57 Years old Man with ST segment elevation acute myocardial infarction (STEMI) complicated with cardiorespiratory arrest

Homem de 57 anos com infarto agudo e elevação do segmento ST complicado con parada cardiorespiratória

From Raimundo Barbosa Barros M.D.
Coronary Center Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza-Ceará-Brazil

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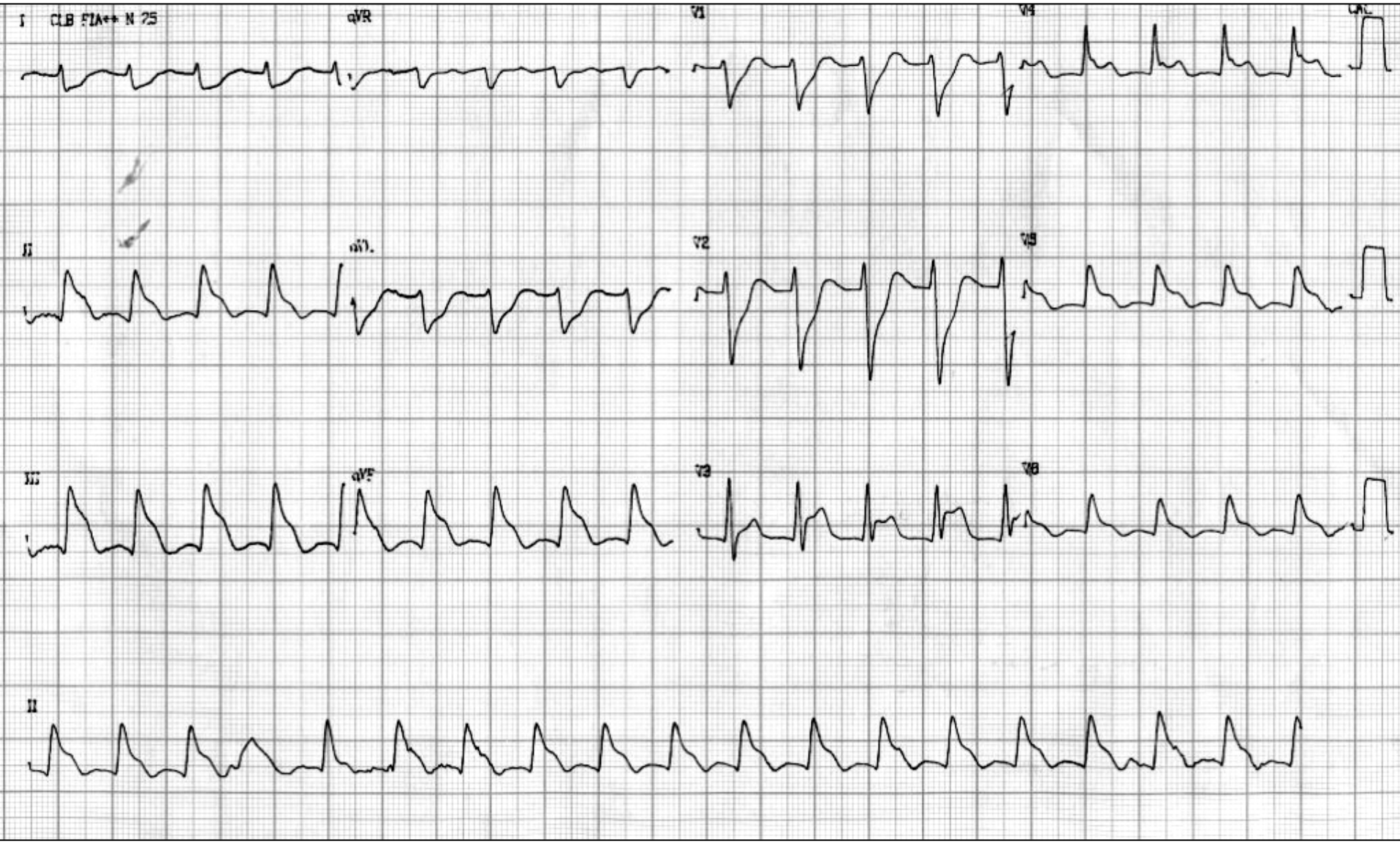
Prezado Andrés, este paciente, homem, 54 anos, hipertenso, deu entrada na emergencia em parada cardiorespiratória por fibrilação ventricular. Segundo familiares, antes do evento arrítmico o paciente relatou ,4 horas antes, dor retroesternal constrictiva com dispnéia seguida de vômitos.
Após manobras de reanimação o paciente foi enviado para o laboratório de hemodinâmica sendo submentido a angioplastia com colocação de stent e implante de Marcapasos.
Atualmente encontra-se sedado sob ventilação mecânica na UCO.Estes traçados apresentam várias aspectos interessantes !!!

Qual é a artéria culpada?
Qual o ritmo de II longo?
Um abraço
Raimundo

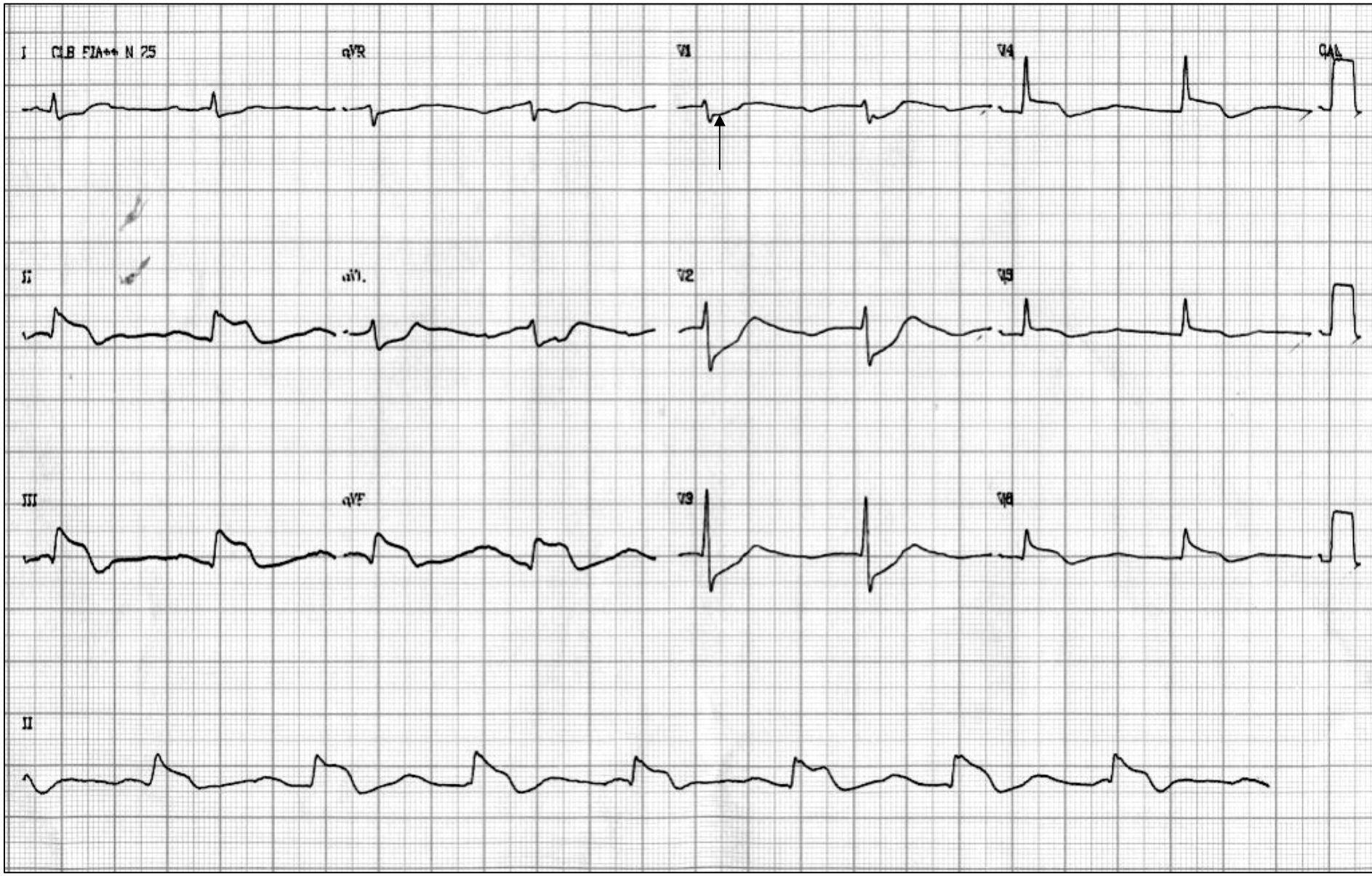
Dear Andrés: male, 54 years, hypertensive, was admitted into the emergency room in cardiorespiratory arrest consequence of ventricular fibrillation (VF). According to family relate four hours before the event, the patient complained, of constrictive retrosternal pain followed by dyspenia and vomit.
After reanimation maneuvers the patient was sent to the hemodynamic lab where preform angioplasty with stent placement and pacemaker implantation.

He is currently sedated with mechanic ventilation.
These ECGs present several interesting aspects
Which is the culprit artery?
Which is the heart rhythm in long II lead?

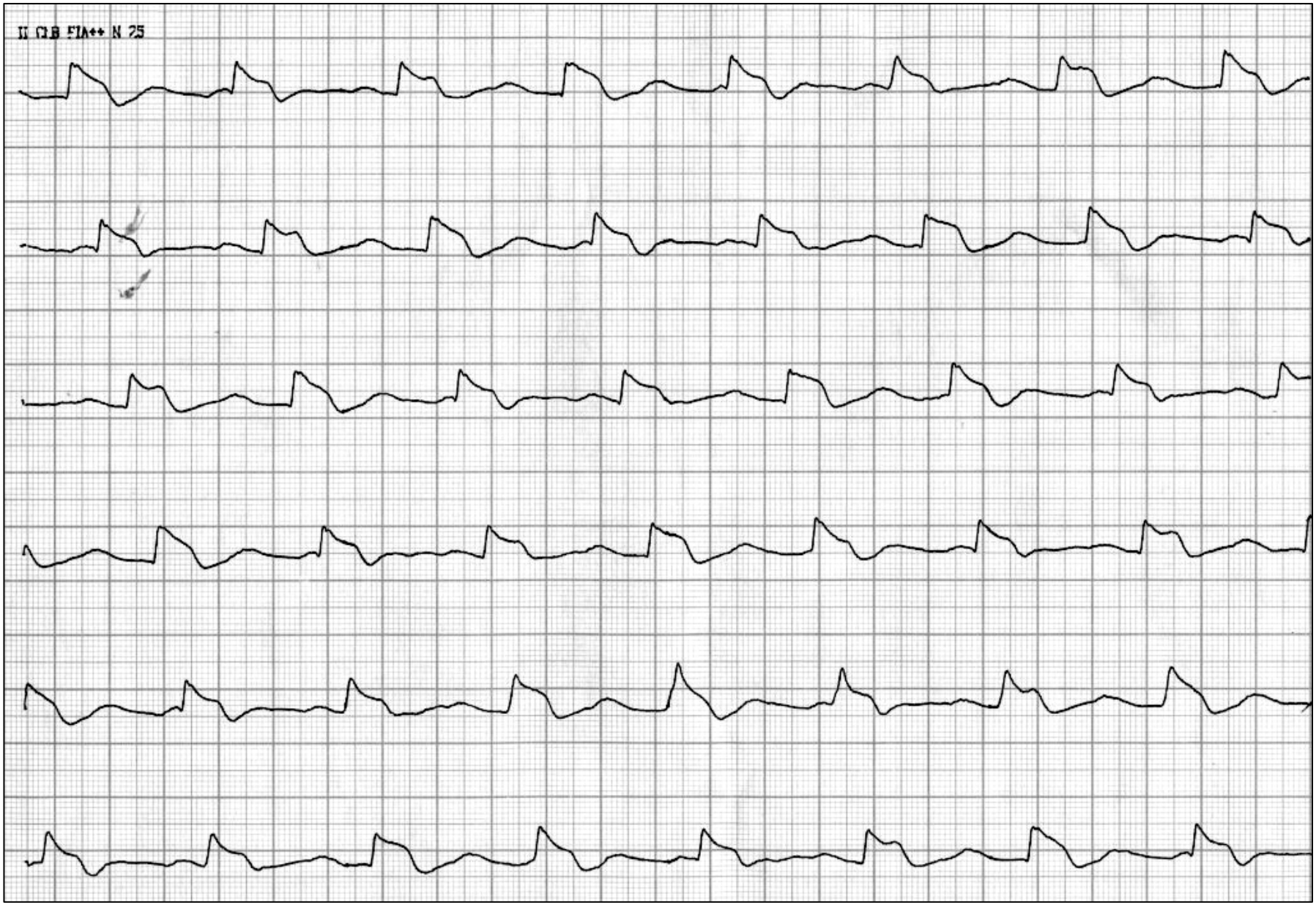
March, 27-2012 immediatly cardioversion 22h 05min



ECG after angioplasty March 28-2012 02:13



Continuous long II lead



Colleagues opinions

Sugiero, preguntando, ¿no será una coronária derecha dominante con afectación de ventrícuo derecho?

