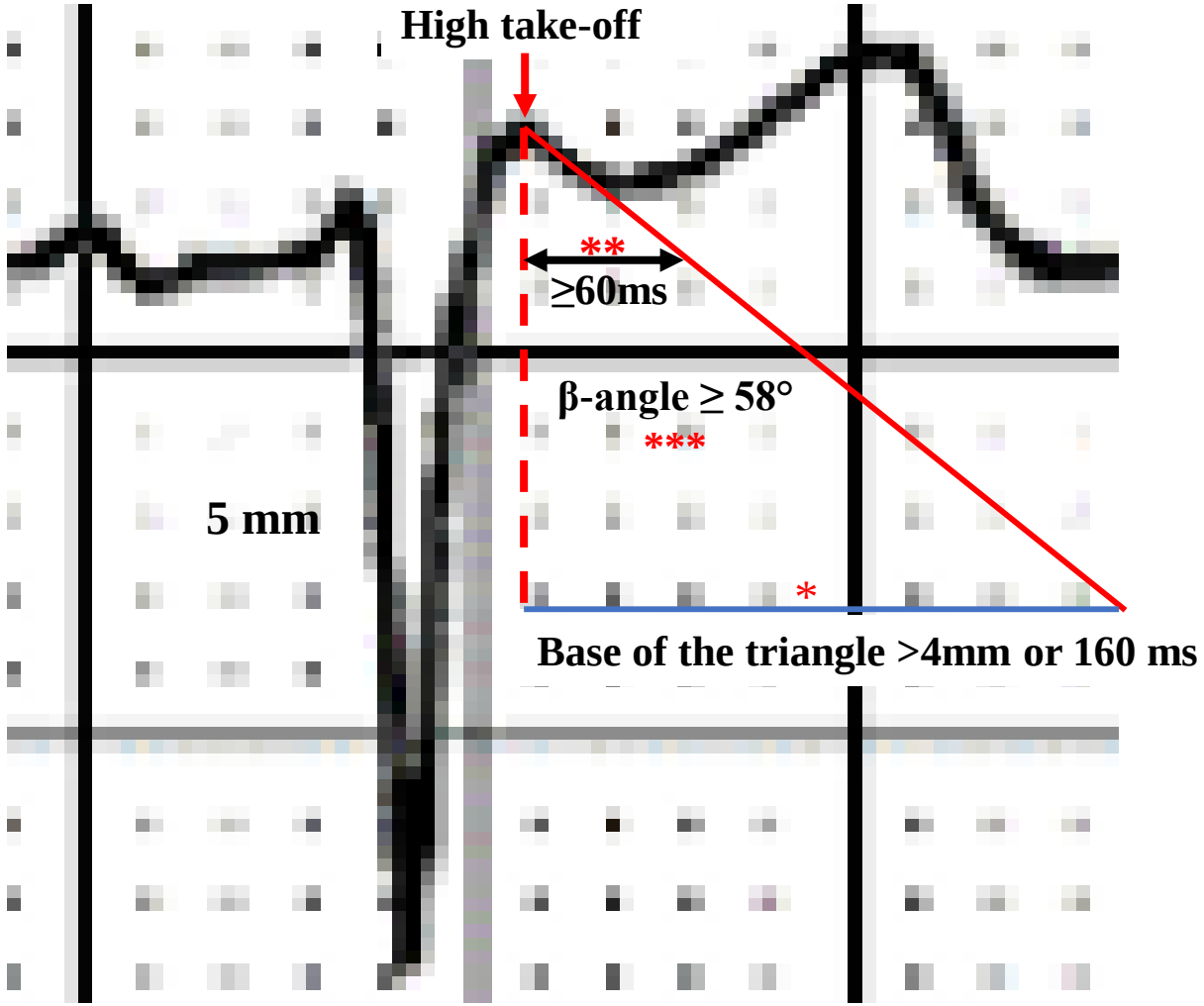
Base of the triangle > 4mm or >160 ms

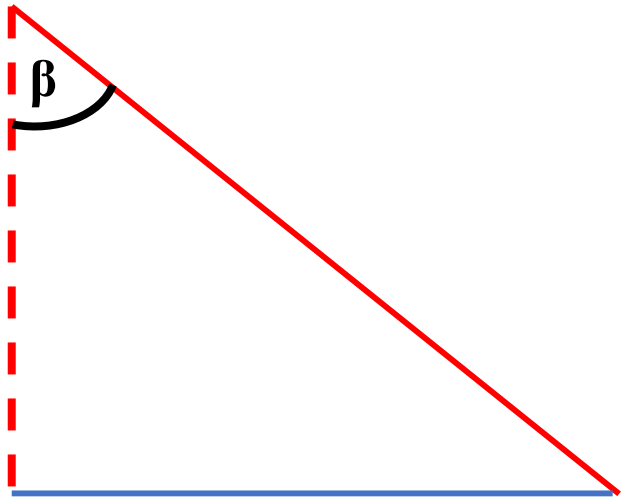


The duration of the base of the triangle formed by r' at 5 mm from the high take-off is greater than 3.5 mm, New ECG criteria based on the r' wave accurately identify rSr' patterns in V1-V2 from potential type 2 Brugada patterns in patients with purposely placed high precordial leads (**Peritz 2016**). See next slide…….

