Sessão Coronariana
Coronary Session

Apresentador/ Presenter: Dr. Mário Alves – R2 da Cardiologia
Orientador/ Adviser: Dr. Daniel

14/12/2011
December 14th, 2011

Portuguese

- Id: masculino, 27 anos, natural e procedente de Fortaleza/Ceará Brasil, pedreiro
- HDA Relata dor precordial em aperto, sem irradiação, associado a náuseas/vômitos e sudorese fria, durante trabalho
- HPP /personal history: uso de ilícitos (parou há 03 anos?)/
- Ao Exame
  - EGR, FC: 65, PA: 140x100mmHg/normal overall condition, HR: 65, BP: 140x100mmHg
  - AC: RCR, 2T, sem sopros
  - AP: MVU
  - Sem edema em MMII

Após medidas iniciais, inclusive nitrato, o paciente apresentou hipotensão arterial enquanto era encaminhado para sala de hemodinâmica.
Trata-se de uma sindrome coronariana aguda com supradesnivelamanto do segmento ST em paciente jovem,
usuário de droga.
Na minha análise o ECG exibia alterações compatíveis com oclusão aguda da coronaria direita.
Para minha surpresa a coronariografia mostrou: CD hipoplásica, Cx normal, Tronco de coronária esquerda normal e oclusão total com imagem de trombo na porção distal da artéria DA.

Pergunta: Como se poderia explicar este fenômeno?
Desculpe pela má qualidade dos traçados depois mando os originais.

Raimundo Barbosa-Barros MD

Male, 27 years old, born and from Fortaleza/Ceará, Brazil, bricklayer
QP/main complaint: chest pain started 4 hs ago.
HDA/history of current disease: he mentions oppressive precordial pain, with no irradiation, associated to nausea and vomiting, and cold sweat during work.
/use of illicit drugs (he stopped 3 years ago?)
in examination:

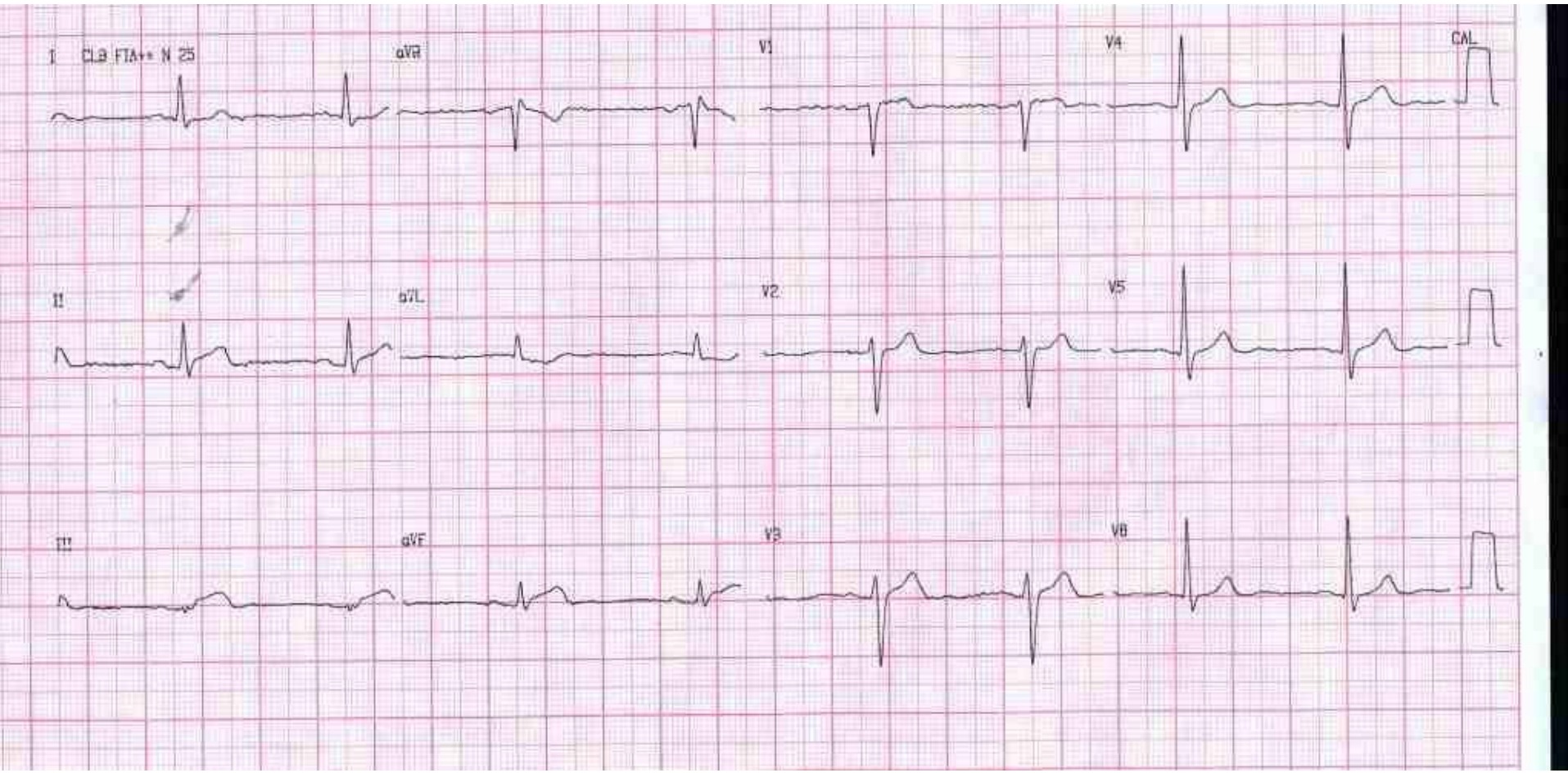
EGR, FC: 65, PA: 140x100mmHg/normal overall condition, HR: 65, BP: 140x100mmHg
AC: /stroke: CPR in two tempos, without murmurs
AP: MVU/Pulmonary auscultation: vesicular murmurs
no edema in inferior limbs.

After the initial measures, even nitrate, the patient presented hypotension while he was taken to the hemodynamic lab.
This is acute coronary syndrome with ST segment elevation in young patient, who uses drugs.
In my analysis of the ECG, he displayed alterations compatible with acute occlusion of the RCA. To my surprise, coronary angiography showed: hypoplastic RCA, normal LCx, normal left coronary artery trunk (LMCA) and total occlusion with image of thrombus in the distal portion of the anterior descending artery (LAD).

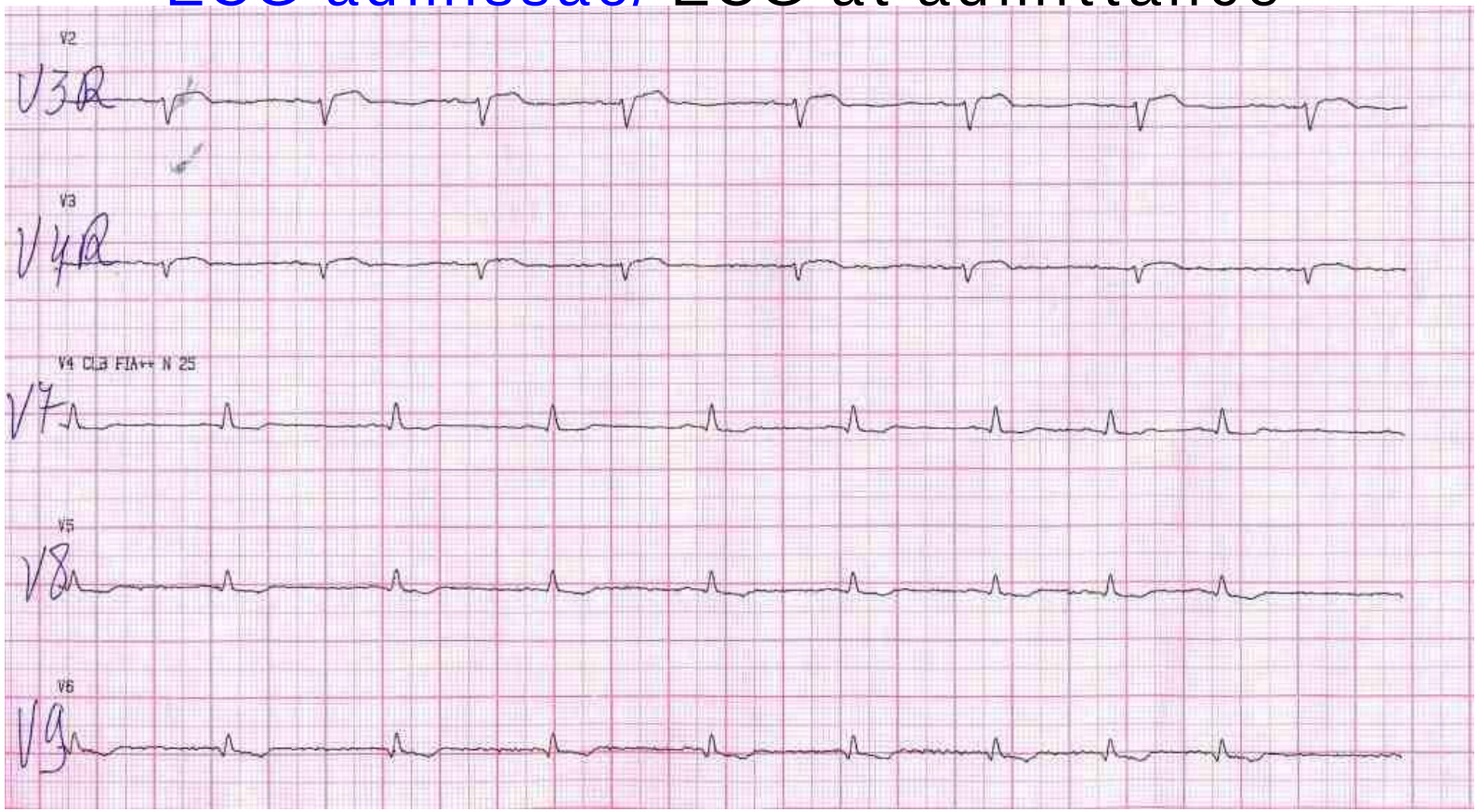
Question: How can we explain this phenomenon?
I apologize for the poor quality of the tracings. Later I will send the originals.

Raimundo Barbosa-Barros MD

ECG admissão/ ECG at admittance



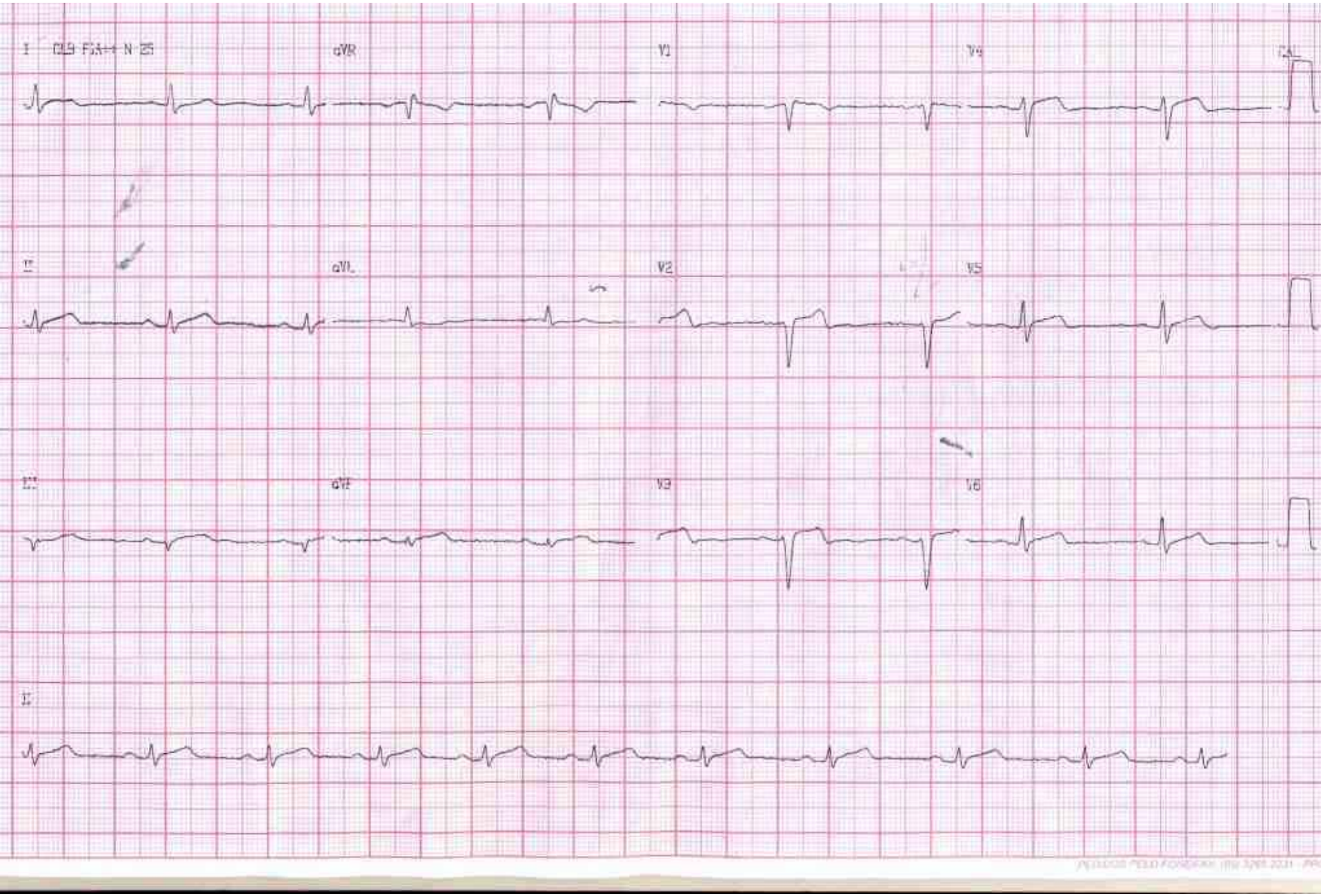
ECG admissão/ ECG at admittance



12-lead ECGs provide insufficient information for the accurate diagnosis of posterior and/or right ventricular AMI in patients with acute chest pain. Right-sided precordial leads (V3R-V5R) and/or posterior chest leads (V7-V9) provide important information from those specific areas, but these additional ECGs are not routinely recorded because of the time-consuming procedure involved. Synthesized posterior and right-sided precordial lead ECGs appear to be highly reliable and useful in the rapid diagnosis of AMI, especially in the early detection of posterior and/or right ventricular involvement, thereby alleviating patient distress.(1)

1. **Katoh T, Ueno A, Tanaka K, et al. Clinical significance of synthesized posterior/right-sided chest lead electrocardiograms in patients with acute chest pain.J Nihon Med Sch. 2011;78:22-29.**

ECG após ATC primária + Stent/ ECG after primary PTCA + stent



Left Main or left coronary artery (LCA)

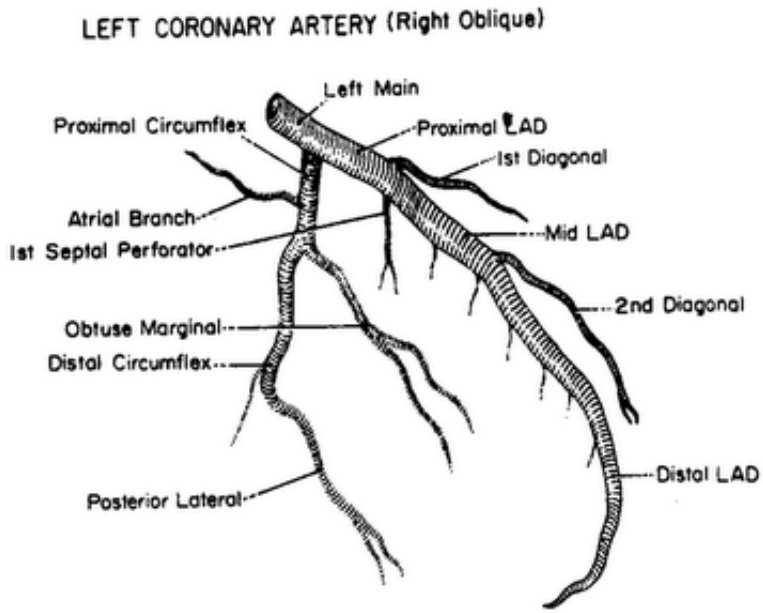
First Dg1

2nd Diagonal

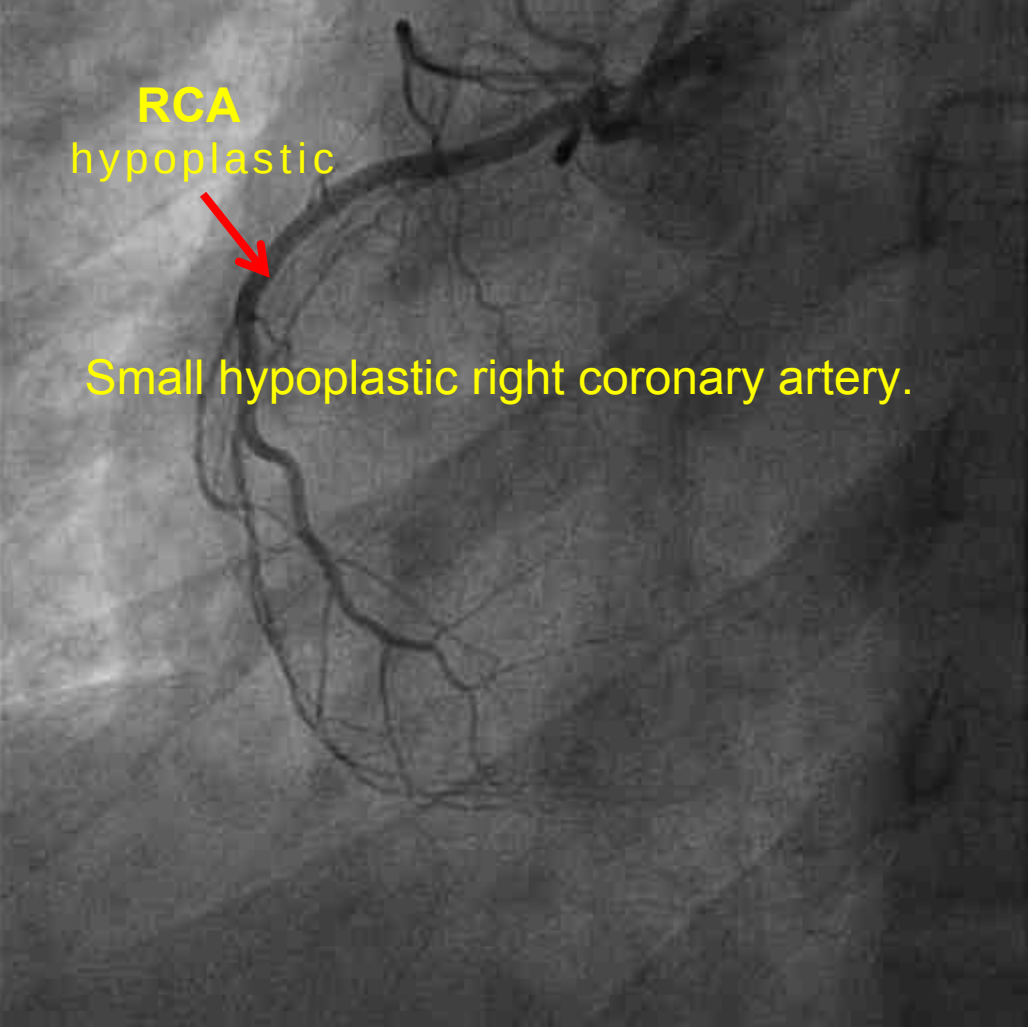
LCX

LAD total occlusion with thrombus

Obtuse marginal



Anatomy of the left coronary tree in the right oblique view.



Coronary artery dominance

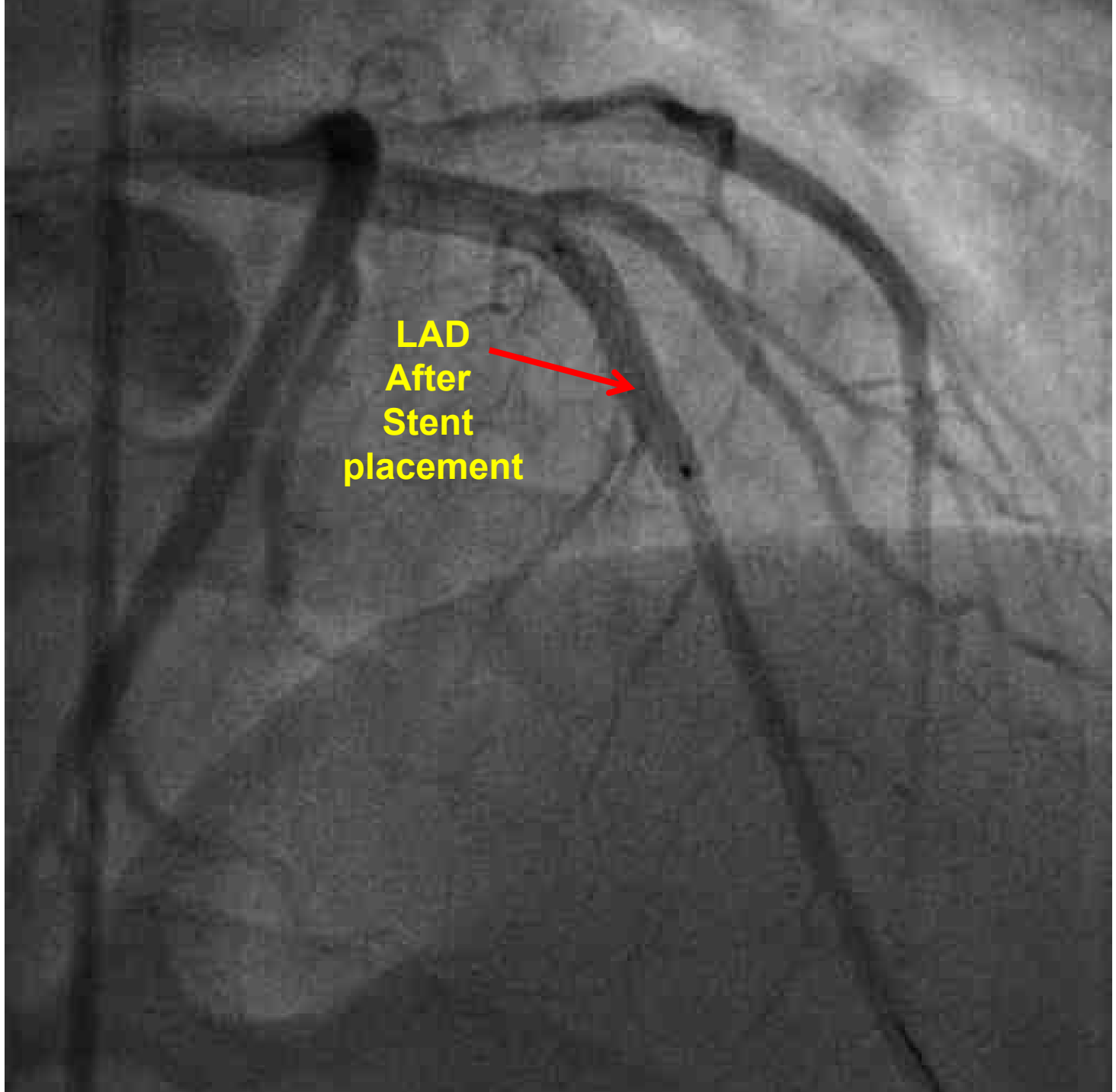
The artery that supplies the posterior descending artery (PDA)(1) (a.k.a. posterior interventricular artery) determines the coronary dominance.(1)

≈ 70% of the general population are right-dominant, 20% are co-dominant, and 10% are left-dominant.(2)

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 right coronary artery.

In left-dominance, the blood supply to the entire interventricular septum is derived from this "hyperdominant" LAD system, consequently, stenosis of LAD can be catastrophic.(3)

1. **The Collaborative Hypertext of Radiology (or "CHORUS") is a free medical reference database. It is based upon a system originally developed at the University of Chicago, but is currently maintained at the Medical College of Wisconsin.**
2. **Fuster, V; Alexander RW, O'Rourke RA (2001). *Hurst's The Heart* (10th ed.). McGraw-Hill. p. 53.**
3. **Javangula K, Kaul P. Hyperdominant left anterior descending artery continuing across left ventricular apex as posterior descending artery coexistent with aortic stenosis. J Cardiothorac Surg. 2007 Oct 21;2:42.**


LAD
After
Stent
placement

Opinião dos colegas
Colleagues opinions

Queridos amigos del forum este ECG es similar a uno ya discutido en el pasado, y está descripto en mi libro donde lo he llamado arterias coronárias similares a las anatomia coronaria de los perros

Las derivaciones de los miembros sugieren obstrucción de una arteria coronária derecha muy pequeña. V1 positivo indica que la obstruccion es proximal, señalando que el septo anterior derecho esta irrigado por la arteria conal o primera marginal, y el septo superior izquierdo y medio estan irrigados por la arteria primera marginal. No hay signos de remodelación (es decir ST -T deprimido en V2-V3) por dos motivos una porque V2-V3 estan isquemicas y II no presenta elevación significativa del segmento ST,sugeriendo que la cara posterior esta irrigada casi exclusivamente por la circunfleja. Esta es la artéria dominante, (como en los canes. En este animal el cierre de la artéria circunfleja ocasiona fibrilación instantánea). En estos pacientes la oclusión de la Cx puede ocasionar grande daño

En fin, el sindrome de coronaria derecha pequeña con obstrucción súbita proximal, arteria pequeña con arteria marginal primera derecha ,no muy desarrollada,(a veces mas grande que estas) y con artéria circunfleja dominante

Samuel Sclarovsky

La asociación clínica entre abuso de cocaína y eventos isquémicos ha sido reconocida desde hace mucho tiempo; Young y Glauber reportaron cambios electrocardiográficos relacionados con el uso de cocaína en el año 1947. A partir de entonces se han incrementado de manera progresiva los reportes de **angina de pecho e infarto de miocardio en pacientes que ingieren cocaína en ausencia de enfermedad coronaria relevante.*FISIOPATOLOGÍA:*** los mecanismos fisiopatológicos de la isquemia y el infarto de miocardio relacionados con el consumo de cocaína son varios y concluyen en el **disbalance de la ecuación aporte /demanda de oxígeno miocárdico** (disminución del aporte y aumento del consumo).

La cocaína aumenta el consumo de oxígeno a través del incremento de los tres determinantes mayores de dicho consumo: la frecuencia cardiaca**, la presión arterial** y la contractilidad miocárdica del ventrículo izquierdo; estas acciones son mediadas por el **efecto simpaticomimético de la cocaína**.

Por otro lado la cocaína produce **vasoconstricción de las arterias coronarias epicárdicas**, lo que lleva a una disminución del aporte de oxígeno al miocardio. La vasoconstricción coronaria se produce tanto en las arterias sanas como en las enfermas, siendo mayor el efecto a nivel de éstas últimas, por lo que los pacientes con coronariopatía previa que consumen cocaína tienen mayor riesgo de padecer un evento coronario agudo que las consumidores sin antecedentes de enfermedad ateroesclerótica coronaria.

El otro elemento patogénico relevante que interviene en la génesis de la cardiopatía isquémica aguda de la cocaína es la formación de trombos. El uso de esta droga se asocia con aumento de la activación y la agregación plaquetaria, incremento de la concentración del inhibidor del activador del plasminógeno, disminución de los niveles plasmáticos de antitrombina III y aumento de la producción de tromboxanos, todo lo cual promueve la **formación de trombos**.