¿En qué me baso?

1. **Supra ST en cara inferior DII, DIII y aVF (con mayor positividad en DIII)**
2. **Infradesnivel del segmento ST en DI y aVL**
3. **Supradesnivel del segmento ST V3 y V4 y pensando en una CD dominante pues interesa también a las derivaciones V5 y V6**

Gracias y saludos cordiales,

Ilíada Cuba

I suggest asking, can not be a dominant right coronary artery(RCA) with involvement of the right ventricle?

How do I draw?

The ST segment elevation in inferior leads II, III and aVF (with more positivity in III)

The ST segment depression in I and aVL

The ST segment elevation V3 and V4 and dominant thinking of a RCA as well interested leads V5 and V6

Thanks and best regards,

Iliada from Cuba

Queridos amigos del forum

Discutiré el caso del Dr Raimundo Barbosa-Barros y Profesor Andrés Ricardo Pérez Riera
En esta etapa existen varios parámetros de gran significancia clinica
Frecuencia cardiaca elevada (de 150 lpm). Pareceria ritmo sinusal dado que en DII, se observa algo parecido a una onda P. Este es un signo de extrema gravedad porque señala la presencia de músculo isquémico desprotegido
El segmento ST está en un nivel mas alto que la onda T en las derivaciones afectadas lo que constituye otro signo de mucha gravedad
Con respecto a la arteria responsable la coronaria derecha dominante es una posibilidad, pero en este caso el ST↑ en V6 tendria que ser mayor que en V3 (ST in decrescendo) Aqui ocurre lo contrario, sugeriendo que la arteria responsable sea una artéria descendente anterior distal a la primera diagonal.
¿Como se explica la desviación del eje del QRS en el plano frontal a la derecha y el segmento ST elevado en las derivaciones inferiores? Respuesta: Una posibilidad es que la artéria coronária derecha esta obstruida previamente irrigada por la descendente anterior
¿ Porque V2 se expresa con Q/S? Respuesta: Porque los vectores de injuria izquierda arrastran los potencialles hacia esta derivación. Esto se debe a que la pared lateral no esta protejida por la arteria segunda marginal izquierda rama de la artéria circunfleja
El eje desviado hacia derecha no es consecuencia de bloqueo fascicular póstero-inferior. Obedece a que el músculo papilar posterior esta mas isquémico que el anterior
Este caso es de extrema gravedad y el pronóstico es muy reservado
Un fraternal abrazo
Samuel Sclarovskv

1. **Sclarovsky S et al Transient right axis deviation during acute anterior wall infarction or ischemia: electrocardiographic and angiographic correlation. Am J Cardiology 1986 Jul;8:27-31.**

I will analyze the case of Dr Raimundo Barbosa-Barros and Professor Andrés Ricardo Pérez-Riera At this stage there are several clinical parameters of major significance.
In the first ECG I observ elevated heart rate (150 bpm). This is a signal of extreme gravity because it indicates the presence of unprotected ischemic muscle. It would seem as sinus rhythm in lead II, there was something like a P wave.
The ST segment is at a level higher than the T wave in leads affected is another ominous sign.

With regard to the right coronary artery(RCA) dominance is a possibility, but in this case the ST ↑ in V6 would have to be higher than in V3 (ST in decrescendo) Here the opposite occurs, suggesting that the culprit artery is left anterior descending artery (LAD) distal to the first diagonal.
What explains the deviation of the QRS axis in the frontal plane to the right and the ST-segment elevation in the inferior leads? Answer: One possibility is that the RCA was obstructed previously and the irrigated territory supplied by the LAD.
Why V2 is given by Q / S? Answer: Because the vectors of the trail left potencials injury to this lead. This is because the lateral wall is not fully protected by no second marginal artery the left circumflex artery

The axis deviated to right is not the result of left posteriro fascicular block. It is because the posterior papillary muscle is more ischemic than the anterior one.

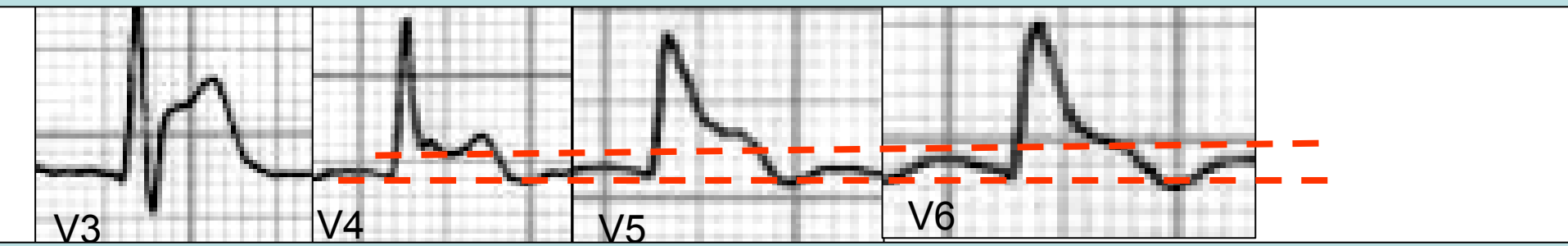
The prognosis of this case is extremely serious
Samuel Sclarovskv

1. **Sclarovsky S et al Transient right axis deviation during acute anterior wall infarction or ischemia: electrocardiographic and angiographic correlation. Am J Cardiology 1986 Jul;8:27-31.**

Querido Profesor Samuel: inicialmente habia pensado en obstrucción de la artéria descendente anterior sin afectacion de primera diagonal, lo cual explicaría el supradesnivel del segmento ST en las precordiales de V3-V6, y siendo una descendente anterior larga que contorna la punta irrigando la pared inferior que perfectamente podría elevar el segmento ST en dichas derivaciones. No obstante continuo con algunas dudas ¿Cómo me explicaría ST mas elevado en V3 y V4 que V5 –V6? ¿Cuando ocurre esto, no se trata de un signo de compromiso del VD?
Gracias. Dra Iliada de Cuba

Dear Professor Samuel had initially thought of obstruction of the left anterior descending artery(LAD) without affectation of the first diagonal, which would explain the ST-segment elevation in leads V3-V6, and being a long LAD surrounds the cardiac apex and irrigating inferior wall could easily to explain the ST segment elevation in inferior leads. However, I continue with some doubts:
How do you explain the ST segment elevation highest in V3 and V4 than in V5-V6?
Is this a sign of RV compromising?
Thank you in advance.
Dr Iliada Cuba

Querida Dra Iliada de Cuba le responde Andres: usted pregunta ¿Cómo me explicar ST mas elevado en V3 y V4 que V5 –V6? . ST↑ V4 es < V5-V6 Apenas V3 tiene ST↑ > V5-V6: Posible artefacto?



Dear Dr Iliada from Cuba. Andres replied: You ask how do you explain that **ST↑** is highest in V3 and V4 that V5-V6? . ↑ ST in V4 is <that V5-V6 Only ST ↑ in V3> V5-V6: Possible artifact?

Remodelamiento IMA ínfero-posterior: concepto algoritmo IAM inferior-remodelación fisiológica y patológica

Querida amiga Iliada de Cuba: El segmento S T y la onda T mas elevados señalan cual es el centro de la isquemia. En la obstrucción de la descendente anterior generalmente el centro de la isquemia se encuentra en V2 y V3 (segmentos S T y la ondas T mas altas).

En el presente caso es una excepción, un patrón infrecuente.

Los vectores isquémicos izquierdos (de V5-V6), inducen este fenómeno cuando la segunda marginal (que irriga la cara apical lateral) es poco desarrollada o está obstruida. En estos casos, la segunda diagonal (rama de la descendente anterior) la substituye irrigando esta área y asi dislocando los vectores hacia la izquierda.

El epicardio de esta area puede ser irrigado por tres differentes ramas secundarias:

1) La segunda marginal, rama de la circunfeja

2) La marginal izquierda, rama de la coronária derecha

3) La segunda diagonal rama de la descendente anterior

A esta superposición de sistemas de irrigación se lo ha denominado "overlaping circulation".

Esta área es la mas sufrida del corazón, por ser la que se contrae mas precóz e intensamente y tambien la que mas sufre cuando ocurren aumentos de las presiones diastólicas finales del ventrículo izquierdo. Por este motivo, la sabia madre naturaleza le da ha dado maxima protección de irrigación en los mamíferos, es decir, la cuando se obstruye la primera y segunda rama (fijese en su material ECG ycoronariografiaen isquemia aguda) siempre los ST y las ondas T estan mas elevados en V6 y V5, V4 es isoeléctrica y V3 y V2 presentan ST deprimidos con T positivas (el tipico infarto en cuña que ya lo hemos discutido en otro caso en el forum)

Este es un trabajo que estoy realizando junto con el Dr Zhon Zhong de China y Dr Kjell Nikus de FInlandia

Un fratenal abrazo

Samuel Sclarovsky

Inferoposterior AMI remodeling: concept, algorithm inferior AMI physiological and pathological remodeling

Dear friend Dr Iliada from Cuba: The ST segment and T wave most elevated point which is the center of ischemia. In the obstruction of the LAD, usually the center of ischemia is found in V2 and V3 (ST segment and T waves higher). The present case is an exception, a rare pattern.

Ischemic left vectors (V5-V6), induce this phenomenon when the second marginal branch (which supplies the lateral apical wall) is poorly developed or obstructed. In these cases, the second diagonal (branch of LAD) irrigates this area and thus disrupting the vectors to the left.

The epicardium in this area can be irrigated by three different secondaries branches:

- The second marginal branch of the LCX
- The left marginal branch of the RCA
- The second diagonal branch of the LAD

This multiple irrigation systems has been called "overlapping circulation".

This area is the most suffering of the heart, because its contractions is most precocious and intense contraction and also that suffer most when there are increases in end diastolic pressure of the LV. For this reason, the wise Mother Nature has given maximum protection of irrigation in mammals, i.e., when obstruction of the first and second branch occur always ST and T waves are more elevated in V6 and V5, V4 and V3 are isoelectric and V2 has depressed ST segment followed by a positive T wave

This is a work that I am doing along with Dr. Zhon Zhong of China and Dr Kjell Nikus of Finland

A fraternal hug

Samuel Sclarovsky Israel.

Caro Andrés: Ao que tudo indica, a artéria culpada é a coronária direita. O infarto é ínfero-lateral. O ritmo de DII longo é um ritmo idioventricular que traduz a reperfusão miocárdica.
Abraços afetuosos do

Hélio Germiniani M.D.Ph.D.Curitiba Brasil

Dear Andrés: Apparently, the culprit artery is right coronary artery. The infarction is inferolateral. The II lead shows a idioventricular rhythm which reflects the myocardial reperfusion. Affectionate hugs
Professor Hélio Germiniani M.D.Ph.D.Curitiba Brasil

Artéria envolvida: A Cx dominante.
Ritmo Sinusal com BAV do 1º grau evolui para BAV 2º grau, Mobitz I (períodos BAV2:1).
Atte
Severiano Atanes Netto

Culprit artery: Dominant Left Circunfex
Sinus Rhythm with first degree AV block that evolves for second degree AV block Mobitz Type I with 2:1 periods

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Prezados colegas Andrés Ricardo Pérez-Riera e Raimundo Barbosa-Barros,
A artéria culpada deve ser uma grande coronária direita dominante que, dando origem a àrtéria do nódulo A-V talvez explique a provável taquicardia sinusal com bloqueio atrioventricular variável devido a reperfusão pós-angioplastia primária.
Um abraço

Dr. Evandro Vidal Osterne General médico retirado das forças Armadas da República Federativa do Brasil Brasilia

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Dear colleagues, Andres Ricardo Perez Riera and Raimundo Barbosa-Barros,
The culprit artery should be a large dominant right coronary artery, giving rise to the AV node artery may explain the probable sinus tachycardia with atrioventricular block variable due to reperfusion after primary angioplasty.
a hug

Dr. Evandro Vidal Osterne Medical General retired Armed Forces of the Federative Republic of Brazil Brasilia

The culprit artery is probably not LAD, because V1 and V2 show ST depression. There are more signs indicating RCA than LCx occlusion. Could even be a proximal LAD occlusion, because ST elevation is higher in V3 than in V6 (cancellation of injury vectors from right ventricle and lateral wall), or the ST elevation in V3 could be explained by a large marginal branch distal to the occlusion. There is severe grade 3 ischemia (Sclarovsky-Birnbaum grading). Post-PCI there are initial signs of myocardial reperfusion (end-T positive). There seems to be AV-dissociation.

