

Caro Andres,

Aqui envio um real desafio eletrocardiográfico.

Este sim é estranho.

Quero ver se vocês acertam o diagnóstico. Este é para gente grande mesmo.

FAS, masculino, 53 anos. Em investigação de dor torácica atípica foi realizado a ECG que mostrou alteração (sic) em 2004. Foi então submetido a teste ergométrico que foi dado como alterado (sic).

Medicado na ocasião com enalapril 10mg/dia. Continuou com dor no peito e novo teste ergométrico foi realizado aqui em janeiro de 2010 (vide anexo).

Abcs,

Dear Andres,

In attachment I'm sending a real electrocardiographic challenge.

This is truly strange.

I want to see if you hit the diagnosis. This is for colleagues really grown-up.

FAS, men's, 53 years. In investigation of atypical thoracic pain was performed ECG who showed alteration (?) in 2004. It was then submitted at Treadmill Exercise Test that was given as alter (?).

In that occasion was medicated with enalapril 10mg/dia. He continued with pain in the chest and new exercise test was performed in our institution in January 2010 (see in attachment).

Sincerely,

Professor Dr. Paulo Jorge Moffa

Chefe do setor de métodos gráficos do InCor

Professor Associado da Faculdade de Medicina da Universidade de São Paulo (FMUSP) Diretor Técnico de Saúde do Serviço de Eletrocardiologia do Instituto do Coração (InCor) – Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo (HC-FMUSP) FMUSP. Av. Dr. Eneas C. Aguiar, 44 – CEP 05403-000 – São Paulo, SP –E-mail:

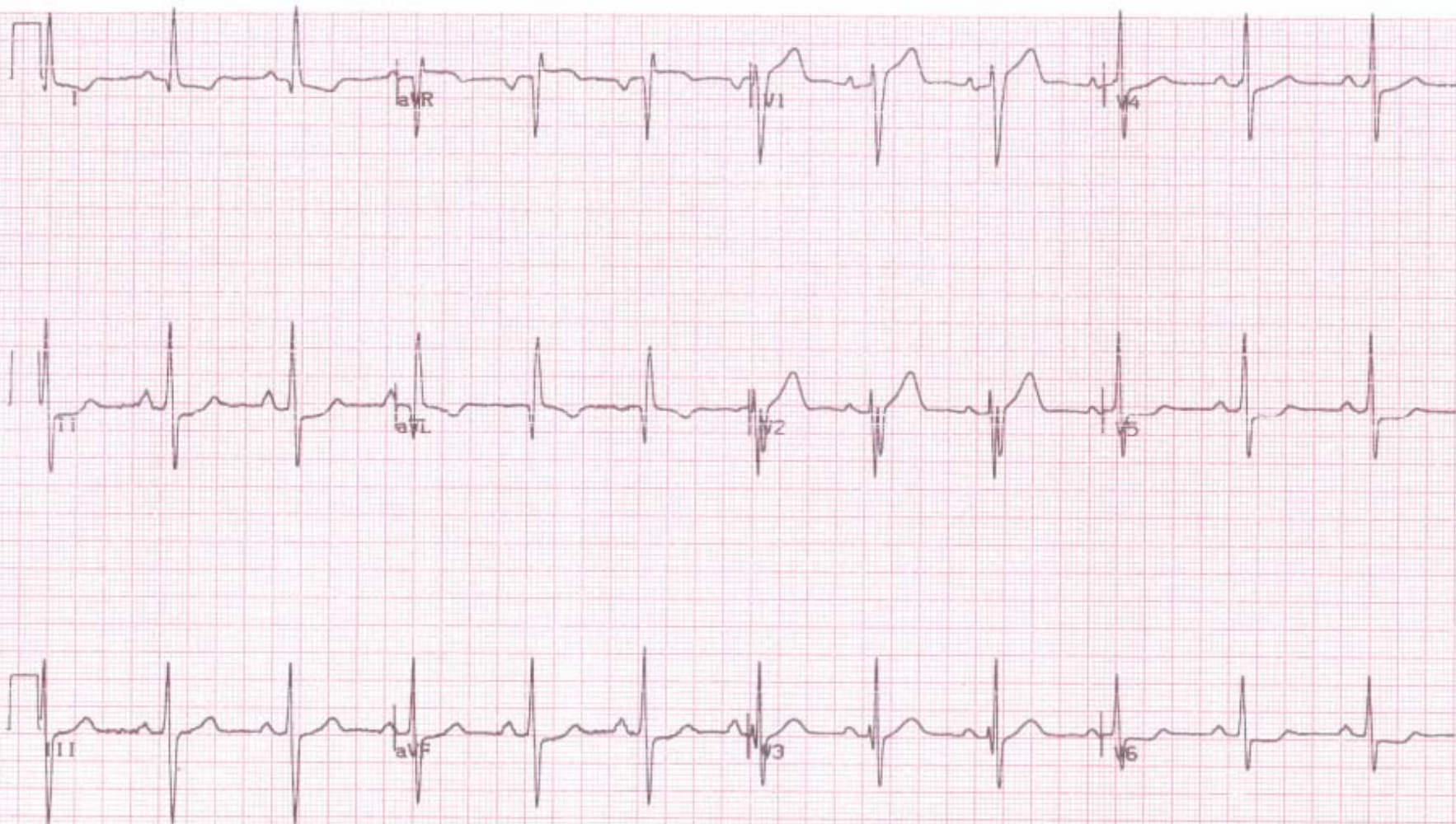
moff@incor.usp.br

10mm/mV
40Hz

HR: 71bpm
BP: 130/90

Speed: 0.0mph
Grade: 0.0x

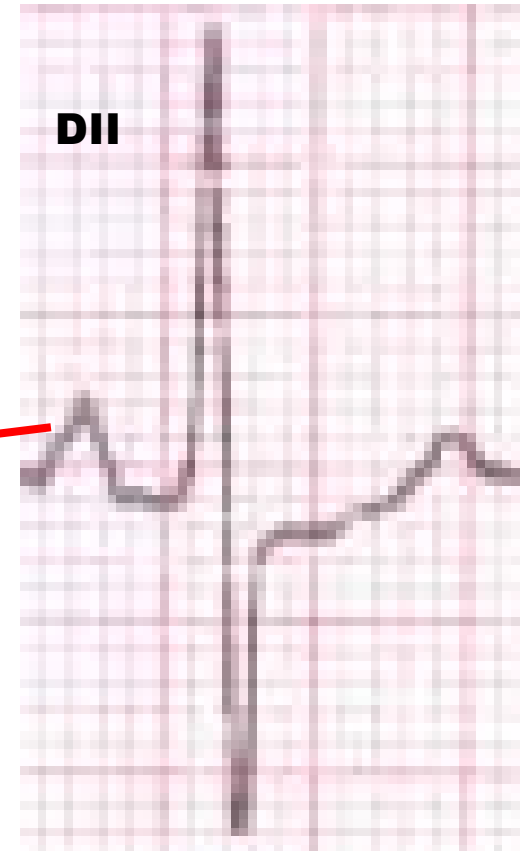
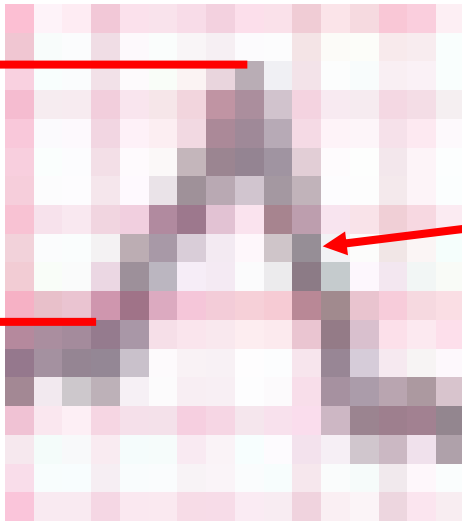
Lead	ST(mm)	Lead	ST(mm)
I	-0.6	V1	3.7
II	-0.8	V2	3.5
III	0.0	V3	1.0
aVR	0.8	V4	-0.2
aVL	-0.2	V5	-0.8
aVF	-0.4	V6	-0.7



