Dyslipemic senior woman, 68 years old of age, admited in emergency room with typical chest pain

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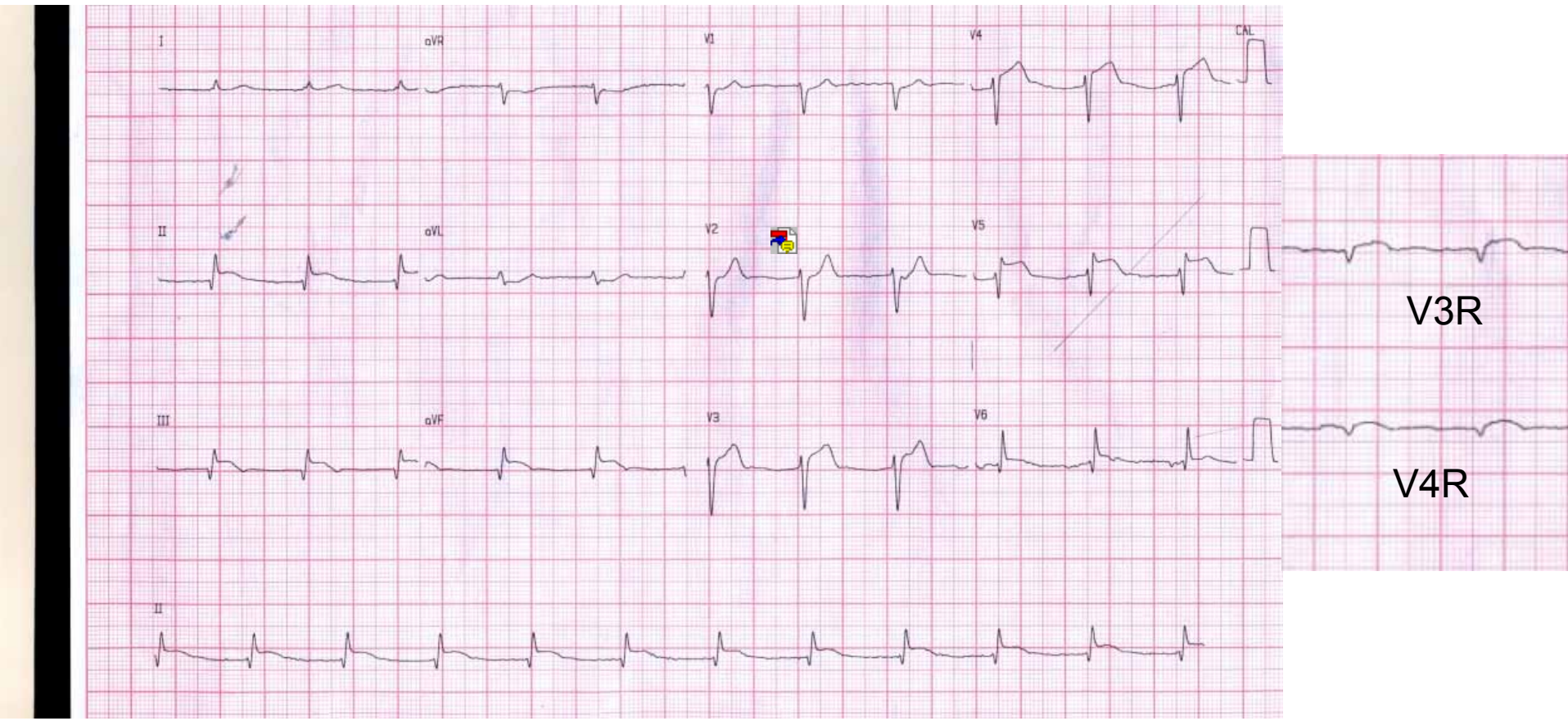
Mulher,68 anos, dislipedemia, admitida com dor no peito típica
Qual a artéria culpada?

Dyslipemic woman, 68 yo, was admited in ER with typical chest pain.
Which is the culprit artery a why?



Colleagues opinions

About this female patient with acute myocardial infarction, first the artery has started to reperfuse and masks the data a bit, because the final part of T waves of III and aVF is starting to get inverted. The culprit artery, very likely, is a dominant right coronary artery (RCA) that also irrigates the posterior side. The obstruction is proximal to the first right marginal artery because there is no ST-T depression in V_1 (is cancelled with the posterior right septal artery ischemia), and V_2 is not depressed due to being cancelled by posterior ischemia in II. The left lateral branches of the RCA irrigate the left anterolateral side, causing ST-T elevation in V_5, V_6, but also involving the anterior septum, because we observed elevated ST-T in V_3 and V_4, higher than V_6. The obstruction of the RCA is proximal to the artery of the sino atrial node (SAN) that originates 3 cm from the origin of the RCA, thus justifying the loss in sinus rhythm with supplying tachycardic nodal rhythm. The left circumflex artery(LCX) is very small, as well as the left anterior descending artery(LAD). These cases entail high mortality and a maximal emergency, in spite of showing a non-dramatic ECG.

In brief, it is a very proximal obstruction of a very dominant RCA that irrigates the inferior anterolateral and inferior anteroseptal sides in the presence of a very underdeveloped left system, as is usual in women. A hormonal analysis could be done, as to why this type of ischemia is occurring, but it would be long to explain.

The differential diagnosis is posed with the first electrocardiographic stage of a Tako-Tsubo that may present an identical ECG but with more signs of right obstruction. We recently described (1) the three ECG stages that develop in this syndrome, almost exclusive to post-menopausal women.

1. First stage: with a duration of only a few hours and manifest by acute circumferential epicardial ischemia.
2. Second stage: it also last a few hours, and is translated as very transient appearance of Q/S waves.
3. Third stage: characterized by reappearance of R waves with deep T waves inversion.

Samuel Sclarovsky MD Israel

1. **Sclarovsky S, Nikus K.The electrocardiographic paradox of tako-tsubo cardiomyopathy-comparison with acute ischemic syndromes and consideration of molecular biology and electrophysiology to understand the electrical-mechanical mismatching. J Electrocardiol. 2010 Mar-Apr;43:173-176.**

Con respecto a esta paciente con infarto agudo en principio la arteria comienza a reperdundirse y enmascara un poco los datos. Porque? Porque la parte final de las ondas T de III y aVF comienzan a invertirse.

La arteria culpada es muy probable que sea una coronaria derecha(CD) dominante que irriga también la cara posterior. La obstrucción es proximal a la primera arteria marginal derecha porque no hay depression del ST-T en V1 (se cancela con la isquemia de la septal derecha posterior) , y V2 no se deprime por esta cancelada por la isquemia posterior de II , La ramas lateral izquierda de la CD irrigan la cara anterolateral izquierda que ocasiona elevación de ST-T en V5 ,V6 ,pero tambien está comprometido el septo anterior por observarse ST-T elevado de V3 y V4 mas alto que V6.

La obstrucción de la CD es proximal a la arteria del nódulo sinusal que se origina a 3 cm del origen de la CD, lo que justifica la pérdida del ritmo sinusal con ritmo nodal taquicárdico de suplencia. La arteria circunfleja es muy pequeña como asi también la descendente anterior. Estos casos acarrean alta mortalidad y son de máxima emergencia a pesar de mostrar un ECG no dramático.

En fin obstrucción muy proximal de la arteria CD muy dominante que irriga la cara anterolateral inferior y anteroseptal inferior en presencia de un sistema izquierdo muy poco desarrollado como suele ocurrir con frecuencia en mujeres. Caberia un análisis hormonal del porque se produce este tipo de isquemia pero seria largo de explicar.

El diagnóstico diferencial se plantea con la primera etapa electrocardiográfica de un Tako-Tsubo que puede presentar un ECG idéntico pero con mas signos obstrucción derecha. Nosotros recientemente describimos (1) las tres etapas electrocardiográficas que se desarrollan en este sindrome casi exclusivo de mujeres postmenopáusicas.

1. **Primera etapa: con duración de apenas unas horas y manifiestado por isquema aguda circunferencial epicárdica,**
2. **Segunda etapa: también dura algunas horas y se traduce por aparición muy transitória de ondas Q /S**
3. **Tercera etapa: caracterizada por la reaparición de las ondas R con inversión de ondas T profundas.**

1. **Sclarovsky S, Nikus K.The electrocardiographic paradox of tako-tsubo cardiomyopathy-comparison with acute ischemic syndromes and consideration of molecular biology and electrophysiology to understand the electrical-mechanical mismatching. J Electrocardiol. 2010 Mar-Apr;43:173-176.**

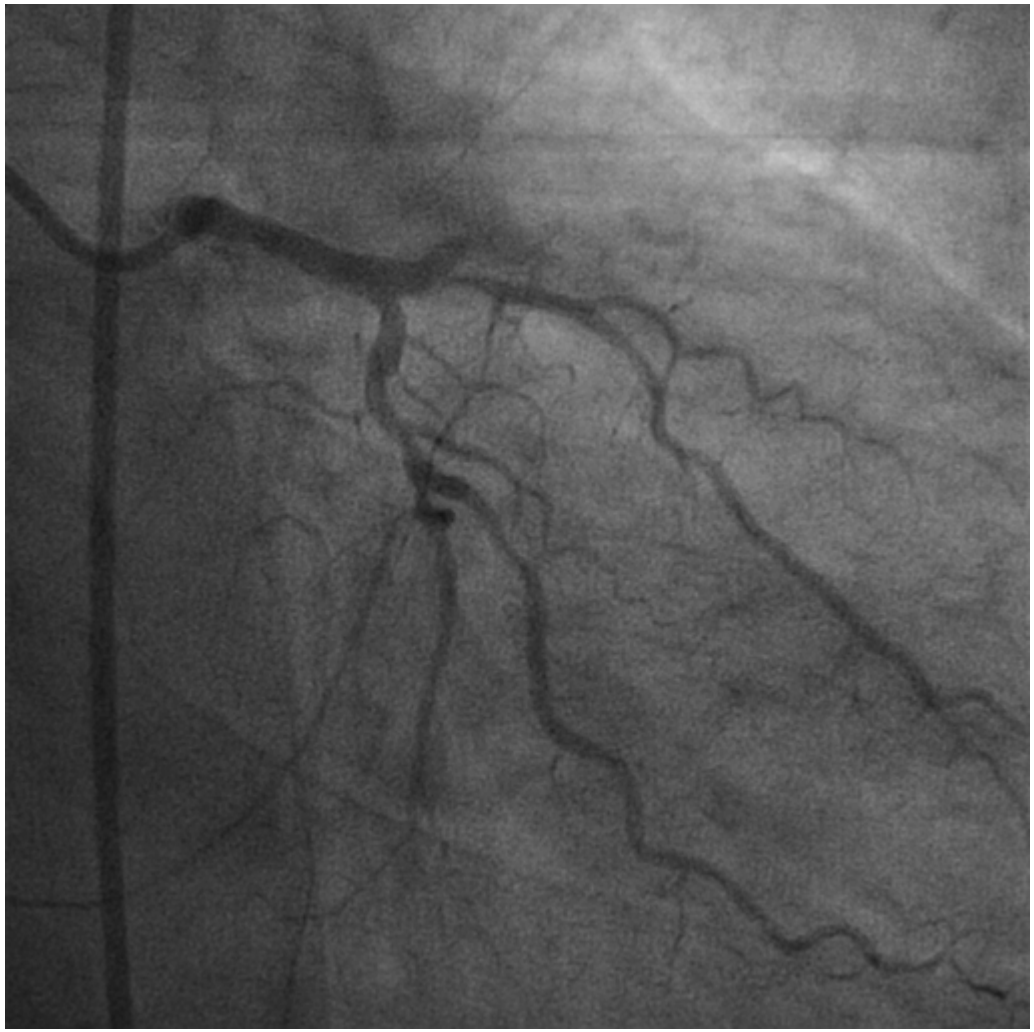
English

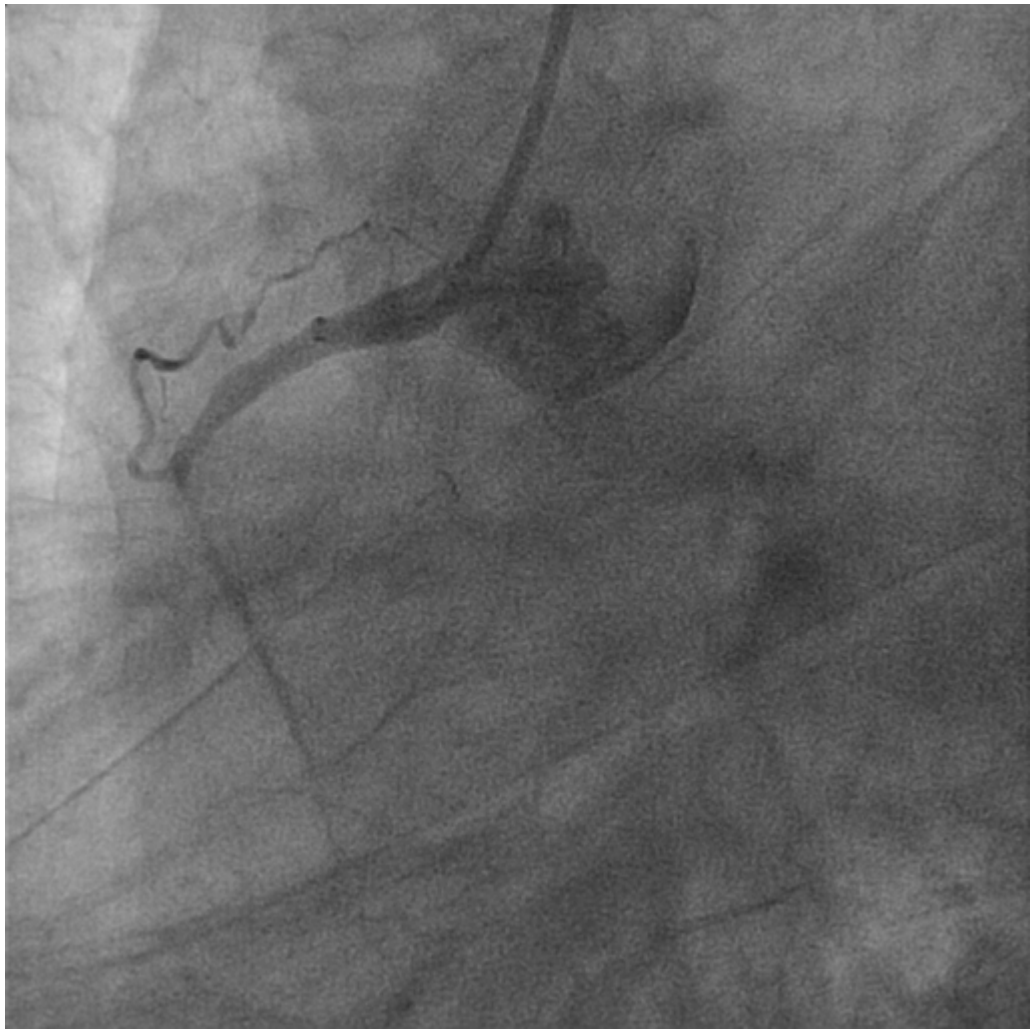
Again a challenging case. Could be LCx occlusion based on 12-lead ECG: J-point elevation higher in LII than in LIII and J-point depression deeper in aVR than in aVL and isoelectric ST in LI. However ST elevation in V3R and V4R indicates right ventricular transmural ischemia. Then there is probably either proximal occlusion of a dominant RCA or an occlusion distal to the first obtuse marginal branch of a dominant LCx, which irrigates the right ventricle. Then there is the strange QRS configuration in V5-V6: rSR', which I am not sure about - could it have to do with rotation of the heart or electrode placement? There is an initial r wave in V1 and thus there should normally be an intial q in V6.

