YOUNG MAN WITH TYPICAL CHEST PAIN
HOMEM JOVEM COM PRECORDIALGIA TÍPICA
HOMBRE JOVEN CON DOLOR PRECORDIAL TÍPICO

Raimundo Barbosa Barros MD
("The Fox" "A raposa" "El zorro")
Fortaleza-Ceará- Brazil&
Andrés Ricardo Pérez-Riera MD "El Potro"

PART I

Prezado Professor Andrés y queridos colegas/amigos:

Homem, 47 anos, com história de precordial tipica. Pometo a coronariografia em 24 horas

Raimundo Babarbosa Barros. " A raposa"

Qual é a artéria culpada?

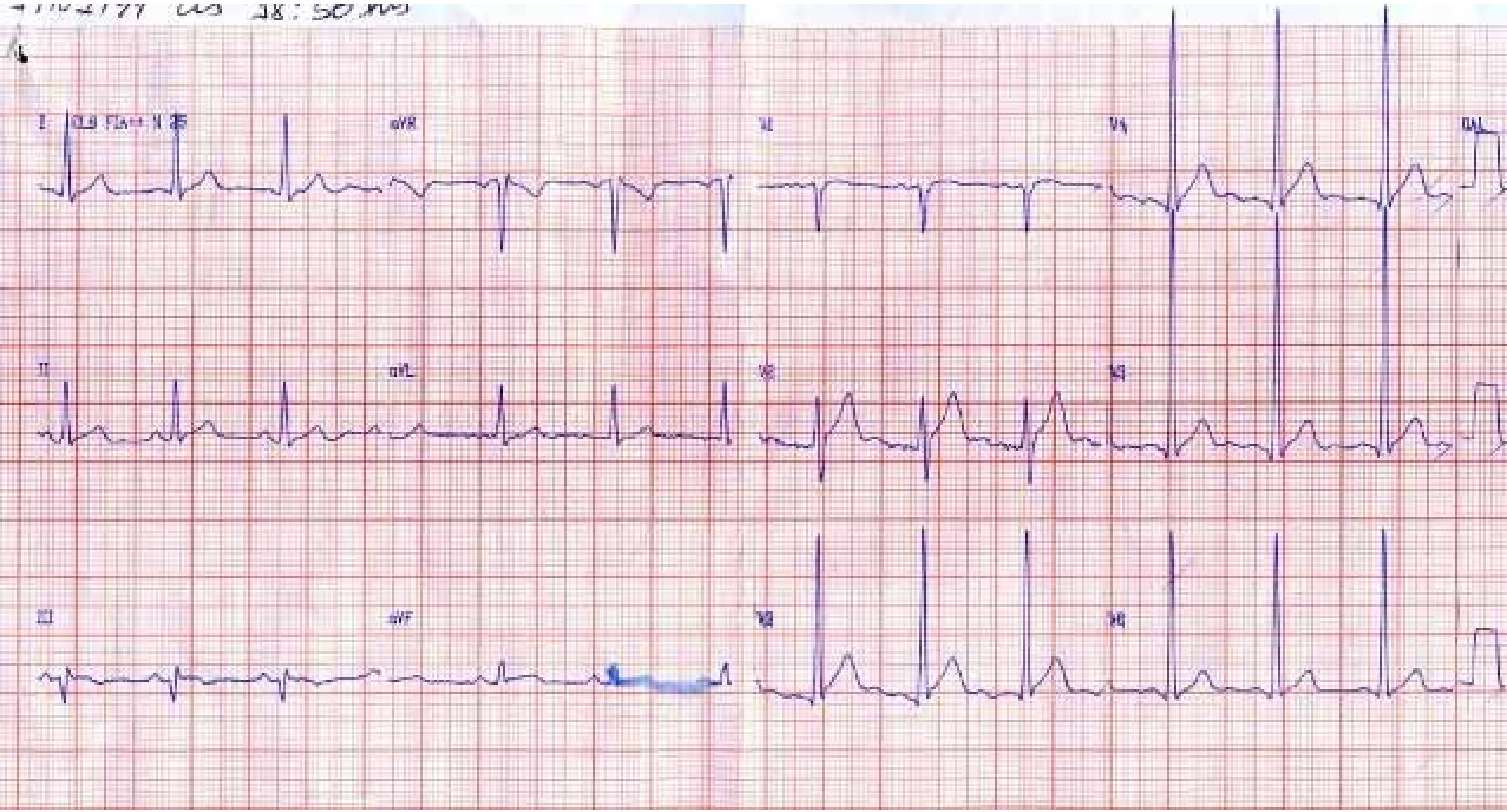
Dear Professor Andrés and dear colleagues/friends: Man, 47yo, with a history of typical chest pain. I promise the coronary angiography within 24 hours Whats is the culprit artery?

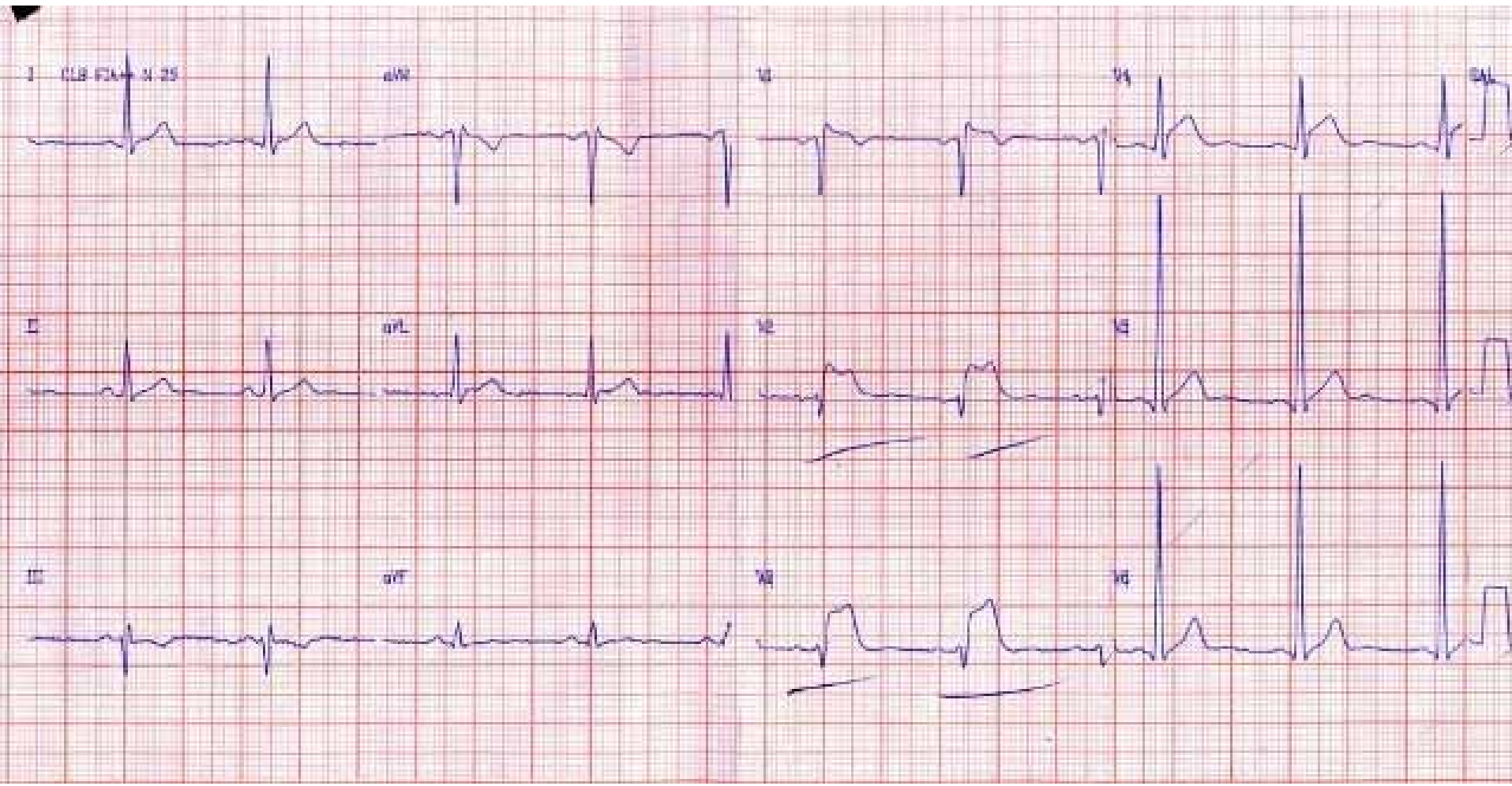
Raimundo Barbosa Barros MD "Te Fox"

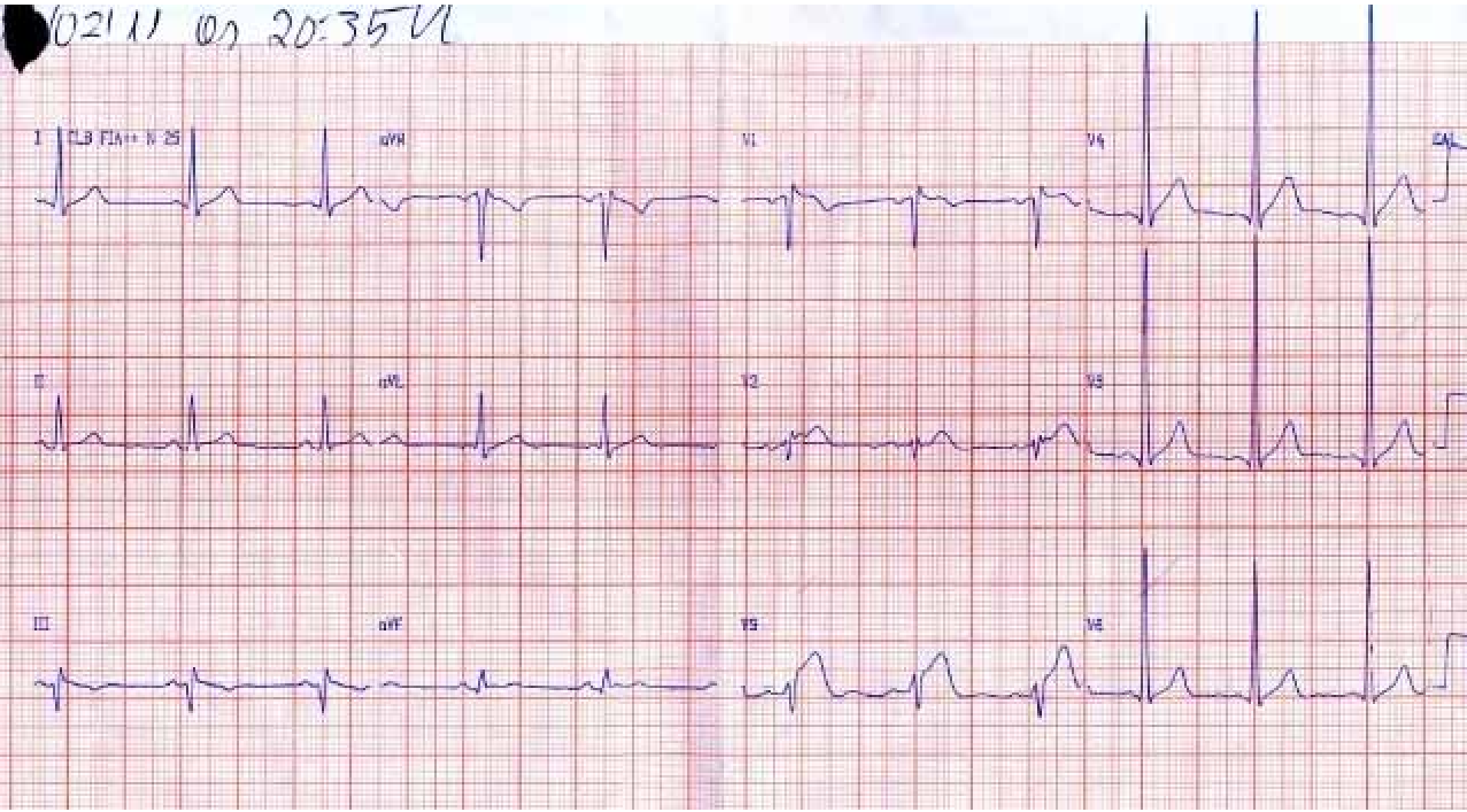
Estimado Profesor Andrés y queridos colegas/amigos: Hombre, de 47 años. Con historia de dolor precordial típico. Prometo la cinecoronariografia dentro de 24 horas.

Cual es la arteria culpada?

Raimundo Barbosa Barros " El zorro" →






02 11 01 20:35 VL
I
aVR
V1
V4
II
aVL
V2
V5
III
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V3
V6

COLLEAGUES OPINIONS
OPINIÃO DOS COLEGAS
OPINIÓN DE LOS COLEGAS

Me parece que este paciente tiene un infarto agudo inferior. Creo que en este caso esta comprometido el septo interventricular tanto izquierdo cuanto derecho por oclusión subita de la arteria coronaria derecha a nivel de la marginal primera,
Saludos Pancho Femenia

I think this patient has an acute inferior myocardial infarction. I think in this case is committed both left interventricular septum as a right by sudden occlusion of right coronary artery(RCA) at the level of the marginal first
Regards
Francisco FemeniaMD Mendoza – Argentina

Looks like LAD occlusion, of note is short ST segment ,is hypercalcemia present? Acute pancreatitis cam mimic MI. Is this a trick?

Parece oclusión de la arteria descendente anterior. El segmento ST es corto. Está presente hipercalcemia? Pancreatitis aguda puede imitar IM. ¿Es esto un truco?

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Dear Andres, The ECG during this young man's acute coronary syndrome suggests a mid or distal LAD lesion, perhaps distal to the 1st diagonal. I believe this is because there is no ST segment depression in the inferior leads.
Regards,
Frank

Querido Andrés, el ECG en este síndrome coronario agudo de este hombre joven sugiere una lesión media o distal de la LAD, tal vez distal a la primera diagonal porque no hay depresión del segmento ST en las derivaciones inferiores.
Un cordial saludo,

Frank

Caro Andrés, O ECG durante a síndrome aguda do jovem homem sugere uma lesão média ou distal da LAD, talvez distal a 1ra diagonal. Eu acredito isto porque não há depressão do segmento ST nas derivações inferiores.
Atenciosamente,
Frank

LAD proximal third
AB

$^{1}/_{3}$ proximal da Descendente Anterior
AB

Hello. Everything else than LAD would be surprising as the maximal ST elevation is in V_2-V_3.
Regards
Kjell Nikus
Tampere, Finland

The changes in the extremity leads are rather mild. In the first ECG, there seems to be slight elevation of the ST segment in the inferior leads, which would point to a distal occlusion in a "wrap-around-the-apex" LAD. However, in the second recording, where there is more severe ischemia, aVL is elevated, while LIII is slightly depressed ("reciprocal" ST depression). Anatomical information should be analyzed from the ECG with maximal ischemia. The findings indicate that there is a medium-sized or large diagonal branch distal to the occlusion. I am not sure about the QRS duration, but there seems to be at least partial RBBB, which also points to a proximal occlusion (proximal to the second septal branch, which usually is a large septal, subtending the right bundle branch). We have no previous ECG, so we don't know if the mild intraventricular conduction defect is old or new. But, summa summarum, I would expect a proximal LAD occlusion in a wrapping LAD. Anatomic prediction is influenced by inter-individual variation of coronary anatomy

Kind regards Kjell Nikus Tampere Finland

FINAL CONCLUSION AND THEORETICAL CONSIDERATIONS
CONCLUSÕES FINAIS E CONSIDERAÇÕES TEÓRICAS

Andrés Ricardo Pérez-Riera MD

Coronariografia: TCE,DA e Cx normais; oclusão de CD no terço distal. Todos nós sabemos que uma oclusão proximal de CD com envolvimento do VD pode simular um IAM anteroseptal. Infelizmente não houve sucesso na tentativa de angioplastia primária; FLUXO TIMI 0.

Coronariography: LMCA, LAD and LCX: normal. RCA with distal obstruction.
We all know that a proximal occlusion of the RCA with right ventricular involvement may simulate an acute anteroseptal MI. Unfortunately there was no success in the attempt of primary angioplasty, TIMI flow 0.

Nem sempre é posivel identificar pelo ECG a artéria relacionada ao infarto (ARI). Traçados de ECGs com 18-derivações foram comparados com os respectivos resultados das angiográfias de 1024 pacientes consecutivos. Foram realizados em todos os pacientes mais de dois ECGs de 18 derivações dentro das 12 horas de iniciados os sintomas.
Foram critérios de exclusão: Pacientes com IM prévio, com cirurgia de revascularização miocárdica, com implante de marcapassos ou com padrão BCRE e angiografia realizada com mais de 12 horas do início dos sintomas

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The infarct-related artery (IRA) could not always be identified by ECG. the reason for failed IRA identification by ECG based on the comparison between ECG records and coronary angiographic findings. All 18-lead ECG records were compared with respective angiographic findings in 1024 consecutive patients with STEM. More than two continous18-lead ECG records were performed within 12 hours of the symptom onset in all patients.
Exclusion criteria: Patients with previous MI, coronary artery bypass surgery, pacemaker implantation or ECG evidence of LBBB and angiography was performed more than 12 hours time from symptom onset.

Of all 1024 patients enrolled, the IRA were correctly identified in 854 cases and identified wrong in 96 cases and could not be identified in 74 cases by ECG. Of the failed identification in these 170 cases, IRA was LCX in 76 (44.7%)cases, RCA in 66 (38.8%) cases, LAD in 20 (11.8%) cases, ramus medianus branch in 7 (4.1%) cases, and LMCA in 1(0.6%) case. Double-vessel and triple-vessel diseases were recorded in 27(15.9%) patients and 47(27.6%) patients respectively. Early repolarization syndrome occurred in 8 (4.7%) patients, and dextrocardia in 1 patient (0.6%). Angiographic study showed acute occlusion of a small branch in 6 (3.5%) patients. The authors concluded that coronary collateral vessel can mislead judgments of the IRA by ECG. When the IRA can not be determined by ECG, LCX is most likely to be the culprit vessel. Occasionally, early repolarization syndrome and anatomic variation of the coronary artery or heart and a small branch occlusion could be causes of misjudgments of IRA by ECG.