Kjell Nikus, Tampere, Finland

La arteria culpable probablemente no es la descendente anterior (DA), ya que existe depresión del segmento ST en V1 y V2.
Hay más señales que indican oclusión de la coronaria derecha RCA que de la circunfleja CX. Incluso podría ser una oclusión proximal de la DA, ya que la elevación del ST es mayor en V3 que en V6 (cancelación de los vectores de lesión de ventrículo derecho y la pared lateral), o la elevación del ST en V3 puede ser explicado por una gran rama marginal distal a la oclusión. El grado de isquemia es severo: (grado 3de la clasificación de Sclarovsky Birnbaum).
Post-ICP hay signos iniciales de la reperfusión miocárdica (las porciones finales de las T positivas). Parece que hay disociación AV.

Kjell Nikus, Tampere, Finlandia

Final comments

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Diagnosis: Proximal obstruction of dominant RCA because RCA supplies the posterior descending artery (PDA) (a.k.a. posterior interventricular artery) determines the coronary dominance. A precise anatomic definition of dominance would be the artery which gives off supply to the AV node (the AV nodal artery). Most of the times this is the RCA. Approximately 70% of the general population are RCA-dominant, 20% are co-dominant, and 10% are left-circumflex (LCX) dominant. The term coronary dominance was introduced by Sclesinger in 1940(1). The RCA originates above the right cusp of the aortic valve. It travels down the right atrioventricular groove, towards the crux of the heart.

At the origin of the RCA is the conus artery. In addition to supplying blood to the RV, the RCA supplies 25% to 35% of the LV. In 85% of patients, the RCA gives off the PDA.

The PDA supplies the inferior wall, ventricular septum, and the posteromedial papillary muscle.

The RCA also supplies the SA nodal artery in 60% of patients. The other 40% of the time, the SA nodal artery is supplied by the LCX.

Clinical electrocardiographic background of present case

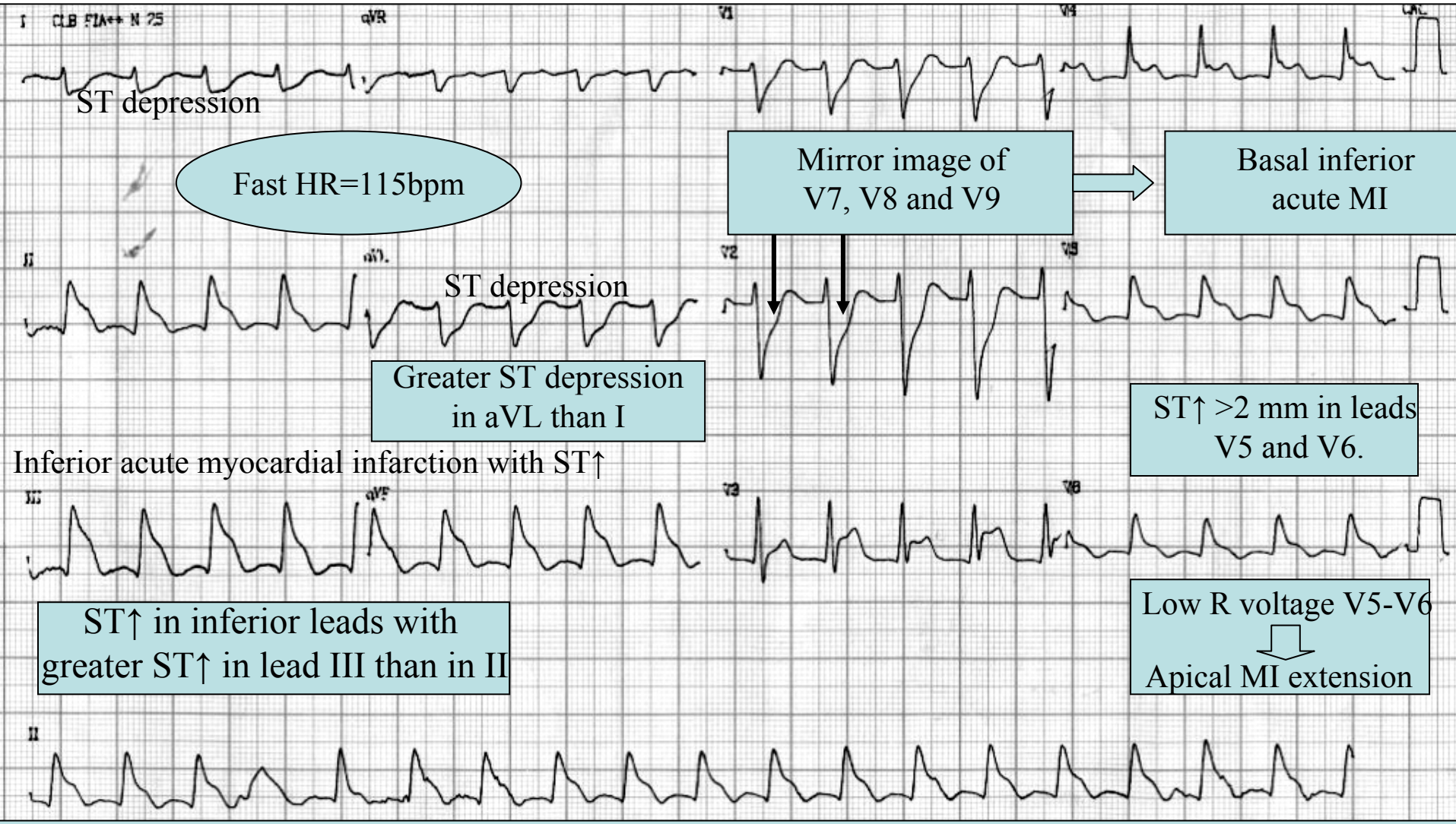
- Early dyspnea in interogatory. Why? Because afectation of posteromedial papillary muscle usually supplied exclusively by the PDA The clinical significance of this is that a MI involving the PDA is more likely to cause mitral regurgitation and precocious dyspneia. Frequently, left ventriculography shows severe diffuse impairment of akinesis of the left ventricular inferobasal (old posterobasal segment).
- Early cardiac arrest
- Necessity of pacemaker implantation giving rise to the AV node artery may explain heart rate tachycardia with variable AV block and dissociation due to reperfusion after primary angioplasty.
- ST↑ in leads V5 and V6 suggests a greater risk area and impaired myocardial reperfusion in patients with inferior acute myocardial infarction(2).

1. ***Sclesinger MJ Relation of anatomic pattern to pathologic condition of the coronary arteries. Archives of Pathology. 1940; 30: 403-415.***
2. ***Kosuge M, Ebina T, Hibi K, et al. Implications of ST-segment elevation in leads V5 and V6 in patients with reperfused inferior wall acute myocardial infarction. Am J Cardiol. 2012 Feb 1;109:314-329***.

- Proximal occlusion of the RCA may, in certain circumstances, cause ST↑ on the anterior wall leads. This can occur more easily when the size of the lesion in the inferior LV wall is smaller that in the RV. The size distribution of the ST↑ in the above leads is different from that seen when the LAD is occluded. More rarely, isolated RV infarctions on a substrate of a non-dominant RCA can cause ST↑ exclusively on the anterior wall leads, with no ECG changes in the inferior leads.(1)

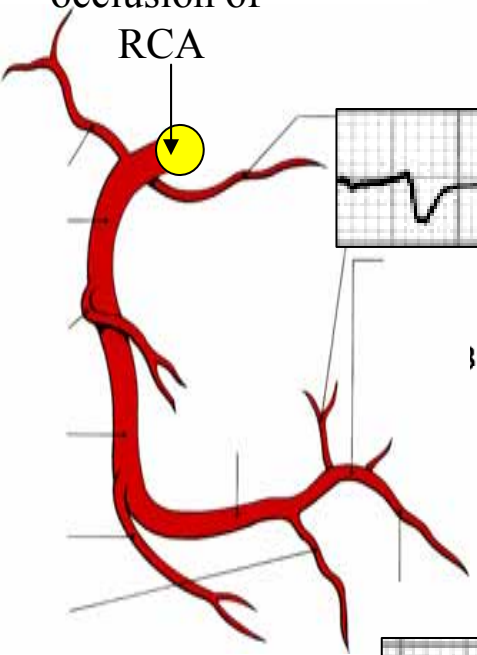
1. **Triantafyllis DG, Vrahatis A, Zaharoulis A. Electrocardiographic picture of acute anterior infarction due to proximal obstruction of the right coronary artery. Hellenic J Cardiol. 2005 Mar-Apr;46:154-157.**

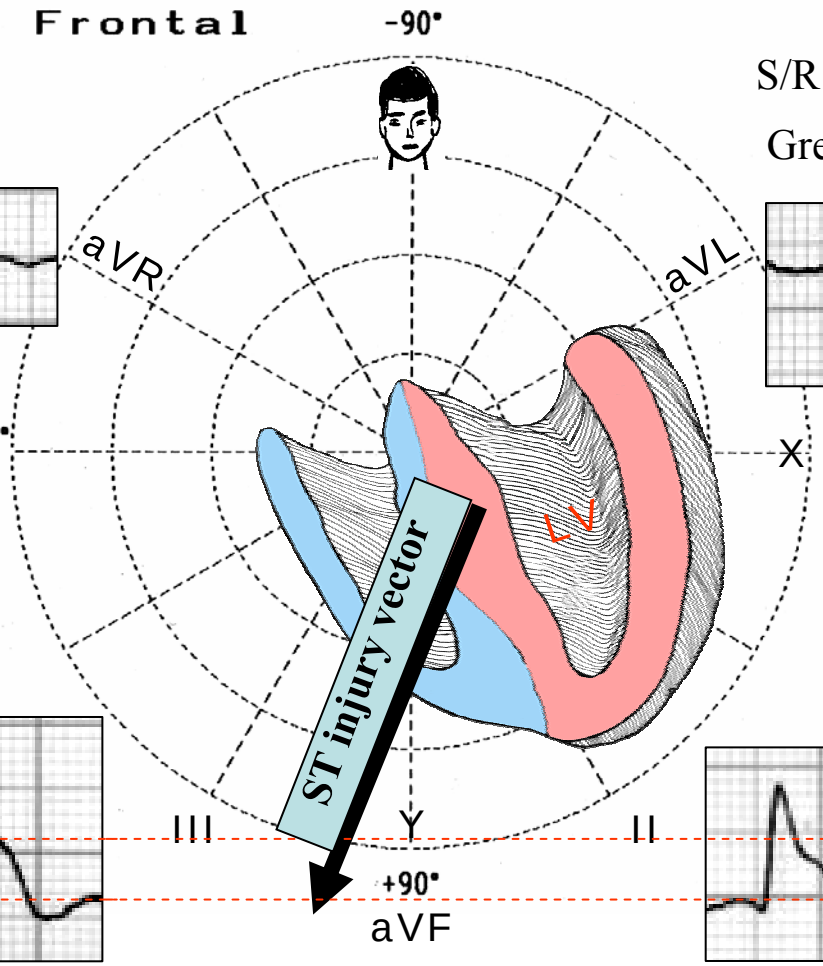
March, 27-2012 immediatly cardioversion 22h 05min



QRS axis near +120º, rS pattern I and aVL, qR pattern in inferior leads, similar to Left Posterior Fascicular Block consequence of posteromedial papillary muscle ischemia. This structure is supplied by PDA.