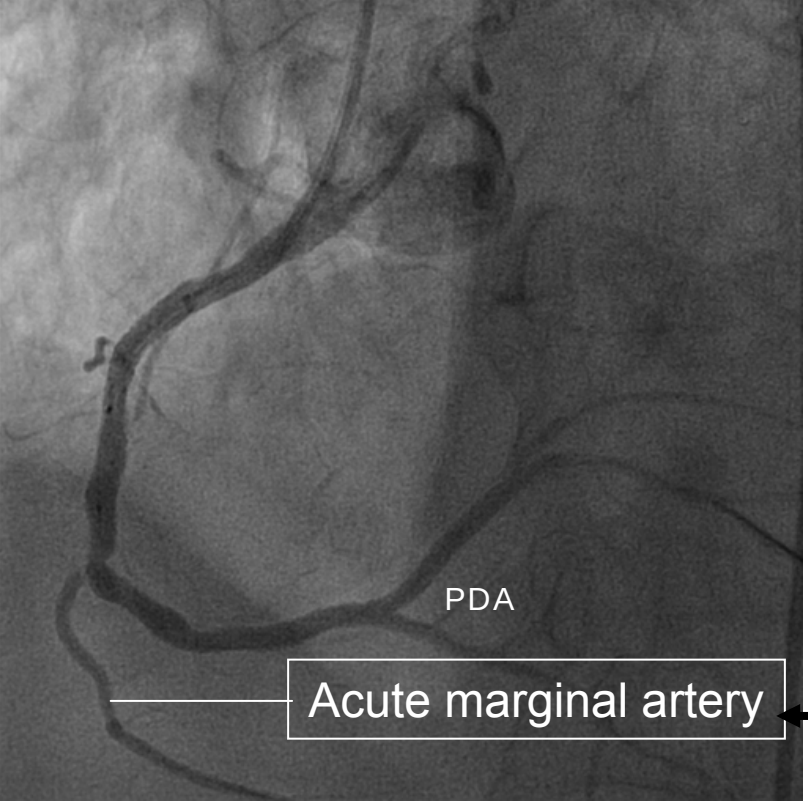
Kjell Nikus, Tampere, Finland

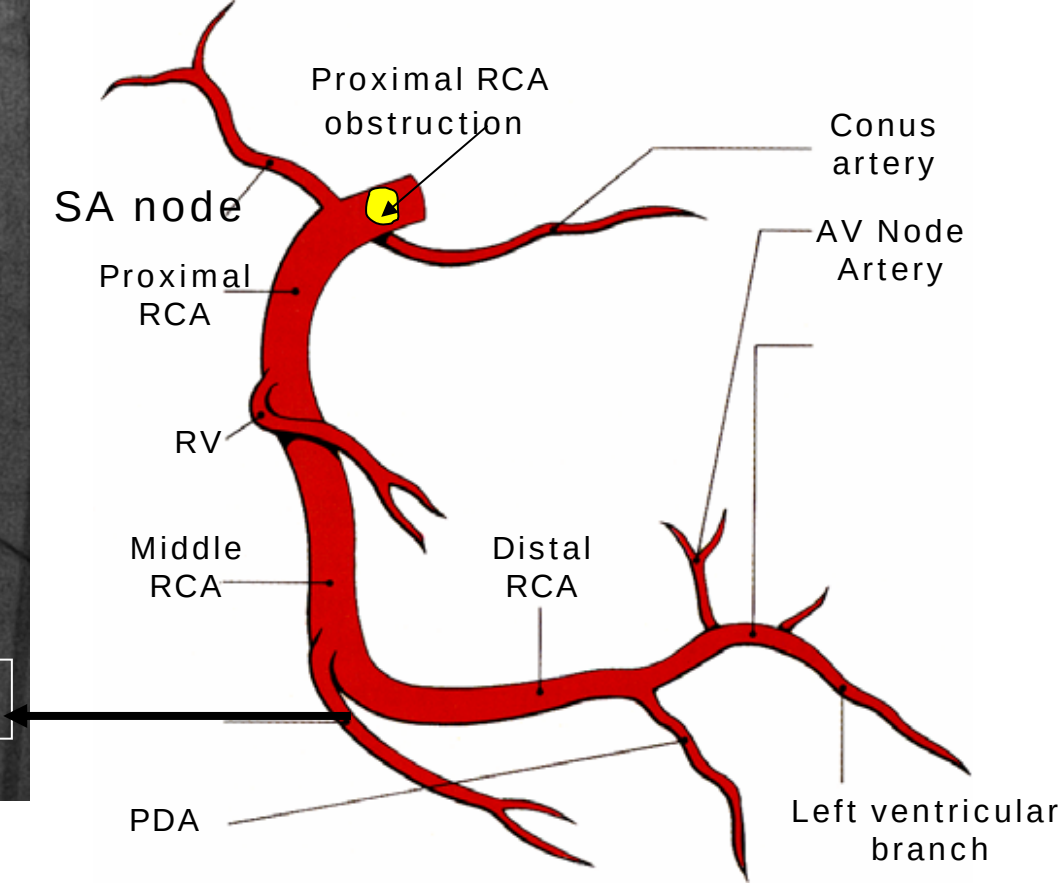
Spanish

Una vez más un caso difícil. Podría ser la oclusión LCx sobre la base de ECG de 12 derivaciones: mayor elevación del punto J en DII > DIII, depresión más profunda del punto J en aVR que en aVL y ST isoeléctrico en DI. Sin embargo, la elevación del ST en V3R y V4R indica isquemia transmural del ventrículo derecho, consecuentemente es probable que sea una oclusión proximal de una artéria coronaria derecha dominante o una oclusión distal a la primera rama marginal obtusa de una CX dominante, que irriga el ventrículo derecho.
Luego está la extraña configuración del QRS en V5-V6: rSR', que no estoy seguro – pero podrían tener que ver con la rotación del corazón o con la colocación de electrodos?
Hay una onda r en V1 y por lo tanto debe corresponder a la primera q en V6.
Kjell Nikus, Tampere, Finland









DOMINANT RCA

The artery that supplies the posterior descending artery (PDA) determines the coronary dominance.

If the posterior descending artery (PDA) is supplied by the RCA, then the coronary circulation can be classified as "right-dominant". Approximately 70% of the general population are right-dominant,

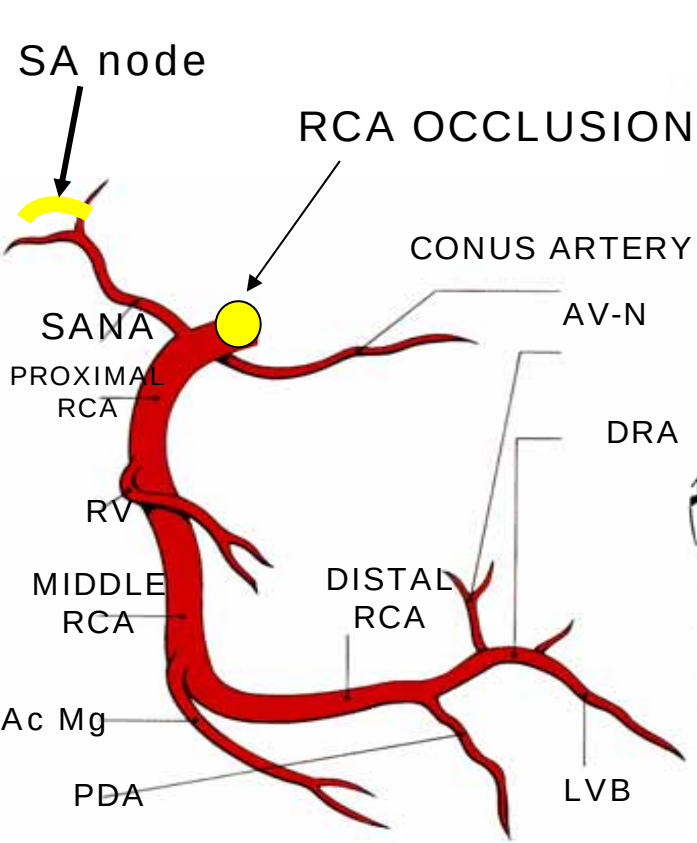
If the PDA is supplied by the LCX then the coronary circulation can be classified as "left-dominant". and 10% are left-dominant.

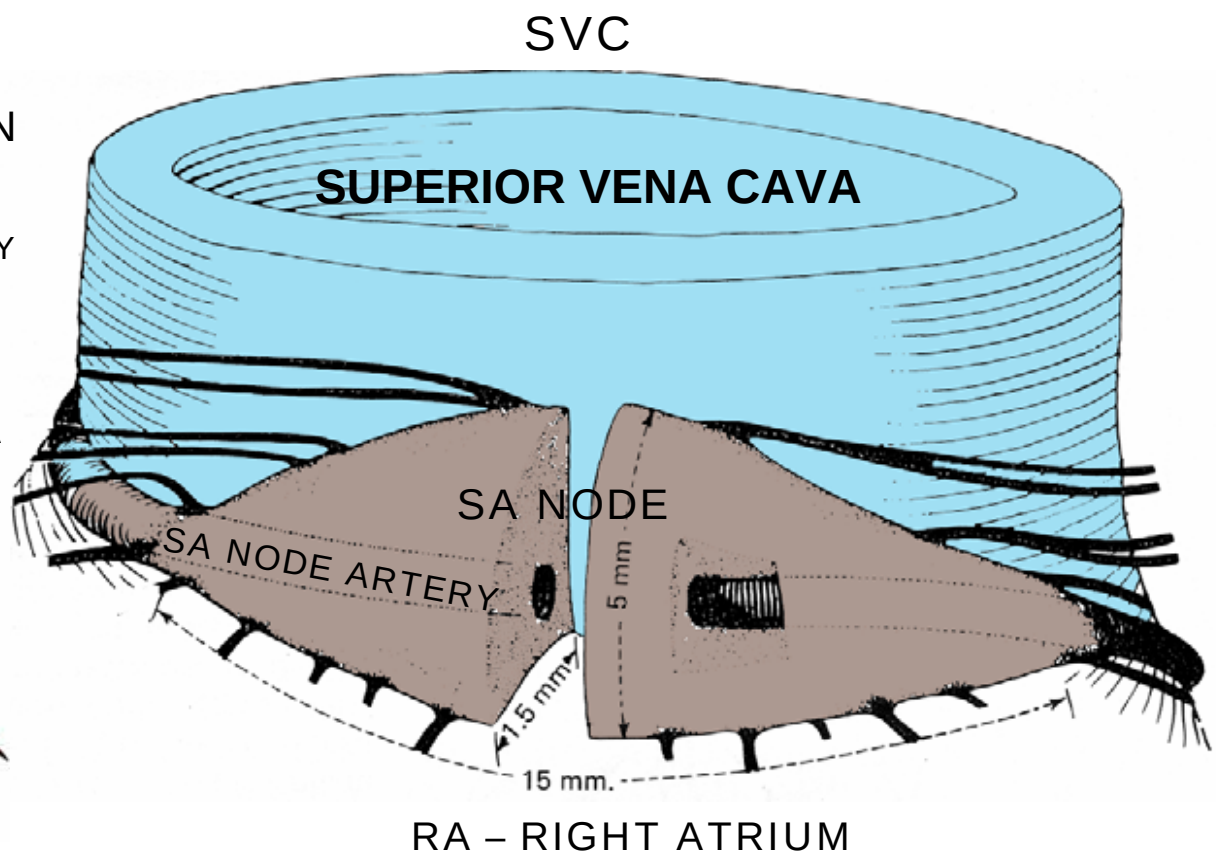
If the PDA is supplied by both the RCA and the LCX artery, then the coronary circulation can be classified as "co-dominant". 20% are co-dominant.

A precise anatomic definition of dominance would be the artery which gives off supply to the AV node i.e. the AV nodal artery. Most of the times this is the RCA.

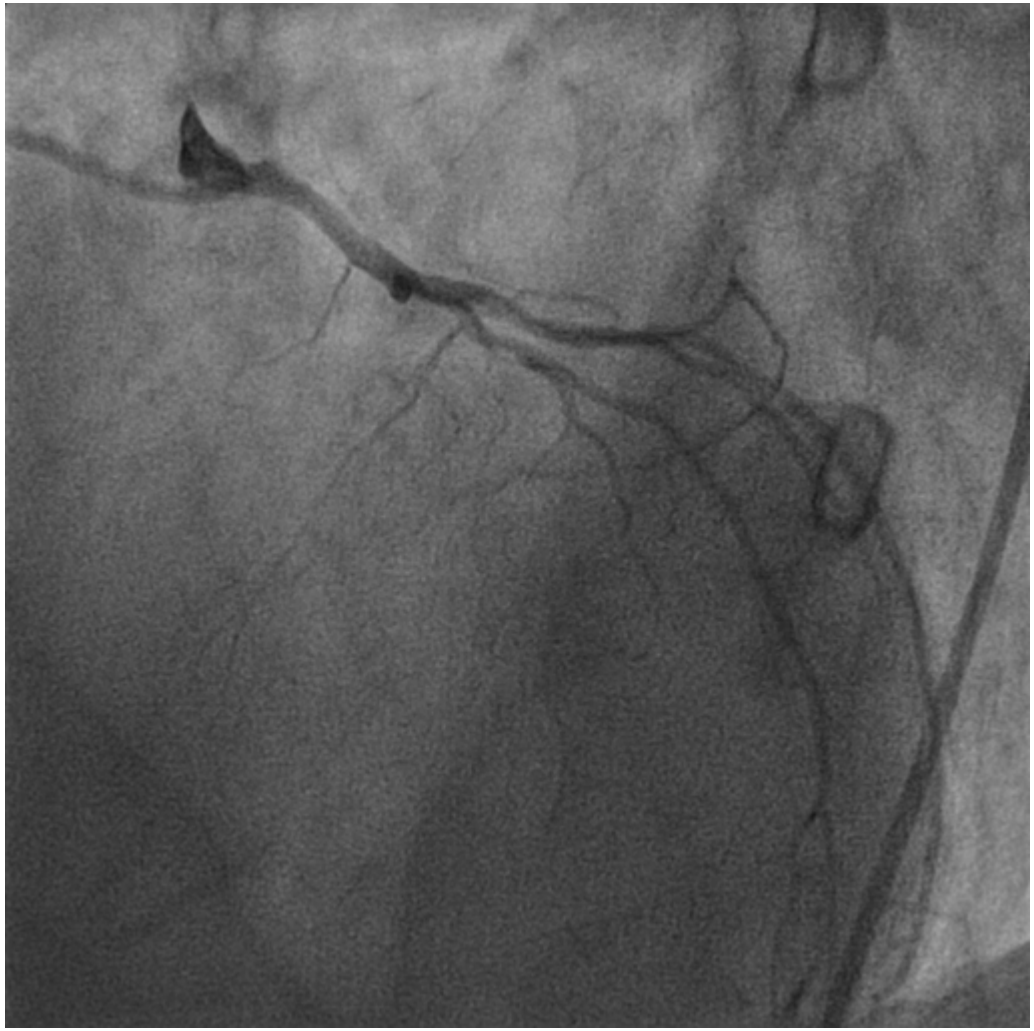
BLOOD SUPPLY OF SA-NODE

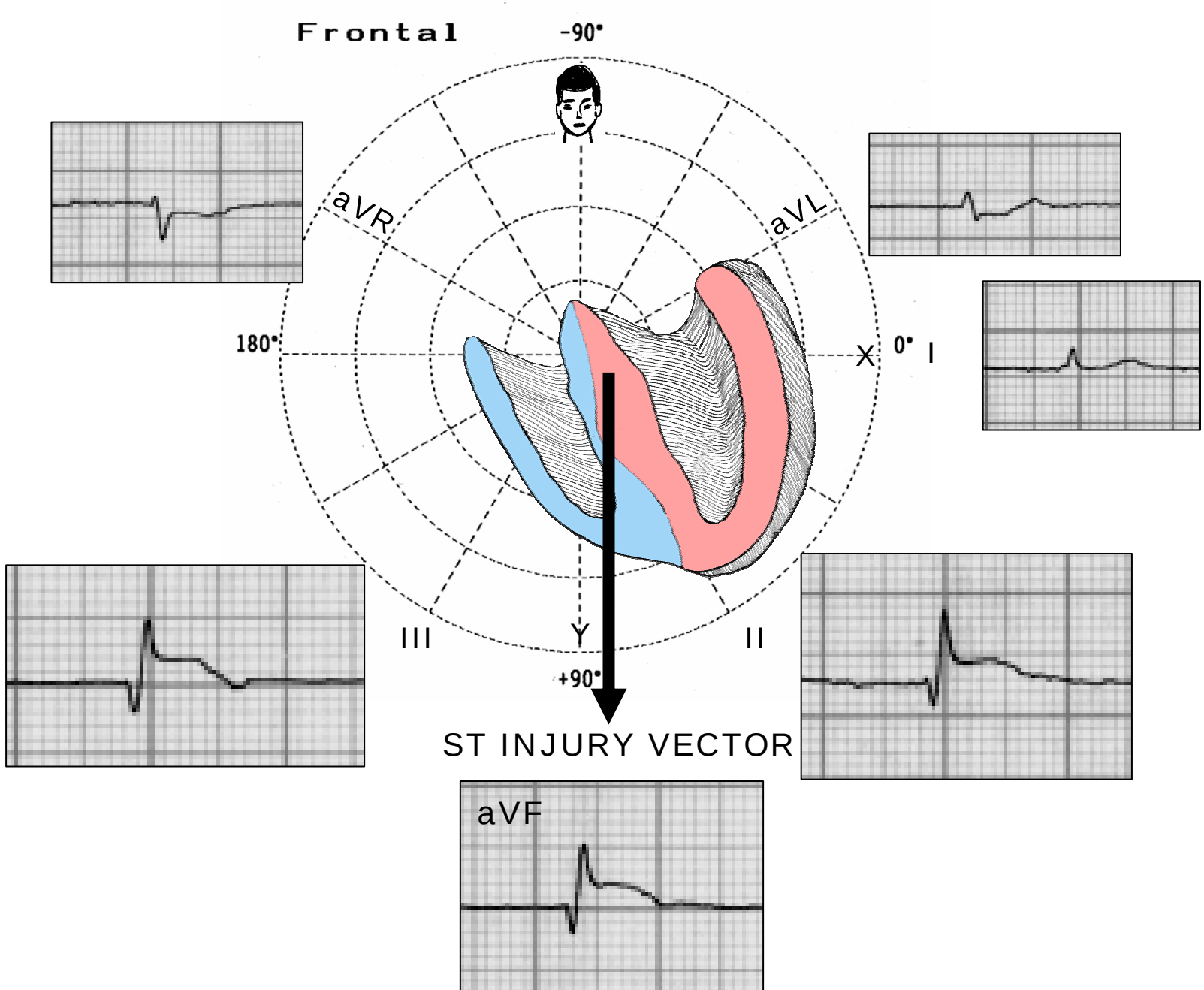
In the majority of individuals (≈ 59% of cases), the SA-node receives blood from a SA node artery. This is the second branch of the RCA (the first one is the conus artery) and in 38% of cases from the LCX and from both arteries in 3%[1].



