Acute Coronary Syndrome in senior man

Case Report

Good afternoon Dear Master: I submit for your consideration the following case: It is 74 years old male patient. He was received at the emergency room complaining of chest pain.

background:

History of acute myocardial infarction eight years ago.

Angina pectoris and heart failure secondary to myocardial infarction

Smoking longstanding currently suspended

He had an ischemic stroke without sequelae a time ago.

And unspecified treatment.

With a history of three days effort angina that does not improve with medication. Episodes of angina becoming longer and more often to come to rest, to start a night prior to admission with severe chest pain, no symptoms added, not giving up to rest or medication.

Vital signs: BP 90/60, HR: 95, Sat 98% without mask, CPK: 453 , CPK MB 91, TGO 108 DHL 616

ECGs attached: ECG-1 is the entrance and ECG 2 is 6 hours later

The patient was referred to the hemodynamics laboratory for Percutaneous Transluminal Coronary Angioplasty (PTCA)

Hope will be of interest

Thanks

Jesus Antonio **Campuzano Chacon** M.D.

Emergency physicians specialist. Public Hospital del Instituto Mexicano del Seguro Social (IMSS) HGZ 33

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Spanish

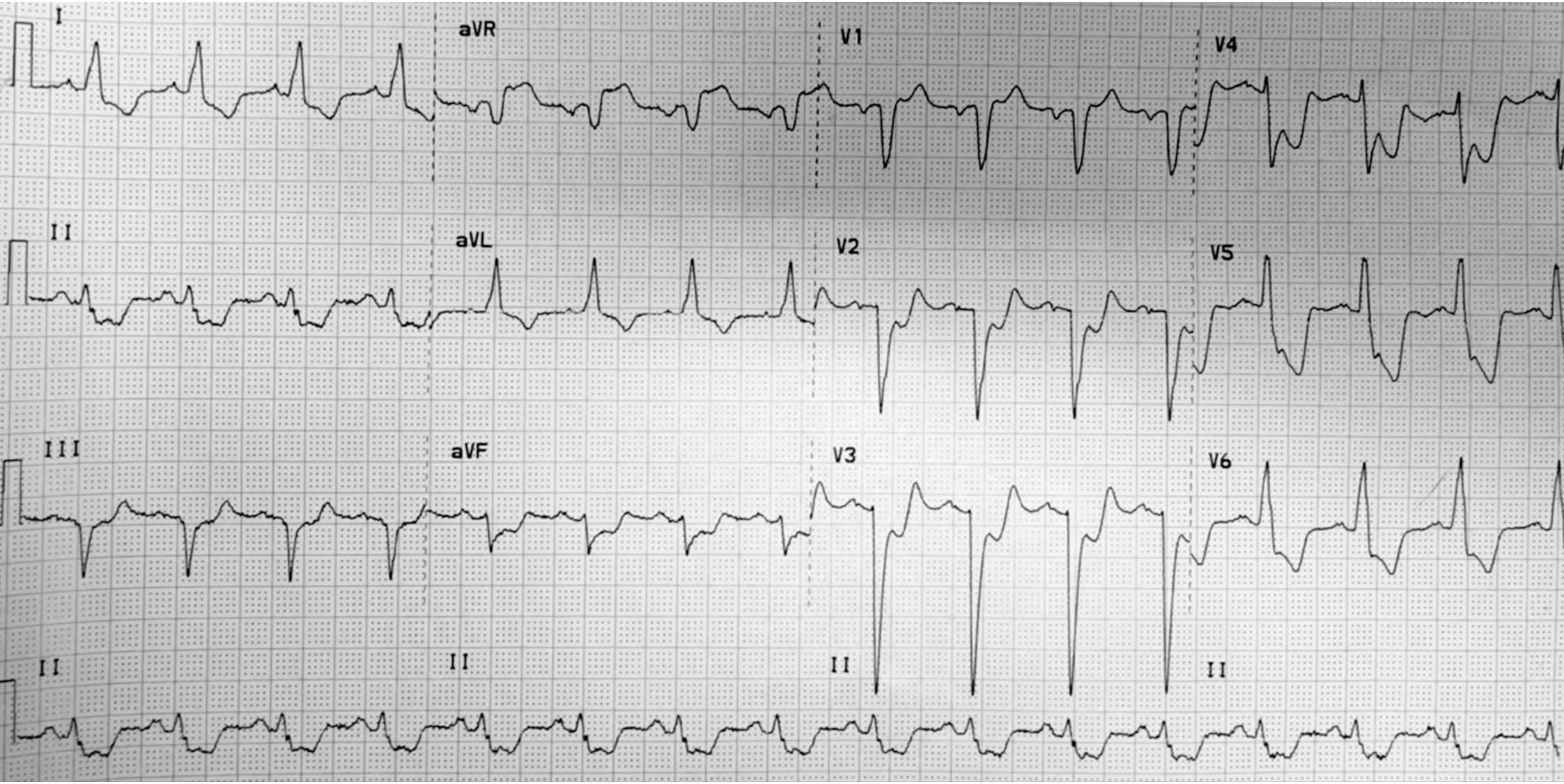
Síndrome coronario agudo en provecto

Buenas tardes Querido Maestro

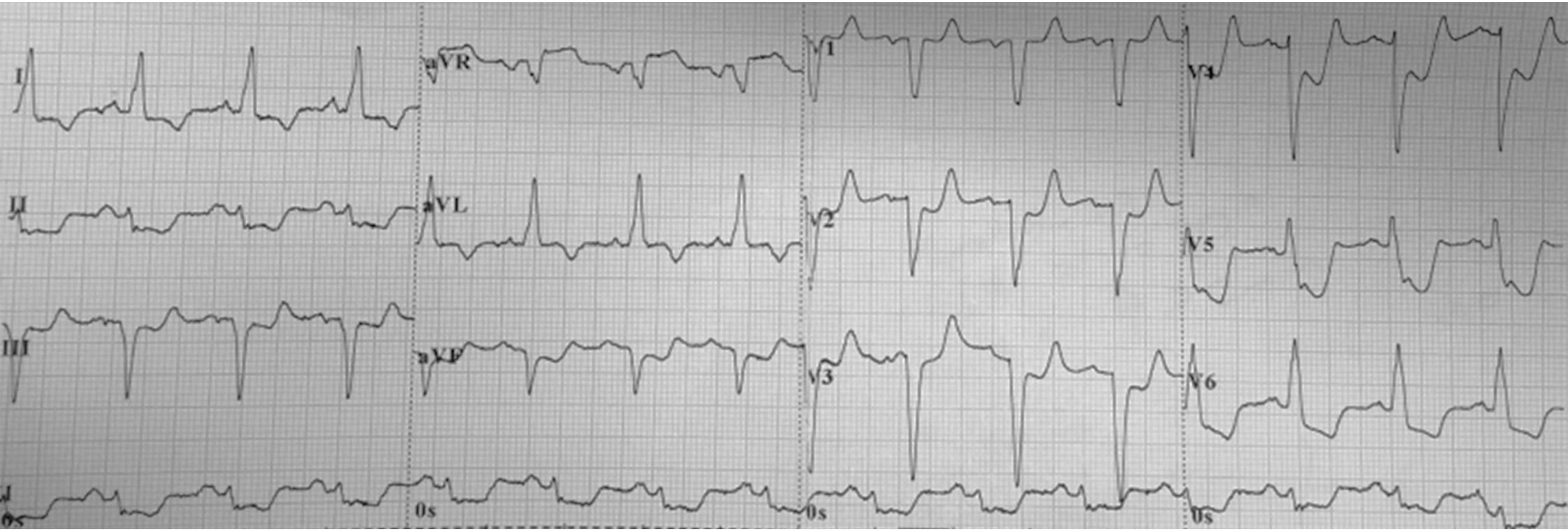
Pongo a su consideración siguiente caso:
Se trata de paciente masculino de 74 años
Que ingresa a cuarto de urgencias con queja de dolor cardiaco
Con antecedentes:
Antecedentes de infarto agudo de miocardio IAM hace 8 años.
Angor pectoris e insuficiencia cardiaca secundarios a IM
Tabaquismo de larga data, actualmente suspendido
EVC isquémico sin secuelas
Y tratamiento no especificado.
Con historia de 3 días de angora de esfuerzo que no sede a medicación, haciéndose los episodios de angor de mayor duración y frecuencia hasta llegar al reposo, para iniciar una noche previo a ingreso con dolor centro torácico intenso, sin síntomas agregados, que no cede al reposo ni a medicación.
Ingresa a sala con SV: TA 90/60, FC: 95, Sat 98% sin 02
CPK 453, CPK MB 91, TGO 108, DHL 616
ECGs anexados: ECG-1 es el de ingreso y ECG 2 es 6 hrs posteriores

El paciente es referido a hospital con laboratorio de hemodinámica.
Espero sea de interés
gracias
Jesus Campuzano
Urgenciologo

ECG-1- at admission



ECG-6h later (Jan 26th 6:48A.M., 2016)



Colleagues opinion

Queridos amigos.(Dear friends)

This ECG pattern has been defined by Samuel Sclarovsky as circumferential subendocardial ischemia. It was the topic of my doctoral dissertation. We have published papers dealing with this ECG change and found that it is associated with severe coronary artery disease: left main, left main equivalent or severe 3-vessel disease. We have published one case (2002) with acute total obstruction of the left main without collateral flow from the RCA caused by aortic dissection. The ECG pattern consists of wide-spread ST depression maximally in V4-V5 with inverted T waves in the leads with maximal ST depression and ST elevation in aVR. The ST may be elevated in V1, but always less than in aVR. It is probably caused by global ischemia of the subendocardium of the left ventricle with high diastolic pressure (in this patient PTF in V1). LBBB and cardiogenic shock may follow. We consider these patients as emergent cases and treat them according to the STEMI protocol (emergent angiography)(except no ADP inhibitor if the patient needs bypass surgery).

Saludos cordials(Best regards)

Kjell Nikus M.D. Ph.D.

Tampere, Finland

I agree with Dr Nikus. This was considered "subendocardial or ST depression" AMI in the classification of Bertolasi used in Argentina. Left main or left main equivalent disease. Mortality upwards of 50%. Unfortunately, the current classification of STEMI vs non-STEMI does not capture these subtleties.

Sergio Pinski MD
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Hola foristas,
Bonito y complicado caso para médicos de urgencias. Cardiopatía isquémica crónica (trastorno de conducción rama izquierda). La onda T positiva de V1-V3 podría ser especular de la cara inferolateral (faltan las derivaciones posteriores). Esta onda T positiva en V1 a V3 también puede ser debida a isquemia subendocardica de cara anterior por suboclusión de la DA. En cualquier caso es un ECG compatible con enfermedad multivasos (descenso global de ST con ST de aVR > V1). El infarto antiguo puede haber sido de circunfleja con disfunción de musculo papilar y insuficiencia mitral que ha determinado la insuficiencia cardíaca.
Lo mas interesante del caso es la imagen de ST-T de V1 a V3
Saludos

Dr Miguel Fiol Sala MD.PhD Servicio de Medicina Intensiva y Unidad Coronaria, Hospital Universitario Son Espases, Palma de Mallorca, Baleares, España

Hello forum members,
Nice and complicated case for emergency physicians. Chronic ischemic heart disease (left bundle branch conduction disturbance). The positive T wave in V1-V3 could be mirror image inferolateral (missing V7-V9 leads). This positive T wave in V1 to V3 may also be due to subendocardial ischemia of anterior wall consequence of LAD sub occlusion . In any case it is an ECG compatible with multivessel disease (overall ST segmente depressions with STSE aVR> V1). The old infarction may have been LCX with papillary muscle dysfunction and mitral regurgitation has determined that heart failure.
The most interesting is the image of ST-T V1 to V3
Cheers

Dr. Miguel Fiol Sala MD PhD. Palma de Mallorca Spain
President of ICE 2016. Scientific Director of Palma Institute of Health Research. Hospital Son Espases, Palma, Balearic Islands, Spain.

