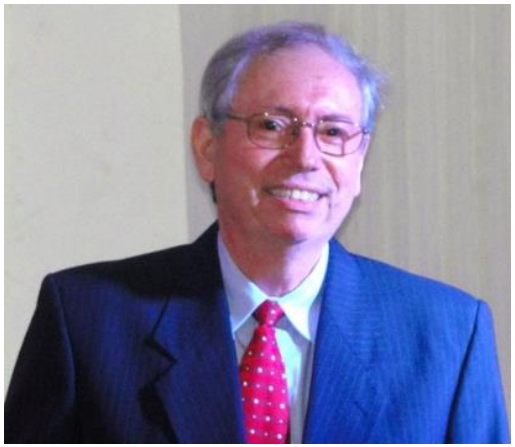
Anterolateral myocardial infarction: a diagnostic challenge



https://ekgvcg.wordpress.com/

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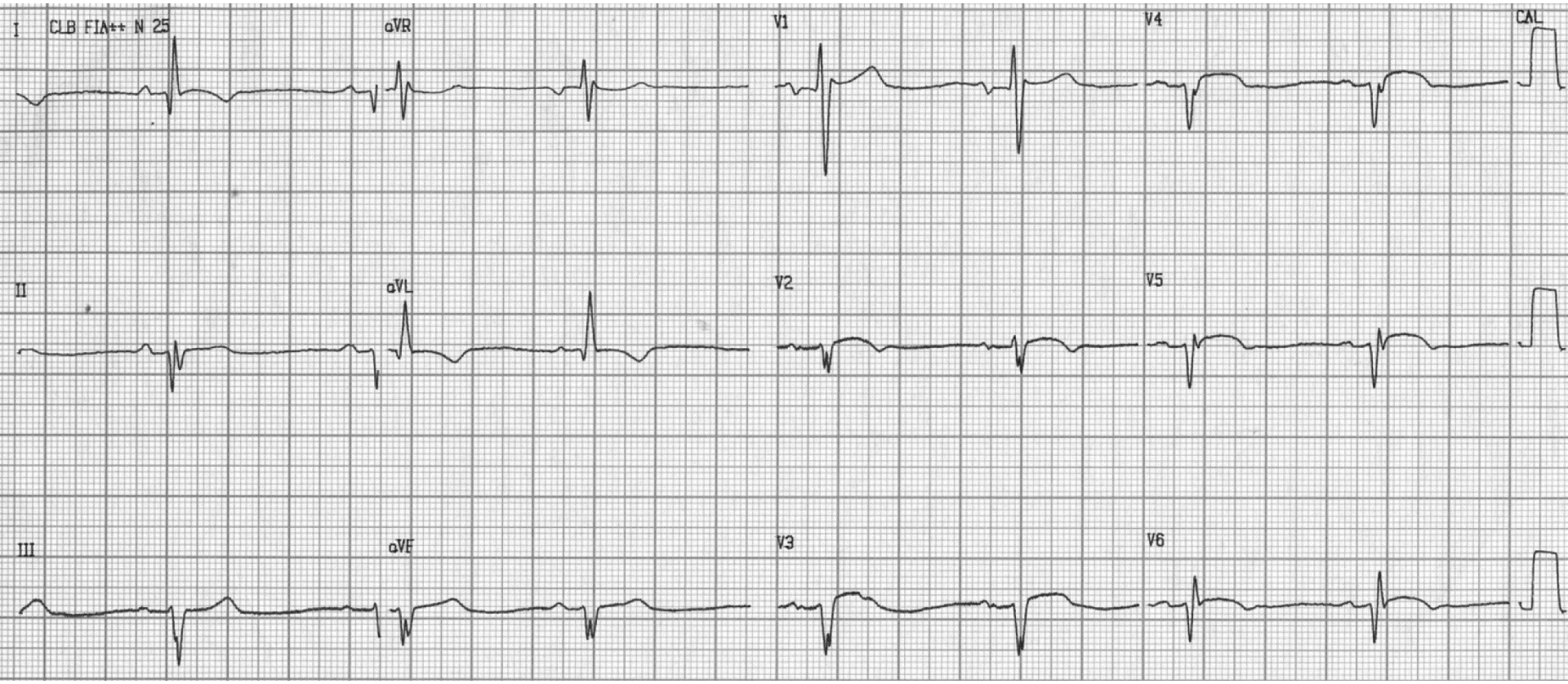
Portuguese: Reporte de caso

Identificação: Masculino, branco, 61 anos, advogado.
Queixas: Dispneia e tonturas há 1 mês relacionados com esforço físico. Relata também precordialgia atípica e palpitações.
Antecedentes pessoais: Hipertenso, dislipidêmico e diabético. Nega síncope.
Antecedentes familiares: Pais falecidos de causa desconhecida. 2 irmãos com “miocardiopatia”.
Medicação em uso: losartana, hidroclorotiazida, anlodipino, sinvastatina, glifage, glicazida e atenolol.
Exame físico: PA=150 x 100; RCR 2T b4 SS+/4 no foco mitral;pulmões limpos. Abdome: NDN. Sem edema periférico; pulsos periféricos palpáveis
Laboratório: Troponina e CK-MB normais
ECG1 e ECG2. Realizamos também ecocardiograma e coronariografia.
Qual é o diagnóstico eletrocardiográfico e clínico?

English: Case report

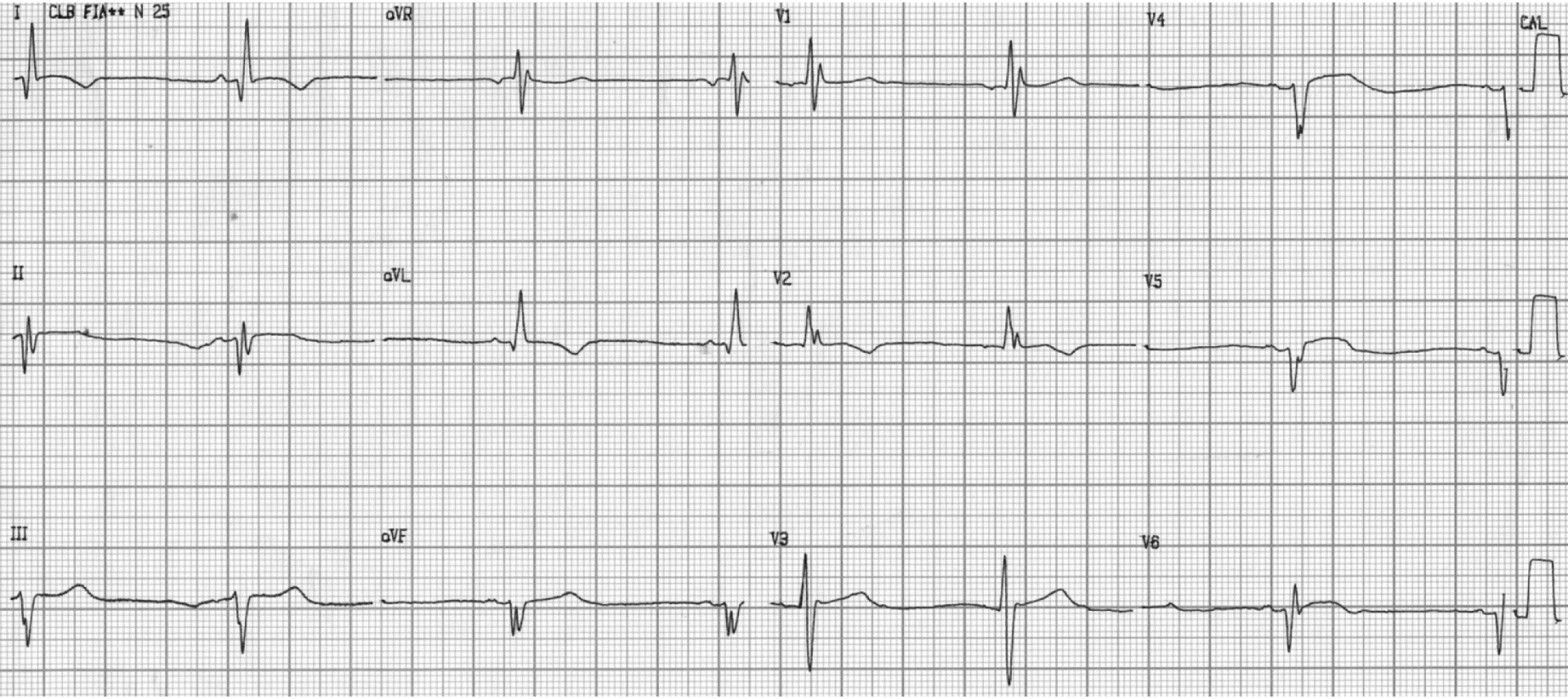
Identification: Caucasian man, 61 years old, lawyer
Complaint: dyspnea and dizziness 1 month ago related to physical efforts. Also he complains atypical chest pain and palpitations
Personal antecedents: history of high blood pressure, dyslipidemia and type 2 diabetes mellitus. He denies syncope
Family background: parents deceased of unknown causes. Two brothers with "cardiomyopathy“.
Current medications: losartan 50mg 2x/day, hydrochlorothiazide 25mg, amlodipine 5mg, simvastatin 20mg, glifage 500mg, gliclazide 80mg 1x/day, and atenolol 50mg
Physical: BP = 150 x 100 mmHg; RCR 2T b4 systolic murmur + / 4 in mitral focus, clear lungs. Abdomen: normal. No peripheral edema. Palpable peripheral pulses.
Lab: Normal troponin and CK-MB
ECG1 and ECG2. Additionally, we performed transthoracic echo and coronary angiography.
Which is the electrocardiographic and clinical diagnosis?

ECG1



ECG diagnosis:

ECG2



ECG diagnosis:

Colleagues opinions

Again a challenging case from your group! I have some problems with 25 mm/sec, because in Finland we use 50 mm/sec. but I will try to analyze. It would have helped if one new the time or other difference between the ECGs.

ECG 1.

In general, the ST/T changes look more like post myocardial infarction than hypertrophic cardiomyopathy.

PTF in V1 – left atrial abnormality, maybe elevated LV filling pressure (diastolic dysfunction).

QRS fragmentation in many leads, probably indicating myocardial fibrosis.

No pathological Q waves, but loss of R waves at least in V2-V6, also low-amplitude R waves in the inferior leads, possibly due to LAFB.

ST elevations in V2-V6 could represent post-MI with dyskinesia of the anterior/anterolateral wall, indicating a myocardial infarction within a couple of weeks.

Strain pattern in aVL - maybe elevated LV filling pressure (diastolic dysfunction).

ECG 2

The P waves are different, especially in V1-V3 – the ECG is probably recorded from a higher intercostal space.

V4 looks like previous V3 and V5 like previous V4 – different lead placement possible.

RBBB (or at least partial RBBB), LAFB as in ECG 1

My guess would be a myocardial infarction with atypical symptoms in a diabetic 1 month ago. Occluded artery (probably LAD) on angiography. RBBB+LAFB could indicate proximal LAD occlusion. Dyskinesia or even aneurysm of LV in echo and also signs of systolic and diastolic dysfunction.

Best regards

Kjell Nikus M.D. P.h.D. Finland Tampere University Hospital (TAUH),
https://www.researchgate.net/profile/Kjell_Nikus2

¡Una vez más un caso difícil de su grupo!. Tengo algunos problemas con la velocidad del papel a25 mm /s, ya que en Finlandia utilizamos 50 mm / seg. pero voy a tratar de analizar. Habría ayudado si esclarecieran la diferencia de tiempo u otras diferencias entre los 2 ECGs.

ECG 1. En general, los cambios en el ST/T sugieren más un post infarto de miocardio que una miocardiopatía hipertrófica. Componente final de la P (PTF) en V1 - sugiere aurícula izquierda por probable aumento de la presión de llenado del VI (disfunción diastólica). Fragmentación del QRS se observa en muchas derivaciones, lo que probablemente indica la fibrosis miocárdica. No hay ondas Q patológicas, pero la pérdida de las ondas R al menos en V2-V6, también ondas R de baja amplitud en las derivaciones inferiores, posiblemente debido a bloqueo del fascículo antero-superior izquierdo.

La elevación de ST de V2-V6 podrían representar post-MI con discinesia de la pared anterior / anterolateral, lo que indica un infarto de miocardio con un tiempo de evolución dentro de un par de semanas. Patrón de tensión “strain pattern” en aVL – por probable presión de llenado del VI elevada (disfunción diastólica).

ECG 2 Las ondas P son diferentes, especialmente en V1-V3 – Probablemente se registraron, a partir de un espacio intercostal más superior. V4 se parece a V3 y V5 anterior como V4 anterior – posible diferente colocación de un electrodo.BRD o al menos incompleto (BIRD), bloqueo del fascículo antero-superior izquierdo como en el ECG 1 Mi conjetura diagnóstica sería: un infarto de miocardio con síntomas atípicos por ser diabético de hace aproximadamente 1 mes. La arteria ocluida en la angiografía sería probablemente la descendente anterior izquierda (LADA). La presencia de BIRD + Bloqueo fascicular antero-superior izquierdo podría indicar una oclusión proximal de LADA.

Discinesia o incluso aneurisma de LV en el eco y también signos de disfunción sistólica

y diastólica.Atentamente

Kjell Nikus M.D. P.h.D. Finland Tampere University Hospital (TAUH),
https://www.researchgate.net/profile/Kjell_Nikus2

Spanish

Me impresiona como un infarto antero-lateral en evolución aún sin reperfusión por tener ST-T elevado en las precordiales. La onda T invertida en aVL corresponde a la región basal izquierda alta y media expresada en I y aVL. Esto suele ocurrir después de la angioplastia o del tratamiento médico. La reperfusión siempre tiene inicio en los bordes siendo en este caso el más afectado el lateral medio porque la Q es más profunda en I que en aVL.

Las ondas R altas en V1 y aVR expresan remodelación de las Q en V5-V6.

El segundo complejo QRS se debe a despolarización lenta de la base cardiaca.

La presencia de Q de II sin Q en III acompaña a V5-V6 en infartos laterales expresando siempre la pared posterior que se encuentra entre la inferior y lateral.

La arteria "culpable" es la circunfleja izquierda distal alrededor de la primera marginal pero la segunda marginal es muy larga porque irriga la pared antero-lateral baja hasta el septo medio (Q de V6 > Q V3), lo que explica una cierta protección de la pared inferior, la cual debe estar irrigada por la descendente posterior rama de la coronaria derecha.

Las ondas P son normales; lo que llama la atención porque la presión diastólica final debe estar aumentada por el compromiso del ápex (V5) lo cual aumentaría la Pd2 del VI alterando la P

El diagnóstico diferencia se plantea con cardiomiopatías congénitas o adquiridas en cuyo caso las coronarias estarían normales en la angiografía.

Samuel Sclarowsky, MD Israel
Observación otras possiblidades son

1. Quiste hidatídico
2. Aneurisma em presencia de hipertrofia apical
3. Aneurisma idiopático da pared lateral baja apical y del septo.

English

I'm impressed as an anterolateral MI without reperfusion yet evolving because we observe ST-T elevated in precordial leads.

Inverted T wave in aVL corresponds to the upper and middle left ventricle basal region expressed in I and aVL. This usually occurs after angioplasty or medical treatment. Reperfusion always start at the edges. In this case being the most affected side because QI is deeper than Q aVL.

High R waves in V1 and aVR remodeling express Q in V5-V6.

The second QRS complex is due to slow depolarization of the cardiac base.

The presence of QII without QIII accompanies apex-lateral infarct(V5-V6). always expressing the dorsal wall located between lateral and inferior walls.

The "culprit" artery is a distal left circumflex artery around the first marginal but the second marginal is very long because irrigates the anterolateral low wall to the middle septum (Q V6> Q V3), which explains some protection the inferior wall, which must be supplied by the posterior descending branch of the right coronary artery.

P waves are normal; what it draws attention because end-diastolic pressure should be increased by the commitment of the apex (V5) which would increase the LV final pressure altering the P

Differential diagnosis arises with congenital or acquired cardiomyopathies in which case would be normal coronary angiography.

Samuel Sclarowsky, MD Israel

Observation: other possibilities are

1. hydatid cysts
2. Aneurysm in the presence of apical hypertrophy
3. Idiopathic aneurysm gives low apical and lateral septal wall.