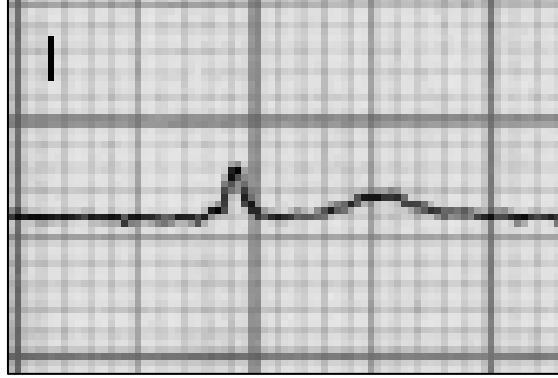


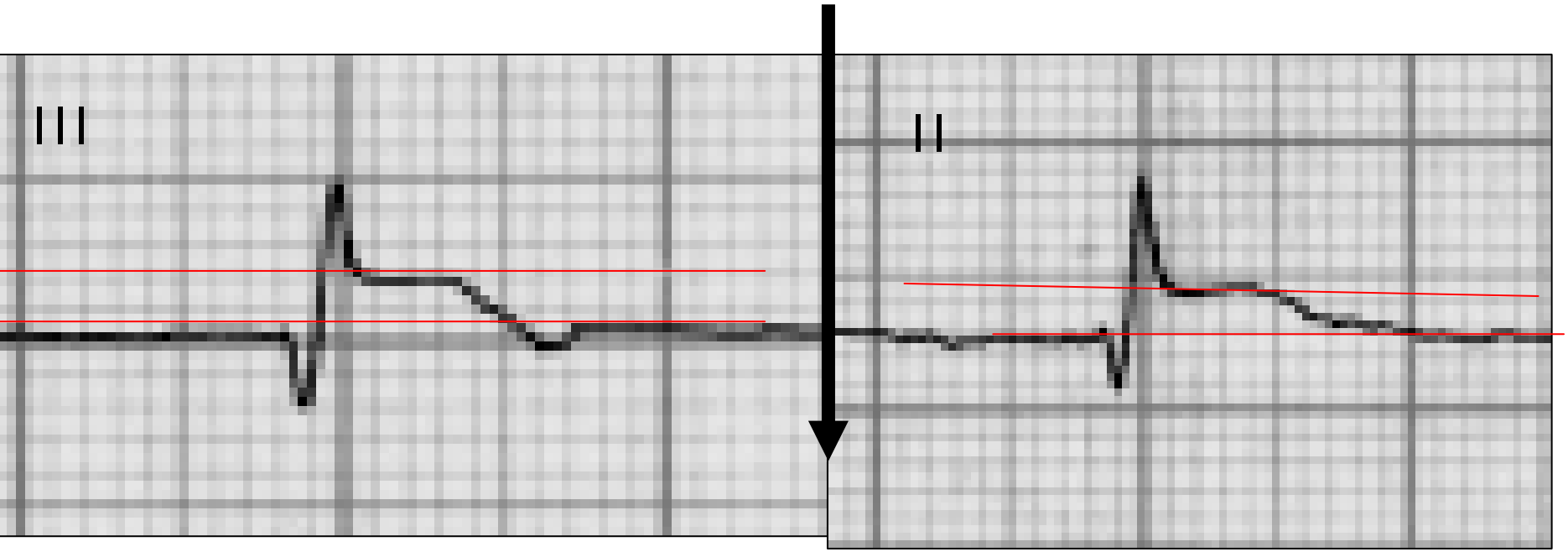
1. **Kyriakidis MK, Kourouklis CB, Papaioannou JT, Christakos SG, Spanos GP, Avgoustakis DG. Sinus node coronary arteries studied with angiography. Am J Cardiol. 1983 Mar 1;51:749-750.**




Frontal
-90°
aVR
aVL
180°
0°
I
X
III
Y
II
+90°
ST INJURY VECTOR
aVF

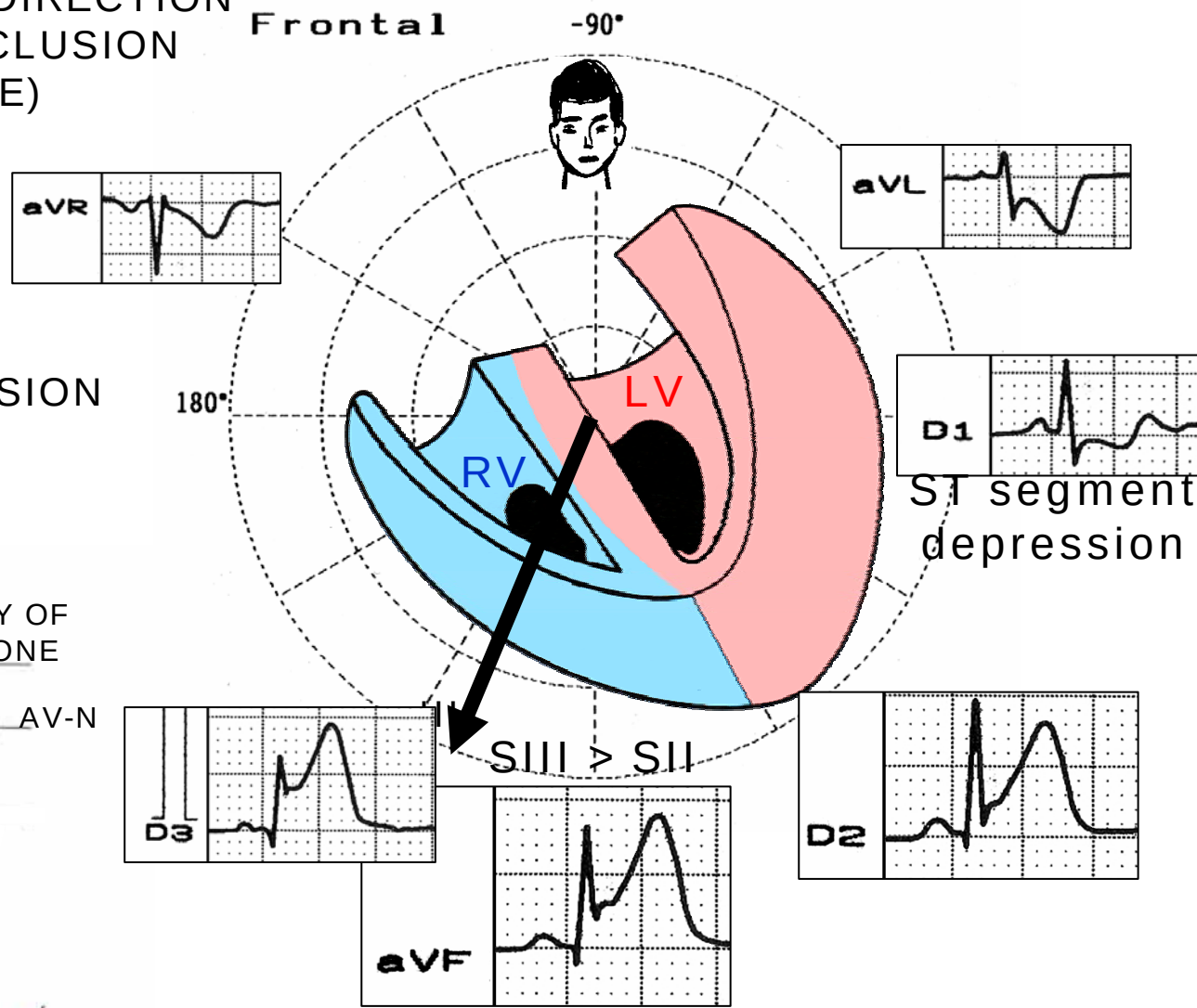
THE ST INJURY VECTOR DIRECTED TO DOWNWARD PERPENDICULAR TO I:
THE ST SEGMENT IS NOT DEVIATED IN I LEAD
IN CLASSIC RCA OCCLUSION THE ST SEGMENT IN I LEAD IS DEPRESSED



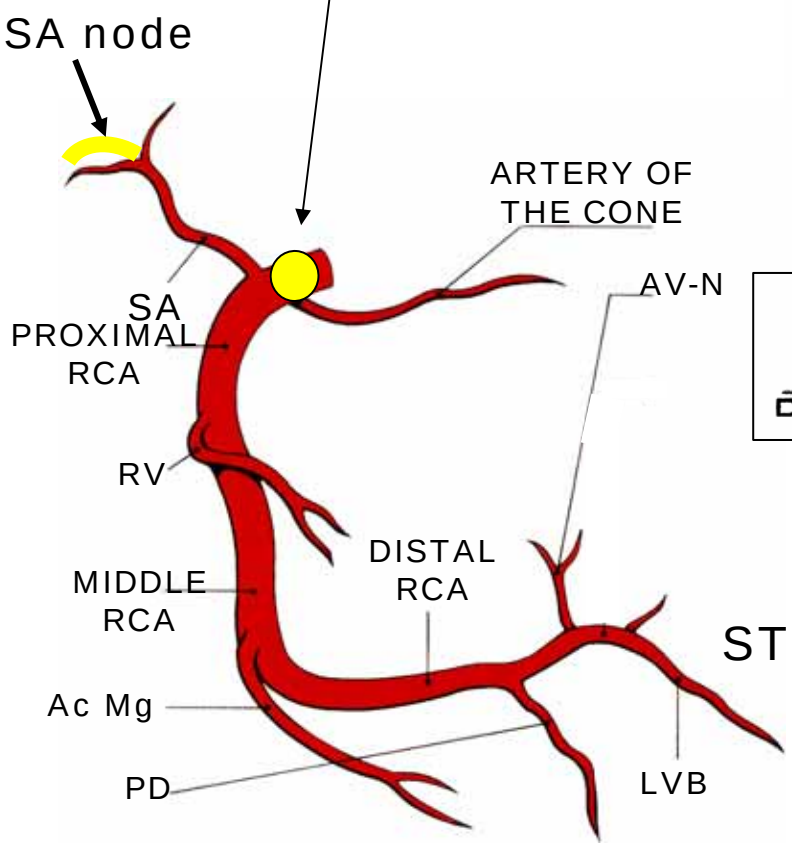


ST segment elevation in inferior leads
STIII=STII
In classic or conventional RCA proximal occlusion STIII>STII (please see the next slide). Here we have in association LAD occlusion

THE ST INJURY VECTOR DIRECTION IN PROXIMAL RCA OCCLUSION (FRONTAL PLANE)

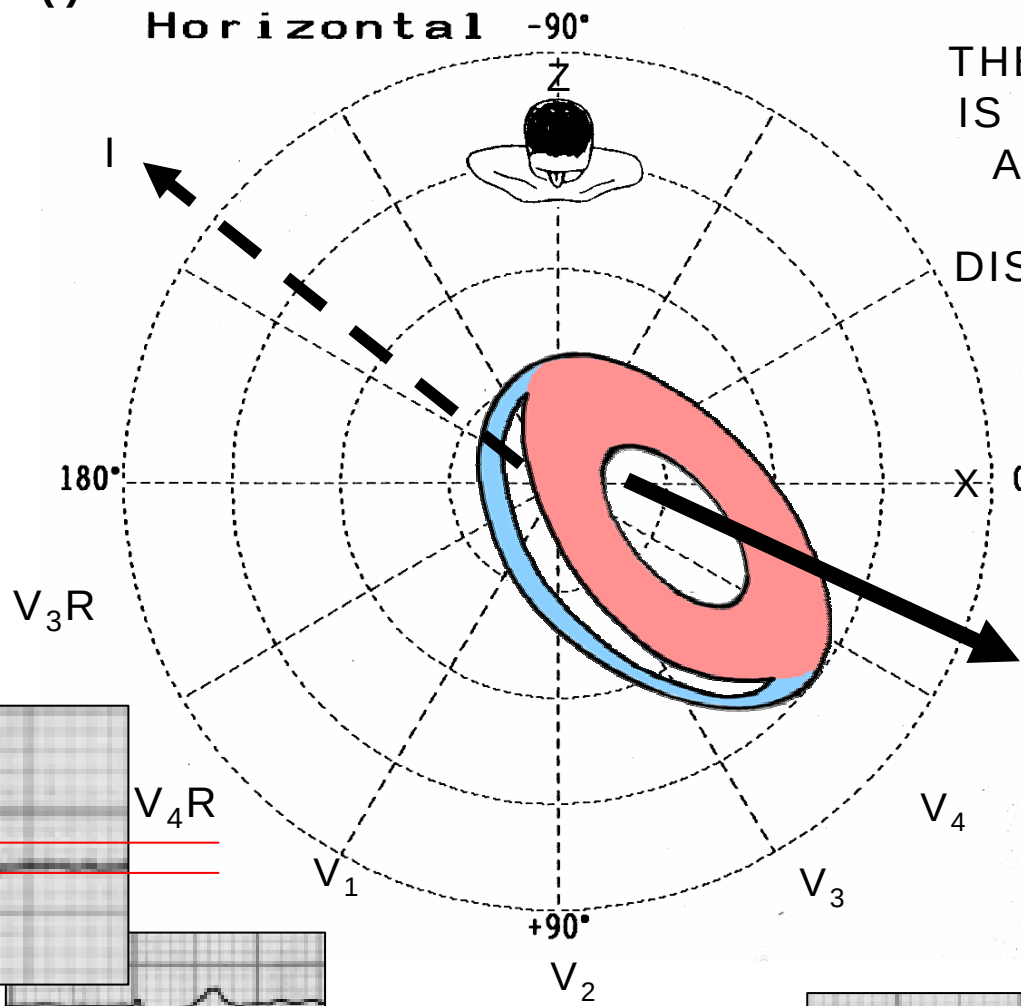


PROXIMAL RCA OCCLUSION



THE ST INJURY VECTOR DIRECTED TO DOWNWARD AND TO RIGHT

IN CLASSIC RCA PROXIMAL OCCLUSION THE ST INJURY VECTOR IS DIRECTED TO BACK AND RIGHTWARD(I)



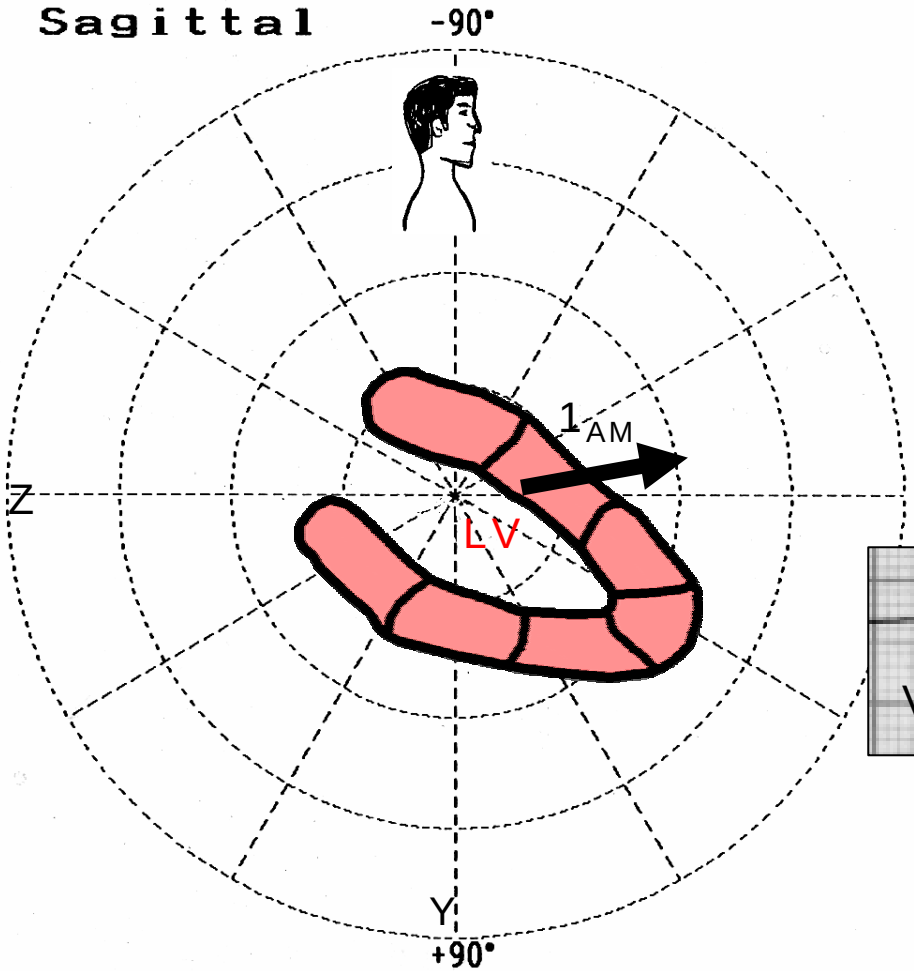
THE ST INJURY VECTOR IS DIRECTED TO FRONT AND TO LEFWARD(II) TYPICAL OF DISTAL LAD OCCLUSION

Voltage of R in V5- V6 of less amplitude

ST segment elevation in V_4R followed by positive T wave: indicative of RV involvement

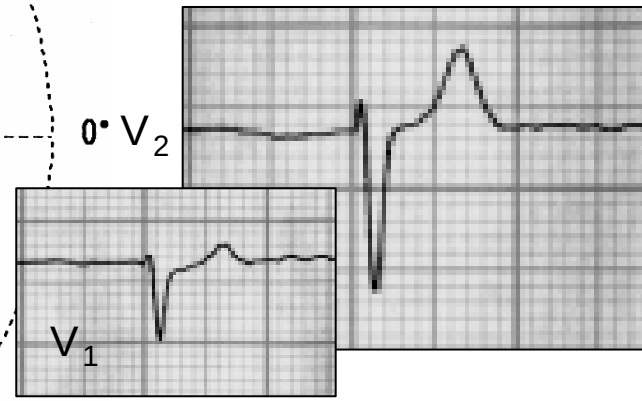
The middle-septal activation is preserved (1_{AM} vector*), explaining the q waves of the left V_5-V_6 leads. The activation of center of the left side of the interventricular septum, which originates the first septal vector, 1_{AM} vector or Peñaloza and Tranchesi vector. ***(1)***

RIGHT SAGITTAL VIEW

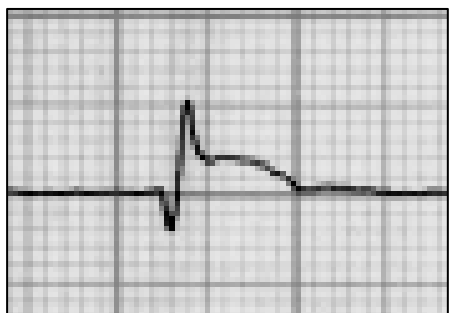


aVF

rS pattern V_1-V_2



The first QRS vector is preserved: middle septal region is undamaged



Inferior acute MI with ST segment Elevation in inferior lead

1. **Penaloza D, Tranchesi J. The three main vectors of the ventricular activation process in the normal human heart. I. Its significance. Am Heart J. 1955; 49: 51-67.**

- 1_{AM}: First anteromedial
- vector.