43rd International Congress on Electrocardiology Palma, Balearic Island, Spain - June, 4-6 2016. He is waiting for us!!!!

Maestro
O Dr Nikus já falou tudo. Estou totalmente de acordo com sua análise. Fica difícil opinar depois dele. Inclusive há algum tempo atrás eu baixei uma cópia da sua tese que aborda exatamente este tema.

Raimundo Barbosa Barros MD Fortaleza Ceará Brasil

Master
Dr Nikus talked all. I'm totally agree with his analysis. It is difficult to express an opinion after him. .Sometime ago I downloaded a copy of his thesis that addresses this issue exactly.

Raimundo Barbosa-Barros MD Fortaleza Ceará Brazil

In USA "Raymond" nick name The fox

Final comments by

Andrés Ricardo Pérez-Riera, MD, PhD.
Post-Graduates Advisor at Design of Studies and Scientific Writing Laboratory
in the ABC Faculty of Medicine - ABC Foundation - Santo André – São Paulo – Brazil
https://ekgvcg.wordpress.com/

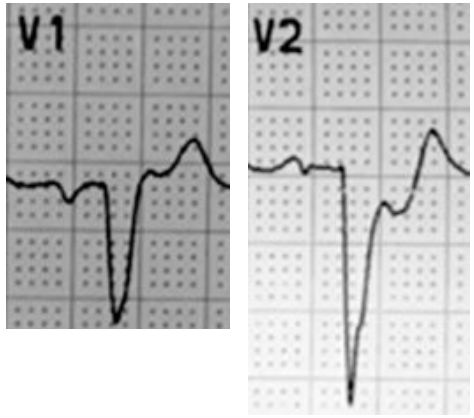
With my "daughter" Evita

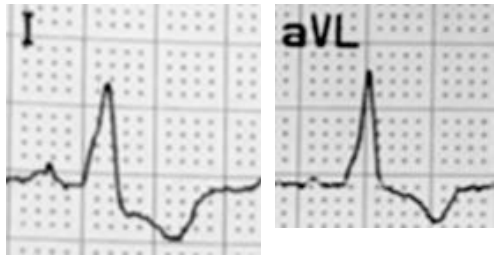
The six degrees of the improvement of physicians devoted to the diagnosis and management of cardiac arrhythmias (**Slama 1973**)

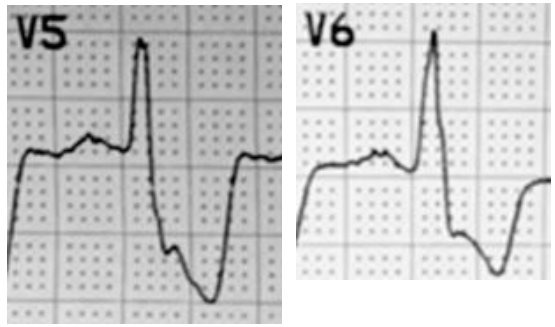
1) Happy stage: he still ignores the problems.
2) The normal stage: he sees there are problems he doesn't understand.
3) The excellent stage: now he believes he understands everything.
4) The depressive stage: he is under the impression he doesn't understanding anything.
5) The stage of self-satisfaction: he doesn't understand anything, but has an explanation for everything.
6) The ideal stage: for which no candidate was ever found: he understands everything and can explain everything. Let's see next slide……

Following the new stricter Straus criteria for "true" LBBB (**Strauss 2011**), it is necessary always after 40ms of the QRS beginning notched/slurred at the apex of R waves in at least two adjacent leads: I-aVL, V1-V2, and V5-V6. This investigator propose stricter criteria for complete LBBB that include a QRS duration ≥ 140 ms for men and ≥ 130 ms for women, along with mid-QRS notching or slurring in ≥ 2 contiguous leads. In contrast, slowly and continuously widened "LBBB like" QRS patterns are mostly occur in LVH or in a metabolic/infiltrative disease (**Préda 2013**). This patient has not a true LBBB. Please see the apex of R wave in left contiguous leads I, aVL, V5-V6 and in right precordial leads V1-V2. These leads have acute apex shape, not "tower". Consequently he has an unspecific intraventricular conduction defect or "non-specific" IVCD with LBBB-like pattern consequence of periinfarction block such us the present case. For this diagnosis is necessary QRS duration > 100ms indicating slowed conduction in the ventricles and criteria for specific bundle branch or fascicular blocks or pre-excitation not met. Causes of nonspecific IVCD's include: ventricular hypertrophy (especially LVH), myocardial infarction (so called periinfarction blocks) such us the present case, drugs effects, especially class IA and IC antiarrhythmics (e.g., quinidine, flecainide), tricycleantidepressant poisoning and hyperkalemia.