Characteristic Pattern of proximal occlusion of RCA



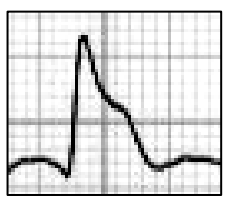


S/R ratio of greater than 1:3 in aVL

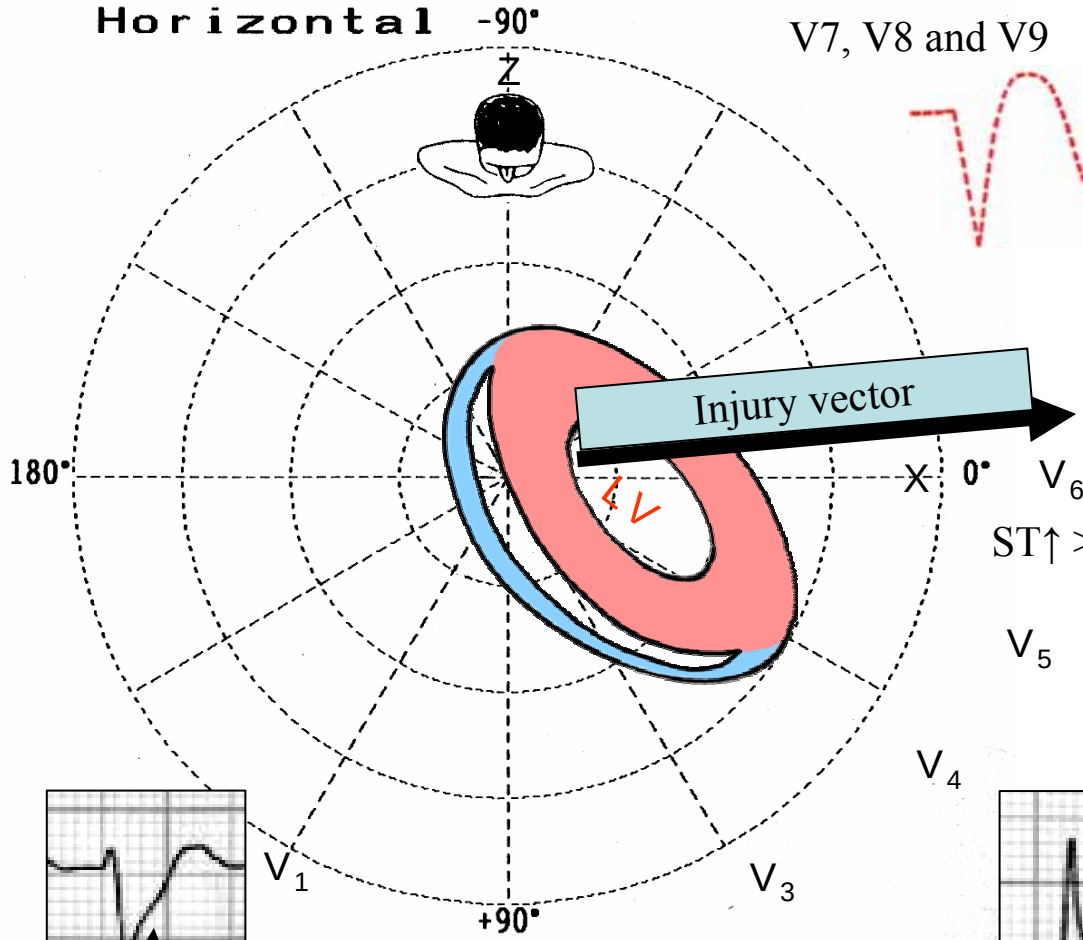
Greater ST depression in aVL than I

ST segment depression

ST↑ in inferior leads with greater ST↑ in lead III than in II



The ST injury vector directed to downward and to right

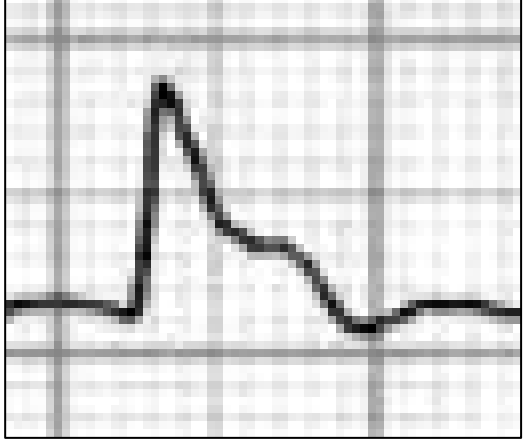


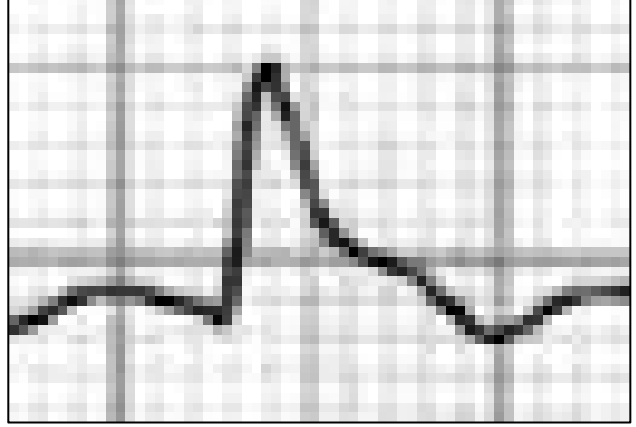
V7, V8 and V9

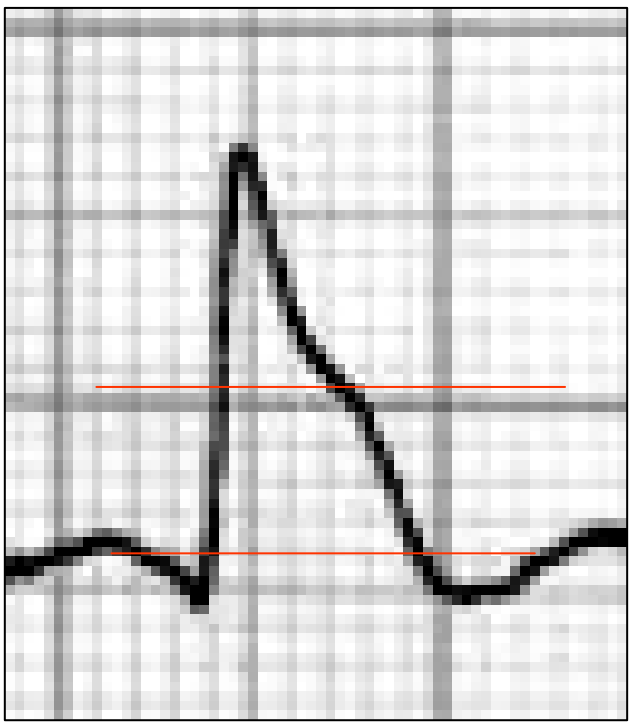
Basal inferior acute MI

ST↑ >2 mm in leads V5 and V6.

Mirror image of V_7, V_8 and V_9



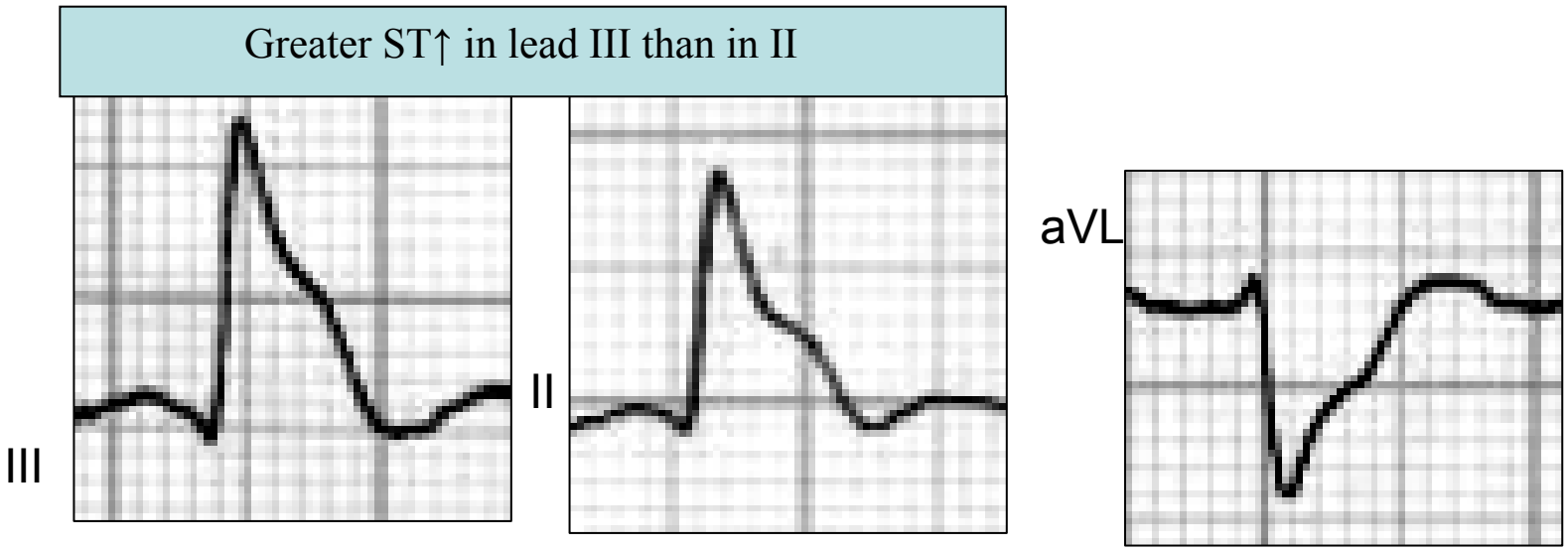




Multivariate analysis showed that ST↑ in leads V5 and V6 with ST↑ in lead III greater than in V6 in patients with first inferior AMI is suggestive of proximal RCA occlusion. Additionally, ST↑ in leads V5 and V6 suggests a greater risk area and impaired myocardial reperfusion in patients with inferior acute myocardial infarction (1).

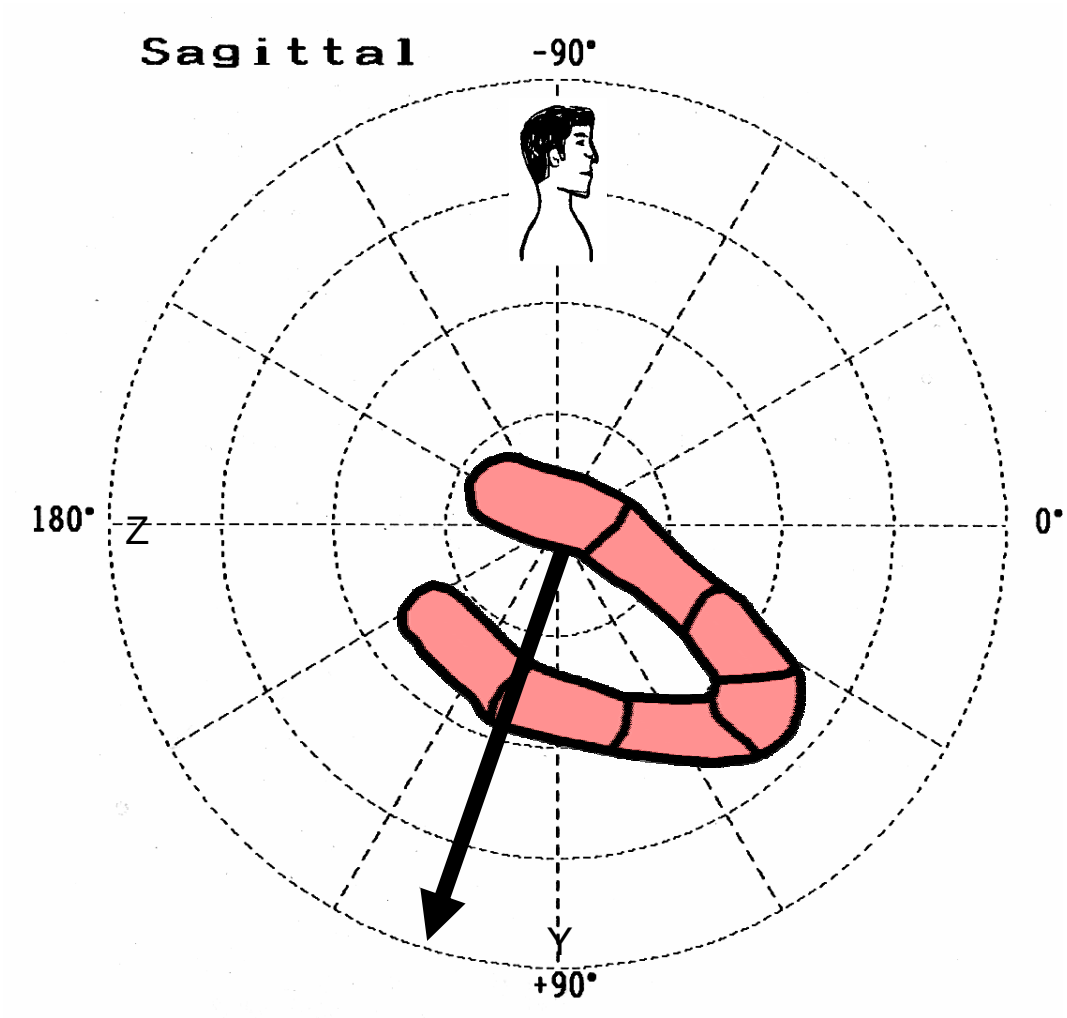
1. **Kosuge M, Ebina T, Hibi K, et al. Implications of ST-segment elevation in leads V5 and V6 in patients with reperfused inferior wall acute myocardial infarction. Am J Cardiol. 2012 Feb 1;109:314-329.**

Greater ST↑ in lead III than in II, greater ST depression in aVL than I, and an S/R ratio of greater than 1:3 in aVL were not useful to discriminate between dominant RCA and dominant LCx occlusion-related inferior AMI.