El electrocardiograma es particularmente difícil de interpretar en pacientes jóvenes debido a la alta incidencia de cambios por repolarización precoz e hipertrofia ventricular izquierda, de manera tal que hasta un 84% de los pacientes con dolor torácico asociado a uso de cocaína pueden tener electrocardiograma anormal;

El valor predictivo positivo del electrocardiograma para detectar infarto de miocardio en los pacientes que consumen cocaína es de apenas el 18 % y su valor predictivo negativo es del 96 %, siendo este último dato el de mayor de relevancia clínica que surge a partir de estudio complementario.

Arritmias que produce la cocaína-

1. Taquicardia **y bradicardia sinusal**
2. Taquicardia supraventricular
3. Bloqueo de rama,
4. bloqueo auriculoventruicular completo
5. Ritmo idioventricular acelerado
6. Taquicardia ventricular, torcida de punta
7. Fibrilación ventricular, asistolia.*1,2,3,4,5,6
8. El electrocardiograma de este caso muestra una bradicardia sinusal y posteriormente existe una supradesnivelamiento del ST.

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Mi opinion de este caso se relaciona con la semiologia del dolor del profesor Macruz. El dolor que refiere el paciente es de cara anterior. En "aperto" sin iradiacion no es de coronaria derecha lo que corresponderia a cara inferior sino de coronaria izquierda que es cara anterior casi siempre. Seguramente la circulación de la cara inferior la mantenia la CI puesto que la CD es atrofica. un abrazo

Emilio Marigliano emiliomarigliano2000@yahoo.com

Hola Eduardo
No se en relacion a que caso Ud presenta este interesante review.
Justamente ayer, salio publicado esta revision sistematica que escribimos con Pancho Femenia.
Tal vez resulte de su interes.
Salud
AB

Electrocardiographic findings associated with cocaine use in humans: a systematic review.

Ramirez FD, Femenia F, Simpson CS, Redfearn DP, Michael KA, Baranchuk A.
Expert Rev Cardiovasc Ther. 2012 Jan;10(1):105-27,
AB

¡Interesante caso! En mi opinión, con este árbol conorario que describes con una coronaria derecha tan corta y con una CX normal, el ascenso del ST en derivaciones inferiores puede ser debido a la oclusión muy distal de una arteria descendente anterior muy larga que irriga gran parte del territorio que habitualmente corresponde a una coronaria derecha normal.
Sin embargo el ECG tras la angioplastia + stent muestra signos de afectación anterior e inferior propio de una lesión de una DA media muy larga. Sería interesante ver algún fotograma de la coronariografía.

Un abrazo,

Javier García Niebla

Estimado Dr Raimundo: 1. Angina vasoespastica, puede o no estar asociada al consumo de drogas ilicitas (cocaina), otra causa es el puente muscular en la DA (no lo refieren) el que provoca el fenomeno del vasoespasmo. 2. El diagnóstico diferencial electrocardiográfico entre dos causas de supradesnivel en precordiales derechas, son el SCA con compromiso de la cara anterior del VI o del VD. El primer dato a favor del de los segundo es que el supradesnivel anterior es descendente desde V1 hacia V4 y el otro dato es el supradesnivel en V3R-V4R. En este caso observo lo contrario y V3R presenta mayor supra que V4R. En este caso el supradesnivel es ascendente de V1 a V3. Los cambios en V4R sugieren compromiso del VD, estos cambios no son solo producidos por IAM de CD, sino por el compromiso hemodinamico del VD, creo en esto vital que el paciente tiene dominancia izquierda y CD hipoplasica.. En este caso la injuria inferior por isquemia de la DA. En estudios el supra ST en II, III o aVF, y se observó DA desarrollada que da la vuelta al ápex (sensibilidad del 30%, especificidad del 100%, valor predictivo positivo [VPP] del 100%, valor predictivo negativo [VPN] del 46%), y en estos el territorio de la diagonal estaba respetado (lesión distal a diagonal). El ascenso del ST en cara inferior se asocia a DA desarrollada que da la vuelta al ápex, y de una lesión distal a la diagonal dominante. Por esto pienso en una oclusion de tercio medio de DA distal a la diagonal. Desgraciadamente en este IAM sin tiempo la angioplastia no logró rescatar el area infartada y se observa post cateterismo una lesion extensa anteroseptoapical y disminucion de los voltajes de las R en cara lateral e inferior, probablemente evolucione a la dilatacion VI, el maestro Samuel sabiamente nos podra explicar el porque de este fenómeno. Una pregunta amigo Raimundo no recibio 2b3a en la angioplastia? porque estaban plenamente indicados

Un cordial saludo

Martin Ibarrola

1. Birnbaum Y, Slarovsky S, Solodky A, Tschori J, Herz I, Sulkes J, et al. Prediction of the level of left anterior descending coronary artery obstruction during anterior wall acute myocardial infarction by the admission electrocardiogram. Am J Cardiol 1993;72: 823-
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FINAL COMMENTS

By Andrés Ricardo Pérez-Riera M.D. PhD.

Dearest friend Raimundo: your concrete question is: How can we explain alterations compatible with acute occlusion of the RCA and observation of total occlusion with image of thrombus in the distal portion of the anterior descending artery (LAD). Answer: because *certain diagnostic tools, including ECG and serial cardiac markers, are not as accurate in identifying MI in cocaine users experiencing chest pain.(1) Additionally another situation where the deviation of the ST is limited is underdevelopment of the coronary arteries.*

Cocaine, a crystalline tropane alkaloid which is obtained from the leaves of the coca plant, acts a powerfully addictive stimulant that directly targets the CNS. The effects of the drug appear almost immediately after a single dose (intravenous, intranasal, or inhaled), and disappear within a few minutes or hours. Although the free commercialization of the drug is illicit and severely penalized in virtually all countries, its use remains widespread in many social, cultural, and personal settings.. There is emerging evidence that cocaine abuse might trigger a variety of cardiac disorders, ranging from arrhythmias to AMI, HF and even SCD, especially in relatively young male patients, in those who concomitantly use tobacco and alcohol, in those having experienced a trauma or a car accident and lack traditional risk factors for atherosclerosis.

Clinical situations where the deviation of the ST segment is limited

1. Presence of a previous infarction
2. Preexisting abnormalities of the ST segment
3. Left Bundle Brach Block/Right Bundle Branch Block
4. Ventricular Preexcitation
5. Multivessel disease
6. Abnormal site of origin of a coronary artery
7. Dominance or underdevelopment of the coronary arteries.

1. **Phillips K, Luk A, Soor GS, et al. Cocaine cardiotoxicity: a review of the pathophysiology, pathology, and treatment options.Am J Cardiovasc Drugs. 2009;9:177-196.**

Since the use of cocaine may influence the treatment strategies of patients being evaluated for possible acute coronary syndrome (ACS) as well as the prognosis of an AMI, it might be advisable to introduce cocaine screening in patients admitted with chest pain at the emergence department, especially in high-risk patients (i.e., young males with concurrent use of tobacco or alcohol, suffering from a recent accident and with no traditional atherosclerotic risk factors), or in those who are unresponsive and unreliable. This strategy might be helpful to adopt the best therapeutic approach for reducing the risks associated with cardiovascular disease in these patients, and also to deter relapse.

Cocaine is a powerful stimulant that gives users a temporary sense of euphoria, mental alertness, talkativeness, and a decreased need for food and sleep. Cocaine intoxication is the most frequent cause of drug-related death reported by medical examiners in the US, and these events are most often related to the cardiovascular manifestations of the drug. Once playing a vital role in medicine as a local anesthetic, decades of research have established that cocaine has the ability to cause irreversible structural damage to the heart, greatly accelerate cardiovascular disease, and initiate SCD. Although pathologic findings are often reported in the literature, few images are available to support these findings, and reviews of cocaine cardiopathology are rare.

Phillips et al (1) conducted a MEDLINE search to identify all English language articles from January 2000 to June 2008 with the subject headings and key words 'cocaine', 'heart', 'toxicity', and 'cardiotoxicity'. Epidemiologic, laboratory, and clinical studies on the pathology, pathophysiology, and pharmacology of the effects of cocaine on the heart were reviewed, along with relevant treatment options. Reference lists were used to identify earlier studies on these topics, and related articles from Google Scholar were also included.

1. **Phillips K, Luk A, Soor GS, et al. Cocaine cardiotoxicity: a review of the pathophysiology, pathology, and treatment options.Am J Cardiovasc Drugs. 2009;9:177-96.**

There is an established connection between cocaine use and myocardial infarction (MI), arrhythmia, HF, and SCD. Numerous mechanisms have been postulated to explain how cocaine contributes to these conditions. Among these, cocaine may lead to MI by causing coronary artery vasoconstriction and accelerated atherosclerosis, and by initiating thrombus formation. Cocaine has also been shown to block K+ channels, increase L-type Ca2+ channel current, and inhibit Na+ influx during depolarization, all possible causes for arrhythmia. Additionally, cocaine use has been associated with LVH, myocarditis, and dilated cardiomyopathy, which can lead to HF if drug use is continued. As a result, clinicians should be suspicious of cocaine use in their differential diagnosis of chest pain, especially in the younger male population, and proceed more cautiously when use is suspected.

Treatment for cocaine-related cardiovascular disease

Treatment for cocaine-related cardiovascular disease is in many ways similar to treatment for traditional cardiovascular disease. However use of ß-receptor antagonists and class Ia and III anti-arrhythmics is strongly discouraged if the patient is likely to continue cocaine use, because of documented adverse effects. The medical community is in urgent need of a pharmacologic adjunct to cocaine-dependence treatment that can deter relapse and reduce the risks associated with cardiovascular disease in these patients.

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

THE ECG IN ACUTE CORONARY SYNDROME(ACS)

ACS accounts for approximately 1.2 million hospital admissions in the United States annually.(2) Patients with ACS include those whose clinical presentations cover the following range of diagnoses. ACS is a broad term encompassing a spectrum of acute myocardial ischemia and injury ranging from unstable angina and non-ST-segment elevation myocardial infarction to ST-segment elevation myocardial infarction:

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(2)

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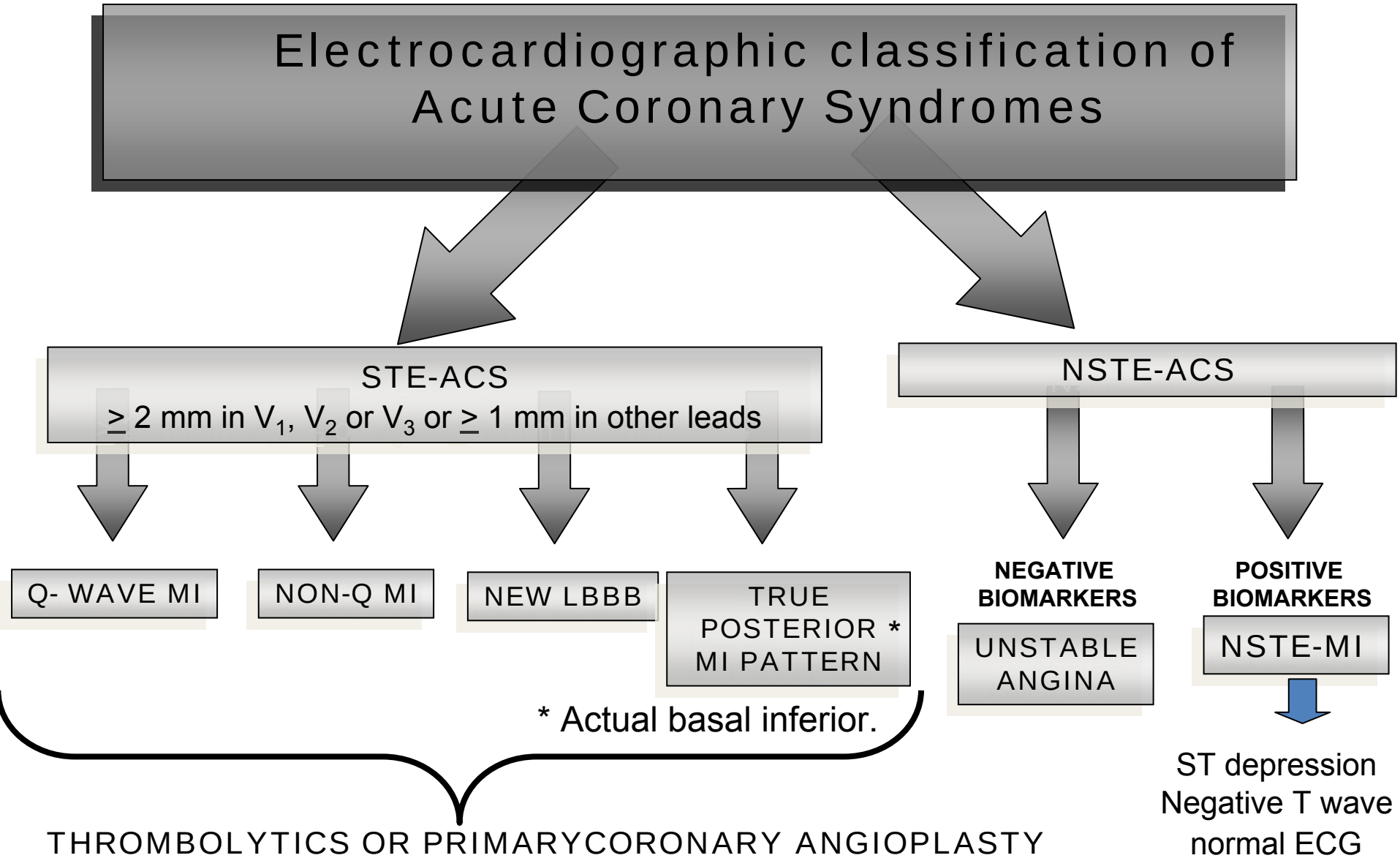
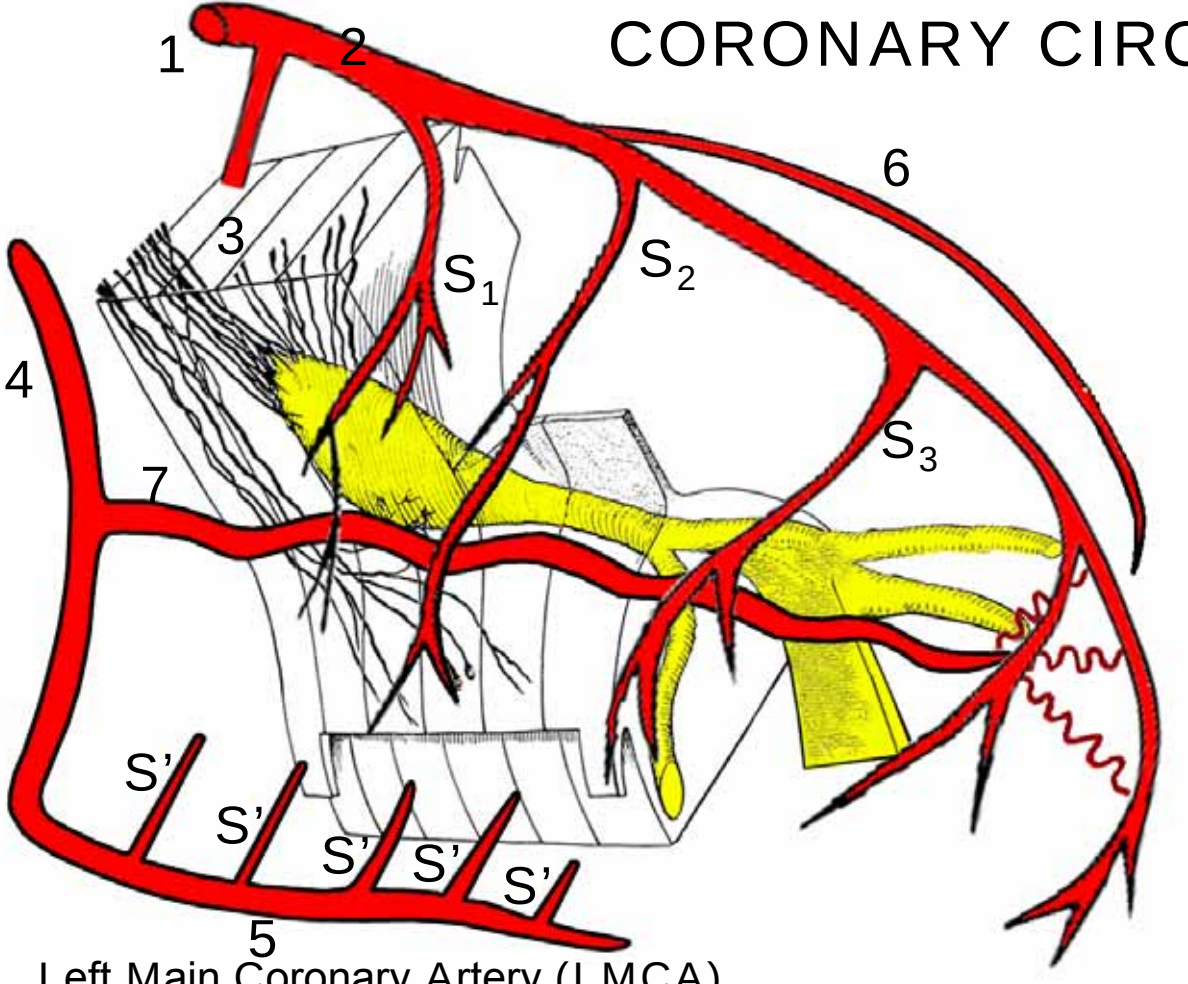
Electrocardiographic classification of Acute Coronary Syndromes



1. **Nikus K, Pahlm O, Wagner G, Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology. J Electrocardiol. 2010 Mar-Apr;43:91-103.**

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. **von Lüdinghausen M. The clinical anatomy of coronary arteries. Adv Anat Embryol Cell Biol. 2003;167:III-VIII, 1-111.**
2. **Cademartiri F, Marano R, Luccichenti G, et al.Normal anatomy of the vessels of the heart with 16-row multislice computed tomography. Radiol Med. 2004 Jan-Feb;107:11-21.**

1. Left Main Coronary Artery (LMCA)
2. Left Anterior Descending Artery (LAD)
3. Left Circumflex Coronary Artery (LCX)
4. Right Coronary Artery (RCA)
5. Posterior Descending Artery (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)

Left Main Coronary Artery (LMCA)

The left main coronary artery (LMCA) is also known as the left main or Left Main Stem(1)
The left main coronary artery have a mean length of 112±55 mm.(2)
The LCA arises from the left coronary cusp.
The aortic valve has three leaflets, each having a cusp or cup-like configuration. These are known as the left coronary cusp (L), the right coronary cusp (R) and the posterior non-coronary cusp (N). Just above the aortic valves there are anatomic dilations of the ascending aorta, also known as the sinus of Valsalva. The left aortic sinus gives rise to the left coronary artery. The right aortic sinus which lies anteriorly, gives rise to the right coronary artery. The non-coronary sinus is postioned on the right side.
The LMCA divides almost immediately into the left circumflex artery (LCX) and left anterior descending artery (LAD). On the left an axial CT-image the LMCA travels between the right ventricle outflow tract anteriorly and the left atrium posteriorly and divides into LAD and LCX. In 15% of cases a third branch arises in between the LAD and the LCX, known as the ramus intermedius or intermediate branch. This intermediate branches behaves as a diagonal branch of the LCX.

1. Liang M, Kelly DJ, Devlin G. Left main stem stenosis in the unstable patient--forewarned is forearmed. N Z Med J. 2011 Jul 8;124:111-113.
2. Cademartiri F, La Grutta L, Malagò R, et al Prevalence of anatomical variants and coronary anomalies in 543 consecutive patients studied with 64-slice CT coronary angiography. Eur Radiol. 2008 April; 18: 781–791.

The Left Anterior Descending (LAD) and it's branches

The LAD or anterior interventricular branch of the left coronary artery, or anterior descending branch travels in the anterior interventricular groove and continues up to the apex of the heart.
It passes at first behind the pulmonary artery and then comes forward between that vessel and the left auricula to reach the anterior interventricular sulcus, along which it descends to the incisura apicis cordis.In 78% of cases, it reaches the apex of the heart.
The LAD supplies the anterior anterior wall of the left ventricle with diagonal branches and the part of the septum with septal branches. The LAD supplies most of the left ventricle (LV). The LAD typically supplies 45-55% of the LV and also the AV-bundle. It supplies the anterolateral myocardium, apex, and interventricular septum.
The LAD gives off two types of branches: *septals* and *diagonals*.

Septals for the antero-septal territory originate from the LAD at 90º to the surface of the heart, perforating and supplying the interventricular septum. The major perforating septal arteries arise from the superior interventricular artery. The first is usually the largest. The location of this artery can be predicted relative to the position of the medial papillary muscle. The location of the first superior septal perforating artery is predictable in many cases. Its course leaves a triangular area on the muscular ventricular septum that is free of major arteries.
Diagonal branches arise from the LA**D**, run along the surface of the heart and supply the lateral wall of the left ventricle and the anterolateral papillary muscle. The diagonal branches come off the LAD and run laterally to supply the anterolateral wall of the left ventricle.
The first diagonal branch serves as the boundary between the proximal and mid portion of the LAD.
There can be one or more diagonal branches: D1, D2 , etc.

1. **Hosseinpour AR, Anderson RH, Ho SY. The anatomy of the septal perforating arteries in normal and congenitally malformed hearts.J Thorac Cardiovasc Surg. 2001 Jun;121:1046-1052.**

Left Circumflex (LCX)

The Cx or Left circumflex artery (LCX) lies in the left AV groove between the left atrium and left ventricle and supplies the vessels of the lateral wall of the left ventricle. The LCX gives three left atrial branches, the superior, medium and inferior branches and 1 or 2 left ventricular (lateral) branches for the corresponding lateral territory of the left ventricle.(1)

These vessels are known as obtuse marginals (M1, M2...), because they supply the lateral *margin* of the left ventricle and branch off with an *obtuse* angle.

In most cases the LCX ends as an obtuse marginal branch, but 10% of patients have a left dominant circulation in which the LCX also supplies the posterior descending artery (PDA).

Mnemonic: **M**arginal branches arise from the LCX and supply the lateral **M**argin of the left ventricle.

Patients with LCX-related AMI were less likely to present with ST elevation in ECG (46.3%, 87.0%, and 82.3%; $p<0.001$) and primary percutaneous coronary intervention (PCI) (43.4%, 78.9%, and 74.5%; $p<0.001$) and door to balloon time <90 min (31.3%, 52.8%, and 51.0%; $p<0.001$), compared with LAD and RCA. However, no statistical difference was found in hospital mortality among the three groups. Multivariate analysis showed primary PCI decreased the hospital mortality in patients with occluded coronary artery.

AMI patients with an occluded LCX presented with less ST elevation and primary PCI. These results suggest that clinical physicians should be careful with patients presenting with chest pain but apparently normal ECG and must rule out LCX occlusion.(2)

Acute LCX territory occlusion often presents as NSTEMI, but patients with NSTEMI and occlusion have a lower mortality risk than those with STEMI, possibly because of factors such as the amount of myocardium involved, the lesion location along the vessel, and/or a dual blood supply.(3)

1. **Cabrol C, Christides C. Usual arrangement and nomenclature of the coronary arteries. Bull Assoc Anat (Nancy). 1976 Dec;60:645-649.**
2. **Kim SS, Choi HS, Jeong MH, et al ; Clinical outcomes of acute myocardial infarction with occluded left circumflex artery. Korea Acute Myocardial Infarction Registry Investigators. J Cardiol. 2011 May;57:290-296.**
3. **Stribling WK, Kontos MC, Abbate A, et al. Left circumflex occlusion in acute myocardial infarction (from the National Cardiovascular Data Registry). Am J Cardiol. 2011 Oct 1;108:959-963.**

Right Coronary Artery (RCA)

The right coronary artery(RCA) arises from the anterior sinus of Valsalva and courses through the right atrioventricular (AV) groove between the right artium and right ventricle to the inferior part of the septum.
In 50-60% the first branch of the RCA is the small conus branch, that supplies the right ventricle outflow tract.
In 20-30% the conus branch arises directly from the aorta.
In 60% a sinus node artery arises as second branch of the RCA, that runs posteriorly to the SA-node (in 40% it originates from the Cx).

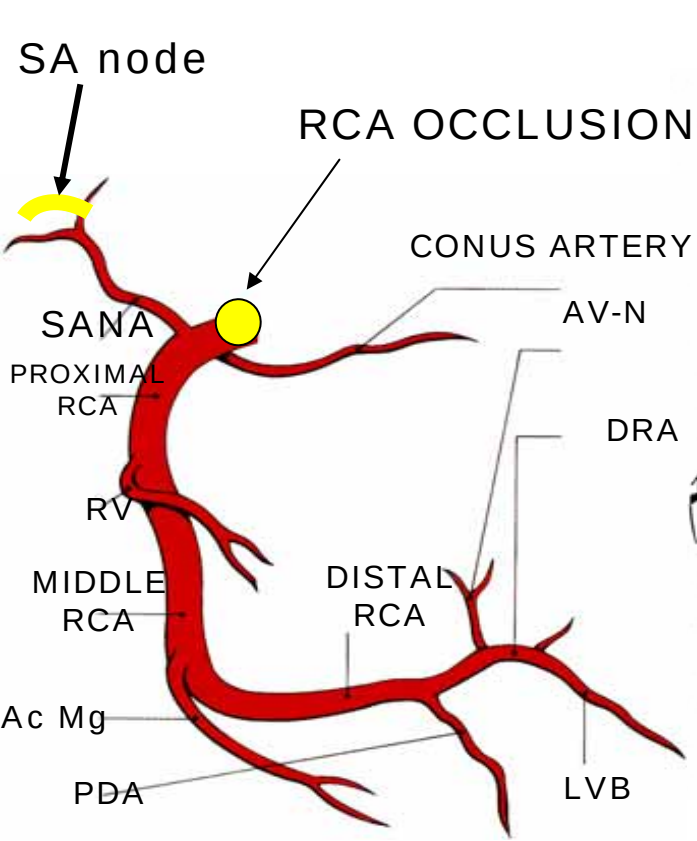
The next branches are some diagonals that run anteriorly to supply the anterior wall of the right ventricle.
The large acute marginal branch (AM) comes off with an *acute* angle and runs along the *margin* of the right ventricle above the diaphragm.