Our physiopathological speculation for the present case

From the electrocardiographic point of view this patient suggests (with atypical directions of the injury vector previously indicated) that the culprit artery is a dominant proximal right coronary artery (RCA) occlusion. In favor of this hypothesis are the following observations:

1. The loss of sinus rhythm that suggests obstruction of the RCA before the SA node
2. The absence of ST segment depression in V_1 and V_2
3. The ST segment elevation in V_3R and V_4R indicative of associated right ventricular free wall acute myocardial infarction.

The cardiac catheterization study revealed total obstruction of the left anterior descending artery (LAD) in association with proximal occlusion of RCA which in our opinion justifies the atypical directions of the injury vectors in both planes (frontal and horizontal.)

The interventionist made the choice to perform angioplasty with implantation of two stents in the RCA.

Unfortunately, the clinical evolution was not favorable and the patient remains hospitalized with heart failure. (Raimundo´s information).

Our pathophysiological reasoning about the clinical/ECG events is as follows:

The patient would have had previously LAD obstruction which was not manifested by anterior MI because collateral supply from dominant RCA. Coronary collateralization is a normal response to hypoxia and may be induced, under some circumstances, by exercise. It is considered to be protective. Coronary collateralization exists latently in the normal heart.

In the moment that occurs the acute occlusion the RCA immediately the anteroapical wall supplied by the collaterals from RCA is affected with lack of collateral irrigation and pump failure by concomitant anterior myocardial infarction.

Microscopic collateral vessels of the heart undergo a process called *transformation* that widens the vessel lumen at the expense of its cell wall in response to myocardial stresses—specifically, myocardial spasm and hypoxia secondary to MI or acutely stressful exercise. The status of the coronary collaterals has also been shown to be influenced by the presence of diabetes mellitus.(1) The functional significance of the coronary collateral vessels is a matter of continuing experimental investigation although their existence has been known for over three centuries and been documented repeatedly in man and beast over the past seven decades. Although a now-classic series of experiments by Schaper(2) in the late 1960s and '70s expanded our understanding of the mechanisms by which these usually redundant, microscopic (40-10 um in diameter in their native state) ur-arterioles are transformed by ischemia or stenosis into vessels with life-preserving blood capacity,(3), equally as many studies have denied the function of these vessels to preserve myocardium by salvaging tissue perfusion and maintaining blood pressure as have documented this. It was only during the 1980s that a consensus among researchers was reached that these vessels can preserve as much as 30 to 40% of coronary blood flow to an otherwise-occluded blood vessel, and, while not capable of preventing ischemia in the event of high-output exercise, can nevertheless maintain aortic, pulmonic, and atrial blood pressure, redirect ST elevation into less serious ST depression in ischemia,(4) and prevent infarction and symptoms of infarction, even in the case of complete left main coronary artery occlusion.

1. Kilian JG, Keech A, Adams MR, Celermajer DS. Coronary collateralization: determinants of adequate distal vessel filling after arterial occlusion. Coron Artery Dis. 2002 May;13:155-159.
2. Schaper W, The collateral circulation of the heart, New York, N.Y.: Elsevier, 1971.
3. Kolibash AJ, et al., "Coronary collateral vessels: spectrum of physiologic capabilities with respect to providing rest and stress myocardial perfusion, maintenance of left ventricular function, and protection against infarction," American Journal of Cardiology 1982; 50: 230-238.
4. Yamagsihi M, "The functional significance of transient collaterals during coronary artery spasm," American Journal of Cardiology 1985; 56: 407-12.

The native collaterals are small vessels, with a narrow endothelial lining, a layer or two of smooth muscle, and a variable amount of elastic tissue. They are rarely if ever observed during angiography in the absence of severe ischemia (vessels less than 200 micrometers are not visible, generally), and only coronary stenosis, anemia, and exercise have experimentally been shown to cause transformation.(1) Most observers agree that a 90% occlusion is necessary to bring about transformation in the absence of other factors, though a recent article suggests that they may appear as a result of coronary spasm in the absence of total occlusion (see below). Within ninety seconds of occlusion, the pressure gradient between the segment of the coronary vessel distal to the occlusion and the incipient collateral vessel precipitates damage to the internal elastic lamina, provoking an inflammatory response; monocytes and polycytes migrate to the vascular wall, which has, as a result of the occlusion, become permeable to the blood's cellular components.(2)

The internal diameter of these vessels expands exponentially in the first hours and days following an occlusion, as mitotic division of the cell wall narrows the wall's diameter and expands each vessel's lumen.Within four weeks, the functional capacity of the vessels has reached a maximum, accompanied by a 90% reduction in their resistance, though structural remodeling continues by cell proliferation and synthesis of elastin and collagen over a period of up to six months.

1. **Yamagsihi M, "The functional significance of transient collaterals during coronary artery spasm," American Journal of Cardiology 1985; 56: 411.**
2. **Freedman SB, et al., "Influence of coronary collateral blood flow on the development of exertional ischemia and Q wave infarction in patients with severe single-vessel disease," Circulation 1985; 71: 681-686.**

Schaper(1) summarizes the status-2009 knowledge of coronary collateral transformation "Following an arterial occlusion outward remodeling of pre-existent inter-connecting arterioles occurs by proliferation of vascular smooth muscle and endothelial cells. This is initiated by deformation of the endothelial cells through increased pulsatile fluid shear stress (FSS) caused by the steep pressure gradient between the high pre-occlusive and the very low post-occlusive pressure regions that are interconnected by collateral vessels. Shear stress leads to the activation and expression of all nitric oxide synthetase (NOS) isoforms and nitric oxide production, followed by endothelial vascular endothelial growth factor (VEGF) secretion, which induces monocyte chemoattractant protein-1 (MCP-1) synthesis in the endothelium and in the smooth muscle of the media. This leads to attraction and activation of monocytes and T-cells into the adventitial space (peripheral collateral vessels) or attachment of these cells to the endothelium (coronary collaterals). Mononuclear cells produce proteases and growth factors to digest the extra-cellular scaffold and allow motility and provide space for the new cells. They also produce NO from inducible nitric oxide synthetase (iNOS), which is essential for arteriogenesis. The bulk of new tissue production is carried by the smooth muscles of the media, which transform their phenotype from a contractile into a synthetic and proliferative one. Important roles are played by actin binding proteins like actin-binding Rho-activating protein (ABRA), cofilin, and thymosin beta 4 which determine actin polymerization and maturation. Integrins and connexins are markedly up-regulated. A key role in this concerted action, which leads to a 2-to-20 fold increase in vascular diameter, depending on species size, are the transcription factors AP-1, egr-1, carp, ets, by the Rho pathway and by the mitogen activated kinases ERK-1 and -2. In spite of the enormous increase in tissue mass, the degree of functional restoration of blood flow capacity is incomplete and ends at 30% of maximal coronary conductance and 40% in the vascular periphery. The process of arteriogenesis can be drastically stimulated by increases in FSS (arterio-venous fistulas) and can be completely blocked by inhibition of NO production, by pharmacological blockade of VEGF-A, and by the inhibition of the Rho-pathway. Pharmacological stimulation of arteriogenesis, important for the treatment of arterial occlusive diseases, seems feasible with NO donors.“

1) Schaper W. Basic Research in Cardiology. 2009 Jan;104:5-21.

As nossas especulações fisiopatológicas para o presente caso

Do ponto de vista eletrocardiográfico esta paciente sugere (com as atipías previamente assinaladas em relação a direção do vetor de lesão em ambos os planos) que a artéria culpada do presente infarto agudo seja a região proximal de uma artéria coronária direita (CD) dominante.

Em favor desta hipótese estão as seguintes observações:

1) A perda do ritmo sinusal que sugere obstrução da coronária direita antes da artéria do Nó SA
2) A ausência de depressão do segmento ST em V_1 e V_2
3) A elevação do segmento ST em V_3R e V_4R indicativo de infarto de VD associado.

O estudo hemodinâmico revelou concomitante obstrução total da artéria descendente anterior, o que a nosso entender justifica as atipías eletrocardiográficas encontradas em referencia a direção do vetor de lesão em ambos os planos.

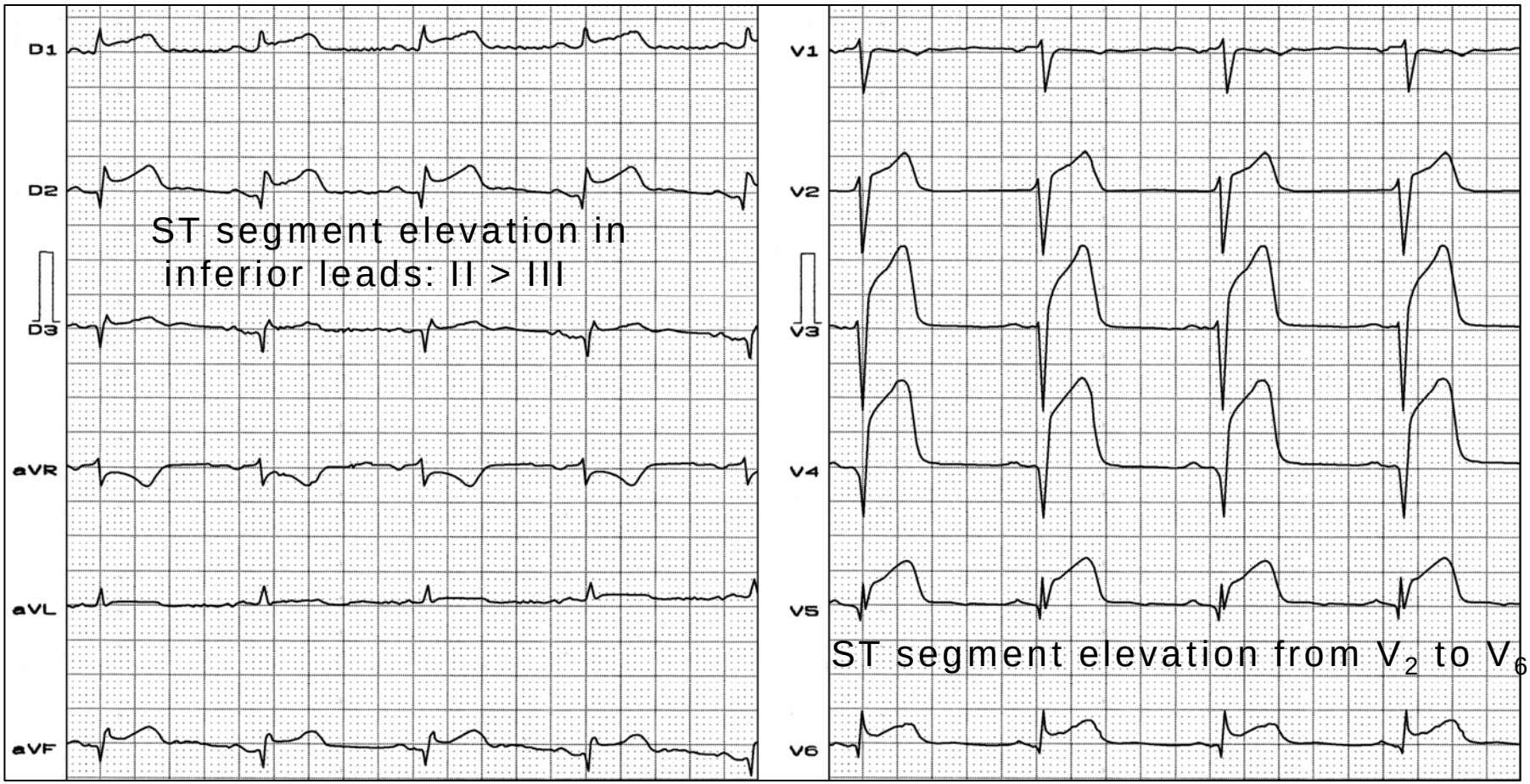
O hemodinamicista fez a opção por realizar angioplastia com implante de dois stents na coronária direita.

A evolução desafortunadamente não foi favorável e a paciente permanece internada com quadro de insuficiência cardiaca.

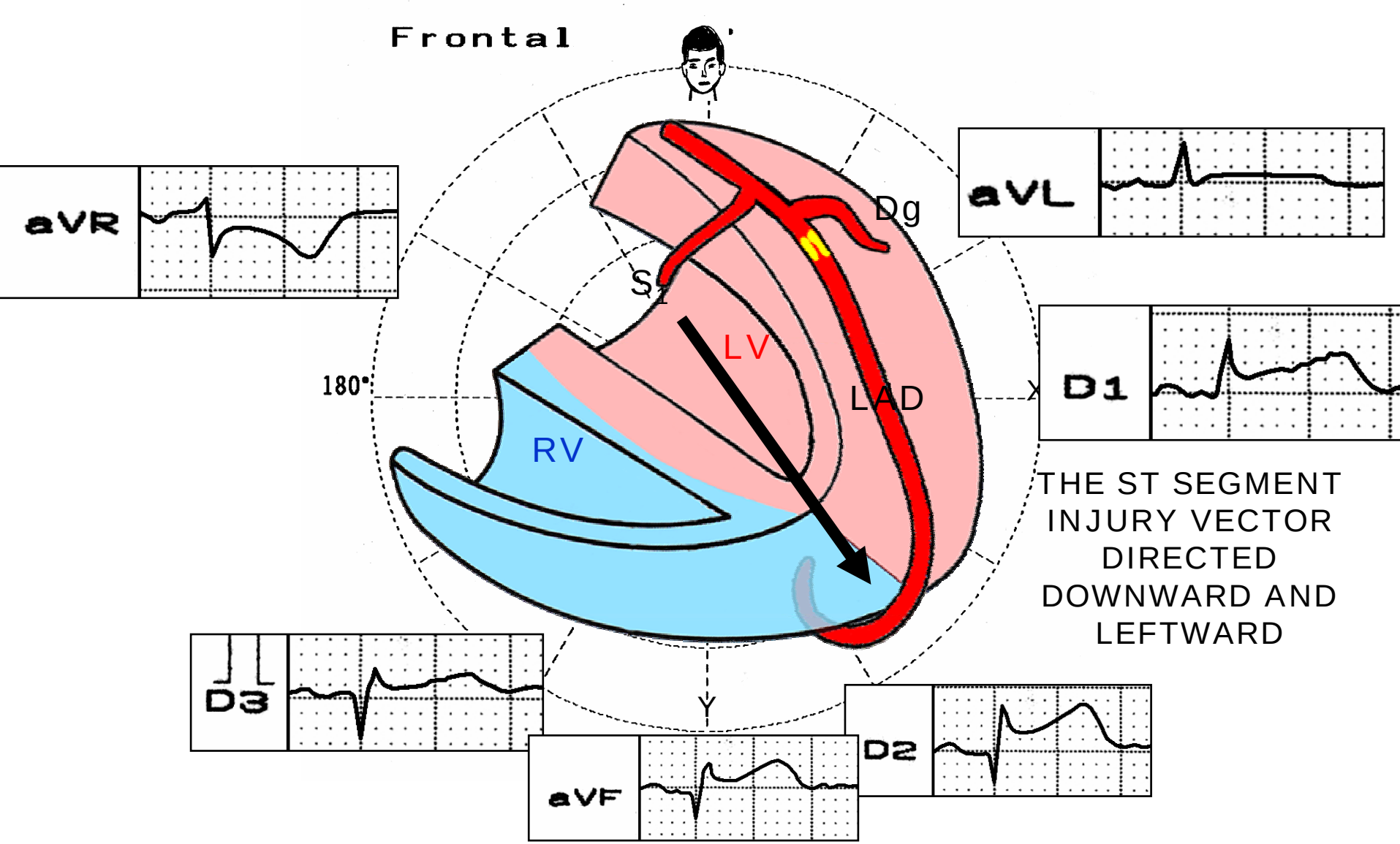
Nosso raciocinio fisiopatológico dos eventos é o seguinte: A paciente teria tido em tempos passados obstrução da DA, que não se manifestara por infarto anterior pela grande dominância da CD que supriria de sangue por colaterais a parede antero-apical.

No momento em que ocorre o infarto agudo por oclussão da CD de imediato a parede anterolateral baixa o apical suprida pelas colaterais da CD manifestaram sua falta de irrigação e falha de bomba por infarto anterior apical e inferior extenso por oclusão distal da artéria DA.