Peritz and Chung (**Peritz 2016**) used three criteria for evaluating rSr' patterns at high precordial ECG lead placement for differentiate the type 2 Brugada ECG pattern from ordinary IRBBB. The 3 criteria used where:

I. Duration of the base of the triangle at 0.5mV or 5mm from the high take-of r'- wave ≥160ms (4mm),*
II. Duration of the base of the triangle at the isoelectric line level ≥60ms (1.5mm) **
III. β-angle ≥ 58°.***

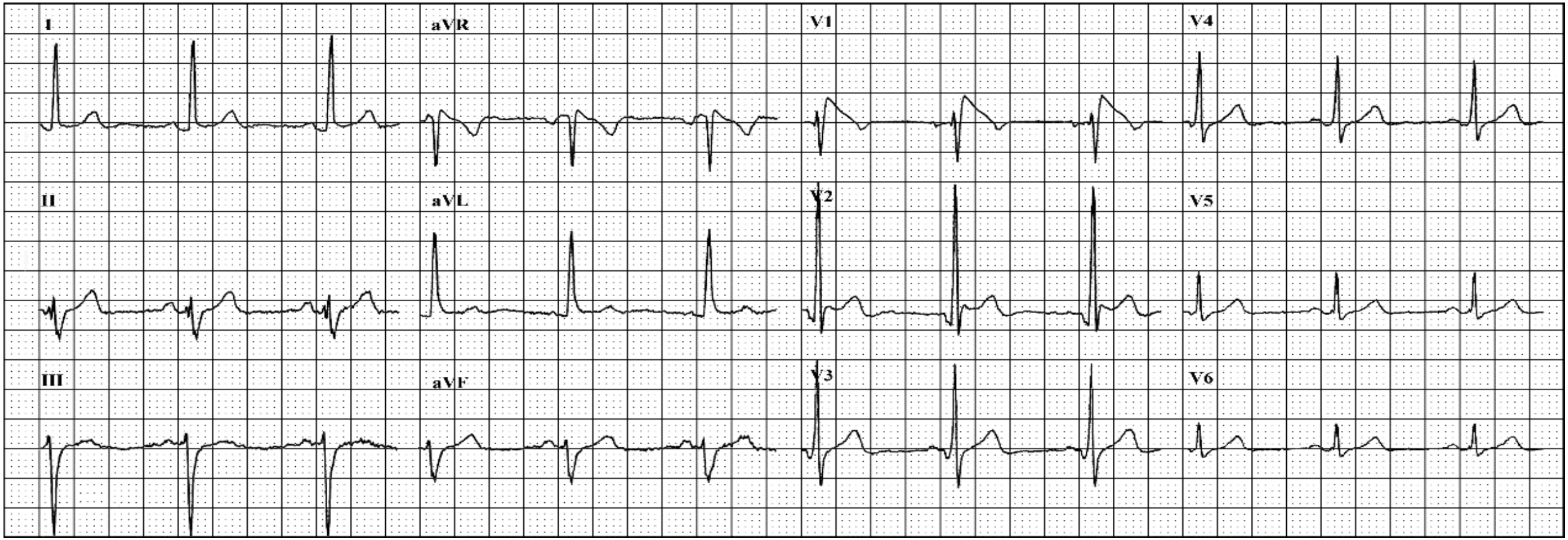




The duration of the base of the triangle at 0.5 mV or 5mm from the high take-off is the easiest to measure and may be used in clinical practice.(**Serra 2014**)

Three new ECG criteria were accurate to distinguish the Type-2 Brugada pattern from the ECG pattern with an r'-wave in healthy athletes (**Serra 2014**).