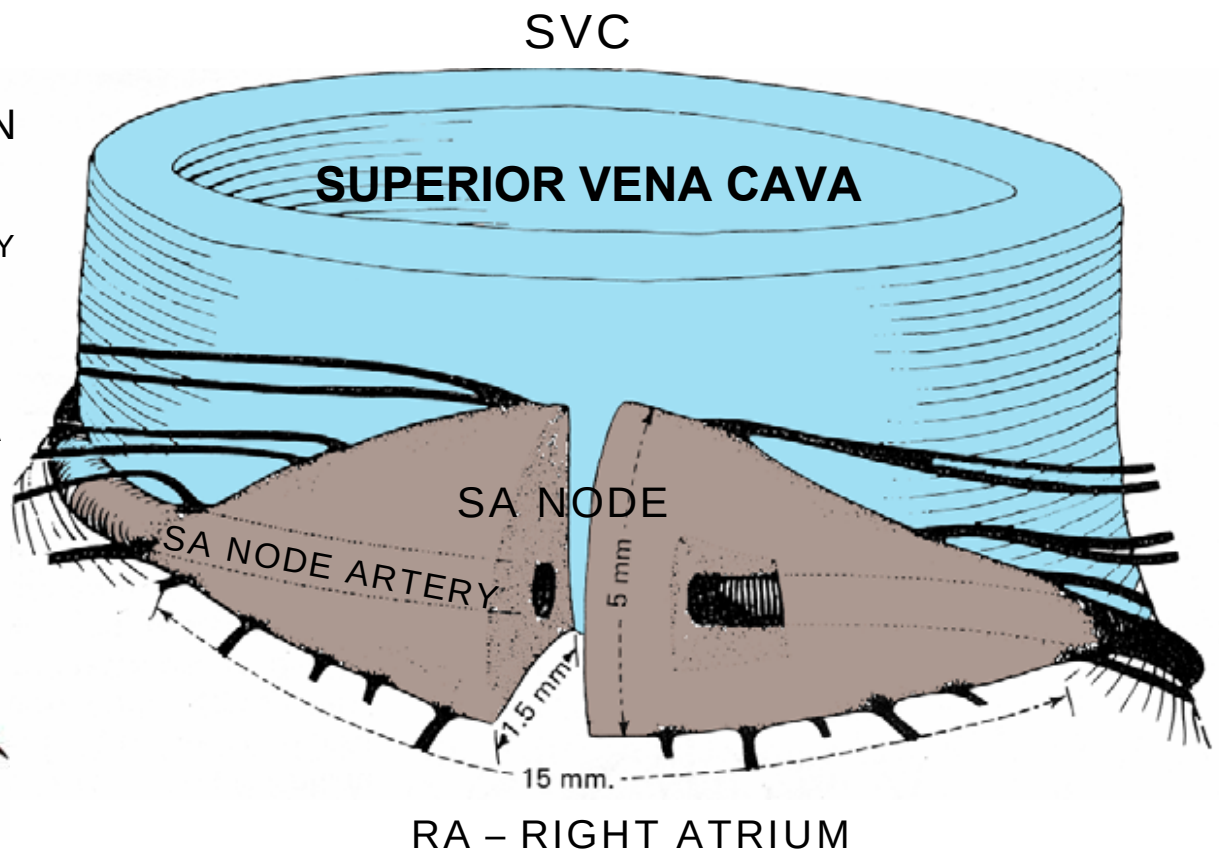
The RCA continues in the AV groove posteriorly and gives off a branch to the AV node.
In 65% of cases the posterior descending artery (PDA) is a branch of the RCA (right dominant circulation).
The PDA supplies the inferior wall of the left ventricle and inferior part of the septum.(1)

1. **Zimmermann E, Schnapauff D, Dewey M. Cardiac and coronary anatomy in computed tomography.Semin Ultrasound CT MR. 2008 Jun;29:176-181.**

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]. Futami et al (2) observed that the sinoatrial node was supplied by the RCA in 73% of cases than by the left (3%), and in 23% of cases this node was supplied by both coronary arteries.





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.**
2. **Futami C, Tanuma K, Tanuma Y, et al. The arterial blood supply of the conducting system in normal human hearts. Surg Radiol Anat. 2003 Apr;25:42-49.**

Normal hearts of cadavers of 100 individuals (69 males), belonging to 24 Caucasians (whites) and 36 non-Caucasians (Negroes and Mulattoes), whose age varied ranged from 7 to 80 years, were studied by Caetano et al(1). The coronary arteries were injected with gelatin mixed to a radiopaque substance and a red or blue pigment.

RESULTS AND CONCLUSIONS

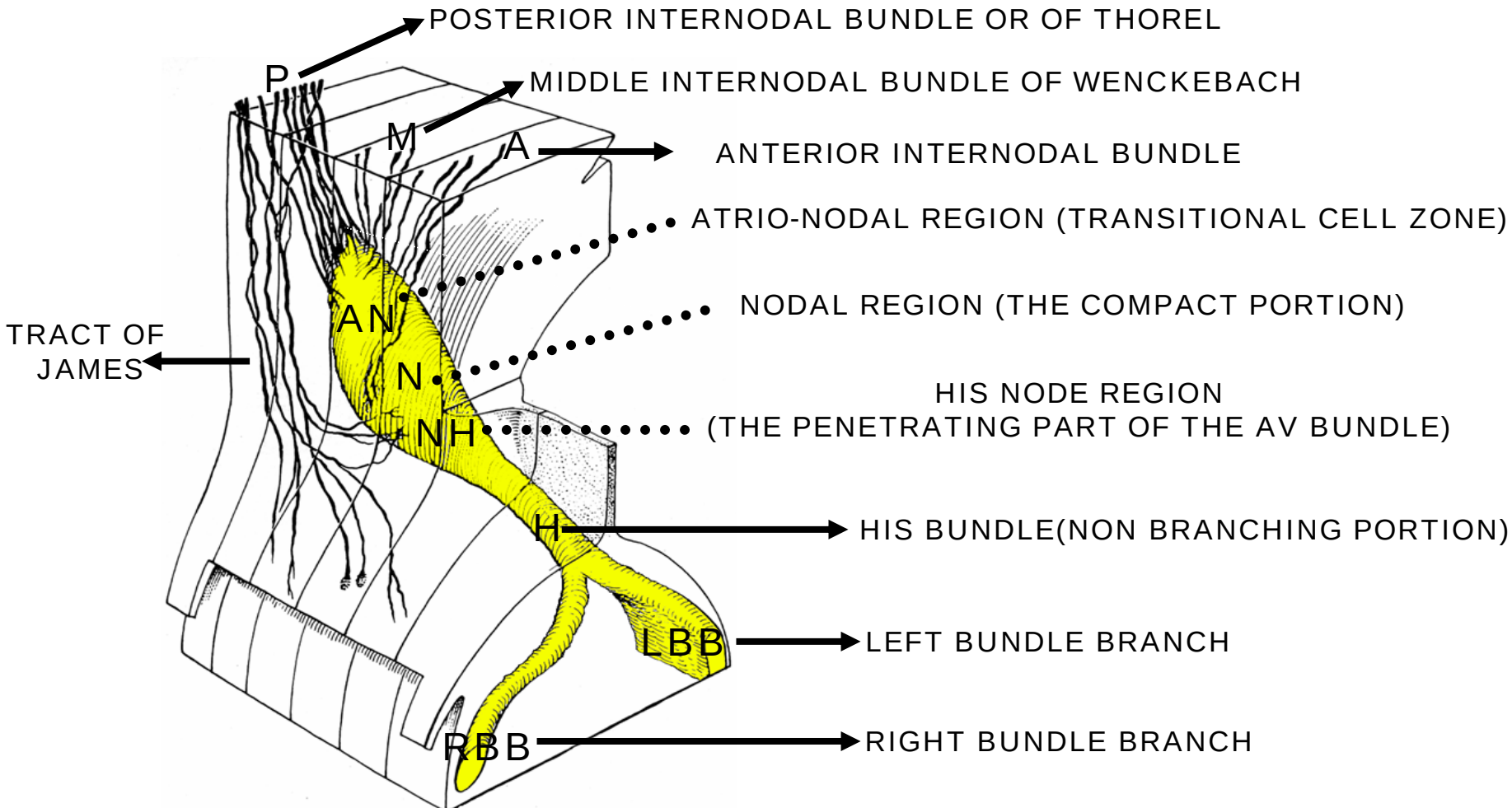
1) The artery of the SA- node was originated more frequently from the RCA (58%) than from the left.
2) When originated from the left coronary artery the artery of the SA-node was more frequently a branch of the LCX (30%) than from the trunk of this artery (12%).
3) No sexual or racial factor influenced the anatomical variations.
4) The most frequent arteries originating the artery of the SA-node were the right anterior medial atrial artery and the left anterior medial atrial artery.
5) No cases of blood supply to the SA-node were found originating neither from other arteries than the coronary arteries nor from both coronary arteries.
6) The distribution of the coronary arteries allows to understand the possible ischemic etiology of the SA-node syndrome and permits to the surgeon a safe approach to cardiac disease.

1. **Caetano AG, Lopes AC, DiDio LJ, Prates JC. Critical analysis of the clinical and surgical importance of the variations in the origin of the sinoatrial node artery of the human heart. Rev Assoc Med Bras. 1995 Mar-Apr;41:94-102.**

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.

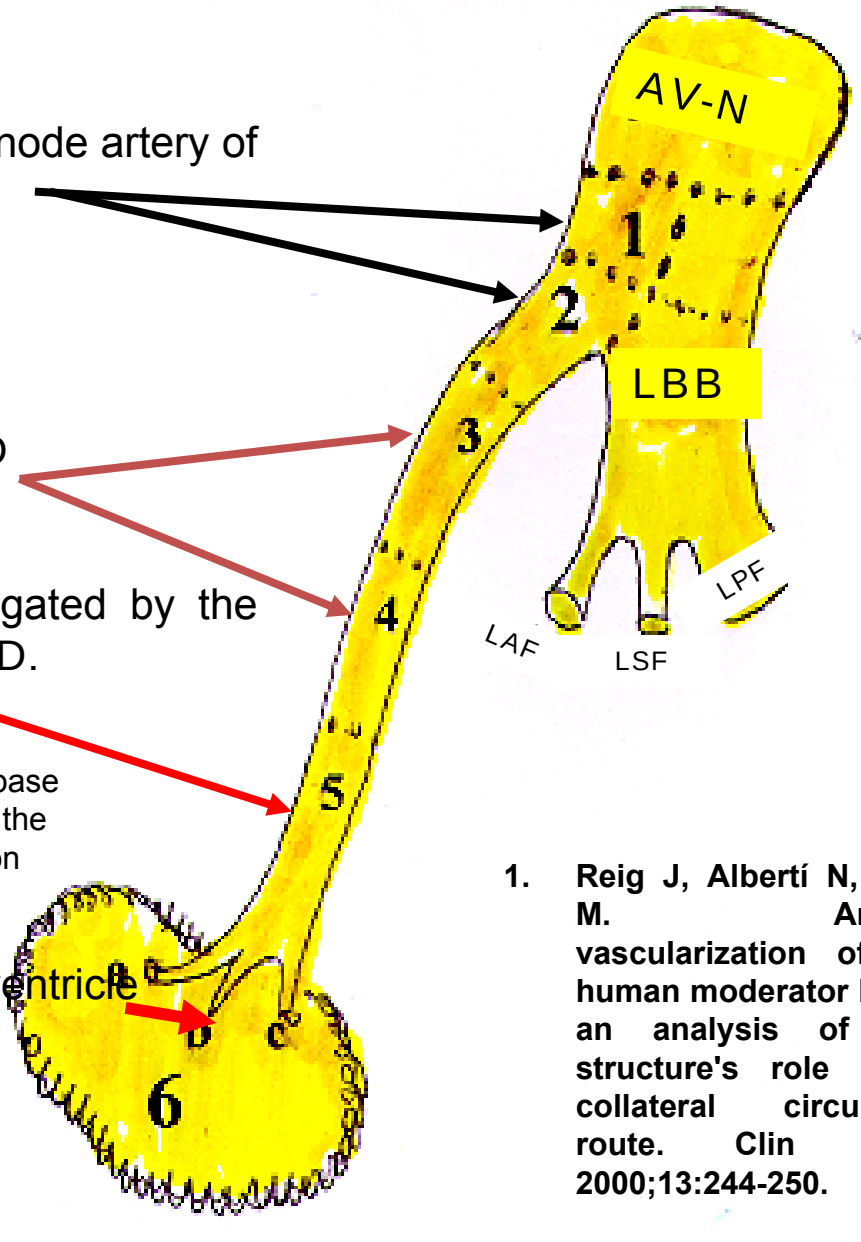
MIDDLE PORTION is irrigated by:
Posterior Septal perforators of the PDA
Second septal perforator artery(S_2) of the LAD
Kugel's artery, branch of the LCX.

MIDDLE AND DISTAL PORTION: are irrigated by the "Ramus limbi dextri", branch of the S_2 of the LAD.
or the first three anterior septal arteries

The moderator band artery made anastomotic connections at the base of the anterior papillary muscle of the RV with various branches of the RCA, which means that it can play a key role in collateral circulation following obstruction of the epicardium coronary arteries.(1)

Base of the anterior papillary muscle of the right ventricle

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



1. **Reig J, Albertí N, Petit M. Arterial vascularization of the human moderator band: an analysis of this structure's role as a collateral circulation route. Clin Anat. 2000;13:244-250.**

BLOOD SUPPLY OF THE TRUNCUS OF LEFT BUNDLE BRANCH (LBB)

I) Length: 10 mm (five times shorter than the RB).
II) Diameter: in its onset 5 mm and at the end 9 mm (four to eight times longer than the RB).
III) Color: white.
IV) Cell type: Purkinje. These are large cells of 15 to 30 mm of diameter and 20 to 100 mm of length.
V) Conduction velocity: 5 m/sec (fast fibers)
VI) Characteristics of AP: fast fiber type, Na^+ dependent, phase 4 with automatism (diastolic depolarization) and with a refractory period shorter than the right bundle branch (RBB): faster depolarization and repolarization.
VII) Related to: very close to the following structures:Noncoronary and right coronary aortic valves, aortic ring (the reason why is frequent in aortic valve disease), membranous septum, subaortic septal endocardium, muscular septum apex, and right bundle branch of the His bundle (the anterior portion of the LBB).
H) Irrigation: assured by two arterial systems:

1) Branches of the posterior descending artery (90% of the RCA):
a) AV node artery: ramus septi fibrosi.
b) Ramus septi ventriculorum superior.
c) Ramus cristae.

2) Branches of LAD:
a) Ramus limbi sinistri (equivalent to ramus limbi dextri of the LAD).

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 %

BLOOD SUPPLY OF THE PAPILLARY MUSCLES

The papillary muscles attach the mitral valve and the tricuspid valve to the wall of the heart. If the papillary muscles are not functioning properly, the mitral valve may leak during contraction of the left ventricle. This causes some of the blood to travel "in reverse", from the left ventricle to the left atrium, instead of forward to the aorta and the rest of the body. This leaking of blood to the left atrium is known as mitral regurgitation. Similarly, the leaking of blood from the right ventricle through the tricuspid valve and into the right atrium can also occur, and this is described as tricuspid insufficiency or tricuspid regurgitation.

The anterolateral papillary muscle (ALPM) more frequently receives two blood supplies:

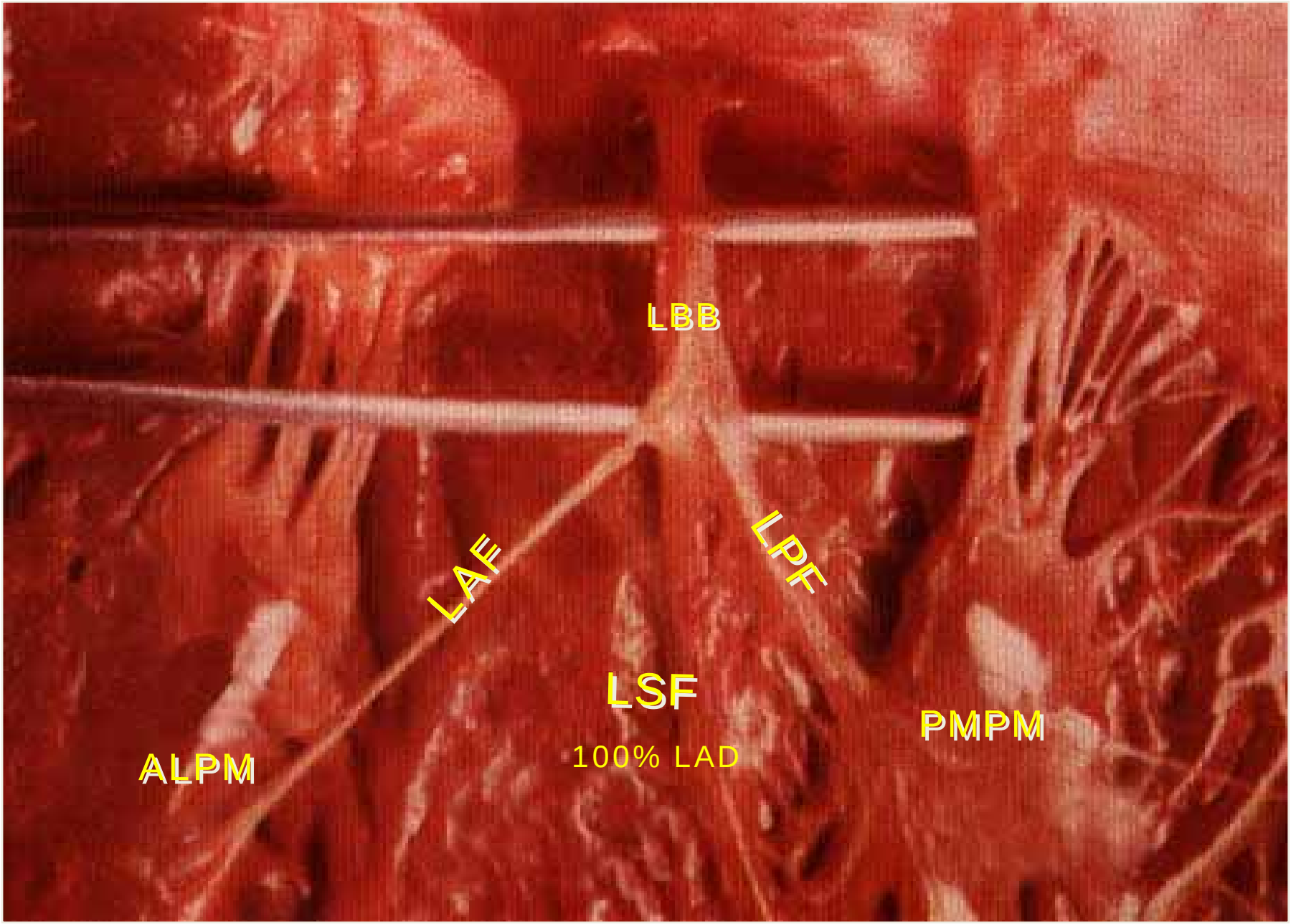
Left anterior descending (LAD) artery and

The left circumflex artery (LCX).(1)

It is therefore more frequently resistant to coronary ischemia.

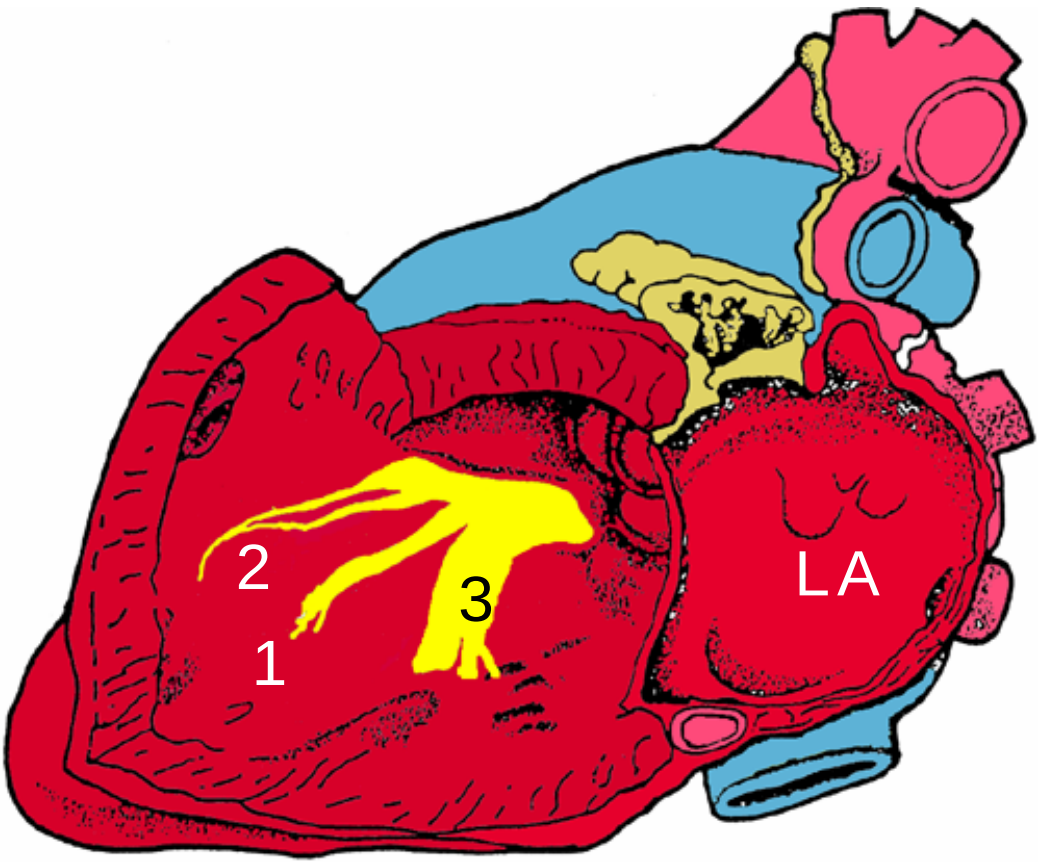
On the other hand, the posteromedial papillary muscle is usually supplied only by the PDA.(1) This makes the posteromedial papillary muscle significantly more susceptible to ischemia. The clinical significance of this is that a myocardial infarction involving the PDA is more likely to cause mitral regurgitation.(See next slide)

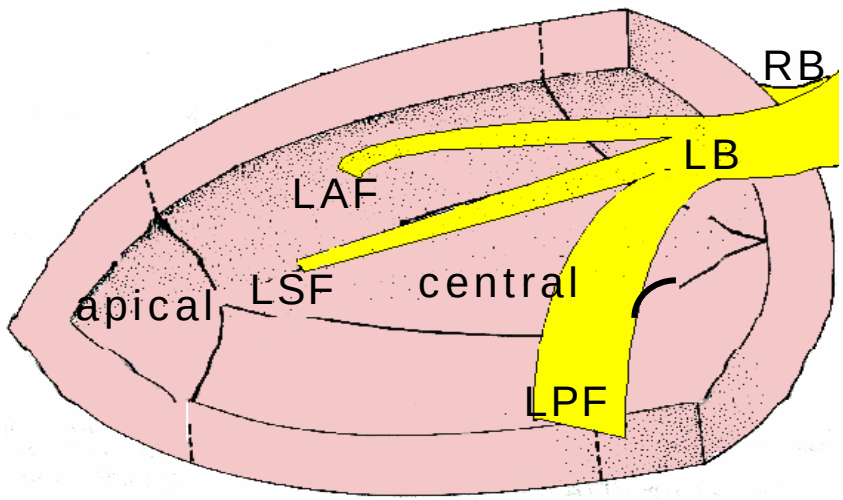
1. **Voci P, Bilotta F, Caretta Q, Mercanti C, Marino B "Papillary muscle perfusion pattern. A hypothesis for ischemic papillary muscle dysfunction". *Circulation* 1995; 91: 1714–1718.**



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.

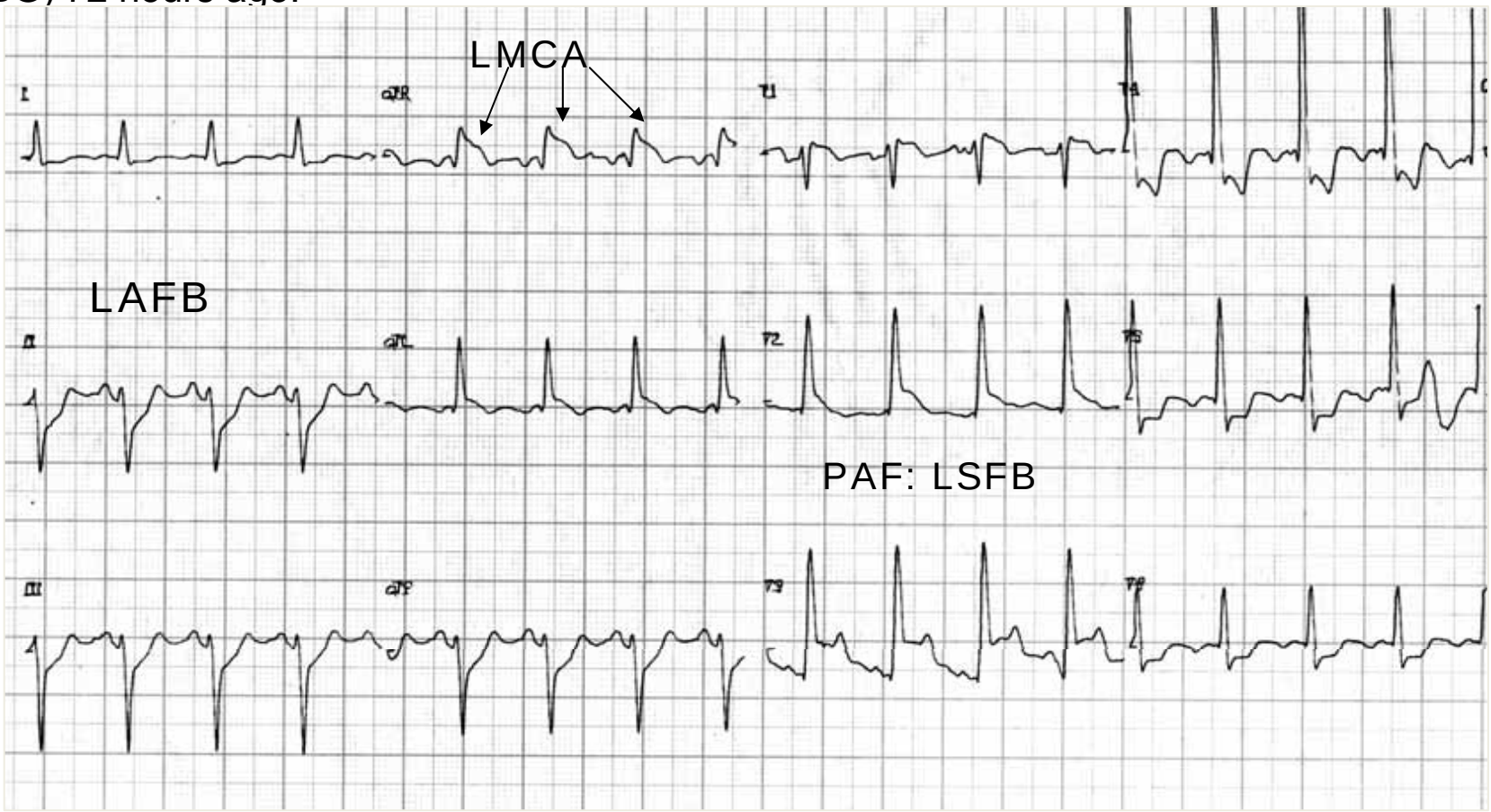
Killip class II: includes individuals with rales or crackles over 50% of the lung fields in the lungs, an S3, and elevated jugular venous pressure. **Forrester:** wedge ≤18mm Hg. Poor perfusion. Hipovolemic.