Spanish

Queridos colegas: El diagnóstico diferencial del infarto antero-lateral e inferior por probable obstrucción de una arteria circunfleja izquierda dominante es la miocardiopatía hipertrófica asimétrica.

El segundo ECG tiene BIRD sin hemibloqueo anterior. Es posible también que haya sido por una oclusión de una DA grande que da vuelta el ápex cardiaco irrigando parte de la cara inferior.

Los estudios para el diagnóstico diferencial están bien encaminados

Con afecto

Gerardo Nau MD
Médico Cardiólogo Intervencionista
Facultad de Medicina de la Universidad del Salvador,
Buenos Aires, Argentina.

English

Dear colleagues: The differential diagnosis with anterolateral and inferior MI by occlusion likely a dominant left circumflex artery is asymmetric hypertrophic cardiomyopathy However, the second ECG shows incomplete right bundle branch block without left anterior fascicular

Studies for the differential diagnosis are on track

With love

Gerardo Nau MD Buenos Aires Argentina

Spanish: Hola foristas: Los trazados de estos dos ECG son tan especiales que casi no me atrevo a opinar porque tengo dudas de que haya problemas técnicos: filtros ?, V2-V3 del 2º ECG cambian radicalmente pero no V4-V6, V2 del 2º ECG se parece mucho a aVL (donde está colocado el electrodo de V2?, cerca del hombro izquierdo?), Si se plantea el diagnóstico diferencial entre infarto y miocardiopatía hipertrófica me inclinaría mas por ésta última.
Saludos
English: Hello forum members,
The ECGs are so special that I hardly dare to say because I have doubts that have technical problems: filters, V2-V3.
ECG-2 change radically but not V4-V6, V2 2nd ECG closely resembles aVL (where is placed the electrode V2 ?, near the left shoulder?) If the differential diagnosis of hypertrophic cardiomyopathy and coronary artery disease I am inclined towards the latter arises.
regards
Miquel Fiol Sala, MD, PhD Director Científico del Instituto de Investigación Sanitaria de Palma (IdISPa)
Hospital Son Espase Ctra de Valldemossa, 79 07010 Palma de Mallorca, España
Scientific Director of Palma Institute of Health Research. Hospital Son Espases, Palma, Balearic Islands, Spain.
President of ICE 2016. President 43rd International Congress on Electrocardiology Palma, Balearic Islands, Spain - June, 4-6 2016
Dear friends and colleagues,
We have the honour of inviting you to the 43rd International Congress on Electrocardiology – ICE 2016. It will take place at Palma de Mallorca, Balearic Islands, Spain, 4 to 6 June 2016. Surface electrocardiography has improved in the last years thanks to correlation with imaging techniques and advances in genetic studies. However, we need to consider that correlation with clinical studies is also just as useful. Electrocardiography is a basic technique that requires continuous learning. Electrocardiographic interpretation is crucial and must be carried out as part of imaging studies. We must make an effort to demonstrate that the electrocardiogram is a technique in constant evolution that provides information about aetiology, electrophysiology, mechanisms,

the indications for treatment and prognosis, not only in heart disease but also in many other aspects, always with a good cost-benefit ratio. This is why we want to give a practical purpose to the ICI-2016 Congress.
In addition to the classical topics in the field of electrocardiology, the ICE 2016 Congress will address new and important themes: atrial block and atrial arrhythmias (Bayes Syndrome), the ECG pattern of Brugada Syndrome (differential diagnosis), mistakes in the ECG interpretation in ACS, new insights into the electrophysiological mechanisms of atrial fibrillation,
technical advances in surface electrocardiography, the role of late-sodium channel modulators, and advances in the genetic diagnosis of arrhythmic disorders.
We very much hope that you will decide to attend the congress, and we will do our best to make you fall in love with Mallorca. Our best regards

Miquel Fiol Sala, MD, PhD

President of ICE 2016.

Antonio Bayes De Luna, MD, FESC, FACC

Honour President of ICE 2016.

Spanish

Saludos a todos los foristas:Con estos ECGs tengo más dudas que certezas; probablemente más que los otros integrantes del foro.El diagnóstico diferencial planteado hasta el momento es de cardiopatía isquémica versus miocardiopatía hipertrofia. Creo que se deben plantear además necrosis más miocardiopatía hipertrófica o hipertrofia ventricular más necrosis.Observo entre ambos ECGs, cambios de la onda P y de los complejos QRS, solo en el plano horizontal que me impresionan como posicionales.,

.**ECG-1:** ritmo sinusal, el primer vector esta aumentado de voltaje y se dirige hacia abajo, adelante y a la derecha. En el plano frontal las fuerzas del complejo QRS presentan una rotación horaria, el eje de la onda R se ubica alrededor de los -10º, luego rota anti horario y finaliza en los -60º.

En el plano horizontal el bucle del QRS presenta una rotación en 8 (primero rota horaria y luego anti-horaria), con el máximo vector que se opone a V1.

La onda T tiene dirección opuesta al complejo QRS y se localizada adelante, a la derecha y abajo. Este hallazgo me hace suponer hipertrofia ventricular izquierda. Me inclino por una hipertrofia septal importante.

No puedo descartar una cardiopatía isquémica ni una necrosis miocárdica secundaria a una miocardiopatía hipertrófica. Si este caso es el de una MH, la presencia de un bloqueo del fascículo izquierdo, no lo puedo diagnosticar; ya que el complejo QRS en su primera parte tiene rotación horaria en el plano frontal y si se trata de una hipertrofia septal, la fuerzas del septum basal aumentadas pueden producir la rotación anti-horaria posterior.

Con respecto al bloqueo incompleto de rama derecha , las últimas fuerzas del complejo QRS como lo demuestra DI terminan hacia la izquierda; la r´ de V1 y V2 yo creo, como mencioné anteriormente, que es por la posición de los electrodos.

Afectuosamente.

Isabel Victoria Konopka M.D. Hospital Argerich Buenos Aires.

English

Greetings to all forum members: With these ECGs I have more doubts than certainties; probably more than the other members of the forum. The differential diagnosis raised so far is coronary artery disease(CAD) versus HCM. I think they should raise also necrosis + hypertrophic cardiomyopathy(HCM) or HCM+ necrosis. I look between the two ECGs, changes of P wave and QRS complex, only in the horizontal plane that impress me as positional.

ECG-1: sinus rhythm, the first vector is increased in voltage and directed downward, forward and to the right. In the frontal plane QRS complex forces have a clockwise rotation, the axis of the R wave is located around -10º, then rotates counterclockwise and ends at -60°.

In the horizontal plane of the QRS loop has in 8 rotation (first portion and then rotates counter-clockwise), with the maximum vector that is opposes to V1.

The T wave has opposite direction related to the QRS complex and located below, to the right and down. This finding makes me think in left ventricular hypertrophy. I am inclined to think in significant septal hypertrophy.

I can not rule out ischemic heart disease or myocardial necrosis secondary to HCM. If this case is that of MCH, the presence of left anterior fascicular block, I can not diagnose; because as the QRS complex in its first part has clockwise rotation in the frontal plane and if it is a septal hypertrophy, increased basal septum forces can cause the rear counter-clockwise rotation.

With respect to incomplete right bundle branch block, the last forces of the QRS complex as shown by DI end to the left; r'of the V1 and V2 I think, as I mentioned earlier, which is malposition of the electrodes.

Affectionately

Isabel Victoria Konopka M.D. Argerich Hospital Buenos Aires

A mí me impresiona como un infarto antero-lateral. No es la imagen de MCH que suelo ver. Este trazado tiene elevación del segmento ST (que no se ve en la MCH). Por otra parte, en el segundo ECG veo una onda P que no conduce, pero al carecer de una tira de ritmo no me animo a decir de qué se trata. Cordiales saludos.

Luciano Pereira MD Ciudad del Este Paraguay

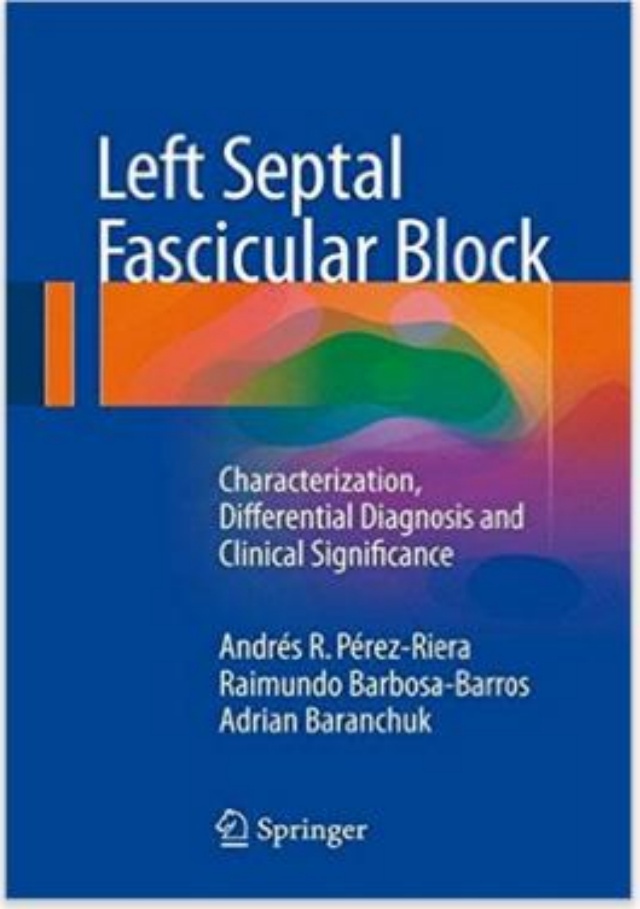
“The Voice of the Guarani People”

It impresses me as an anterolateral myocardial infarction. It is not the image I usually see in HCM. This ST-segment elevation observed is not seen in HCM. Moreover, in the second ECG,
I see a non conducted P wave, but lacking a long rhythm strip not dare to say what it is.

Greetings

Luciano **Pereira MD** Ciudad del Este Paraguay.

Final conclusions



Avaliable at:

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In the first place, like a living organism, Truth grows and its gradual evolution may be traced form the tiny germ to the mature product. Never springing, Minerva-like, to full stature at once, Truth may suffer all the hazards incident to generation and gestation. Much of history is a record of the mishaps of truth which have struggled to the birth, only to die or else to wither in premature decay, Or the germ may be dormant for centuries, awaiting the fullness of time -- All scientific truth is conditioned by the state of knowledge at the time of its announcement.

William Osler, MD, FRS.
Harveian Oration,
Royal College of Physicians
18th October 1906

Final diagnosis: obstructive septal asymmetric hypertrophic cardiomyopathy with a slight LVOT gradient.

Clinical and electrocardiographic diagnostic clues in this case:

I. **Complaint:** he complains of **atypical** chest pain and palpitations

II. **Family background:** he has two brothers with "cardiomyopathy".

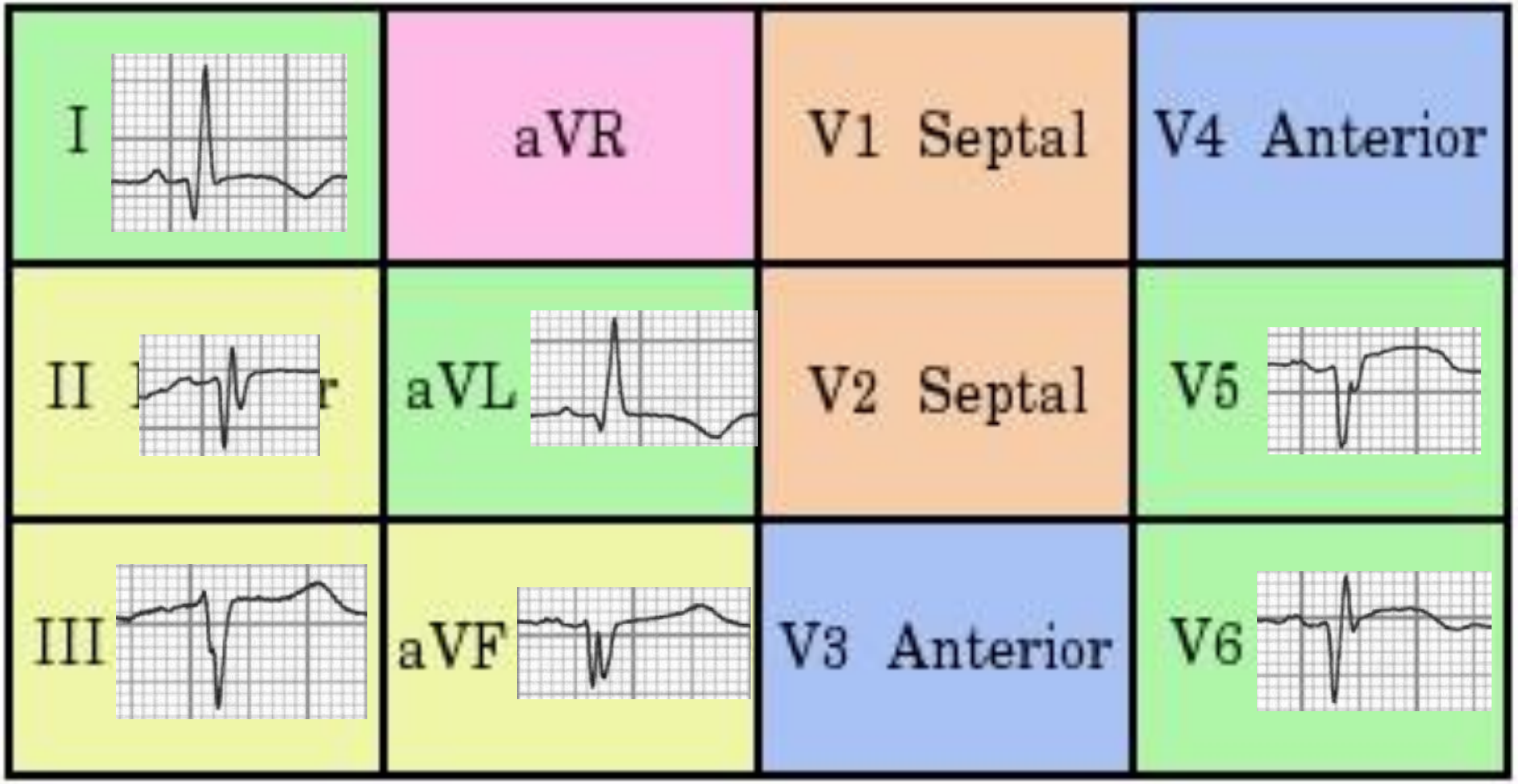
III. **Electrocardiographic:** Asymmetrical septal hypertrophy produces deep, narrow ("dagger-like") Q **waves** in the lateral (V5-6, I, aVL) and inferior (II, III, aVF) leads, such as this case. These may mimic prior MI, although the Q-wave morphology is different: infarction Q waves are typically ≥ 40 ms duration while septal Q waves in HCM are < 40 ms.