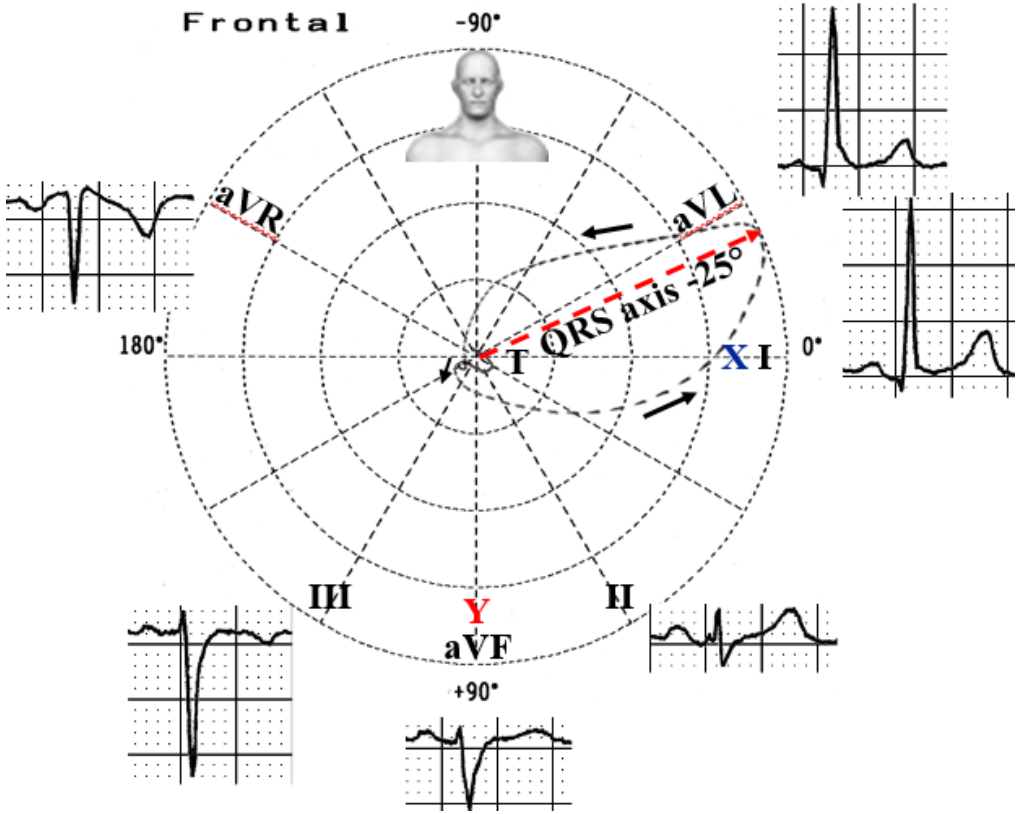
ECG-2



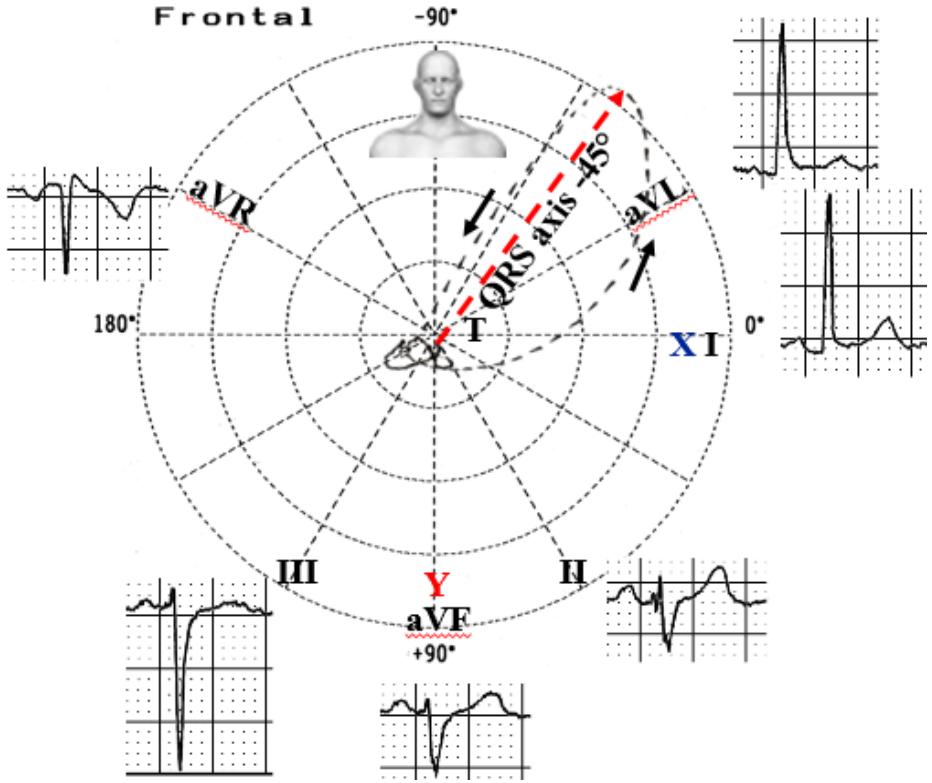
ECG-2 diagnosis: sinus rhythm, heart rate 75bpm, normal P wave, PR interval duration 144ms, QRS axis -45°, SIII> SII, absence of initial q wave in the lateral leads I, aVL, V5-V6. In the precordial leads, type 1 BrP, wide initial Q wave and prominent QRS anterior forces in V2-V3 and absence of q wave in V5-V6.

Conclusion: LAFB + LSFB (bifascicular block) + Type 1 BrP + probable electrically inactive forces in the septal wall.

ECG-1/VCG-1 correlation in the Frontal Plane



ECG-2/VCG-2 correlation in the Frontal Plane

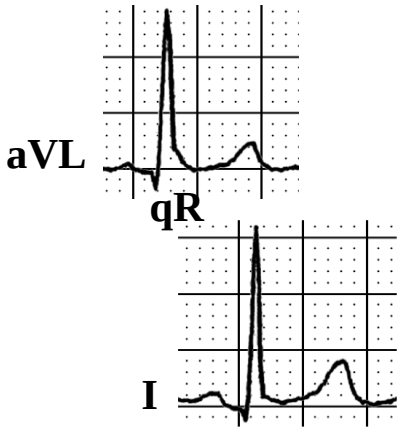


QRS axis	-25° -30° * (See comments in next slide)	-45°
QRS in I and aVL	qR	R
QRS in aVR	QS	Qr
Initial 10 to 20 vector	Directed to down and rightward	Directed to down and leftward: absence of first middle septal vector dependente of left septal fascicle
QRS rotation	Counterclockwise	Counter clock wise
Final QRS loop forces	On top left quadrant	On top right quadrante
T-wave/T-loop	Negative in III	Positive in III

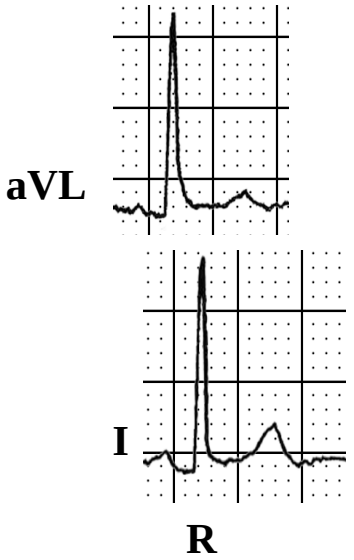
- -25° -30° or higher is considered a degree of LAFB. Earlier studies (**Watt 1965**) has indicated that an axis of was present in no more than 1.5% of patients with left ventricular hypertrophy or horizontal hearts. Rosenbaum et al (**Rosenbaum 1968; 1969a; 1969b; 1970; 1972; 1973a; 1973b**) however , felt that an axis of -45° was required for the confident diagnosis of LAFB, although realized that this was and arbitrary limit. In addition, other electrocardiographic criteria should be present in LAFB standard type. LAFB usually shifts the main forces of the QRS axis to −45°, −60°, or even to −75° (complete LAFB). Because the QRS axis may be <−45° in cases of incomplete LAFB, the degree of left axis deviation required for the accurate diagnosis of complete LAFB is −45°. All these changes occur with a QRS that widens no more than 0.02 seconds in pure and uncomplicated LAFB (**Elizari 2007**).