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.**

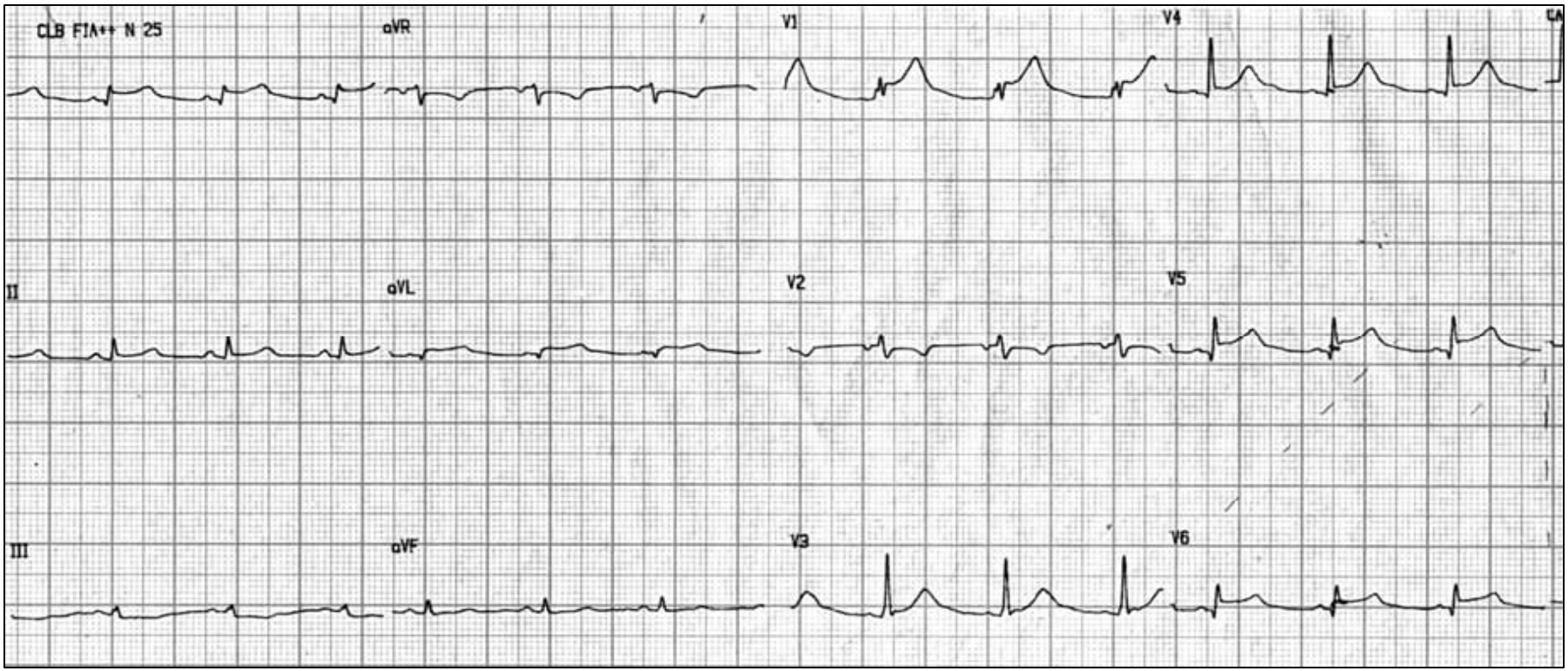
Name: AR.; **Date:** 02/01/2009.; **Age:** 72 yo.; **Gender:** Male.; **Ethnic Group:** Caucasian **Weight:** 72 Kg.; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours ago.



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 + 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 grafting (CABG) surgery.)

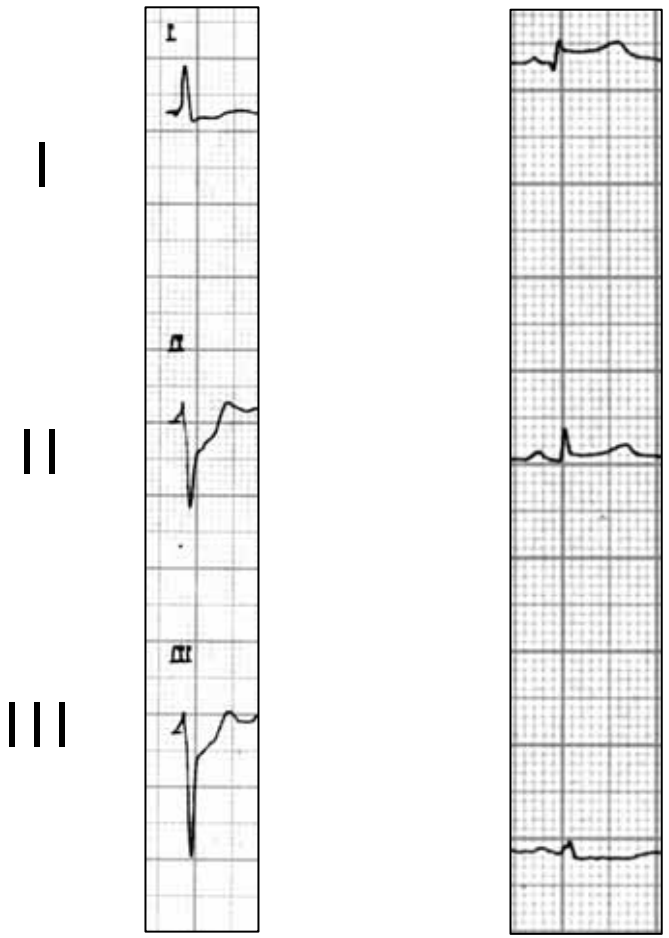
Name: AR; **Date:** 05/01/2009; **Age:** 72 yo;**Gender:** Male
Ethnic Group: Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic;
Management: Coronary Artery Bypass Graft (CABG) 72 hours ago.



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

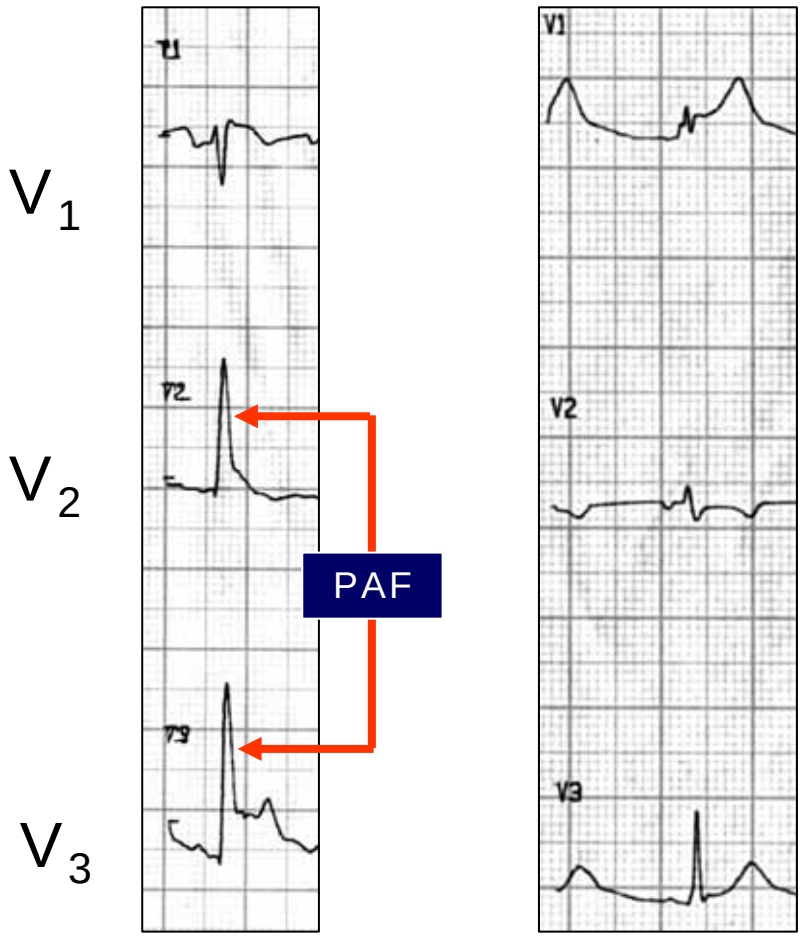


WITH LAFB

WITHOUT LAFB

Date:
02/01/2009

Date:
05/01/2009



WITH PAF: LSFB

WITHOUT PAF: LSFB

PAF: Prominent Anterior Forces

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

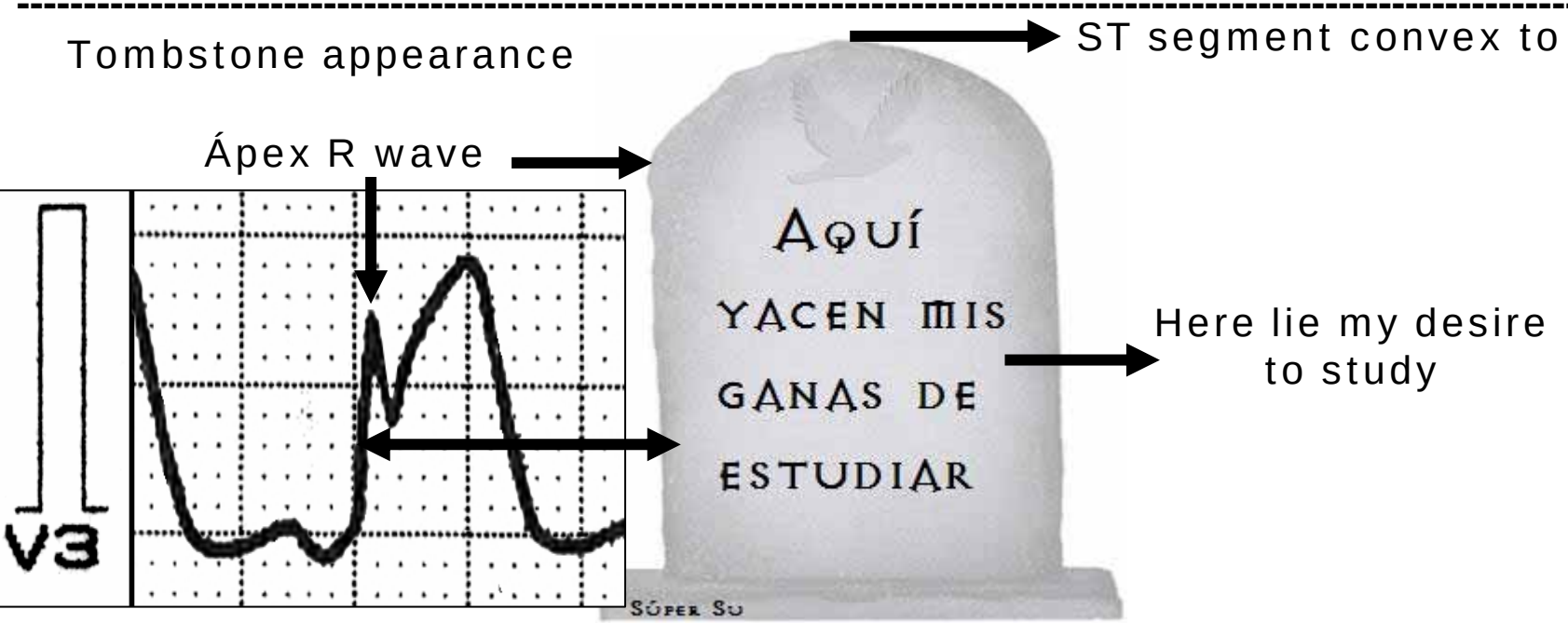
Tombstoning (**TOMB-ST**) is manifested by a particular changing the shape of repolarization: monophasic action potential-like patter consequence of proximal occlusion of LAD.
TOMB-ST has been associated with a poor prognosis ever since Wimalaratna's first description[1;2] of this clinical entity, and 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.

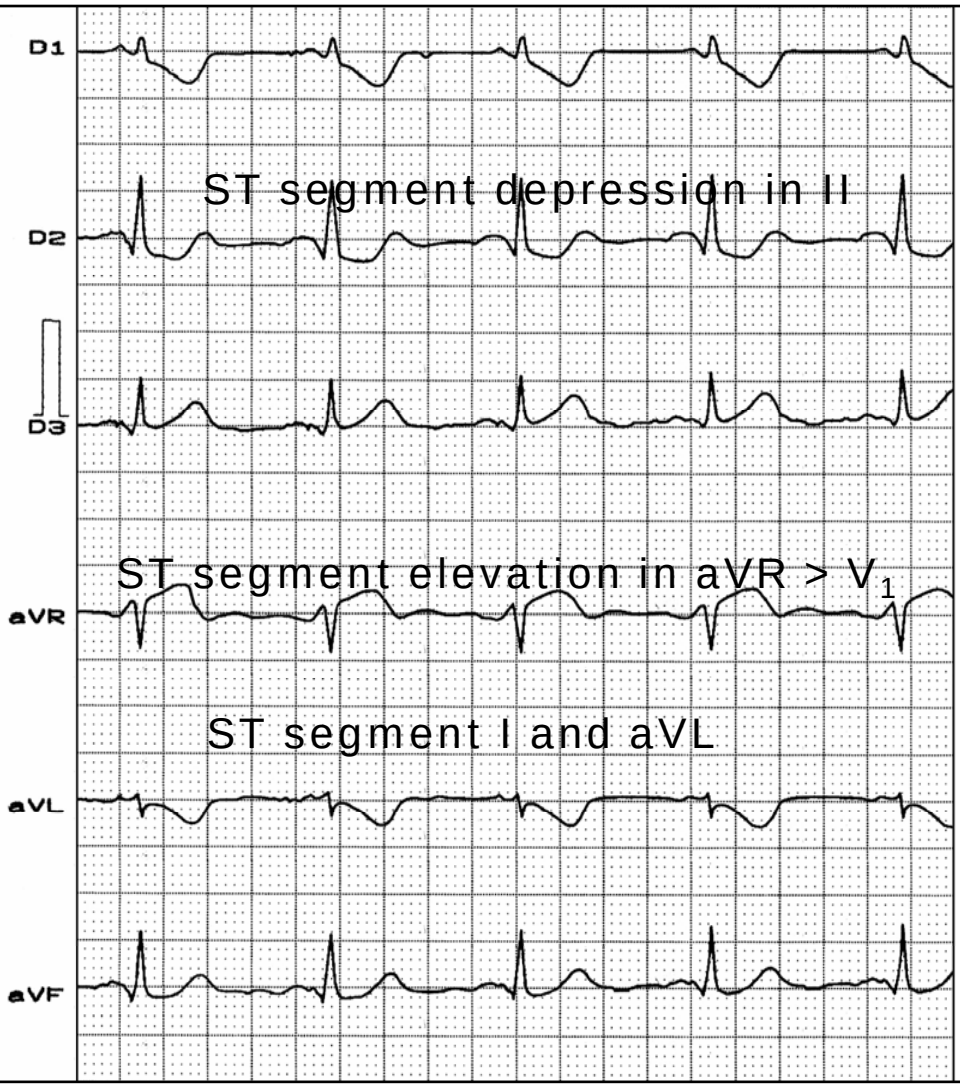


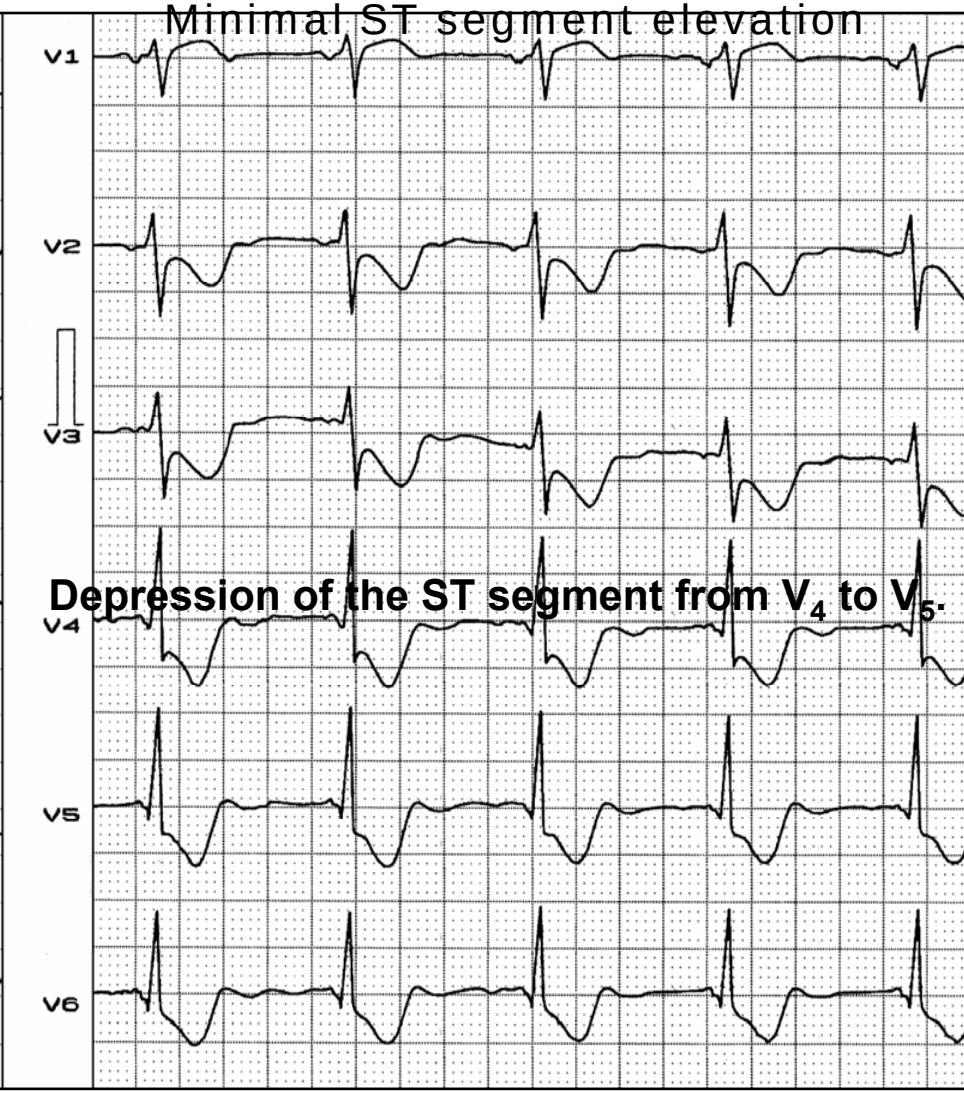
Two electrophysiological mechanisms play a role in the formation of a tombstone appearance: delayed transmural conduction and intramyocardial conduction block.

1. **Spodick DH. Tombstone ST segments. Am Heart Hosp J. 2005 Winter;3(1):61.**

TYPICAL ECG PATTERN OF LMCA OCCLUSION

Diffuse ST segment depression in the inferolateral leads





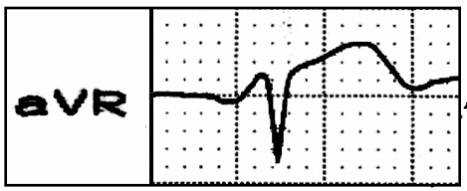
Why this pattern is observed?

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

The ST injury vector pointing to aVR

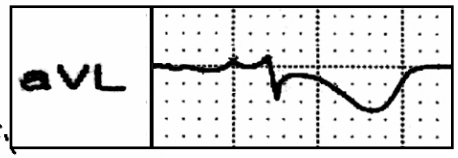
Frontal

ST segment elevation



LMCA

LCx



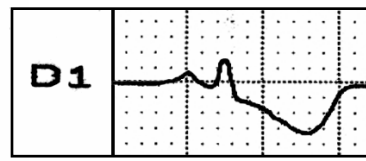
LAD

180°

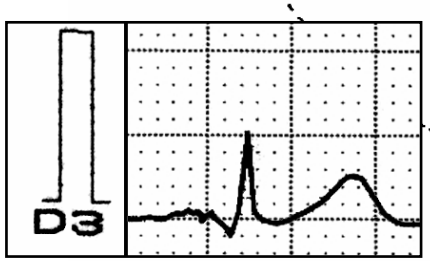
0°

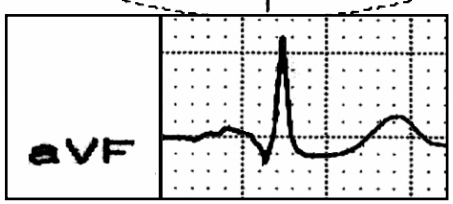
LV

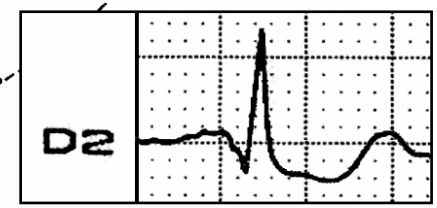
RV

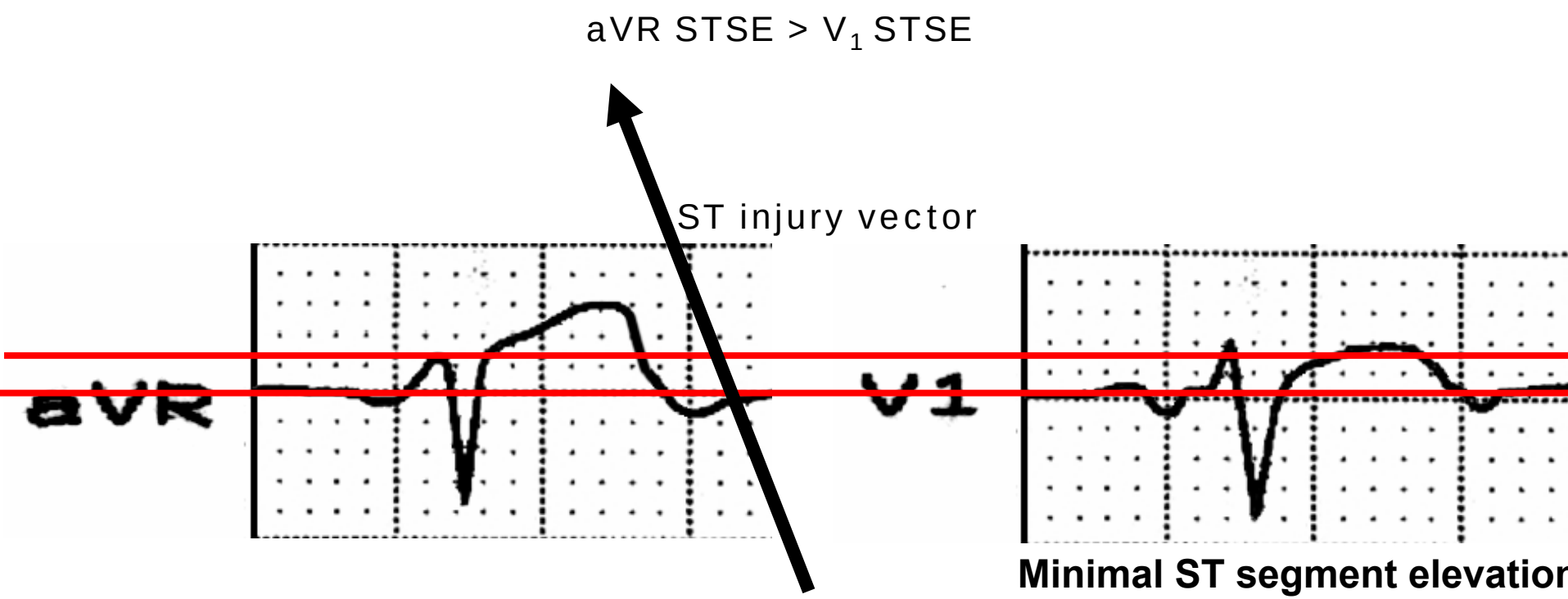


ST segment depression in II I and aVL

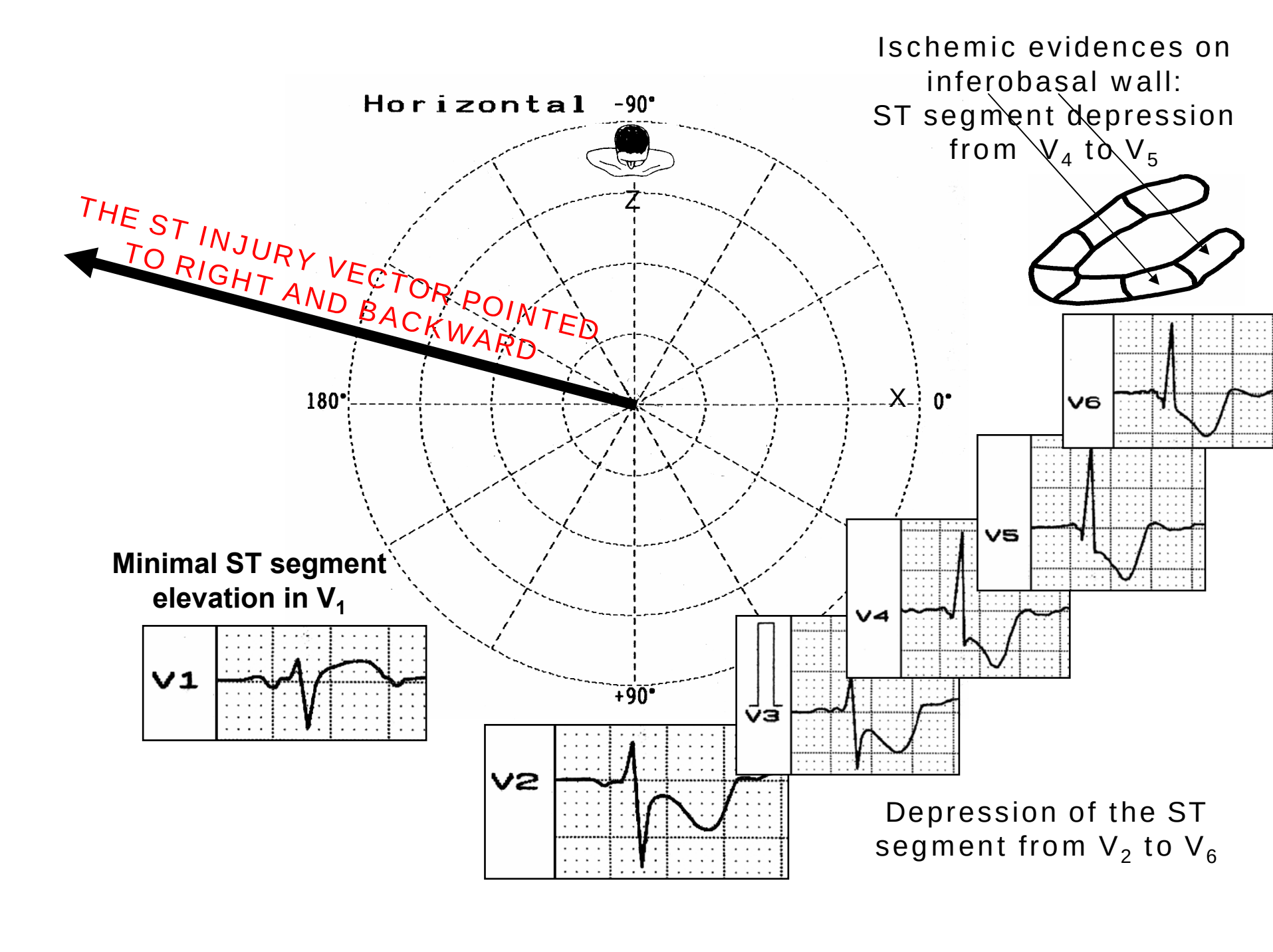






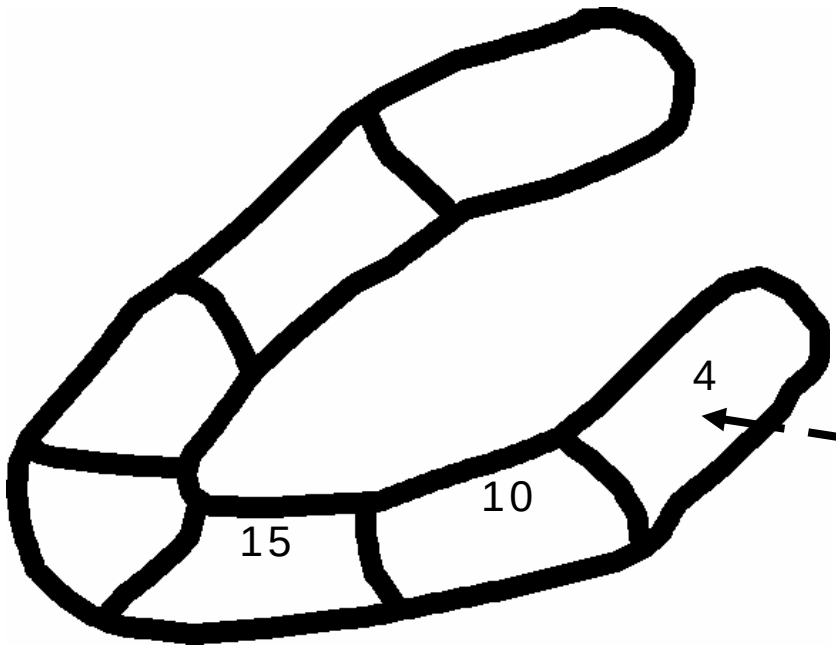


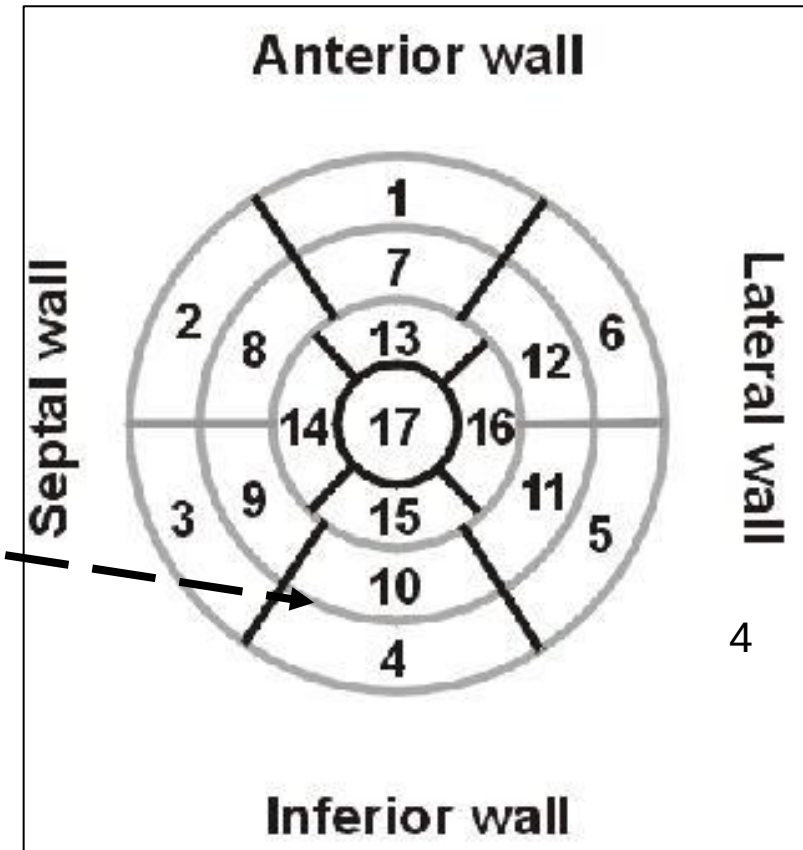
ST segment elevation in aVR > V_1. Why?
Because the ST injury vector is directed to upward and rightward, pointing to aVR lead (RVOT) ST segment elevation **in lead aVR, a less recognized finding associated with severe left main disease, may be present.**