De todos os 1024 pacientes incluídos, foram corretamente identificados pelo ECG 854 casos. Identificação equivocada ocorreu em 96 casos e não pôde ser identificada em 74. Dos 170 casos com falha na identificação 76 (44,7%) foram da circunflexa esquerda, casos 66 (38,8%), da coronária direita e em 20 pacientes (11,8%) a DA. O ramo mediano em 7 (4,1%) casos, e O Tronco da CE em 1 (0,6%) caso. Doença de dois vasos foi observado em 27 (15,9%) e de três vasos em 47 (27,6%), respectivamente. Síndrome de repolarização precoce ocorreu em 8 (4,7%) e dextrocardia em um paciente (0,6%). O estudo angiográfico mostrou oclusão aguda de um pequeno ramo em 6 (3,5%). Os autores concluíram que a presença de circulação colateral coronária é o fator principal de falsa identificação da artéria culpada pelo ECG. Quando a artéria relacionada a obstrução não pode ser determinado pelo ECG a circunflexa é o vaso mais provávelmente culpado. Ocasionalmente, a síndrome de repolarização precoce e variação anatômica da artéria coronariana ou cardíaca e oclusão de ramos pequenos poderiam ser causas de incorreções de identificação da artéria culpada pelo ECG.

1. **Zhang XJ, Yan HB, Zheng B, Song L, Wang J, Chi YP. Reasons for failed electrocardiographic identification of the infarct-related artery in patients with ST-elevation acute myocardial infarction. Zhonghua Xin Xue Guan Bing Za Zhi. 2010 Oct;38:914-917.**

Anterior STE is the classic ECG feature of anterior LV myocardial infarction due to occlusion of the LAD. However, anterior ST-segment elevation has rarely also been described in patients with RCA occlusion at several levels (Mainly proximal) without inferior ST-segment elevation. It is hypothesized that the inferior LV wall is protected by left-to-right collaterals, as seen on coronary angiography, with resultant isolated RV infarction RCA occlusions. Isolated RV infarction resulting in an ECG pattern mimicking anterior-wall LV infarction.

STE na parede anterior é a característica clássico do ECG de infarto do miocárdio do VE anterior, devido à oclusão da artéria descendente anterior. No entanto, a elevação do segmento ST anterior raramente tem sido descrita também em pacientes com oclusão RCA em diversos níveis (principalmente proximal) sem elevação concomitante do segmento ST em parede inferior. A hipótese que tenta explicar esta ausência de isquemia na parede inferior do VE é a proteção oferecida por colaterais da esquerda para a direita, como visto na angiografia coronária, com conseqüente infarto isolado do VD oclusão da coronária direita.

O infarto isolado do VD,pode resultar em um padrão de ECG que imita o infarto de parede anterior do VE.

Right ventricular infarction (RVI) during inferior MI is readily diagnosed when STE is recorded in lead V4R. RVI may also yield precordial STE and such an ECG pattern may be misinterpreted as a sign of anterior MI. Inferior-RVMI due to occlusion of a dominant RCA manifesting STE in precordial and right chest leads[1]. RV dilation due to acute ischemic insult facilitated STE in leads V1-V4 despite the dominant opponent inferior and posterolateral LV injury current. Dilation of an infarcted RV should be considered when such an ECG pattern is encountered during inferior MI, specifically a dominant one. Awareness of the circumstances under which this ECG pattern develops facilitates avoidance of misinterpretation as a sign of anterior MI and proper management.

1. **Andreou AY, Georgiou GM. Dominant right coronary artery occlusion entailing diffuse ST-segment elevation in the precordial leads. J Cardiovasc Med (Hagerstown). 2010 Nov;11:843-847.**

ECG VALUE FOR LOCALIZATION OF "CULPRIT" ARTERY IN ACUTE CORONARY SYNDROMES (ACS) WITH ST SEGMENT ELEVATION (STEMI)

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THE ECG IN ACUTE CORONARY SYNDROME(ACS)

Patients with ACS include those whose clinical presentations cover the following range of diagnoses:

I) ***Unstable angina:*** ***New-onset exertion angina, angina increasing frequency or duration or refractory to nitroglycerin, or angina at rest.***

II) ***Non–ST-Elevation Myocardial Infarction (NSTEMI)***

III) ***ST-elevation Myocardial Infarction (STEMI).***

ST-segment Elevation Myocardial Infarction (STEMI)

New or presumably recent J point and ST segment elevation in 2 or more adjacent leads $\geq$ 2 mm in V_1, V_2 or V_3 or $\geq$ 1 mm in other leads

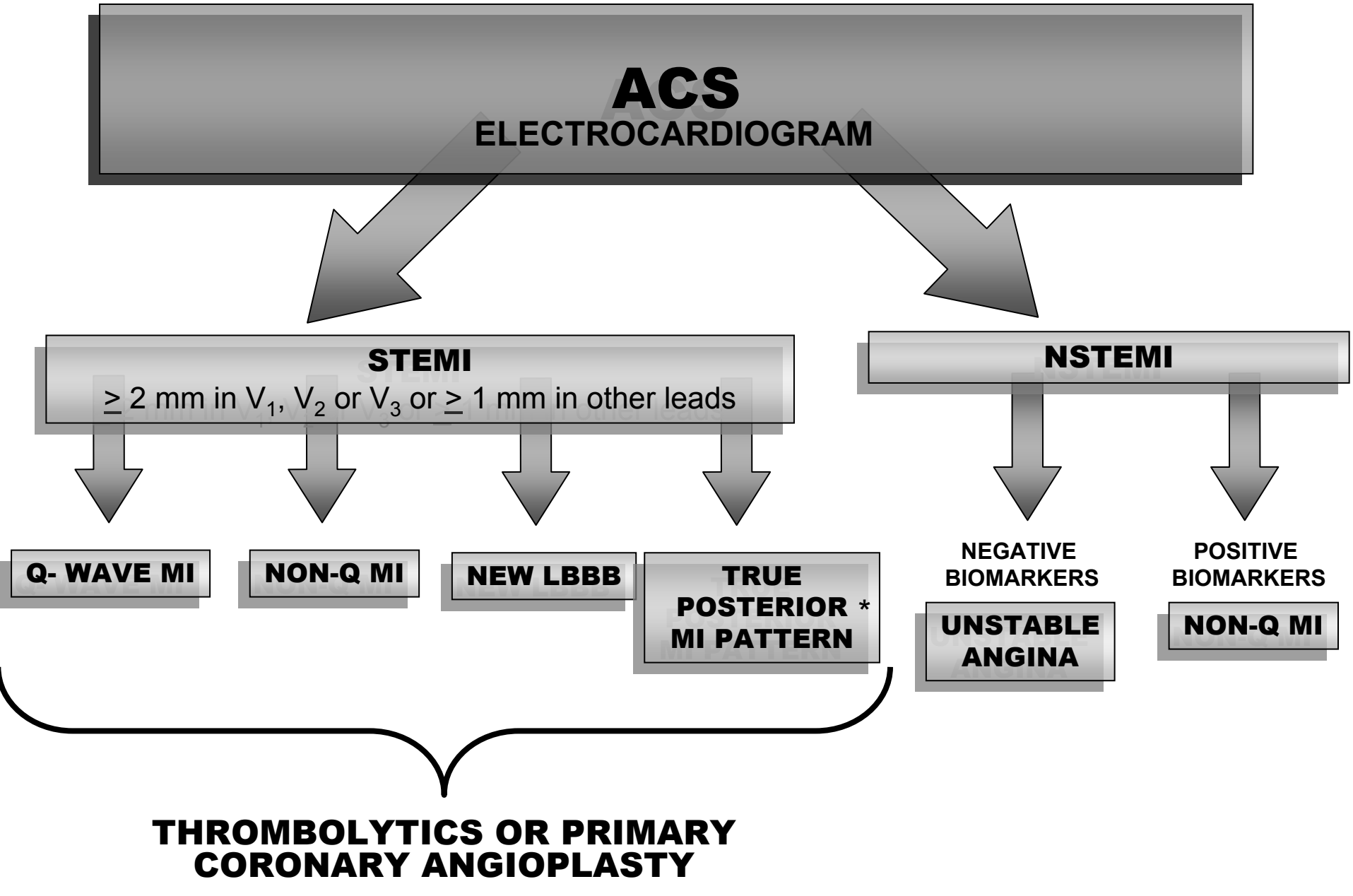
Non-ST segment Elevation Myocardial Infarction (NSTEMI)

ST segment depression

Isolated alterations of the T wave

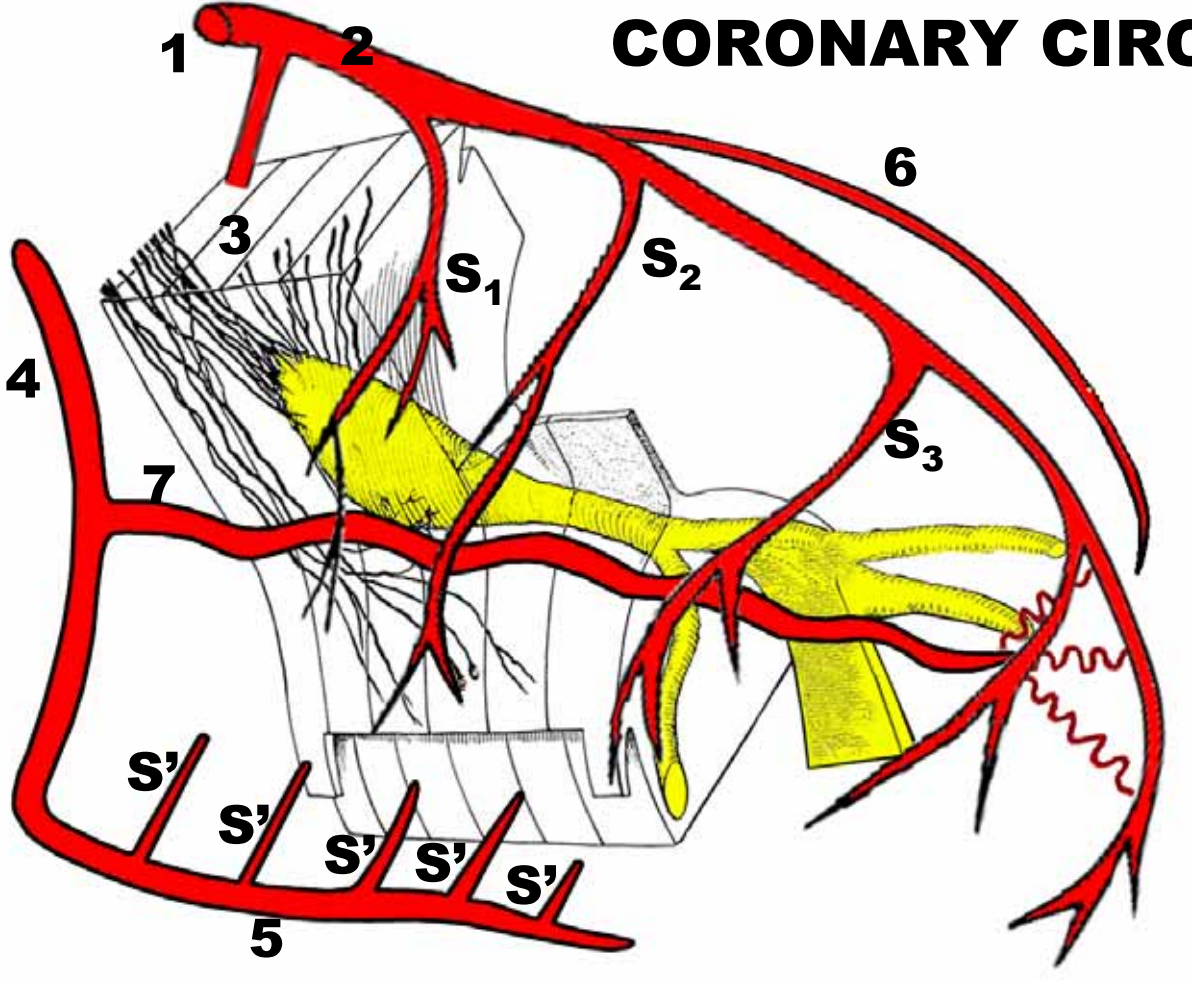
This ACS spectrum concept is a useful framework for developing therapeutic strategies

1. **Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined--a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol. 2000 Sep;36:959-969. Erratum in: J Am Coll Cardiol 2001 Mar 1;37:973.**



* Actual basal inferior.

CORONARY CIRCULATION



Anterior Septal Perforator Branches

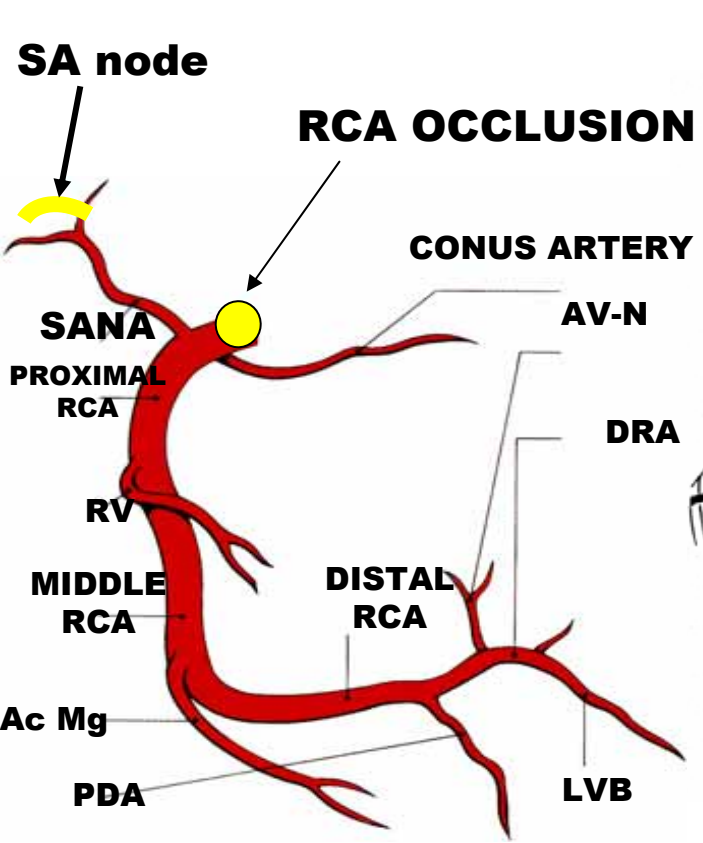
S_1: First Septal Perforator branch
S_2: Second Septal Perforator
S_3: Third Septal Perforator

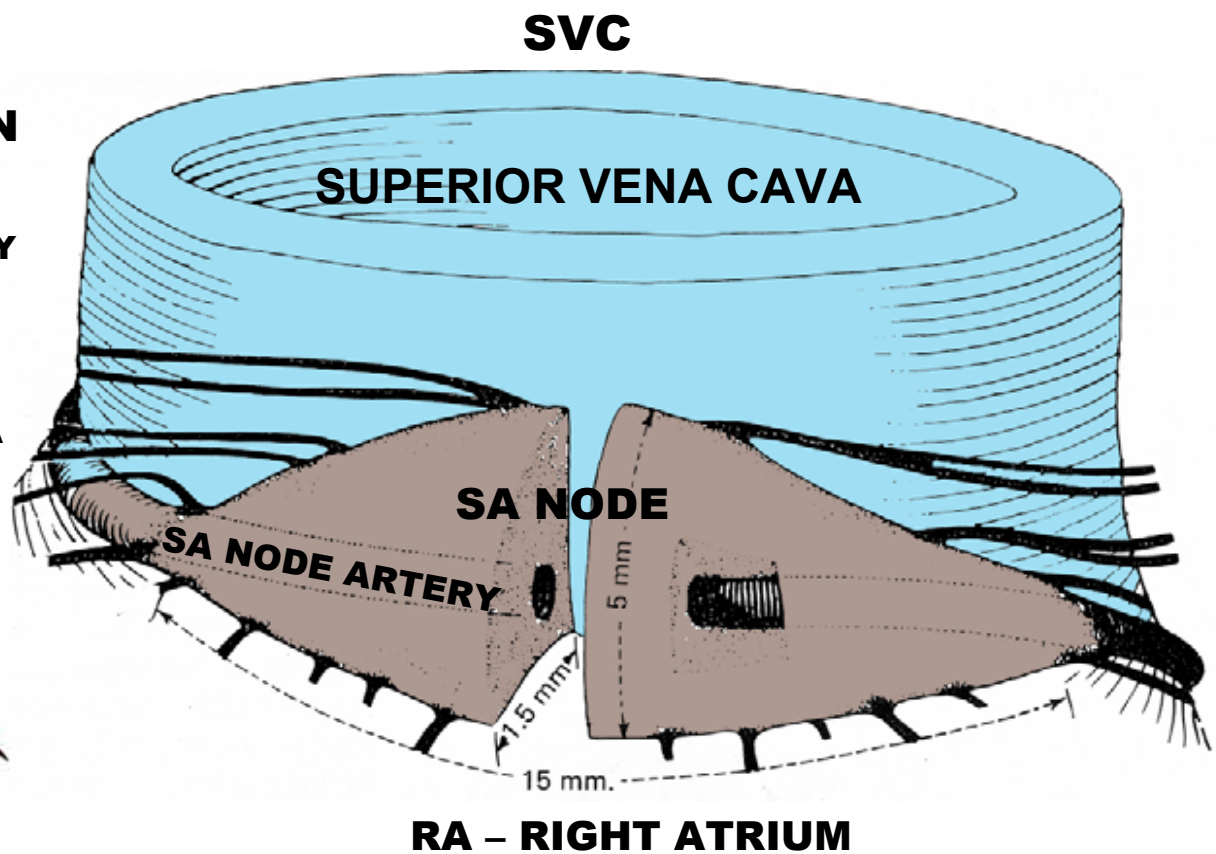
S': Posterior Septal Perforators

1. **L**eft **M**ain **C**oronary **A**rtery **(LMCA)**
2. **L**eft **A**nterior **D**escending Artery **(LAD)**
3. **L**eft **C**ircumflex Coronary Artery **(LCX)**
4. **R**ight **C**oronary **A**rtery **(RCA)**
5. **P**osterior **D**escending **A**rtery **(PDA)**. In this case is supplied by the RCA, then the coronary circulation can be classified as "right-dominant“
6. First Diagonal **(Dg)**
7. Acute Marginal **(A. Mg)**

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].