- Lateral Q waves are more common than inferior Q waves in HCM.
- abnormal Q waves reflect the interrelation between upper anterior septal thickness and other regions of the left and right ventricles
- The depth of negative T waves is related to craniocaudal asymmetry and apical late-enhancement. wider Q waves are associated with late-enhancement.
- Conduction disturbances and absent septal Q waves are associated with late-enhancement.
- Loss of electrical forces due to transmural myocardial fibrosis, altered direction of resultant initial QRS vector due to increased electrical forces of disproportionate hypertrophy of the basal septal and/or ventricular free wall, unopposed by apical forces.(**Koga 2004**) such us the present case in ECG-2
- RVH is significantly more common in patients without abnormal Q waves (**Lemery 1990**)
- Only the ratios of upper anterior septal to mean RV wall thickness (p less than 0.005) and to upper posterior wall thickness (p less than 0.05) are significantly related to the presence of abnormal Q waves and predicted Q wave location with a sensitivity, specificity and predictive accuracy of 90%, 88% and 89%, respectively.
- In HCM, abnormal Q waves on the ECG is primarily a function of the relation of RV wall thickness and upper anterior septal thickness.(**Dumon 2006**)
- Diagnostic deep Q waves were detected more frequently in female patients with HCM than in their male counterparts. (**Ohmoto-Sekine 2007**)

- In HCM with deep Q waves in limb leads, morphologic and electrocardiographic analysis showed similar features in both sexes. (**Ohmoto-Sekine 2007**)

Many patients with hypertrophic cardiomyopathy (HCM) have clinical – electrocardiographic picture of myocardial ischemia and dysfunction in the absence of extramural coronary atherosclerosis. Although hypertrophy and increased left ventricular pressure can account for such abnormalities, altered small intramural coronary arteries have also been described in such patients (**Maron 1986**). To determine the prevalence and extent as well as the clinical relevance of abnormal intramural coronary arteries, Maron et al preformed a histologic analysis of LV myocardium obtained at necropsy in 48 patients with HCM without atherosclerosis cand in 68 control patients with normal heart or acquired heart disease. In HCM, abnormal intramural coronary arteries were characterized by thickening of the vessel wall and a decrease in luminal size. The wall thickening was due to proliferation of medial or intimal components, or both, particularly smooth muscle cells and collagen. 83% of patients with HCM , had abnormalities of intramural coronary arteries located in the interventricular septum (33 patients), anterior LV free wall (20 patients) or posterior free wall (9 patients); an average of 3.0 ± 0.7 abnormal arteries were identified per tissue section. Altered intramural coronary arteries were also significantly more common in tissue sections having considerable myocardial fibrosis. HCM is a cardiac disease with a variety of clinical and morphologic features. Many patients with HCM manifest evidence of myocardial ischemia or damage, including angina. Abnormal intramural coronary arteries were also identified in three of eight infants who died of HCM before 1 year of age. In contrast, only rare altered intramural coronary arteries were identified in the 68 control patients and those arteries showed only mild thickening of the wall and minimal luminal narrowing. Moreover, of those patients with abnormal intramural coronary arteries, such vessels were about 20 times more frequent in patients with HCM than in control patients. Hence, abnormal intramural coronary arteries with markedly thickened walls and narrowed lumens are present in increased numbers in most patients with HCM studied at necropsy and may represent a congenital component of the underlying cardiomyopathic process. Although the clinical significance of "small vessel coronary artery disease" in HCM is unclear, the occurrence of

structurally altered intramural coronary arteries in areas of substantial myocardial fibrosis suggests a causal role for these arteries in producing ischemia.



Deep, narrow (“dagger-like”) Q **waves** in the lateral (V5-V6, I, aVL) and inferior (II, III, aVF) leads, such as this case. These may mimic prior MI, although the Q-wave morphology is different: infarction Q waves are typically ≥ 40 ms duration while septal Q waves in HCM are < 40 ms. Additionally, we observe clear notched or fQRS in II, III, aVF, V5 and V6.

Chest pain in Hypertrophic cardiomyopathy

Many patients complain of chest pain at rest or on exertion. Pain may also be precipitated by large meals or alcohol (**Gilligan 1996;Feiner 2013; Paz**). The causes of chest pain include myocardial ischemia due to microvascular dysfunction, increased LV wall stress and LVOTO. Congenital coronary artery anomalies, including tunneled left anterior descending artery or atherosclerotic coronary artery disease, may also be responsible (**Sorajja2003**). Systolic compression of epicardial and intramural vessels is very common but is not usually of clinical importance (**Mohiddin 2000; Sorajja 2003; Basso 2009**). Resting ECG abnormalities and a high prevalence of perfusion abnormalities on nuclear imaging and CMR mean that these techniques are of limited use in differentiating obstructive coronary disease from other causes of chest pain and in determining pre-test probability of coronary disease in patients with HCM (**Yamada 1998; Elliott 1996; Romero-Farina 2004; Soler R, Rodriguez 2006; Barbosa 2009**). Patients with typical angina on exertion should be considered for invasive or CT coronary angiography on the basis of their symptoms, age, gender and atherosclerosis risk factors, as outlined in existing ESC Guidelines (**Schroeder 2008; Montalescot 2013**). Coronary angiography is recommended in adult survivors of cardiac arrest, in patients with sustained ventricular arrhythmia and in symptomatic patients with previous coronary revascularization procedures (**Zipes 2006**). Invasive or CT coronary angiography should be considered before septal reduction therapy in all patients aged ≥40, irrespective of the presence of typical angina.

Transthoracic Echocardiographic diagnosis in the present case: septal asymmetric hypertrophic cardiomyopathy: Septal diastolic thickness = 20 mm; diastolic thickness of posterior wall = 8mm: septum/posterior wall ratio = 2.5; tele systolic acceleration. systolic anterior motion (SAM) of the mitral valve and dynamic left ventricular outflow tract (LVOT) mild obstruction .

Diastolic LV dysfunction.

Preserved biventricular systolic function index

LVEF = 66%. Normal

LV mass (265g). Normal!!!!

Normal diastolic and systolic LV volume.

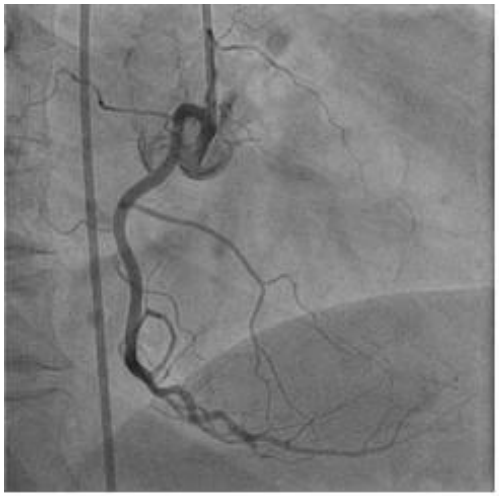
Normal LA size.

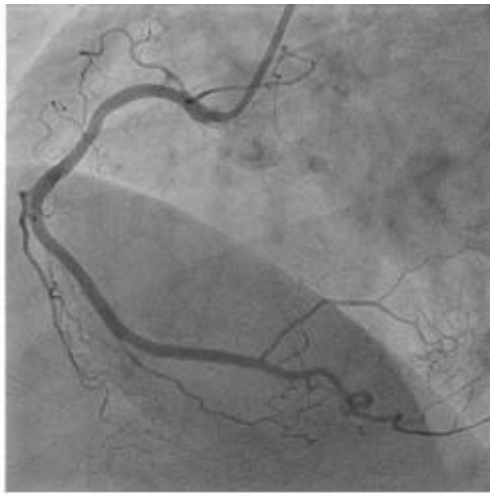
Cine coronary angiography: epicardial coronary arteries without obstruction of epicardial arteries.

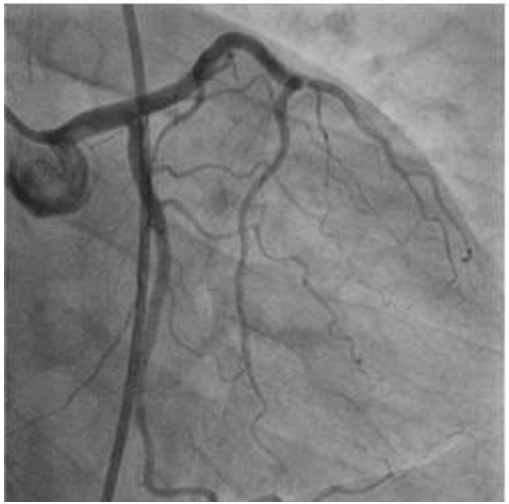
1. Microcirculation disease?
2. Decrease of vasodilator capacity?
3. Systemic compression of septal and subepicardial vessels;

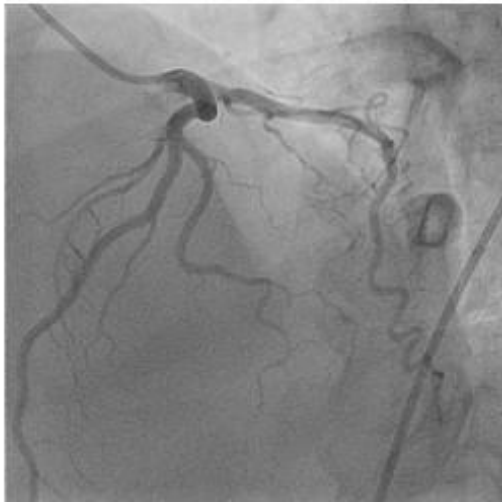
1. Fall of pressure in aorta root;
2. Difficulty in coronary filling by hypertrophy;
3. Coronary atherosclerosis in patients older than 50 years old;

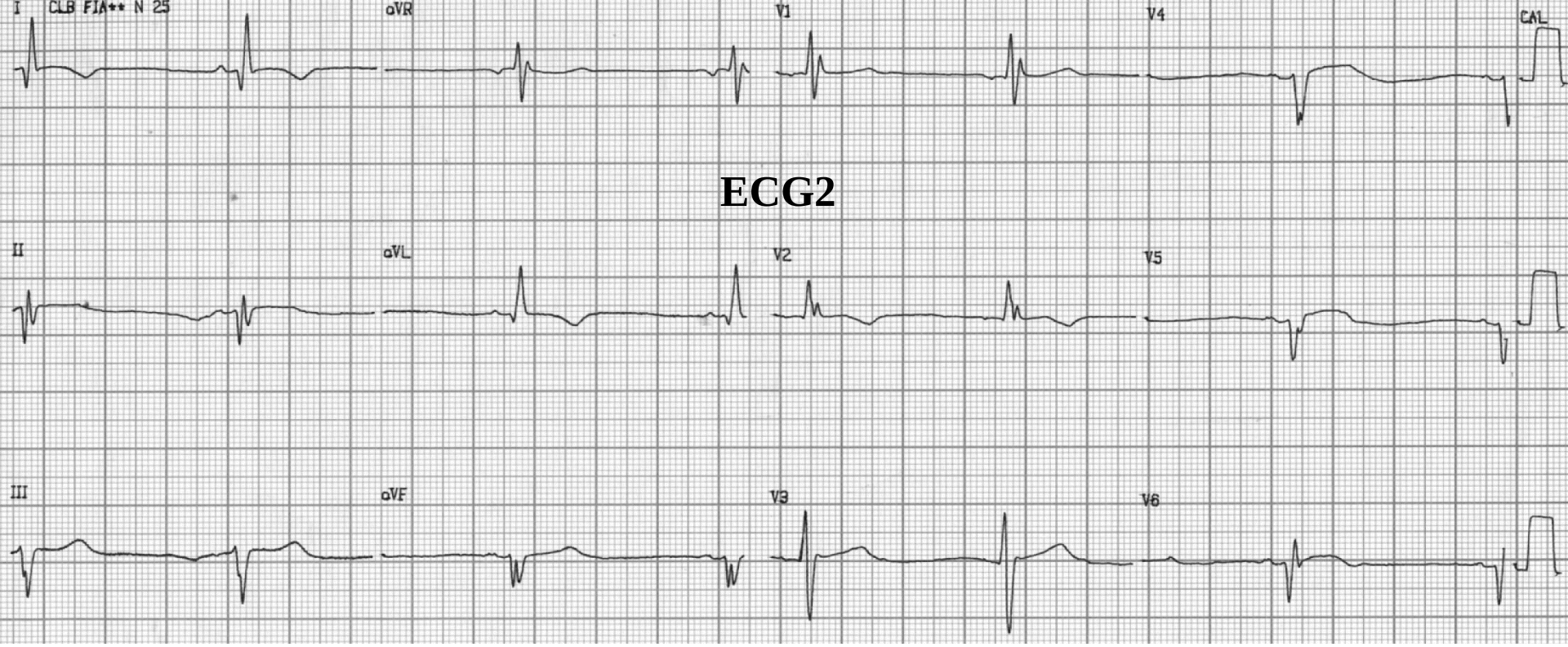
1. Excessive increase of mass and subsequent offer/demand disproportion.





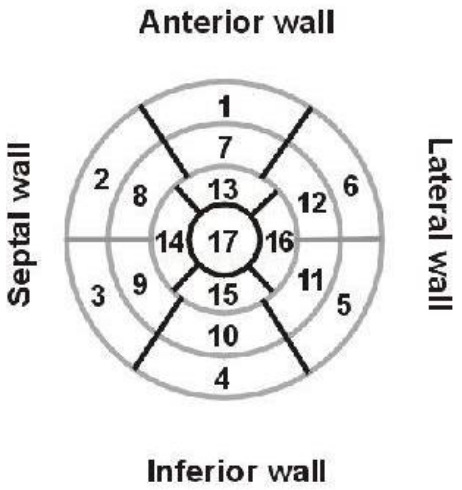




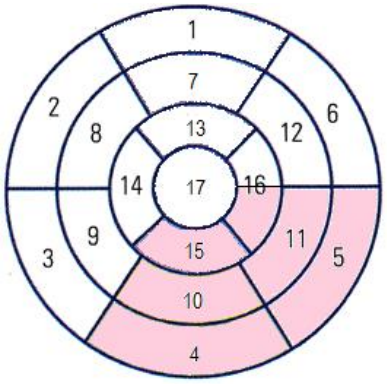


ECG diag.: Inactive anterolateral area: Q in II, III, aVF + Q in I, aVL, V5-V6 and/or RS in V1. SE: 70%. SP: 100%

Polar map. Short axis. "Bull's-eye"

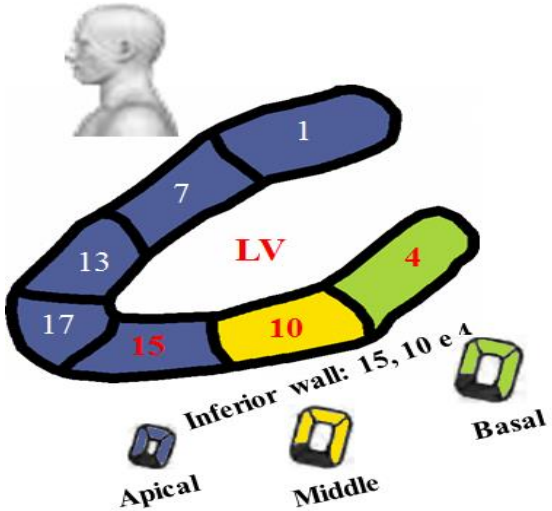


B3



inferior wall: 15, 10 and 4

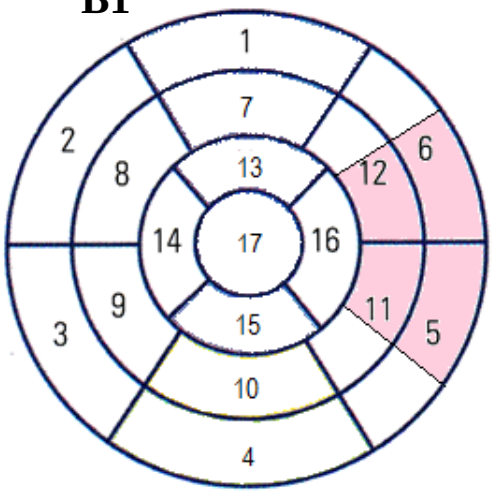
Left lateral view



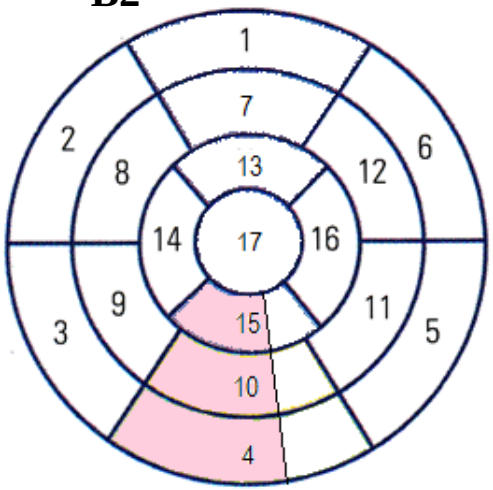
II –Inferolateral Zone

B1 – Q, qr or r in I, aVL, V5-V6 and/or RS in V1. Lateral. SE: 50%. SP:98%
B2 – Q in II, III and aVF. Inferior. SE: 87,5%. SP: 98%
B3 – Q in II, III, aVF + Q in I, aVL, V5-V6 and/or RS in V1. Inferolateral. SE: 70%. SP: 100%