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 V4 to V5
Minimal ST segment elevation in V1
V1
V2
V3
V4
V5
V6
Depression of the ST segment from V2 to V6

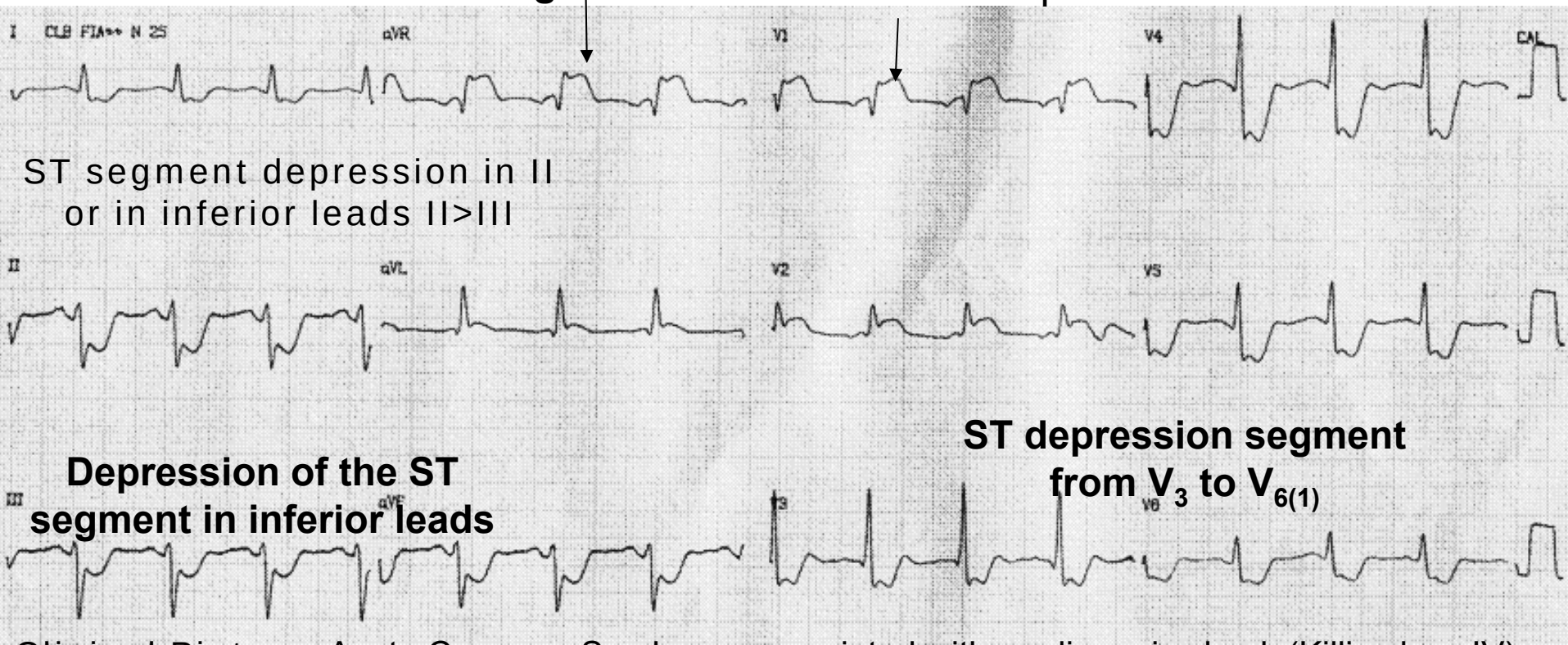
LMCA OCCLUSION ECG CRITERIA

- 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
- Widespread depression of ST segment maximally in leads V4- V_6 > ST with inverted T waves segment elevation in V_1
- Diffuse ST segment depression in the inferolateral leads
- Eventually observation of RBBB, LAFB and/or LSFB.





ST segment elevation in aVR > V_1 Raimundo´s case



ST segment depression in II or in inferior leads II>III

Depression of the ST segment in inferior leads

ST depression segment from V_3 to $V_{6(1)}$

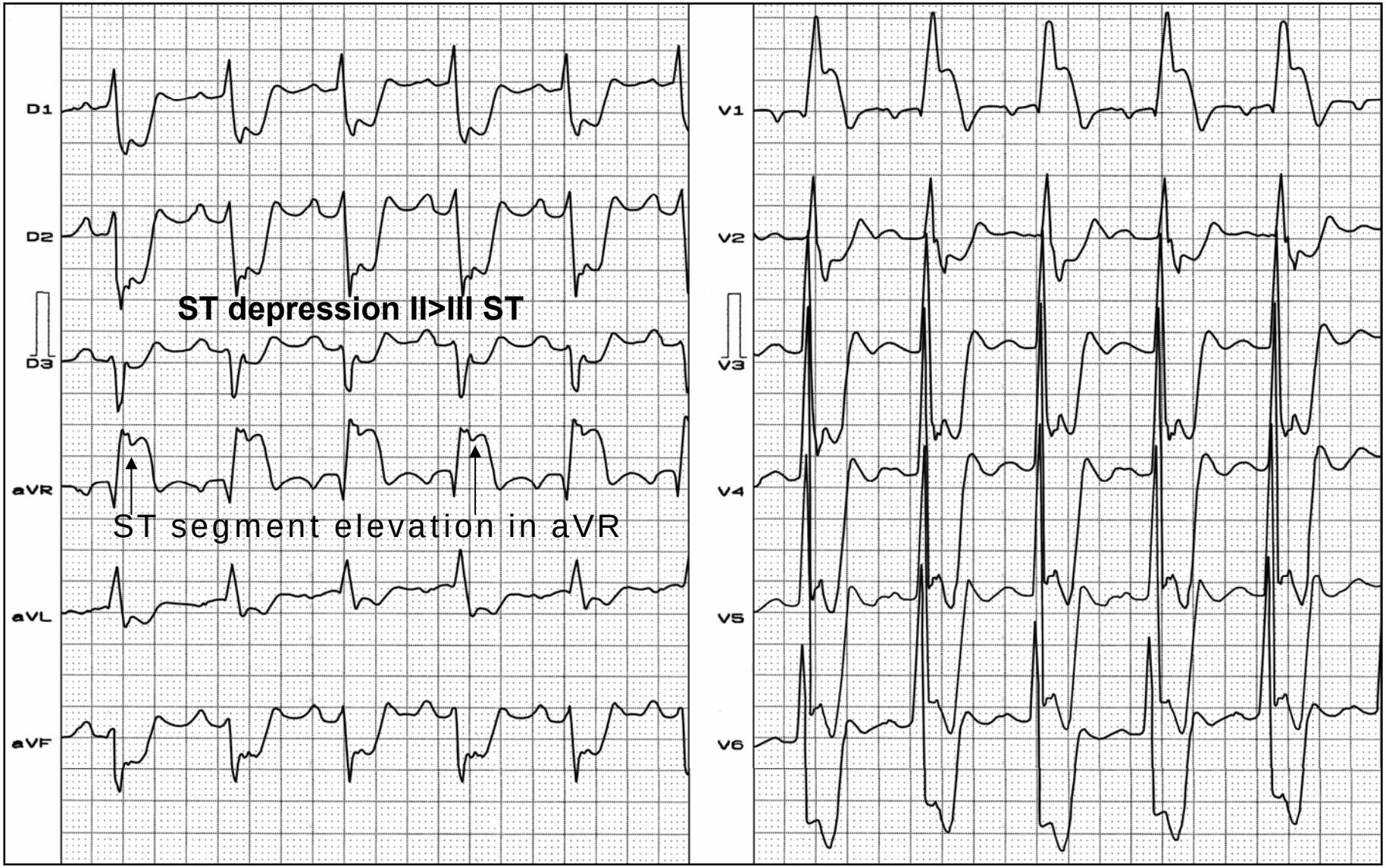
Clinical Picture: Acute Coronary Syndrome associated with cardiogenic shock (Killip class IV) consequence of total occlusion of LMCA.(2)

Primary Angioplasty was performed, with immediately hemodynamic stabilization.

Global ischemia ECG, pattern (wide-spread ST depression and/or T waves inversion) predicted an unfavorable outcome, when compared to other ECG patterns in patients with ACS.(3)

1. **Nikus KC, Eskola MJ. Electrocardiogram patterns in acute left main coronary artery occlusion. J Electrocardiol. 2008 Nov-Dec;41:626-629.**
2. **Nikus KC. Acute total occlusion of the left main coronary artery with emphasis on electrocardiographic manifestations. Timely Top Med Cardiovasc Dis. 2007 Aug 1;11:E22.**
3. **Nikus KC, Sclarovsky S, Huhtala H, et al. Electrocardiographic presentation of global ischemia in acute coronary syndrome predicts poor outcome. Ann Med. 2011 Jun 17. [Epub ahead of print]**

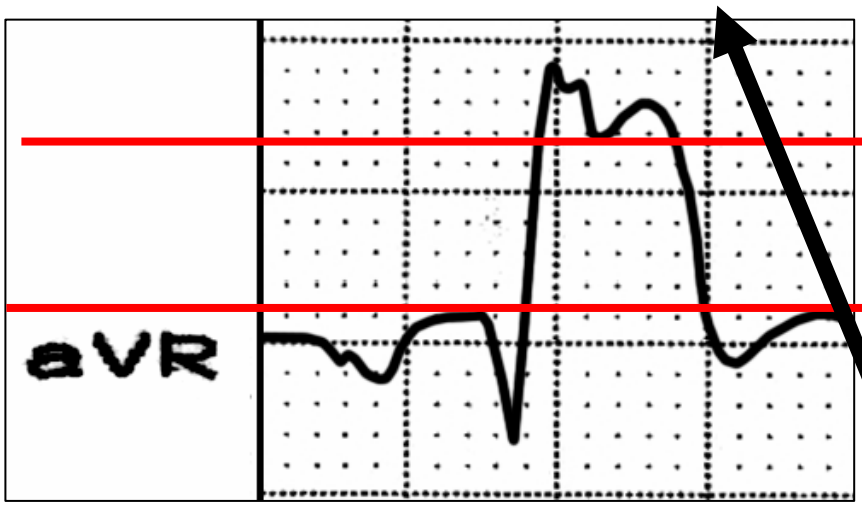
LMCA Occlusion complicated with Complete RBBB.

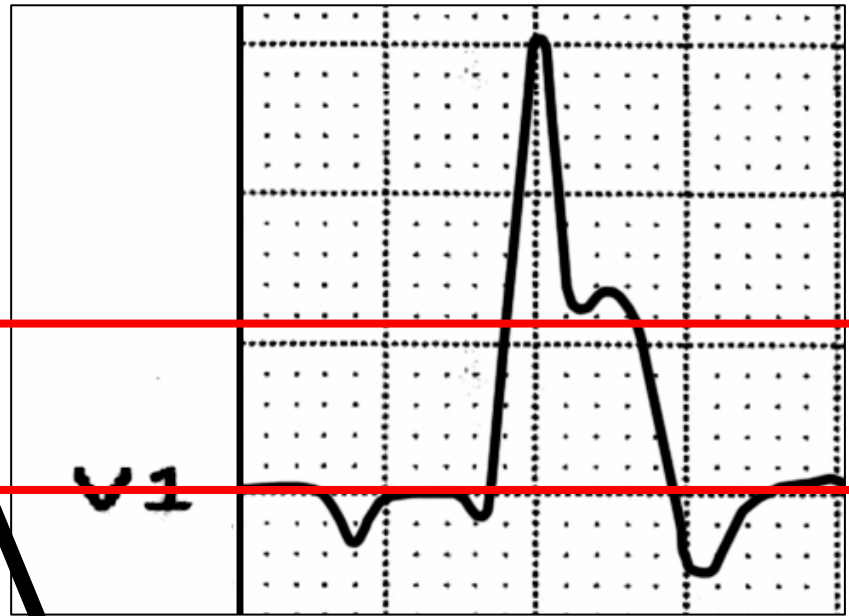


ST segment elevation in aVR and V_1 (aVR > V_1.). ST depression II>III ST depression segment from V_2 to V_6.

ST segment elevation in aVR > V_1

The ST injury vector is directed to upward and rightward, pointing to aVR lead (RVOT)

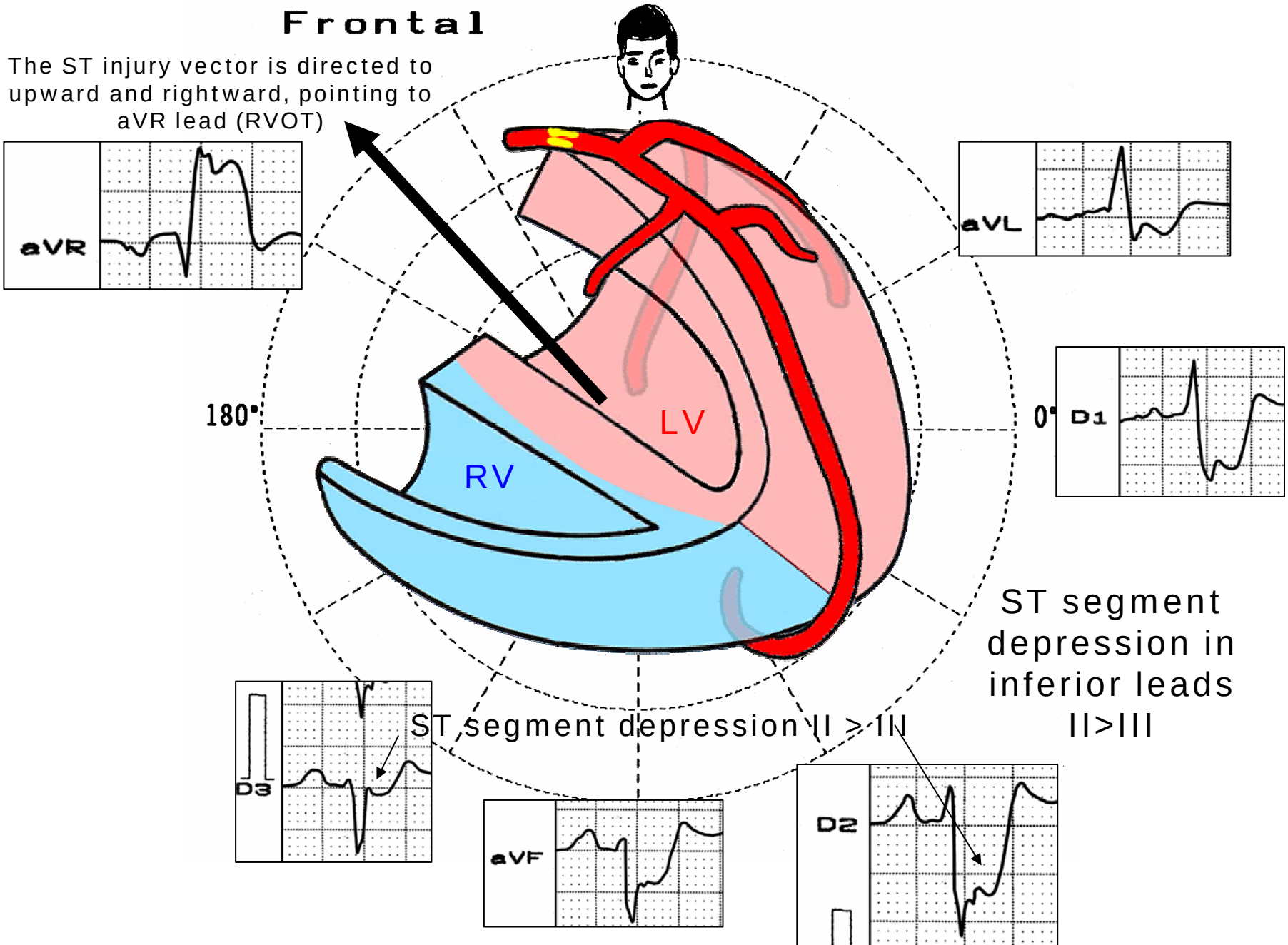




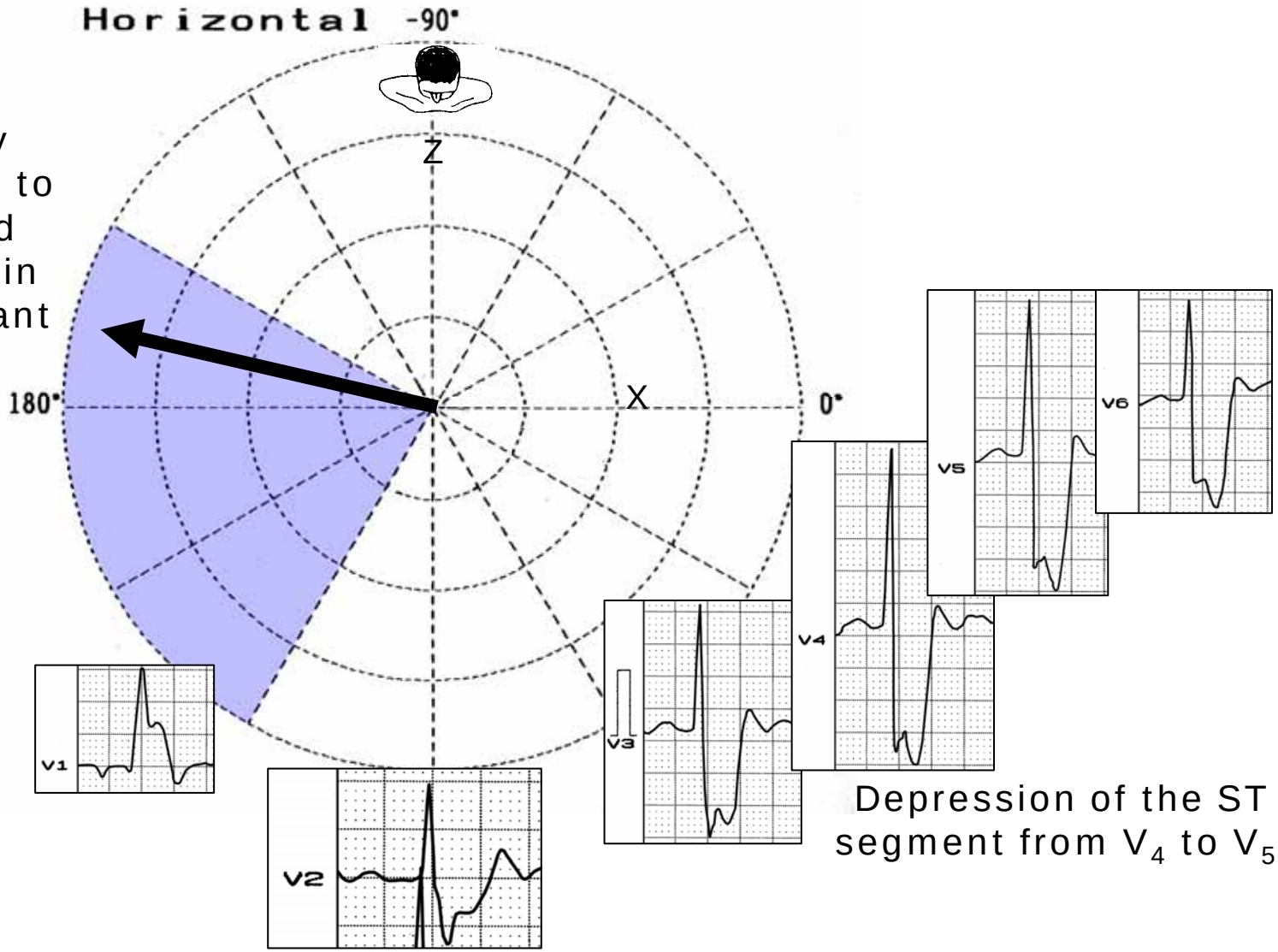
ST segment elevation = 6mm

ST segment elevation = 5,5mm

QRS AXIS LOCATED IN RIGHT SUPERIOR QUADRANT



The ST injury vector pointing to rightward and backward but in positive quadrant of v_1

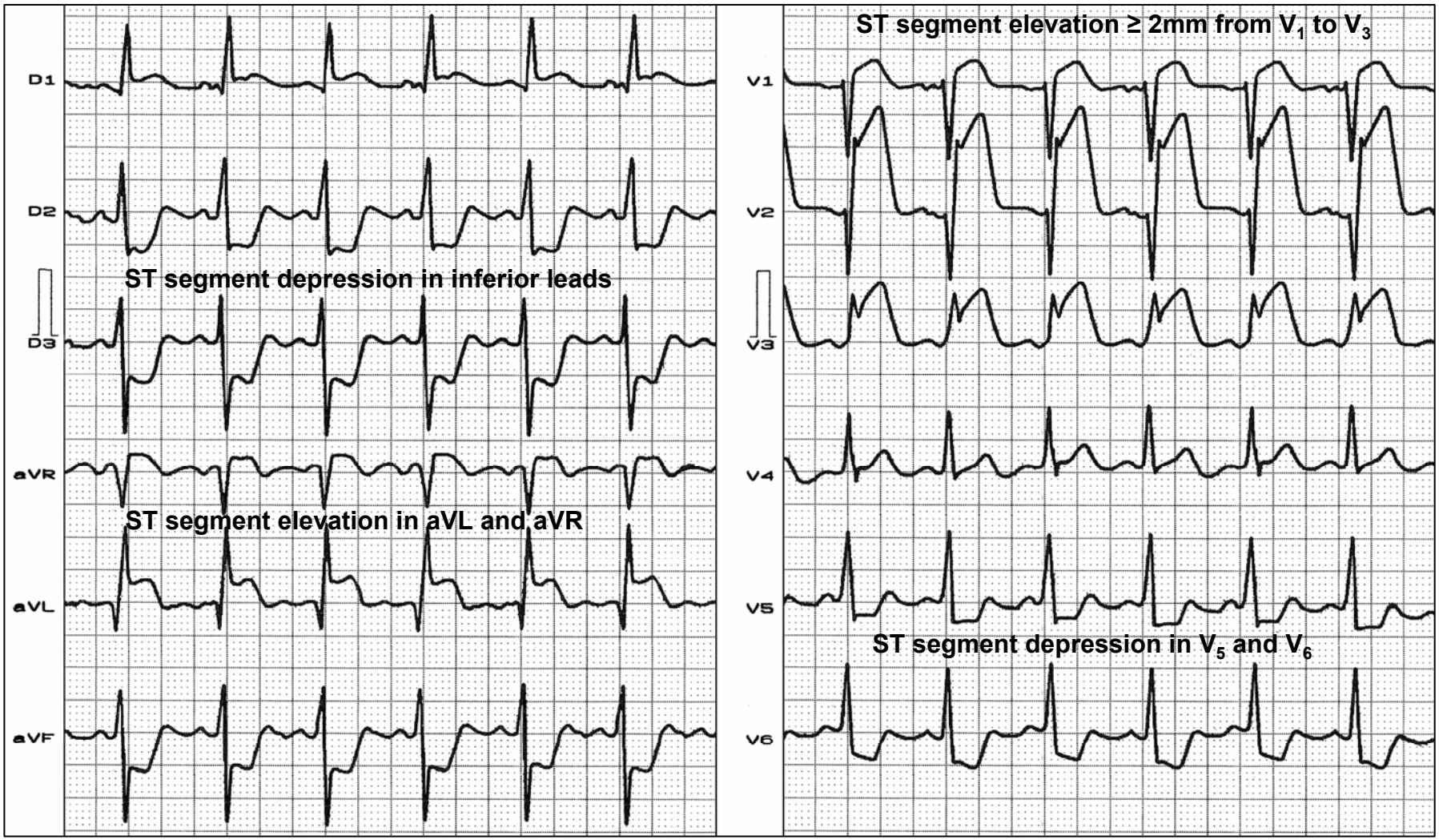


Depression of the ST segment from V_4 to V_5.

= Positive quadrant of V_1. The ST lesion vector is inside of positive quadrant of V_1 consequently ST segment elevation is present.