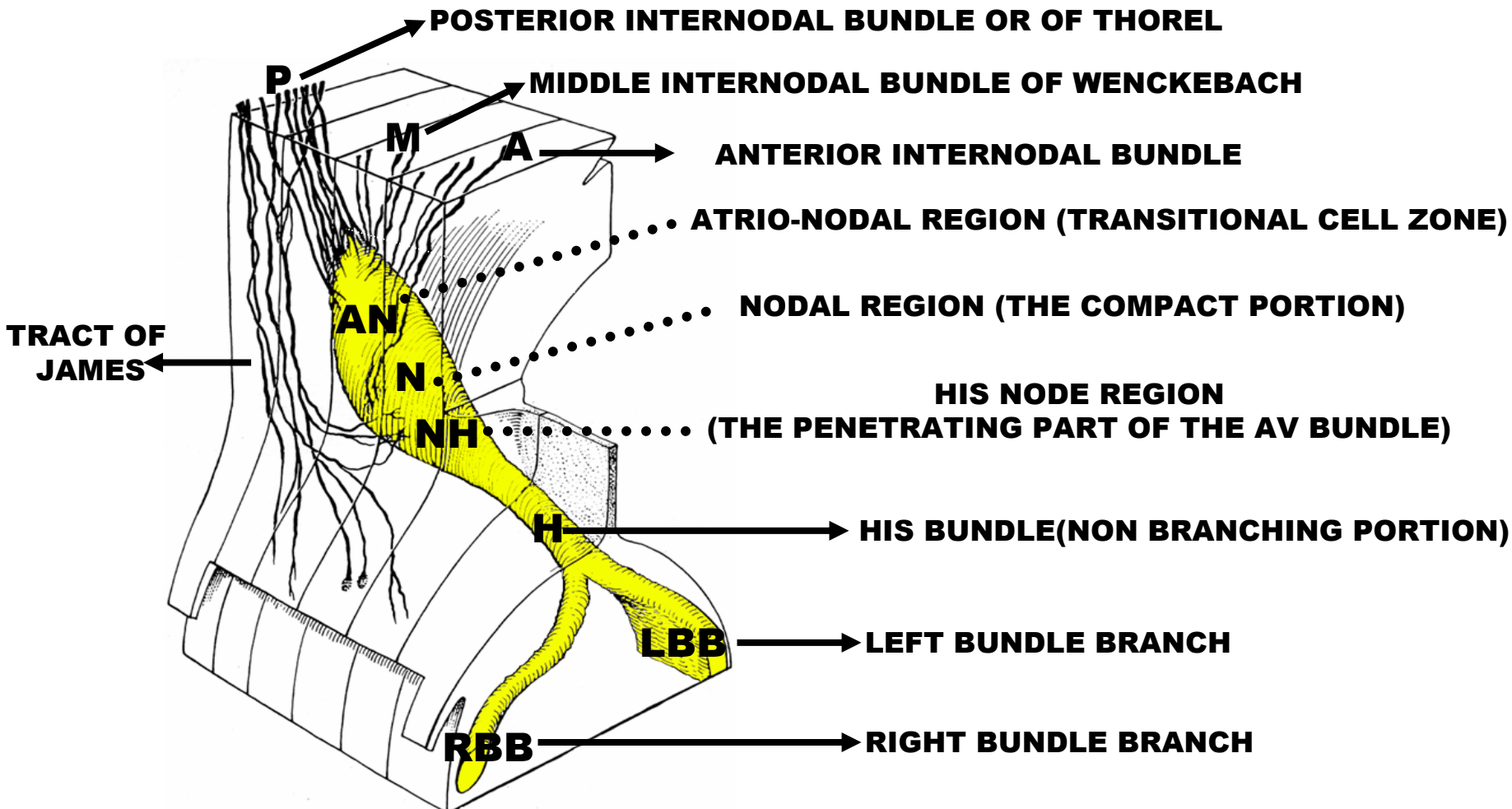


1. Kyriakidis MK, Kourouklis CB, Papaioannou JT, Christakos SG, Spanos GP, Avgoustakis DG. Sinus node coronary arteries studied with angiography. Am J Cardiol. 1983 Mar 1;51:749-750.

THE JUNCTIONAL OR ATRIOVENTRICULAR AREA

The AV junction can be divided into 3 regions as follows:

1. Transitional cell zone = AN: Atrionodal Region
2. Compact AV node = N: Nodal Region
3. Penetrating portion of the AV bundle = NH
4. Penetrating portion of His bundle = H



BLOOD SUPPLY OF THE COMPACT AV NODE

In 85% of cases AV node receives its blood supply from the **RCA**. In the remaining 13% by the **LCX** and in 2% by both arteries[1].
The AV node becomes the AV His bundle at the point where the overall axis for conduction penetrates into the central fibrous body[2].

BLOOD SUPPLY OF THE HIS BUNDLE

This structure has double blood supply: from branches of the **LAD** and **PDA**[3].

BLOOD SUPPLY OF THE LEFT BUNDLE BRANCH (LBB)

- **Branches of the PDA (90% of the RCA):** AV node artery: ramus septi fibrosi, ramus septi ventriculorum superior and ramus cristae.
- **Branches of LAD:** Ramus limbi sinistri (equivalent to ramus limbi dextri of the **LDA**).

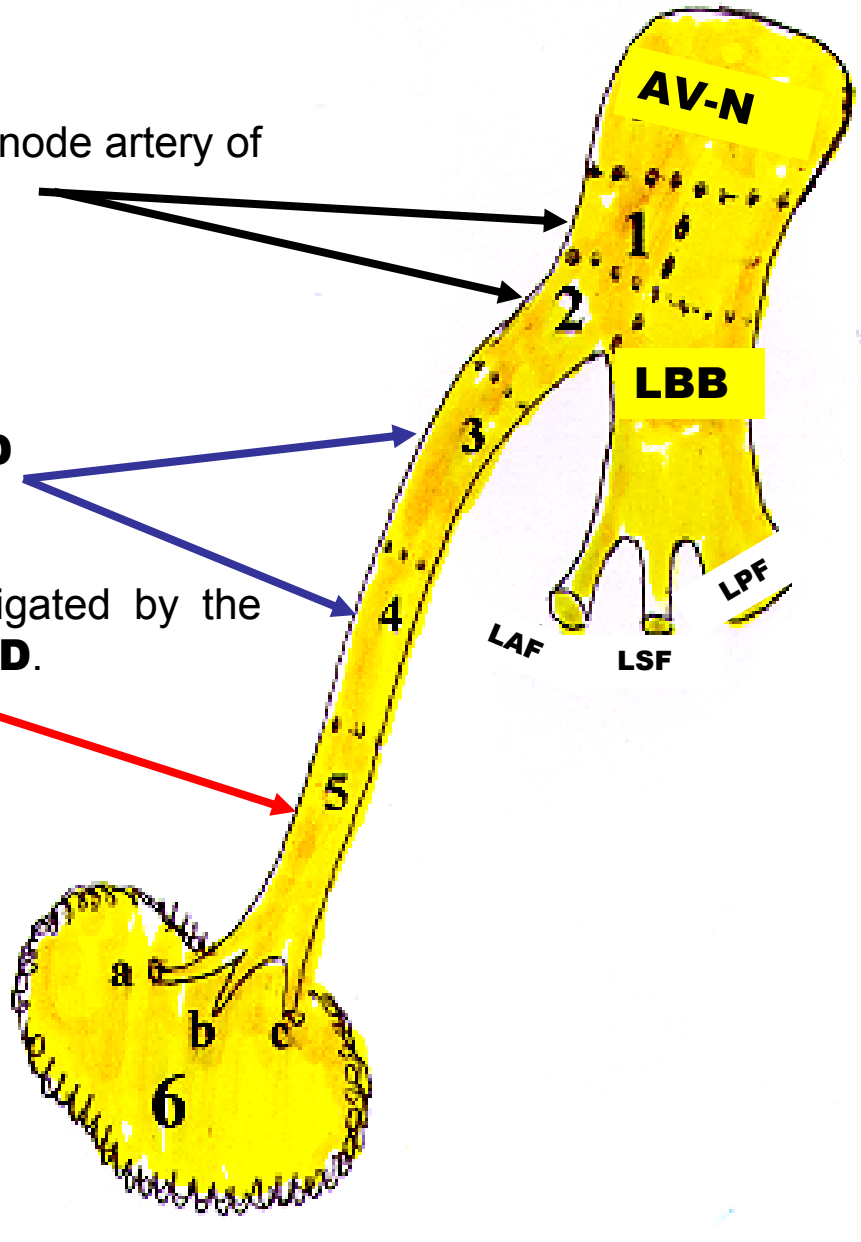
1. **Hadziselimović H. Vascularization of the conducting system in the human heart. Acta Anat (Basel). 1978;102:105-110.**
2. **Anderson RH, Ho SY, Becker AE. Anatomy of the human atrioventricular junctions revisited. Anat Rec. 2000 Sep 1;260:81-91.**
3. **Lumg G, Singletary HP. Blood Supply to the Atrioventricular Node and Bundle of His: A Comparative Study in Pig, Dog, and Man Am J Pathol. 1962 Jul;41:65-75.**

BLOOD SUPPLY OF THE RIGHT BUNDLE BRANCH (RBB)

PROXIMAL PORTION is irrigated by the AV node artery of the **RCA** and the first septal perforator artery (S_1) of the **LAD**.

MIDDLE PORTION is irrigated by:
Posterior Septal perforators of the **PDA**
Second septal perforator artery(S_2) of the **LAD**
Kugel's artery, branch of the **LCX**.

MIDDLE AND DISTAL PORTION: are irrigated by the "Ramus limbi dextri", branch of the S_2 of the **LAD**.

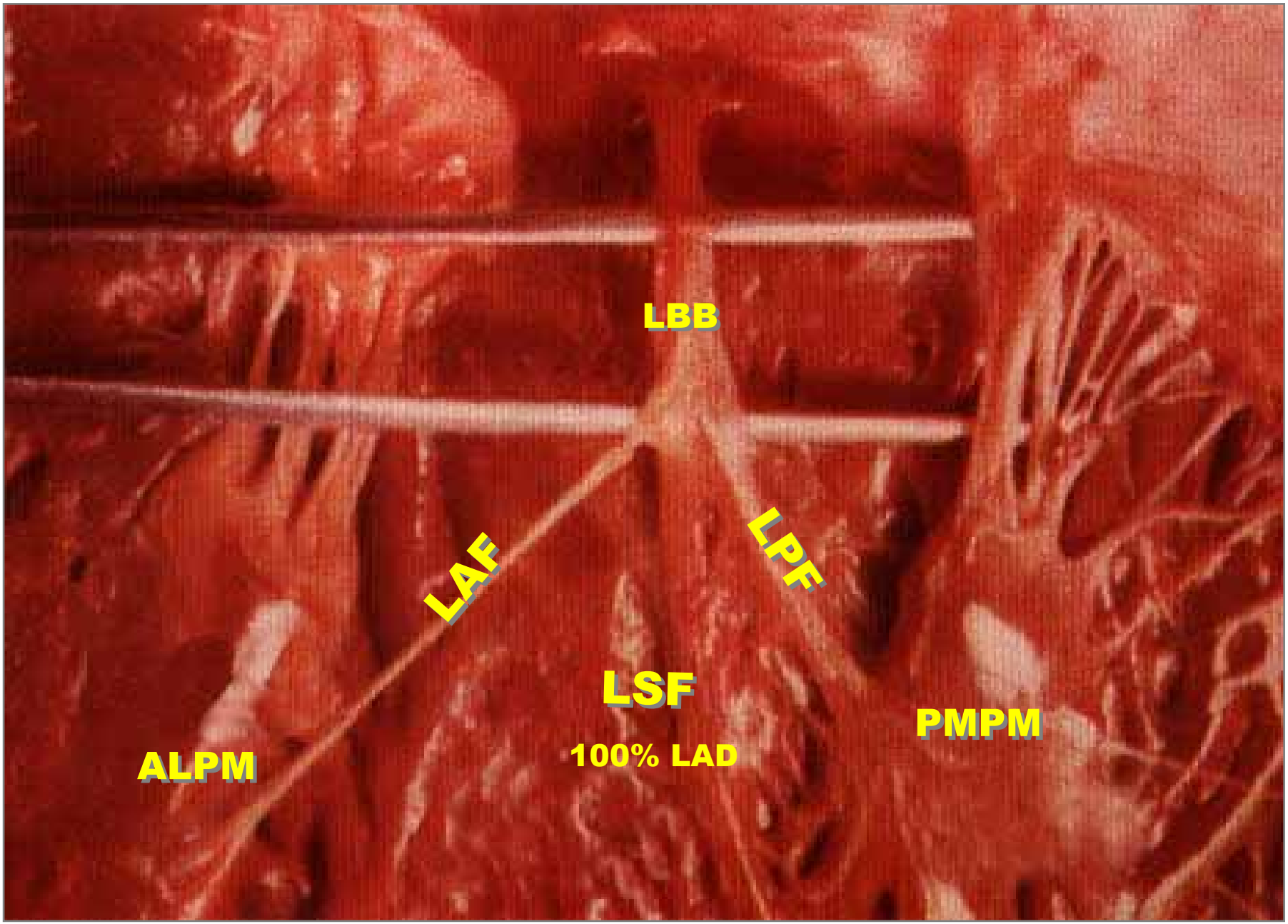


1) His Right Penetrating Portion.
2) His Right Branching Portion
3) Proximal or membranous of RBB
4) Middle, intramyocardial or mimetic
5) Inferior, distal or intra-moderator Band.

BLOOD SUPPLY OF THE LEFT FASCICLES

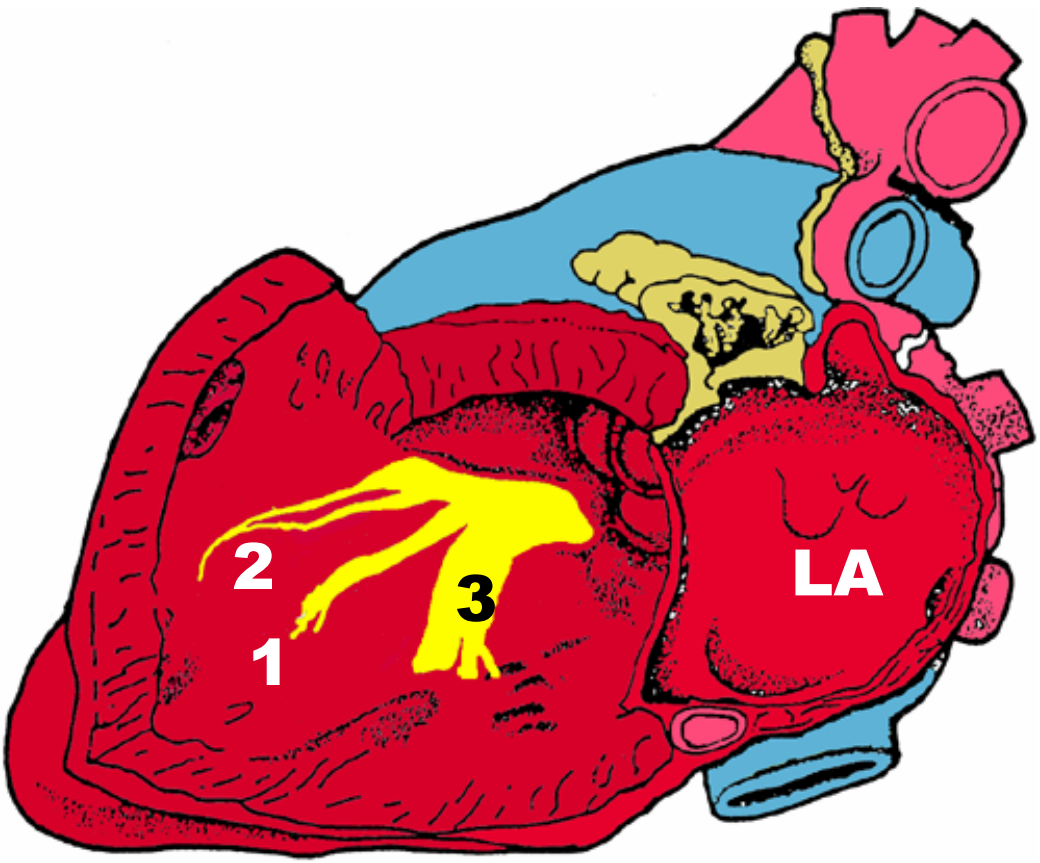
1. **Left Anterior Fascicle (LAF)** Is supplied either by septal branches of the **LAD** or by the AV nodal artery
2. **Left Posterior Fascicle (LPF):** The proximal part of LPF is supplied by the artery to the AV Node and, at times, by septal branches of the **LAD** artery. The distal portion has a dual blood supply from both anterior (S) and posterior (S`) Septal Perforator Arteries.
3. **Left Septal Fascicle (LSF) or Left Median Fascicle:** It is supplied exclusively by septal branches of the **LAD**. Critical lesions of the **LAD** before the first septal perforator, constitute the main cause of **LSFB** in the first world.

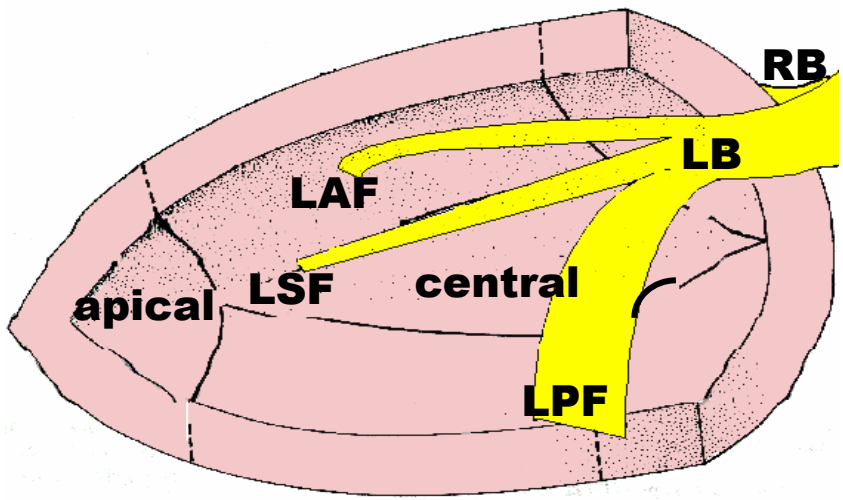
RESPONSIBLE SYSTEM	**LAF**	**LPF**	**LSF**
Branches of the LAD	40 %	10 %	100 %
Double irrigation (LAD & RCA)	50 %	40 %	0 %
RCA branches	10 %	50 %	0 %



ALPM: ANTEROLATERAL PAPILLARY MUSCLE
PMPM: POSTERO MEDIAL PAPILLARY MUSCLE

LSF DISTRIBUTION AND TRAJECTORY





1) **Left Septal Fascicle:** exclusively by septal branches of the **LAD**. Critical lesions of the **LAD** before the **S_1,** constitute the main cause of **LSFB** in the first world.
2) **Left Anterior Fascicle**
3) **Left Posterior Fascicle**.