2 A- H- S+ F+ 60 Sf23 H442 #20479 CASE 006A

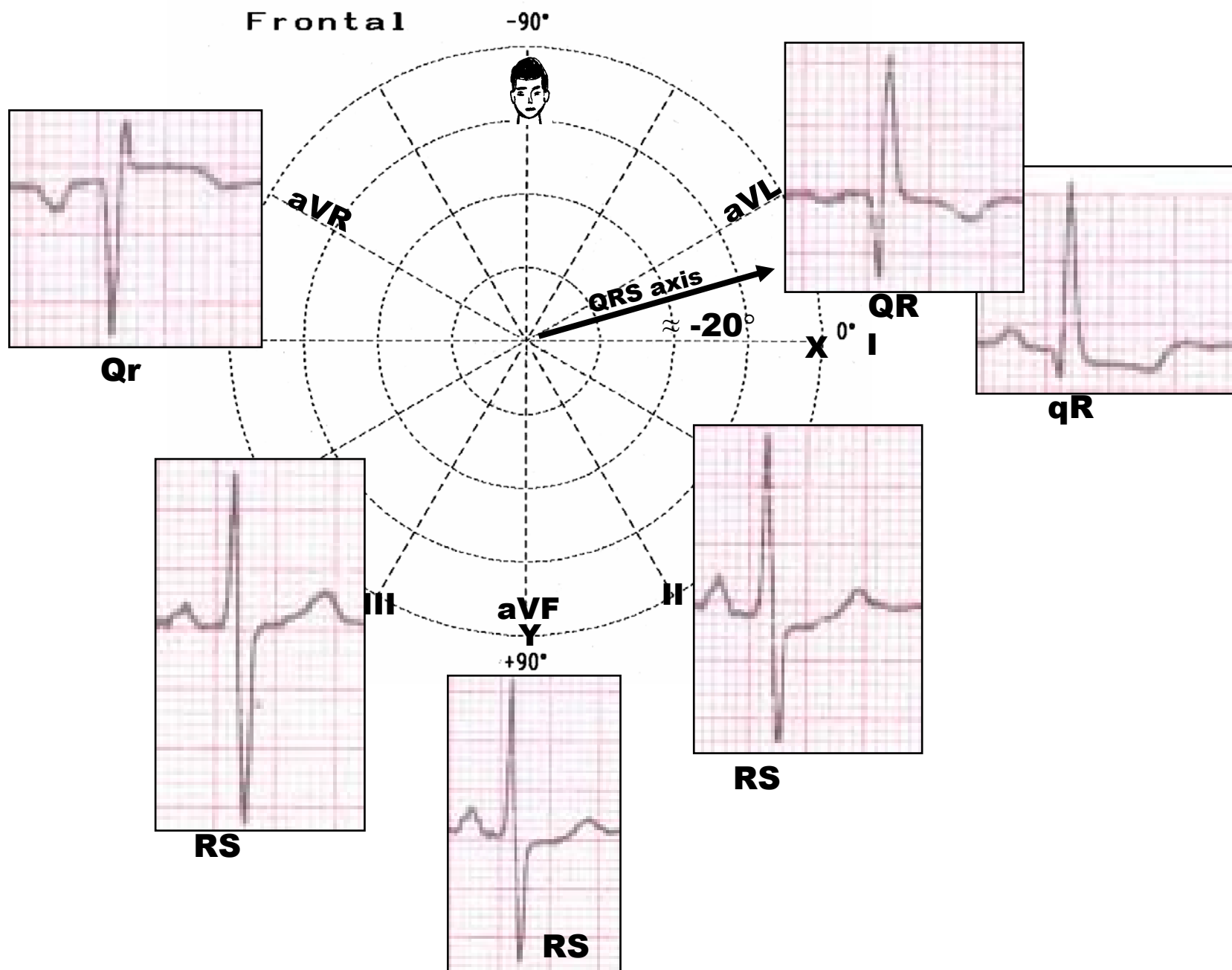
P WAVE SHAPE AND VOLTAGE

P VOLTAGE = $\geq 2,5\text{mm}$



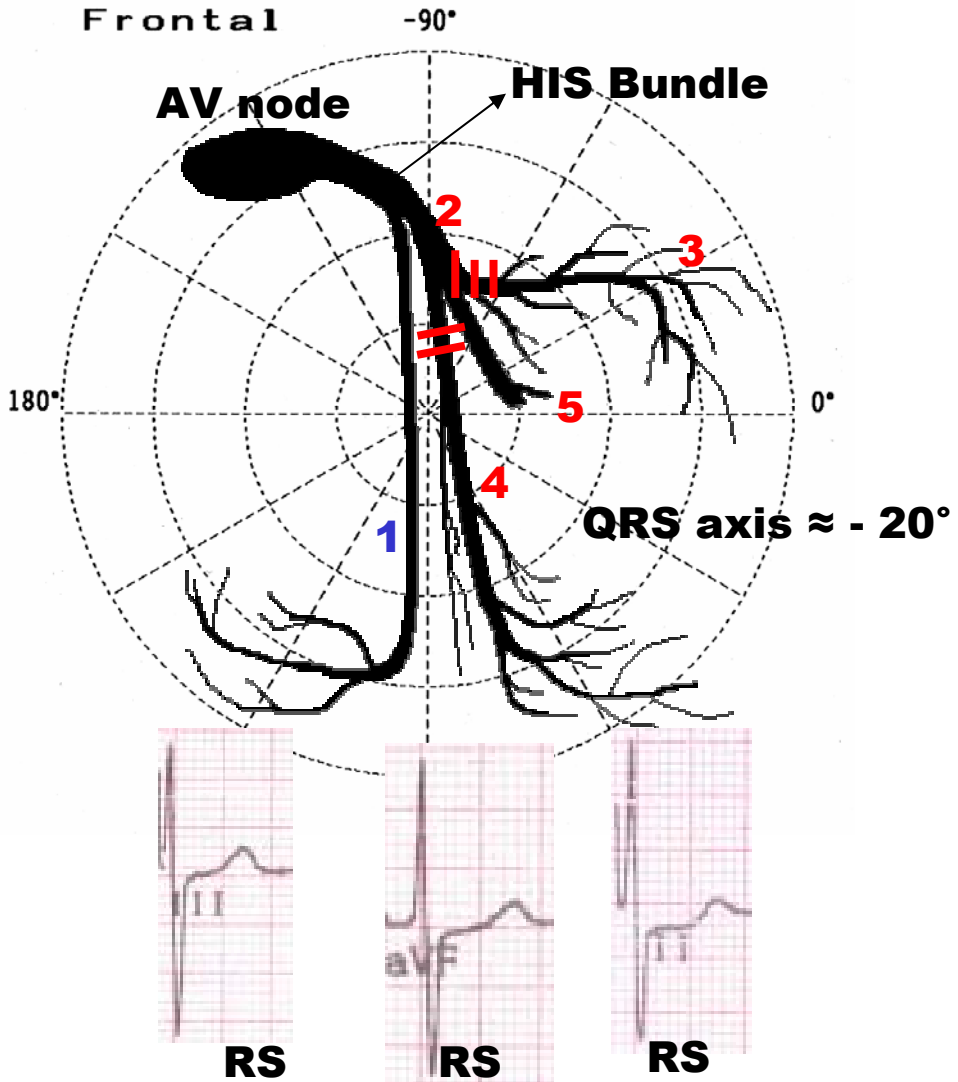
PROPER MEASUREMENT OF VOLTAGE OF P WAVE



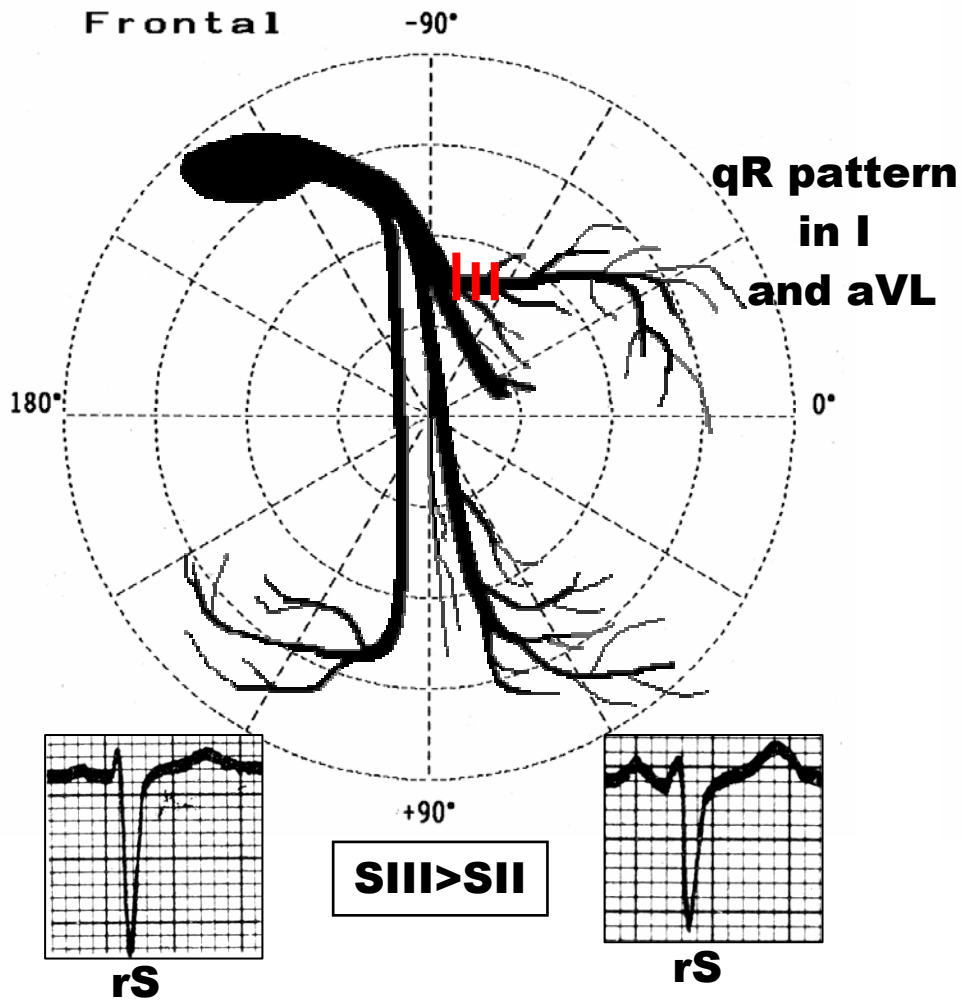


LAF larger blockade degree. IIII
LPF smaller blockade degree. II

- 1: Right Bundle Branch (RBB)**
- 2: Left Bundle Branch (LBB)**
- 3: Left Anterior Fascicle (LAF)**
- 4: Left Posterior Fascicle (LPF)**
- 5: Left Septal Fascicle (LSF)**