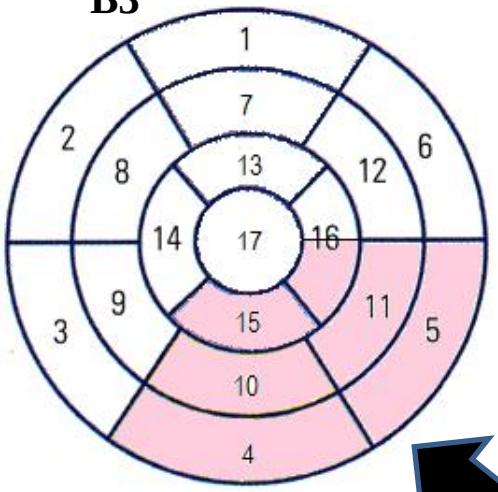
B1



B2



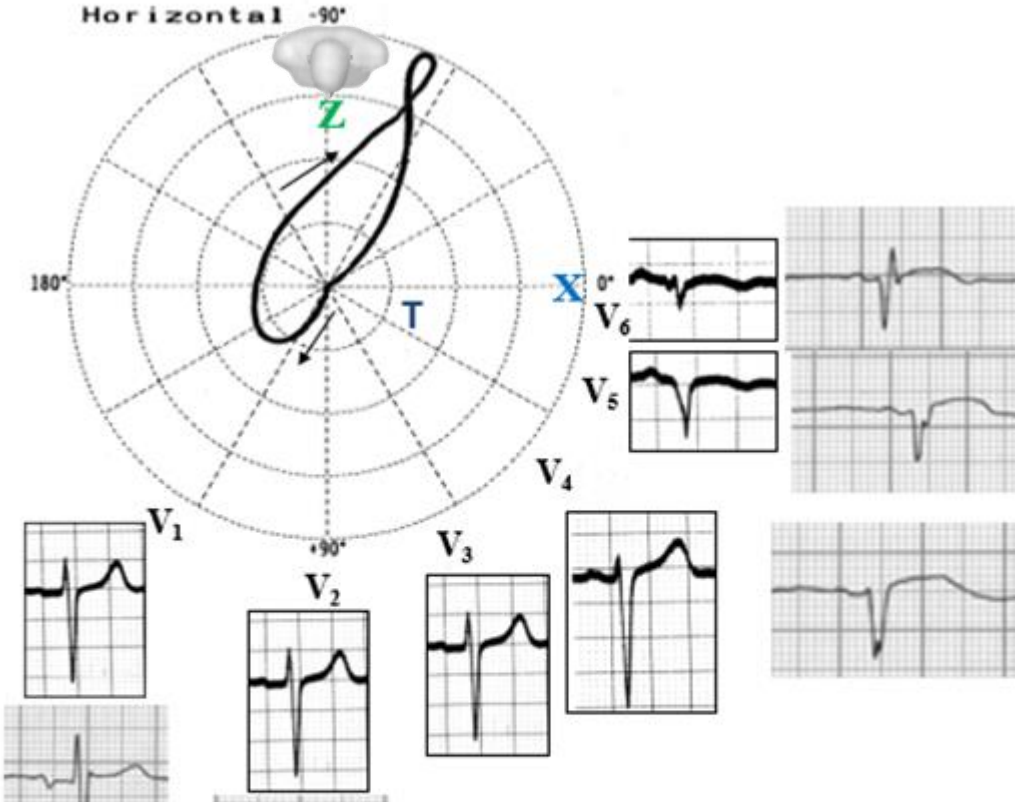
B3

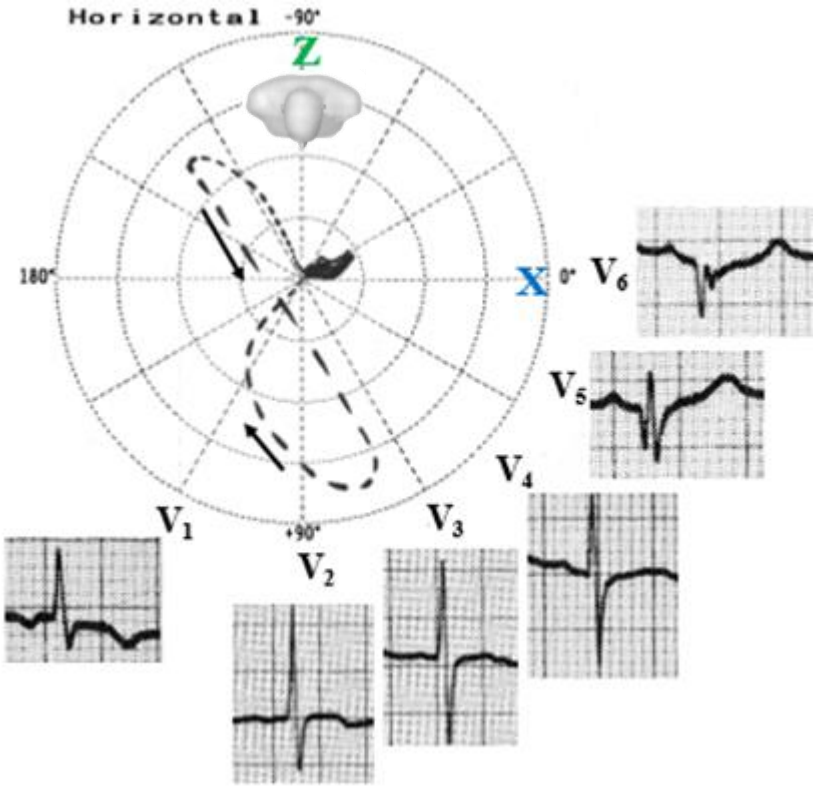


Inferolateral

I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

Lateral myocardial infarction (old laterodorsal)

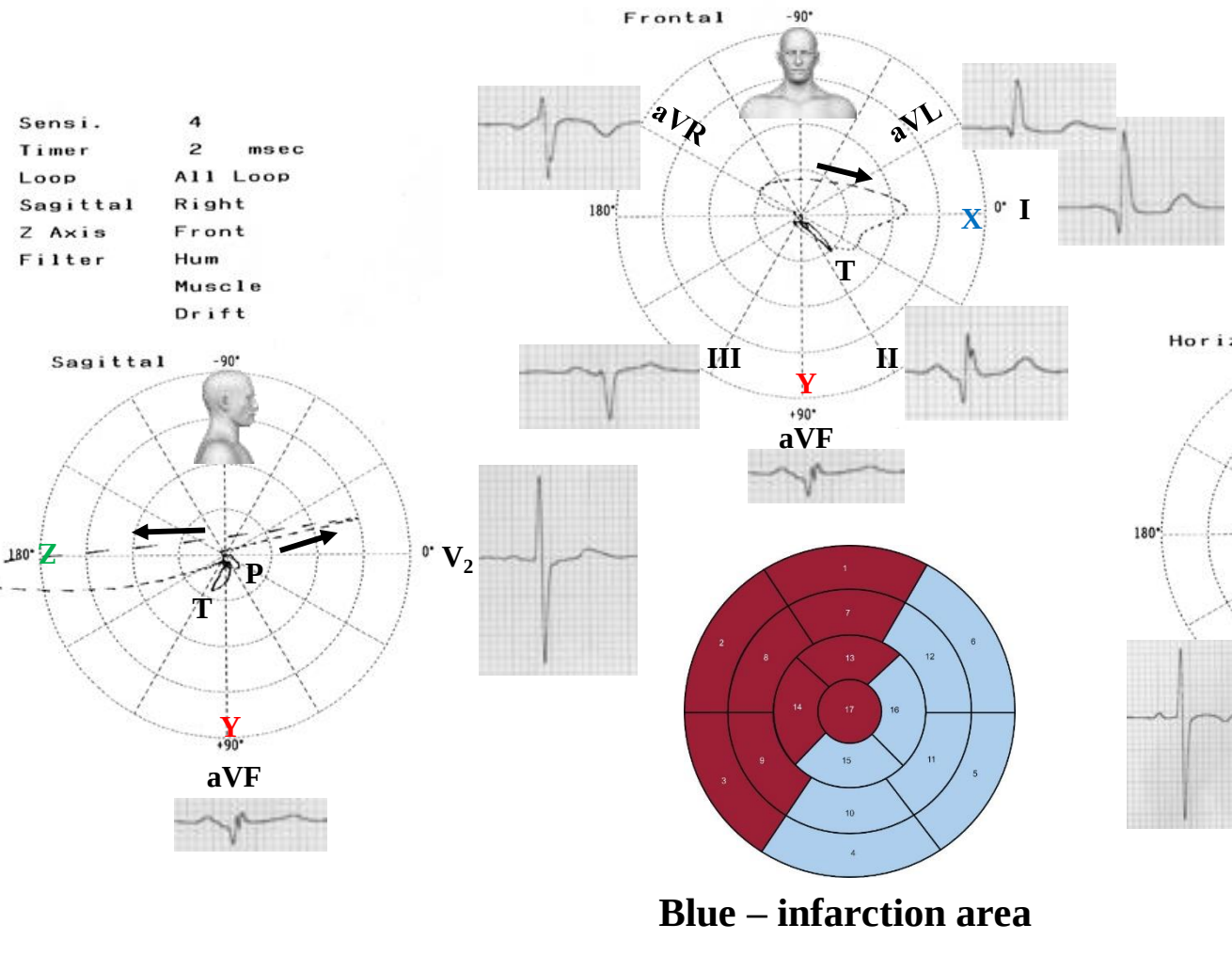




I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

Typical inferolateral myocardial infarction

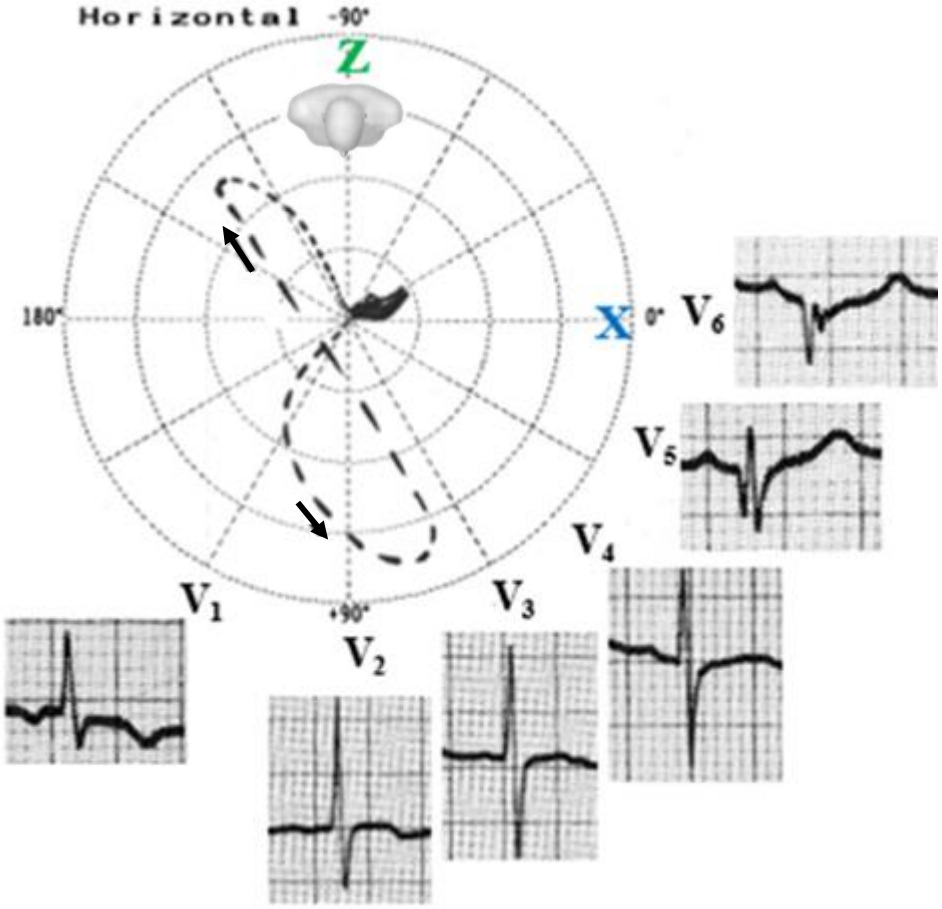
ECG/VCG correlation



Blue – infarction area

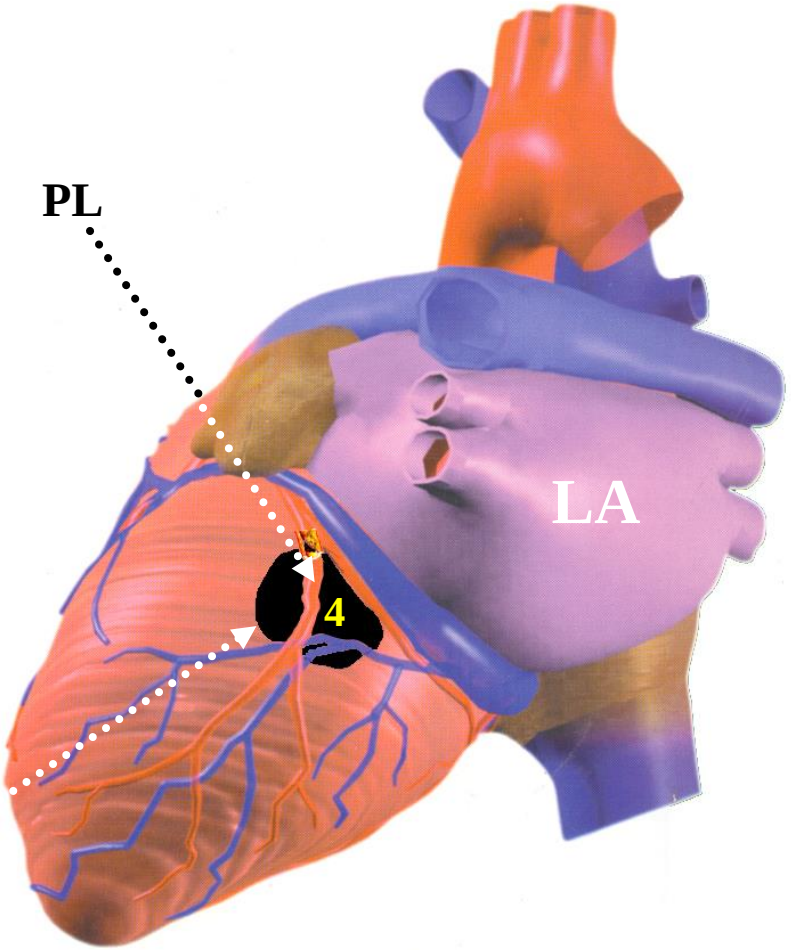
Lateral MI de Luna criteria

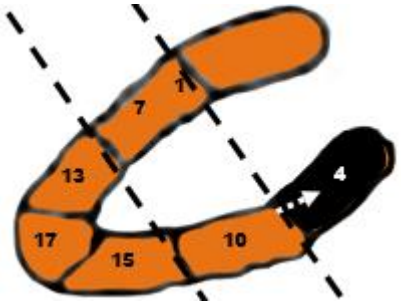
R/S ratio in V1 ≥ 0.5
R amplitude in V1 > 3 mm
Both have very high specificity and lower but very acceptable sensitivity for lateral MI.



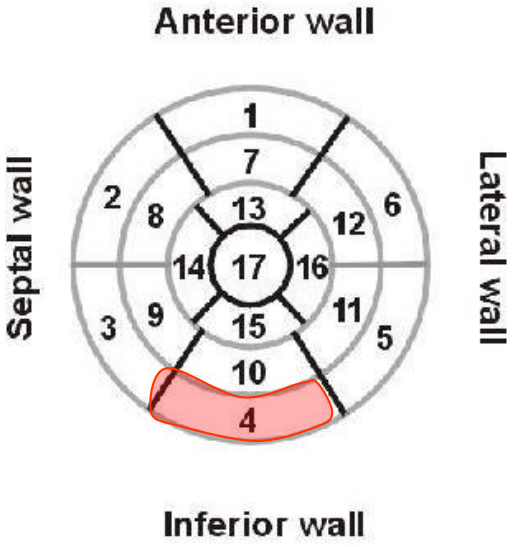
New criteria based on R waves in V2 or T waves in V1 to V2 do not discriminate between inferior and lateral MI.
The classical criteria (R/S amplitude ratio ≥ 1 and R duration ≥ 40 ms in V1 attain very high specificity but much lower sensitivity than the new criteria (**de Luna 2008**).