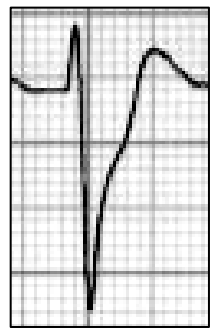


ST-segment deviation in lead V4R and the ratio of ST downward arrowV3/ST upward arrow (III) were useful in predicting the dominant artery occlusion-related inferior AMI.

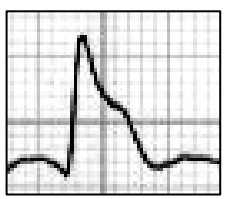
1. **Zhan ZQ, Wang W, Dang SY, et al. Electrocardiographic characteristics in angiographically documented occlusion of the dominant left circumflex artery with acute inferior myocardial infarction: limitations of ST elevation III/II ratio and ST deviation in lateral limb leads. J Electrocardiol. 2009 Sep-Oct;42:432-9.**



V_2



aVF



ECG after angioplasty March 28-2012 02:13

V7, V8 and V9

Mirror image of V7, V8 and V9

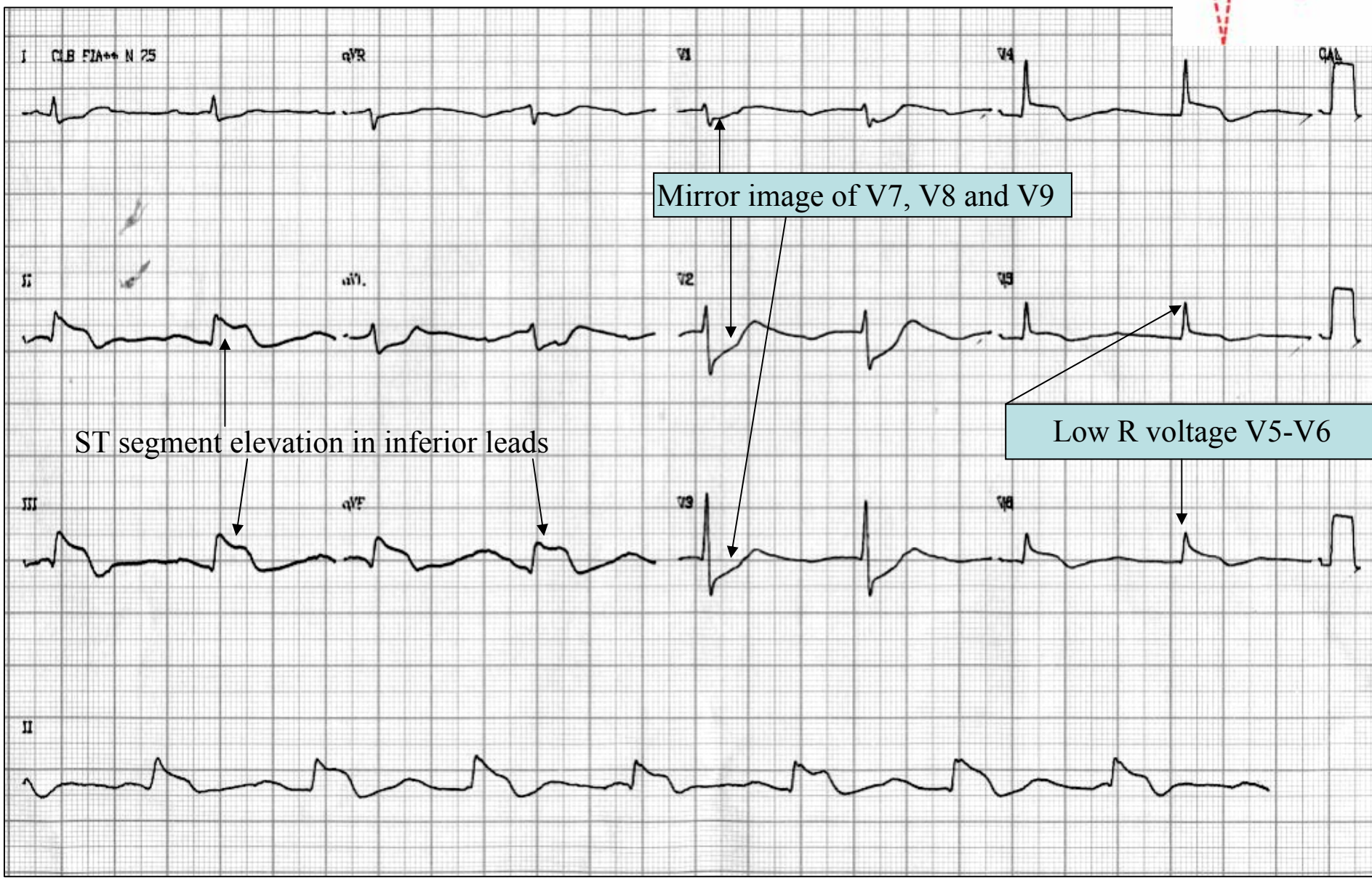
ST segment elevation in inferior leads

Low R voltage V5-V6

Acute phase IM inferobasal with lateralapical wall extension is indicative of higher risk. Patients with inferobasal and lateral wall extension can be identified by the presence of ST depression in the right precordial leads

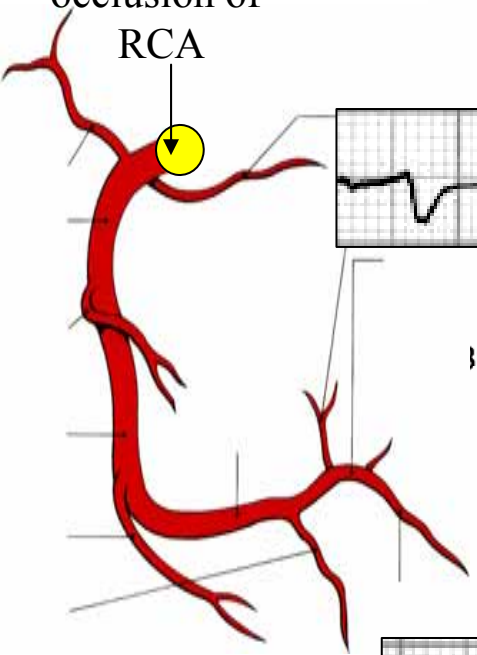
ECG after angioplasty March 28-2012 02:13

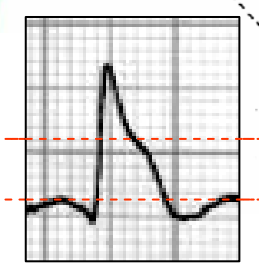
V7, V8 and V9

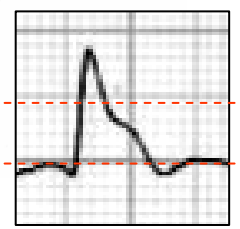


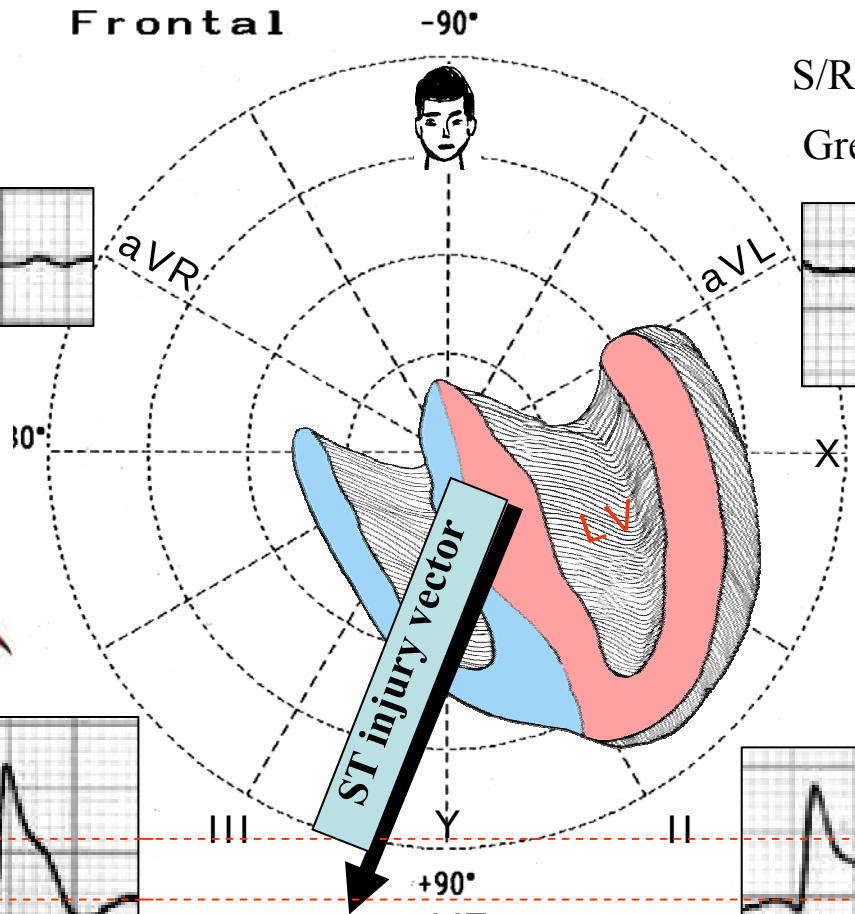
Acute phase IM inferior lateral dorsal wall in old nomenclature

Characteristic Pattern of proximal occlusion of RCA



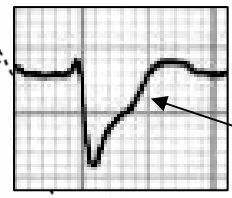




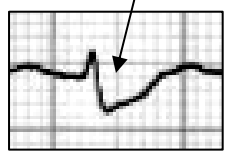


S/R ratio of greater than 1:3 in aVL

Greater ST depression in aVL than I

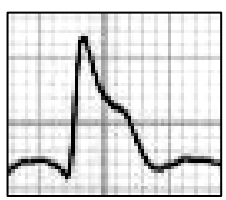


ST segment depression



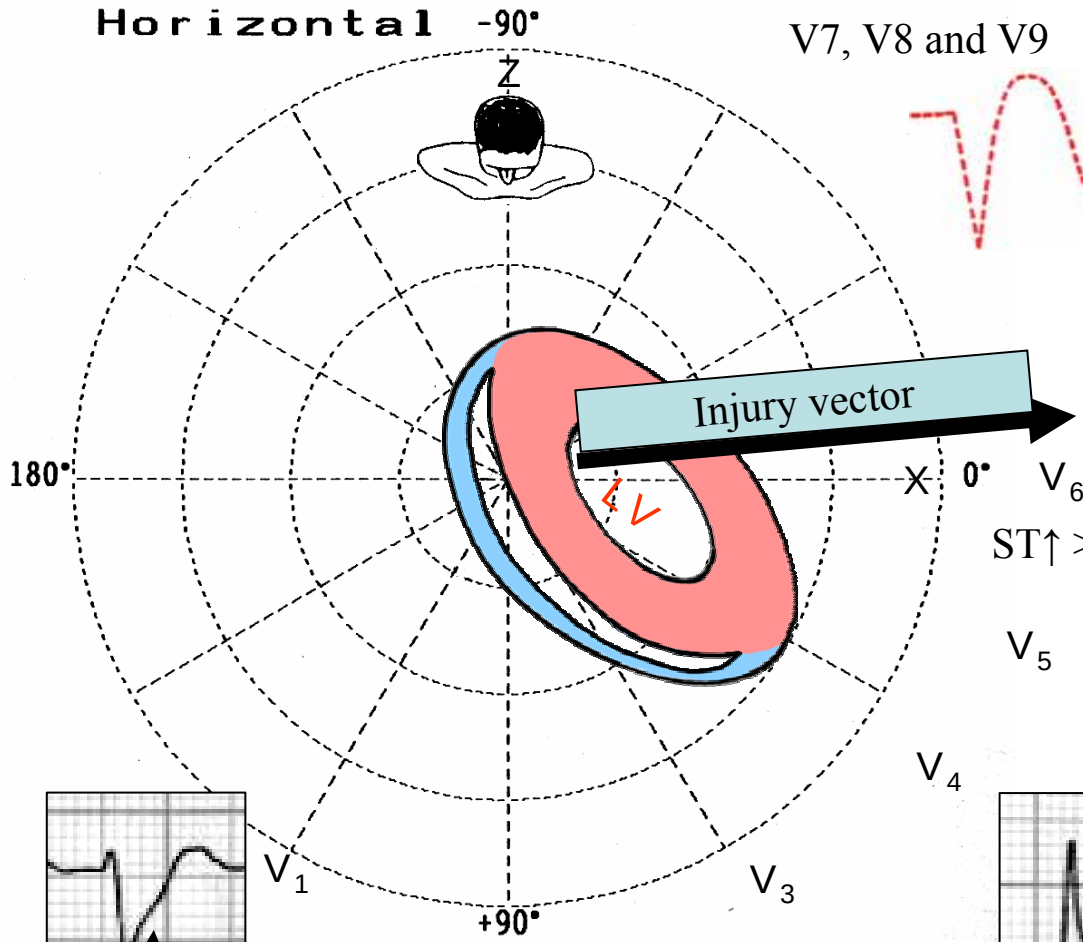
The reciprocal image in I and aVL suggests the possibility of RCA obstruction.(1)

ST↑ in inferior leads with greater ST↑ in lead III than in II

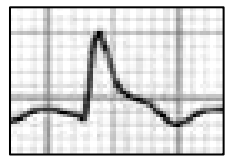


The ST injury vector directed to downward and to right

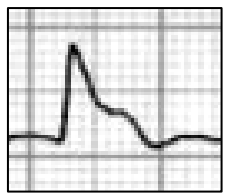
1. Parale GP, et al. J Assoc Physicians India. 2004;52:376-379.