LEFT ANTERIOR DESCENDING ARTERY (LAD) OCCLUSION AFTER BOTH FIRST SEPTAL PERFORATOR AND FIRST DIAGONAL BRANCH (LAD DISTAL OBSTRUCTION)

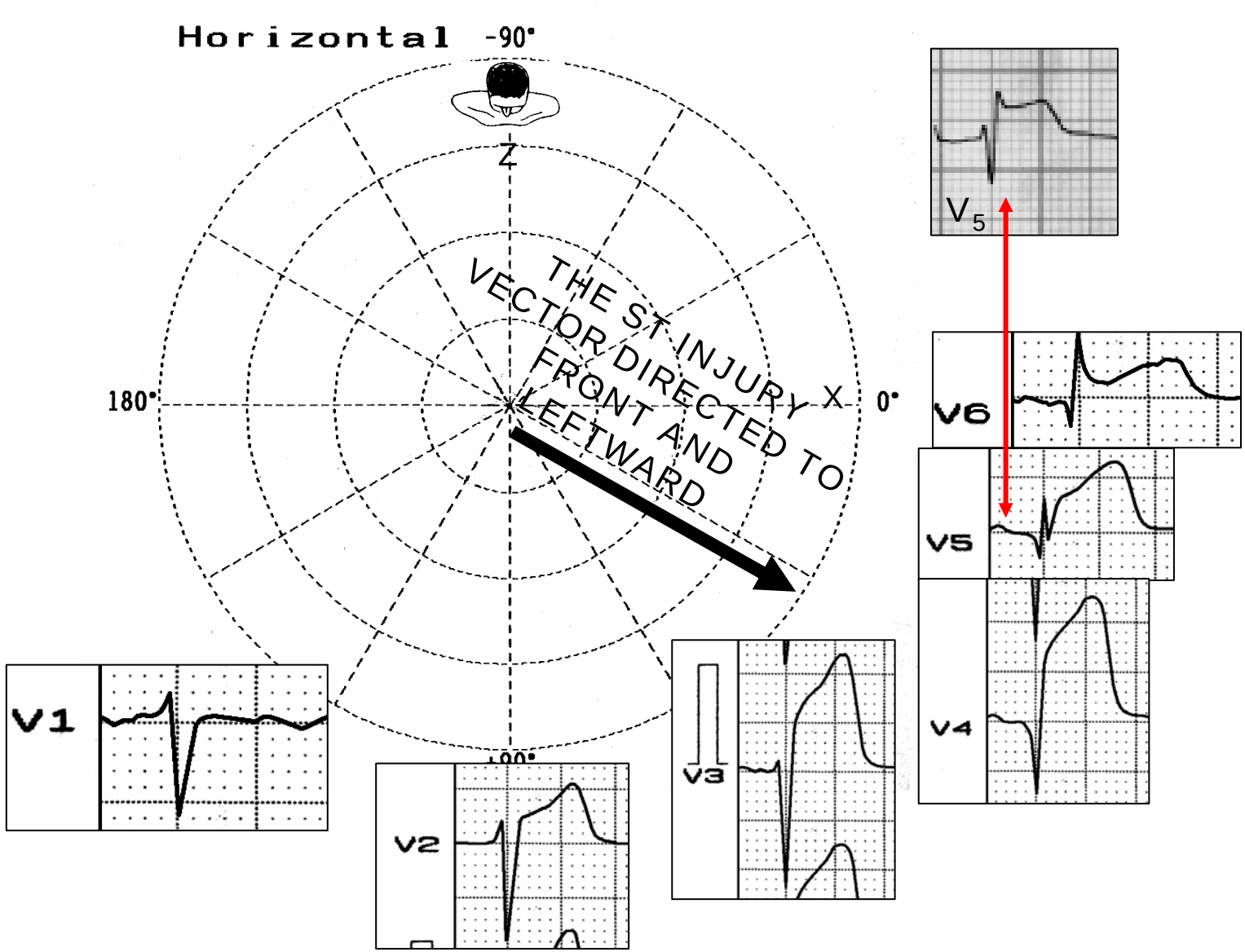


Why this pattern ?

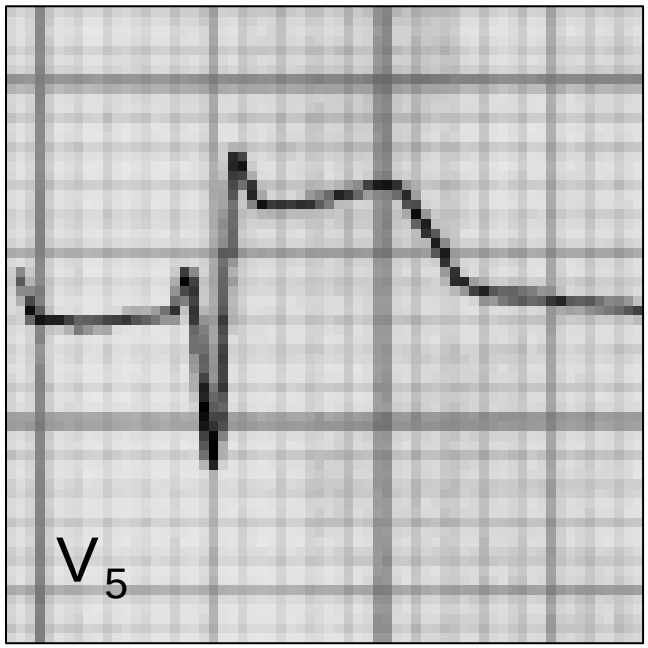


ST segment elevation in inferior leads: ST II > ST III

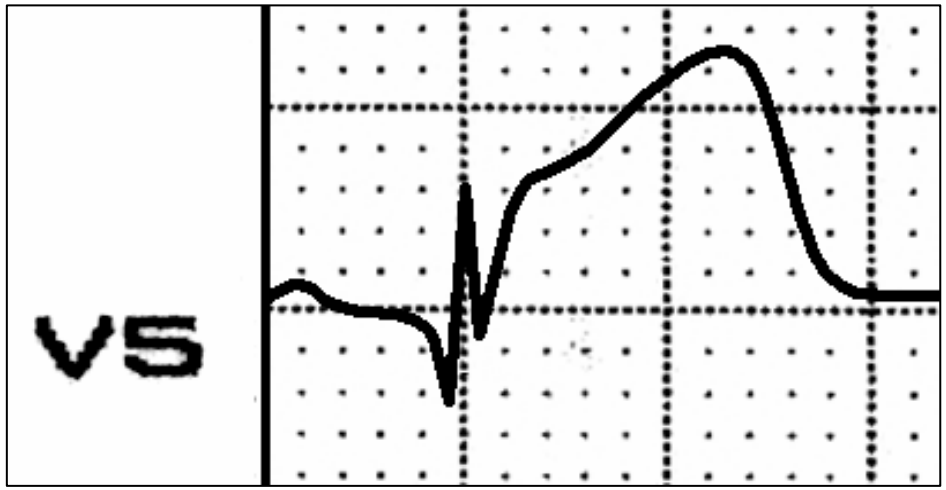
The present case very similar to



THE PRESENT CASE



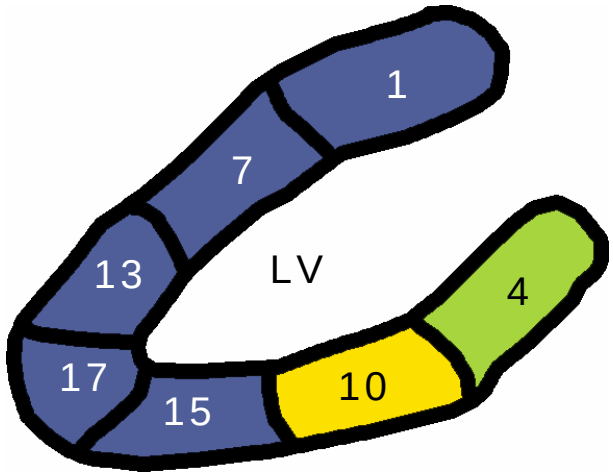
LAD DISTAL OBSTRUCTION

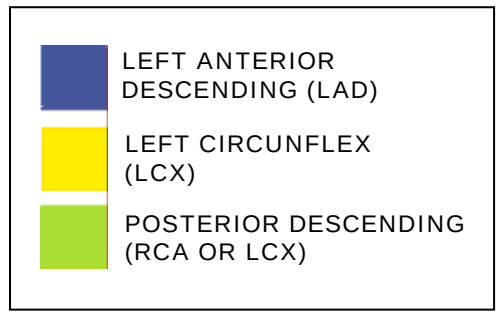


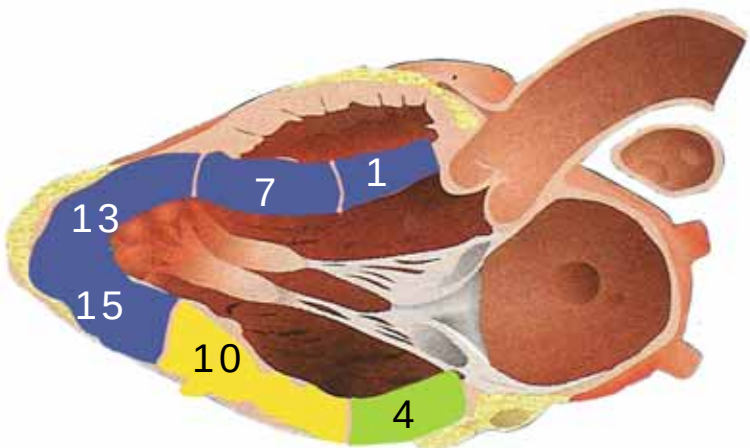
Both have similar QRS/ST-T pattern. The first one has not necrotic Q wave.
The ST injury vector directed to front and leftward in both circumstances like distal obstruction of LAD.

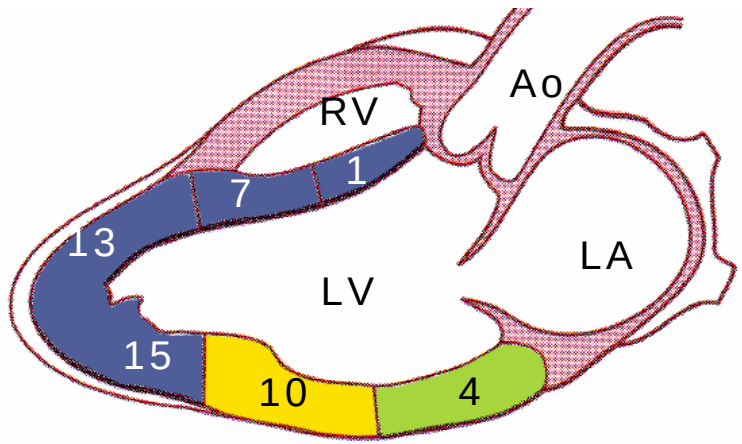
CURRENT MODEL OF VENTRICULAR SEGMENTATION AND WALL/ARTERY IRRIGATION

LONGITUDINAL PARAESTERNAL AXIS

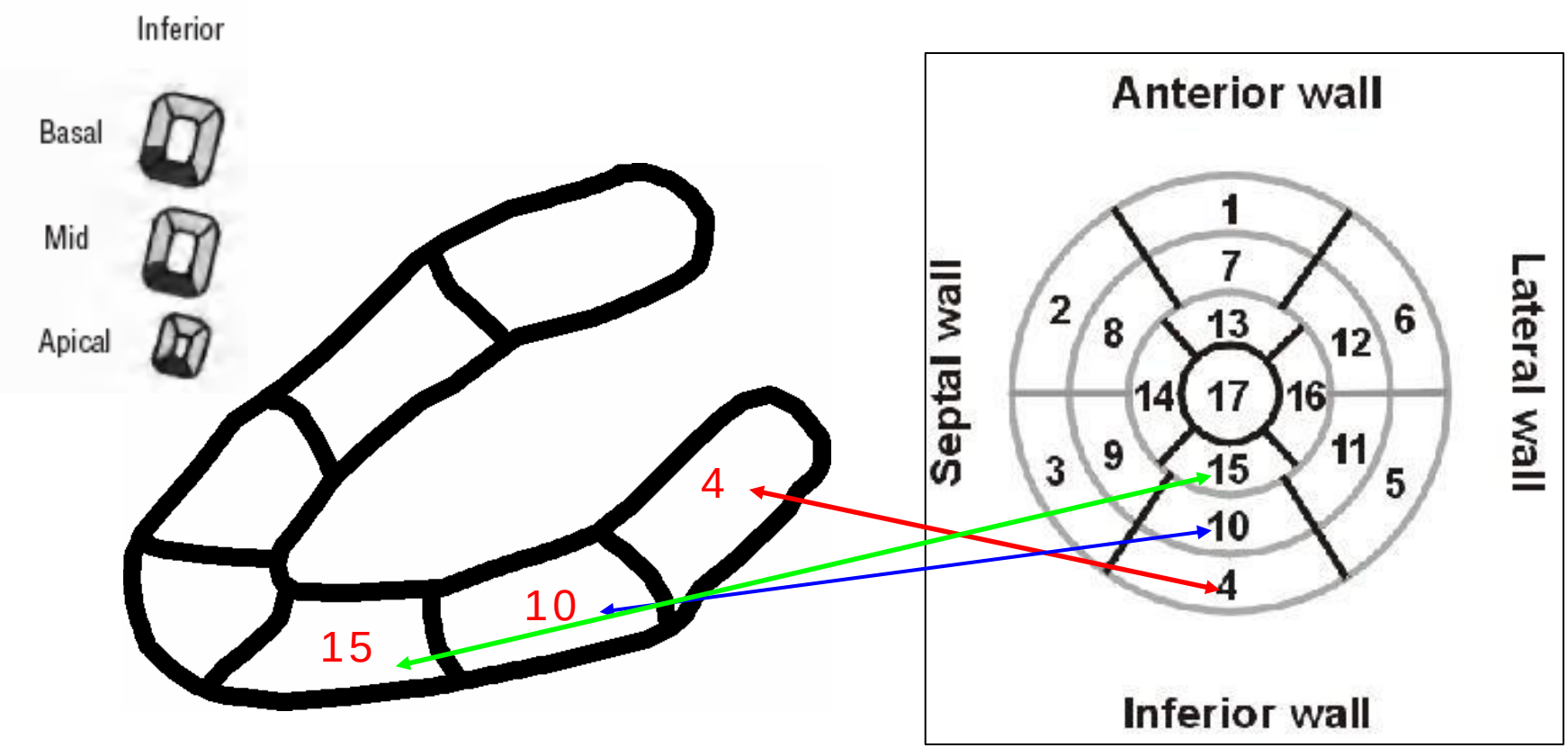








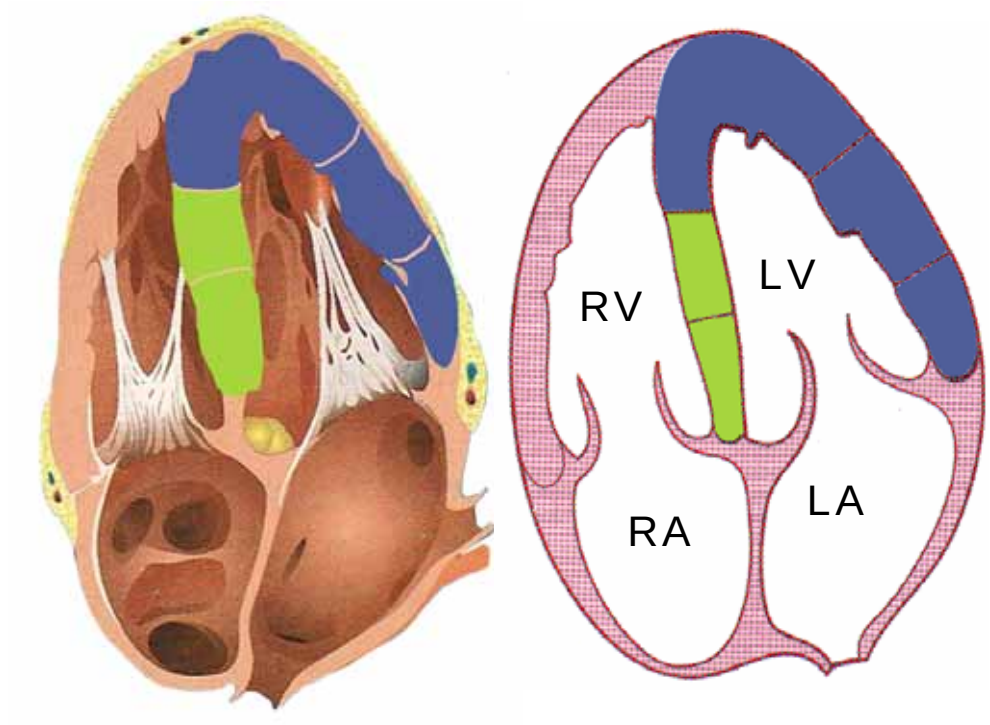
DEMONSTRATION OF THE INEXISTENCE OF THE DORSAL WALL



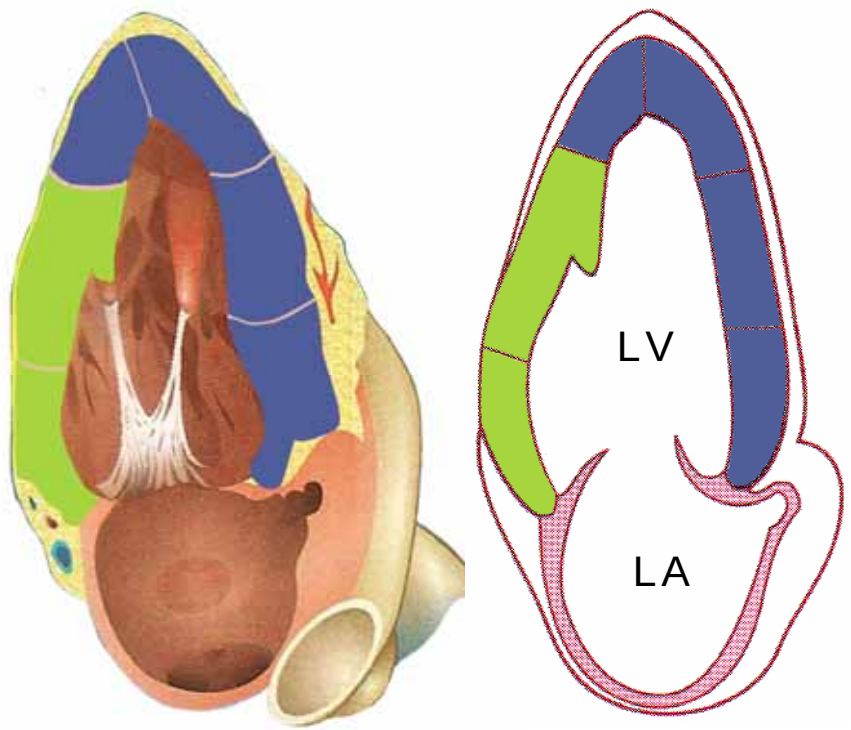
Segment number 4 correspond to the basal inferior wall, not dorsal.