KILLIP SCORING SYSTEM - KILLIP CLASS OR THE KILLIP-KIMBALL CLASSIFICATION[1]

The Killip classification is a system used in individuals with an acute myocardial infarction (AMI) in order to risk stratify them. Individuals with a low Killip class are less likely to die within the first 30 days after their AMI than individuals with a high Killip class. Mortality rises dramatically through the classes form I to IV. Patients were ranked by Killip class in the following way:

Killip class I: includes individuals with no clinical signs of heart failure: Absence of rales over the lung fields and absence of a third heart sound(S3).**Forrester:** wedge ≤18mm Hg. Normal perfusion.

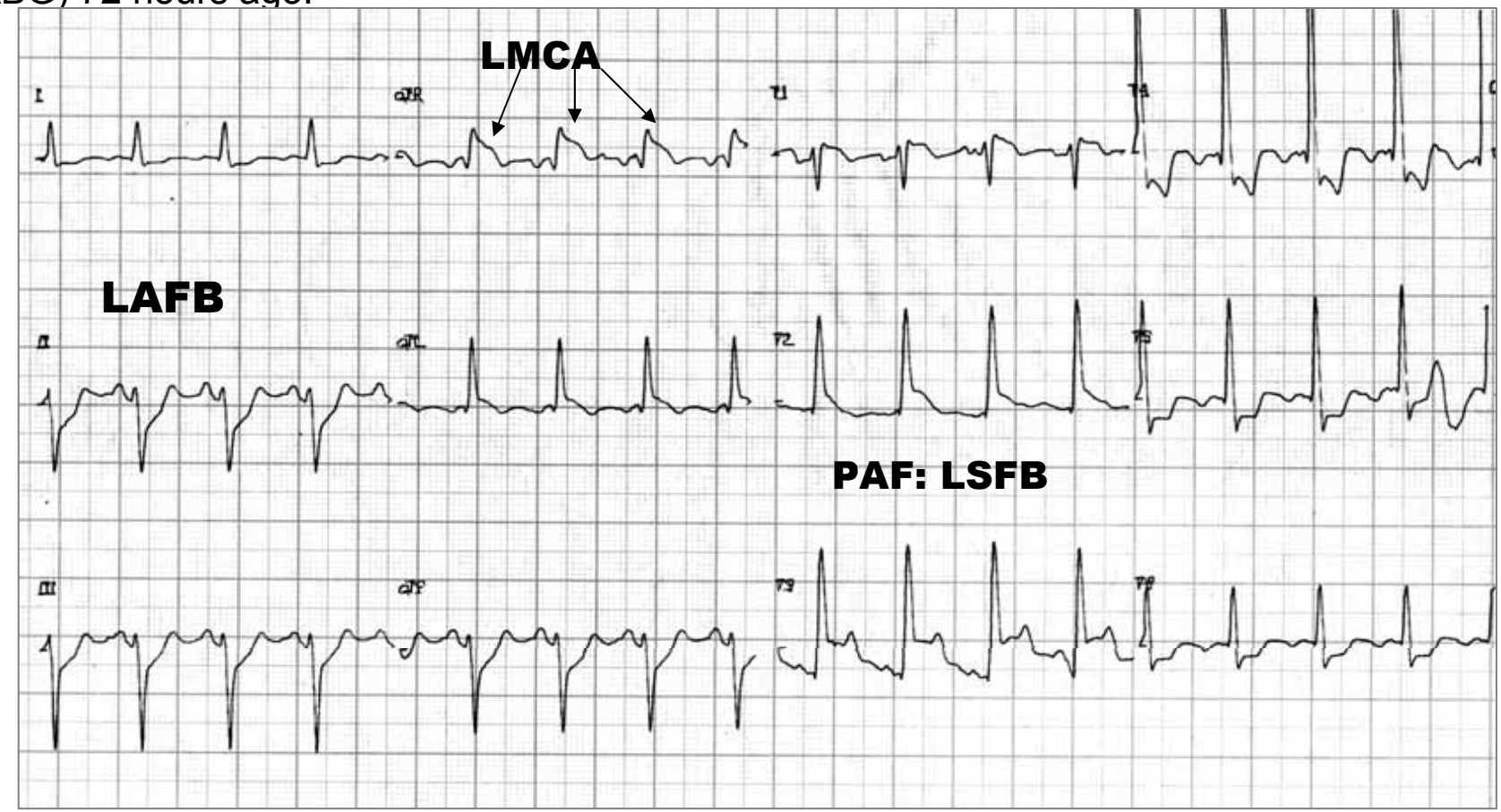
Killip class II: includes individuals with rales or crackles over 50% of the lung fields in the lungs, an S3, and elevated jugular venous pressure. **Forrester:** wedge ≤18mm Hg. Poor perfusion. Hipovolemic.

Killip class III: describes individuals with frank acute pulmonary edema: Rales over > 50% of the lung fields and S3. **Forrester:** almost normal perfusion, increased pulmonary capillary pressure and pulmonary congestion.

Killip class IV: describes individuals in cardiogenic shock or hypotension (measured as systolic blood pressure < 90 mmHg), and evidence of peripheral vasoconstriction (oliguria, cyanosis or sweating). Patients with or without lung congestion can be placed in class IV if they are in cardiogenic shock.

1. **Killip T 3rd, Kimball JT.Treatment of myocardial infarction in a coronary care unit : A Two year experience with 250 patients. Am J Cardiol. 1967 Oct; 20:457-464.**

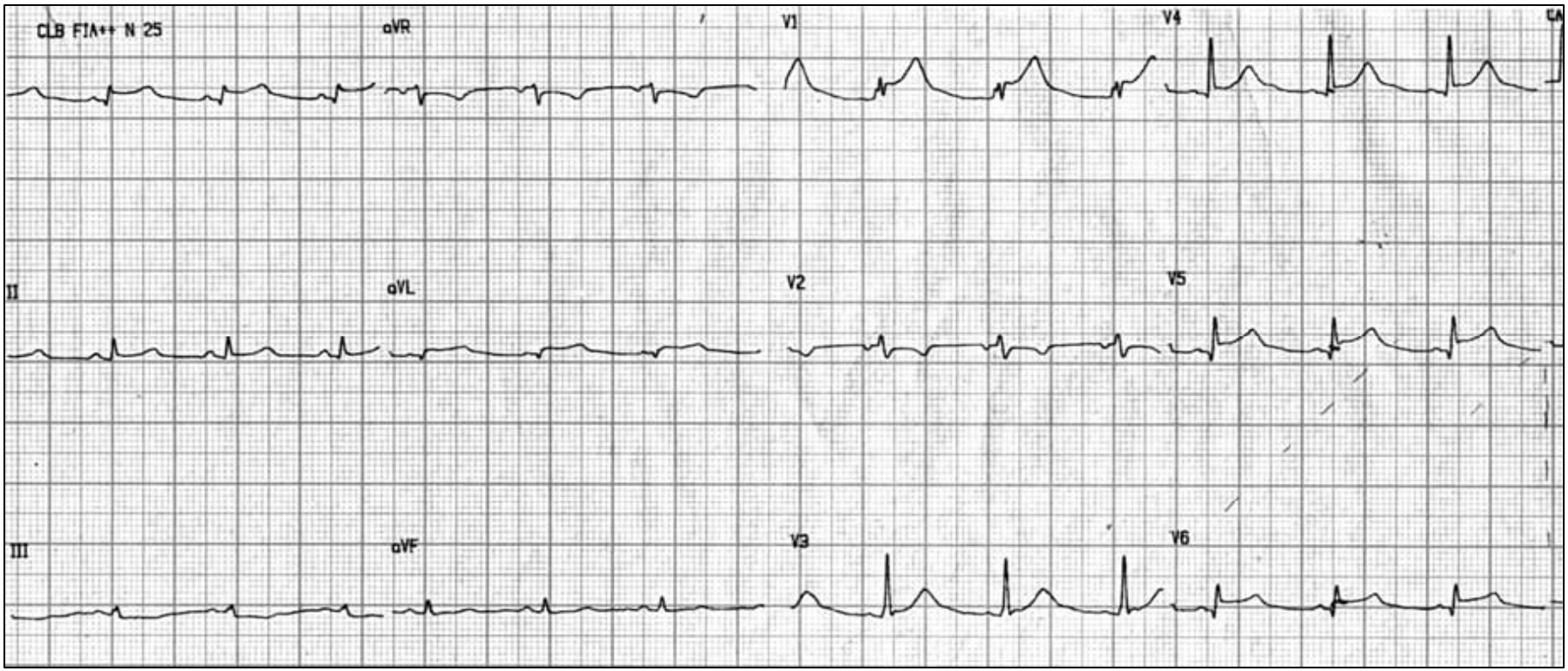
Name: AR.; **Date:** 02/01/2009.; **Age:** 72 yo.; **Gender:** Male.; **Ethnic Group:** Caucasian **Weight:** 72 Kg.; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours ago.



Clinical features: ACS: 72-year-old male patient, admitted in the emergency room with typical precordial pain that yielded after the administration of IV nitroglycerin.

ECG diagnosis: 1) LAFB + 2) LSFB: PAF + Lesion block + aVR lead with ST segment elevation suggestive of obstruction in the LMCA. **Laboratory:** There was no increase of necrosis markers (CK-MB/troponin).**The coronary angiography** revealed LMCA spasm + proximal critical lesion of the LAD. **Management:** The patient was urgently revascularized, successfully. (Coronary Artery Bypass Graft).

Name: AR; **Date:** 05/01/2009; **Age:** 72 yo;**Gender:** Male
Ethnic Group: Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic;
Management: Coronary Artery Bypass Graft (CABG) 72 hours ago.



Electrocardiogram conducted on the third day after successful surgery.
Both divisional blocks have disappeared: the extreme shift of QRS electric axis to the left in the frontal plane (LAFB) is not seen, and prominent anterior forces (LSFB) has disappeared.

Date:
02/01/2009

Date:
05/01/2009

Date:
02/01/2009

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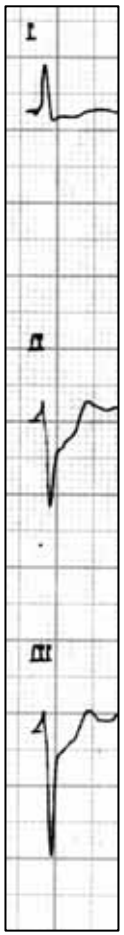
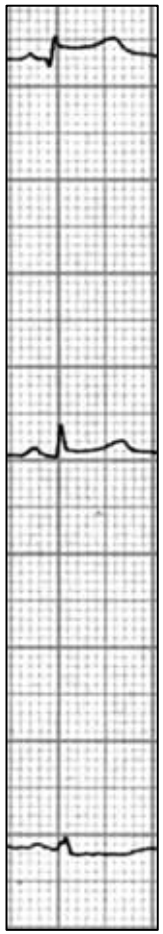
05/01/2009

I

II

III

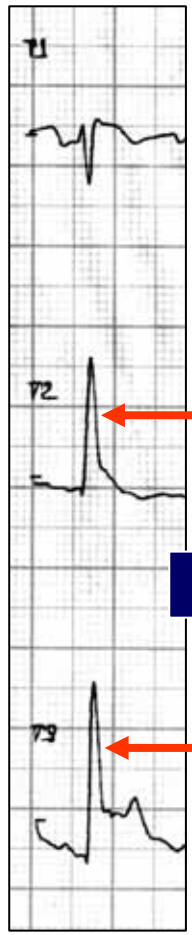




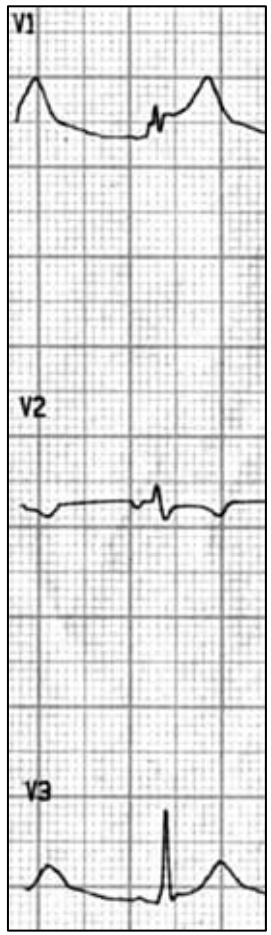
V_1

V_2

V_3



PAF



WITH LAFB

WITHOUT LAFB

WITH PAF: LSFB

WITHOUT PAF: LSFB

"TOMBSTONING" OF ST SEGMENT IN ACUTE MYOCARDIAL INFARCTION.[1]

Tombstoning (**TOMB-ST**) is manifested by a particular changing the shape of repolarization: monophasic action potential-like patter consequence of proximal occlusion of LAD.
TOMB-ST has been associated with a poor prognosis ever since Wimalaratna's first description[1;2] of this clinical entity, and the reasons for this are not fully understood.
Reperfusion injury reflected as **TOMB-ST** in patient following successful AMI PTCA[3].
TOMB-ST can be seen also in an agonal ECG
TOMB-ST pattern suggest large infarction, low LVEF, increased mortality rate, HF, VF and higher initial N-terminus pro-brain natriuretic peptide (NT-pro-BNP) level[4].
In the population with **TOMB-ST,** increased mortality was independent of the total amplitude of ST segment displacement; this relation was, however, observed in patients with STEMI without **TOMB-ST.** The sum of amplitudes of ST segment deviations (SigmaST) >20 mm Is indicative for the subgroup of patients with **TOMB-ST** and trend towards higher mortality. However, in patients without **TOMB-ST**, SigmaST >20 mm identified two subgroups with significantly different mortality rates (20% vs 4%, p=0.001) [5].
Rarely **TOM-ST** is secondary to acute pericarditis. In these rare cases the role of two-dimensional echocardiogram is important[6].

1. **Wimalaratna HS. "Tombstoning" of ST segment in acute myocardial infarction. Lancet. 1993 Aug 21;342(8869):496.**
2. **Birnbaum Y, Sclarovsky S. "Tombstoning" of ST segment in acute myocardial infarction. Lancet. 1993 Dec 11;342(8885):1494.**
3. **Dalal J, Chambers CE.Marked ST elevation after successful PTCA for acute myocardial infarction. J Invasive Cardiol. 1994 Oct;6:263-266.**
4. **Tomcsányi J, Marosi A, Bózsik B, Somlói M, Zsoldos A, Vecsey T, et al. N-terminal pro-brain natriuretic peptide and tombstoning ST-segment elevation in patients with anterior wall acute myocardial infarction. Am J Cardiol. 2005 Nov 1;96:1197-1199.**
5. **Kukla P, Dudek D, Szczuka K. "Tombstoning" of ST segment in acute myocardial infarction -- effect on clinical course. Kardiol Pol. 2006 Mar;64:275-80.**
6. **Jain A. "Tombstone" anterior ST-segment elevations secondary to acute pericarditis: the role of two-dimensional echocardiogram. Clin Cardiol. 1997 Apr;20:404-406.**

TOMB-ST ECG CHARACTERISTICS

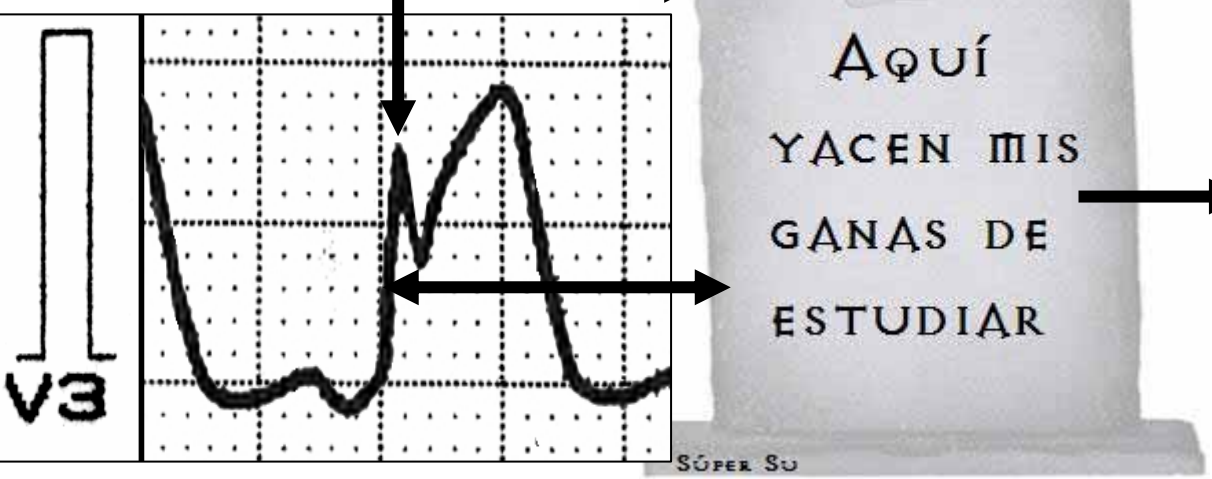
An ST-segment elevation with a specific pattern is the principal element of TOMB-STEMI. ST-segment elevation is often the earliest detected sign of acute MI. Initially, the ST segment may straighten, with loss of the ST-T wave angle. Then the T wave becomes broader and the ST segment elevates, losing its normal concavity. As further elevation occurs, the ST segment tends to become convex upwards. As ST-segment elevation can be minimal, in some cases, it may surpass the peak level of the R wave.

Thus, ST-segment elevation surpassing the R wave exhibits such a morphological appearance that it reminds a tombstone.