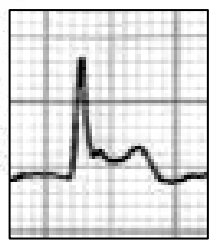


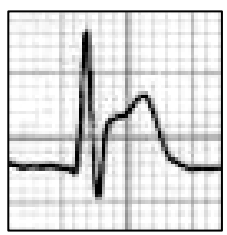
V7, V8 and V9

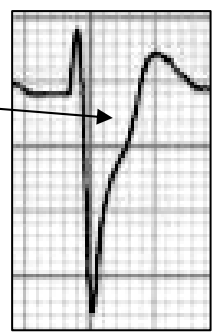


ST↑ >2 mm in leads V5 and V6.

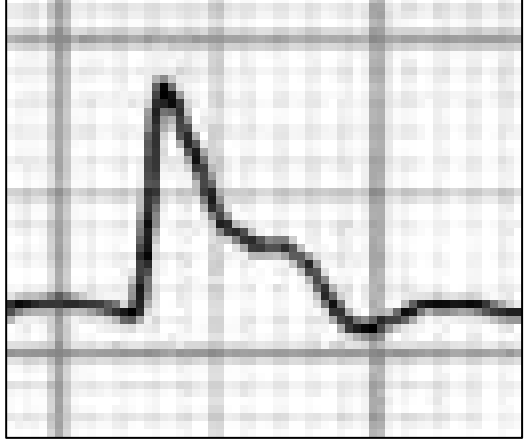


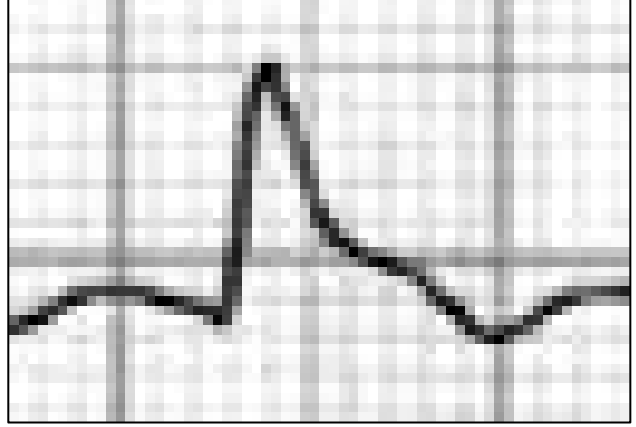


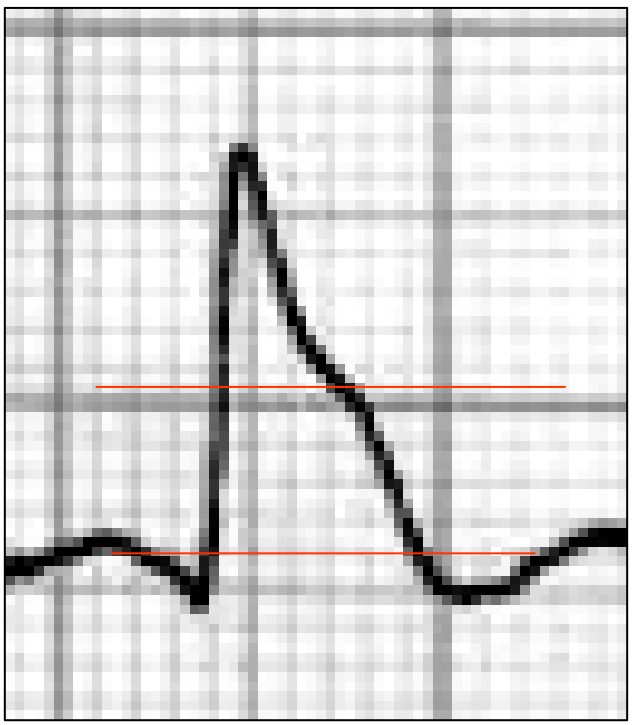




Mirror image of V_7, V_8 and V_9



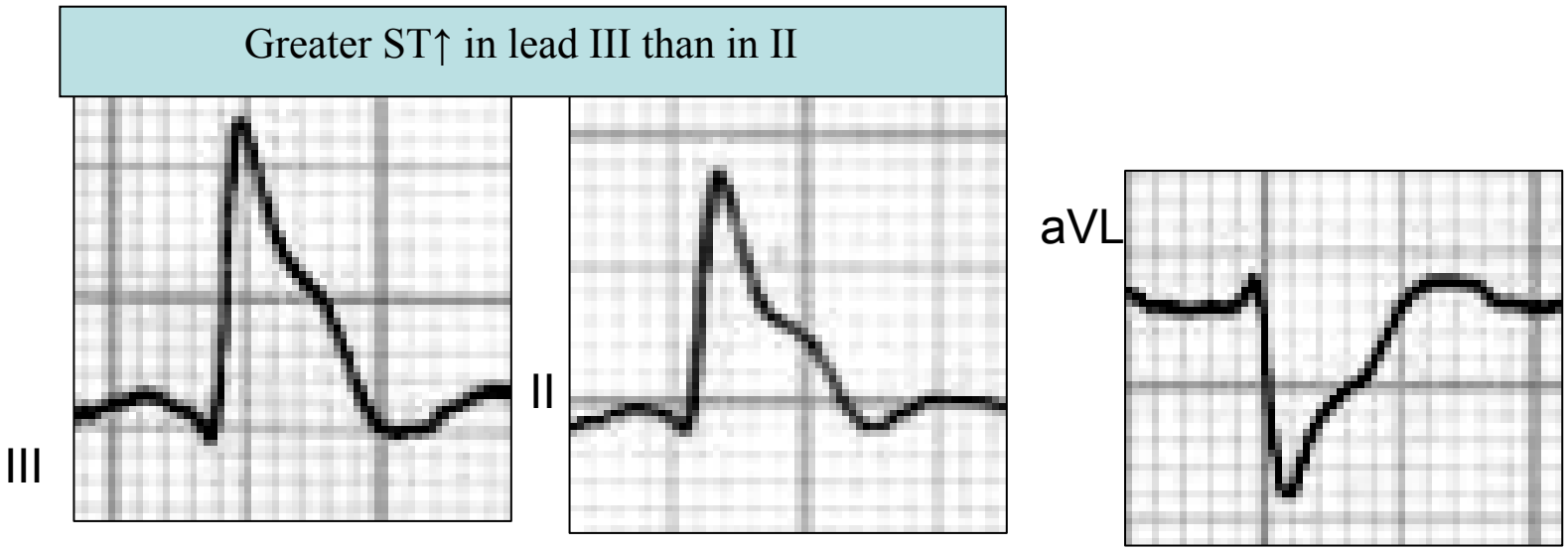




Multivariate analysis showed that ST↑ in leads V5 and V6 with ST↑ in lead III greater than in V6 in patients with first inferior AMI is suggestive of proximal right coronary artery occlusion. Additionally, ST↑ in leads V5 and V6 suggests a greater risk area and impaired myocardial reperfusion in patients with inferior acute myocardial infarction(1).

1. **Kosuge M, Ebina T, Hibi K, et al. Implications of ST-segment elevation in leads V5 and V6 in patients with reperfused inferior wall acute myocardial infarction. Am J Cardiol. 2012 Feb 1;109:314-329.**

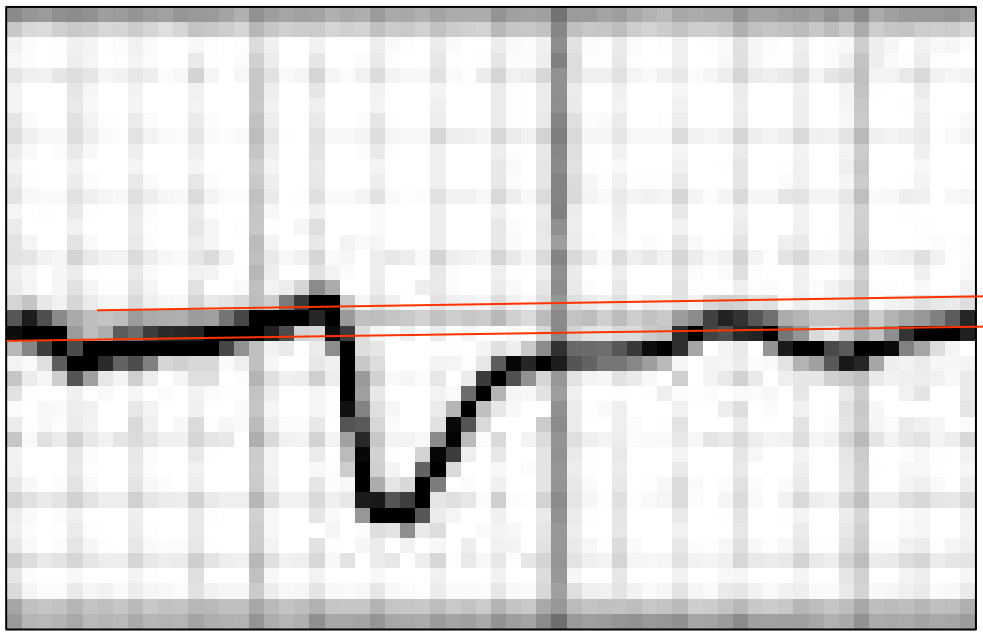
Greater ST↑ in lead III than in II, greater ST depression in aVL than I, and an S/R ratio of greater than 1:3 in aVL were not useful to discriminate between dominant RCA and dominant LCx occlusion-related inferior AMI.



ST-segment deviation in lead V4R and the ratio of ST downward arrowV3/ST upward arrow (III) were useful in predicting the dominant artery occlusion-related inferior AMI.

1. **Zhan ZQ, Wang W, Dang SY, et al. Electrocardiographic characteristics in angiographically documented occlusion of the dominant left circumflex artery with acute inferior myocardial infarction: limitations of ST elevation III/II ratio and ST deviation in lateral limb leads. J Electrocardiol. 2009 Sep-Oct;42:432-9.**

The degree of ST-segment depression in lead aVR is a useful independent predictor of impaired myocardial reperfusion in patients who have experienced inferior AMIs.(2)