LBBB-like pattern consequence of periinfarction block

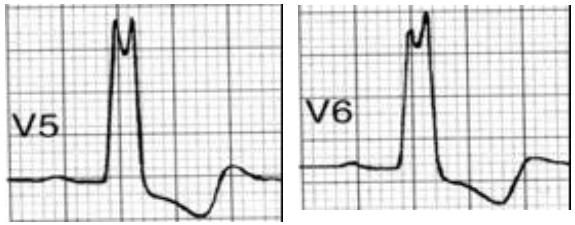






I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

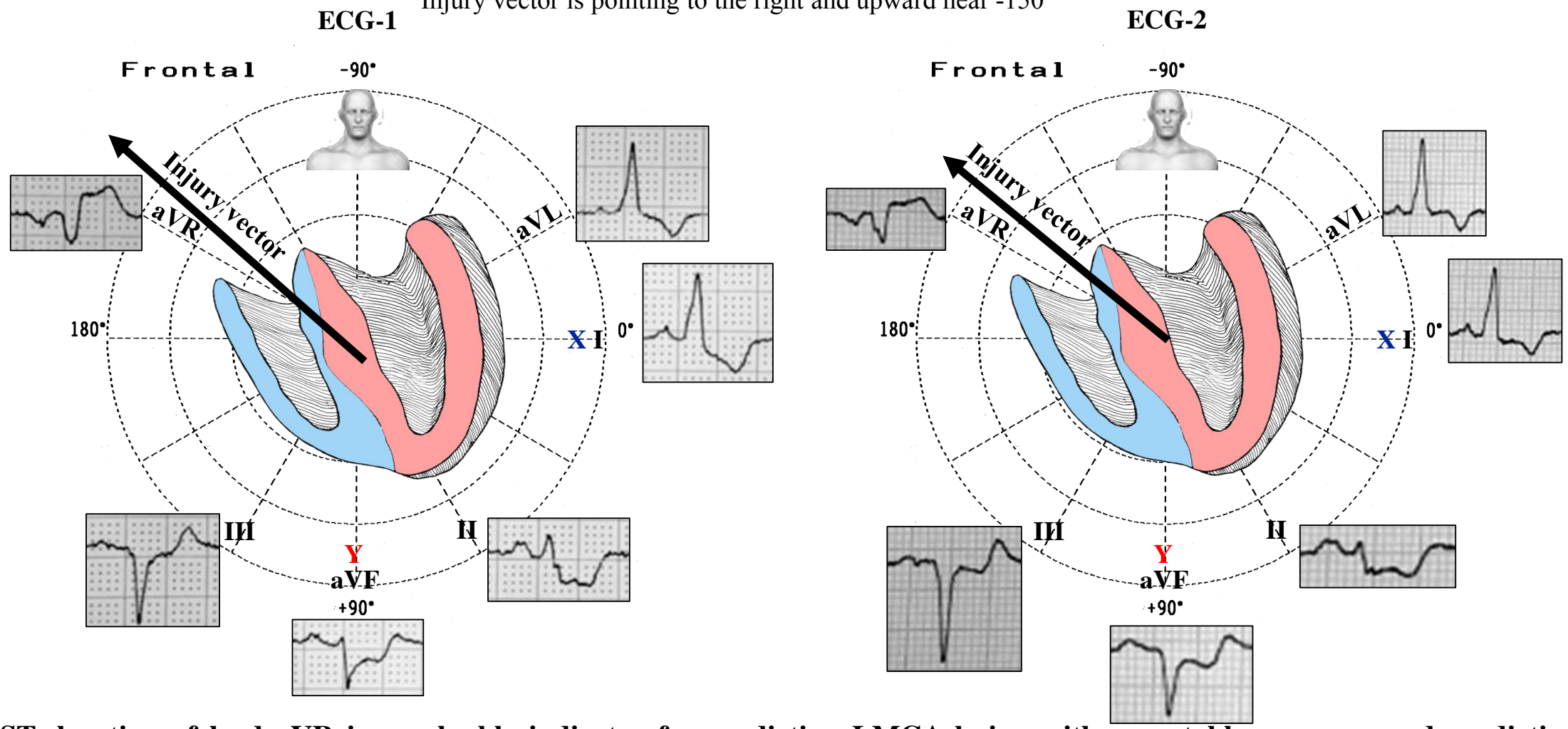
True LBBB



Mid-QRS notching or slurring in ≥ 2 contiguous leads

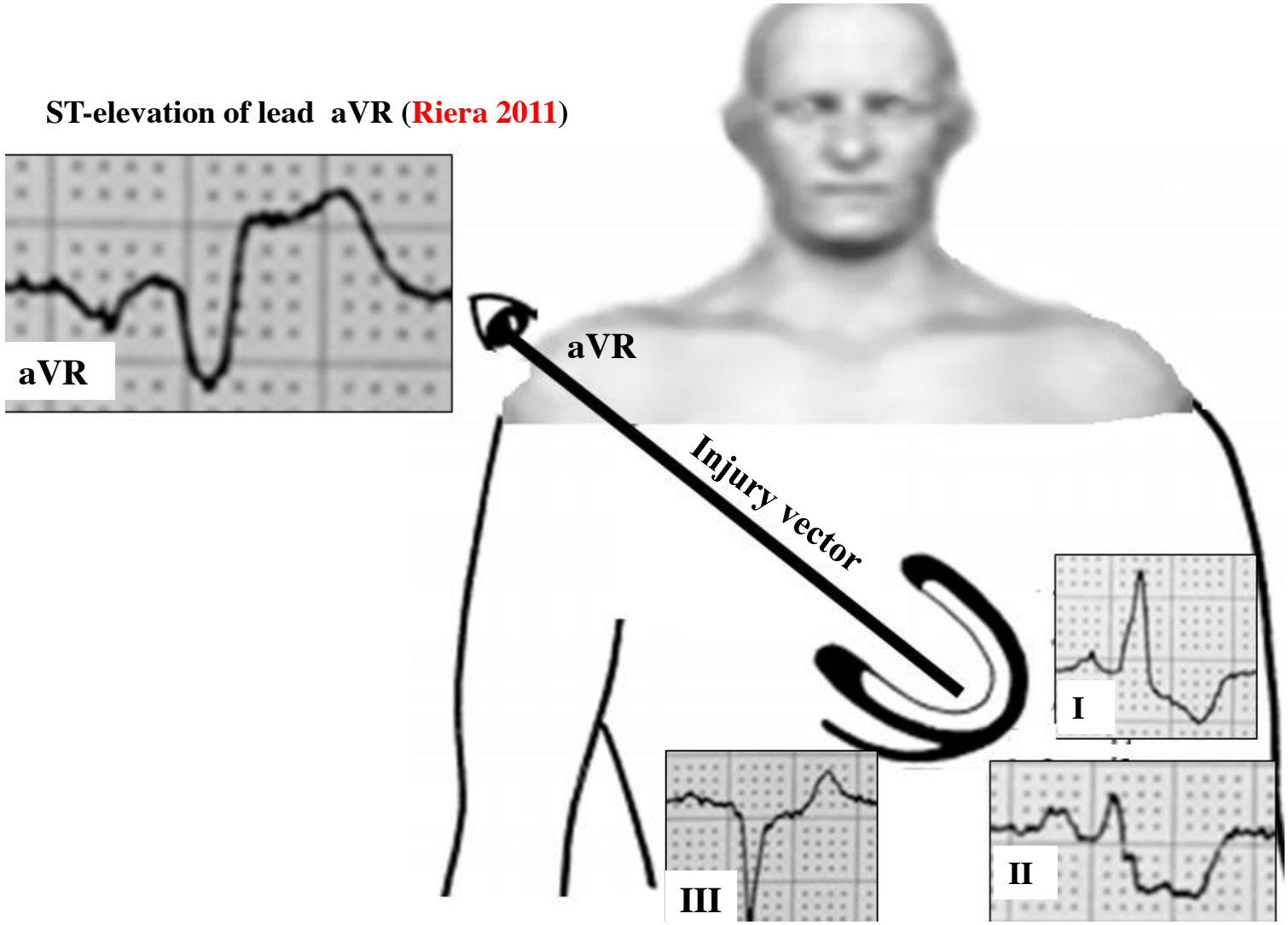
Frontal Plane

Injury vector is pointing to the right and upward near -150º



ST-elevation of lead aVR is a valuable indicator for predicting LMCA lesion with acceptable accuracy and predictive value (Nough 2012). Concomitant ST segment depression in leads II and III with II>III

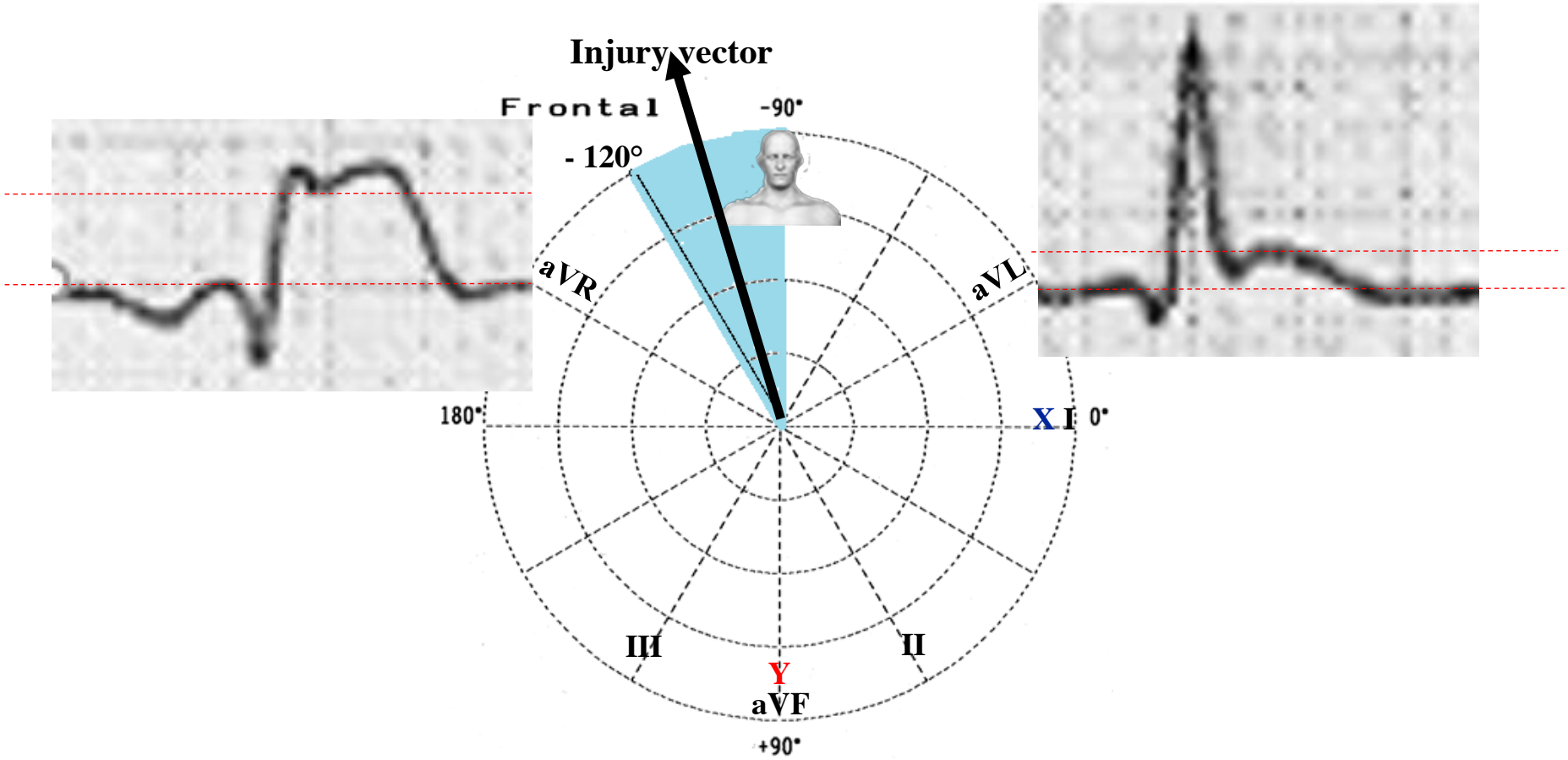
ST-elevation of lead aVR (Riera 2011)



Acute occlusion of the proximal LAD, proximal to the first major septal perforator branch(S1), most frequently presented with STSE in lead aVR, due to the transmural nature of ischemia to the basal septum. Further characterization has demonstrated the presence of inferior ST segment depression during acute LMCA occlusions, independent of the presence of RCA stenosis, as a result of the profound ischemia caused to the basal inferior (ancient dorsal) aspect of the left ventricle.

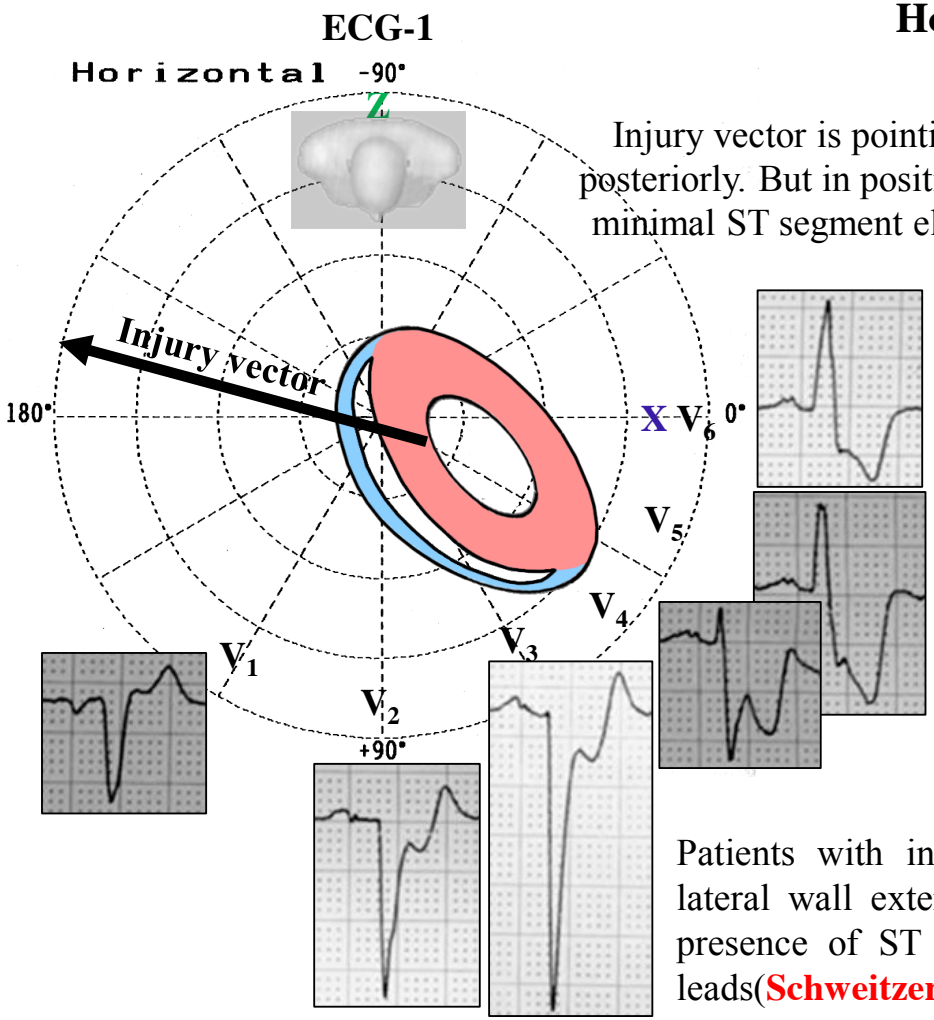
The finding of lead aVR ST segment elevation being greater than or equal to lead V1 ST segment elevation is said to distinguish the LMCA group from the LAD group, with 81% sensitivity, 80% specificity, and 81% accuracy (**Yamaji 2001**). ST segment depression in inferior leads in patients with LMCA occlusion may represent reciprocal changes of the injury current from the basal anterior septum as well as the injury current arising for the basal anterolateral region

Significance of ST segment elevation in both aVR and aVL leads in LMCA obstruction (Hori 2000)