Tombstone appearance

ST segment convex to the top

Ápex R wave

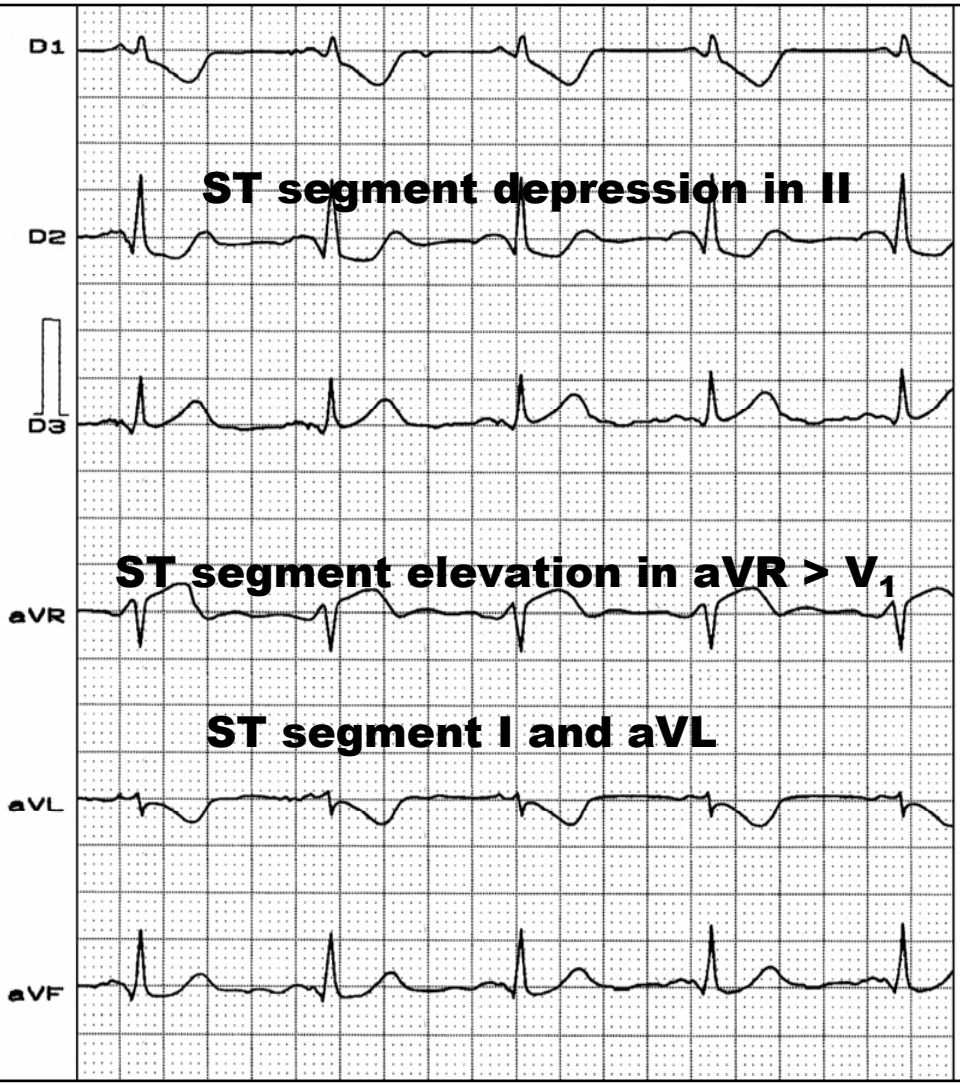


Here lie my desire to study

Two electrophysiological mechanisms play a role in the formation of a tombstone appearance: delayed transmural conduction and intramyocardial conduction block.

TYPICAL ECG PATTERN OF LMCA OCCLUSION

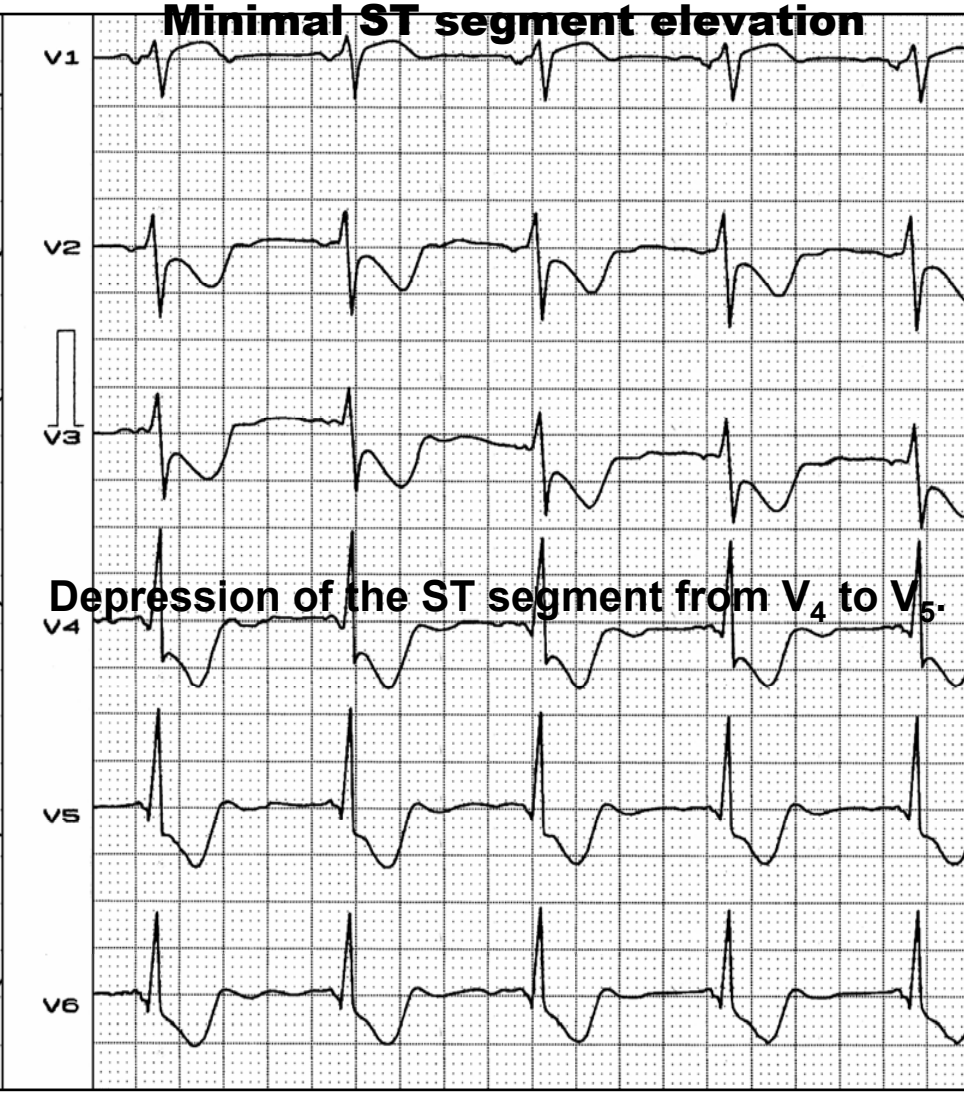
Diffuse ST segment depression in the inferolateral leads



ST segment depression in II

ST segment elevation in aVR > V_1

ST segment I and aVL

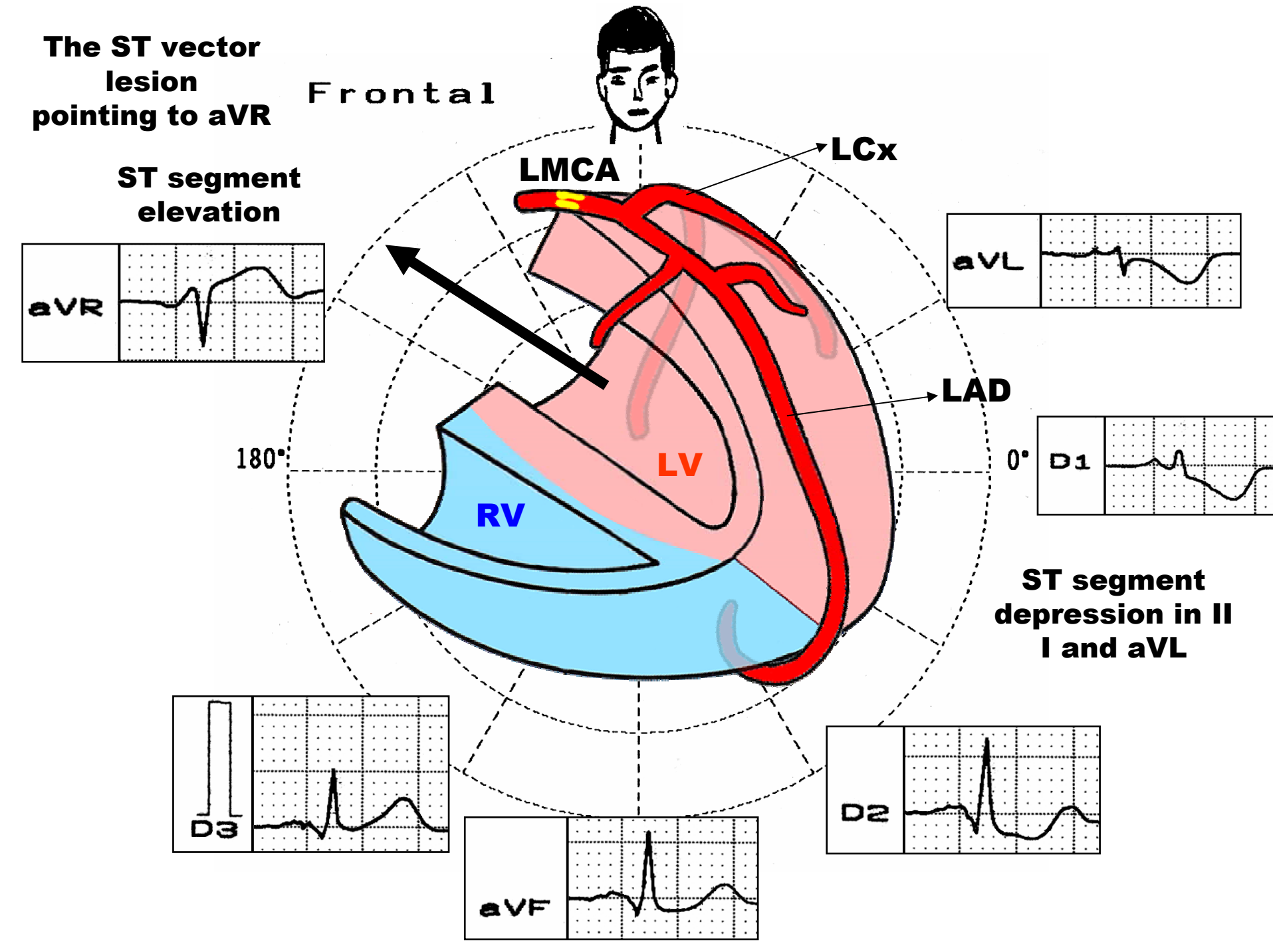


Minimal ST segment elevation

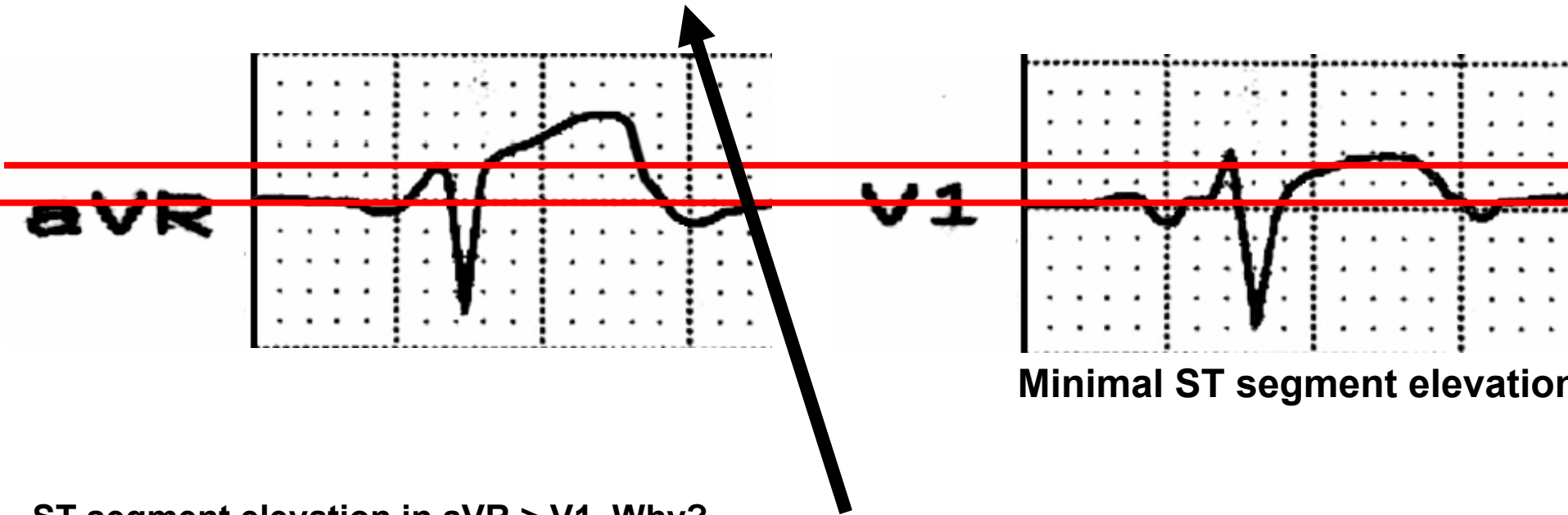
Depression of the ST segment from V_4 to V_5.

Why this pattern is observed?

ST segment depression in V_6 > ST segment elevation in V_1.

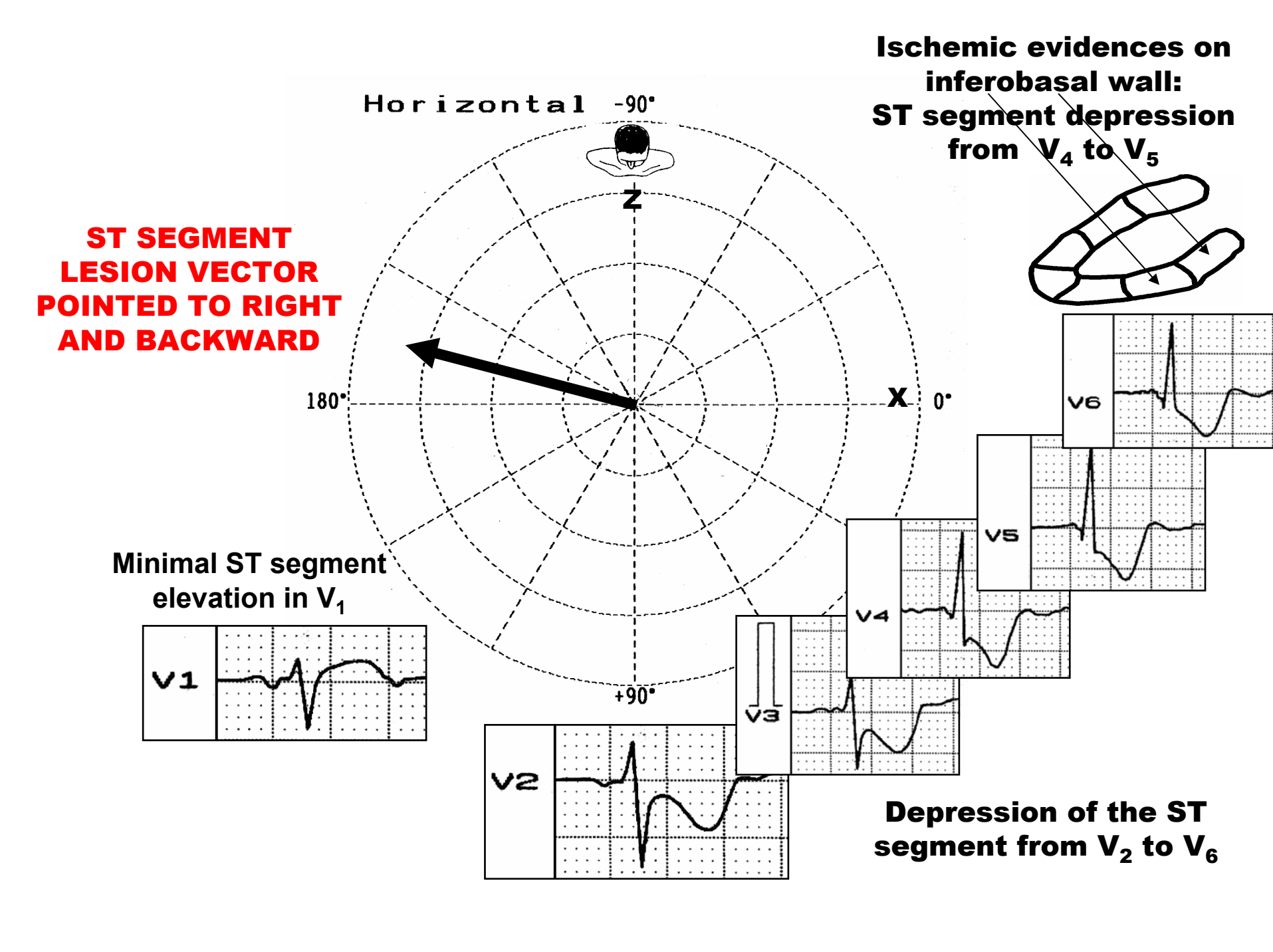

The ST vector lesion pointing to aVR
Frontal
ST segment elevation
LMCA
LCx
LAD
aVR
aVL
D1
180°
0°
LV
RV
ST segment depression in II I and aVL
D3
D2
aVF