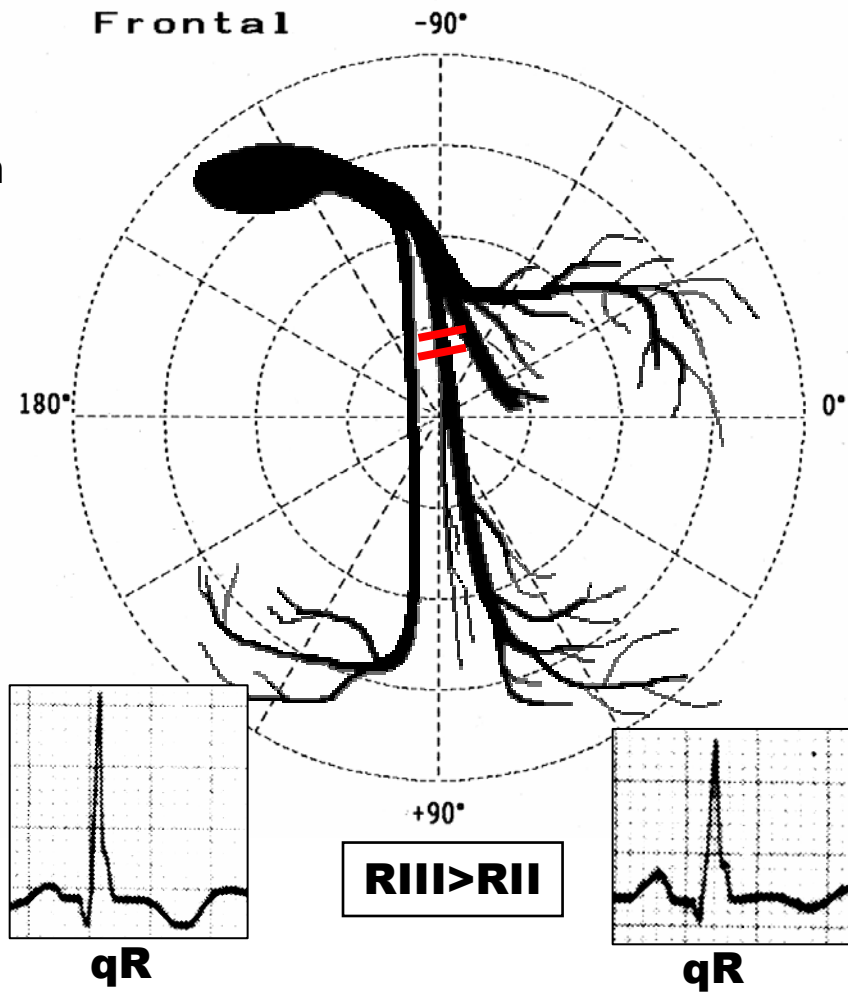


Only LAFB



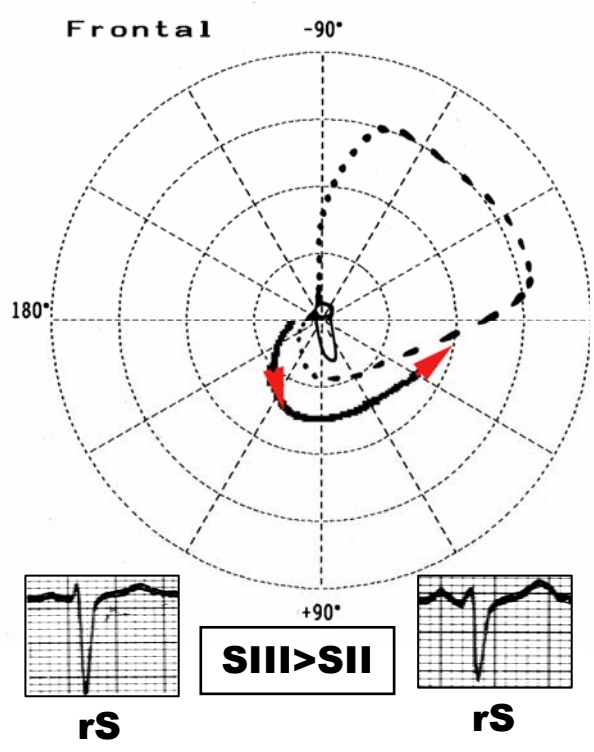
Extreme shift of QRS axis in the left superior quadrant (beyond 30° up to - 90°). Some authors accept between - 45° and - 90°

Only LPFB

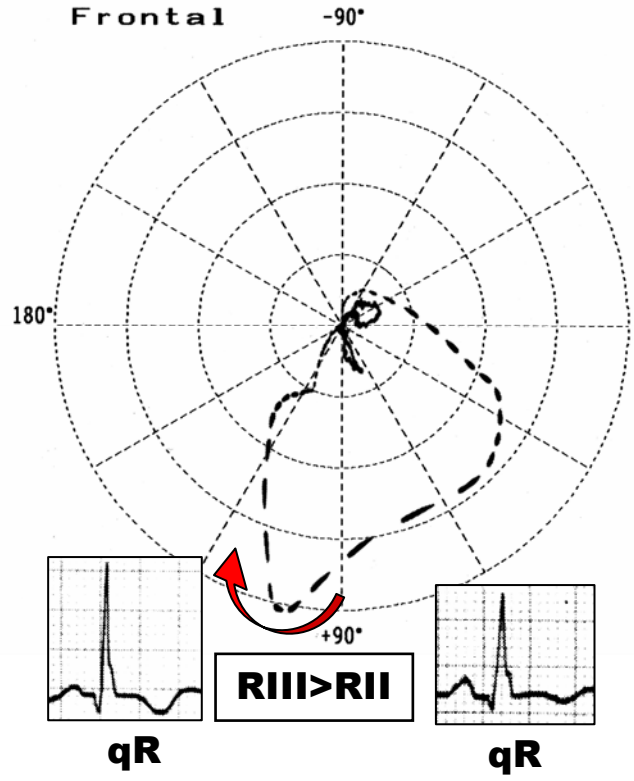


Maximal vector near +110° (+80° to +140°)