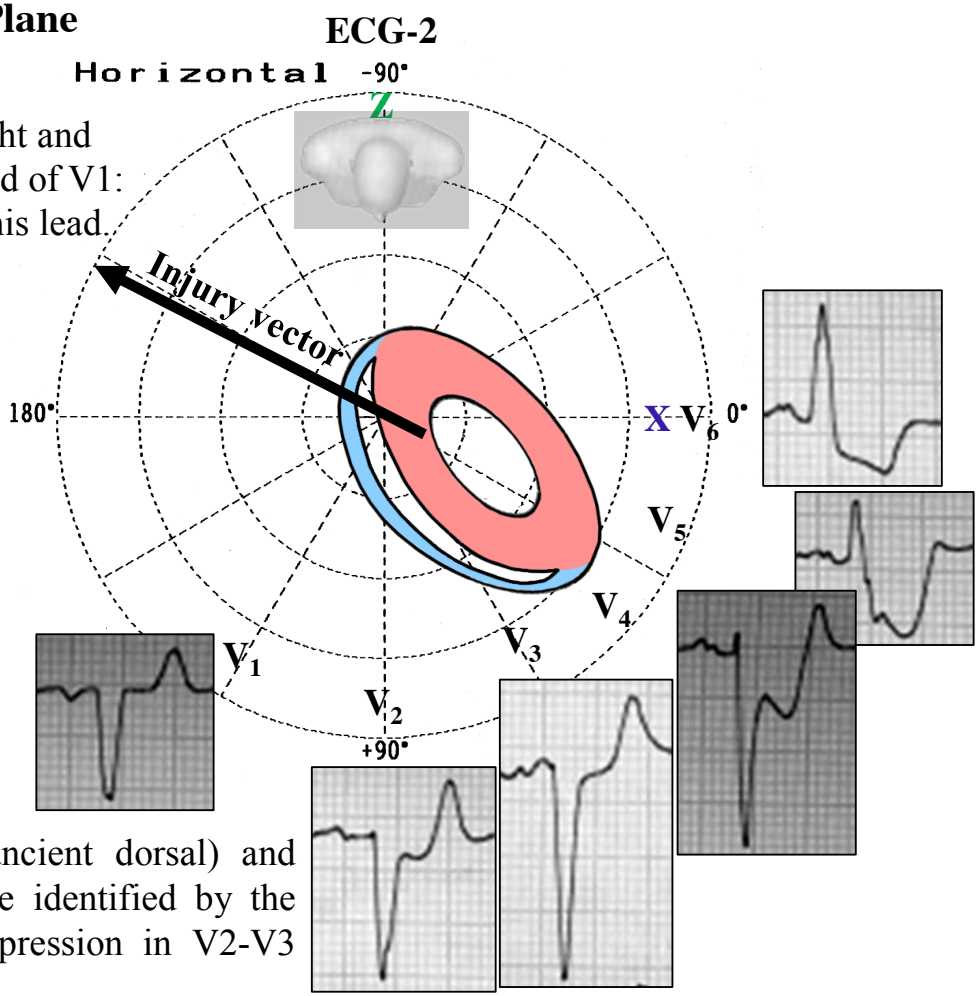


If ST segment elevation is registered in both the aVR and aVL leads in cases of LMCA obstruction it is indicative that injury vector pointed between -90° to - 120°, because in this range the injury vector is located on positive hemifield of both leads aVR and aVL(light blue). The clinical outcome of patients with LMCA-AMI is strongly dependent on the degree of intercoronary collaterals. In electrocardiographic findings, ST segment elevation in both the aVR and aVL leads may reflect widely spreading transmural ischemia, including that of the basal part of the septum. This finding is a useful predictor indicating a poor prognosis.

Horizontal Plane



Injury vector is pointing to the right and posteriorly. But in positive hemi field of V1: minimal ST segment elevation in this lead.

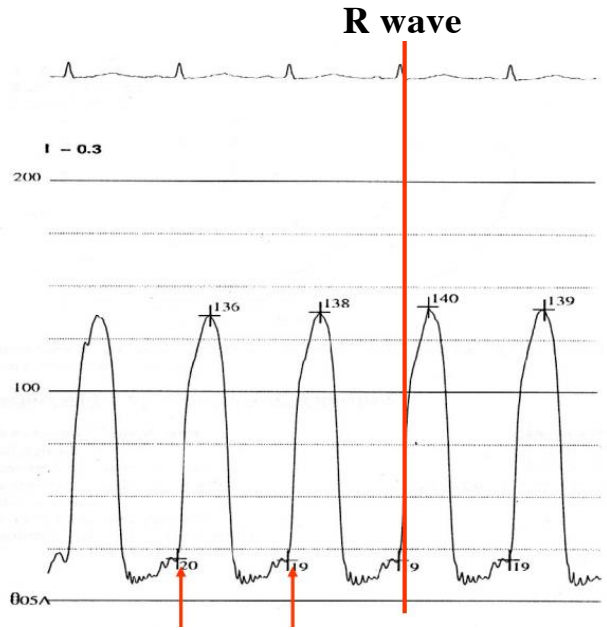


Patients with inferobasal (ancient dorsal) and lateral wall extension can be identified by the presence of ST segment depression in V2-V3 leads(**Schweitzer 2001**).

The tracing shows a pattern compatible with global circumferential subendocardial ischemia. Downsloping ST segment depression is seen in ≥7 leads with negative T waves from V4, V5 and V6. This pattern strongly suggests either ***acute partial obstruction of the left main coronary artery*** **(LMCA)** (**Yamaji 2001; Kosuge 2005; Bayés de Luna 208**) or significant three-vessel involvement, which is considered a left main equivalent (LMEQ). ST segment depression in V6 > ST segment elevation in V1 (**Mahajan 2006**). A transient ST-segment depression and negative T waves maximally in leads V4-5, is associated to LMCA obstruction or severe triple vessel disease (**Nikus 2006**) or others causes (see next slide).

Causes of diffuse transient ST depression in > 7 leads in the inferior and anterolateral leads combined with ST elevation in aVR and V1: "suspect circumferential subendocardial ischemia"

I. Acute subtotal occlusion of the LMCA: ECG manifestation of LMCA stenosis is diffuse ST depression in both the inferior and precordial leads (**Kim 2013**). It was shown in both the experimental laboratory and in clinical studies that a sudden obstruction of a LMCA induces an increase of the left ventricular end-diastolic pressure (LVEDP) without increasing the end diastolic volume, thus shifting the pressure/volume curve upright (**Palacios 1976**). End diastolic pressure can be measured on the R wave of the ECG, which will coincide just after the 'a' wave on the LV trace. This is called the post 'a' wave measurement of EDP. The sudden increase of the end diastolic pressure reduces the subendocardial coronary flow, resulting in a circumferential subendocardial ischemia (**Visner 1985**).



II Significant multi-vessel CAD, "left main equivalent" narrowings or left main equivalent (LMEQ) disease or 3-vessel disease (**Kosuge 2009**).

III Infarction caused by a first diagonal branch occlusion (**Sclarovsky 2002**).

IV Acute neurological disorders (**Kim 2013**).

V Anemia, causing decreased O_2 delivery.

VI Hypoxia, causing decreased O_2 delivery.

VII Sepsis, causing increased O_2 demand.

VIII Severe hypertension, causing increased O_2 demand and decreased subendocardial perfusion.

IX PE, causing decreased O_2 delivery and increased O_2 demand.

X Acute hypertensive pulmonary edema, causing decreased O_2 delivery and increased O_2 demand.

XI Tachycardia, most often AF or SVT, causing increased O_2 demand.

XII Generalized inflammatory states, causing increased O_2 demand and decreased O_2 delivery.

XIII COPD or CHF exacerbation, causing decreased O_2 delivery and increased O_2 demand.

XIV Hypotension, due to decreased O_2 delivery.

Electrocardiographic evidence for partial occlusion of the left main coronary artery

ST segment elevation in aVR (>0.05 mV) and possibly in V1: injury vector is pointing to the right and above the -150º; location of the aVR lead. ST segment elevation in aVR > V1. The greater ST segment elevation in aVR compared to V1 is an important indicator of acute LMC stem occlusion (**Yamaji 2001**) and the amount of ST segment elevation in aVR is an important predictor of adverse outcomes including death (**Barrabes 2003**). ST segment depression in lead II and leads V4 to V6 (inferobasal ischemia with negative T waves from V4 to V6). Consequence of sudden and severe increase of the LVEDP (**Nikus 2002**). ST segment depression in the inferior leads. In the present case the depression is only in lead II and not in lead III because the injury vector is directed from inferior to superior and from the left to right. ST segment depression in V6 > ST segment elevation in V1. Diffuse depression of ST segment in inferolateral leads.

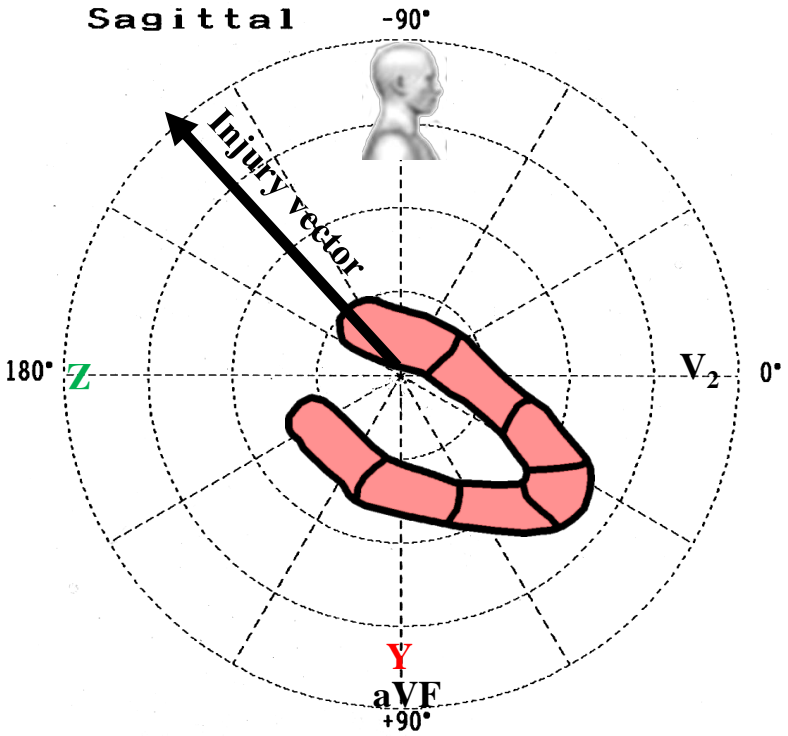
Possible presence of dromotropic disturbance, such as nonspecific IVCD's, complete right bundle branch block pattern, left anterior fascicular block, and/or left septal fascicular block. Diffuse ST segment depression associated with ST segment elevation in aVR is not specific to partial occlusion of the LMC stem but this pattern indicates global circumferential subendocardial ischemia. Multivessel coronary occlusions may also result in this pattern (**Knotts 2013**). ACS cases with total occlusion of the LMCA trunk, unlike those with partial occlusion, present with STSE pattern (STEMI) and not diffuse depression as in this case. These patients generally die before reaching the hospital (**Fiol 2012**) or develop cardiogenic shock (**Kimuran 2012**), acute pulmonary edema, tachyarrhythmia or intraventricular dromotropic disturbances (**Nikus 2007-2008**). Although the surgical management is still the gold standard for these cases, recent investigations have shown that percutaneous coronary intervention with stent implant could be an efficient and safe alternative for carefully selected patients that have good ventricular function and favorable anatomy (**Jean 2012; Park 2011; Capodano 2010**).

The risk-benefit ratio of stenting versus coronary artery bypass graft surgery. These issues are currently being addressed in two seminally important trials including the SYNTAX trial, which randomizes patients with LMCA and/or three-vessel disease to either CABG surgery or a TAXUS drug-eluting stent. CABG remains the standard of care for patients with three-vessel or LMCA disease, since the use of CABG, as compared with PCI, resulted in lower rates of the combined end point of major adverse cardiac or cerebrovascular events at 1 year. (**Serruys 2009**). The other trial is the COMBAT trial, which is focused exclusively on LMCA stenosis and randomizes patients with LMCA disease either to a Sirolimus-eluting stent (Cypher, Johnson and Johnson Cordis USA) or to CABG surgery.