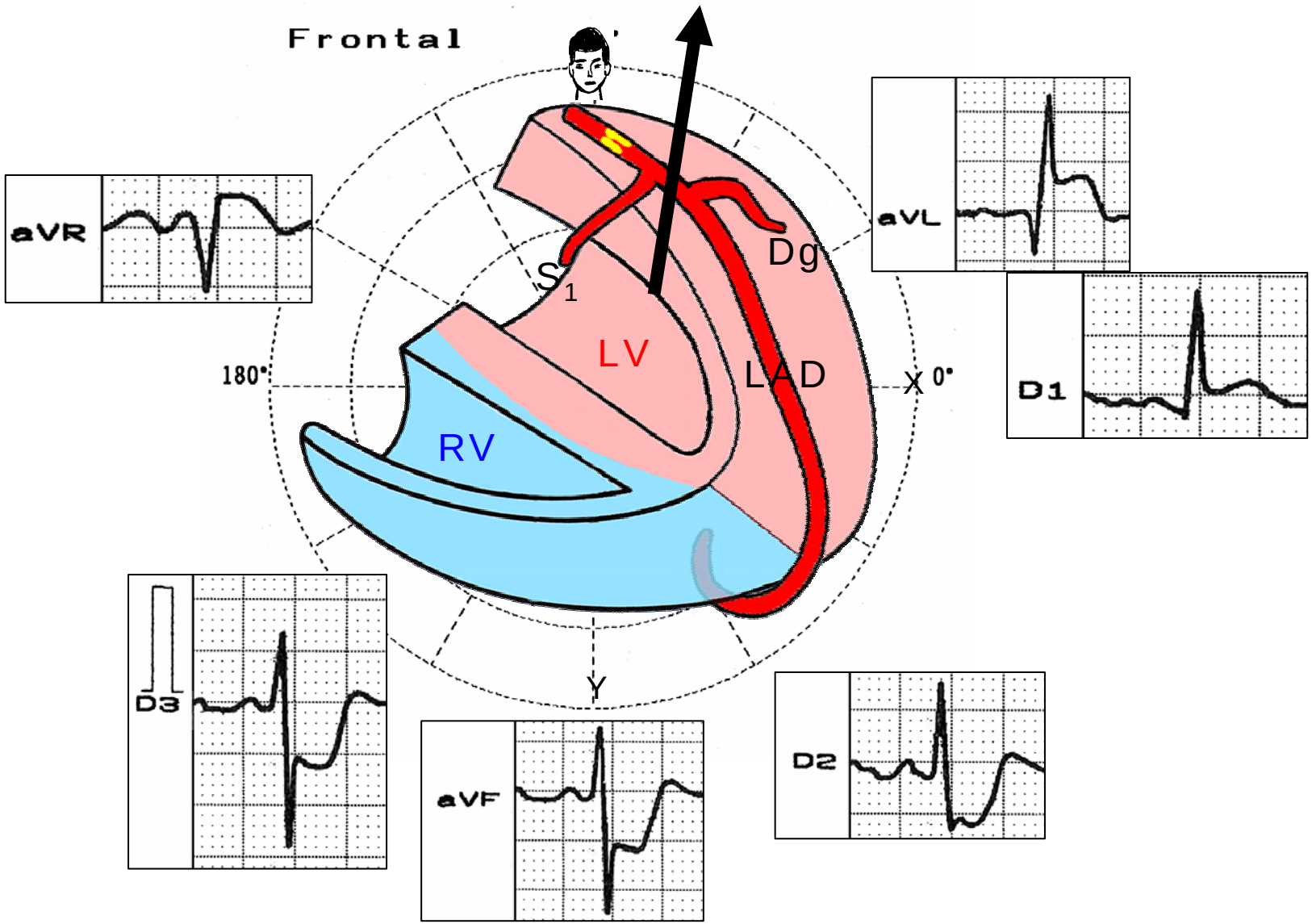
LEFT ANTERIOR DESCENDING ARTERY (LAD) OCCLUSION BEFORE FIRST SEPTAL PERFORATOR(S_1): PROXIMAL LAD OCCLUSION

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



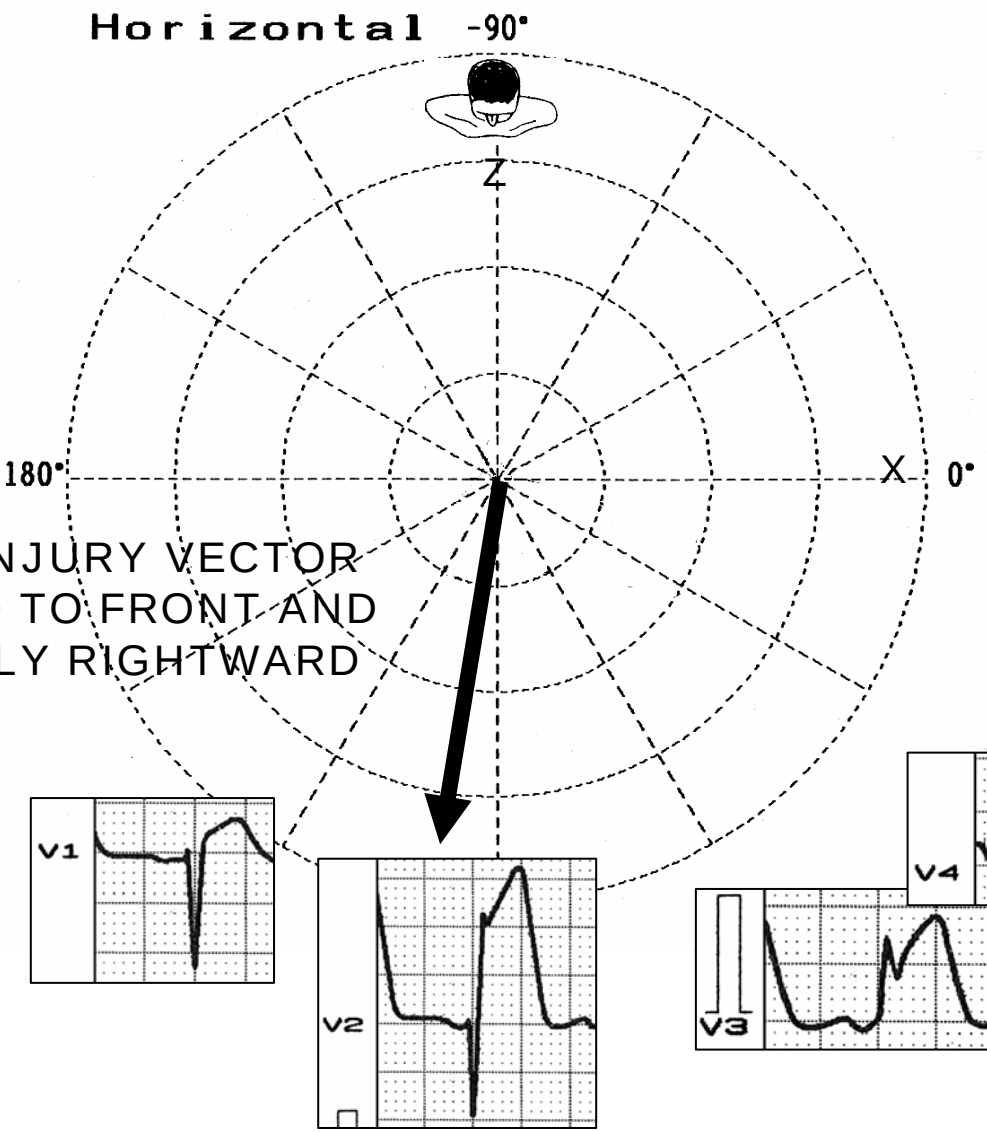
Why we observe this pattern?

THE ST INJURY VECTOR POINTING TO UP



THE ST INJURY VECTOR 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 injury vector directed to front). ST segment depression in V_5 and V_6 or isoelectric. Eventually CRBBB and/or LAFB and/or LSFB.



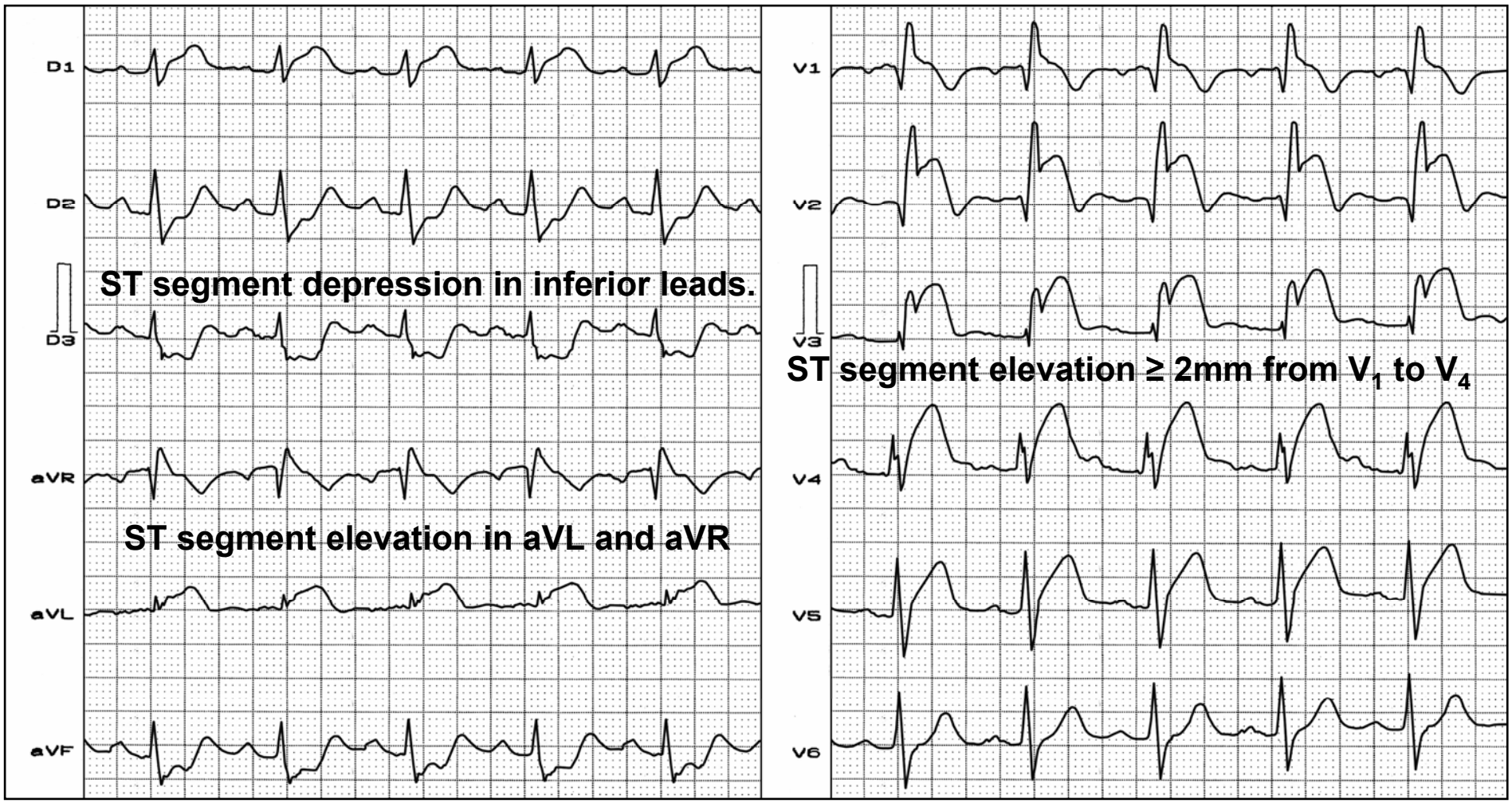
ST segment depression in V_5 and V_6 or isoelectric.

THE ST INJURY VECTOR DIRECTED TO FRONT AND MINIMALLY RIGHTWARD

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.

qR pattern



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

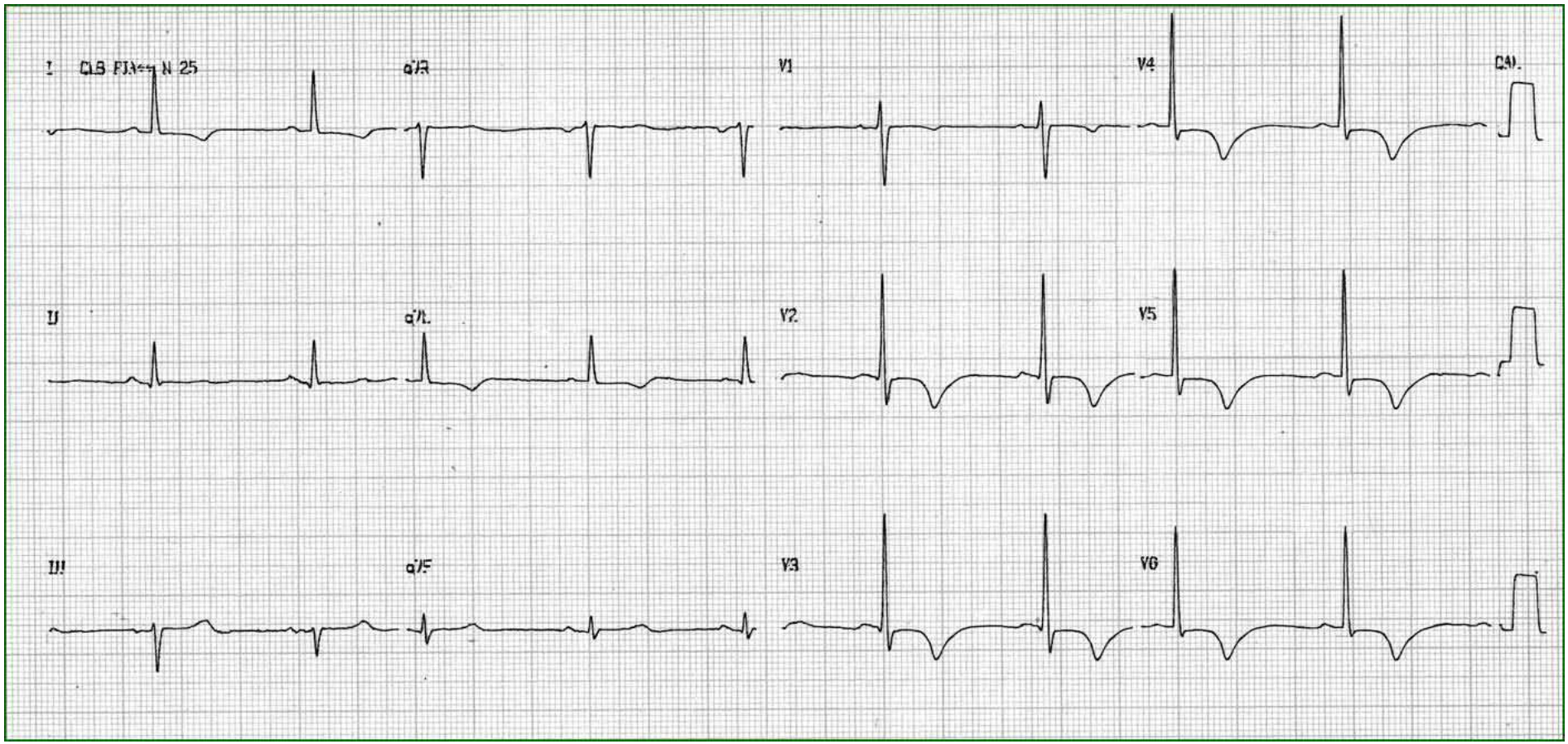
Wellens' syndrome is a clinical-electrocardiographic entity.

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

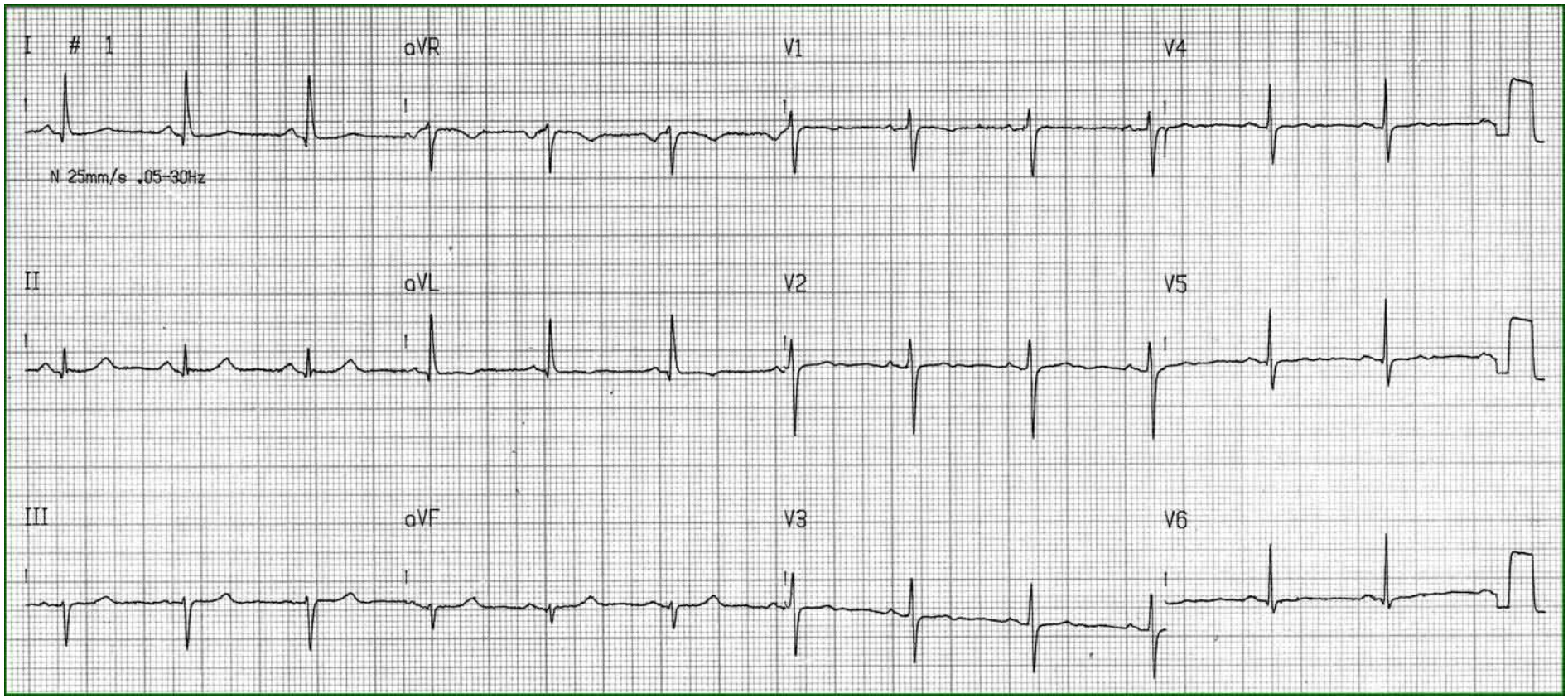
Sensitivity and specificity for significant (≥ 70%) stenosis of the LAD artery was found to be 69% and 89% respectively with positive predictive value 86%.[2]

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.

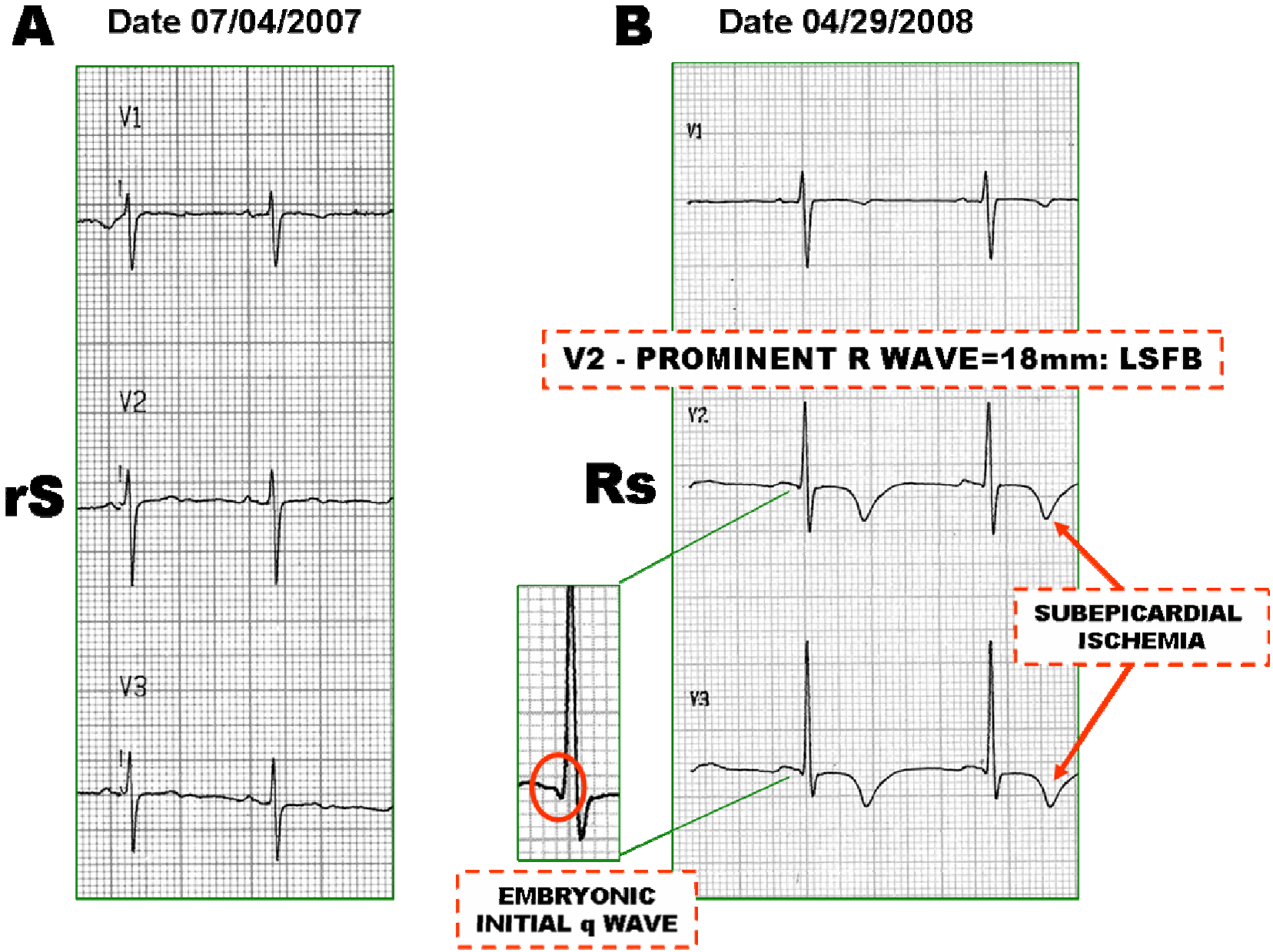


ECG performed upon arrival to the Emergency Department (04/29/2008), and while having chest pain. Deep negative and broad-based T-wave inversions in precordial leads from V_2 through V_6, with high voltage R wave in V_2 (R=18mm). Initial small q waves were observed in V_2-V_3. Left septal initial q waves 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.**

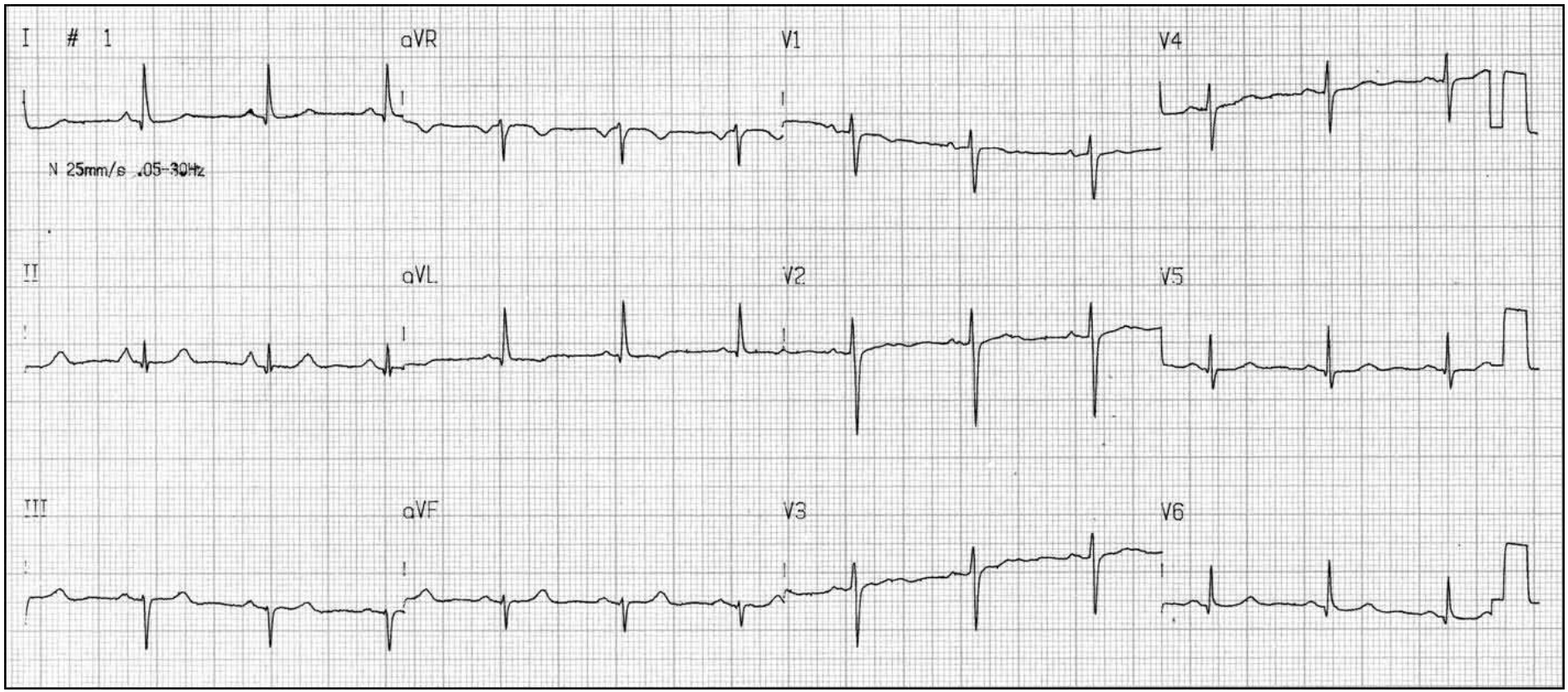


ECGs performed one year before clinical manifestation (07/04/2007). There are not subepicardial T wave ischemic pattern and QRS complexes of the rS type in V_2.
Initial q wave are observed in left leads I, aVL, V_5 and V_6.



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

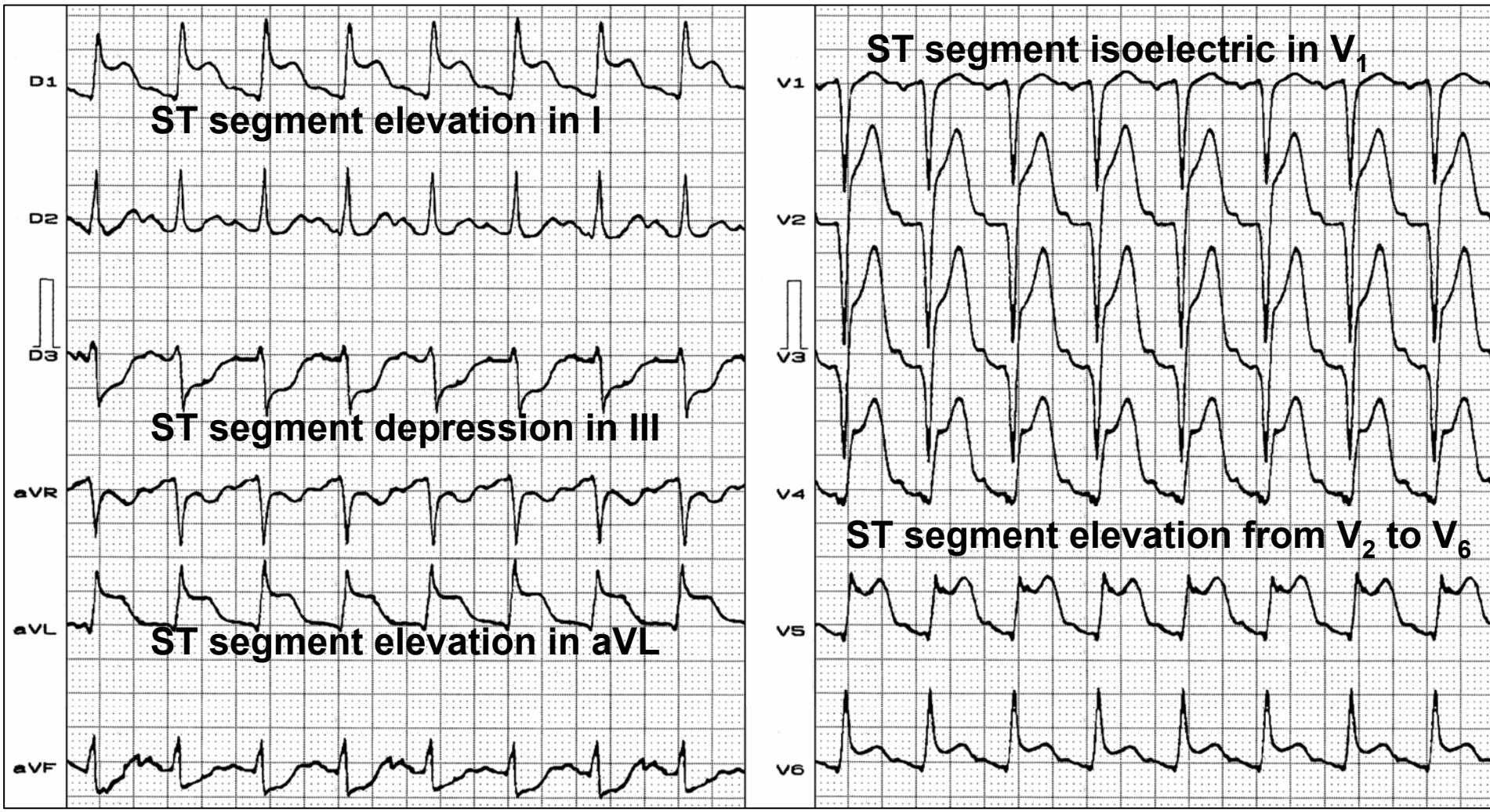
B) The same leads performed during the clinical manifestation.