1. A small Q wave (not wider than 2ms) an a tall R wave in leads I and aVL. The Q wave in lead I is no longer a prerequisite for the diagnosis of LAFB.As Rosenbaum had already observed. It is often absent, for instance of vertical hearts, when some degree of incomplete left bundle branch block is present, or in cases of left septal fascicular block consequence of loss of the first middle septal vector of ventricular activation such as in the present case

ECG-1

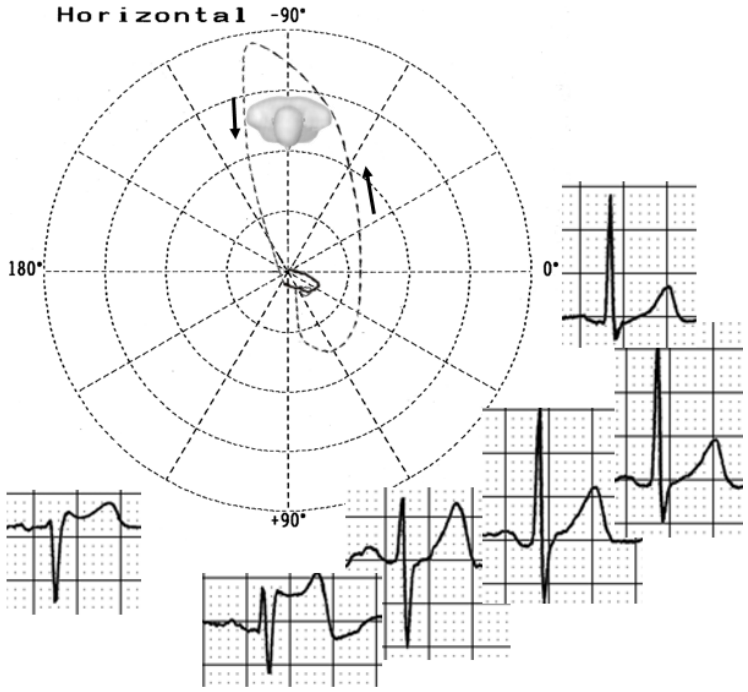


ECG-2

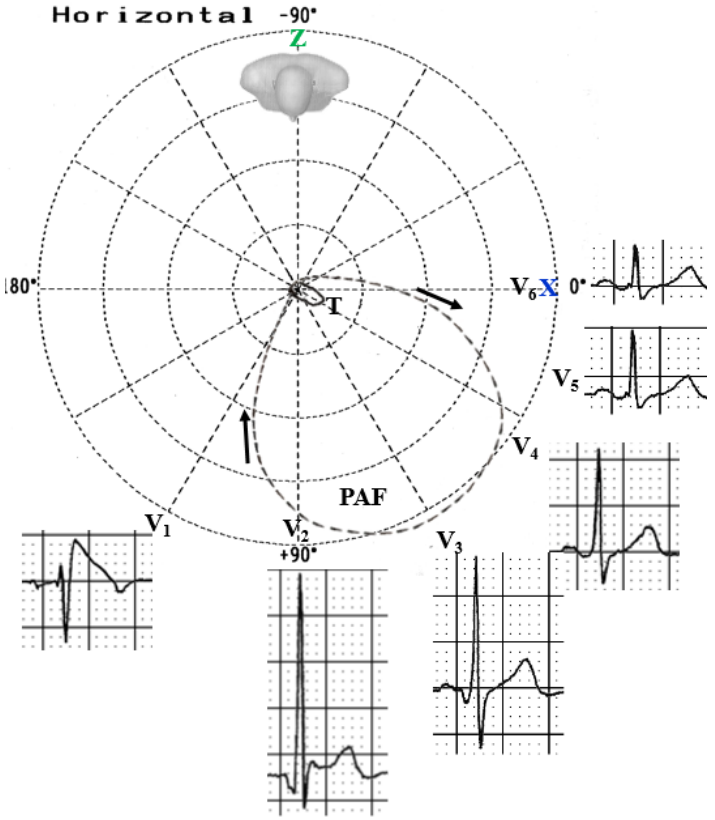


2. A small R wave and deep S wave in leads II, III and aVF with SIII > SII.
3. A total QRS duration of less than 120ms.