aVR STSE > V_1 STSE



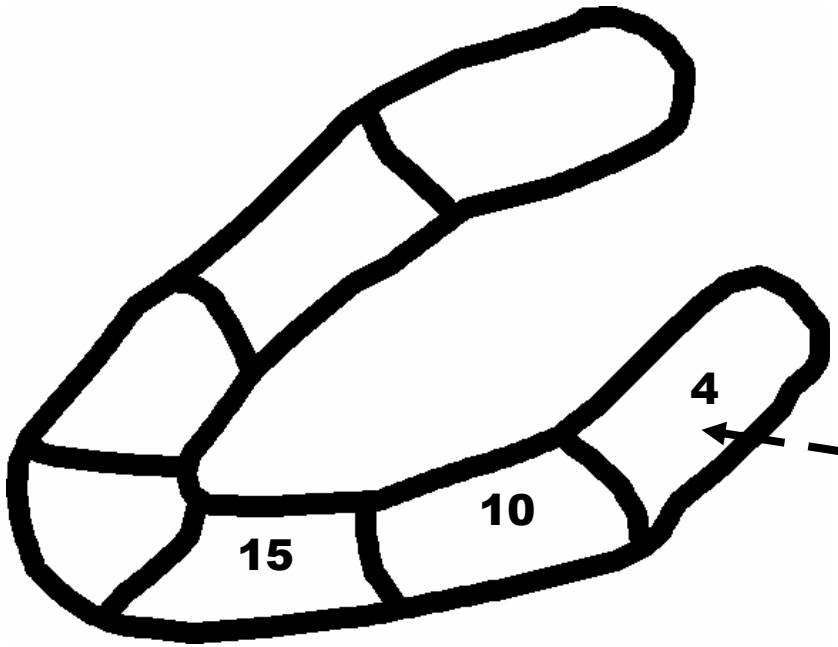
Minimal ST segment elevation

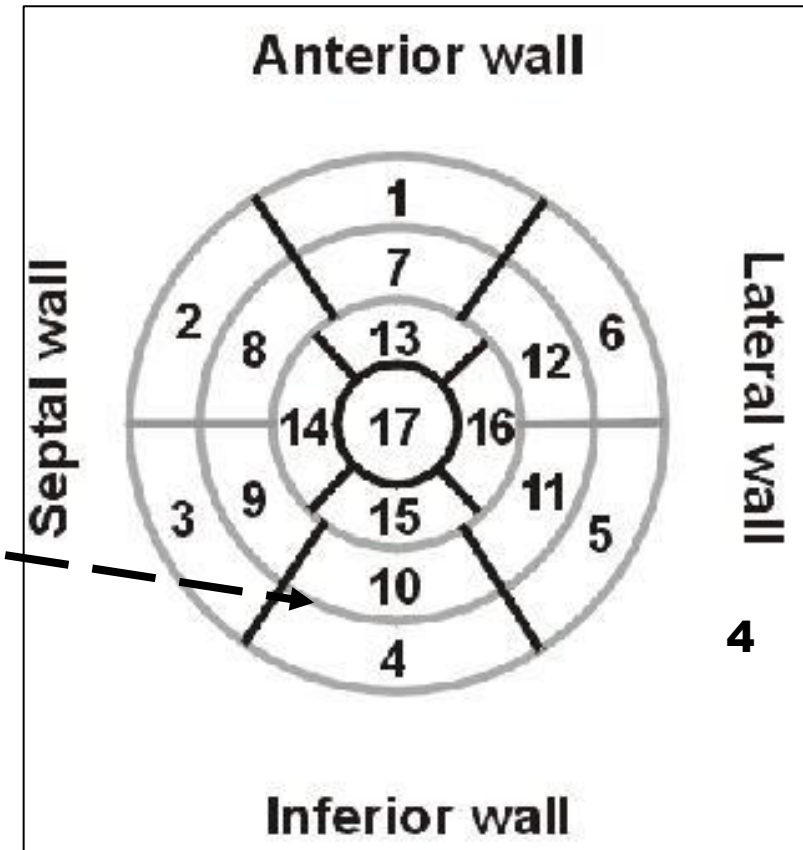
ST segment elevation in aVR > V1. Why?
Because ST segment lesion vector is directed to upward and rightward, pointing to aVR lead(RVOT)


Horizontal -90°
Z
180°
X
0°
+90°
ST SEGMENT LESION VECTOR POINTED TO RIGHT AND BACKWARD
Ischemic evidences on inferobasal wall: ST segment depression from V4 to V5
Minimal ST segment elevation in V1
V1
V2
V3
V4
V5
V6
Depression of the ST segment from V2 to V6

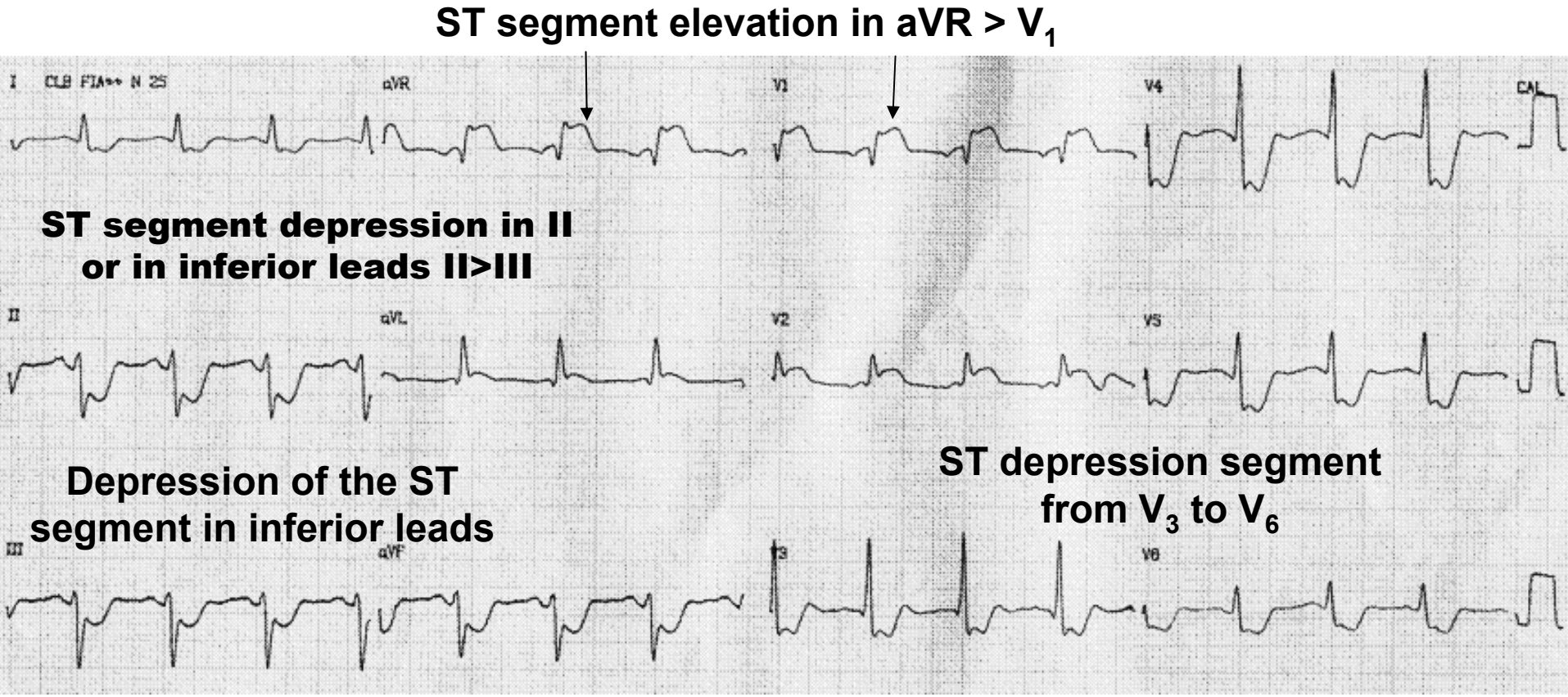
LMCA OCCLUSION ECG CRITERIA

- **ST segment elevation in aVR, and V_1**
- **ST segment elevation in aVR > V_1**
- **Ischemic evidences in inferobasal* wall: depression of the ST segment in II and from V_4 to V_5**
- **ST segment depression in II or in inferior leads II>III**
- **Depression of ST segment in V_6 > ST segment elevation in V_1**
- **Diffuse ST segment depression in the inferolateral leads**
- **Eventually observation of RBBB, LAFB and/or LSFB.**



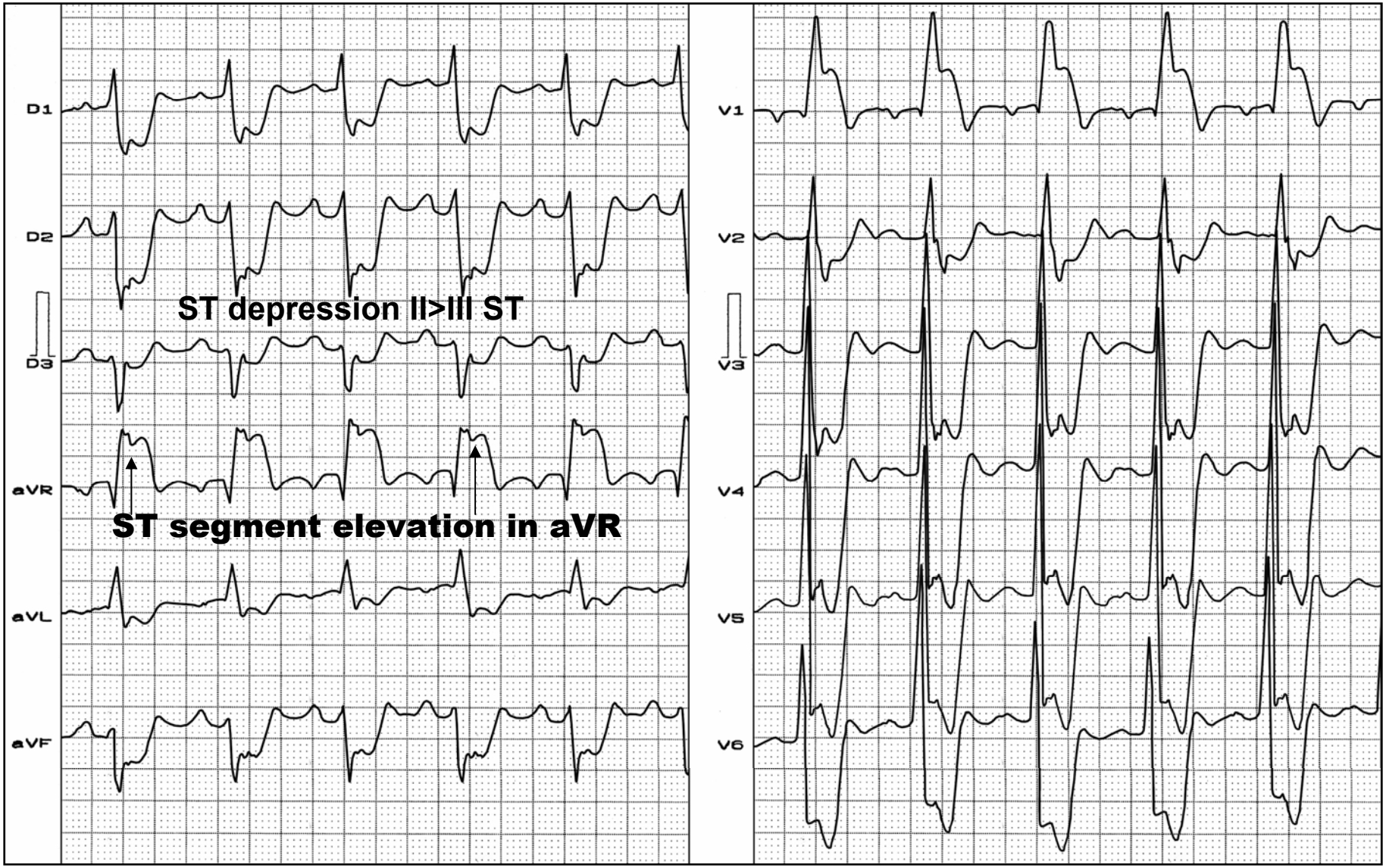


* Formally called inferodorsal wall



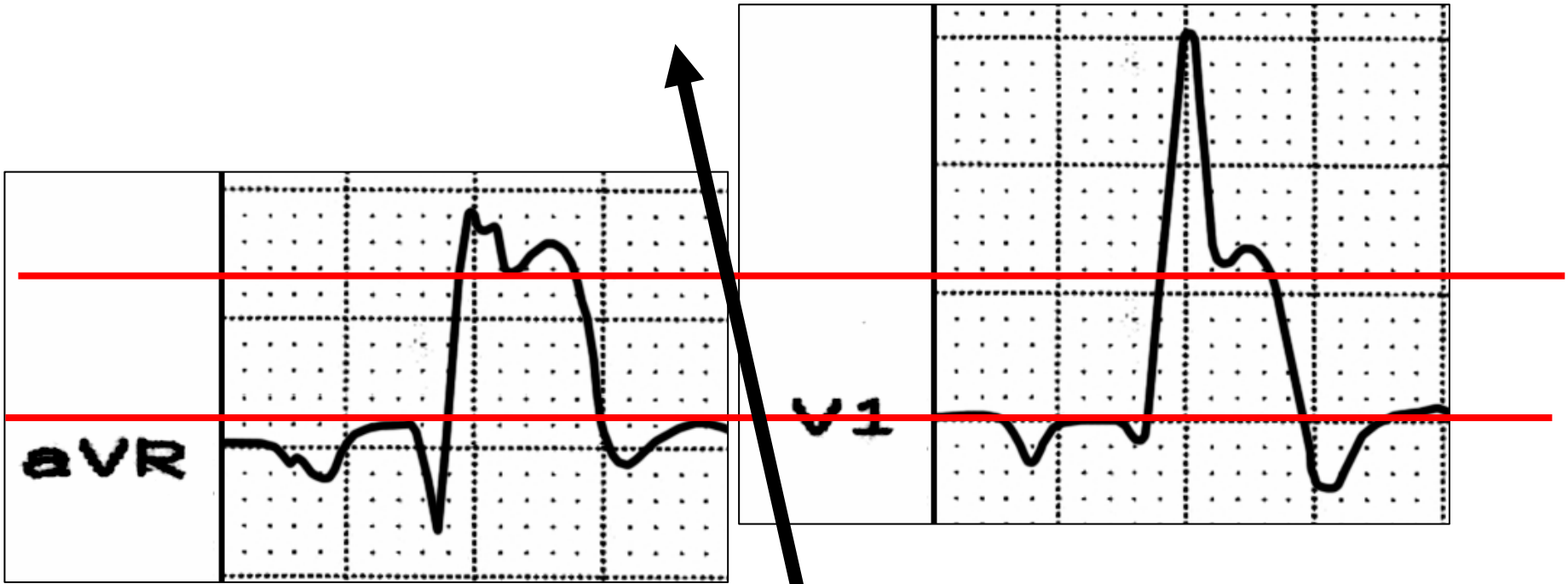
Clinical Picture: Acute Coronary Syndrome associated with cardiogenic shock (Killip class IV) consequence of total occlusion of LMCA.
Primary Angioplasty was performed, with immediately hemodynamic stabilization.

LMCA Occlusion complicated with Complete RBBB.



ST segment elevation in aVR and V_1 (aVR > V_1.). ST depression II>III ST depression segment from V_2 to V_6.