Left posterior oblique view of the heart in the old days called strictly dorsal MI



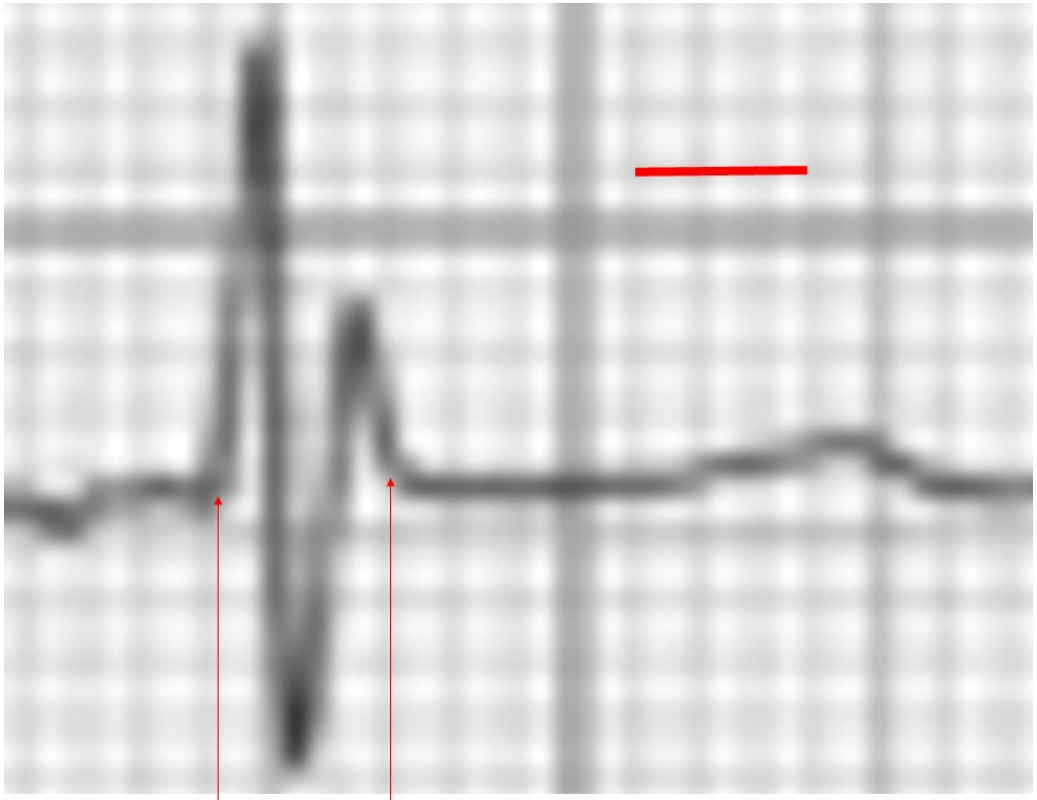


4 inferobasal segment



PL: Posterolateral branch of the left circumflex artery.; LA: Left Atrium/; 4 inferobasal segment old dorsal. The dorsal wall and the high lateral does not exist. Additionally, Prominent R wave in V1 is due to the lateral MI and not to the involvement of inferobasal segment of inferior wall (old dorsal or posterior wall) (**Goldwasser 2015**).

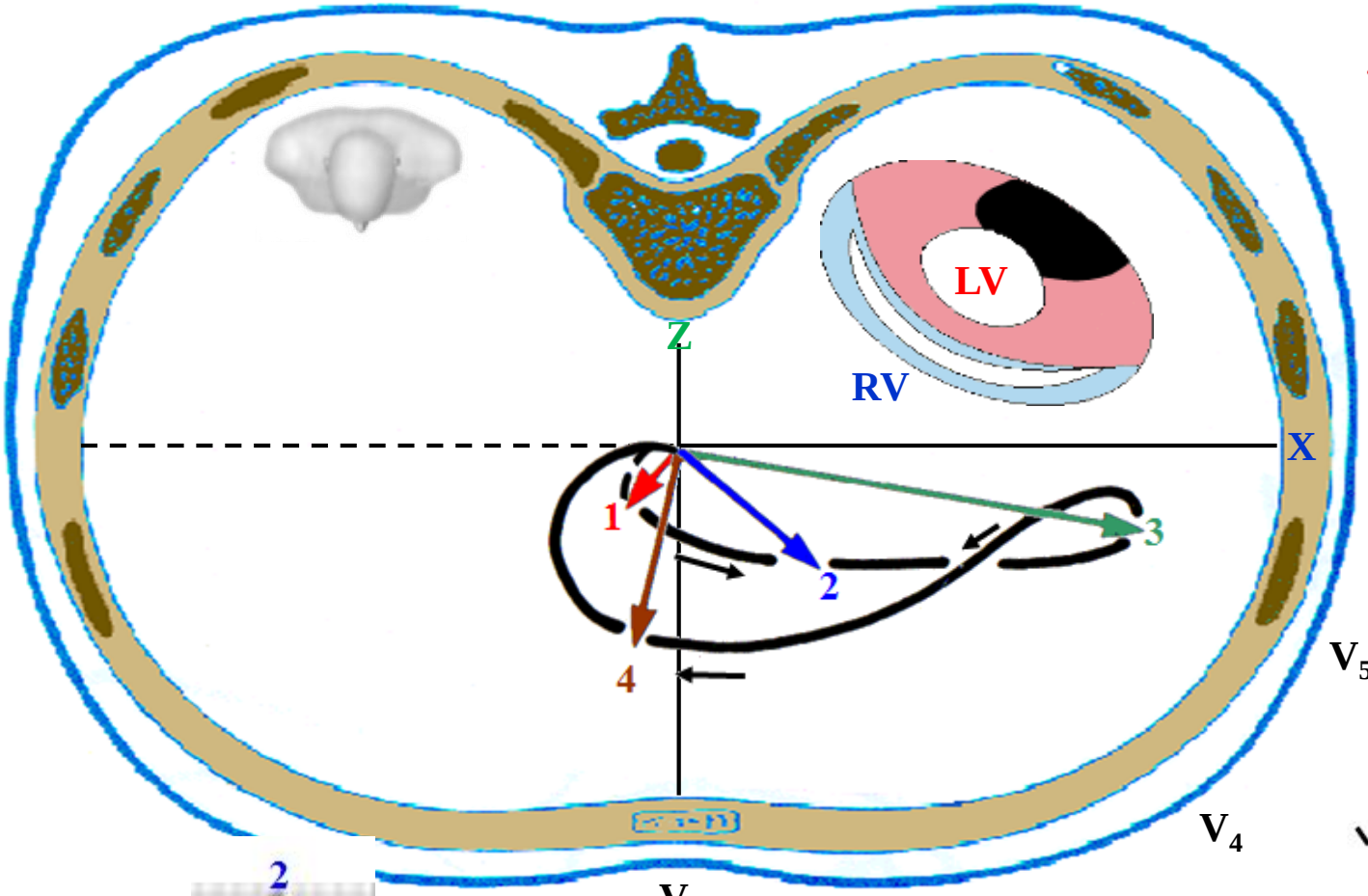
Question: In the ECG-2 can we make the diagnosis of incomplete or complete right bundle branch block based on the triphasic QRS (RSr') pattern of V1?

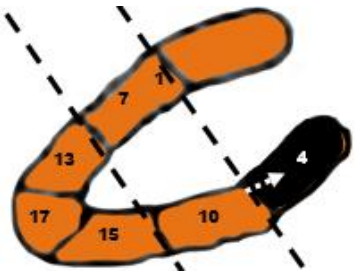


Answer Triphasic QRS complexes that remind IRBBB of the RSr', rSr, rSR' or rsR' type in V_3R and V_1 appear in ≈ 40% of the cases in the in the old days called strictly dorsal myocardial infarction (currently called basal inferior correspondent to segment 4). This infarction affects only the middle and final portions of the QRS loop between 30 and 100ms, the second half of the QRS loop in the horizontal plane.

Vector 1 (**red** arrow) or middle septal vector and the second one (**blue** vector of the low portion of the septum) are not affected. A mild anterior dislocation of the vector of the free walls (vector 3 **green** arrow) is observed and important anterior dislocation o vector 4 (basal. **Brown** arrow). This explains the triphasic pattern in V3R/V1 observed in ≈ 40% of cases: pseudo IRBBB. **See next slide**.

Vectorial representation of ventricular activation in inferobasal infarction in the Horizontal Plane





This pattern correspond in the in the old days called strictly dorsal myocardial infarction (currently called basal inferior correspondent to segment 4).

Myocardial ischemia and another element present in the MCH. It is secondary to microcirculation disease, decrease of vasodilator capacity, systemic compression of septal and subepicardial vessels, fall of pressure in aorta root, difficulty in coronary filling by hypertrophy, CAD (> 50 yo), and excessive increase of mass and subsequent offer/demand disproportion. Prominent deep-narrow Q waves on ECG are considered characteristic of HCM (**Rao 2011**) and with importance in early diagnosis Abnormal Q waves may mimic MI and at times reflect septal hypertrophy. Of the 200 consecutive patients with HCM who underwent CMR imaging, 10 male and 8 female patients had deep Q waves. Deep Q waves were more prevalent in females with HCM than in their male counterparts (**Ohmoto-Sekine 2007**). Of the 18 patients with deep Q waves, maximum wall thickness was localized at the basal anterior wall or the mid-ventricular septum in 90 of the 10 male patients and 75% of the 8 female patients. In both sexes, the Q wave distribution pattern of I and aVL and of II and aVF indicated location of maximum hypertrophy at the mid-ventricular septum in 75% of the 8 patients with the former pattern, and at the basal anterior wall in 9 (90%) of the 10 patients with the latter pattern. Diagnostic deep Q waves were detected more frequently in females with HCM than in their male counterparts. In HCM with deep Q waves in limb leads, morphologic and ECG analysis showed similar features in both sexes. Abnormal Q-waves observed on ECG are divided in MI Q-waves and non-MI Q-waves. This last group is divided in transient and permanent non-MI Q-waves. Transient abnormal Q waves are defined as abnormal Q-waves, which disappear within ten days. They are most often seen in patients with CAD but are also seen in other conditions (**Zhang 2010**) such us advanced hyperkalemia, septic shock, acute pancreatitis, localized metabolic and electrolyte disturbances and hypothermia.

The numerous causes of pseudo-infarct patterns

Since the introduction of cardiac plasma troponin measurements, a significant number of patients were seen with chest pain, elevated troponin levels but no significant coronary artery disease. Pulmonary embolism, aortic valve disease, myocarditis, sepsis, trauma, arrhythmias, stress cardiomyopathy and dilated cardiomyopathy stand among possible causes for this syndrome. In some cases, myocardial strain could be the mechanism underlying this phenomenon, since it is known that the stimulation of stretch-responsive integrins may lead to the release of cardiac troponin I. In the present text, a case is made in favor of classifying this syndrome, of chest pain with increased values for plasma cardiac troponin, with or without ECG changes, in the absence of definite MI or CAD, as pseudo MI (PMI). This constitutes a new definition for a concept with decades, formerly centered on clinical and ECG changes mimicking infarct. The case is based on the search of scientific truth, on avoidance of unnecessary cardiac examinations, on avoidance of unnecessary drug therapy and on avoidance of unnecessary legal liability. PMI should be seen as a working diagnosis, since a more definitive diagnosis can be reached at all time. It should also be seen as a heterogeneous group of patients - several different diseases and conditions can lead to this phenomenon. But it must certainly not be seen as a benign condition, since published studies point in a totally different direction. The following are the main causes of PMI.

I. In **left ventricular hypertrophy**, there is often a QS deflection or poor R-wave progression in the right precordial leads that suggests anterior MI. The secondary ST-segment elevation in these leads may be mistaken as a current of injury. (**Edhouse 2002**)

II. **Chronic Obstructive Pulmonary Disease (COPD):** In pulmonary emphysema, the R waves in the right precordial and sometimes mid-precordial leads become quite small or are absent, suggesting anterior MI. These QRS changes are explained by the vertical displacement of the heart secondary to a low-lying diaphragm and the intervention of hyperinflated lungs. In patients with emphysema, QS pattern appears occasionally from V1 to V4, offering a false image of anteroseptal MI . The QS pattern in the right and middle precordium may be present in the Cor Pulmonale with bronchospasm, being absent if there is no airway obstruction. The low position of the diaphragm leads the heart to a relatively inferior position in relation to the exploring electrodes of the precordium, manifesting by images of the rS or even QS type, which may be confused with anteroseptal MI. The differential diagnosis is made taking into account the typical manifestations of the emphysema in the ECG: 1) "P pulmonale", i.e. of great voltage in II and aVF (> 2.5 mV); 2) SAP with shift to the right beyond + 65º (negative P wave in aVL); 3) Shift of SÂQRS to the right (+120º to +180º). Possible significant shift of SÂQRS to the left; 4) Occasional positive Leo Schamroth's sign: SAP, SAQRS and AT with axes in +90º, with isodiphasic rs recorded in DI, without visible P or T waves.; 5) Universal low voltage of QRS, caused by the electrodes being pushed away from the heart by hyperinsuflated lung, and by scant pulmonary tissue conductivity; 6) Occasional SI Q3 or S1, S2, S3 pattern; 7) Significant S waves in the left precordium (V5 and V6) by shift of the transition area to the left (S wave of V6 of more than 5 mm); 8) The VCG shows in the HP, RVE of type C or special: 1) QRS loop with counterclockwise rotation or in 8 in the HP; 2) Predominant area of the QRS loop located in the right posterior quadrant (by the hypertrophy predominant in the RV outflow tract); 3) Area of the QRS loop located in the right posterior quadrant > 20% of the total area.(**Catemole 2006**)

III. **Pneumotorax:** The pseudo-infarction pattern may be seen in patients with pneumothorax. The voltage of the QRS complex may be reduced. QS deflection may appear in the right precordial leads (**Brywczynski 2013**).

IV. In **acute pulmonary embolism,** the Q waves in lead III (as part of the S1Q3 pattern), and sometimes in lead aVF, that are accompanied by ST-segment and T-wave changes are often interpreted as inferior MI. QS complexes with ST-segment elevation may occasionally develop in these leads and mimic acute anterior myocardial infarction. From the electrocardiographic point of view, the APE may cause ECG modifications that may be confused with anteroseptal MI. These modifications are:1) T wave inversion from V1 to V3 and more rarely from V1 to V4. This manifestation is quite frequent in APE and it is estimated that it is present in 40% of cases. On the other hand, the phenomenon appears to be persistent (it may last 40 days).2) Discrete ST segment elevation in the same precordial leads. This phenomenon is found less frequently (11%). 3) Association of both (ST-T) present in 54%. The ST-T segment alterations found in acute pulmonary embolism, are attributed to RV strain due to abrupt chamber enlargement, as well as subendocardial ischemia secondary to coronary perfusion pressure fall by shock and the always-present hypoxemia of arterial blood. 4) Qr or Qs pattern in V3R, V1 and V2, simulating middle-septal necrosis, reinforcing even more if present, T wave inversion in these leads. 5) R/S ratio regression along precordial leads. Normally, the R/S ratio in the precordium from V1 to V4 is progressive, i.e. the r wave increases its voltage in comparison to the S wave. The regression phenomenon of the R/S ratio is due to selective hypertrophy of the posterior basal region of the RV (vector 3), with r wave being observed as decreasing from V1 to V3. The anteroseptal MI may be ruled out if the SAQRS is concomitantly to the right or the S wave of I is > 1 mm (**Hsieh 2016**).