ECG-1/VCG-1 correlation in the HP

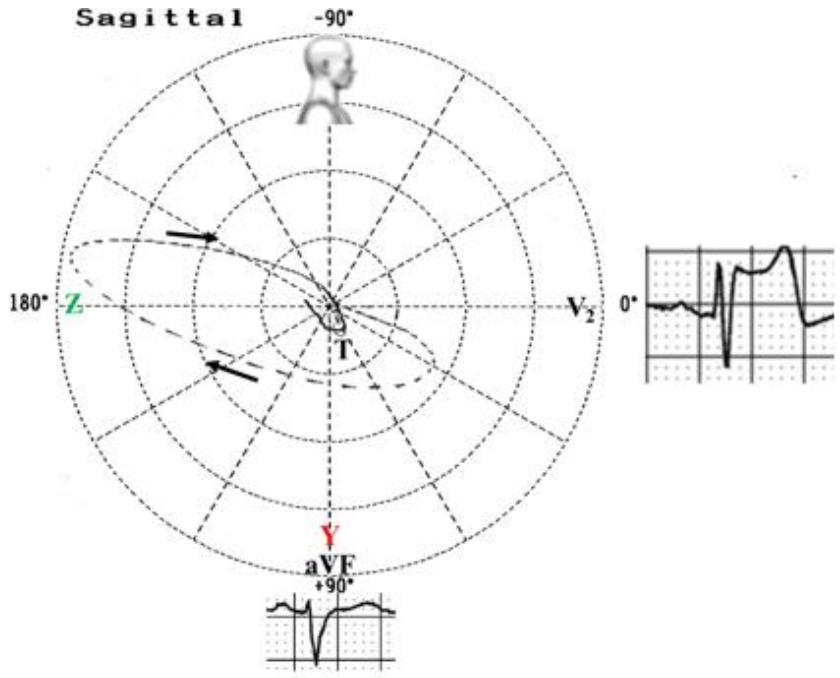


ECG-2/VCG-2 correlation in the HP

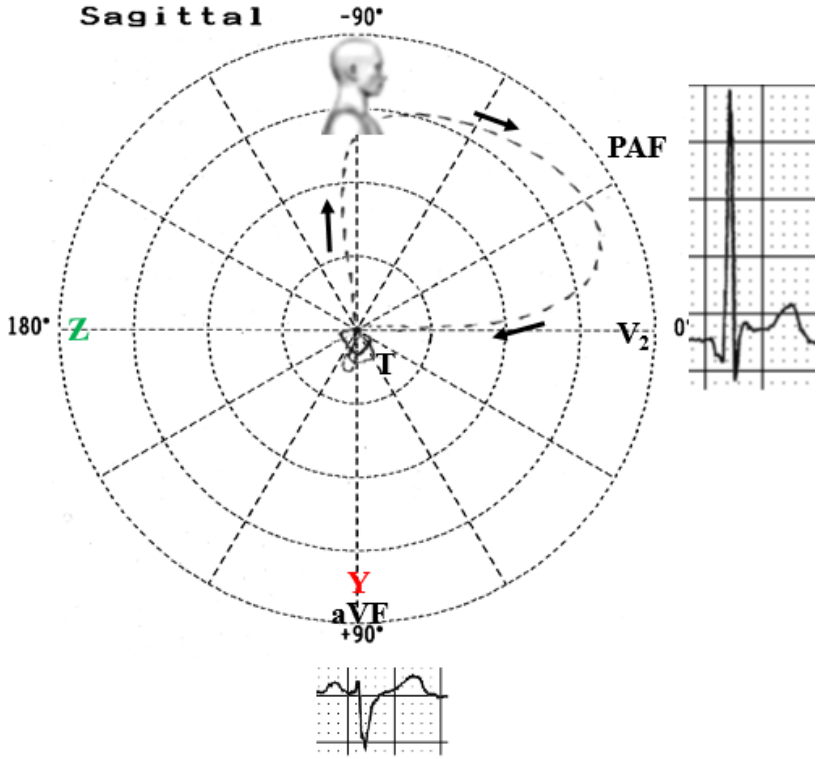


Repolarization eleads V_1-V_2	Type 2 Brugada ECG pattern	Type 1 Brugada ECG pattern in V_1 and Type 2 in V_2
QRS in V_1-V_2	RSr´	qRs
R-voltage in V_2	5 mm	21 mm: prominent anterior QRS forces (PAF)
QRS loop	Predominantly located in the posterior quadrants	Predominantly located in the anterior left quadrante: PAF
Initial 10-20 ms QRS loop	Directed to front	Directed to back and leftward
Final QRS loop	With RECD in the posterior right quadrant	With RECD in the anterior right quadrante
T-loop	Directed to front and leftward (+30°)	Negative hemifield of V_1