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.

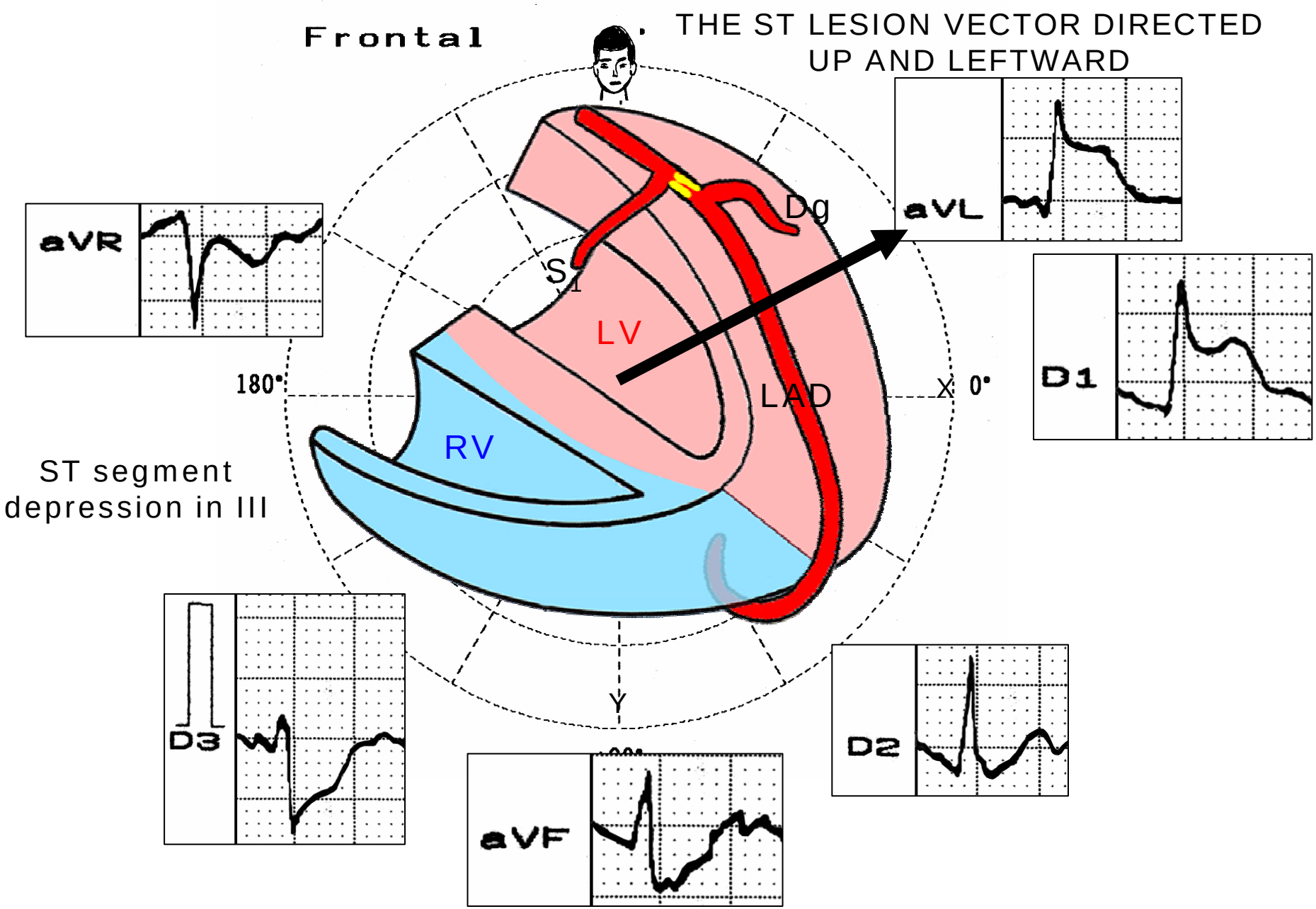
LEFT ANTERIOR DESCENDING ARTERY (LAD) OCCLUSION AFTER FIRST SEPTAL PERFORATOR AND BEFORE FIRST DIAGONAL BRANCH

AMI caused by occlusion of LAD after the first septal perforator and before the first diagonal branch

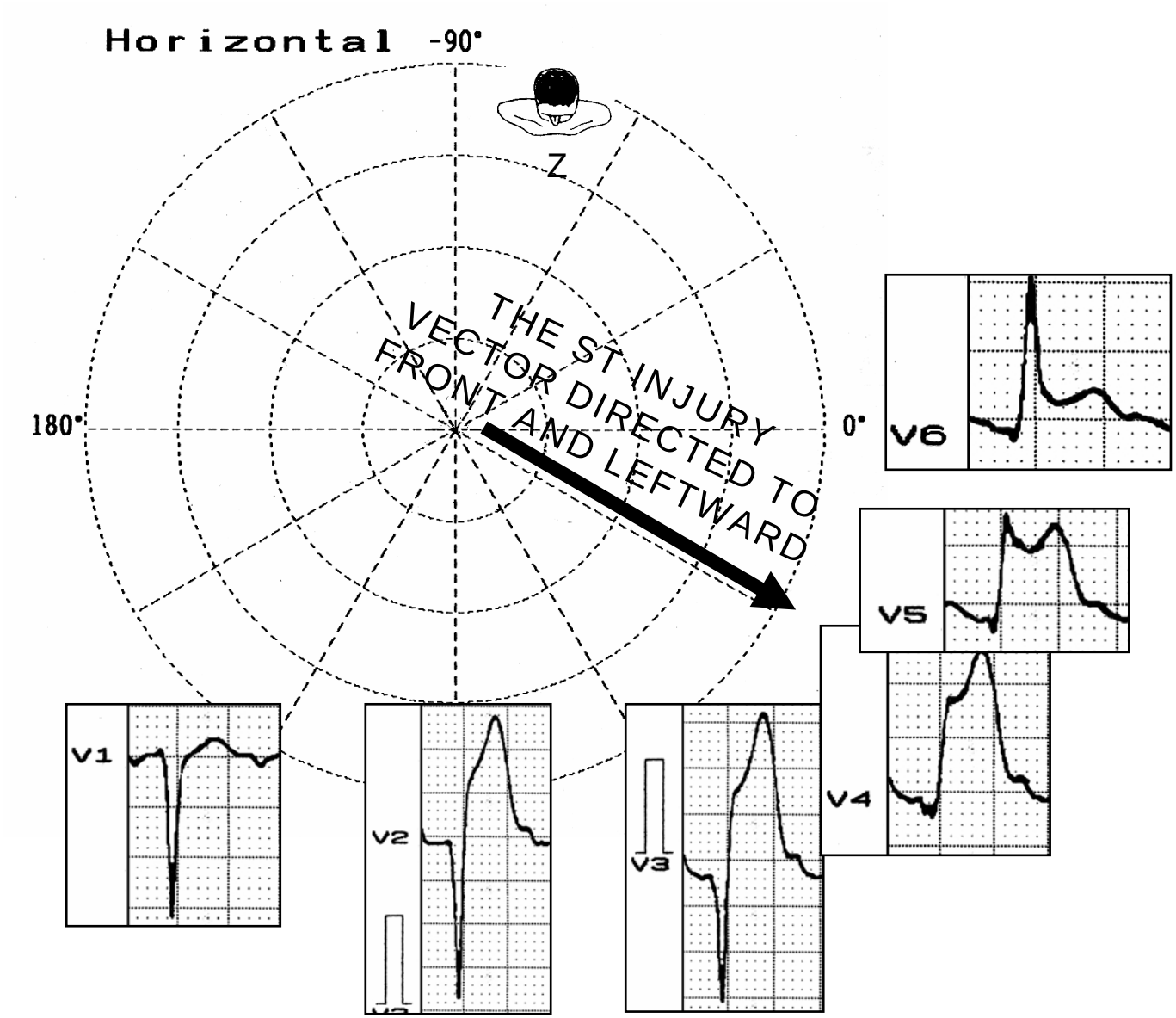


Why this pattern?

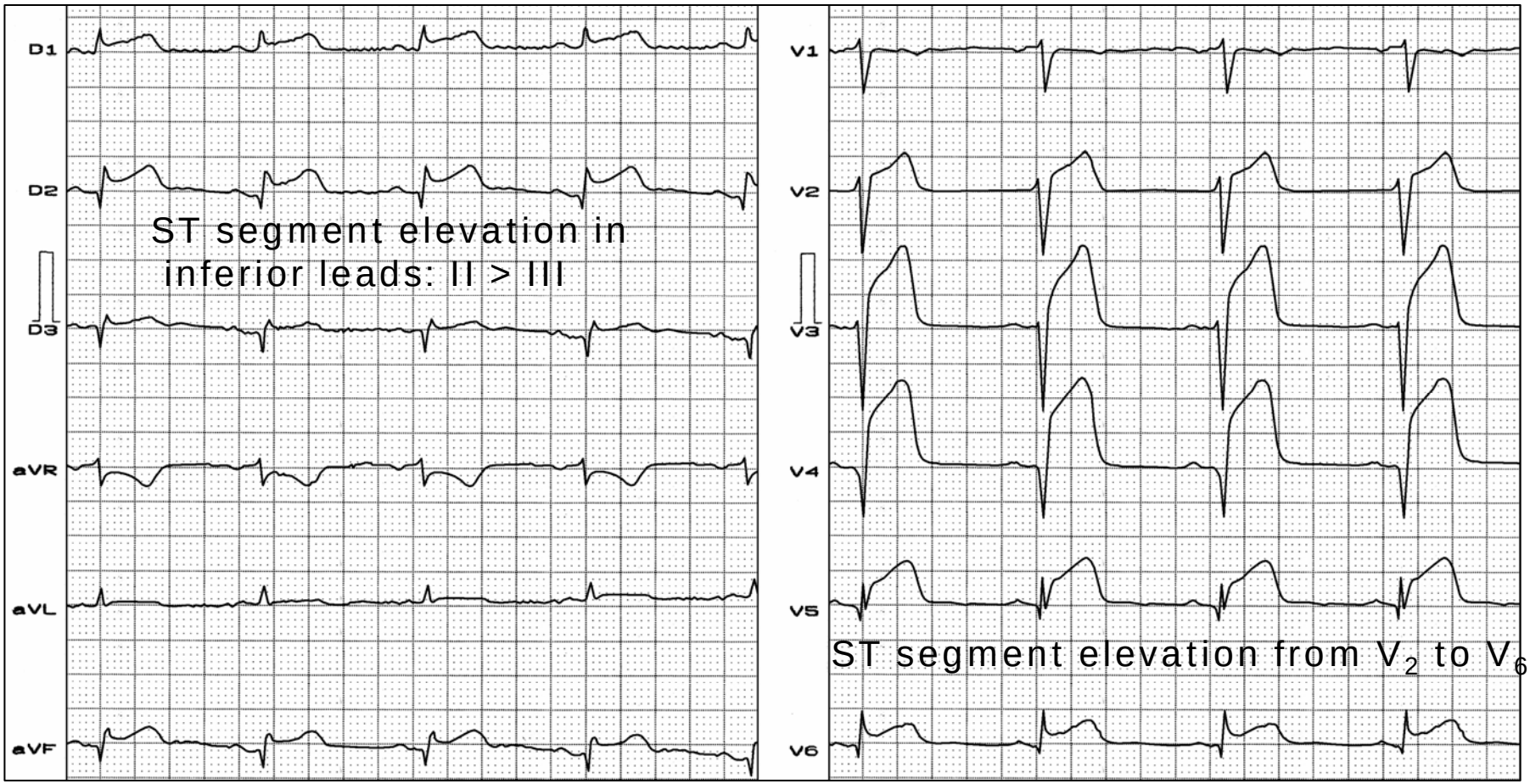
ST segment elevation in I and aVL. ST segment depression in III



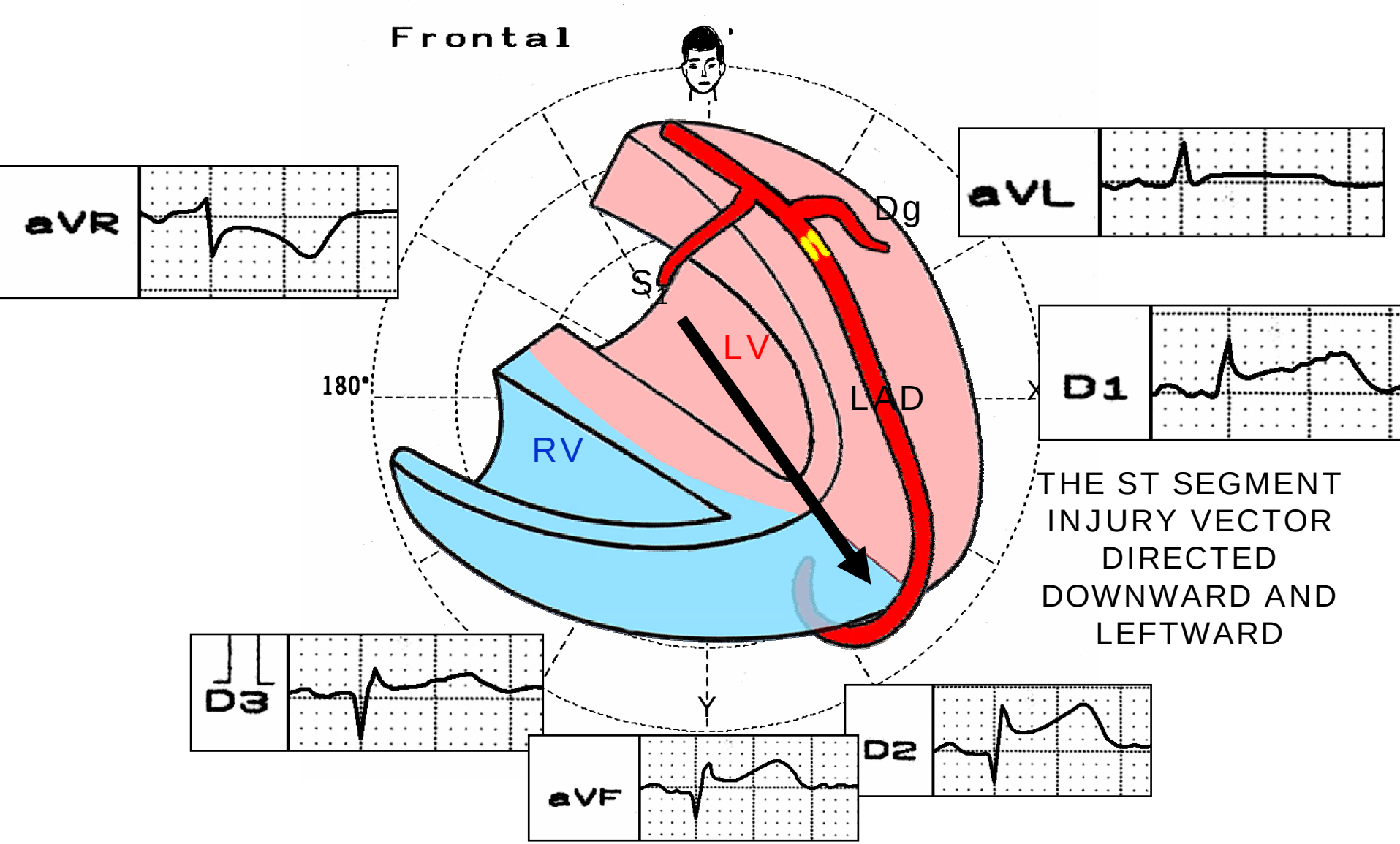
ST segment elevation from V_2 to V_6 and isoelectric in V_1



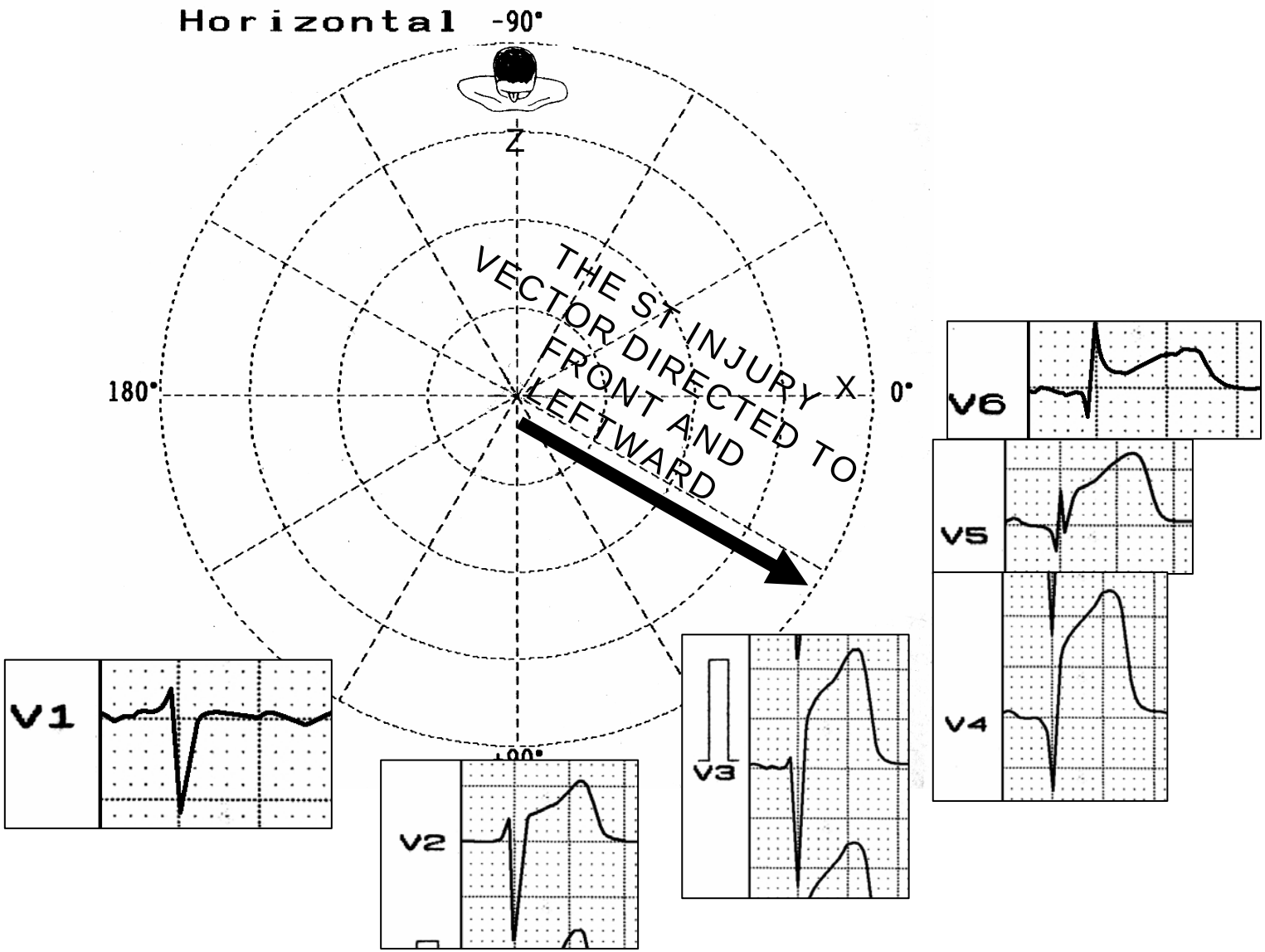
LEFT ANTERIOR DESCENDING ARTERY (LAD) OCCLUSION AFTER BOTH FIRST SEPTAL PERFORATOR AND FIRST DIAGONAL BRANCH (LAD DISTAL OBSTRUCTION)
(THE PRESENT CASE REPORT)



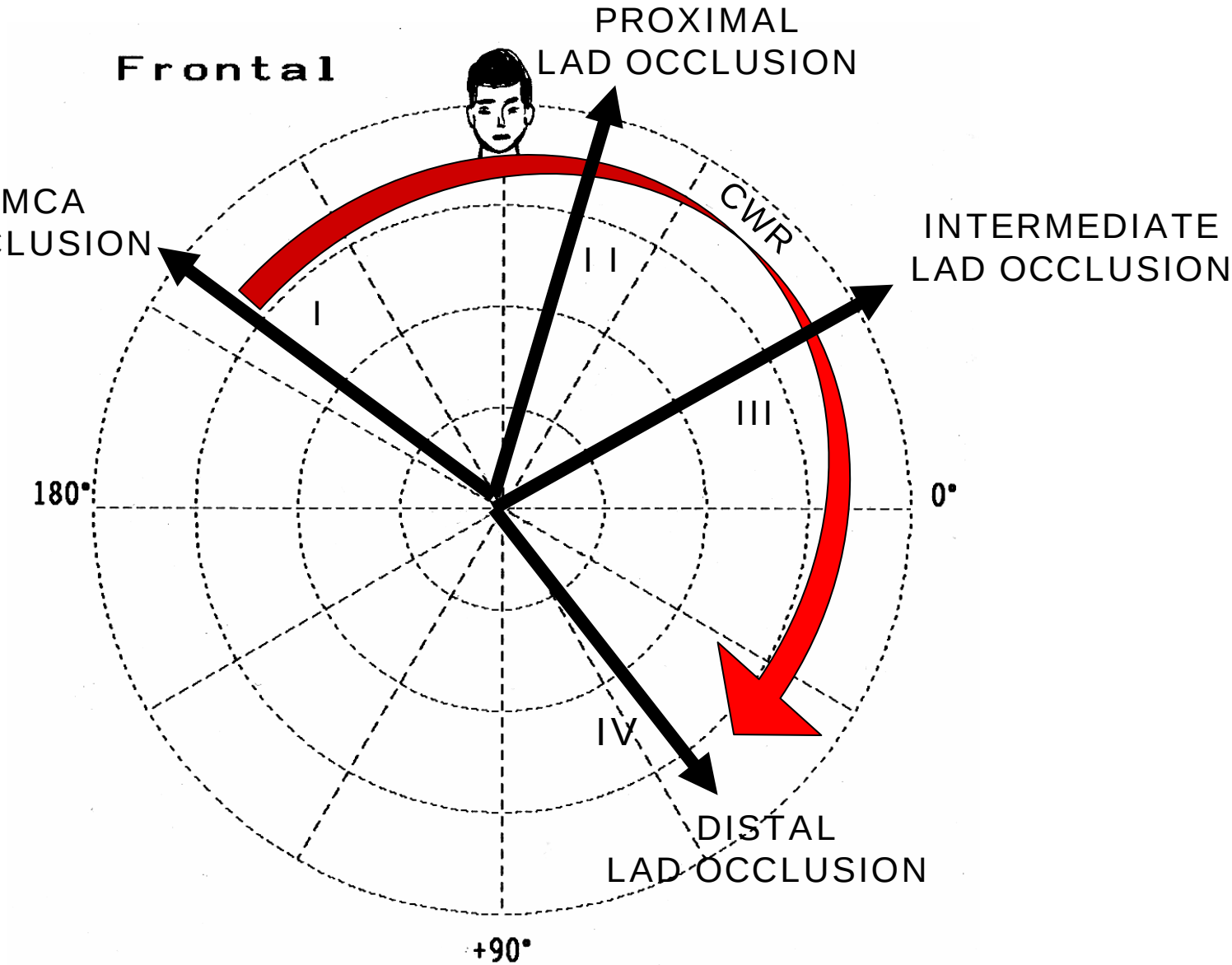
Why this pattern ?