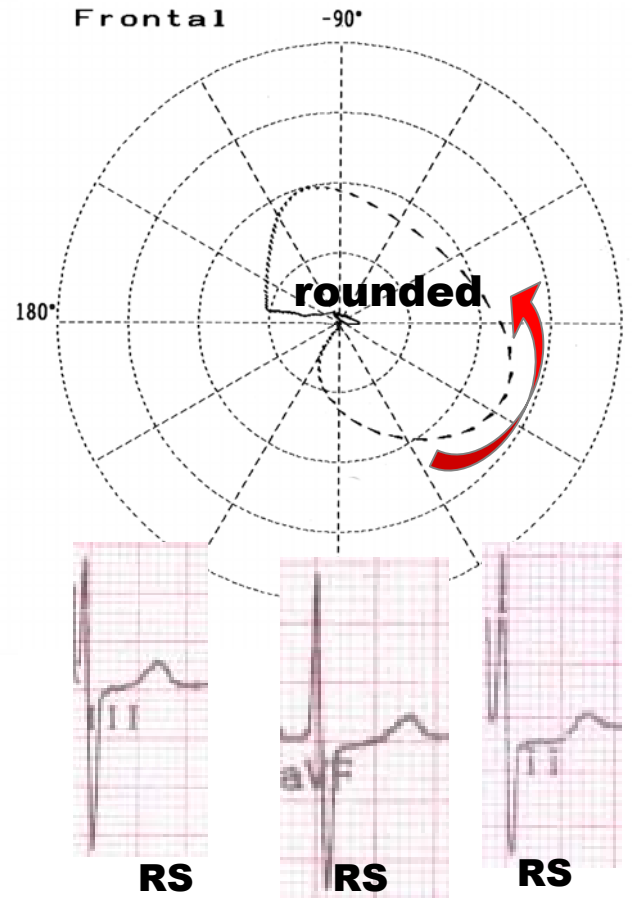
LAFB



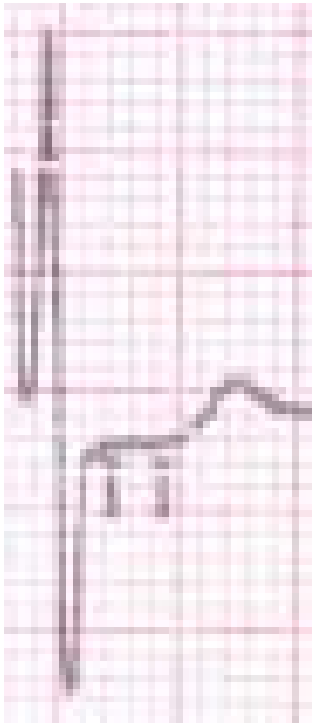
LPFB



**LAF larger blockade degree.
LPF smaller blockade degree.**



RS PATTERN IN INFERIOR LEADS



RS

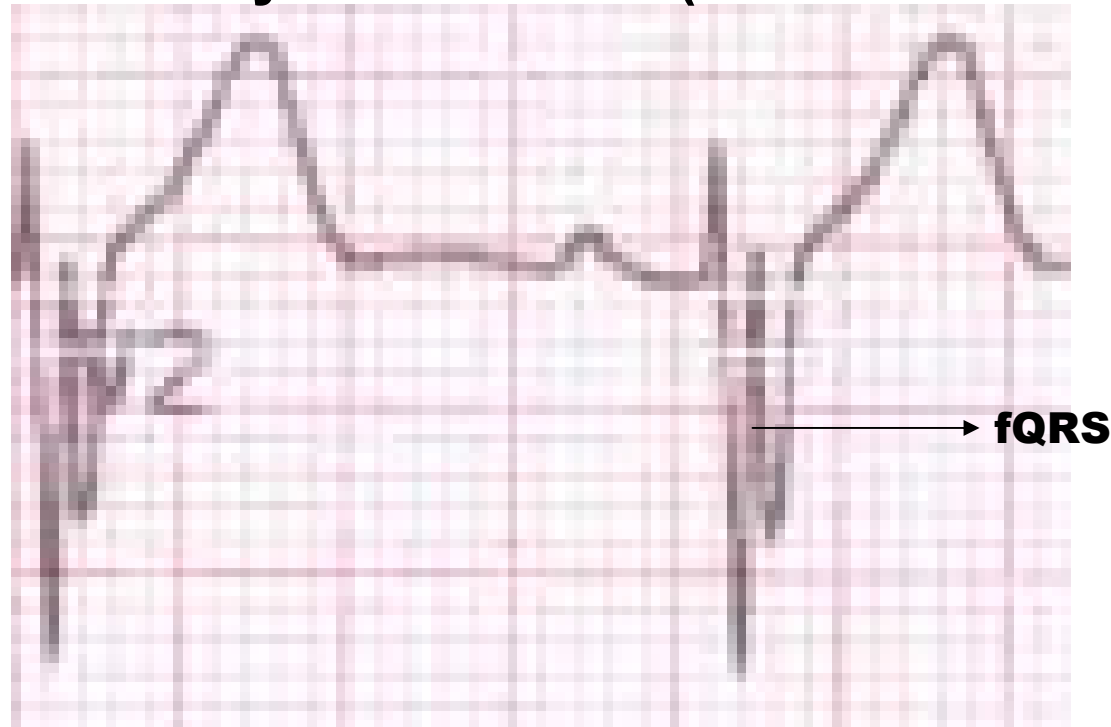


RS



RS

Fragmented QRS complexes (fQRS) With relatively narrow QRS(QRSd = 118ms)



fQRS is associated with increased mortality and arrhythmic events in patients with CAD. Additionally, it is a marker of ARVD/C and BrS. In BrS, the presence of fQRS predicts episodes of VF during follow-up. fQRS may be of value in determining the risk for SCD and guiding selection for device therapy in patients with structural heart disease and BrS. It is possible that the predictive value of fQRS for SCD can be enhanced further by combining a marker of repolarization abnormality such as microwave T wave alternans.

1. Das MK, El Masry H. Fragmented QRS and other depolarization abnormalities as a predictor of mortality and sudden cardiac death. *Curr Opin Cardiol.* 2010 Jan;25:59-64.

Several invasive and noninvasive tests for risk stratification of SCD have been studied, mostly in the context of structural heart disease such as CAD, cardiomyopathy, and heart failure. Tests such as MTWA (repolarization abnormality) and SAECG (depolarization abnormality) have high negative predictive values but a low positive predictive value in patients with MI or cardiomyopathy(1).

The presence of a fQRS on a ECG is another marker of depolarization abnormality.

fQRS represents conduction delay caused by myocardial scar in patients with CAD(2).

The fQRS is an independent predictor of cardiac events in patients with CAD. It is associated with significantly lower event-free survival for a cardiac event on long-term follow-up(3). However, fQRS is not specific for CAD and is also encountered in other **myocardial diseases such as cardiomyopathy and congenital heart disease**. fQRS is associated with increased mortality and arrhythmic events in patients with CAD. The utility of fQRS in risk stratification of SCD needs to be explored further, especially in nonischemic cardiomyopathy and heart failure.

Diffuse Fragmented QRS is as an Index of Extensive Myocardial Scar(4) and poor prognosis. f-wQRS on a standard 12-lead ECG is a moderately sensitive and highly specific sign for myocardial scar in patients with known or suspected CAD. f-wQRS is also an independent predictor of mortality(5).

1. Das MK, Zipes DP. Fragmented QRS: a predictor of mortality and sudden cardiac death. Heart Rhythm. 2009 Mar; 6(3 Suppl):S8-14.
2. Das MK, Saha C, El Masry H, Fragmented QRS on a 12-lead ECG: a predictor of mortality and cardiac events in patients with coronary artery disease. Heart Rhythm. 2007 Nov;4:1385-1392.
3. Jose F, Krishnan M. Fragmented QRS Electrocardiogram - The Hidden Talisman? Indian Pacing Electrophysiol J. 2009 Sep 1; 9: 238-240.
4. Aslani A, Tavoosi A, Emkanjoo Z. Diffuse Fragmented QRS as an Index of Extensive Myocardial Scar. Indian Pacing Electrophysiol J. 2010 Jan 7;10:67-68.
5. Das MK, Suradi H, Maskoun W, et al. Fragmented wide QRS on a 12-lead ECG: a sign of myocardial scar and poor prognosis. Circ Arrhythm Electrophysiol. 2008 Oct;1:258-268.