ECG-1/VCG-1 correlation in the RSP



ECG-2/VCG-2 correlation in the RSP

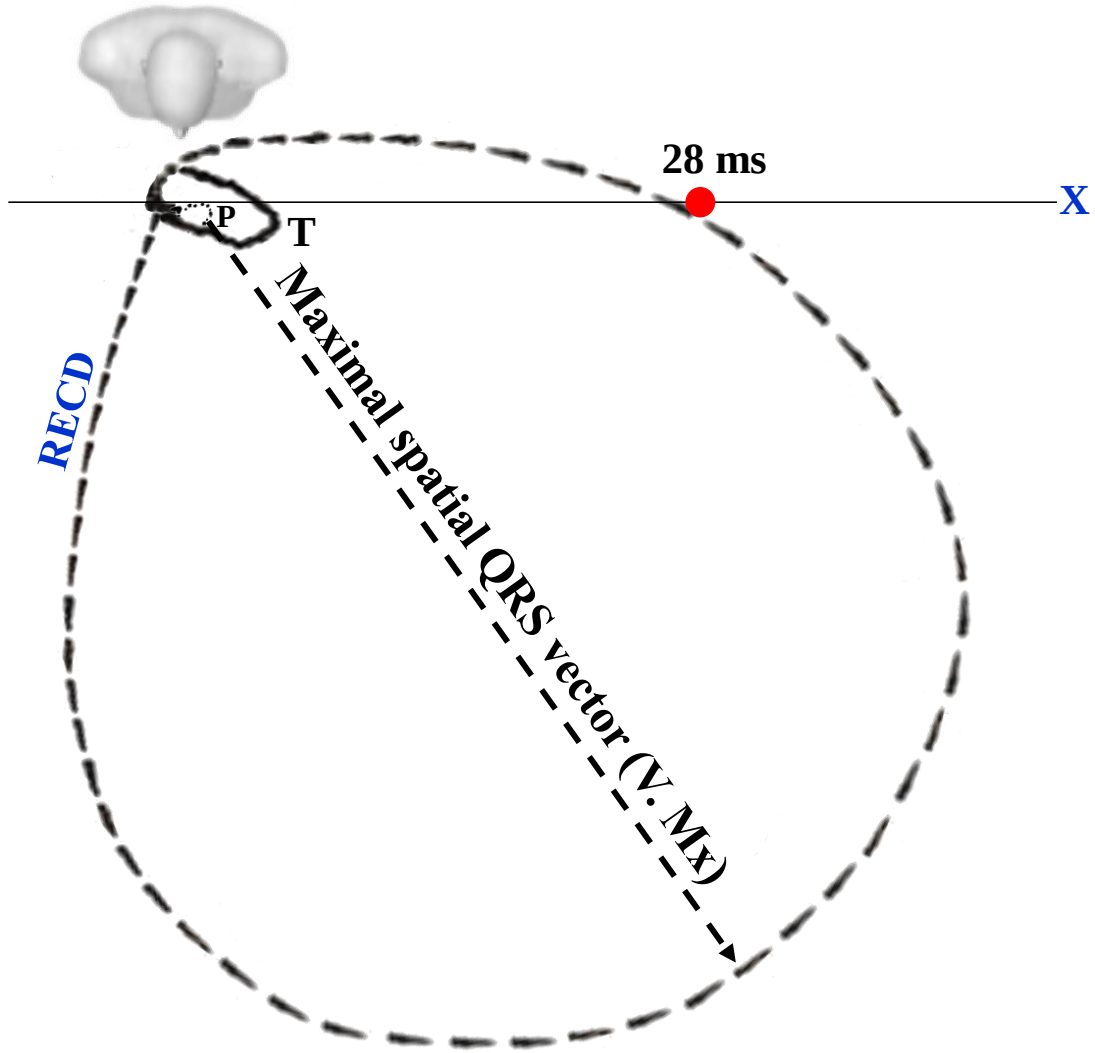


Repolarization in V_2	Type 2 Brugada ECG pattern	Type 2 Brugada ECG pattern in V_1 and Type 2 in V_2
R-voltage in V_2	5 mm	21 mm: prominent anterior QRS forces (PAF)
QRS in aVF	rS	rS
QRS loop	Predominantly located in the posterior quadrants	Predominantly located in the anterosuperior quadrant: PAF
Initial 10-20 ms QRS loop	Directed to front and downward	Directed to back and upward
Final QRS loop	With ECD in the posterosuperior quadrant	With ECD in the Z orthogonal lead
T-loop	Directed to down and forward (+60°)	Directed to down and forward (+80°)

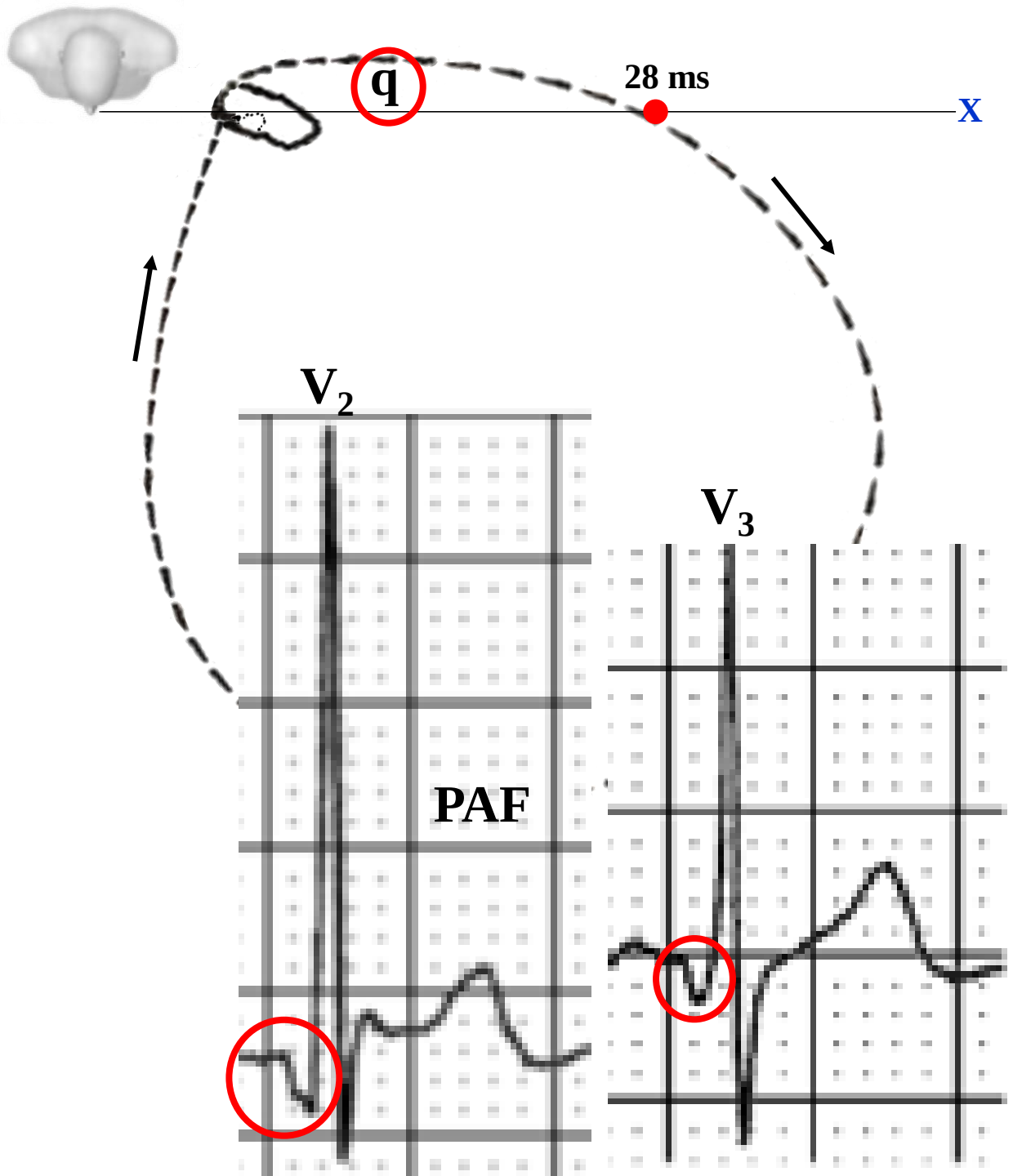
VCG criteria for Left Septal Fascicular Block