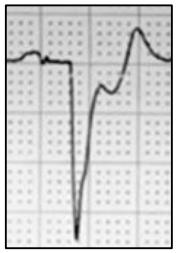
Indications for PCI Favorable for stenting low-risk patients, with good LV function, non-distal and non-calcified LM stenosis, ostial LM lesions and mid-shaft LM lesions, and very few additional lesions on the other coronary vessel (low or intermediate SYNTAX score). These patients have been shown to have excellent outcomes following LM stenting. Patients with STEMI, LM acute occlusion during catheterization, and shock. In these cases, PCI is a fast way to recanalize the LM but clinical outcome is poorer compared with stable patients (**Fajadet 2012**).

Right Sagittal Plane

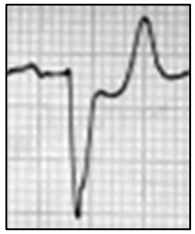
Injury vector is pointing to back and upward: ST segment depression in aVF and V2.



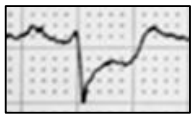
ECG-1



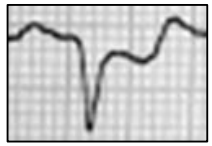
ECG-2



ECG-1



ECG-2



Typical pattern of LMCA occlusion

ST segment depression in V6 > ST segment elevation in V1 (**Mahajan 2006**).
ST segment depression in II and III
ST segment depression II > III
ST segment depression in I and aVL. ST segment elevation has poor prognosis (**Hori 2000**).
ST segment elevation in aVR > V1 because the ST vector lesion pointing to aVR An ST vector directed between 90° and 180° in the frontal plane is observed in 100% of patients with an LMCA obstruction. The specificity of this observation is 78% (**Prieto Solís 2008**).
Widespread horizontal ST depression, most prominent in leads I, II and V4-6: ≥7 leads, known as circumferential subendocardial ischemia
ST elevation in aVR ≥ 1mm
ST elevation in aVR ≥ V1. However, ST elevation in aVR is not entirely specific to LMCA occlusion. It may also be seen with:
Proximal left anterior descending artery (LAD) occlusion
Severe triple-vessel disease (3VD)
Diffuse subendocardial ischaemia – e.g. due to O_2 supply/demand mismatch, following resuscitation from cardiac arrest
NB. Some authors argue that using the term "LMCA occlusion" is inaccurate, as most of these patients have at least some flow in their LMCA (i.e. incomplete LMCA occlusion); whereas a complete LMCA occlusion would rapidly lead to STEMI, cardiogenic shock and death. ST elevation is aVR is therefore postulated to result from two possible mechanisms:
Diffuse subendocardial ischaemia, with ST depression in the lateral leads producing reciprocal change in aVR (= most likely).
Infarction of the basal septum, i.e. a STEMI involving aVR
In the context of widespread ST depression + symptoms of myocardial ischemia
STE in aVR ≥ 1mm indicates proximal LAD / LMCA occlusion or severe 3VD
STE in aVR ≥ 1mm predicts the need for CABG
STE in aVR ≥ V1 differentiates LMCA from proximal LAD occlusion
Absence of ST elevation in aVR almost entirely excludes a significant LMCA lesion

In the context of anterior STEMI

STE in aVR ≥ 1mm is highly specific for LAD occlusion proximal to the first septal branch (S_1)

Left main Coronary Artery occlusion ECG criteria

In patients undergoing exercise stress testing

STE of ≥ 1mm in aVR during exercise stress testing predicts LMCA or ostial LAD stenosis (**Uthamalingam 2011**)

Magnitude of ST elevation in aVR is correlated with mortality in patients with ACSs:

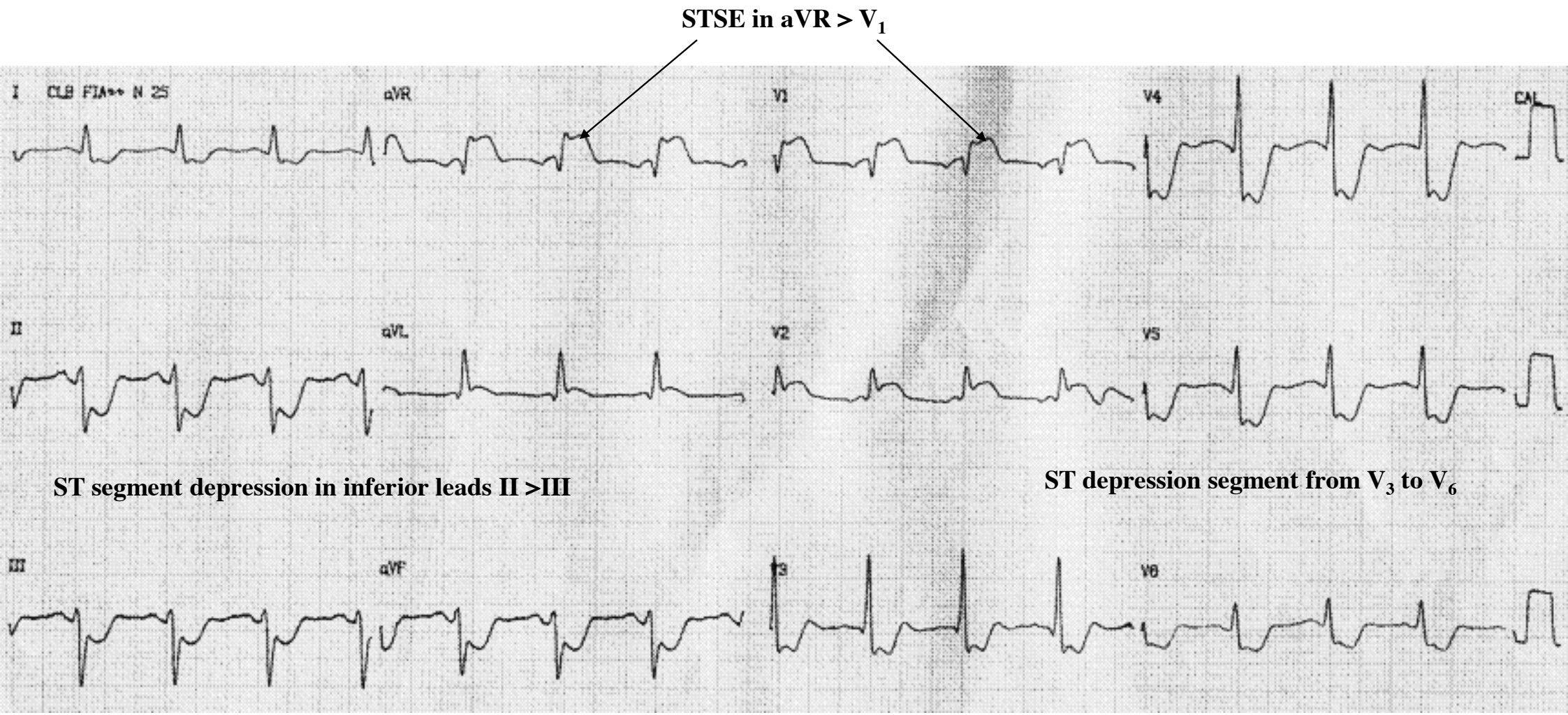
I. STE in aVR ≥ 0.5mm was associated with a 4-fold increase in mortality

II. STE in aVR ≥ 1mm was associated with a 6- to 7-fold increase in mortality

III. STE in aVR ≥ 1.5mm has been associated with mortalities ranging from 20-75%

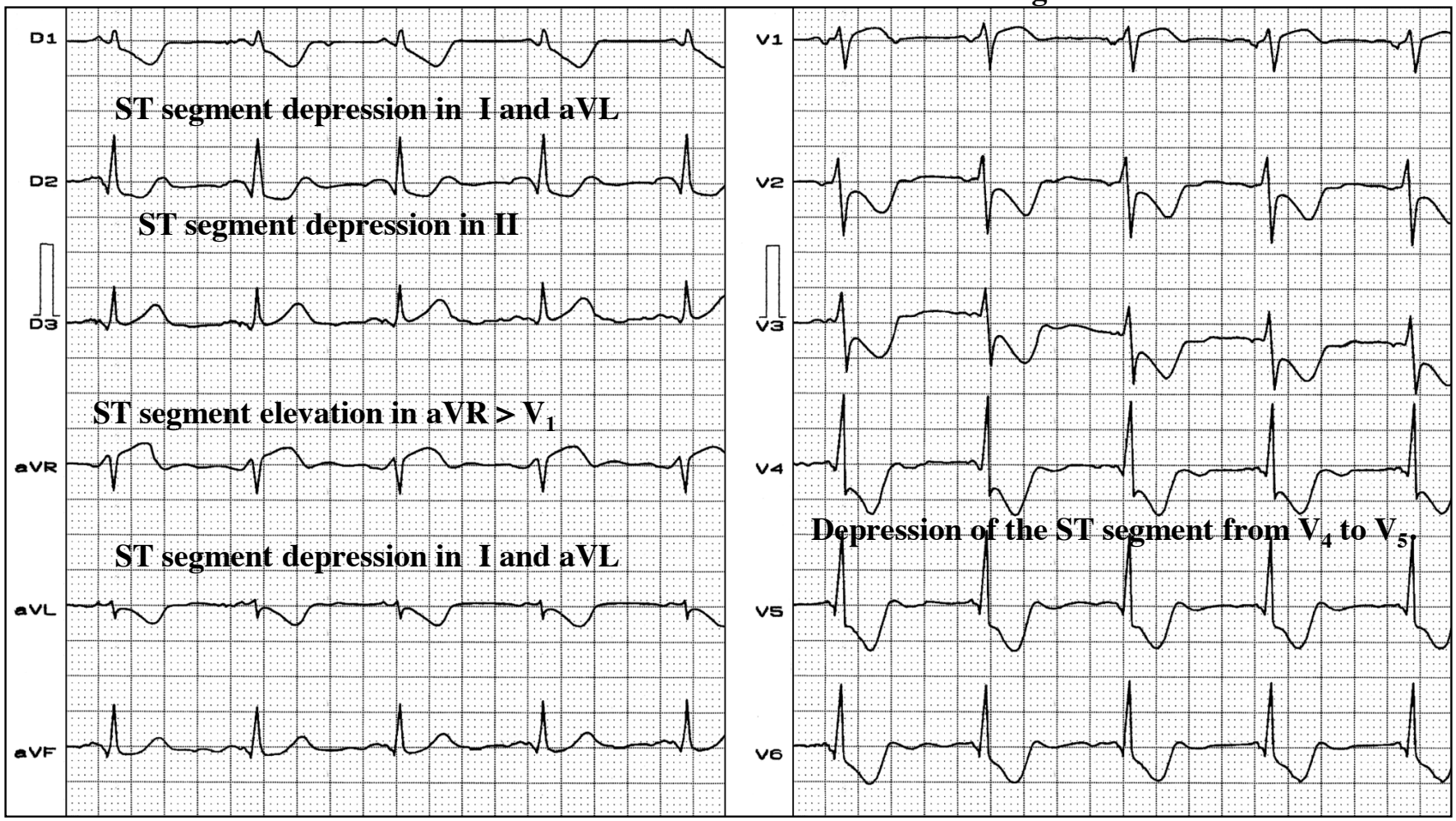
Electrocardiographic examples of Left Main Coronary Artery Oclussion