ECG analysis(first ECG

Rhythm: Sinus.; **HR:** 71bpm.; **P wave:** P axis: 62° and to front: normal; P voltage: 2,5mm: augmented. Suggestive of Right Atrial Enlargement (RAE); P duration: 85ms: normal; **PR interval:** 160ms: Normal.; **QRS axis:** the QRS axis in FP is located near -20° and to back. QRS axis between 0° and -30° may be observed in endomorphs biotype and pregnant women.; **QRS duration:** 118ms. Partially prolonged. QRS duration > 90 ms = 1 point in Romhilt Score System for LVE.; **QRS pattern:** In this case the inferior lead originating RS pattern. This pattern suggest smaller degree of LPFB associated with larger blockade degree of LAFB. This suspicion will be reinforced if the presence of first degree AV block, which may be pointing dromotropic difficulty in the posterior fascicle. Additionally a persistent S waves in left precordial leads V5-V6 is suggestive of fascicular block. If we have a VCG probably the QRS loop morphology will be rounded and not elliptical as in typical LAFB. QR and qR pattern in aVL (R<11m) and I(R<14mm) respectively followed by ST segment depression and asymmetrical negative T wave ("strain pattern"). Only repolarization pattern is suggestive of LVH. ST-T abnormalities 3 points in Romhilt Score System for LVE. ; Fragmented QRS complexes (fQRS) very visible in V2 and suggestive of scar and amputation of R wave in V2-V3. This is a risk marker.; **ST segment:** ST segment depression with upward convexity followed by a negative T wave inversion in the left high precordial leads I and aVL and positive in high apical leads(V5-V6), suggestive of left ventricular enlargement(strain pattern) 3 points in Romhilt Score System for LVE.; **T wave:** Strain pattern in I and aVL: Asymmetrical negative T wave.

QT interval: normal.

Conclusions of ECG diagnosis: Right Atrial Enlargement, probable Left Ventricular Enlargement (4 point in Romhilt Score System = probable LVE); bi- left fascicular block (complete LAFB associated with incomplete or smaller degree of LPFB) and fragmented QRS complex (fQRS).

Possible etiological diagnosis

This patient owns several elements suggestive of coronary anomalous origin, Bland-White-Garland syndrome or ALCAPA syndrome (from the: English acronym Anomalous Left Coronary Artery Arising from Pulmonary Artery)

The following present criteria are suggestive of this diagnosis:

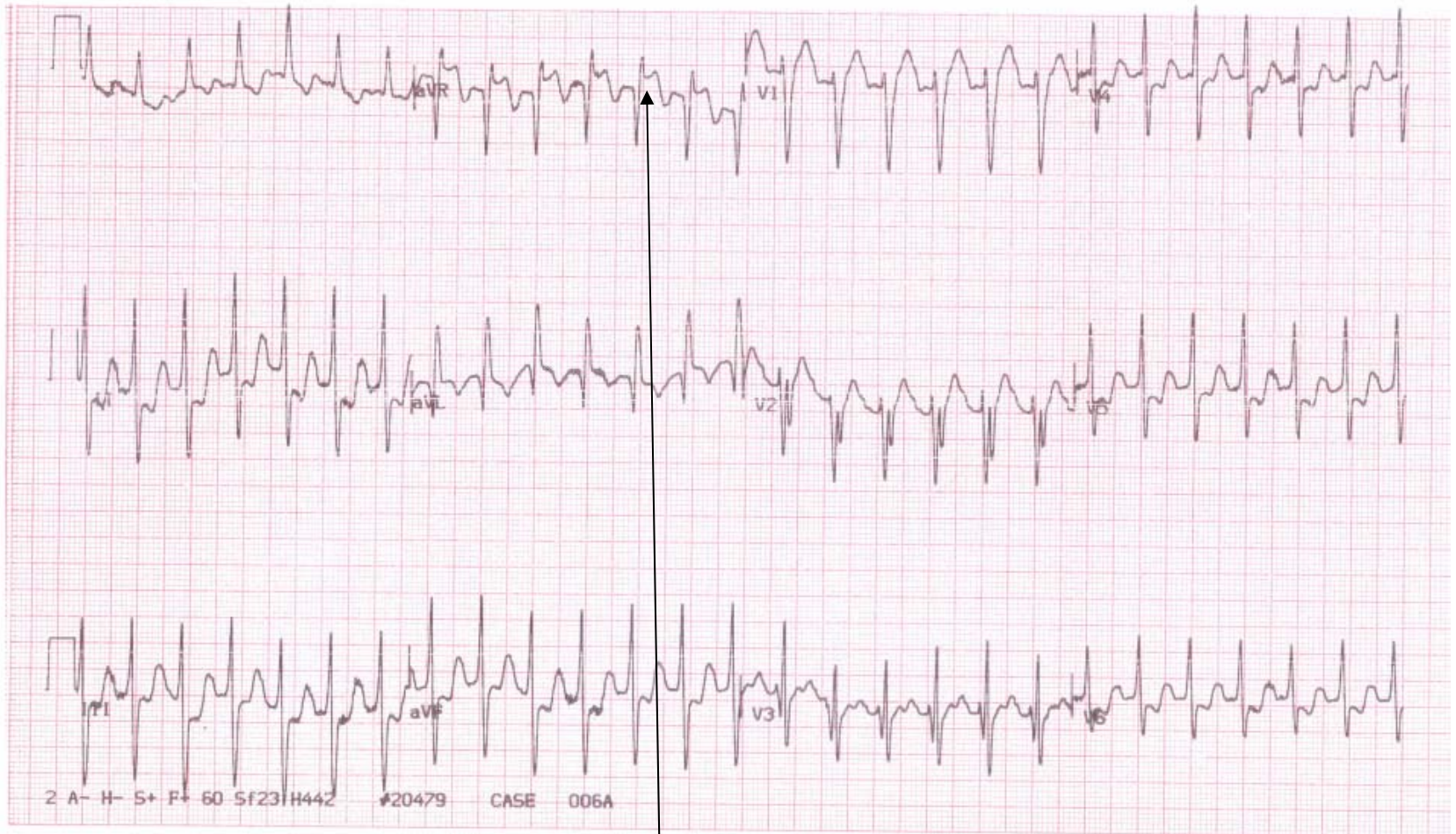
1. Suggestive or probable LVH or LVE pattern
2. Left axis deviation tendency secondary to chronic hypoxia
3. QRS duration prolongation not reaching values compatible to complete block. In this case we have QRSd 118ms!!!
4. Amputation of R waves in V2 and V3
5. Characteristic deeper, but not wide pattern in I, aVL. The QR pattern in I is found in more than 80% of the cases.
6. Absence of Q wave in inferior wall leads, which is considered typical and highly sensitive signal
7. Paroxysmal tachyarrhythmia's tendency.