All criteria are described in the horizontal plane:

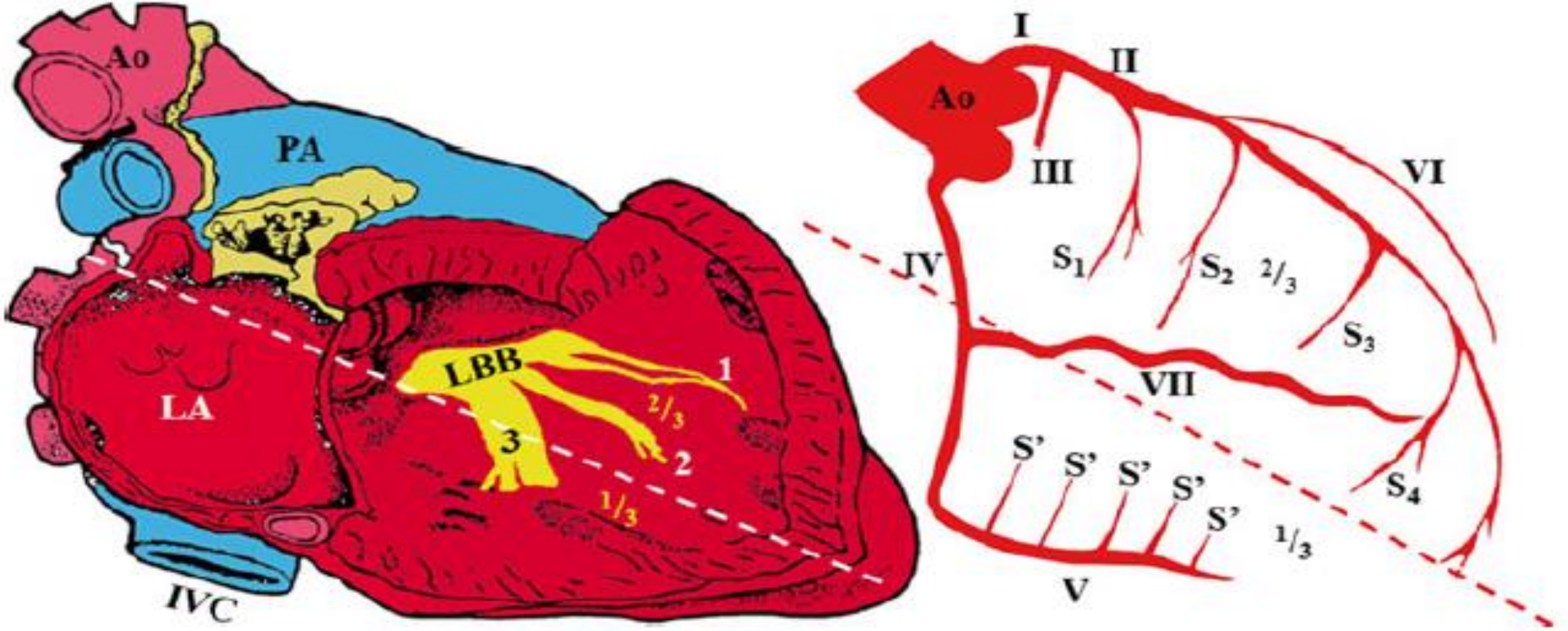
1. QRS loop in the HP with an area prominently located in the left anterior quadrant: ≥2/3 of the loop area facing the orthogonal X lead (0 to ±180°).
2. Absence of normal convexity to the right of the initial 20 ms of the QRS loop.
3. Discrete dextro or rightward orientation with moderate delay of the vector from 20-30 ms.
4. Anterior location of the 40-50 ms vector.
5. Posterior location with reduced magnitude of the vector of 60-70 ms.
6. Maximal vector of the QRS loop located to the right of +30°.
7. Intermittent or transient anterior displacement of the QRS loop.
8. The QRS loop rotation may be counterclockwise rotation: incomplete LSFB; or clockwise rotation: advanced or complete LSFB; or in association of CLBBB, LAFB or LPFB.



Maximal spatial QRS vector or V. Mx: this is defined as the vector that stretches from the 0 point up to the faster point of the QRS loop. In this case it is located +45°. In normal adults it is located in average at -15°(range +40 to -50°). In pediatric vectorcardiography is called left maximum spatial voltage (LMSV). In this case, the sequence of ventricular activation begins at the base of the posteromedial papillary muscle of the mitral valve (PMPM) dependent of the left posterior fascicle (LPF) because left anterior fascicle and left septal fascicle are blocked. The initial predominance of the vector dependent of LPF causes initial 10-20 ms directed to back and leftward showing qR in the right precordial leads.



The block of the anterosuperior (LAFB) and medio-septal fascicles (LSFB) causes only the posterolateral fascicle (LPF) to activate the posteromedial papillary muscle of the mitral valve, where it ends. Consequently, it will originate a first vector directed backward. Additionally, in this case the initial Q waves may also be caused by electrically inactive area in the anteroseptal wall. Remember that electrical and biological death are not the same thing.