CURRENT MODEL OF VENTRICULAR SEGMENTATION AND WALL/ARTERY IRRIGATION

APICAL FOUR CHAMBERS



APICAL TWO CHAMBERS



LEFT ANTERIOR DESCENDING (LAD)

LEFT CIRCUNFLEX (LCX)

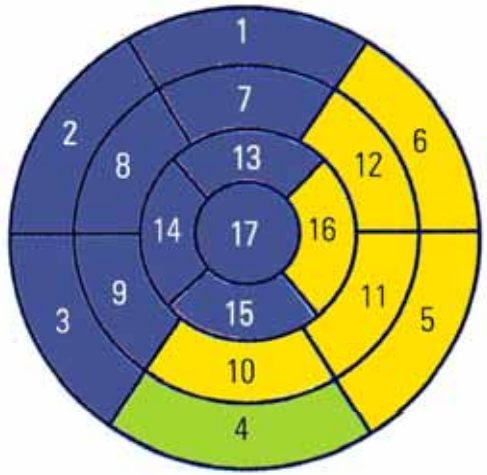
POSTERIOR DESCENDING (RCA OR LCX)

SHORT AXIS POLAR MAP IN "BULL'S-EYE"

ANTERIOR WALL

SEPTAL WALL

LATERAL WALL

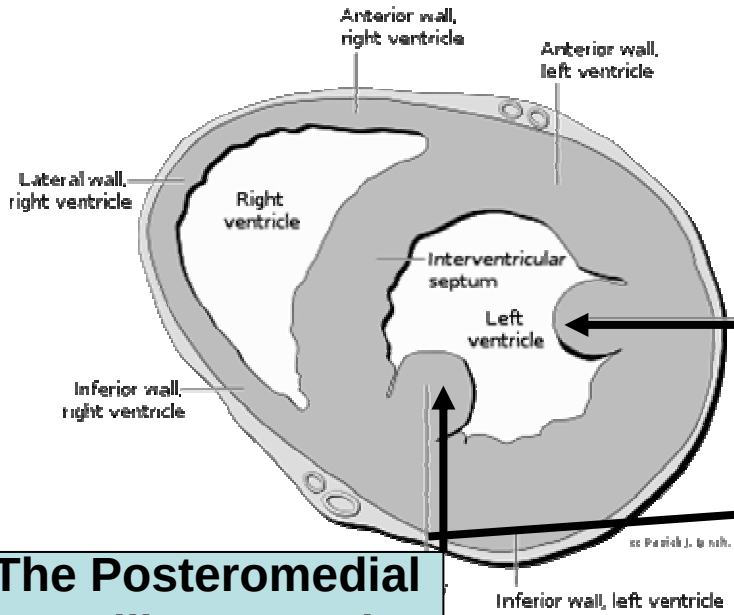


INFERIOR WALL

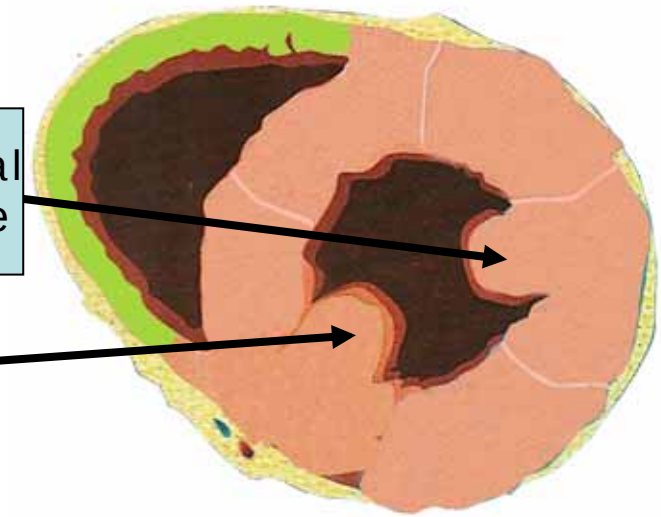
LEFT ANTERIOR DESCENDING (LAD)

LEFT CIRCUNFLEX (LCX)

POSTERIOR DESCENDING (RCA OR LCX)



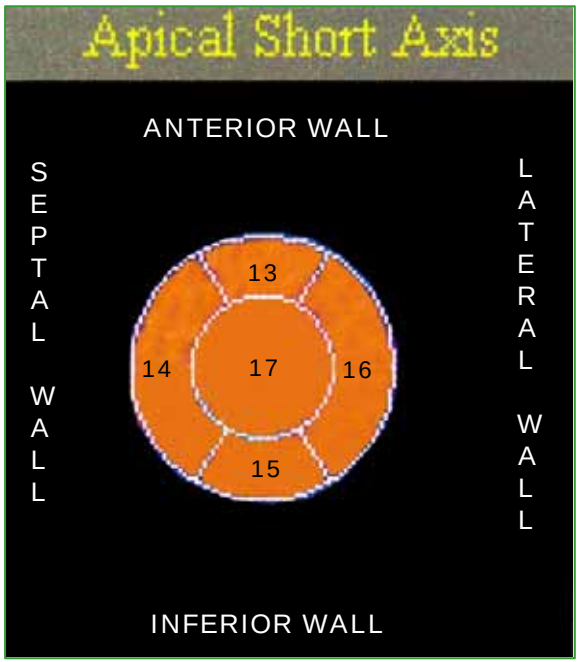
The Anterolateral Papillary Muscle



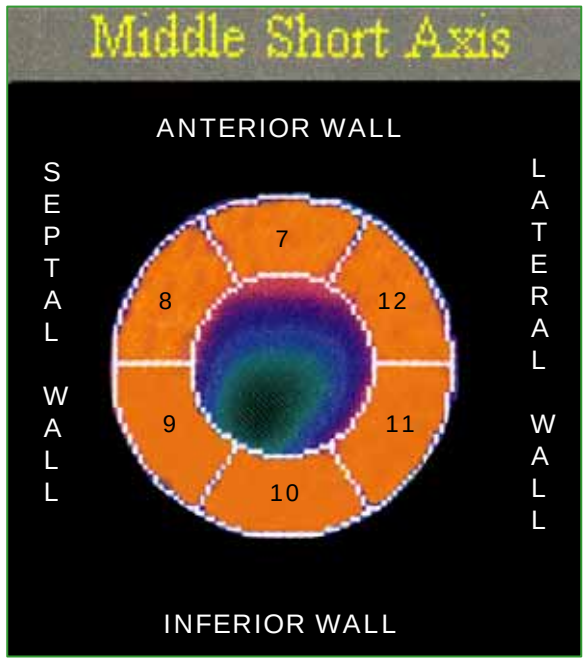
The Posteromedial Papillary Muscle

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

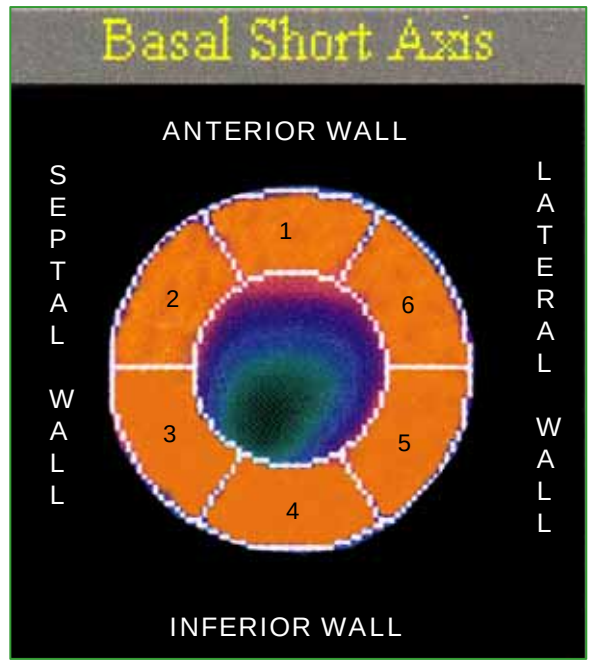
APICAL SHORT AXIS

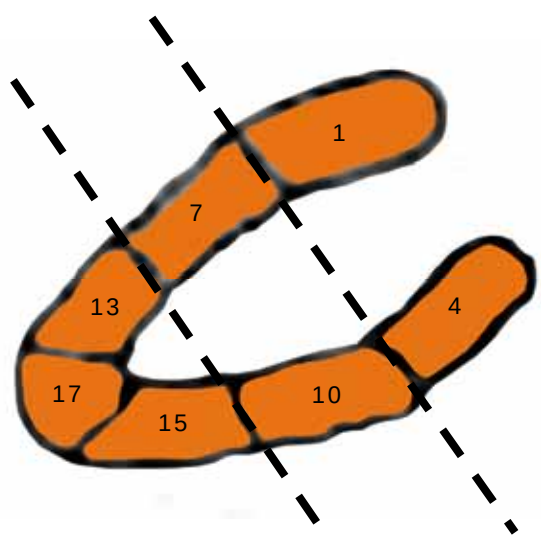


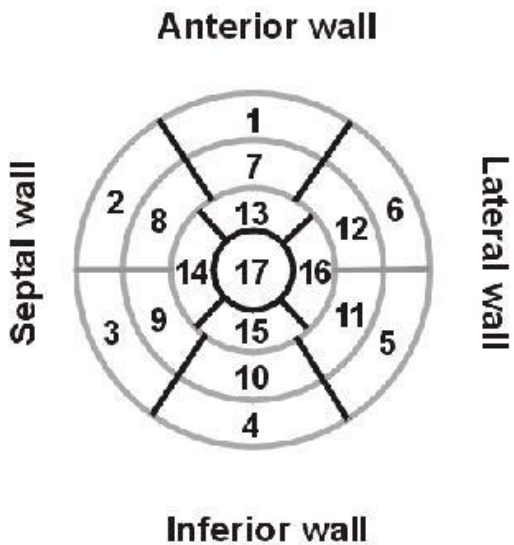
MIDDLE SHORT AXIS



BASAL SHORT AXIS

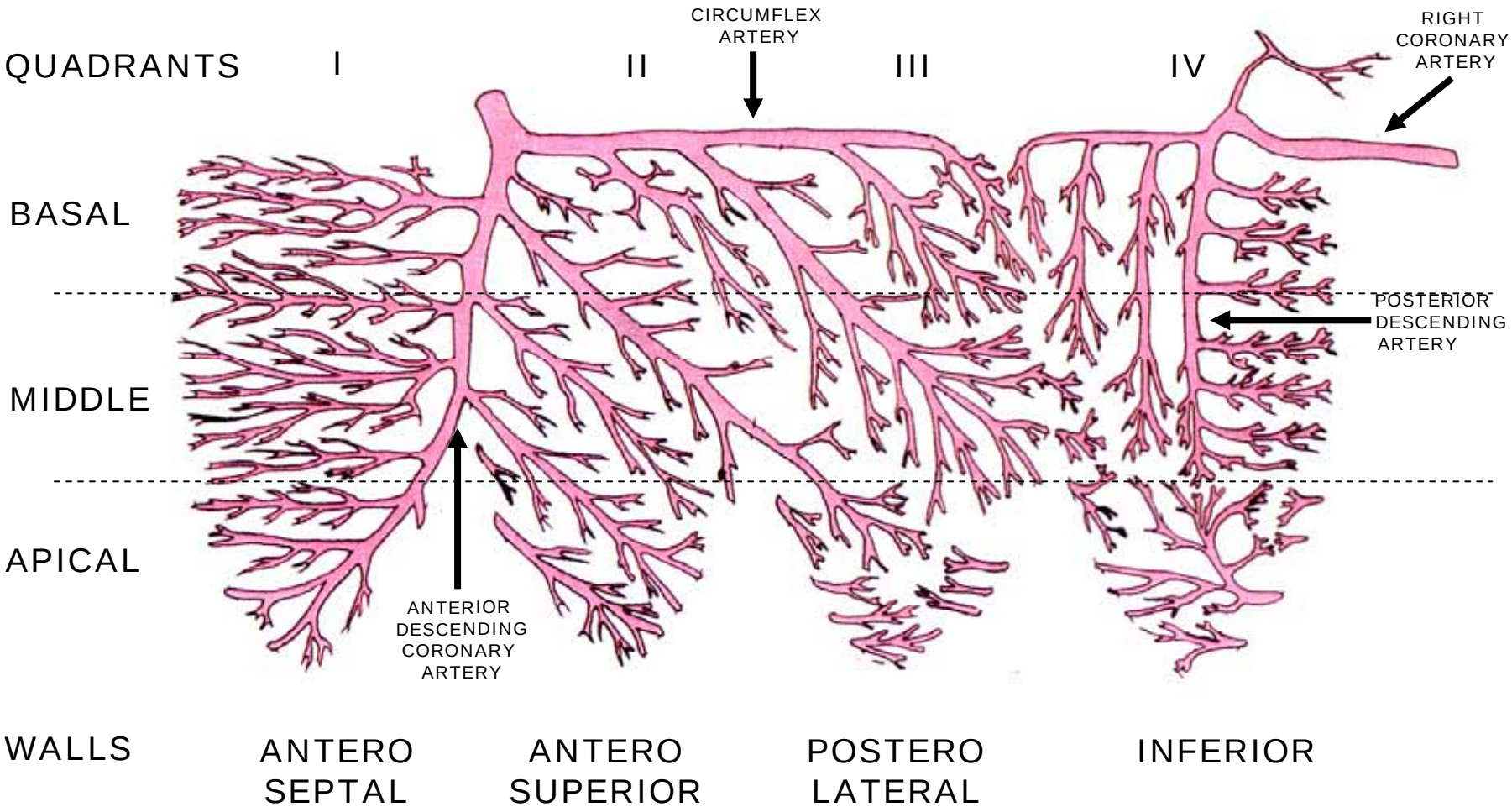






POLAR MAP
SHORT AXIS IN
“BULL’S-EYE”

DISTRIBUTION OF THE THREE MAIN CORONARY ARTERIES ON THE EPICARDIAL SURFACE OF THE LV



1. **Wagner GS, Selvester RH: Part I: Clinical electrocardiography for quantification of myocardial infarction. In: Braunwald E (ed): Atlas of heart disease. Vol 5 Chronic Ischemic Heart Disease. St Louis: Mosby-Year Book; 6.1-6.12,1995**

Outline of the main coronary arteries and their distribution.