10mm/mV
40Hz

HR: 164bpm
BP: 150/80

Speed: 0.0mph
Grade: 10.0%

Lead	ST(mm)	Lead	ST(mm)
I	-2.1	V1	6.8
II	-3.4	V2	6.1
III	-1.6	V3	1.0
aVR	3.0	V4	-2.0
aVL	-0.2	V5	-3.3
aVF	-2.5	V6	-2.9



aVR ST-segment elevation during narrow QRS complex tachycardia favors the atrioventricular reentry through an accessory pathway as the mechanism of the tachycardia. Why? Because see the next slide

Ho et al(1) analyzed ST-segment elevation in lead aVR during tachycardia to differentiate the narrow QRS complex tachycardia. A total of 338 12-lead ECGs during narrow QRS complex tachycardia were analyzed. Each patient underwent a complete EPS.

There were 161 episodes of atrioventricular nodal reentrant tachycardia (AVNRT), 165 episodes of atrioventricular reciprocating tachycardia (AVRT), and 12 episodes of atrial tachycardia (AT).

The prevalence of aVR ST-segment elevation was 71% for AVRT, 31% for AVNRT, and 16% for AT. For ST-T changes in different leads, logistic regression analysis showed aVR ST-segment elevation was the only significant factor to differentiate the types of narrow QRS complex tachycardia.

The sensitivity, specificity, and accuracy of aVR ST-segment elevation to differentiate AVRT from AVNRT and AT were 71%, 70%, and 70%, respectively. Among 117 episodes of AVRT with aVR ST-segment elevation, there were 76 (65%) left side, 23 (20%) right side, 14 (12%) posterior septal, and 4 (3%) antero-and mid-septal accessory pathways

The authors concluded that:

aVR ST-segment elevation during narrow QRS complex tachycardia favors the atrioventricular reentry through an accessory pathway as the mechanism of the tachycardia.

1. Ho YL, Lin LY, Lin JL, et al. Usefulness of ST-segment elevation in lead aVR during tachycardia for determining the mechanism of narrow QRS complex tachycardia. *Am J Cardiol.* 2003 Dec 15;92:1424-1428.

1mm/mv
40Hz

HR: 97bpm
BP: 150/90

Speed: 0.0mph
Grade: 0.0%

Lead	ST(mm)	Lead	ST(mm)
I	-1.2	V1	5.5
II	-1.0	V2	5.9
III	0.2	V3	2.1
aVR	1.3	V4	-0.1
aVL	-0.6	V5	-1.3
aVF	-0.3	V6	-1.3



Colleagues Opinions

First commentaries:

My greetings to the Teacher Moffa. I esteem respects for associating himself to this project.

First ECG: Sinus rhythm , QRS axis located in 0° , right atrial enlargement, fractioned QRS complex suggesting parietal blockade. R waves DI + aVL > 15mm suggesting some degree of left ventricular hypertrophy, even in the absence of Sokolow-Lyon criteria.

Second ECG: (It I think it was performed during the threadmill stress test?) The electrodes seem dislocated in reference to the first ECG. Tachycardia of QRS regular wide 165bpm. The differentiated diagnosis should be done among atrial flutter 2:1, atrial tachycardia atrial 2:1 and re-entered atrioventricular tachycardia. Even so in the atrial flutter we expect a heart rate near 150bpm. Because the ECG suggests right atrial and ventricular overload, I put atrial flutter and the atrial tachycardia atrial, before the re-entered atrioventricular tachycardia.

Third ECG: Sinus tachycardia, right atrial enlargement. And minimal ST segment depression in lateral wall.

Adrian Baranchuk

Mi agradecimiento al Prof Moffa, y mis respetos por sumarse a este proyecto.

ECG 1: RS, Eje en 0, agrandamiento auricular predominantemente derecho, fraccionamiento del QRS por posible bloqueo parietal. La suma de DI y aVL es $>$ a 15 mm, sugiriendo algun grado de hipertrofia ventricular izquierda (a pesar de S-Lyon negativo)

ECG2: *(entiendo que es inducido durante la prueba ergometrica?) Los electrodos parecen desplazados respecto de ECG 1. Taquicardia de QRS ancho regular a 165 lpm. Diagnostico diferencial: a. Aleteo auricular 2:1, b. taquicardia auricular 2:1, c. reentrada auriculoventricular. Si bien en el aleteo esperaríamos una FC mas cercana a 150 lpm, como me impresiona que hay sobrecarga decavidades derechas, he colocado al aleteo y a la taquicardia auricular, antes que a la reentrada.*

ECG 3: Taquicardia sinusal agrandamiento auricular predominantemente derecho. Ligerio infradesnivel del segmento ST en territorio lateral.

Adrian Baranchuk-

Minhas saudações ao Professor Moffa. Estimo respeitos por associar-se a este projeto.

ECG 1: Ritmo sinusal, eixo em elétrico do QRS em 0° , sobrecarga atrial direita, complexo QRS fracionado sugerindo bloqueio parietal. A somatória de DI e aVL $>$ 15mm sugerindo algum grau de hipertrofia ventricular esquerda, mesmo na ausência do critério de Sokolow-Lyon.

ECG 2: (penso que foi realizado durante a prova de esforço?) Os eletrodos parecem deslocados em referência ao primeiro ECG. Taquicardia de QRS largo regular 165bpm. O diagnóstico diferenciado deve ser feito entre flutter atrial 2:1, taquicardia atrial 2:1 e reentrada atrioventricular. Mesmo assim no flutter esperaríamos um frecuencia cardíaca próxima a 150bpm, com me sugere sobrecarga das camaras direitas, tenho colocado o flutter e a taquicardia atrial, antes que a reentrada.

ECG 3: Taquicardia sinusal, sobrecarga atrial direita predominante. Discreto subdesnivelamento do segmento ST em parede lateral.