V. Myocardial fibrosis in dilated cardiomyopathy

VI. Myocardial fibrosis in Progressive muscular dystrophy (Secchi 1982)

VII. Myocardial fibrosis in Friedreich's ataxia (Calvo Iglesias 1995)

VIII. Myocardial fibrosis in Scleroderma or systemic sclerosis (Nordin 2014)

IX. Myocardial fibrosis in Amyloidosis (Murtagh 2005)

X. Tumors of the heart (Cheon 2014)

XI. Cardiac pseudo tumor (Chao 2009)

XII. In HCM, abnormal Q waves are often seen, especially in the left leads. These Q waves have been attributed to ventricular septal hypertrophy. Possible mechanisms of permanent non infarction Q-waves are: Loss of viable myocardium, altered distribution of myocardial mass, altered sequence of depolarization, and altered position of the heart. Infarction Q-waves are characterized by Q duration≥40 ms (in VCG ≥ 30 ms) and with ST-T abnormalities which are stronger events predictors and total mortality than isolated Q-wave abnormalities (**Chatterjee 2010**). Permanent non-infarction Q-waves are characterized by Q-wave duration ≤ 35 ms (those of infarction≤40 ms), "clean" and deep Q-wave aspect (those of truly infarction present notches and are usually accompanied of injury current and ischemia). Additionally they may be observed in children and young people (those of infarction are found in adults and elderly people), are frequently asymptomatic and serum enzymes and troponin are normal (those of infarction in the acute phase, with increased CKMB, TGO, DHL and Troponin). In ≈10% of HCM cases, very wide R waves in V1 and aVR associated to deep and "clean" Q waves in V5 and V6 and/or in inferior leads are observed as a consequence of augmented first left septal vector. Prominent R waves in intermediary precordial leads are observed in ≈80% of cases of Ap-HCM subtype (**Engler 1979**).

XIII. Complete left bundle branch block QS deflections are often seen in the right precordial leads in patients with **complete left bundle branch block** in the absence of MI (**Wegmann 2015**).

XIV. Incomplete LBBB QRS duration between 110 and 119 ms in adults, between 90 and 100 ms in children 8 to 16 years of age, and between 80 and 90 ms in children less than 8 years of age (**Surawicz 2009**).

XIV. Left anterior fascicular block is occasionally associated with small Q waves in the precordial leads that mimic anterior MI. Certain LAFBs may simulate anteroseptal MI by causing a QS pattern in V_2 and V_3 or r wave with decreasing voltage from V_1 to V_3 .Sometimes, a qrS pattern is observed in V_2 and V_3 that may be confused with anteroseptal MI.For a differential diagnosis, it is enough to move the exploring electrodes of V_1, V_2 and V_3, one intercostal space below. When the pattern is secondary to LAFB it disappears, while if it is a consequence of anteroseptal MI it remains. By moving the exploringelectrodes of V_1, V_2 and V_3 one intercostal space above, in the case of LAFB the pattern intensifies or appears (**Elizari 2007**). Usually, the phenomenon is observed in patients with a vertical heart: ectomorph, with a narrow chest and emphysematous. On the other hand, the LAFB may mask an anteroseptal MI causing the appearance of an initial r wave from V_1 to V_3. This fact occurs when the exploring electrodes of the right precordial leads are placed slightly below their correct position. The doubt is cleared by placing the electrodes in a position slightly superior. In this case, the initial r wave that masked the anteroseptal MI disappears. The VCG may be a valuable auxiliary method, since in the LAFB, the QRS loop in the HP always presents counterclockwise rotation. The appearance of the loop in "eight" suggests associated anteroseptal MI and in this case, the initial 20 ms vector is always heading backward, which does not occur in LAFB, where the initial vectors are heading to the front and the right, and only after 30 ms the direction becomes posterior.

XV. Wolff-Parkinson-White syndrome The δ waves in Wolff-Parkinson-White syndrome are frequently interpreted as abnormal Q waves of MI. Type B WPW with anomalous anterior pathway (pseudo anterior MI) (**Khan 2000**).

XVI. Right End Conduction Delay by the divisions of the right bundle branch (ECD) (Pérez-Riera 2012)

XVII. Pheochromocytoma may be associated with striking ECG changes mimicking ischemic heart disease (**Cheng 1976**)

XVIII. Subarachnoid hemorrhage and other **intracranial hemorrhage:** Increased release of catecholamines from local nerve endings in the heart may mediate these cardiac abnormalities. Transient severe coronary vasoconstriction leads to ischemia followed by postischemic ventricular failure and subendocardial myocardial damage. In addition, a direct cardio toxic effect of catecholamine may cause the development of subendocardial damage **(Elrifai1996**). Autopsies have revealed areas of subendocardial myocardial lesions, called contraction band necrosis, in the hearts of patients with subarachnoid hemorrhage. The myocardial damage resembles lesions produced in animal experiments by infusion of norepinephrine. The characteristic pattern of myocardial lesions has suggested that damaging catecholamines are released from intramyocardial nerve endings rather than from the general circulation.

XIX. Hyperkalemia (Burris 1980). T-waves become abnormally tall, peaked/pointed, symmetrical, with narrow base: "Eiffel tower T-waves" or in "desert tend T-waves" (**Light hyperkalemia** 5.5-6.5 mEq/L); **Moderate hyperkalemia** (6.5-7.0 mEq/L) shows P wave become broader and flatter (slow interatrial conduction): reduction in P wave amplitude, prolonged PR interval (first degree AV block. R wave height decreases, QRS complexes become wider and ST segments have elevation in some leads and depression in others ST-segment deviation simulates "acute injury" pattern or "dialyzable injury current". Brugada phenocopy; **Severe hyperkalemia** (7.0-7.5 mEq/L) we observe further widening and distortion of QRS occurs non specific intraventricular conduction pattern, prolonged QT interval, and premature ventricular beats become frequent. Finally, extreme hyperkalemia (>7.6 mEq/L) is characterized by absent P waves, frequent escape beats. Sinoventricular rhythm The combination of an irregular rhythm The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded). Absent P weave may simulate atrial fibrillation. atrioventricular block, very broad and bizarre QRS complexes. ventricular tachycardia. Ventricular fibrillation or ventricular asystole with potassium concentration above 12 to 14 mEq/L.

XX. Acute pericarditis

	Coronary artery disease	Pericarditis
Number of involved leads	Lesser (segmentar)	More (diffuse) extensive
Intensity of the phenomena	More	Lesser
Reciprocal effect	Present	Absent
Prolongation of QRS complex and shortening of QT interval in ECG leads with ST-segment elevation	Present	Absent (**Rossello 2014**)

XXI.Diabetic ketoacidosis with normokalemia: eventually it is observed acute inferior pseudoinfarction pattern (**Aksakal 2009**).

XXII.After intravenous flecainide (Nakamura 1998)

XXIII.In postoperative period after cardiac transplantation (Aziz 2004)

XXIV.Hyperparathyroid heart mimicking acute myocardial infarction (Ker 2009)

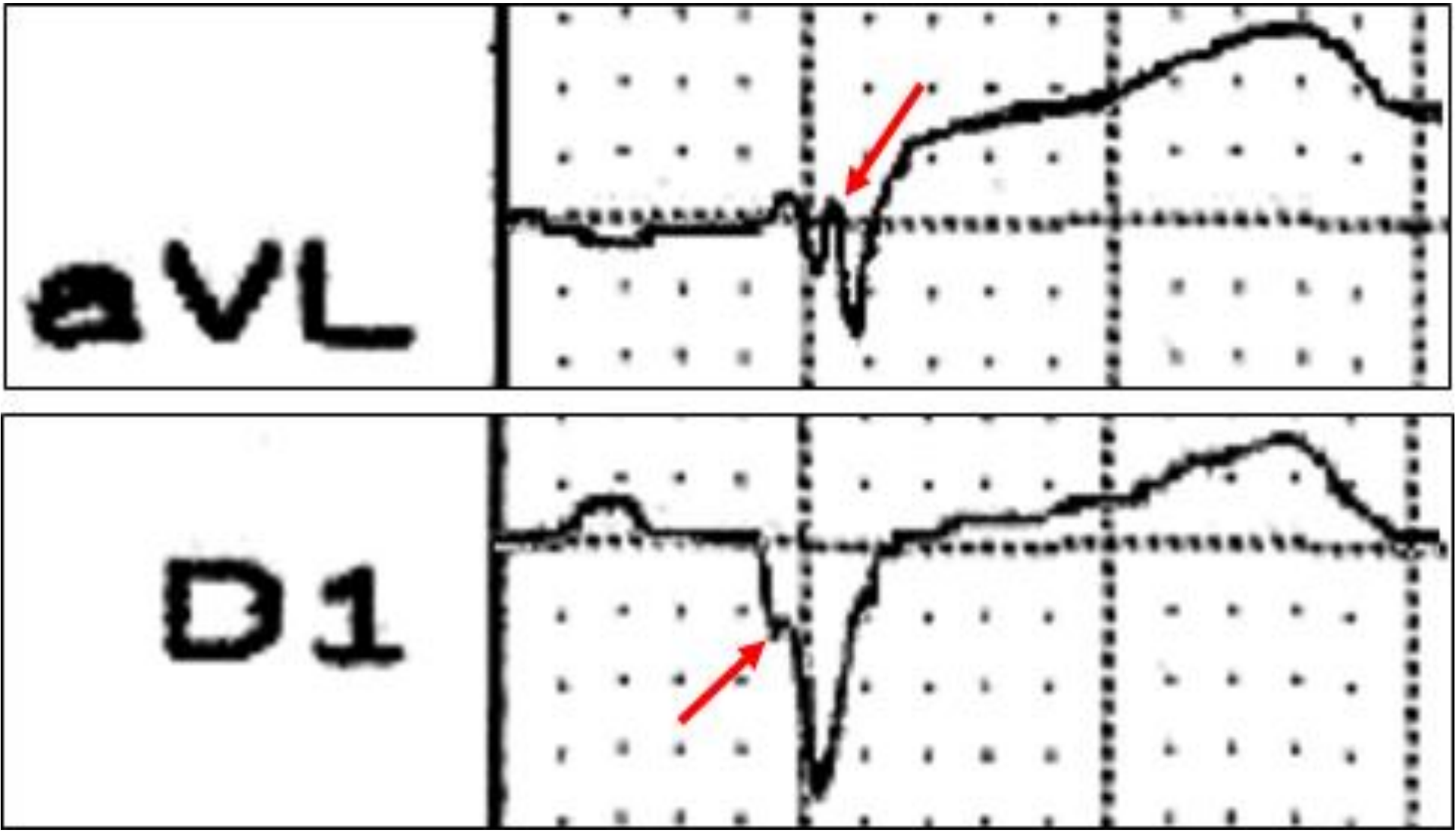
Fragmented (fQRS)

Myocardial scar causes heterogeneous ventricular activation, is a substrate for reentrant ventricular arrhythmias and is associated with poor prognosis. Fragmented QRS (fQRS) on 12-lead ECG represents myocardial conduction delays due to myocardial scar in patients with CAD. The presence of a fragmented QRS (fQRS) complex on a routine 12-lead ECG is another marker of depolarization abnormality together with SAECG. 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. fQRS has also been defined as a marker of arrhythmogenic right ventricular dysplasia/cardiomyopathy and Brugada syndrome. In Brugada syndrome, the presence of fQRS predicts episodes of ventricular fibrillation during follow-up.

QRS fragmentation (fQRS) characteristics

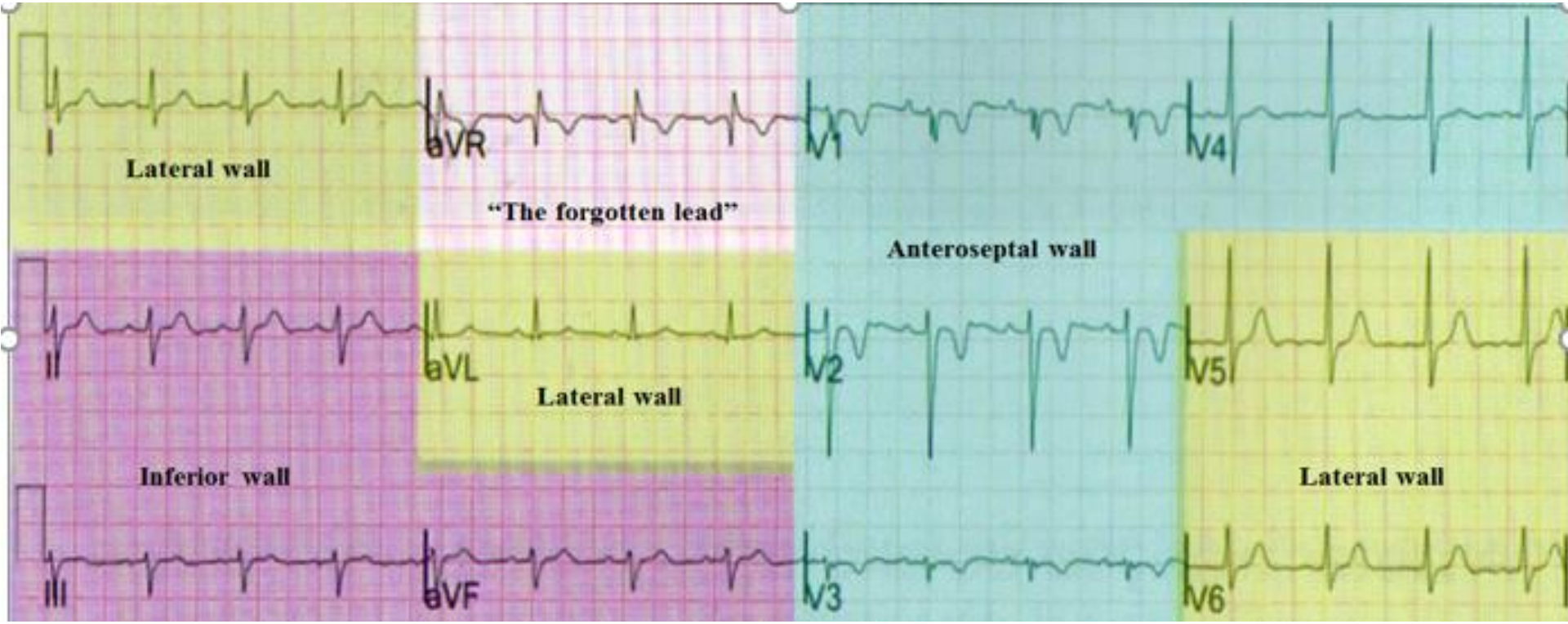
1. QRS duration < 120ms;
2. The presence of fragmentation within the QRS complex, with ≥4 spikes (**Zhang 2014)** in a single lead or ≥ 8 spikes in leads V1, V2, and V3 (**Morita 2008; Calò 2016**).
3. Presence of an additional R’ wave: >1 R’ in two contiguous leads, corresponding to a major coronary artery territory of ECG: inferior (II, III, and aVF), anterior (from V_1 to V_4) or lateral (I, aVL, V5, V_6) (**Das 2006**).
4. Notching on ascendant ramp, on top or in the descending ramp of R-wave;
5. Notched or slurred in the nadir or upstroke of the S wave;
6. Various RSR' in the mid precordial lead or inferior lead in either one or more right precordial lead or in more than one lead including all remaining standard ECG leads;

7. fQRS is a sign comparable to the epsilon (ε) wave seen in ARVC/D, indicating the presence of myocardial scar (**Varriale 1992**). Figure below.



Typical example of fragmented QRS complex (f-QRS) notched in at least ≥ 2 contiguous lateral leads (I and aVL). Notched in the descending ramp of S wave in I, and RSR'S' polyphasic QRS pattern in aVL with normal QRS duration = 100 ms (< 120 ms).

The figure bellow shows the contiguous leads in the current clinical ECG walls of the heart.