By Raimundo **Barbosa-Barros** & Andrés Ricardo **Pérez-Riera**



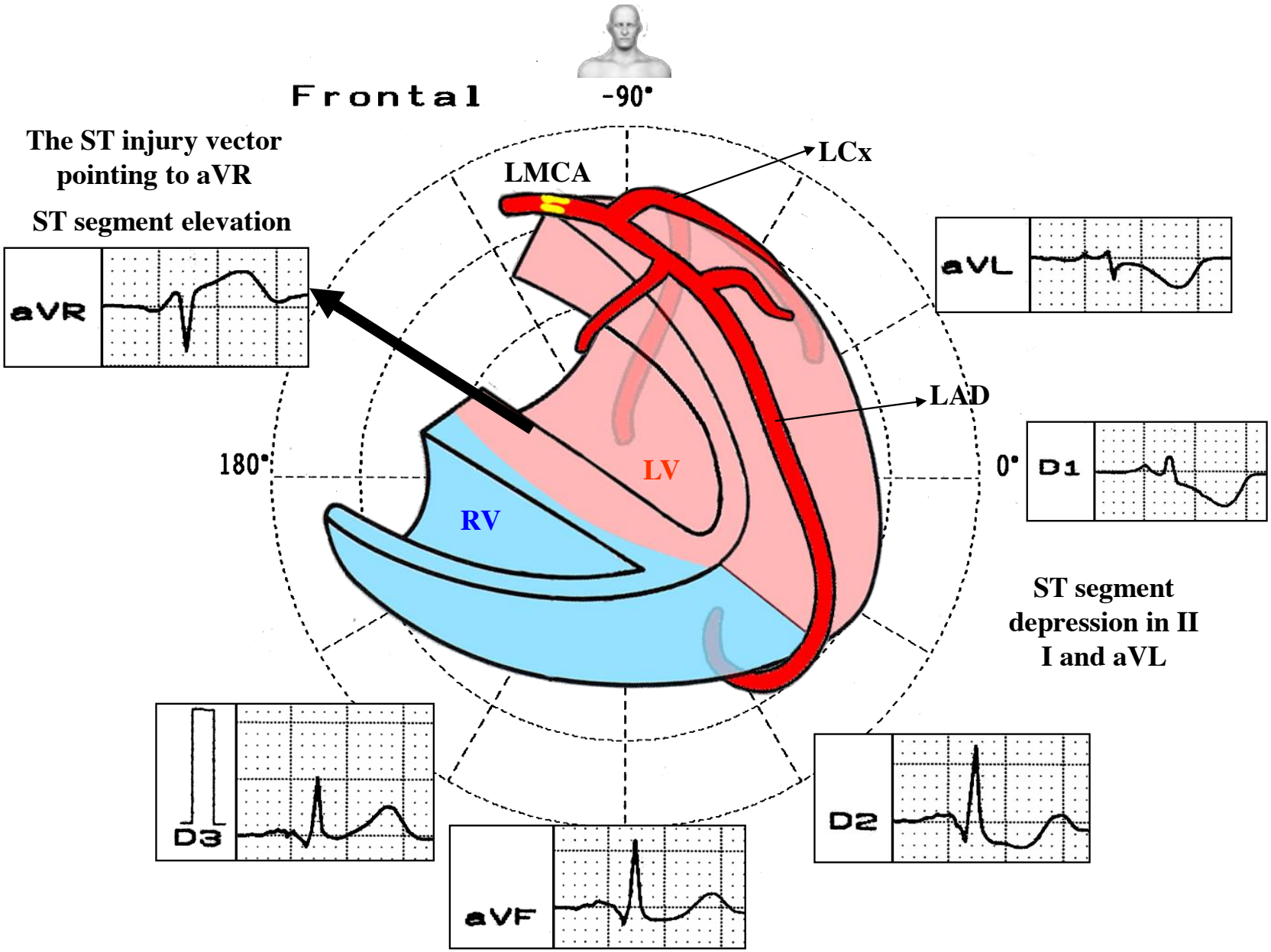
Clinical Picture: Acute Coronary Syndrome associated with cardiogenic shock (Killip class IV) consequence of total occlusion of LMCA. Primary Angioplasty was performed, with immediately hemodynamic stabilization.

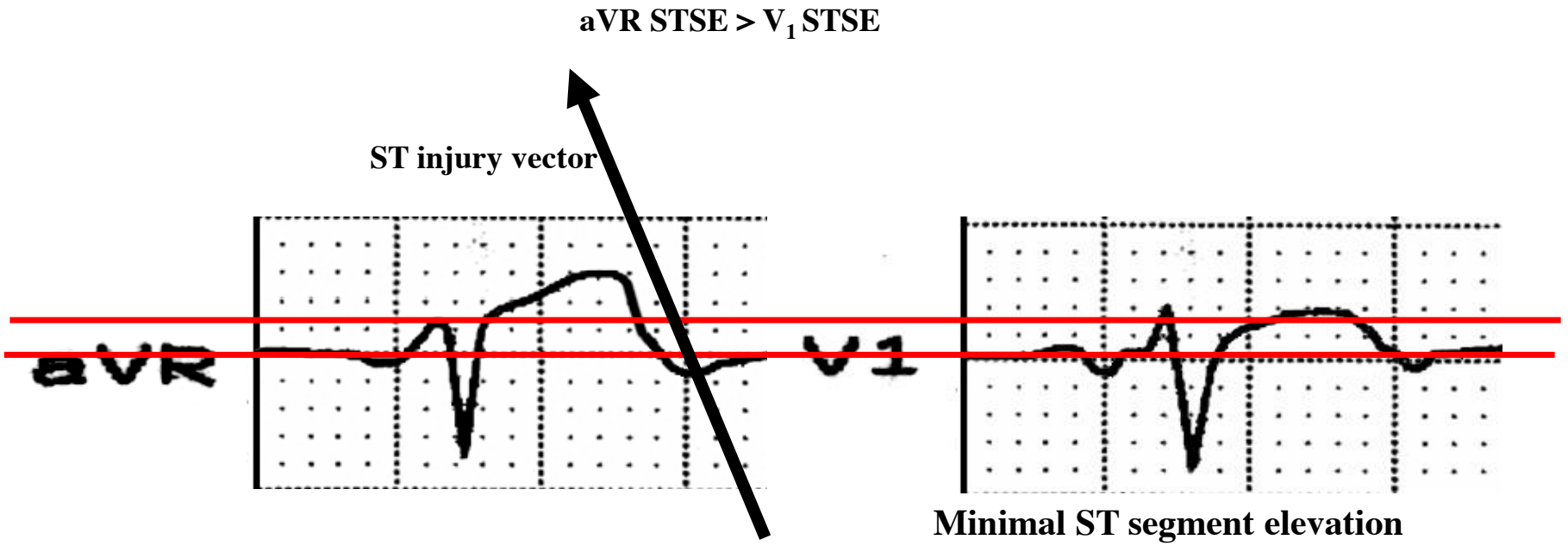
Typical ECG pattern of LMCA occlusion
Diffuse ST segment depression in the inferolateral leads



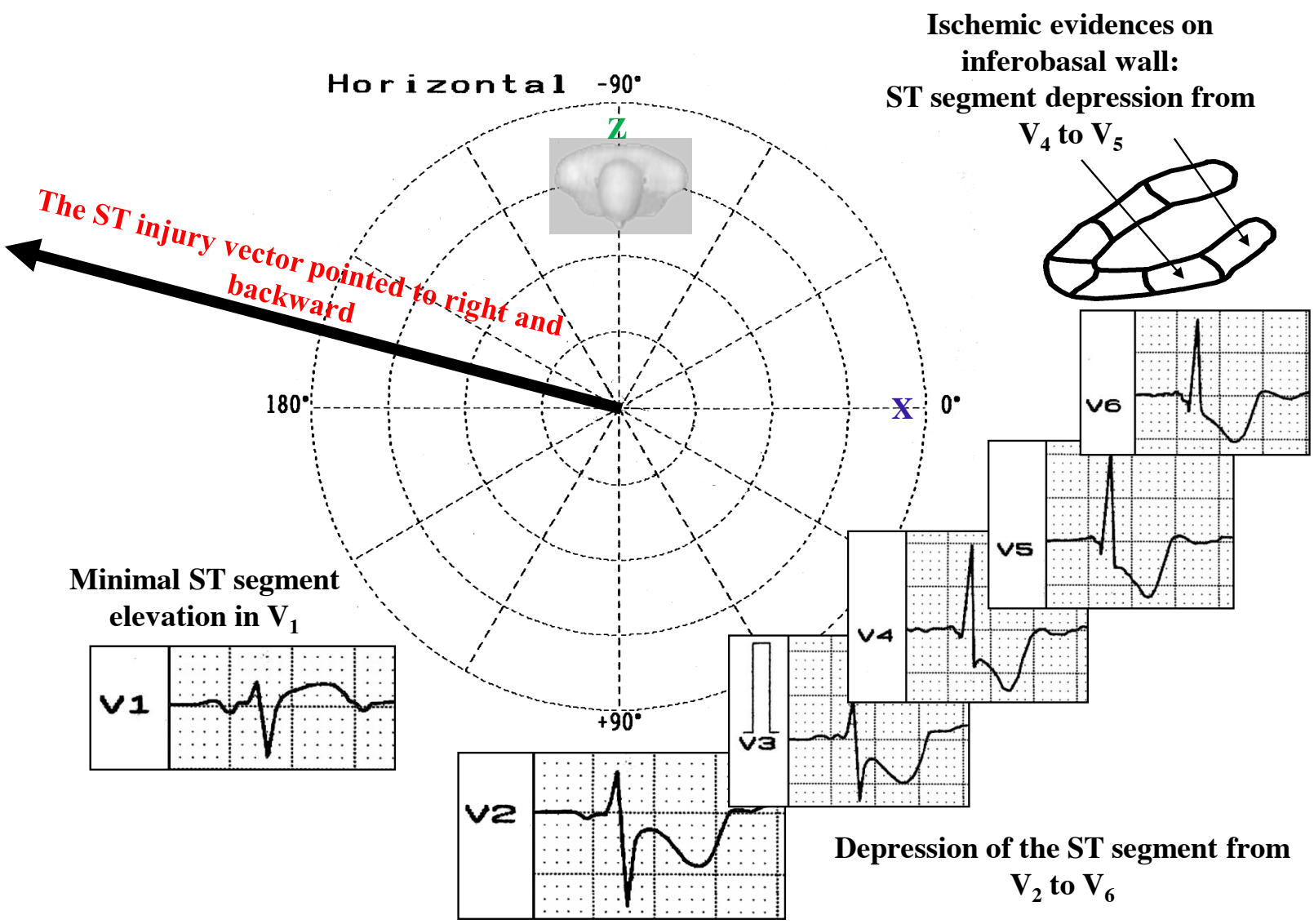
Why this pattern is observed? See next slide sequence.