Adrian Baranchuk

Ritmo sinusal, crecimiento auricular, HBAI, hipertrofia VI, infra ST DII, V5 y V6 (puede estar relacionado a la hipertrofia VI), estos cambios aumentan en el maximo esfuerzo (parece tener una taquicardia no sinusal, flutter?) y se mantienen en la etapa de recuperaci3n, con una FC cercana a 100/min. Pueden ser todos cambios relacionados a una HTA de larga data, HBAI, hipertrofia del VI y la predisposicion a arritmias auriculares (FA, Flutter),

saludos

Pancho Femenia

Ritmo sinusal, sobrecarga atrial, bloqueio divisional ntero-superior, sobrecarga ventricular esquerda, segmento ST infradesnivelado em II, V5-V6(consecuencia da sobrecarga ventricular esquerda?). Estes cambios aumentam no mximo esforo (parece ter uma taquicardia no sinusal, flutter?,que persiste na etapa de recuperao com uma frecuencia cardiaca pr3xima dos 100bpm. Todos estes cambios podem estar relacionados com uma hiertenso arterial de longa data, bloqueio divisional ntero-superior, sobrecarga ventricular esquerda, e tendncia a desenvolver arritmias atriais tipo FA o flutter.

Saudaes

Chico Femenia

Sinus rhythm, atrial overload, LSFB, LVH, ST segment depressed in II, V5-V6 (consequence of the left ventricular overload?). These changes increase at most effort (it seems to have a not sinus tachycardia AF ou flutter, It persists in the recovery stage with a hear rate next of the 100bpm. All of these changes can be related with a long date High blood pressure, LAFB, LVH, and tendency to develop atrial arrhythmias types AF or flutter

Greetings

ChicoFemenia

Amigos disculpen el atrevimiento me llama la atención de este trazado dos cosas: la onda Q profunda de aVL con T negativa y em D1 semejante pero de menor voltage.

El rSRS de V1 y rsRS de V2 me hace pensar en una necrosis focal.

Si imaginamos el vectocardiograma tenemos una zona donde faltaria actividad electrica. y no hay q de V3 a V6.

Podria ser una coronaria izquierda anomala.

Seguro aprenderé mas de las criticas que del silencio.

Dr. Emilio Marigliano emilio@SANMARTINMZA.COM.AR

Prezados Amigos: neste traçado chama minha atenção duas coisas: a onda Q profunda de aVL com onda T negativa e um padrão semelhante porém de menor voltagem em DI.

Os complexos QRS do tipo rSRS´de V1 e rsR`S`deV2 sugerem-me necrose focal.

Se imaginamos o vetorcardiograma teremos uma zona de inatividade elétrica.

Por outra parte, não há onda q de V3 a V6.

Poderia tratar-se de uma coronária anômala?

Tenho certeza que irei a aprender mais com as críticas do que com o silêncio.

Appreciated Friends: In this traced mine flame attention two things: The profound wave Q of aVL with negative T wave and a similar pattern however of smaller voltage in I lead.

The QRS complexes type Rsr´S´of V1 and rsR´´S`deV2 suggest me focal necrosis.

If we imagine the ECG it will have electrically inactive zone.

Additionally, there is no q wave q from V3 to V6.

Could it be a anomalous coronary artery?

I am sure that I will go to to learn more with the criticisms than with the silence.

Dear Emiliano far from his criticism reasoning seemed to me highly appropriated

In fact this patient owns several elements in his ECG who can very well lift the coronary anomalous origin suspicion, syndrome of Bland-White-Garland or ALCAPA syndrome (from the: English acronym is Anomalous Left Coronary Artery Arising from Pulmonary Artery)

Which ones are these elements

1) Suggestive of LVH

2) Left axis deviation tendency consequence of myocytes replication secondary to chronic hypoxia. QRS axis it may be normal; however there are cases –particularly in adults- with a tendency to extreme shift to the left. The cause of the shift is controversial. It has been proposed that it may be the consequence of selective hypertrophy of the LV postero-basal wall without irrigation involvement (irrigated by the right coronary artery).

The anterior and lateral walls, irrigated by the left coronary artery, are thin and possibly fibrotic.

3) QRS duration: there usually is mild increase for the age, not reaching values compatible to block. In this case we have QRSd 118ms!!!

4) Amputation of R waves in V2 and V3. As this case.

They are characteristically deeper, but not wide. Q wave with depth ≥ 3 mm and ≥ 30 ms with QR pattern in at least of the following leads: I, aVL, and from V5 to V7, and absence of Q wave in inferior wall leads, which is considered typical and highly sensitiv

5) There is a description in literature of paroxysmal AV block complicated with syncope, which requires permanent pacemaker implantation Three pathophysiologic mechanisms have been proposed to explain the origin of ventricular arrhythmias:

1) Local ischemia caused by short circuit;

2) Reentry circuit that originates in the peripheral region of the area involved by necrosis;

3) Electrical instability secondary to endocardial fibrosis².

The QR pattern in DI is found in more than 80% of the cases in DI and aVL. In this last lead, Q is usually greater than 50% of the voltage of R wave. In this age range, the average voltage of R wave in V6 is 14 mm. Maximal 24 mm (our case: 21 mm).

Congratulation is a good choose.... I think

Samuel Sclarovsky

My first diagnosis is Asymmetric Hypertrophic Cardiomyopathy. I would like, to analyze the changes occurring in this ECG the frontal axis is shifted to the left, but the R waves in II and III. are very high, and the q waves are deep in aVL and I.

This pattern: left axis deviation with high R is seen mostly in mid chronic anterior wall infarction in which the potentials of the basal and mid anterior wall are weakened and then the healthy inferior wall shifts the vector to the right and down but this ECG is not a myocardial infarction. Why the vector shifts down and right? Because the basal wall is weakened by disorganization of the myofibrils or replaced by fibrosis

It is important to remark the appearance of q waves in V2, V3. V2 expresses the upper paraseptal area and neighbor of the basal area, and V3 expresses the mid septum

The lack of q in V5, V6 is due to the fibrotic area which cannot induce the septal vector as in the normal depolarization. Another important element worthy to discuss is the ST depression in the apical lateral wall. The acute and chronic ST depression due to an increase in diastolic pressure are expressed by ST depression with inverted T waves in acute cases such as circumferential subendocardial ischemia or in chronic systolic overload.

In this case the ST depression is accompanied by a positive T wave. In my opinion this pattern indicates high wall tension due to the low compliance of the apex.

My final diagnosis is asymmetric hypertrophic cardiomyopathy with fibrotic or myocyte disorganization in the basal and high and mid septal of left ventricle with altered diastolic function and as the case of the elderly repeated frequently that the ECG is a cardiac method of investigation able to supply sophisticated information.

My kind regards

**This is the final word of all Einthoven's letters to Sir Thomas Lewis
Samuel**