Schematic diagram of blood supply to the cardiac conduction system. Left Main Coronary Artery (I); Left Anterior Descending Artery (II); Left Circumflex (III); Right Coronary Artery (IV); Posterior Descending Artery (V); First Diagonal (VI); Acute Marginal (VII); First Septal Perforator branch (S_1); Second Septal Perforator (S_2); Third Septal Perforator (S_3); Fourth Septal Perforator (S_4); $^2/_3$ of IVS (2/3); Posterior Septal Perforating branches (S'); $^1/_3$ of IVS (1/3); Left Anterior Fascicle (1); Left Bundle Branch (LBB); Left Septal Fascicle (2); Left Posterior Fascicle (3); Pulmonary artery (PA); Inferior vena cava (IVC); Left atrium (LA); Aorta (Ao).

Discussion

Interventricular septum is the most densely vascularized portion of the heart containing important elements of the cardiac conduction system and providing mechanical support for both right and left ventricular function. This structure receives blood from both the LAD as well as the posterior descending artery (PDA). This latter artery runs in the posterior interventricular sulcus to the apex where it meets with the LAD (**Levin 1988**). In ≈ 70% of cases PDA is a branch of the right coronary artery (RCA) (right dominance) and in 10% is a branch of the left circumflex coronary artery (LCx) (left dominance) which itself is a branch of the left coronary artery. It can also be supplied by an anastomosis of the RCA and LCx in≈ 20% of cases (co-dominance).Anterior septal perforator branches-arteries originate from the LAD and irrigate $^{2}/_{3}$ of the upper portion of the interventricular septum (IVS). Through this area runs the left septal fascicle (LSF) of the left bundle branch (LBB). Septal perforator branches originate in the LAD with an angle of nearly 90°. In the IVS, there are two areas with different blood supplies: The upper portion of IVS which includes the AV node, the atrioventricular (AV) bundle and the proximal segments of the two main bundle branches, that are supplied by a branch of the RCA. The first septal perforator branch (S_1) also supplies a significant portion of the conduction system, including the His bundle, right bundle branch and the AV node in 50 % of patients (**Ozdemir 2001**). The lower area, which comprises the greater mass of the septum, including most of the two main bundle branches, left fascicles, and the Purkinje arborization of the IVS, supplied mainly by the anterior septal perforating branches. There are 4 to 13 anterior septal perforator branches with an average of 8 branches (**Topaz1992**) (S_1 to S_8). The size and anatomy of septal perforator branches vary widely. The length of these vessels ranges from 40 to 80 mm and become shorter near the apex (**James1958 a**). The first septal perforator branch (S_1) is between 4 to 6 cm, and usually is the largest and the longest one. Its external diameter at the origin ranges from 1.0 mm to 2.35 mm. (**Possatti 20015**) Most commonly the S_1 originates close to the takeoff of the first diagonal branch. Less frequently the S_1 might be a short artery and there are two or three major septal arteries comparable in size. (**Ozdemir2001**). Rarely, septal perforators arise from the diagonal branch, the RCA or the LMCA. (**Takeguchi 2012**)

Blood Supply to the left fascicles of the left bundle branch

Left anterior fascicle (LAF) in 50 % of cases, the blood supply to the LAF of the LBB originated not only from the anterior septal branch of the LAD, but also from the atrioventricular (AV) nodal artery, a branch of the RCA in 90 % of the cases and of the LCX in 10 % (**Frink 1973**). Thus, anatomic data support the observation that occlusion of the proximal segment of the LAD is not a prerequisite for the occurrence of LAFB. The appearance of LAFB during AMI is not a sign of a coexistent significant stenosis of the LAD or of more severe or extensive coronary artery disease (CAD). In these patients, other mechanisms such as the degree of the coronary collateral circulation may play a role in the occurrence of this conduction disturbance and supports experimental and clinical reports that LAFB may be due to lesions involving the His bundle by means of a longitudinal dissociation of this structure (**Bosch1985**).

Left posterior fascicle (LPF) The broad nature of the LPI, its protected location in the left ventricular inflow tract as well as its dual blood supply (**James 1965.b**) makes isolated left posterior fascicular block (LPFB) very rare (**Rokey 1984**). The posteromedial papillary muscle where LPF ends is supplied by those arteries that terminate on the diaphragmatic surface of the LV, and most commonly by a junction of terminal branches of the LCX and of the RCA. When the LCX supplies nearly all the diaphragmatic surface of the LV (10 % of human hearts), its branches provide the entire blood supply for the posteromedial papillary muscle. In 10 % of cases, the LPF is irrigated by LAD only, in 40 % of cases by LAD and RCA and in 50 % of cases by RCA only.

Left septal fascicle (LSF) It is irrigated exclusively by the septal perforator arteries from the LAD. Septal perforator branches from the LAD supply the upper $^{2}/_{3}$ superior portion of IVS at this site. Most of the blood supply to the IVS is provided by the LAD. Branches into the septum from PDA rarely penetrate more than 10 mm from the epicardium (slightly more than the normal thickness of the free wall of the left ventricle), so that for practical purposes one may consider the entire blood supply of the IVS to be derived from anterior septal perforator branches of the LAD. Critical lesions of the LAD before the S_1 constitute the main cause of LSFB in the first World. It is a major determinant of high R wave voltage during acute myocardial ischemia. The LSFB may be exercise-induced, transient or intermittent, and may be the cause of large anterior precordial R waves (**Pérez-Riera a;b;c**). The appearance of LSFB in critical LAD lesions speaks in favor of the proximal lesion, and therefore of a worse prognosis. Intermittent or transient LSFB, secondary to critical obstruction of LAD, was observed during exercise testing (**Uchida 2006**), intermittently in acute coronary syndrome scenarios including so called Wellens' syndrome (**Riera 2008**).

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