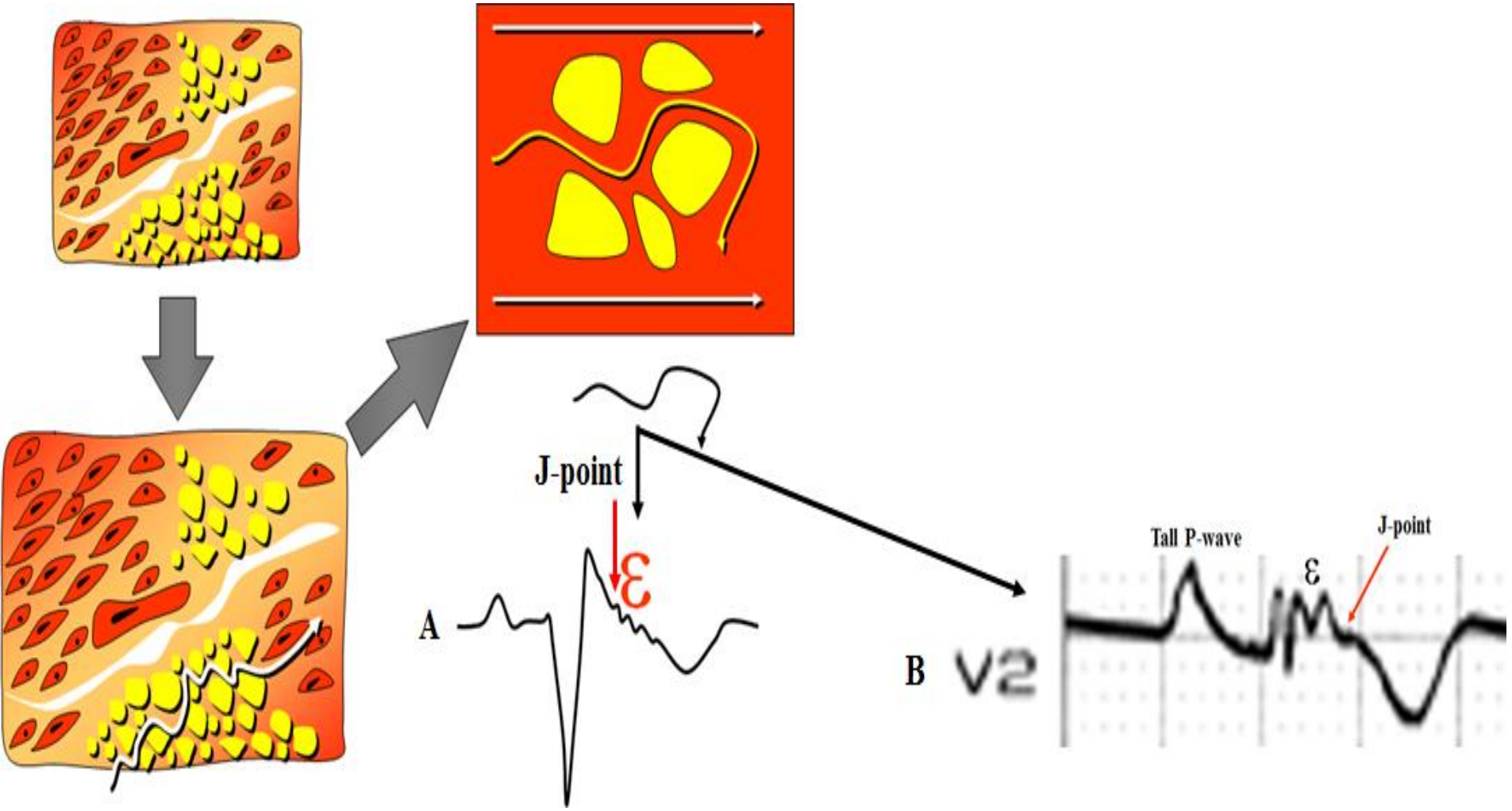


The figure shows the current walls of the heart, following the last consensus about a new terminology for LV walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography (**Bayés de Luna 2006**).

The dorsal or posterior wall and high lateral wall do not exist. The presence of prominent the R wave in V1 is due to the lateral MI and not to the involvement of inferobasal segment of inferior wall (old posterior wall) (**Goldwasser 2015**). We must abandon this wrong and old nomenclature.

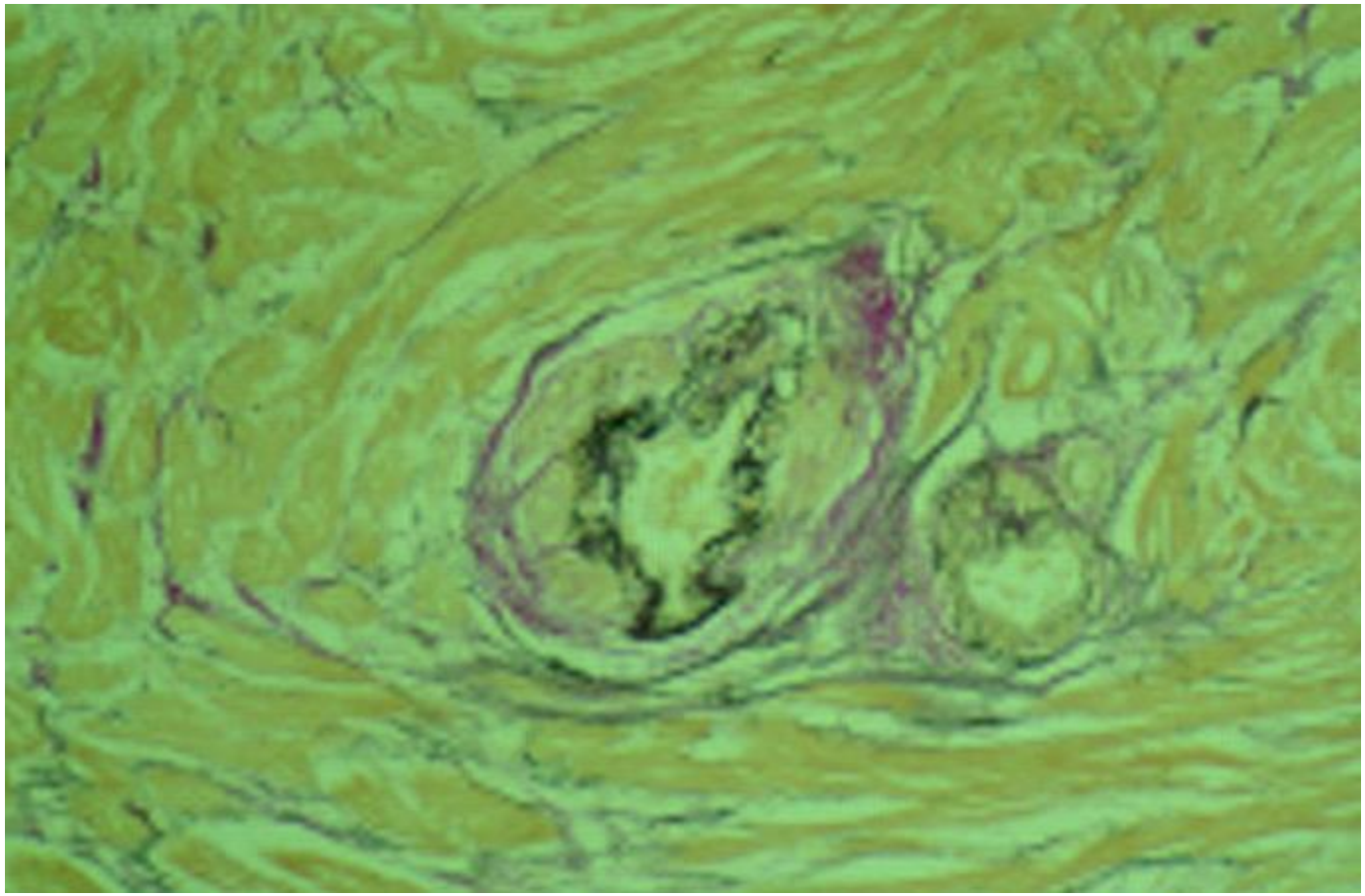
f-QRS of right precordial leads is probably the same ECG phenomenon as epsilon waves (**Peters 2015b**). In ARVD/C fQRS has a high diagnostic value similar to epsilon potentials by a highly amplified and modified recording techniques, such as right-sided precordial lead electrocardiography (R-ECG), higher right precordial leads (HR-ECG) (**Kukla 2012**) and Fontaine bipolar precordial lead electrocardiography (F-ECG) (**Peters 2008**). In real world practice, nevertheless, most ECGs available from ARVC/D patients and family members were obtained by using only the S-ECG. The fQRS is easily recognizable from S-ECG and they are much more common in ARVC/D patient when compared with control subjects: 51% of ARVC/D vs 26% in controls. In ARVC/D, fQRS is often seen in multiple leads (**Zhang 2014**). Such changes, however, are common in control subjects as well. In the latter, the QRS complex is w-fQRS (**Dechering 2013**). fQRS complex, with various morphology, has been described as a diagnostic criterion of ARVC/D. Since fQRS is also prevalent in other types of cardiomyopathies (both ischemic and non-ischemic) (**Das 2006; 2010**). fQRS is induced by radiotherapy in patients with breast cancer (**Adar 2015**), and in normal subjects, its use in ARVC/D diagnosis is limited. The figure of next slide shows the two admitted possibilities of location for ε waves:

A) ε waves after the J point; and B) ε waves before J-point.

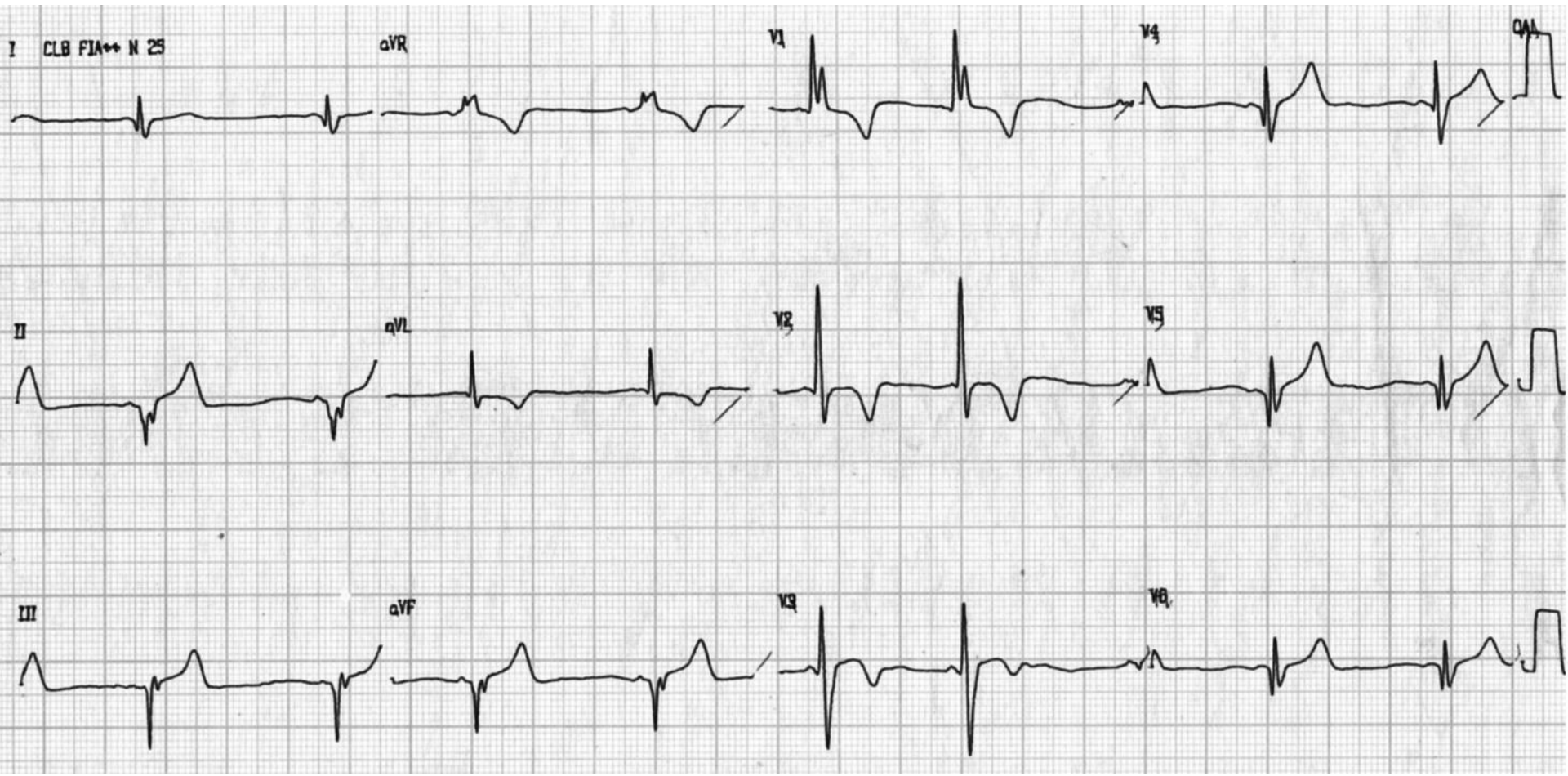


The figure shows the two admitted possibilities of ε wave location: A: Oscillations registered after J-point at the beginning of ST segment. B: Oscillations registered inside the QRS complex. In this case, eventually, ε waves are indistinguishable of fQRS. Additionally, residual myocytes (red color) entrapped within fibrous and fatty tissue(yellow) responsible for the dromotropic disturbance on the so called dysplasic triangle on subepicardial layer of right ventricle represented by the "zigzag" arrows pattern.

Myocyte disarray is a direct response to functional or structural abnormalities of the mutated sarcomeric protein in HCM, while fibrosis and small vessel disease are secondary phenomena unrelated to disarray, but modified by factors such as left ventricular mass, sex, and perhaps local autocrine factors (**Varnava 2000**).



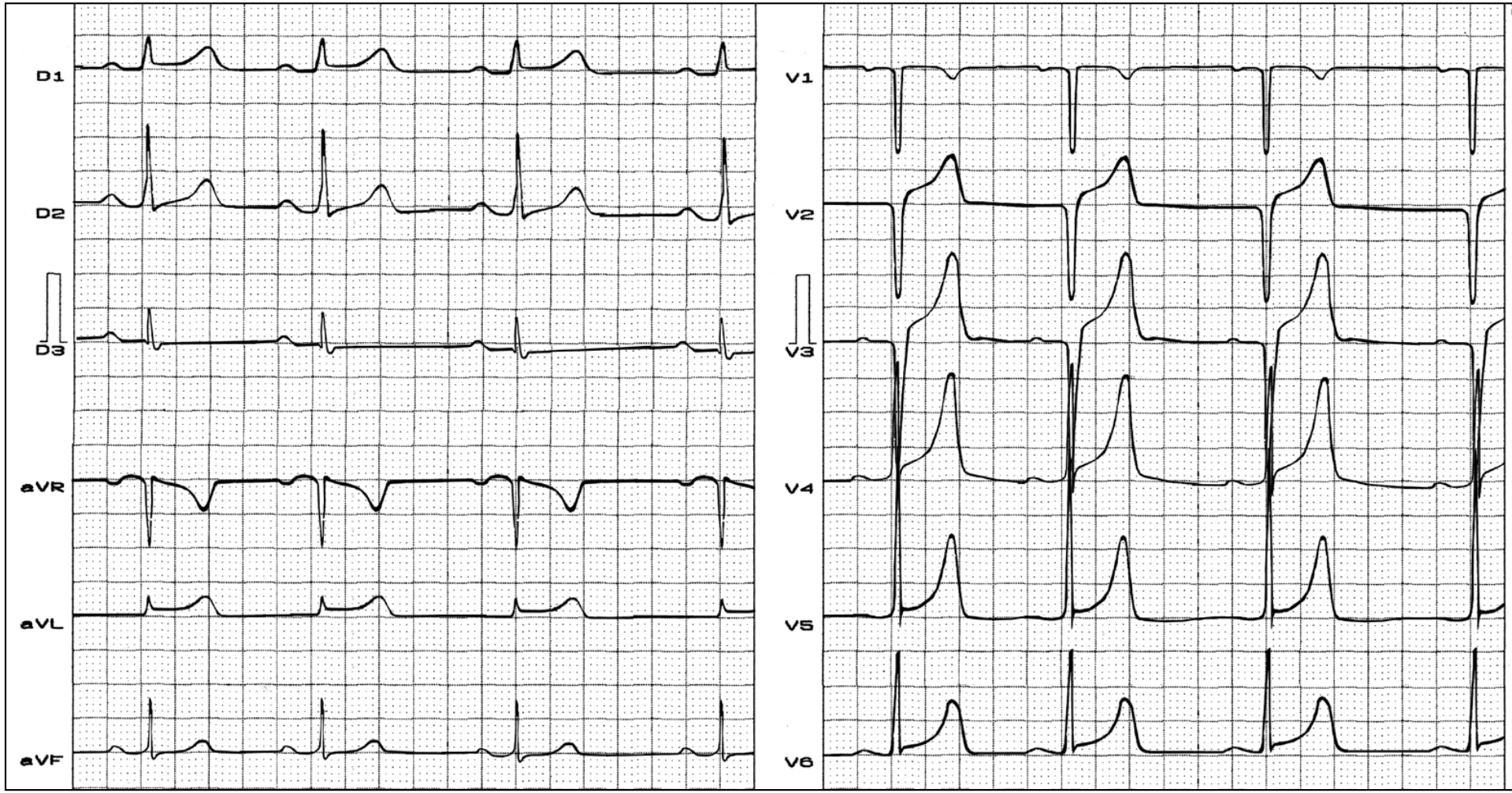
Dysplastic small vessel with no surrounding microscopic fibrosis (×10 objective) in a patient with HCM.