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


Frontal
-90°
The ST injury vector pointing to aVR
ST segment elevation
LMCA
LCx
LAD
aVR
aVL
180°
0°
LV
RV
D1
ST segment depression in II I and aVL
D3
D2
aVF

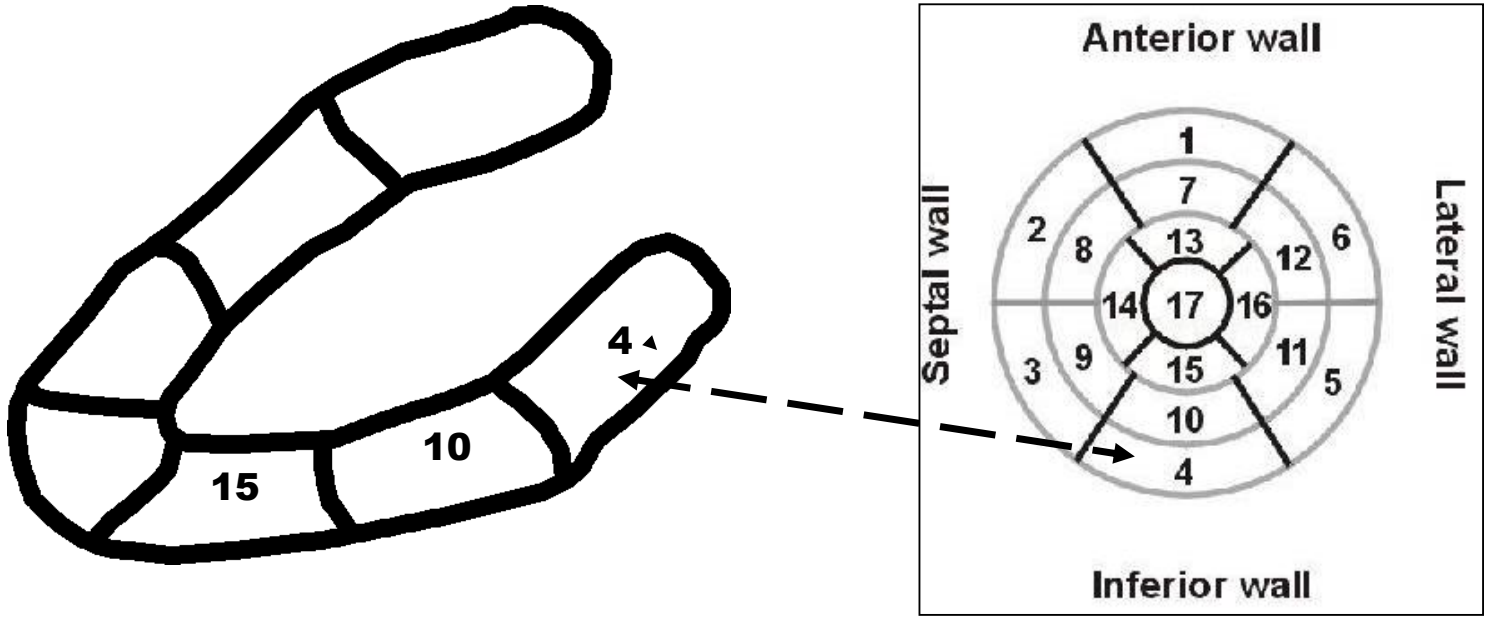


ST segment elevation in aVR > V_1. Why?
Because the ST injury vector is directed to upward and rightward, pointing to aVR lead (RVOT)


Horizontal
-90°
Z
180°
X
0°
+90°
The ST injury vector pointed to right and backward
Ischemic evidences on inferobasal wall:
ST segment depression from V_4 to V_5
Minimal ST segment elevation in V_1
V1
V2
V3
V4
V5
V6
Depression of the ST segment from V_2 to V_6

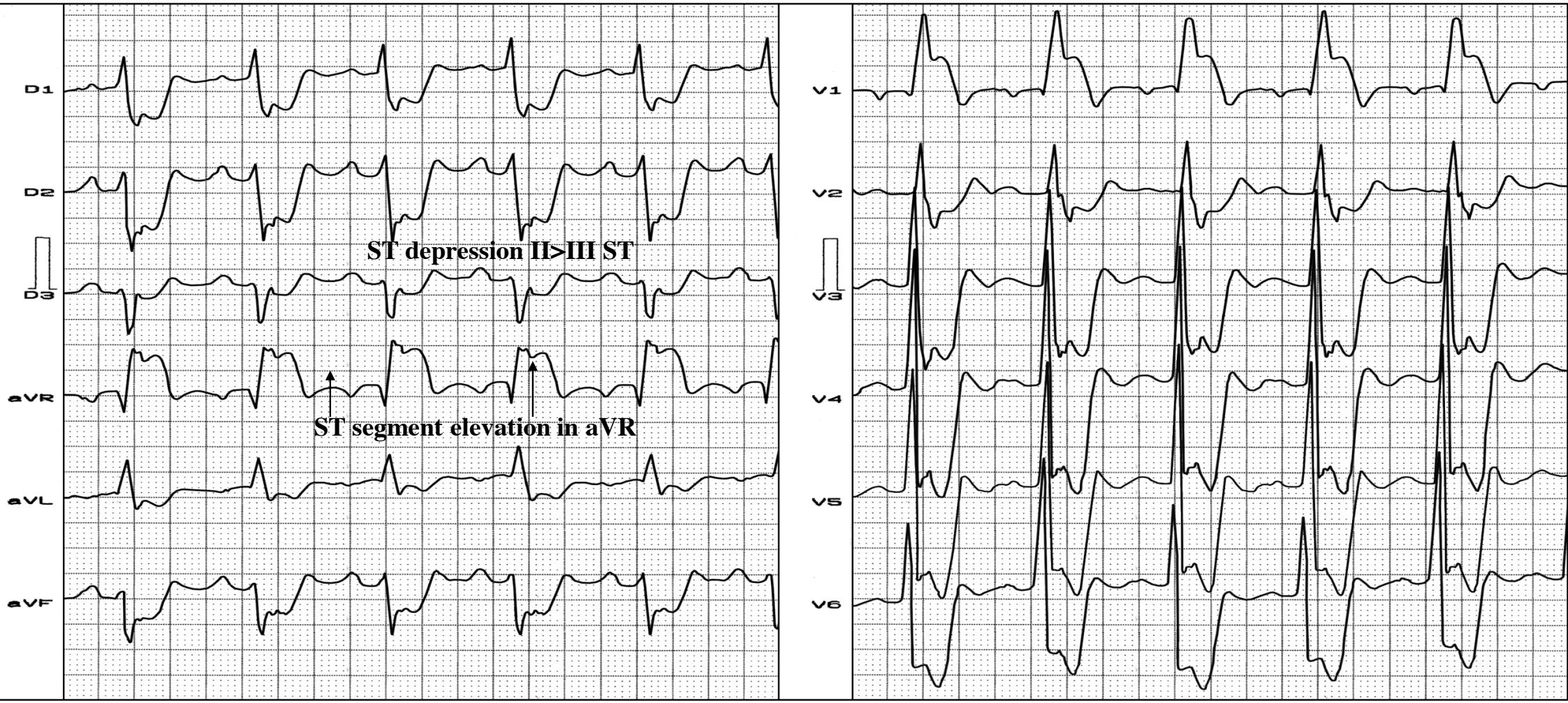
LMCA occlusion ECG criteria

- 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 a new right bundle branch block (RBBB) , LAFB and/or LSFB or unspecific IVCD. A new right bundle branch block (RBBB) pattern, especially when associated with ST elevation in aVR and V1, should raise suspicion of this diagnosis (**Ionescu 2009: Bitigen 2006**).