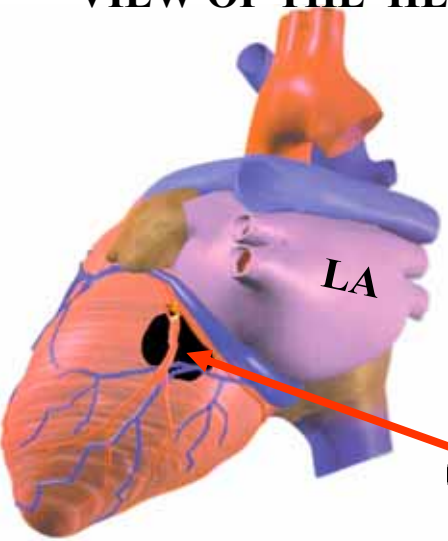


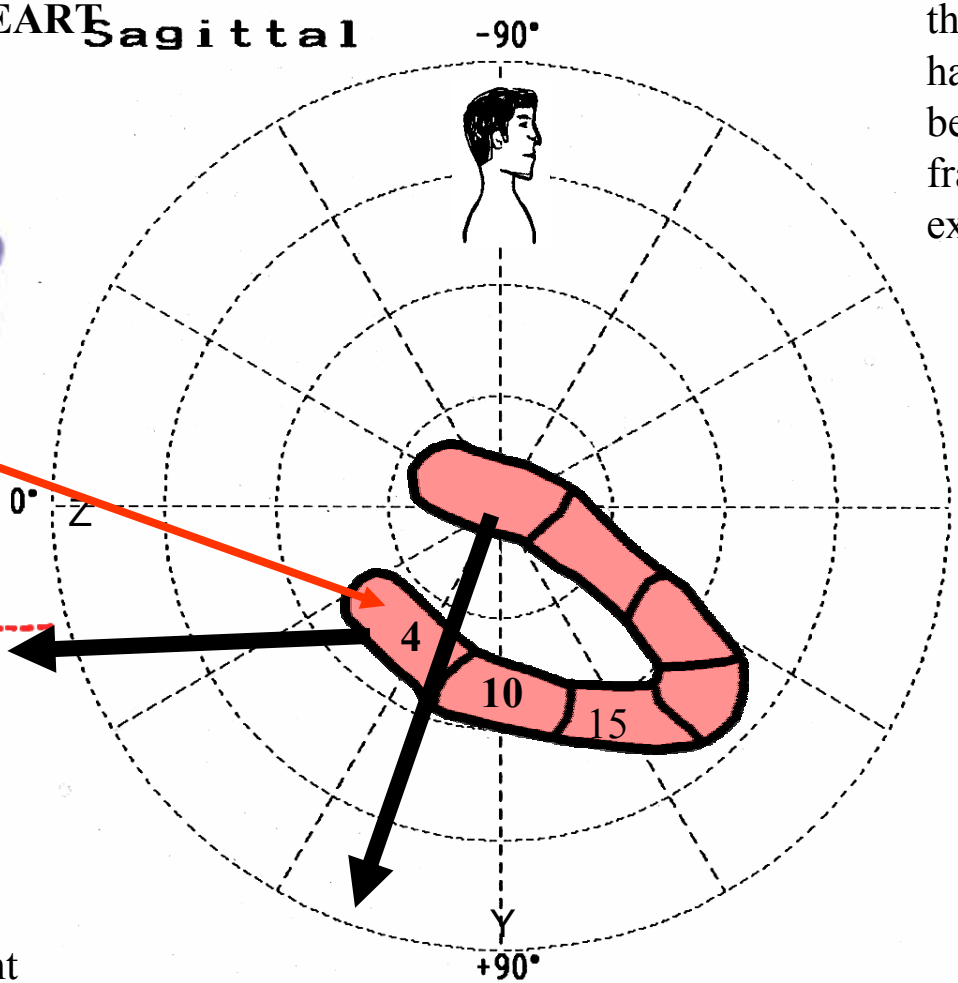
Minimal ST-segment depression =1mm

ST segment changes (elevation or depression) in lead aVR is observed in approximately 50% of cases of inferior wall STEMI patients. The presence of such ST segment changes is associated with a poorer prognosis during the hospital stay, and the changes are not associated with the type of reperfusion treatment.(1)

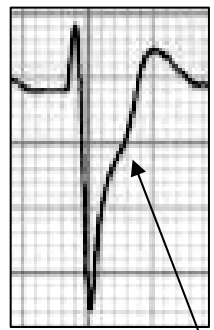
1. **Kukla P, Bryniarski L, Dudek D, Królikowski T, Kawecka Jaszcz K. Prognostic significance of ST segment changes in lead aVR in patients with acute inferior myocardial infarction with ST segment elevation. Kardiol Pol. 2012;70:111-118.**
2. **Kosuge M, Kimura K, Ishikawa T,et al.ST-segment depression in lead aVR: a useful predictor of impaired** myocardial **reperfusion in patients with** inferior acute myocardial infarction**. Chest. 2005 Aug;128:780-786.**

LEFT POSTERIOR OBLIQUE VIEW OF THE HEART





Diaphragmatic infarctions that present reciprocal image have a worse prognosis because they have less ejection fraction and are more extensive infarctions.

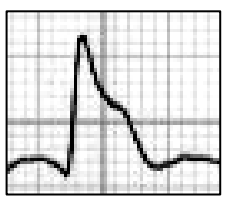


Mirror or reciprocal image of V7, V8 and V9#

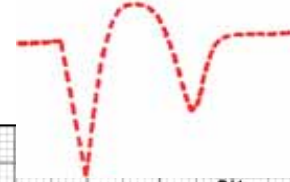
4: Basal inferior segment

Ancient dorsal or posterior or strictly posterior

4;10 and 15: inferior wall



ECG after angioplasty March 28-2012 02:13



V7, V8 and V9

Mirror image of V7, V8 and V9

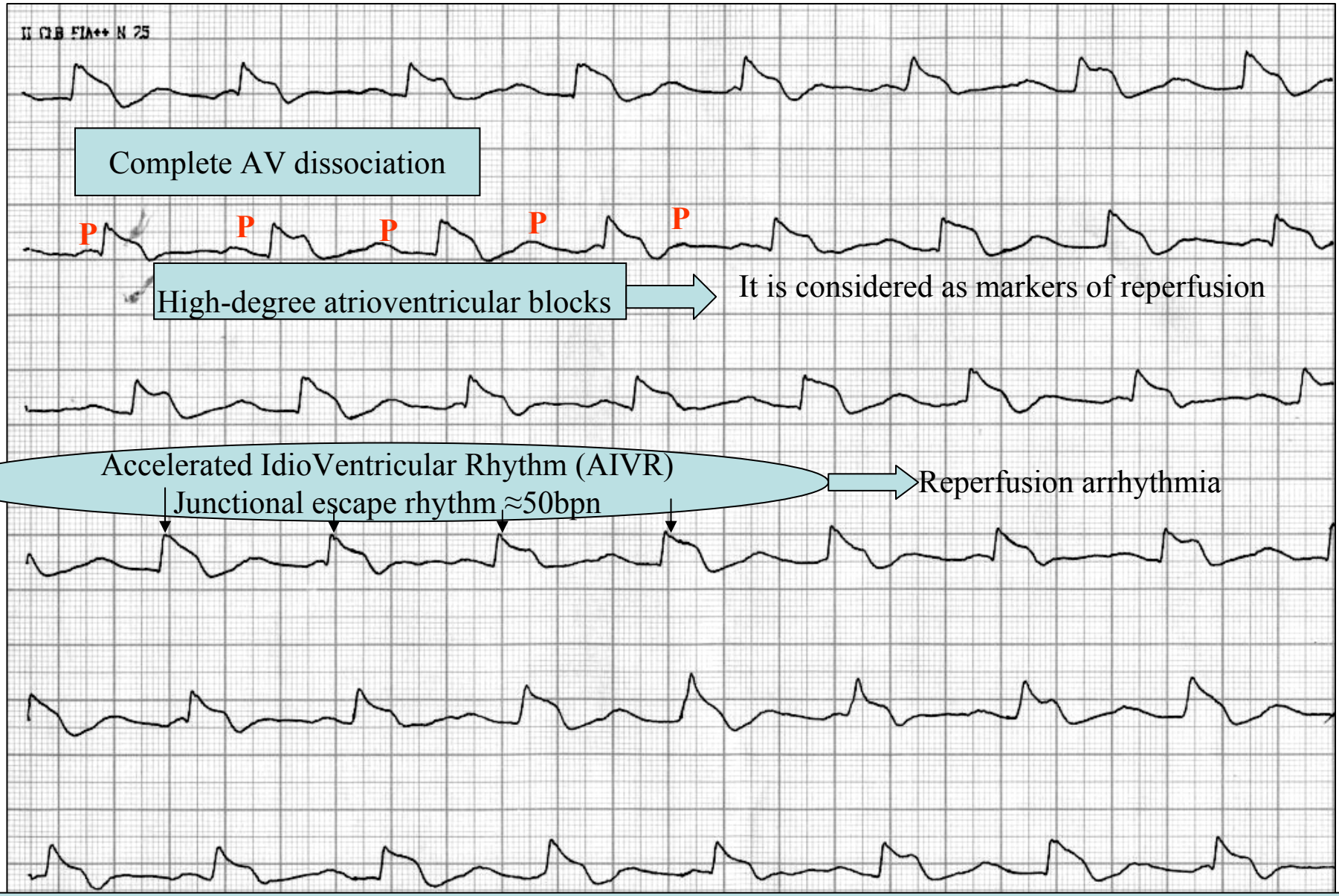
ST segment elevation in inferior leads

Low R voltage V5-V6

Complete AV dissociation due to high grade or complete AV block with a junctional escape rhythm ≈50 bpm and a sinus rhythm ≈60 bpm. Since this is in close proximity to the acute MI with ischemia in the AV junction, it may be reversible. (Dr. Frank Yanowitz comment)

Acute phase IM inferobasal lateral wall (in old nomenclature inferolateral and dorsal). New nomenclature inferolateral AMI or Type: B3. Most probable place of occlusion: proximal RCA

Continuous long II lead

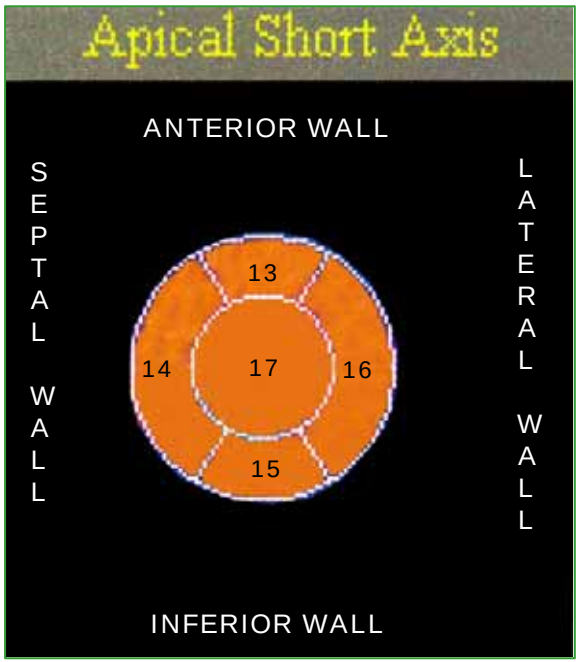


Complete AV dissociation due to high grade or complete AV block with accelared idioventricular rhythm or a junctional sustained escape rhythm ≈50 bpm and a sinus rhythm ≈60 bpm typical of reperfusion.

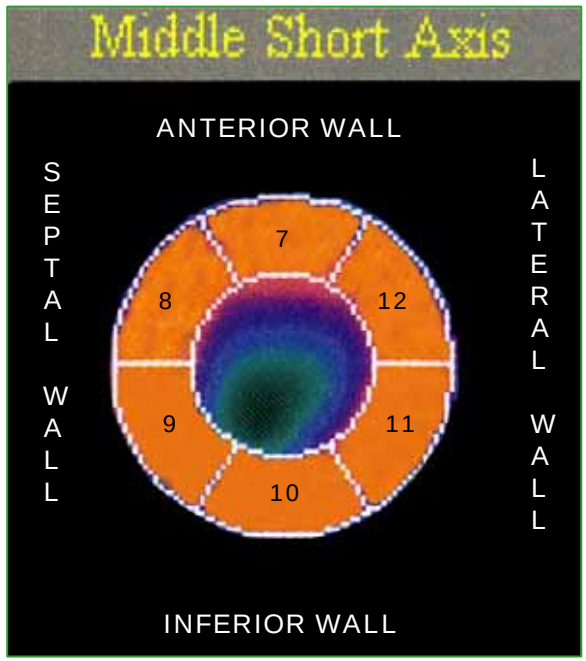
Reperfusion arrhythmias originate as a consequence of the complex of cellular and humoral reactions accompanying the opening of coronary artery. As the primary cause of their generation are considered the chemically defined substances that are produced and accumulated in myocardium during reperfusion. The key role is ascribed to free oxygen radicals but of importance are also other substances such as calcium, thrombin, platelet activating factor, inositol triphosphate, angiotensin II and others. These chemical mediators of reperfusion arrhythmias operate as modulators of cellular electrophysiology causing the complex changes at the level of ion channels. It is supposed that in the genesis of reperfusion arrhythmias unlike ischemic arrhytmias operate nonreentrant mechanisms such as abnormal or enhanced automacy and triggered activity due to afterdepolarizations. As a typical reperfusion arrhythmia is considered an early (within 6 hours after start of thrombolysis), frequent (> 30 episodes/hour) and repetitive (occurring during > 3 consecutive hours) accelerated idioventricular rhythm (AIVR). AIVR with such characteristics has a high specificity and positive predictive accuracy but relative low sensitivity as a predictor of reperfusion. Thus, in occurrence of AIVR, recanalization of infarction-related coronary artery is very probable, but in absence of AIVR, reperfusion is still not excluded. The following arrhythmias are regarded also as markers of reperfusion: frequent PVCs (> twofold increase in frequency within 90 minutes after the start of thrombolysis), a significant increase of episodes in NS-VT, sinus bradycardia and probably also high-degree atrioventricular blocks. At present, there is no definite evidence, as to whether SVT and especially VF can be caused by reperfusion. Reperfusion arrhythmias are an important noninvasive marker of successful recanalization of infarction-related coronary artery. However, they are also a sign of reperfusion injury and a finding which may limit the favorable effect of reperfusion. In account of that, there is a very intensive search for pharmacologic interventions which could protect or attenuate the reperfusion injury and thereby also the genesis of reperfusion arrthythmias. Although promising results were obtained with many substances antagonizing the effects of mediators of reperfusion injury, there is no definite recommendation for their use under clinical conditions. However, the results from the latest clinical trials with ACE inhibitors are very promising. These trials render relative conclusive evidence, that ACE inhibitors could have a protective effect against reperfusion arrhythmias.