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

1) ANTEROSEPTAL ZONE

Septal.....	A-1
Apico-anterior.....	A-2
Extensive anterior.....	A-3
Mid-anterior….....	A-4

2) INFEROLATERAL ZONE

Lateral.....	B-1
Inferior.....	B-2
Inferolateral.....	B-3

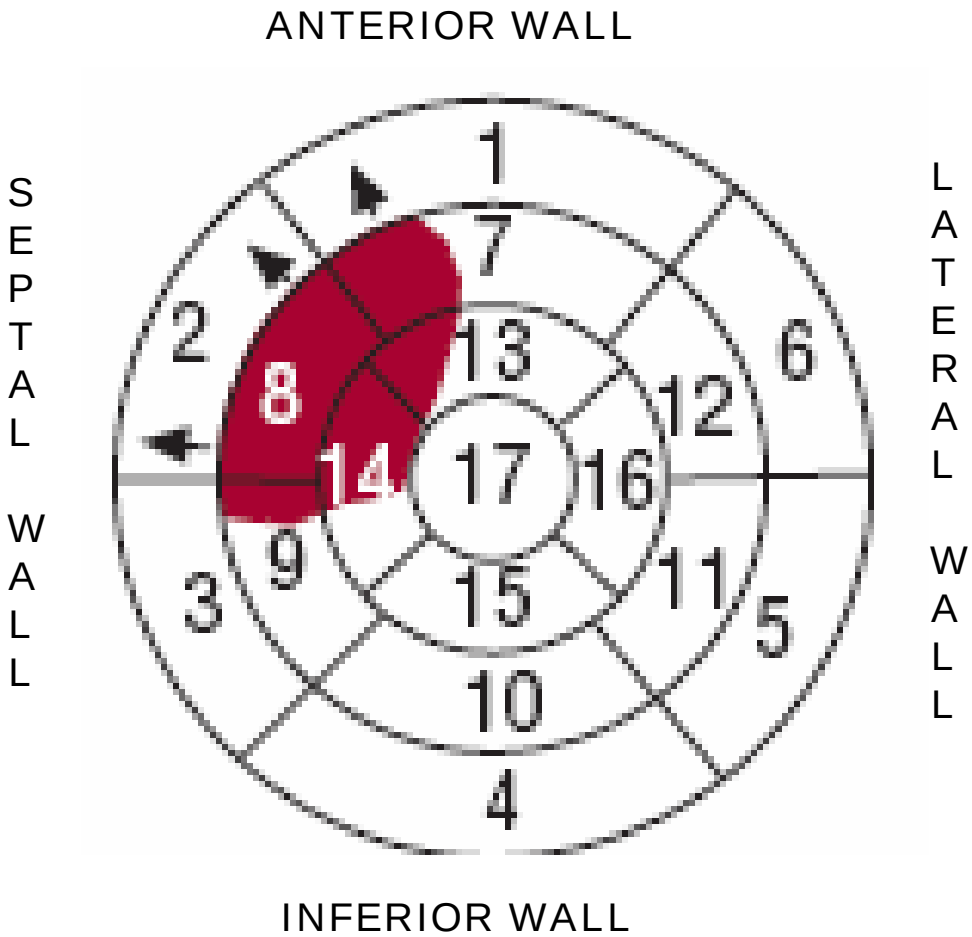
NEW ELECTROCARDIOGRAPHIC TERMINOLOGY FOR Q-WAVE INFARCTIONS BASED ON THE CORRELATION WITH CE-CMR

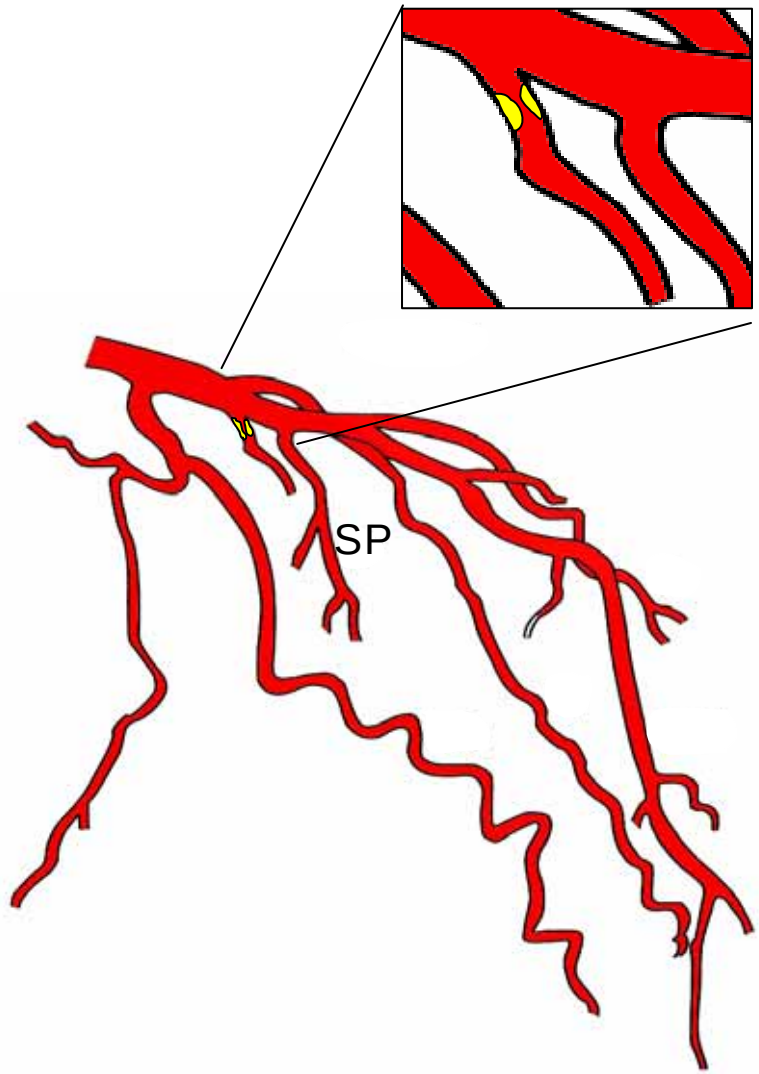
1) ANTEROSEPTAL ZONE

- **Septal myocardial infarction**
- **Type:** A-1.
- **Most likely site of occlusion**: perforating branch (S1) of LAD.
- **ECG pattern**: Q in V_1-V_2.
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **Sensitivity (SE):** 100%
- **Specificity (SP):** 97%.

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;8:325-326.

SEPTAL INFARCTION
A-1



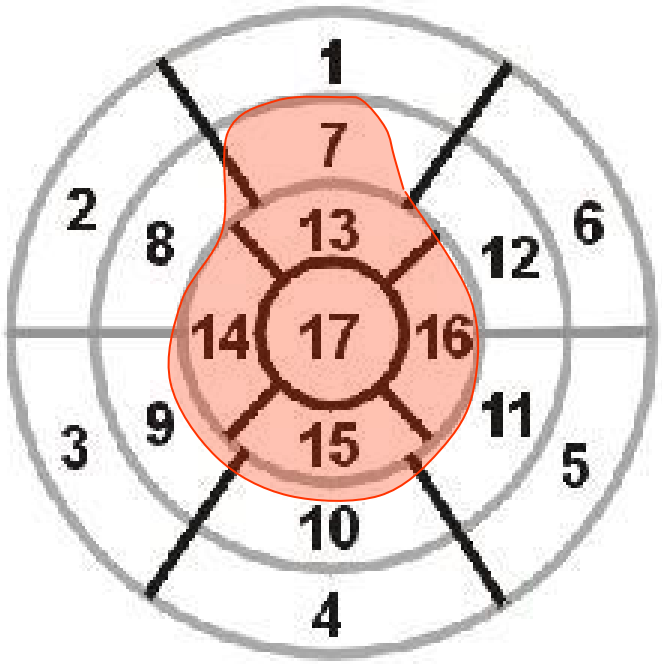


ECG pattern: Q in V1-V2

APICOANTERIOR INFARCTION
A-2

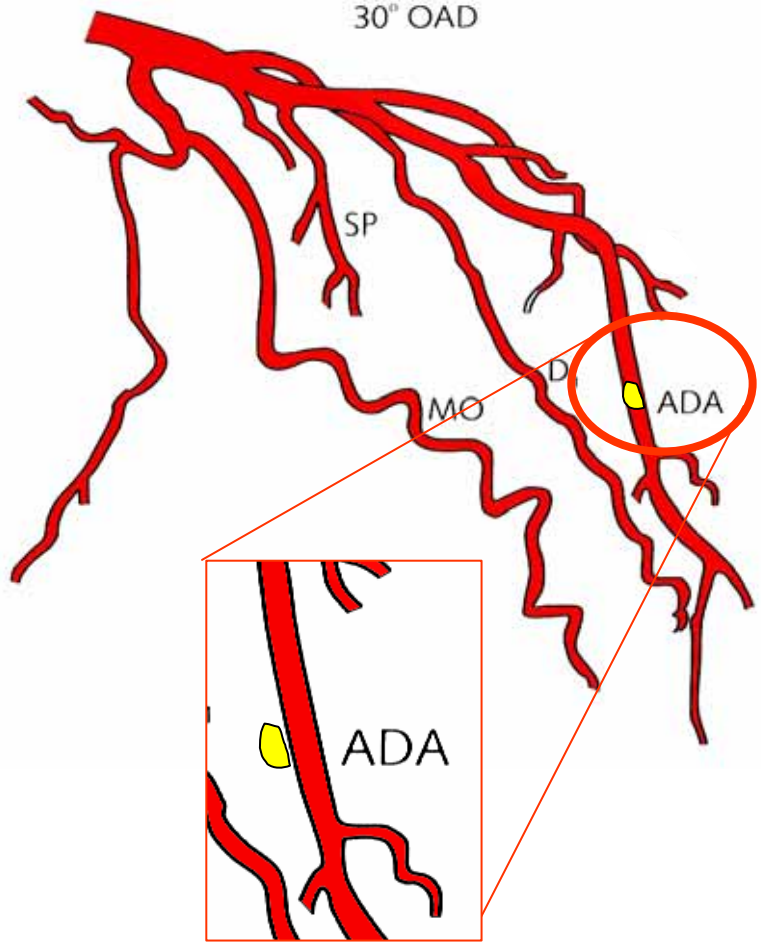
ANTERIOR WALL

SEPTAL WALL



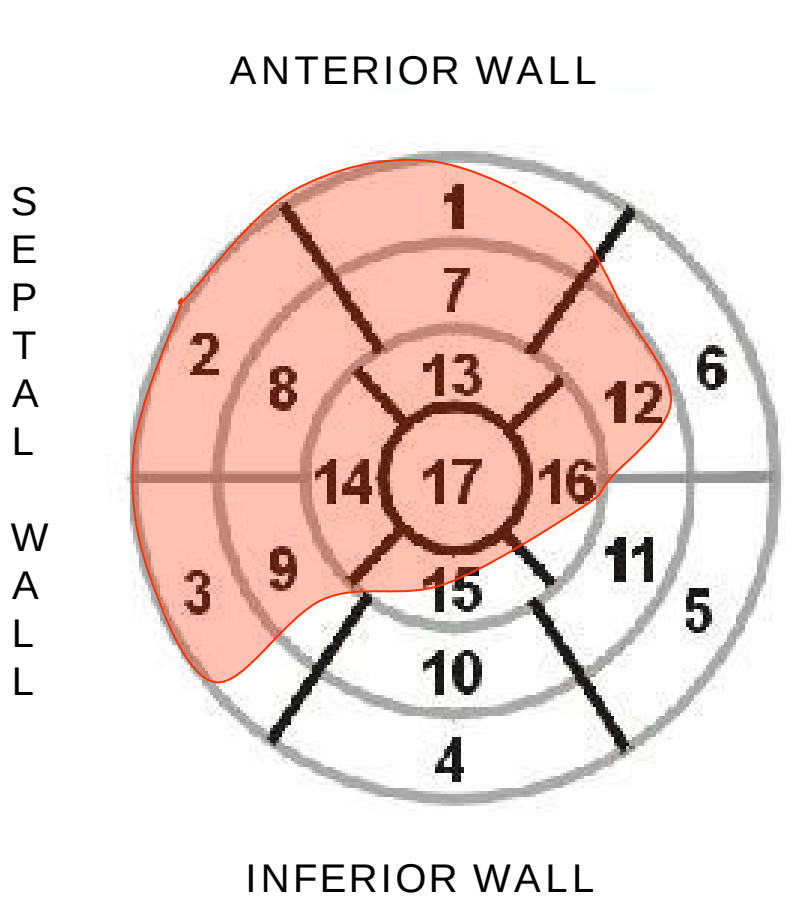
INFERIOR WALL

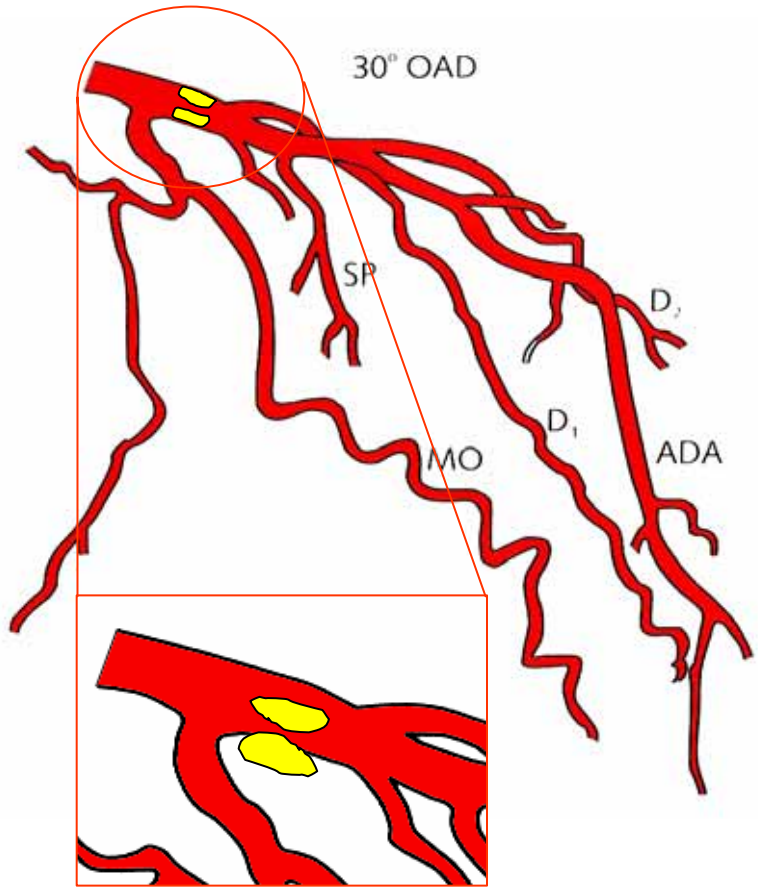
LATERAL WALL



ECG pattern: Q in V1-V2 through V3-V6

EXTENSIVE ANTERIOR INFARCTION A-3





ECG pattern:
Q from V1 through V6, VL, possibly I and VL

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

2) INFEROLATERAL ZONE

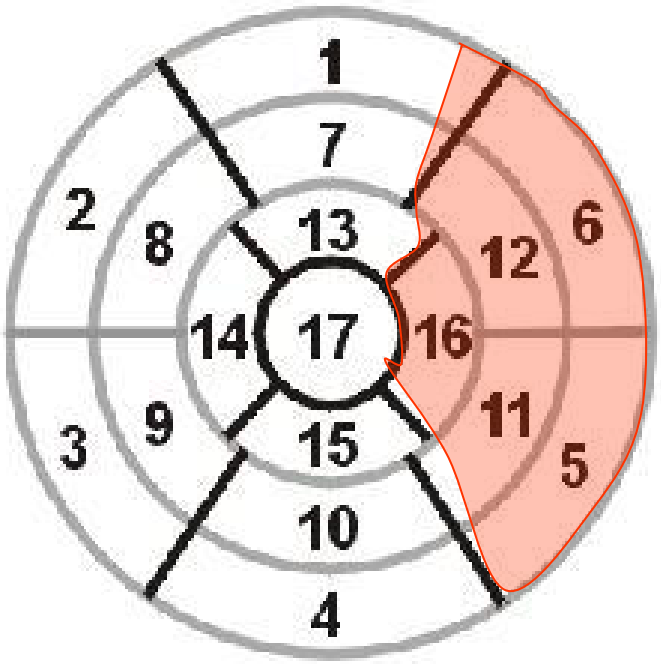
- **Lateral**
- **Type:** B-1
- **Most likely site of occlusion:** LCx artery or its oblique marginal branch (OM)
- **ECG pattern:** RS in V_1-V_2 and/or Q in I, VL, V_5-V_6. Voltage of R wave in V_6 of less amplitude
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **SE:** 67%
- **SP:** 99%.

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.