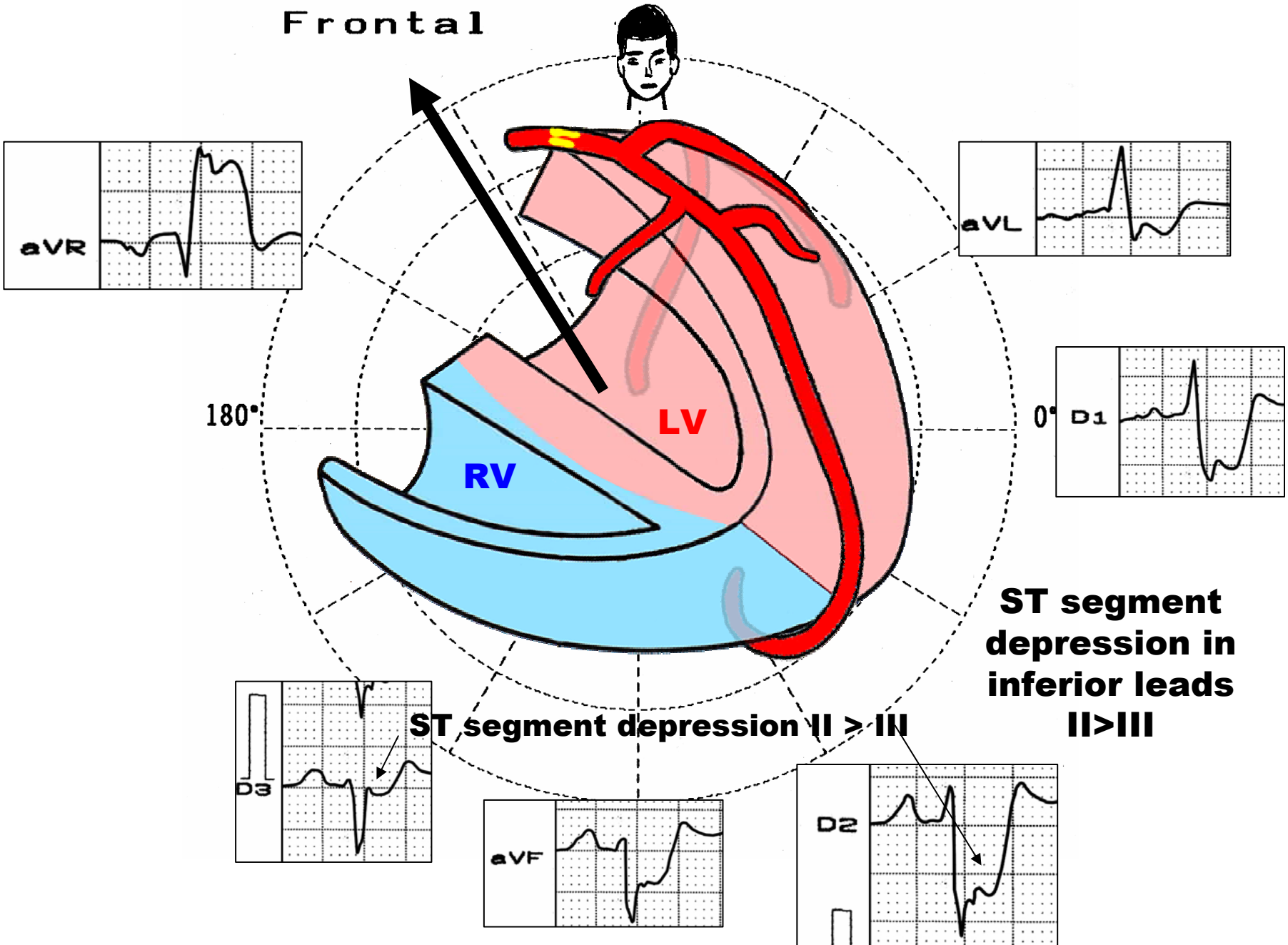
ST segment elevation in aVR > V_1

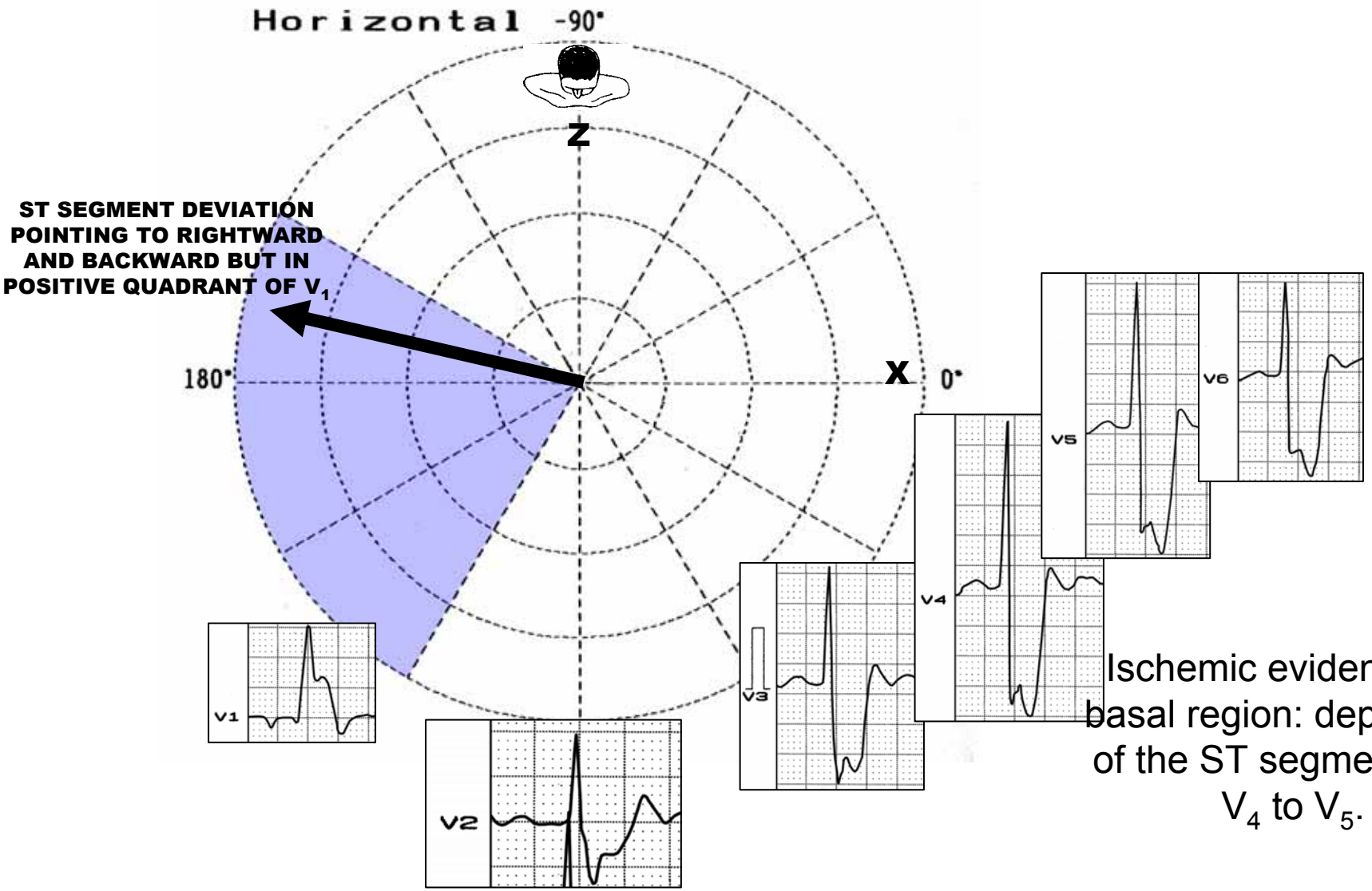


ST segment elevation = 6mm

ST segment elevation = 5,5mm

QRS AXIS LOCATED IN RIGHT SUPERIOR QUADRANT



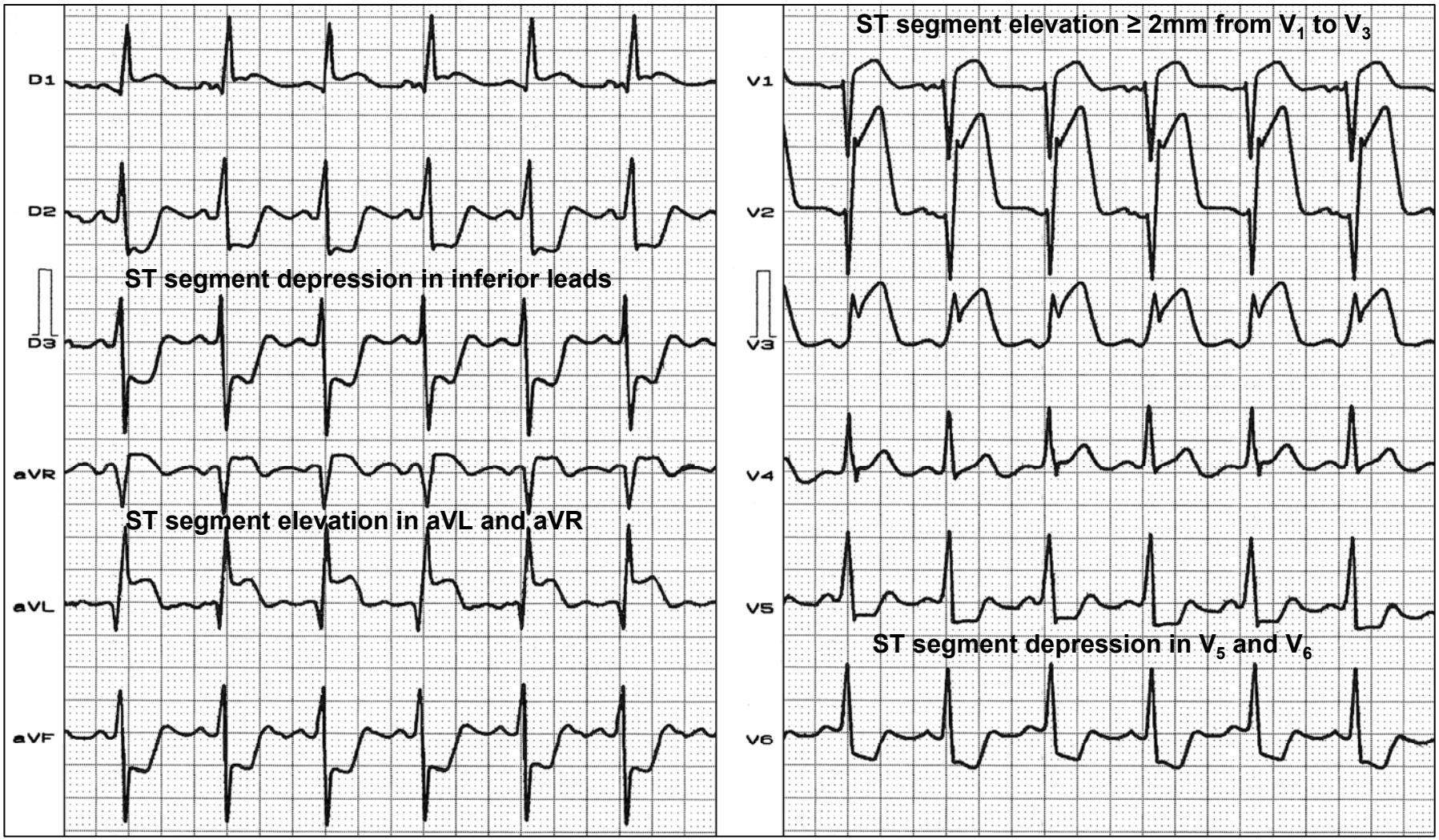


Ischemic evidences in basal region: depression of the ST segment from V_4 to V_5.

= **Positive quadrant of V_1. The ST segment vector is inside of positive quadrant of V_1**

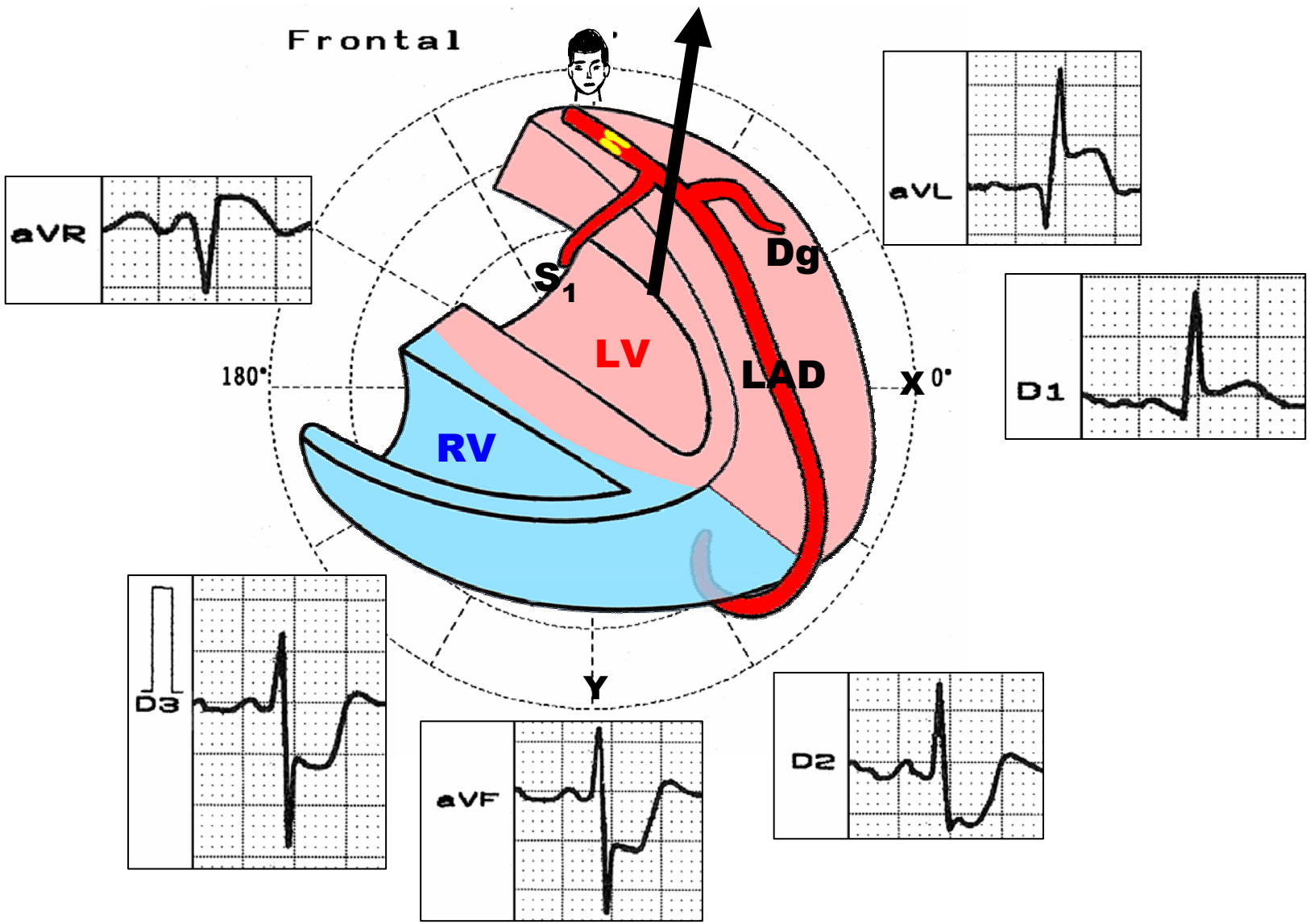
LEFT ANTERIOR DESCENDING ARTERY (LAD) OCCLUSION BEFORE FIRST SEPTAL PERFORATOR(S_1): PROXIMAL LAD OCCLUSION

AMI consequence of occlusion of LAD before the first septal perforator and the first diagonal branch



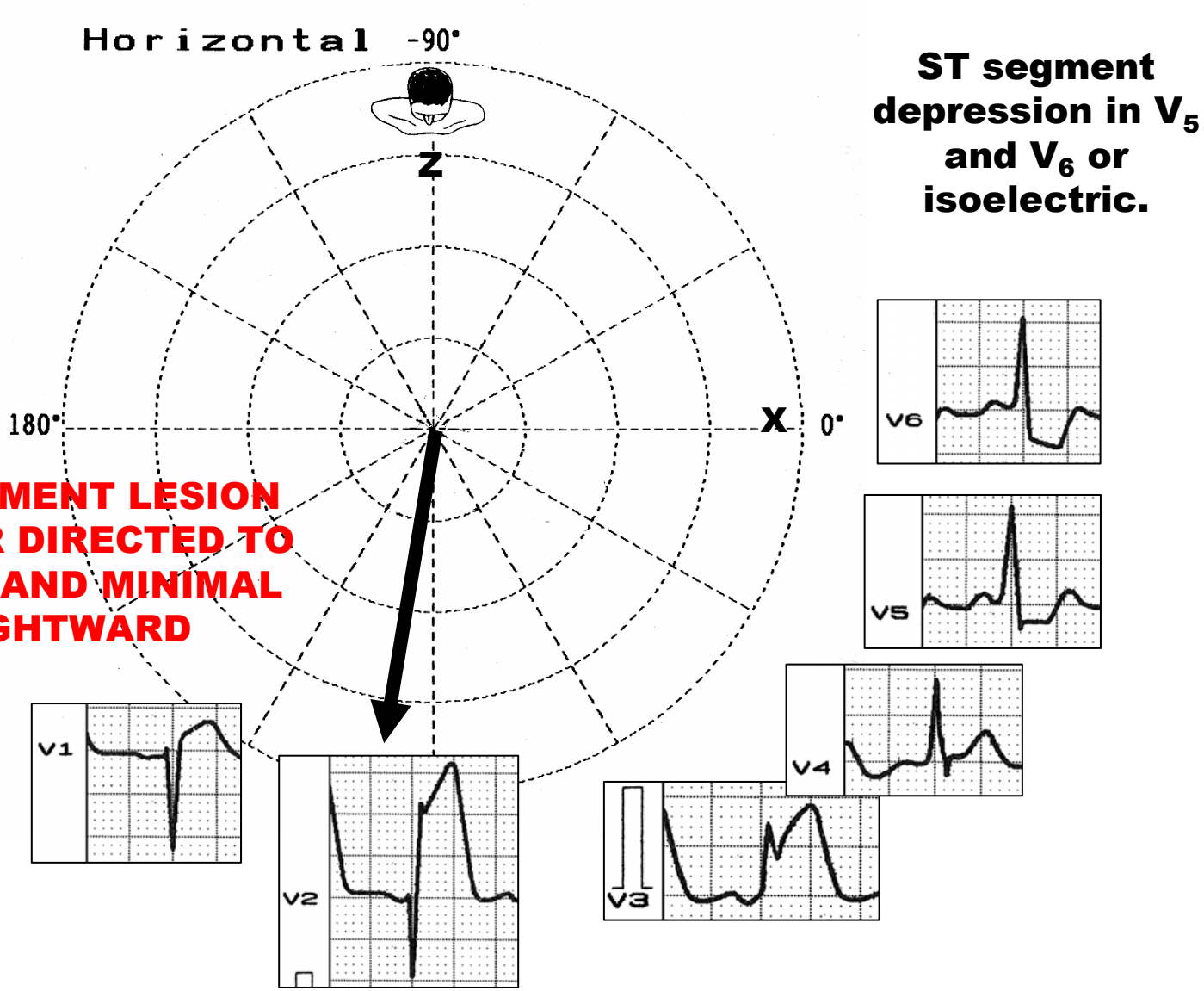
Why we observe this pattern?

ST SEGMENT LESION VECTOR DEVIATION



ST SEGMENT VECTOR LESION POINTING TO UP, CAUSING ST SEGMENT ELEVATION IN aVL AND aVR AND ST SEGMENT DEPRESSION IN INFERIOR LEADS.

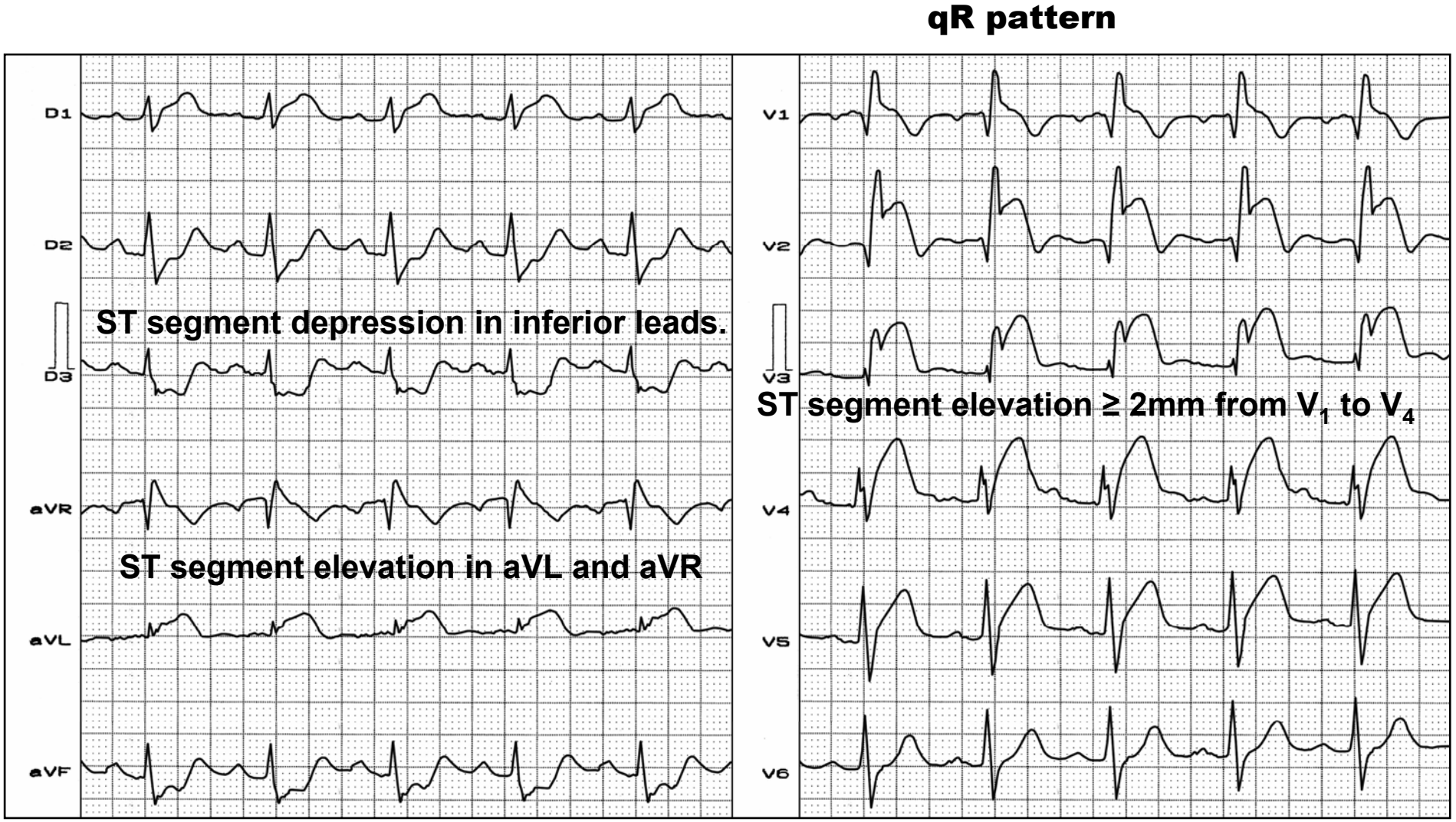
ST segment elevation ≥ 2mm from V_1 to V_3 or V_4 (ST segment lesion vector directed to front). ST segment depression in V_5 and V_6 or isoelectric. Eventually CRBBB and/or LAFB and/or LSFB.