HEART WALLS WITH CONTRAST-ENHANCED CARDIOVASCULAR MAGNETIC RESONANCE (CE-CMR)

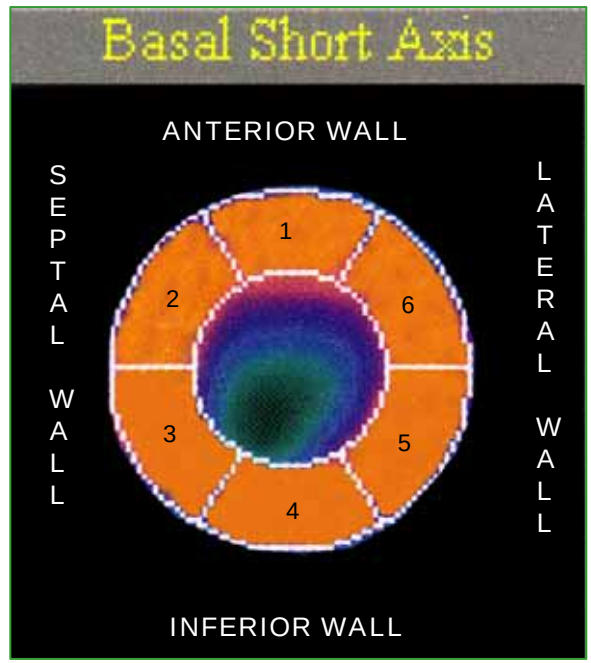
APICAL SHORT AXIS

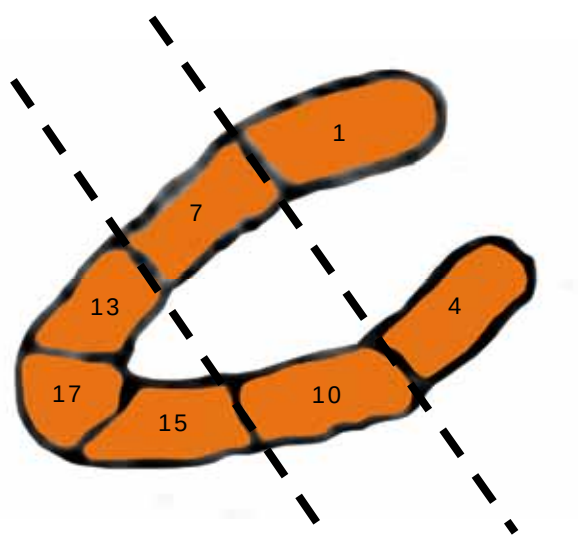


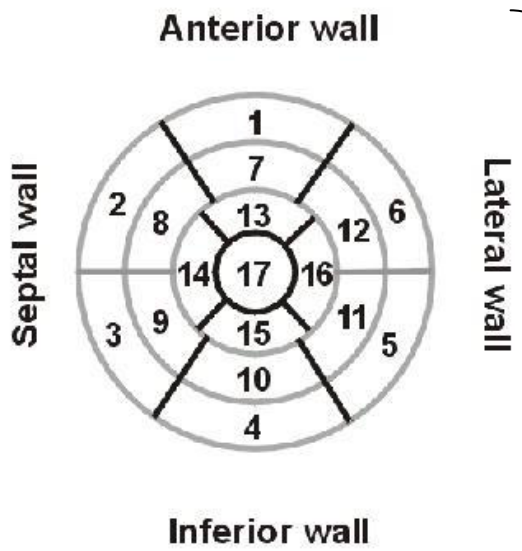
MIDDLE SHORT AXIS



BASAL SHORT AXIS

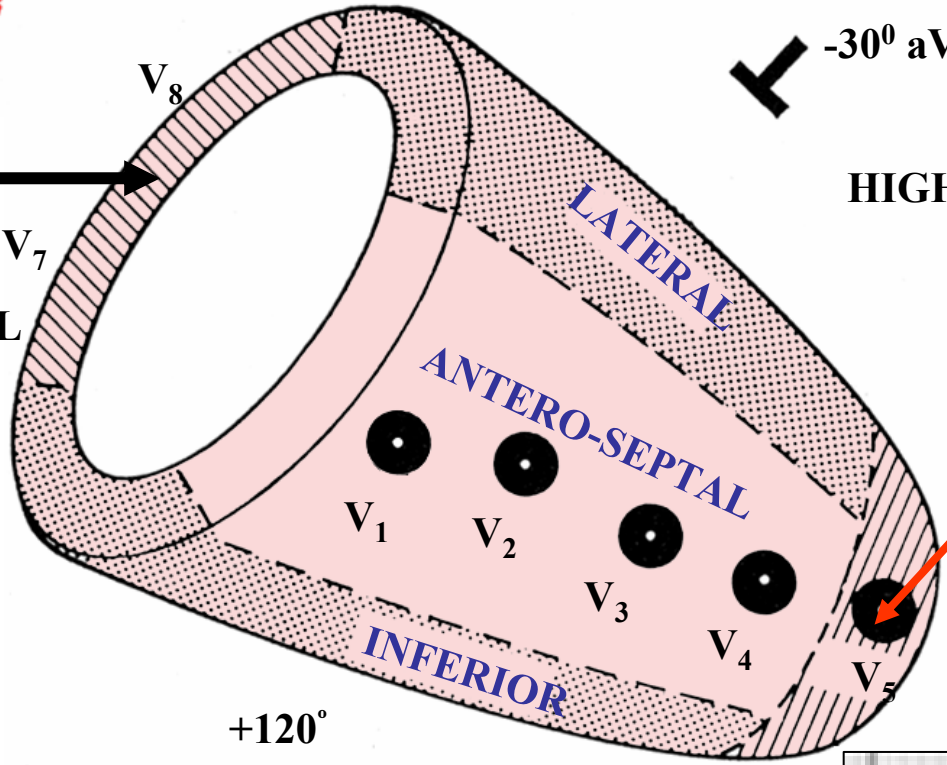






Polar map short axis in "Bull's-eye"

CLASSICAL ELECTROCARDIOGRAPHIC CLASSIFICATION OF MYOCARDIAL INFARCTIONS



POSTERIOR, DORSAL OR POSTERO-BASAL

-30⁰ aVL

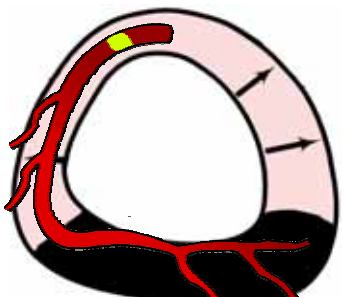
HIGH LATERAL

0⁰ I

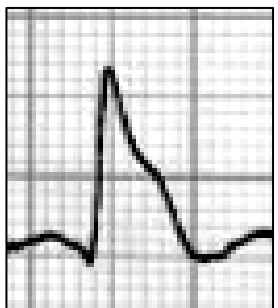
APICAL OR LOW LATERAL

The cardiac cone, its sides and corresponding leads: Antero-septal: V1 to V4; Apical or low lateral: V5 and V6; High lateral: I and aVL; Inferior or diaphragmatic: II, III and aVF; Dorsal, posterior or postero-basal: V7, V8 and V9.

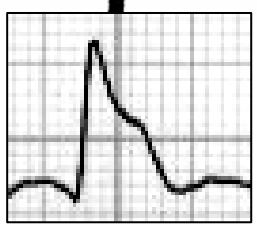
Short axis



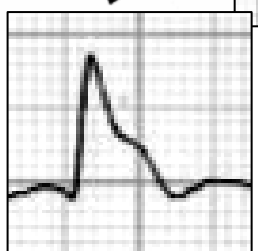
+120°
III

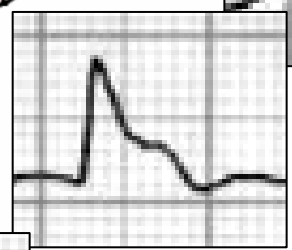


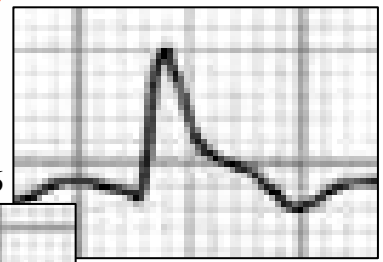
+90⁰
aVF



+60°
II



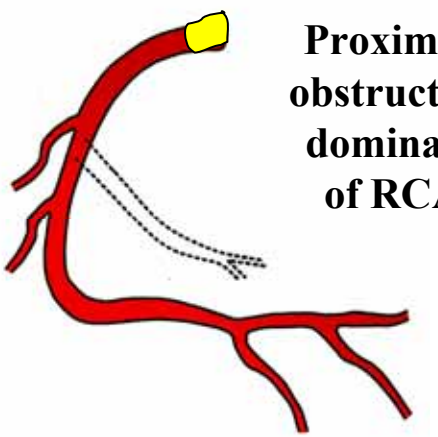




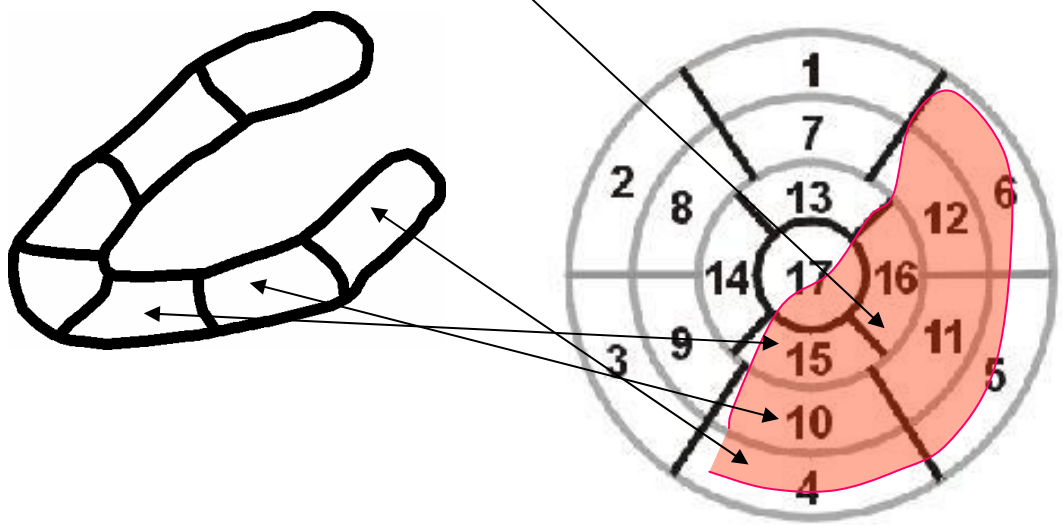
NEW ELECTROCARDIOGRAPHIC TERMINOLOGY FOR Q-WAVE INFARCTIONS BASED ON THE CORRELATION WITH CE-CMR

2) INFEROLATERAL ZONE

- **Inferolateral or inferobasal-lateral**
- **Type:** B-3
- **Most likely site of occlusion:** dominant RCA or dominant LCX
- **ECG pattern:** signs of inferior (Q in II, II, aVF: B2) and/or lateral infarction
- **Segments compromised by infarction in CE-CMR:**
- **SE:** 73%.
- **SP:** 98%.



Proximal obstruction dominant of RCA



1) Bayés de Luna A, et al.Am J Cardiol. 2006;97:443-451.
2) Bayés de Luna A, et al. Circulation 2006; 114:1755-1760.
3) Bayés de Luna A, et al. J Electrocardiol. 2006; 39 (4 Suppl):S79-81.
4) Bayés de Luna A, et al. J Electrocardiol. 2007;40:69-71.
5) Bayés de Luna A,et al. Ann Noninvasive Electrocardiol. 2007; 12:1-4.
6) Bayés de Luna A, et al. Cardiology Journal 2007;14 : 417-419.
7) Cino JM, et al. J Cardiovasc Magn Reson. 2006;8:335-44.
8) Pons-Lladó G, et al. J Cardiovasc Magn Reson. 2006;8:325-6.