LATERAL INFARCTION
B-1

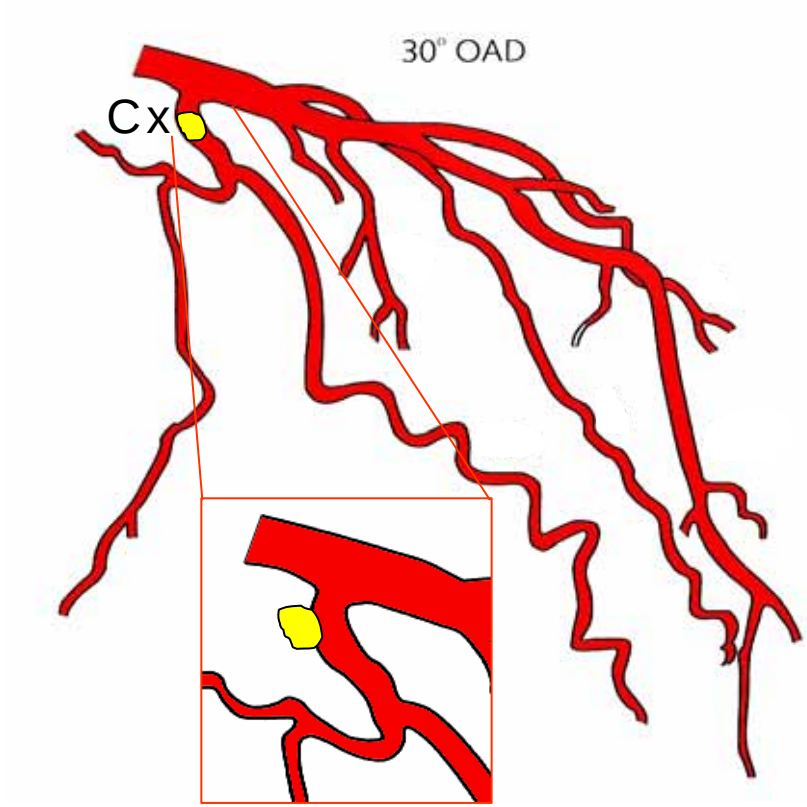
ANTERIOR WALL

SEPTAL WALL



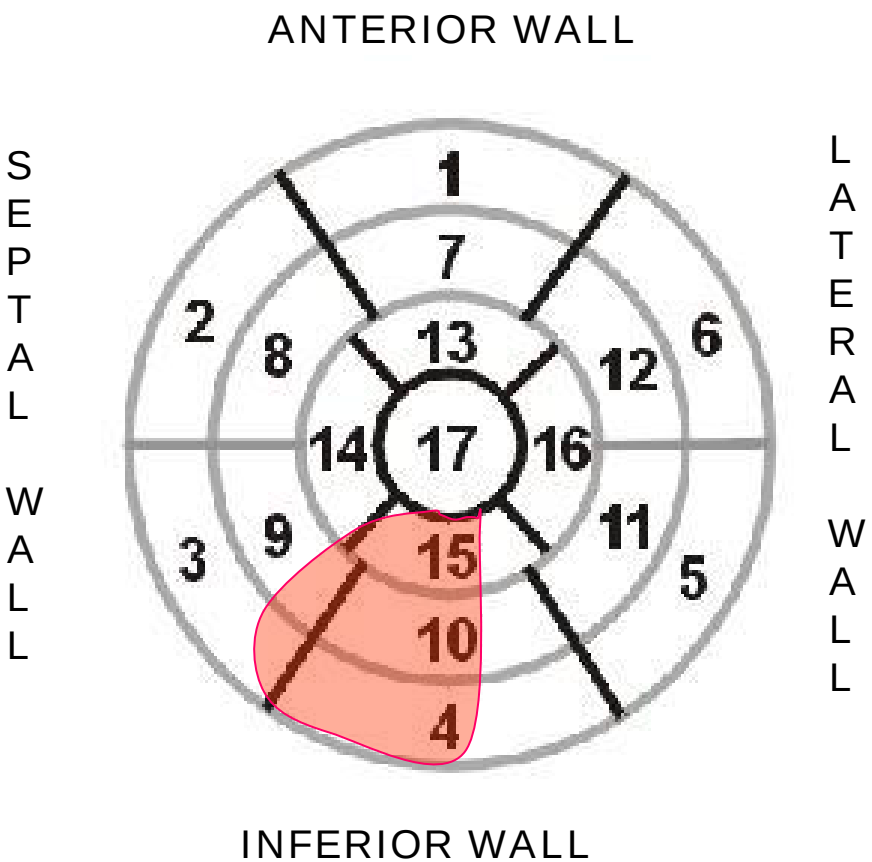
LATERAL WALL

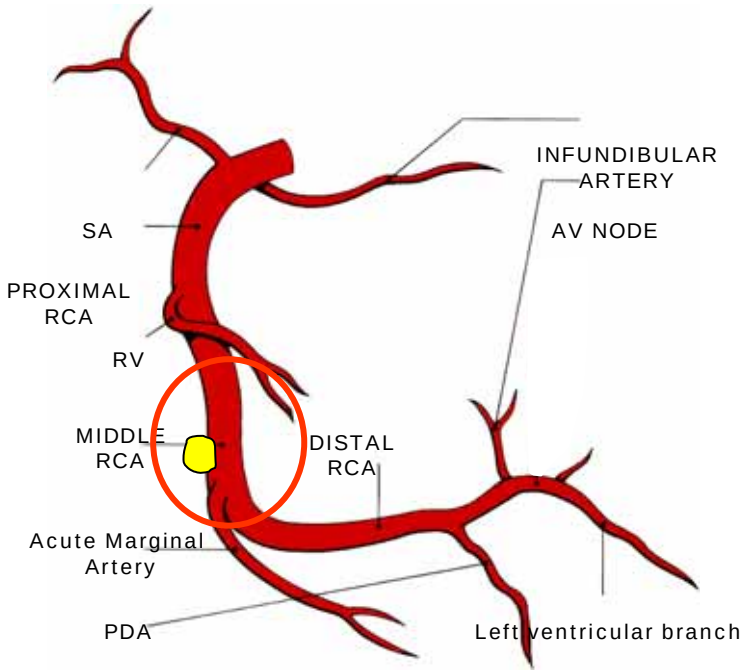
INFERIOR WALL



ECG pattern:
RS in V_1-V_2 and/or Q in I, aVL, V_5-V_6.
Voltage of R in V_6 of less amplitude

LATERAL INFARCTION
B-2





Obstruction of the middle 1/3 of RCA or distal from LCx

ECG pattern: Q in II, III, VF

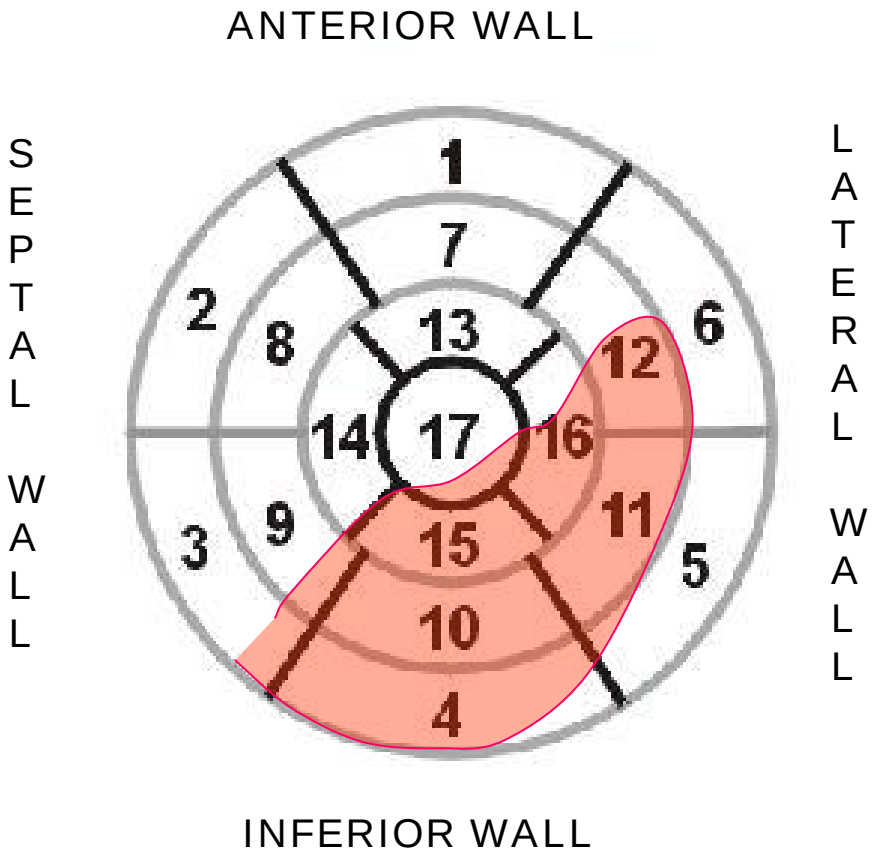
NEW ELECTROCARDIOGRAPHIC TERMINOLOGY FOR Q-WAVE INFARCTIONS BASED ON THE CORRELATION WITH CE-CMR

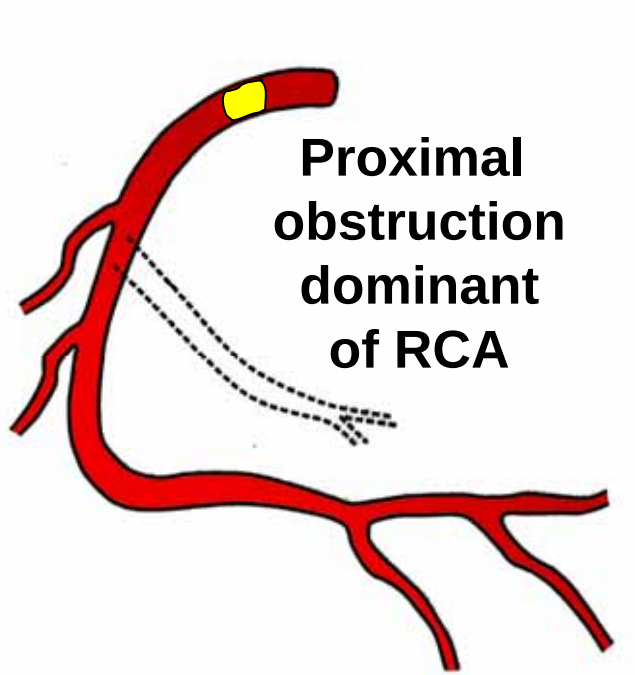
2) INFEROLATERAL ZONE

- **Inferolateral**
- **Type:** B-3
- **Most likely site of occlusion:** RCA or dominant LCx
- **ECG pattern:** signs of inferior (Q in II, II, VF: B2) and/or lateral infarction (RS in V_1).
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **SE:** 73%.
- **SP:** 98%.

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.

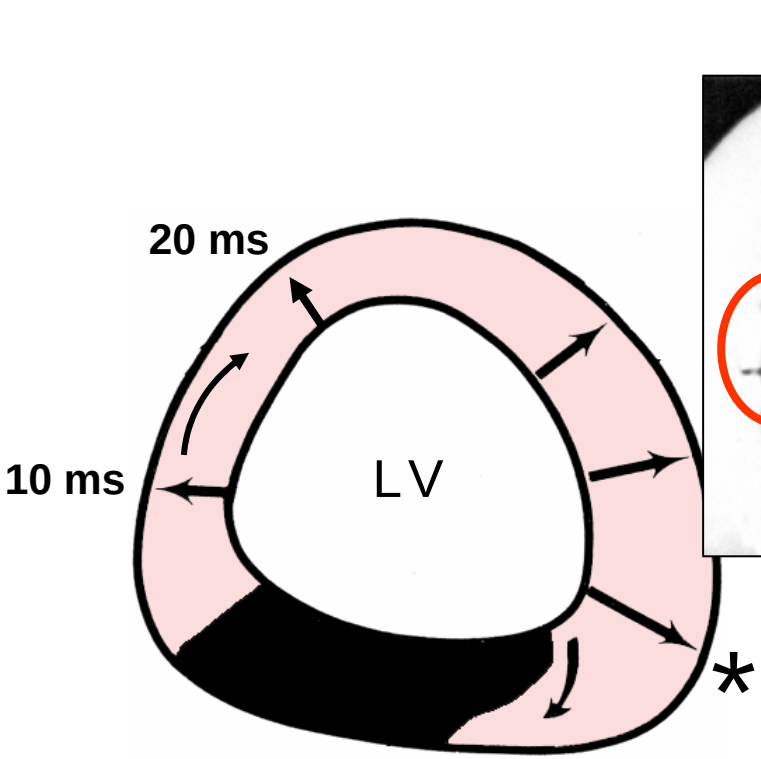
LATERAL INFARCTION
B-3

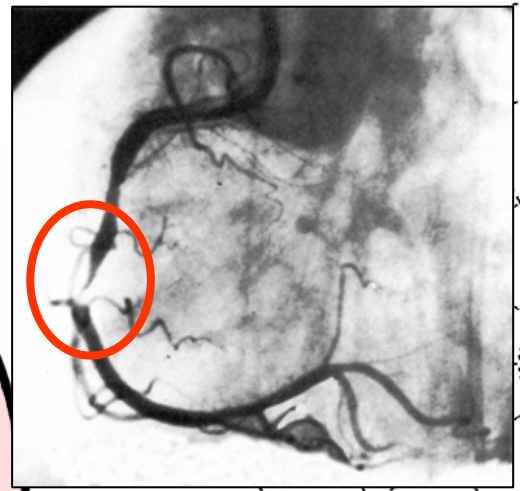




ECG pattern: signs of inferior (Q in II, II, aVF: B2) and/or lateral infarction (RS in V1).

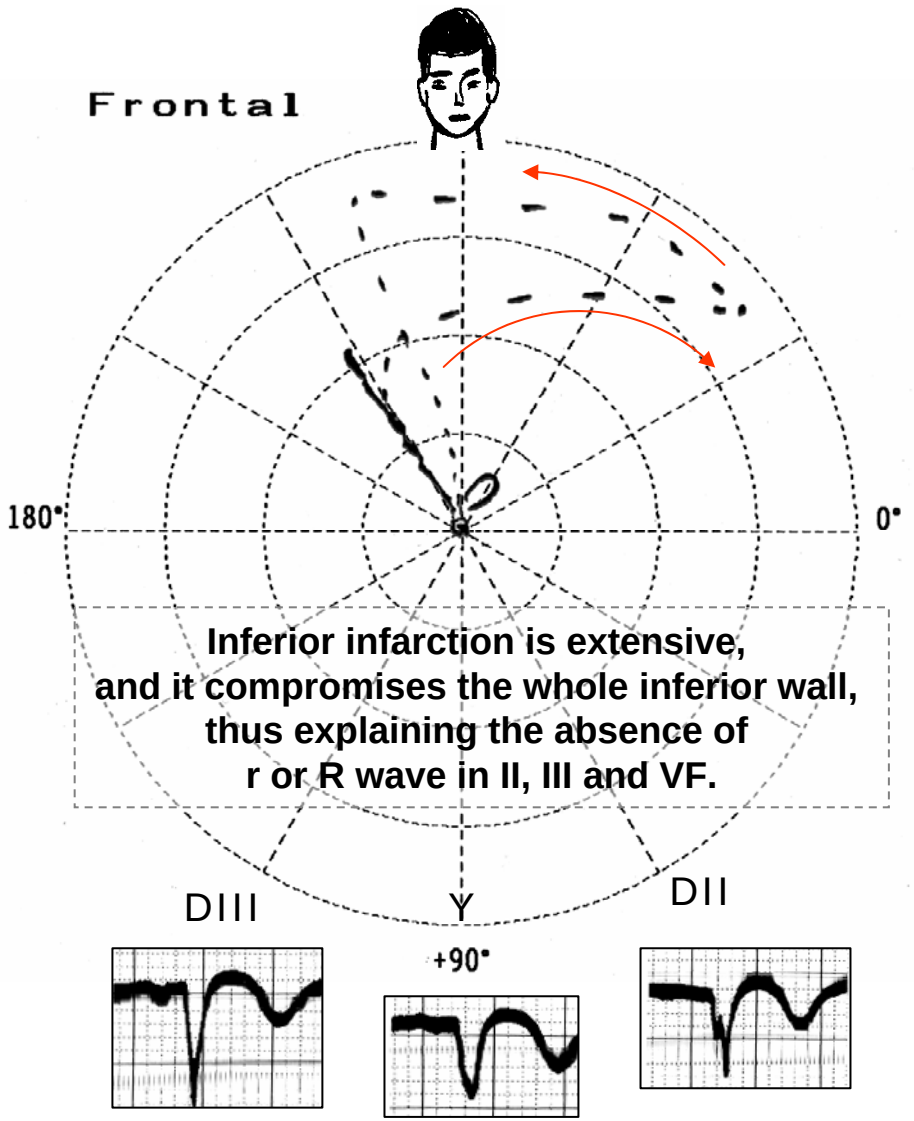
INFERIOR INFARCTION





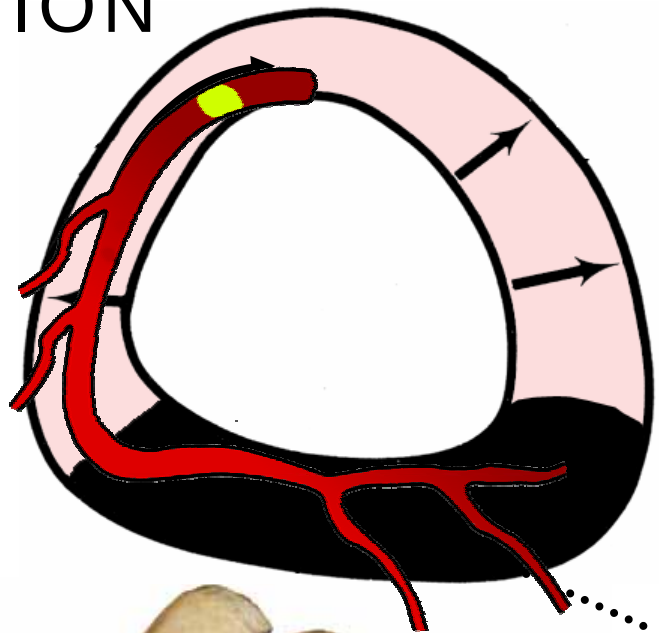
*This vector is responsible for the final R wave in II, since the left part of the inferior wall was not affected.

INFEROLATERAL INFARCTION

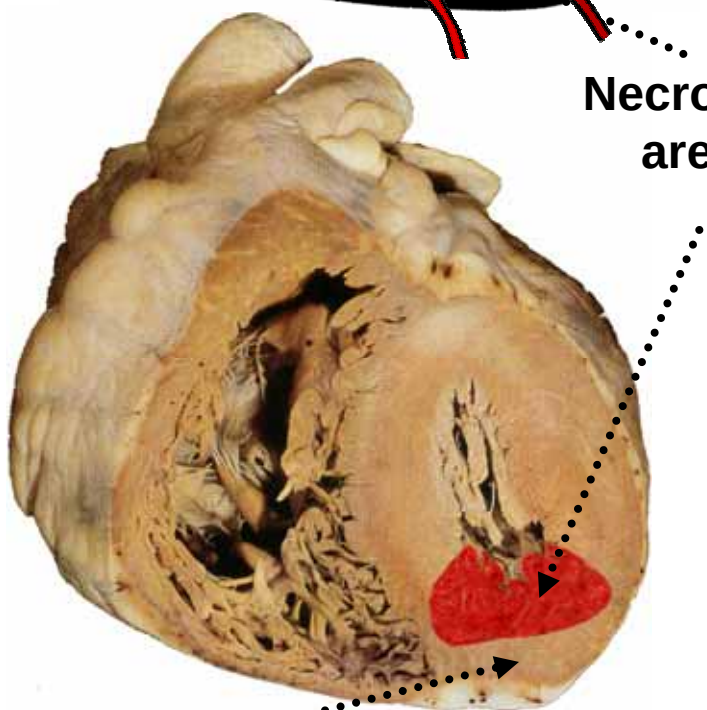


Inferior infarction is extensive, and it compromises the whole inferior wall, thus explaining the absence of r or R wave in II, III and VF.

QS in the three inferior leads



Necrosis area



Apical region