Male, 23-year-old patient, with history of tiredness at strain and syncope episode.

Extreme left axis deviation In HCM the QRS axis between 0 and –90º is observed in 30% of cases, pseudo infarction pattern in inferolateral wall, a very wide R waves in V1 and aVR associated to deep and deep, narrow ("dagger-like") Q waves in the lateral > inferior leads "clean" Q waves in V5 and V6 and/or in inferior leads, In ≈ 10% of cases in HCM is observed by > of septal vector 1 in 10%, LAFB (10%) with extreme shift of AQRS (beyond –30º), important Q waves of pseudo infarction, with less duration (<40 ms) and deep, Q waves in young patients with absence of MI history, arrhythmias: 85%, NS-SVT (30%), AF (10%), frequent premature ventricular contractions (>10/h) in 20%, isolated, coupled, (25%) polymorphic (20%), NS-VT and S-VT.

Name: BCA **Age:** 22yo **Sex:** Male **Race:** Black **Weight:** 82 kg
Height: 1.96m **Biotype:** Athletic Profession: professional basketball player **Date:** 2/09/2001



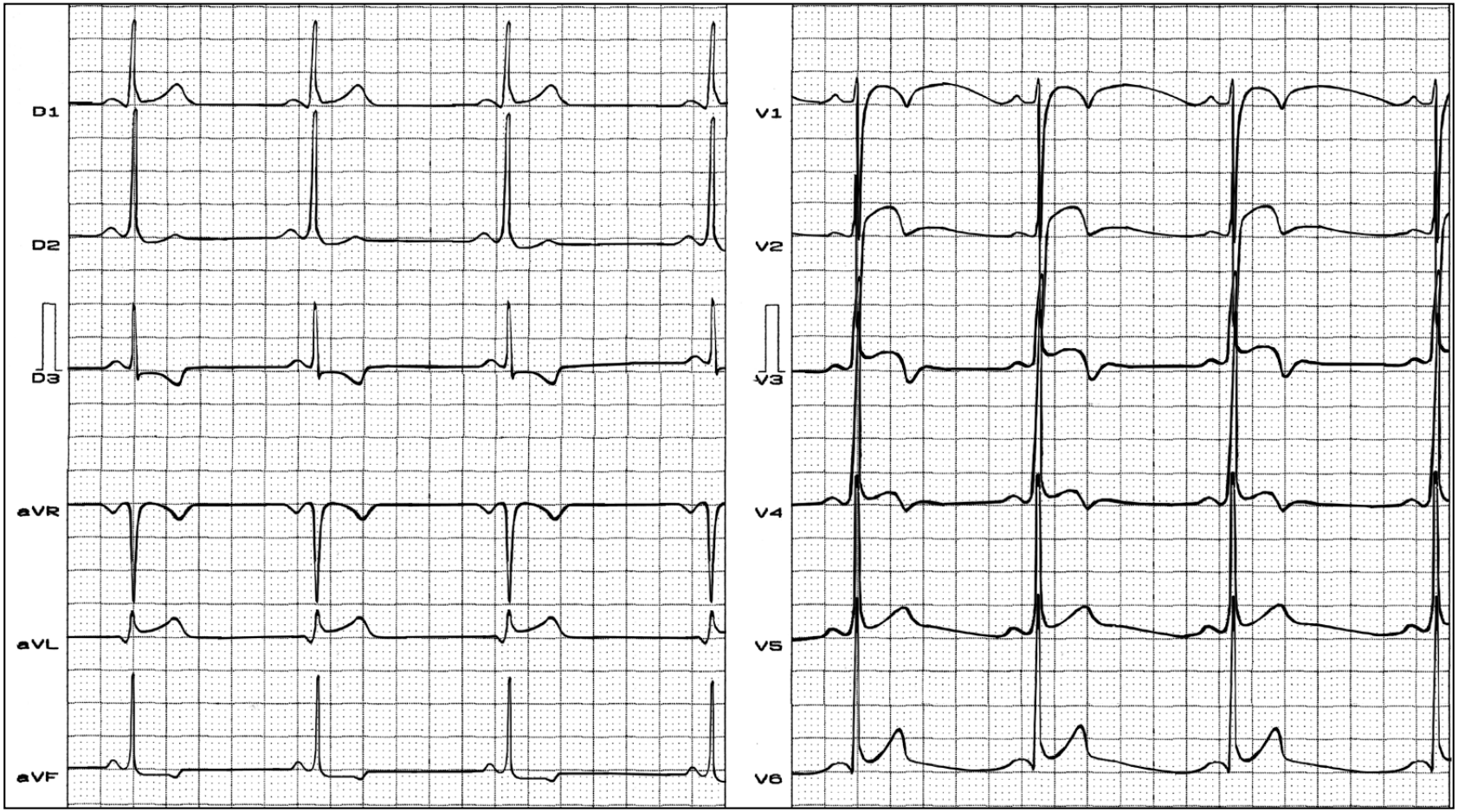
Clinical diagnosis: athlete's heart. Normal variant.

ECG diagnosis: sinus rhythm; HR: between 50 bpm and 57 bpm: phasic or respiratory sinus bradyarrhythmia; QS from V1 to V3: pattern of pseudo infarction in anteroseptal wall. Peaked T waves from V3 to V6. Normal X-rays of chest and echocardiogram.

Pattern of pseudo anterior infarction in an athlete, professional player of basketball with normal heart.

Pattern of pseudo injury and ischemia in anterior wall in an athlete, professional player of basketball with normal heart.

Name: BCW; **Age:** 24yo.; **Sex:** Male; **Race:** Black; **Weight:** 86 kg; **Height:** 2.02 m**; Biotype:** Asthenic; **Profession:** professional basketball player; **Date:** 05/01/1999



Clinical diagnosis: healthy patient. Tracing obtained in a periodical evaluation.

ECG diagnosis: sinus bradycardia, phasic sinus arrhythmia. Positive voltage criterion for LVE. SV_1 or V_2+RV_5 or V_6 >35 mm (Index of Sokolow Lyon). ST segment elevation from V_2 to V_6 and with negative T from V_1 to V_4. Early repolarization, pattern of pseudo injury and anterior subepicardial ischemia. Normal chest X-rays and echocardiogram.

Case report

Male, Caucasian, 41 years old, with history of chest pain and exhaustion in strain.

Personal antecedents: He mentions systemic hypertension, without treatment currently. He doesn't smoke or have diabetes.

Family background: His father died with 77 years old due to AMI? His brother died suddenly when he was 37 y/o during the sleep.

Physical: Cardiac auscultation: regular heart rhythm; HR=68 bpm + fourth heart sound without murmur. Normal pulmonary artery and limbs. BP=140/100 mmHg.

After the ECG was performed (attached), we also suggested performing Echo to rule out apical hypertrophic cardiomyopathy.

Echo: LV end diastolic diameter: 49 mm. LV end systolic diameter: 26 mm. Septal diastolic thickness: 14 mm. LV posterior wall diastolic diameter: 14 mm. Aorta: 29 mm; LA: 40 mm EF=78% Mass: 355 g.

Conclusion: moderate LV concentric hypertrophy. Moderate LV diastolic dysfunction. Heart valves with normal morphological aspect. Absence of gradient in the LVOT. Mild mitral reflux.

Supplementary tests

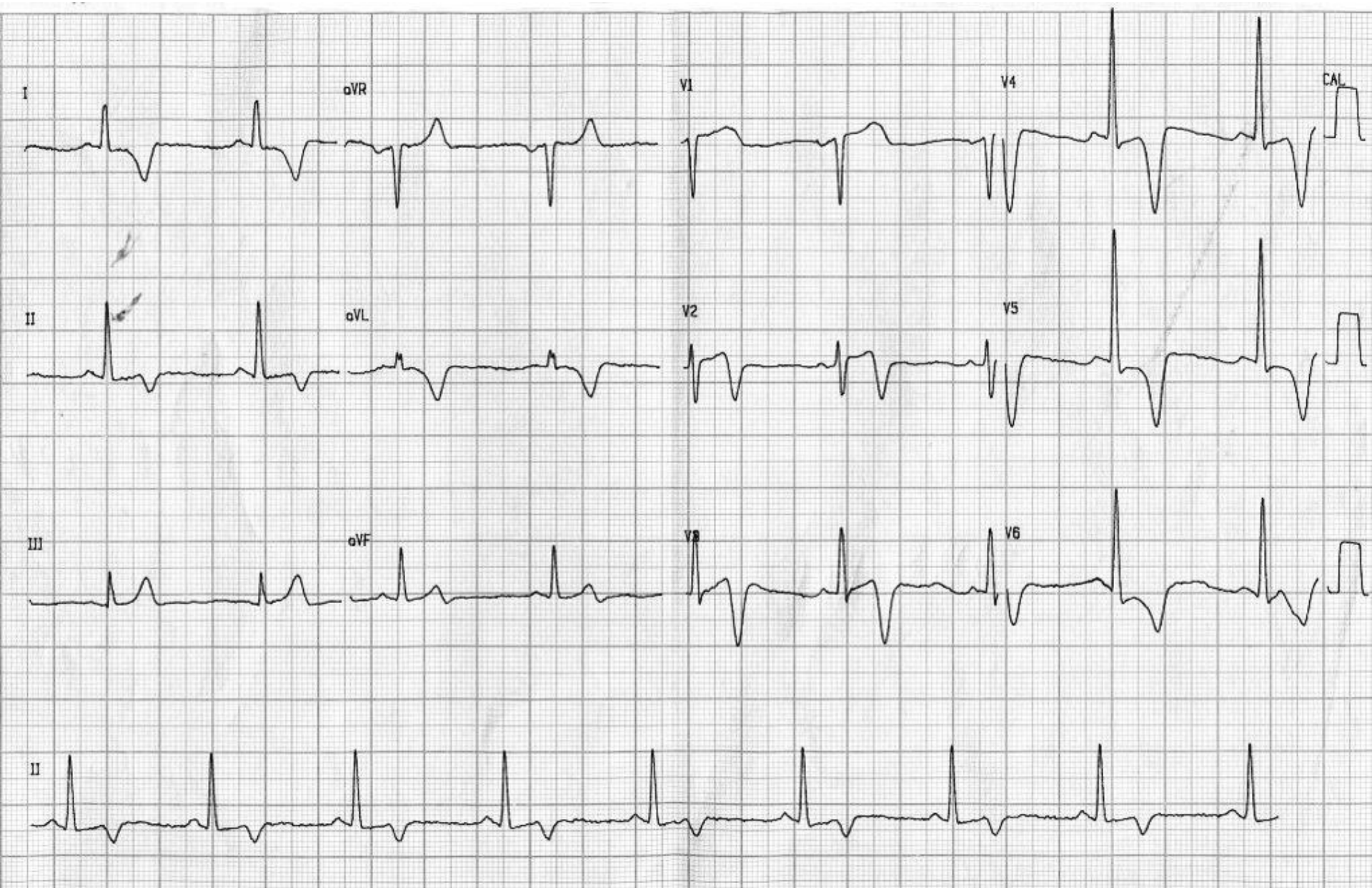
Several ECGs with the same morphology.

Holter monitoring: Sinus rhythm predominates asymptomatic isolated ventricular ectopic beats (210).

Absence of NSVT.

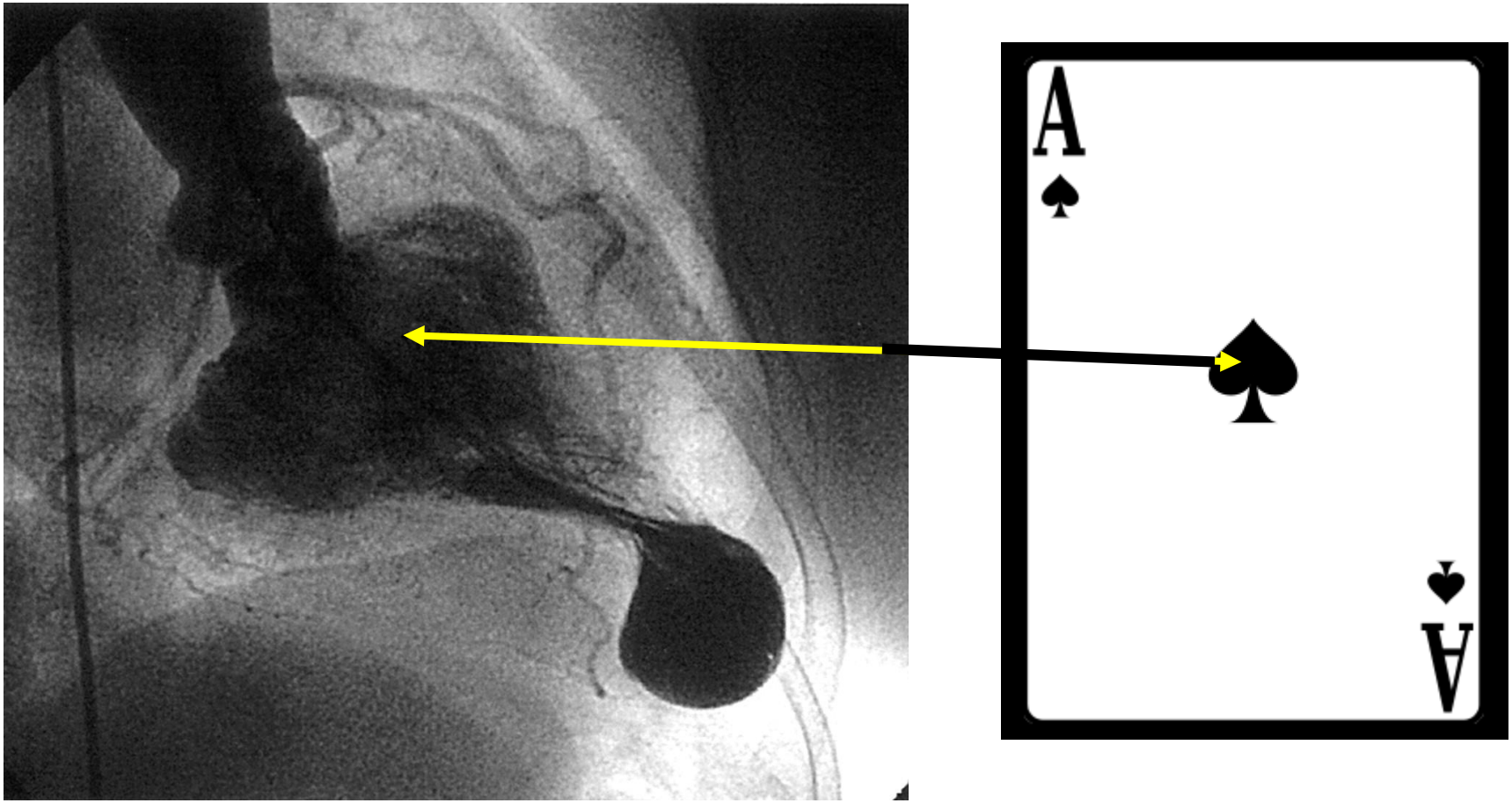
Left heart catheterization: Presence of myocardial bridge in the proximal $^{1}/_{3}$ of the LAD.

LV with asymmetrical hypertrophy with apical predominance (ventriculography shows spade-like morphology). Normal coronary arteries.


I
aVR
V1
V4
CAL
II
aVL
V2
V5
III
aVF
V3
V6
II

Left Ventriculography

The "ace-of-spades" sign on left ventriculography being pathognomonic (**Olearczyk 2008**).



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