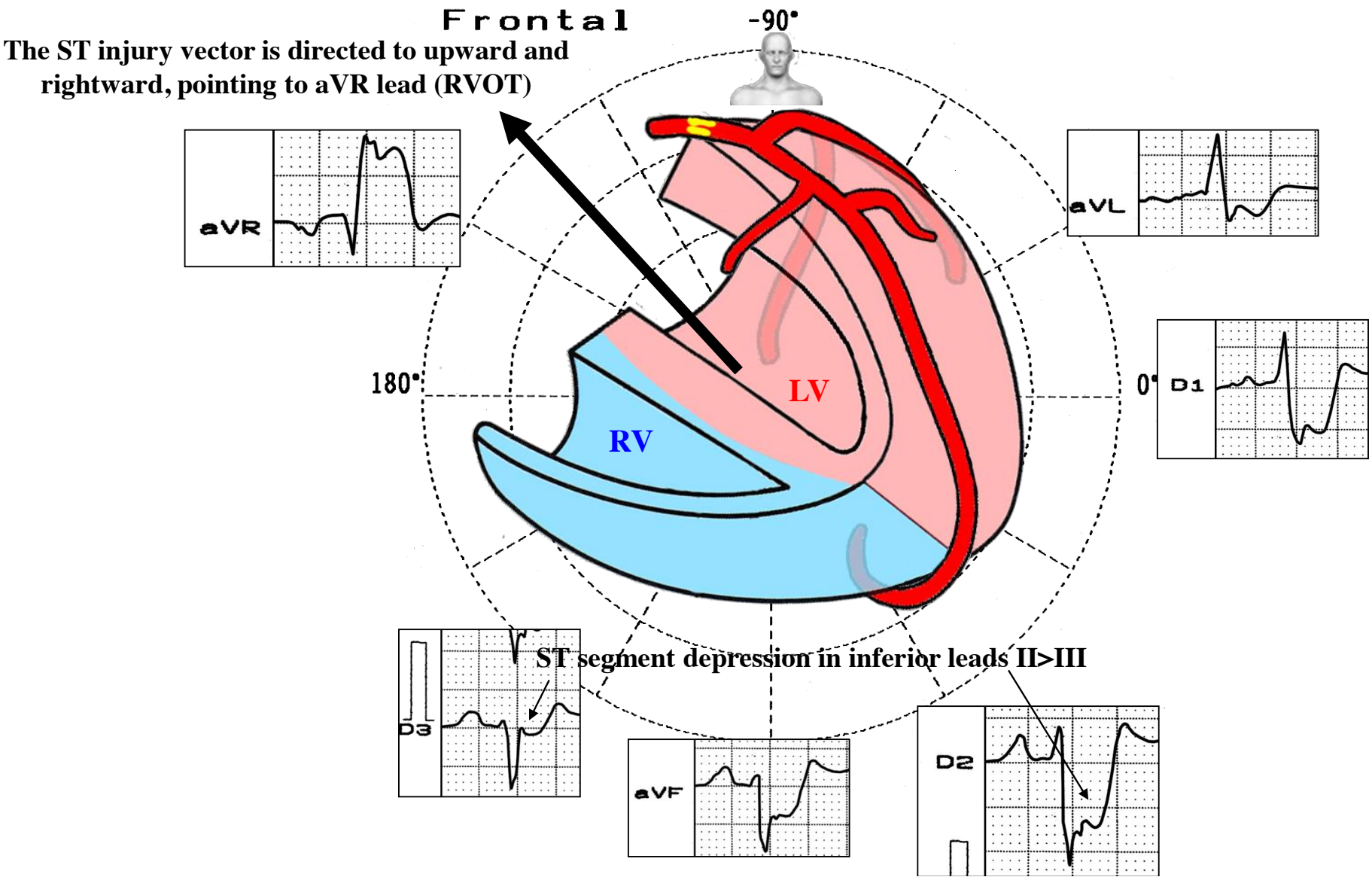


*** Formally called inferobasal wall**

LMCA Occlusion complicated with Complete RBBB

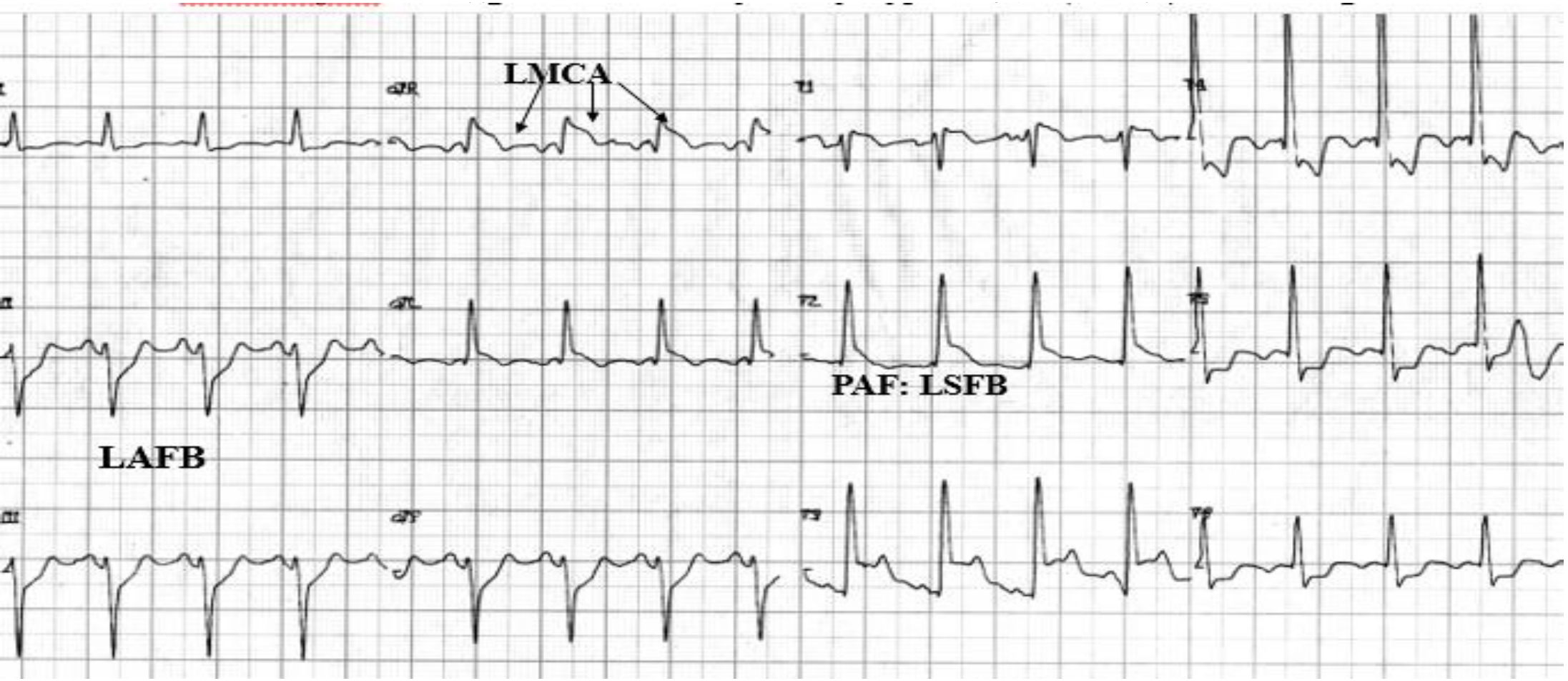


ST SE in aVR and V_1 (aVR > V_1.). ST depression II>III ST depression segment from V_2 to V_6.


Frontal
-90°
The ST injury vector is directed to upward and rightward, pointing to aVR lead (RVOT)
aVR
aVL
180°
0°
D1
LV
RV
D3
ST segment depression in inferior leads II>III
aVF
D2

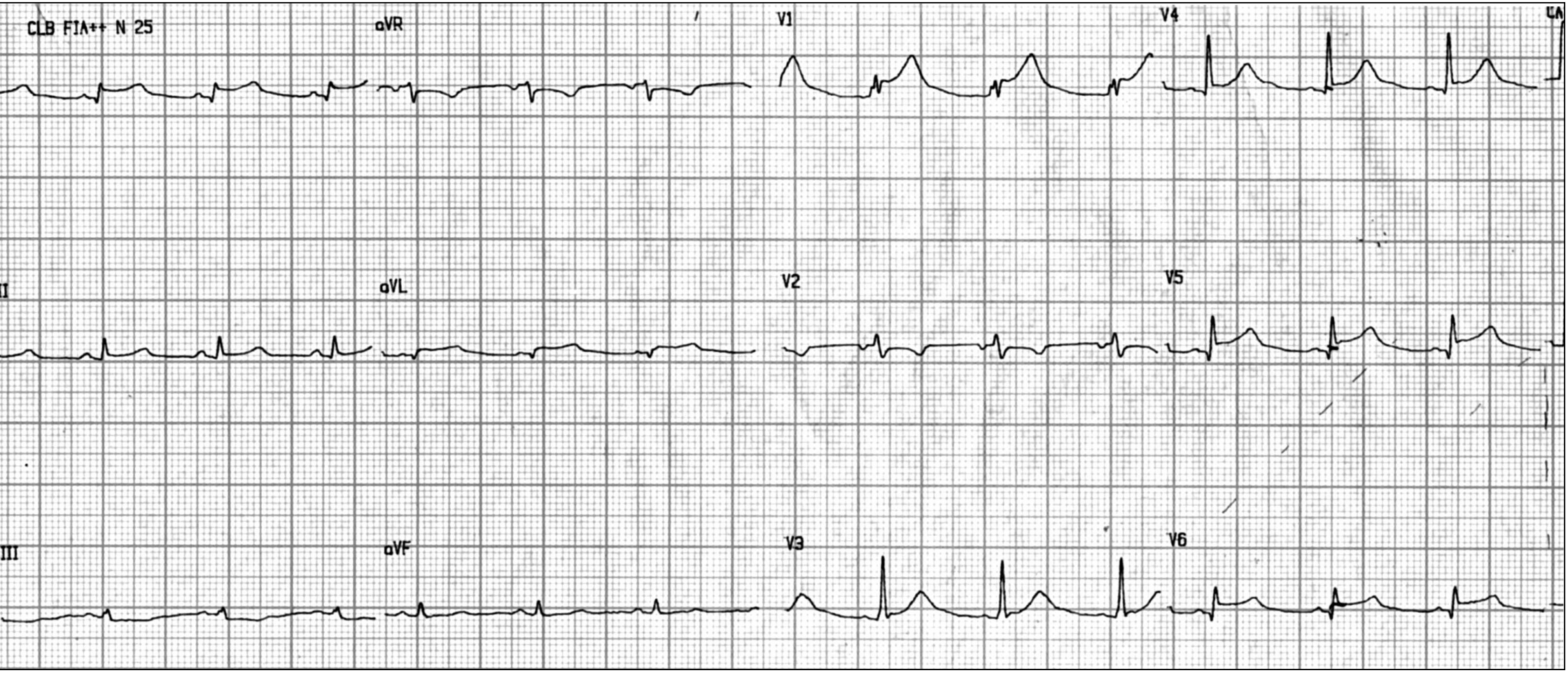
Left Main Coronary Artery occlusion complicated with transient Left bifascicular block: Left Anterior Fascicular Block and Left Septal Fascicular Block

Name: AR.; **Date:** 02/01/2009.; **Age:** 72 yo.; **Gender:** Male.; **Ethnic Group:** Caucasian **Weight:** 72 Kg.; **Height:** 1.74 m; **Biotype:**

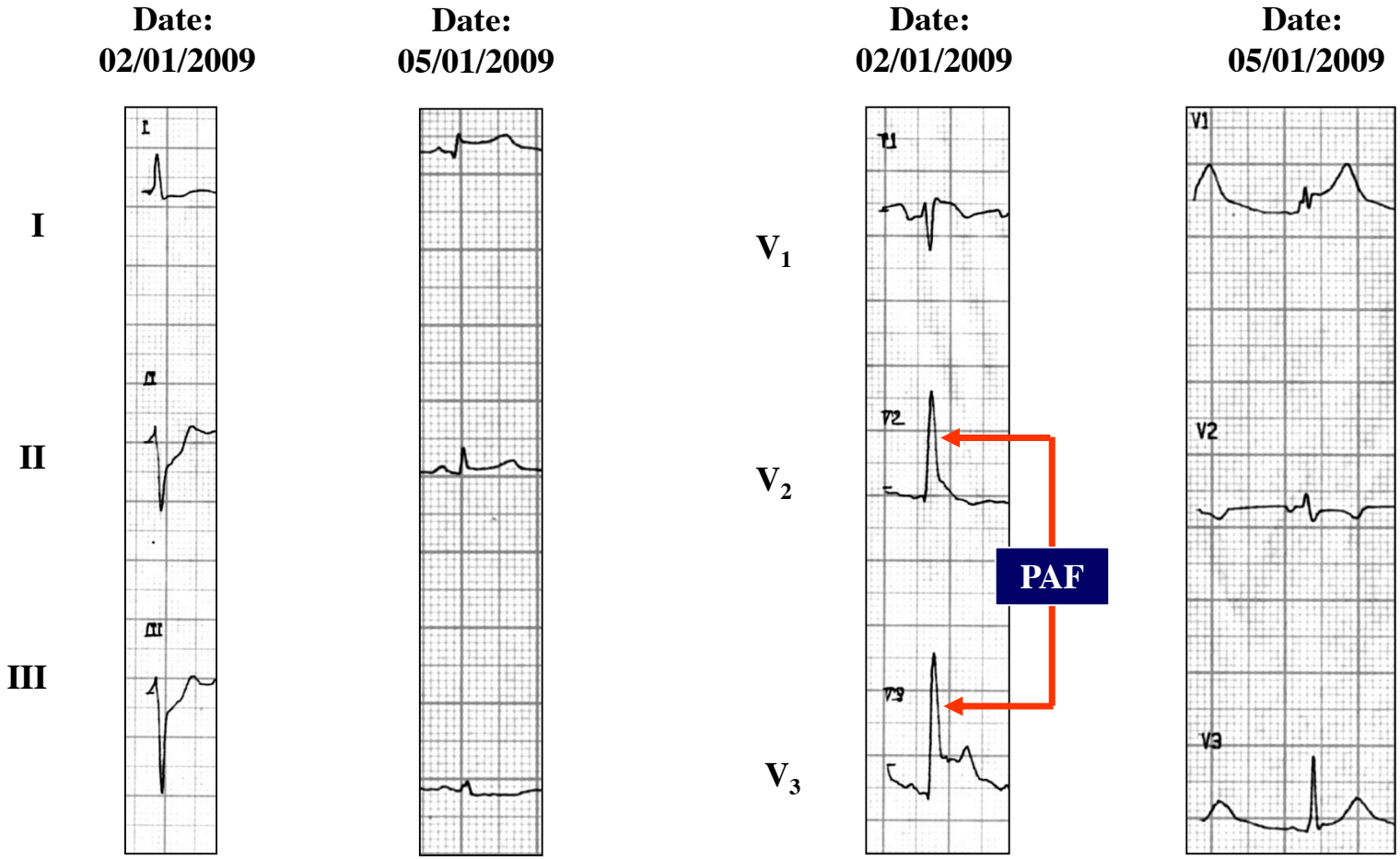


Clinical features: ACS: 72-year-old male patient, admitted in the emergency room with typical precordial pain that yielded after the administration of IV nitroglycerin.
ECG diagnosis: 1) LAFB + 2) LSFB: PAF + Injury block + aVR lead with ST segment elevation suggestive of obstruction in the LMCA. **Laboratory:** 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.



PAF: Prominent Anterior Forces

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