ST segment depression in V_5 and V_6 or isoelectric.

ST segment elevation ≥ 2mm from V_1 to V_3 or V_4

AMI consequence of proximal LAD occlusion before S_1 complicated with RBBB.



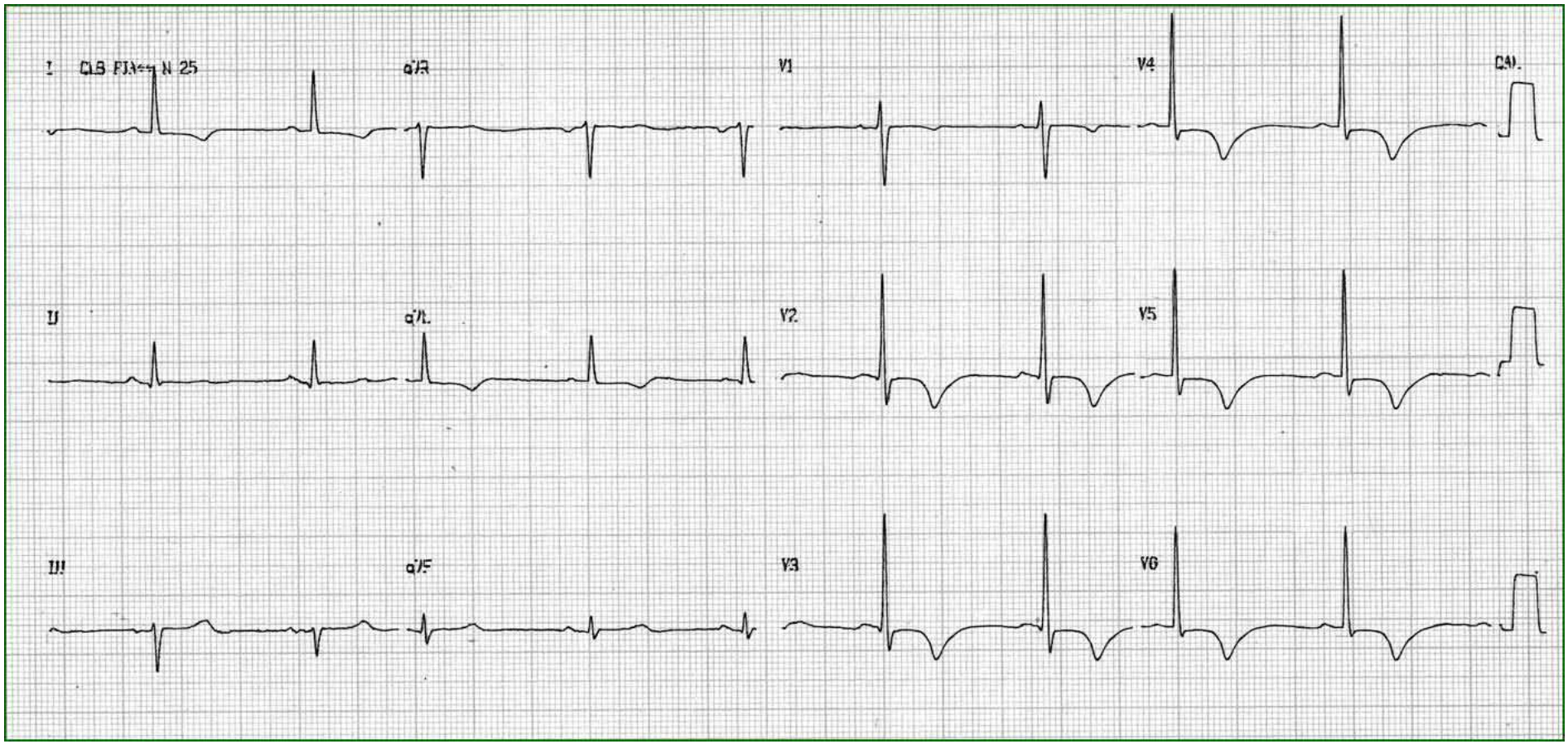
WELLENS' SYNDROME, LAD CORONARY T-WAVE SYNDROME OR ACUTE CORONARY T-WAVE SYNDROME.

Wellens' syndrome is a clinical-electrocardiographic entity.

It is a complex of symptoms and signals indicating the existence of an undesirable condition secondary to critical high-grade proximal stenosis of the LAD coronary artery characterized by the association of:

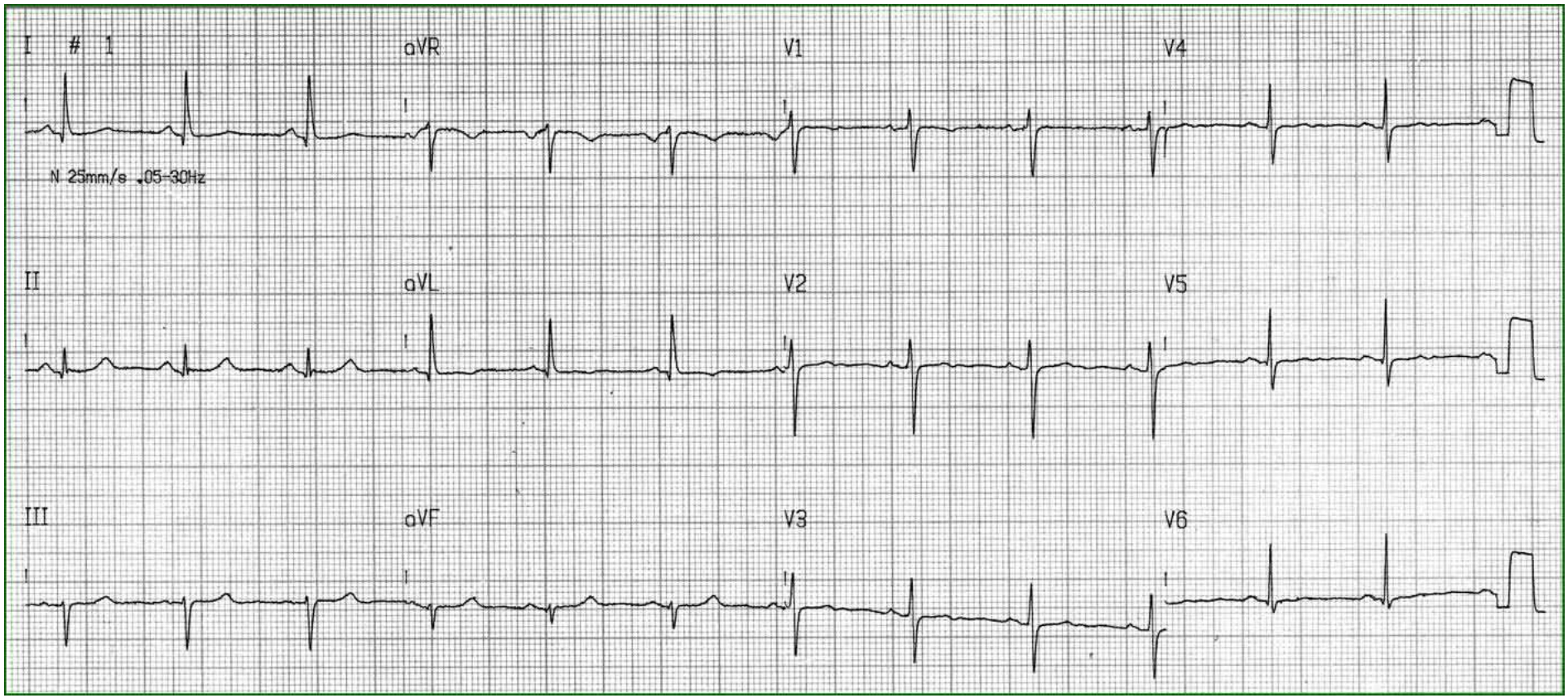
1) **Prior history of ACS**
2) **Little or no elevation of markers of myocardial damage (unstable angina)**
3) **Characteristic ECG changes consistent with subepicardial anterior ischemic pattern in the LAD territory (V_1 through V_5 or V_6):**
   - **Plus-minus T waves with inversion of the terminal portion: Type 1**
   - **Persistently symmetrical, deep negative and broad-based T-waves: Type 2**
4) **Sensitivity and specificity for significant (≥ 70%) stenosis of the LAD artery was found to be 69% and 89% respectively with positive predictive value 86%.[2]**

1. de Zwaan C, Bär FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. Am Heart J. 1982 Apr;103:730-736.
2. Haines DE, Raabe DS, Gundel WD, Wackers FJ. Anatomic and prognostic significance of new T-wave inversion in unstable angina. Am J Cardiol. 1983 Jul;52:14-18.

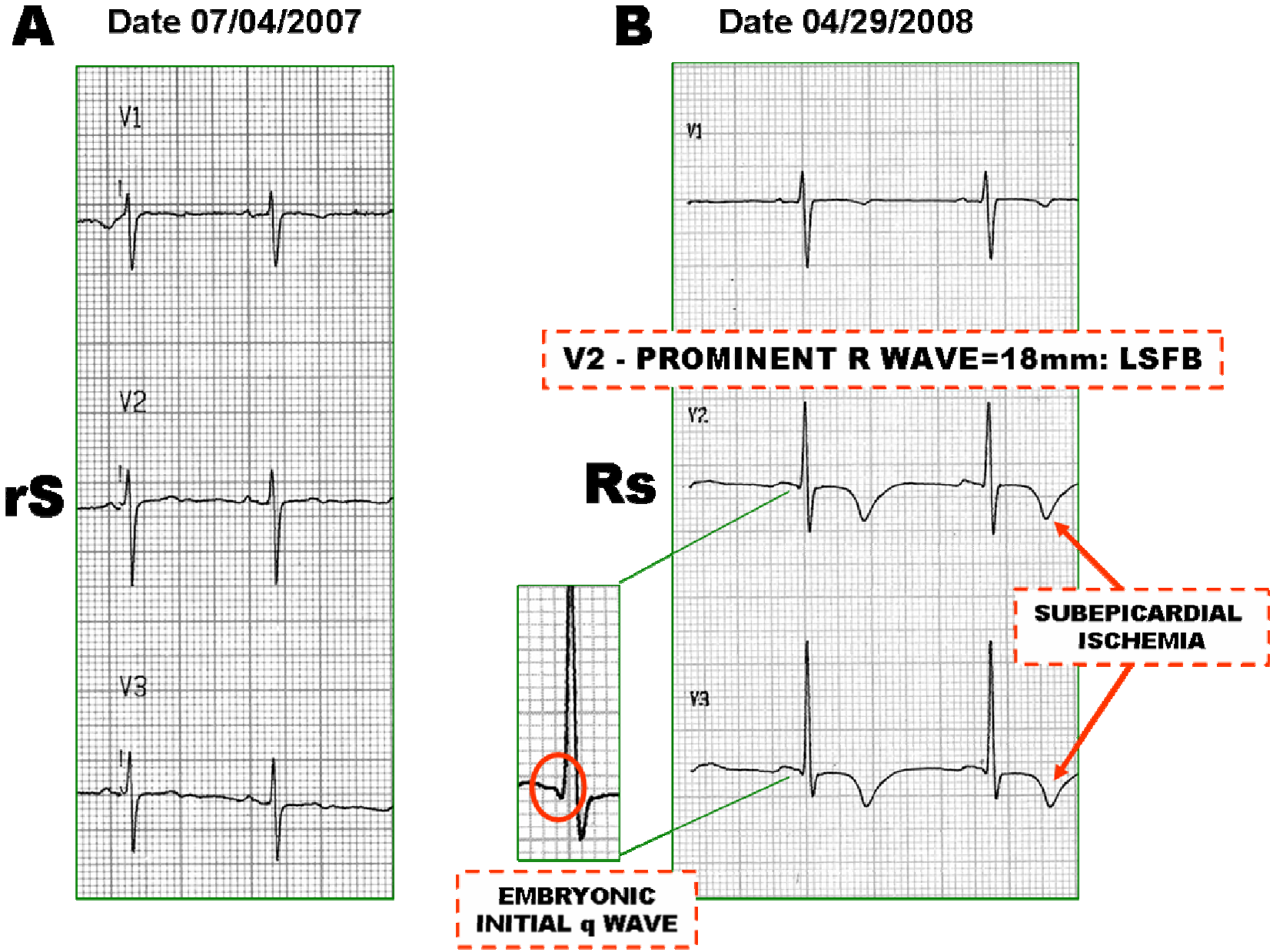


ECG performed upon arrival to the Emergency Department (04/29/2008), and while having chest pain. Deep negative and broad-based T-wave inversions in precordial leads from V_2 through V_6, with high voltage R wave in V_2 (R=18mm). Initial small q waves were observed in V_2-V_3. Left septal initial q waves in left leads are absent. R/S ratio in V2 > 2. S wave depth in V_2 < 5 mm. Conclusion: Type 2 Wellens' pattern associated with prominent anterior forces: several Left Septal Fascicular Block criteria are present.

1. **Riera AR, Ferreira C, Ferreira Filho C, Dubner S, Schapachnik E, Uchida AH, Moffa P, Zhang L, de Luna AB. Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block? J Electrocardiol. 2008 Nov-Dec;41:671-674.**

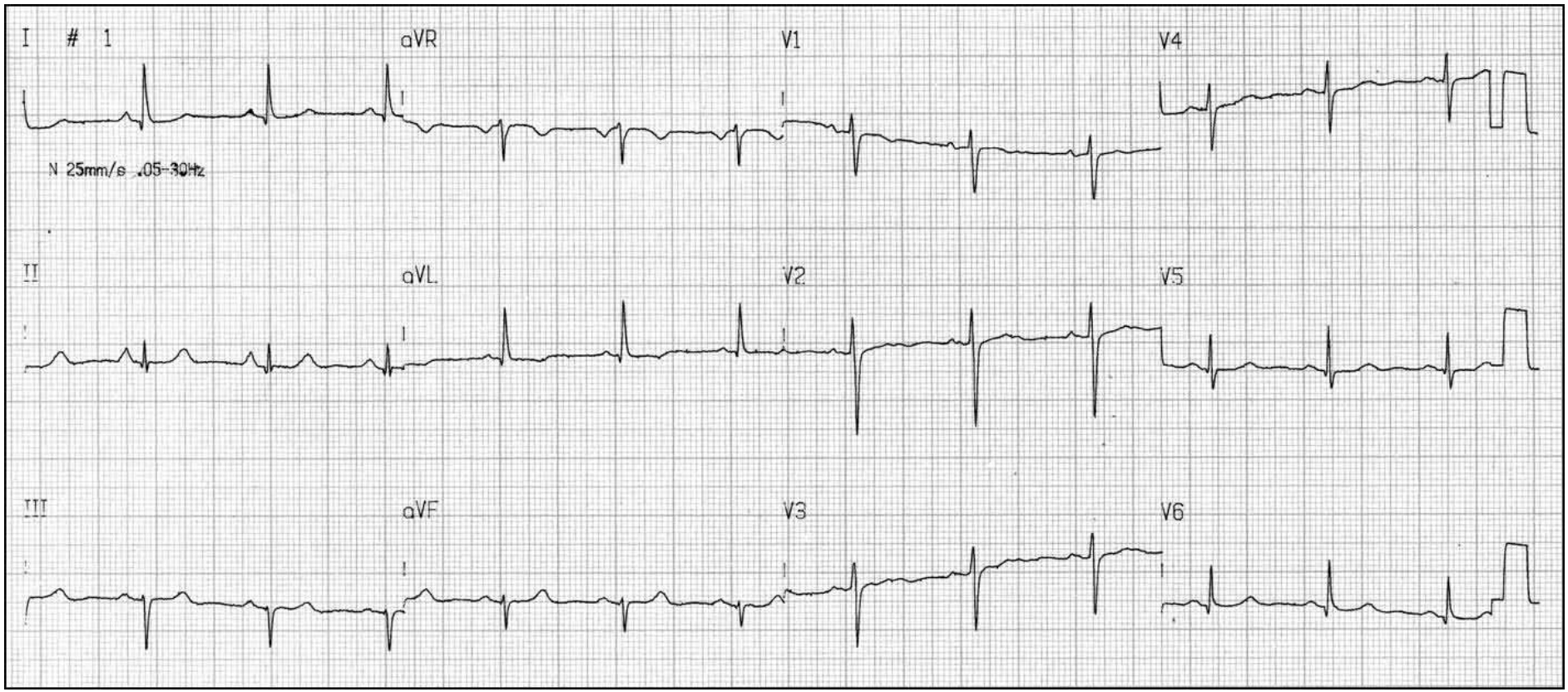


ECGs performed one year before clinical manifestation (07/04/2007). There are not subepicardial T wave ischemic pattern and QRS complexes of the rS type in V_2.
Initial q wave are observed in left leads I, aVL, V_5 and V_6.



A) Basal anteroseptal precordial leads performed approximately one year before onset of clinical picture.

B) The same leads performed during the clinical manifestation.



ECG performed ten days after (05/09/2008) the successful placement of the stents in LAD. The ischemic pattern had disappeared, the lead V_2 returned to rS, the initial q wave in V_3 disappeared, and small septal q waves appeared in the left leads I, aVL, V_5 and V_6.