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


Horizontal
-90°
Z
180°
X
0°
THE ST INJURY VECTOR DIRECTED TO FRONT AND LEFTWARD
V1
V2
V3
V4
V5
V6

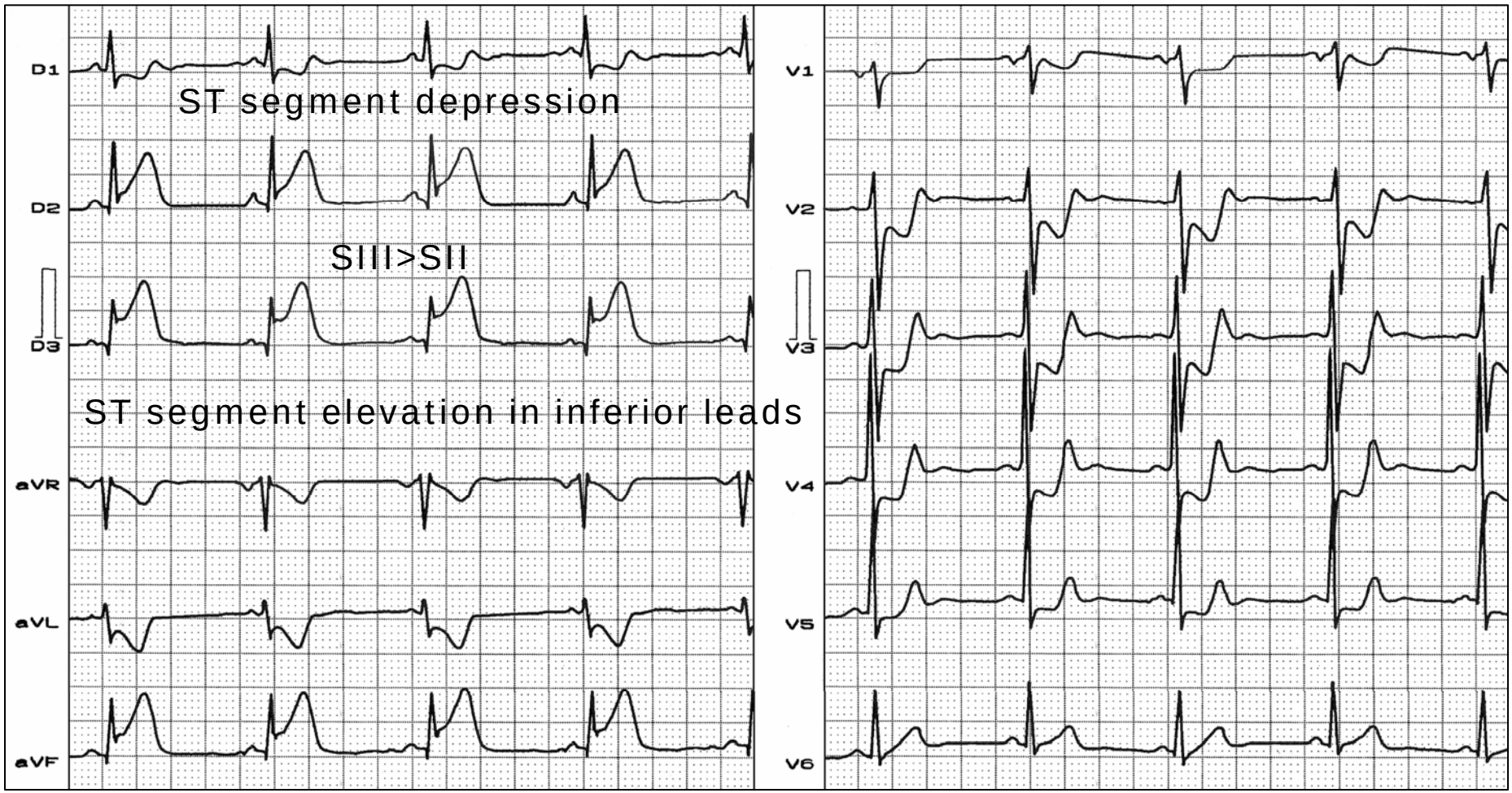
SUMMARY OF THE ST INJURY VECTOR DIRECTION ON FP



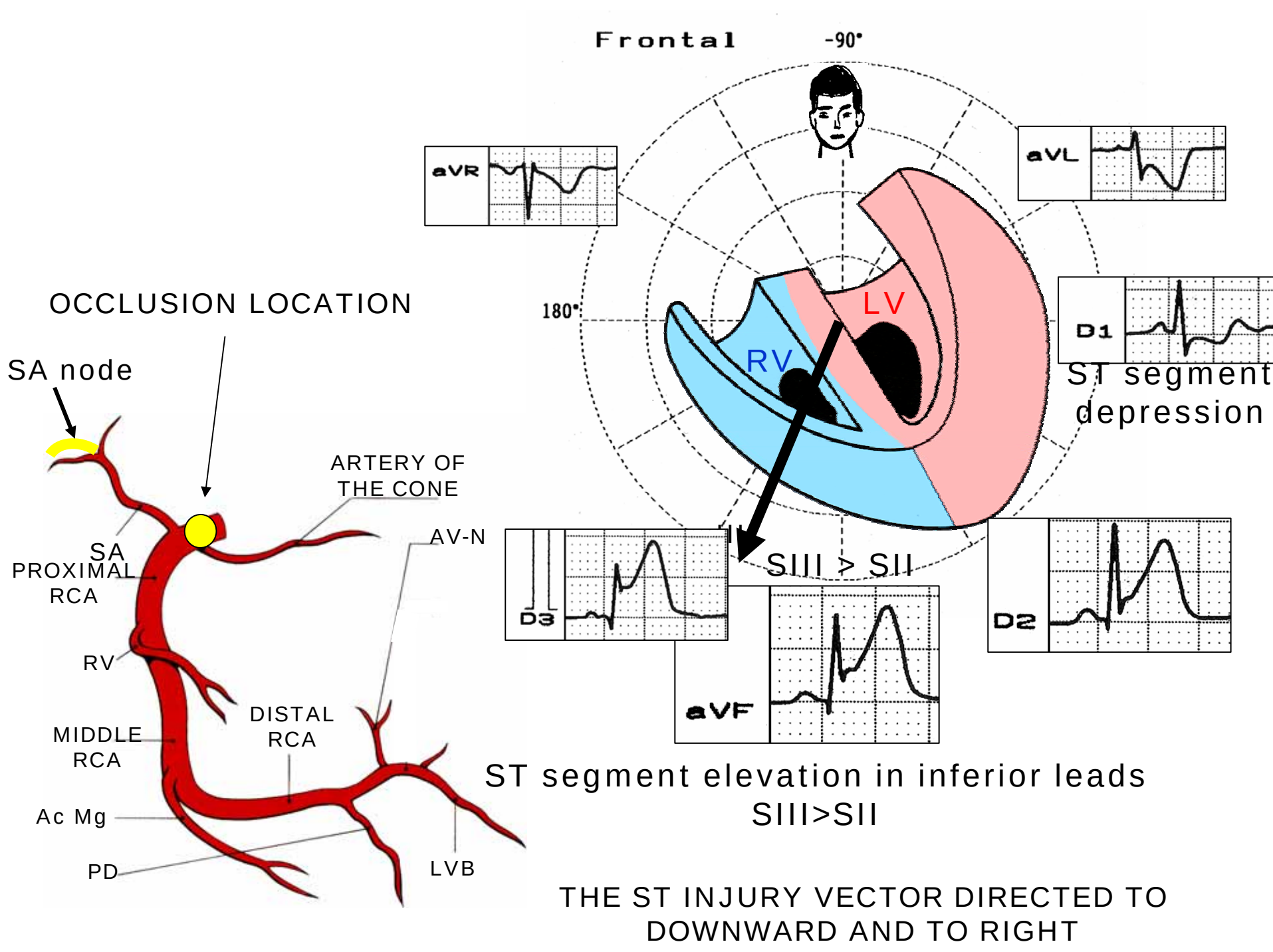
CWR: CLOCK WISE ROTATION

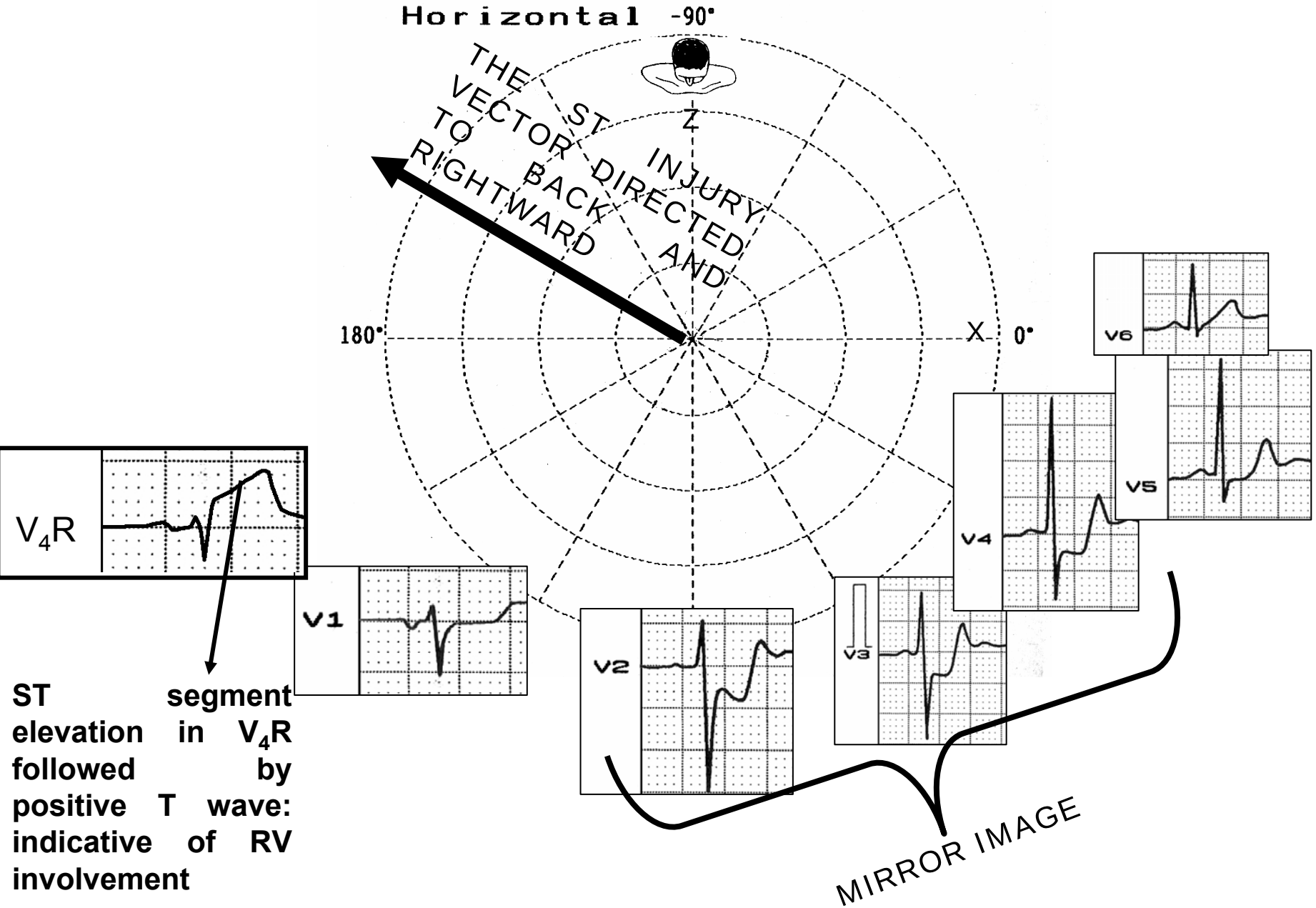
PROXIMAL OCCLUSION
RIGHT CORONARY ARTERY (RCA)

PROXIMAL RCA OCCLUSION



MIRROR IMAGE OF V_7, V_8 AND V_9


OCCLUSION LOCATION
SA node
ARTERY OF THE CONE
AV-N
SA PROXIMAL RCA
RV
DISTAL RCA
MIDDLE RCA
Ac Mg
PD
LVB
Frontal
-90°
180°
aVR
aVL
D1
D3
D2
aVF
LV
RV
SIII > SII
ST segment depression
ST segment elevation in inferior leads
SIII>SII
THE ST INJURY VECTOR DIRECTED TO DOWNWARD AND TO RIGHT



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

RIGHT VENTRICULAR ACUTE MI

Right Ventricle irrigation[1]

1. Inferior and posterior walls: The PDA of the RCA.
2. Lateral wall: A. Mg
3. Anterior wall: Conus artery of the RCA and the moderator branch artery form LAD

Proximal RCA occlusions result in larger RV infarctions[2]
LCX occlusion eventually RV infarction

The classic clinical triad of RV acute MI includes[3]

1) Distended neck veins
2) Clear lung fields
3) Hypotension

1. **Forman MB, Goodin J, Phelan B. Electrocardiographic changes associated with isolated right ventricular infarction. *J Am Coll Cardiol*. Sep 1984;4(3):640-643.**
2. **Giannitsis E, Potratz J, Wiegand U. Impact of early accelerated dose tissue plasminogen activator on in- hospital patency of the infarcted vessel in patients with acute right ventricular infarction. *Heart*. Jun 1997;77:512-516.**
3. **Mavric Z, Zaputovic L, Matana A. Prognostic significance of complete atrioventricular block in patients with acute inferior myocardial infarction with and without right ventricular involvement. *Am Heart J*. Apr 1990;119:823-828.**

ELECTROCARDIOGRAPHY

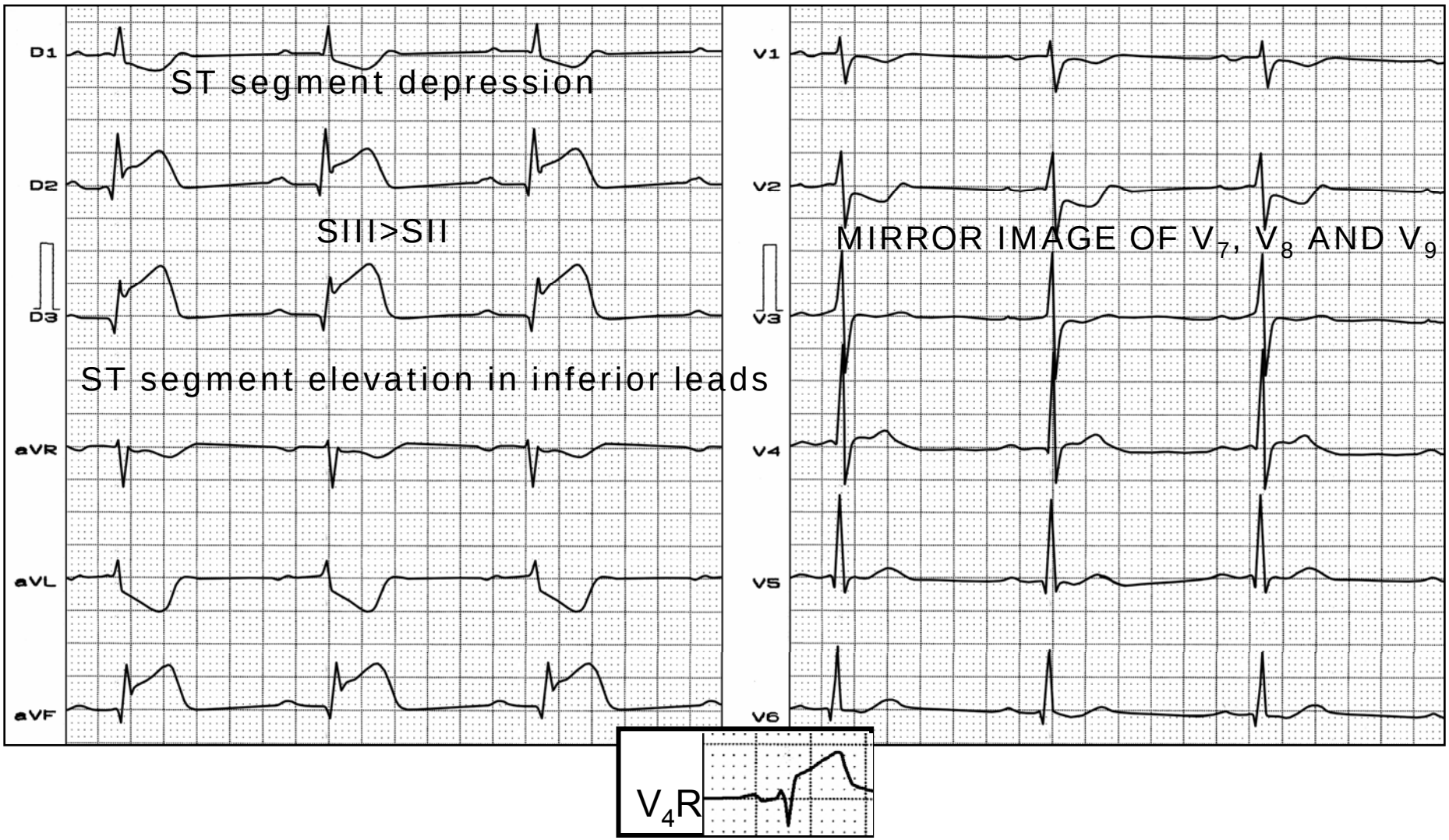
All patients with inferior wall MI should have a right-sided precordial leads.
ST-segment elevation in lead V_4R is the single most powerful predictor of RVMI, The ST-segment elevation is transient, disappearing in < 10 hours following its onset in half of patients. The following table demonstrates the sensitivity and specificity of > 1 mm of ST-segment elevation in V_1, V_3R, and V_4 R[1].

LEADS	SENSITIVITY(%)	SPECIFICITY(%)
V_1	28	92
V_3R	69	97
V_4R	93	95

Isolated RVMI is extremely rare and may be interpreted erroneously as LV anteroseptal infarction on ECG because of ST-segment elevation in leads V_1 -V_4[2;3].
The mean ST-segment lesion vector in RVMI usually is directed anteriorly and to the right: >100°.

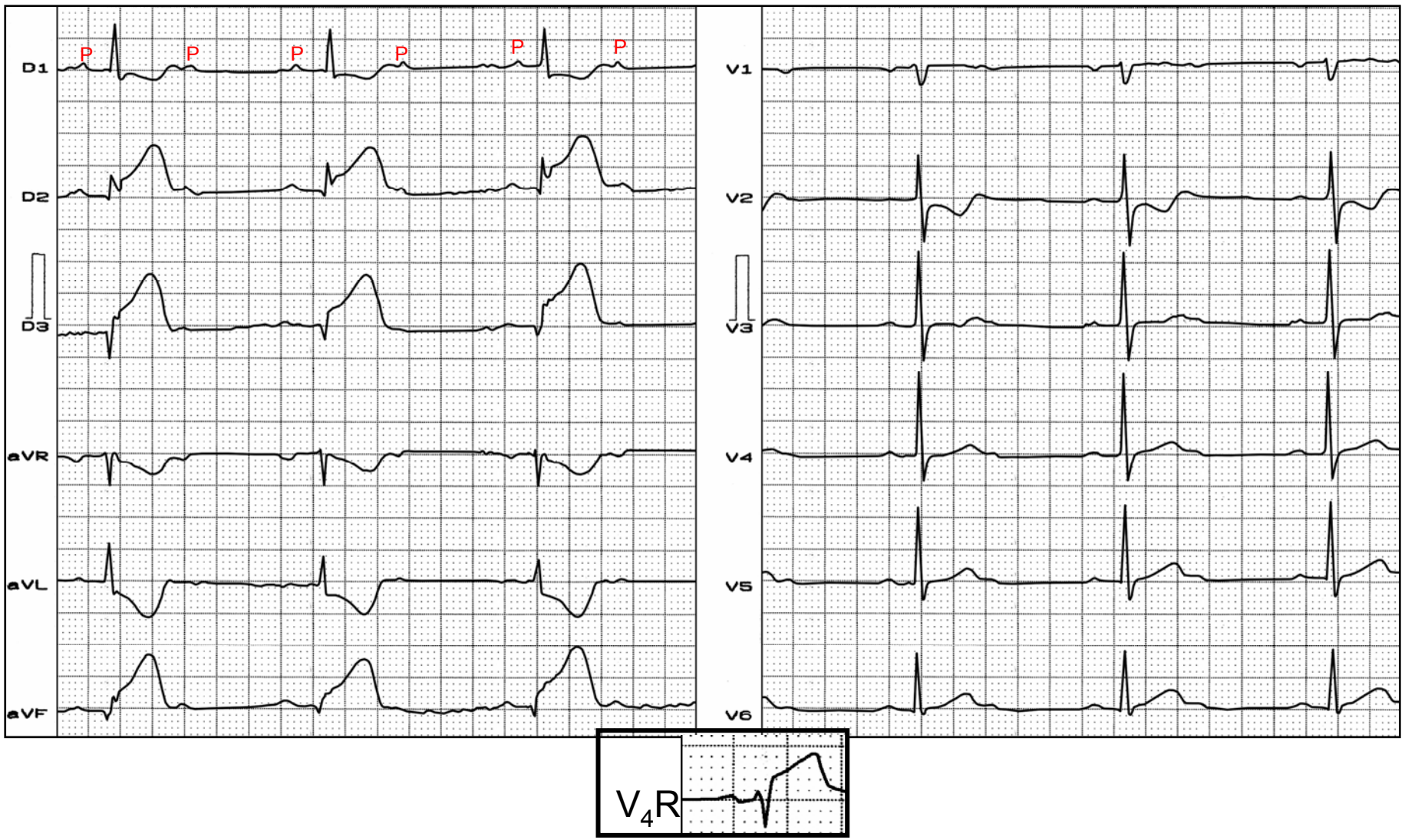
1. **Roth A, Miller HI, Kaluski E. Early thrombolytic therapy does not enhance the recovery of the right ventricle in patients with acute inferior myocardial infarction and predominant right ventricular involvement. *Cardiology*. 1990;77(1):40-9.**
2. **Schuler G, Hofmann M, Schwarz F. Effect of successful thrombolytic therapy on right ventricular function in acute inferior wall myocardial infarction. *Am J Cardiol*. Nov 1 1984;54:951-957.**
3. **Sharpe DN, Botvinick EH, Shames DM. The noninvasive diagnosis of right ventricular infarction. *Circulation*. Mar 1978;57:483-490.**

AMI consequence of proximal RCA occlusion complicated with sinus bradicardia, first-degree AV block and RV envolvement: ST segment elevation followed by positive T wave in V_4R

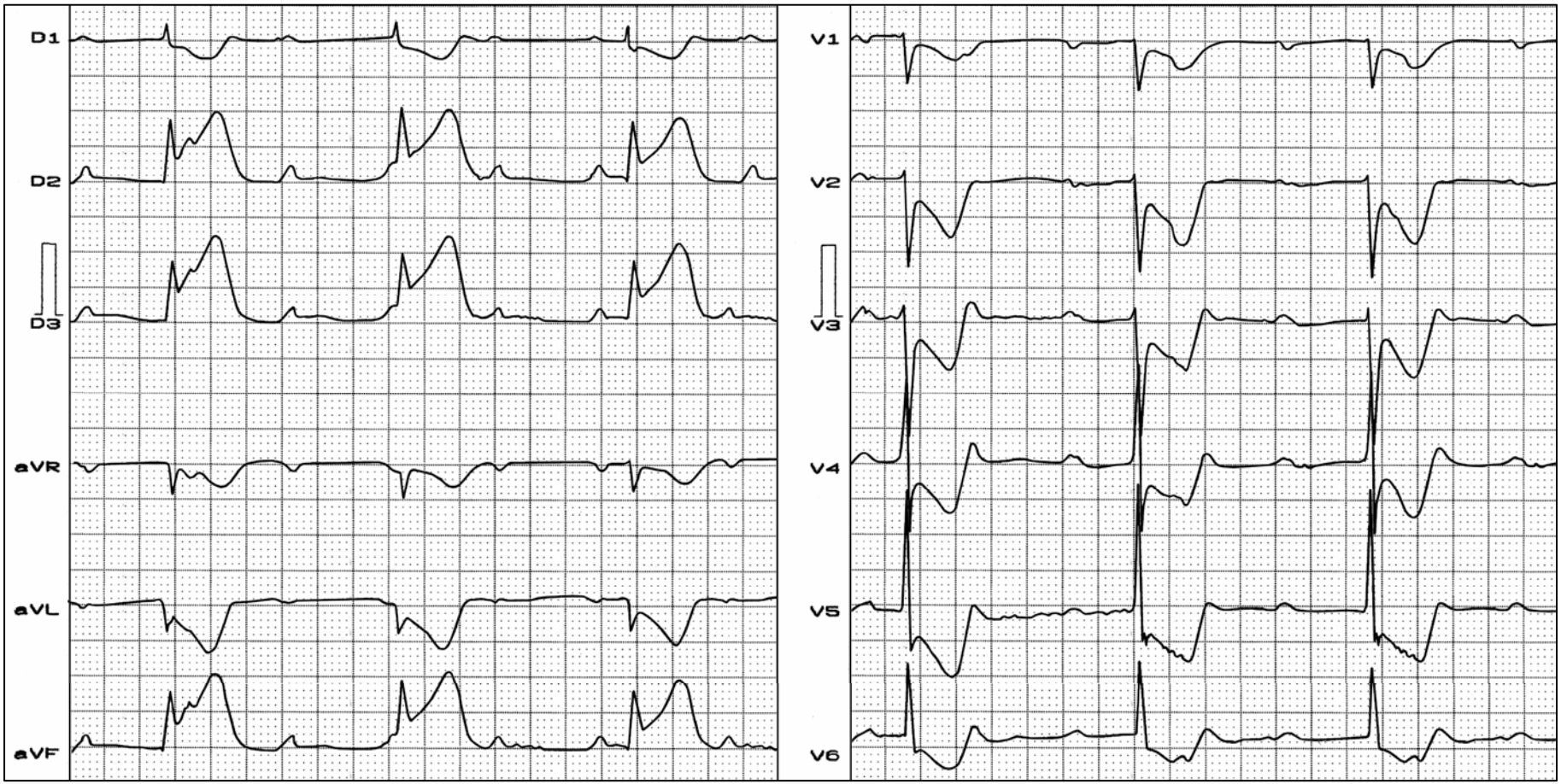


ST-segment elevation in lead V_4R is the single most powerful predictor of RVMI

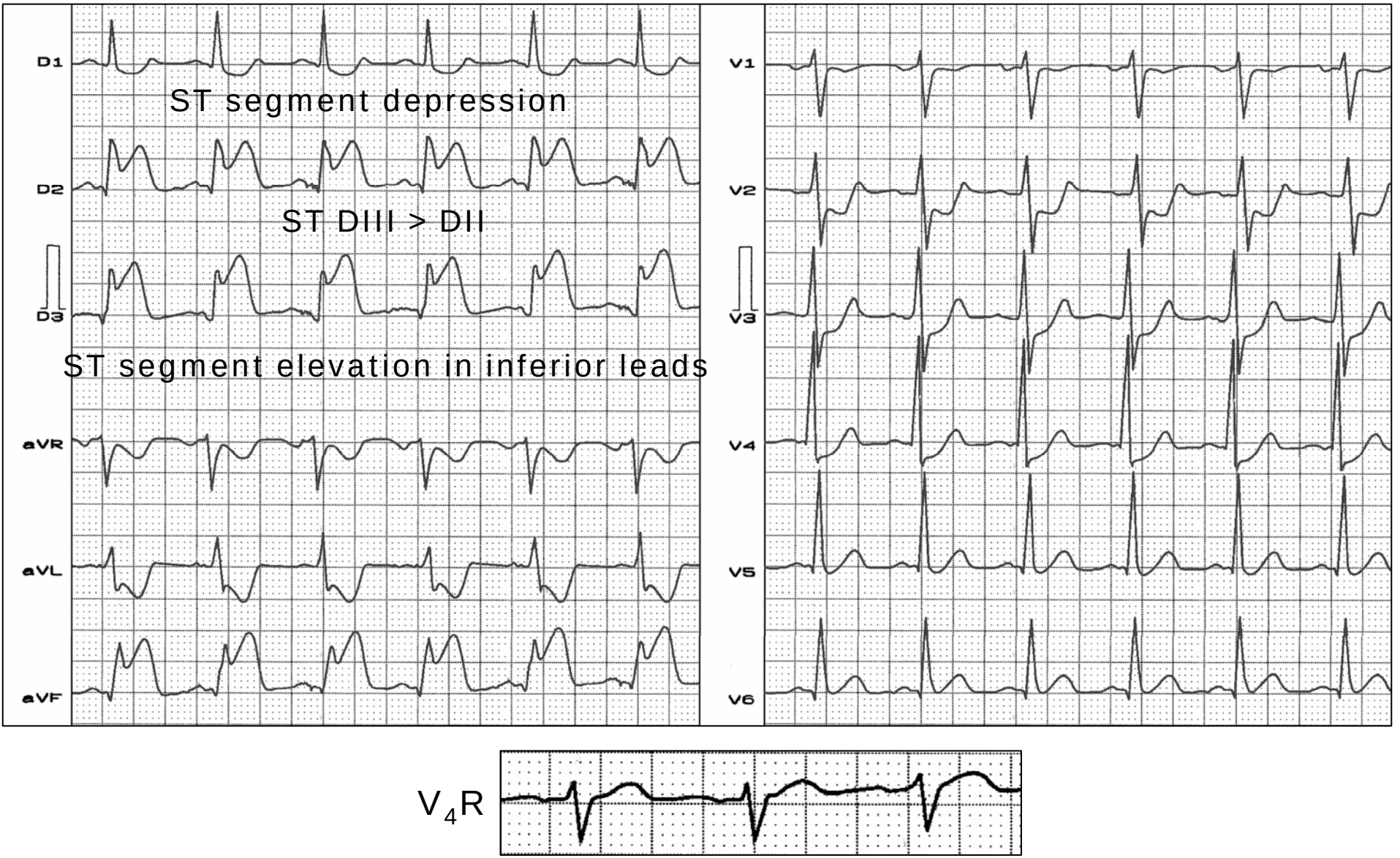
AMI consequence of proximal occlusion RCA complicated with 2:1 AV block and right ventricular envolvement: ST segment elevation in V4R followed by positive T wave



Third degree AV block consequence of AMI by obstruction of RCA. QRS complexes are narrow indicating suprahisian block.

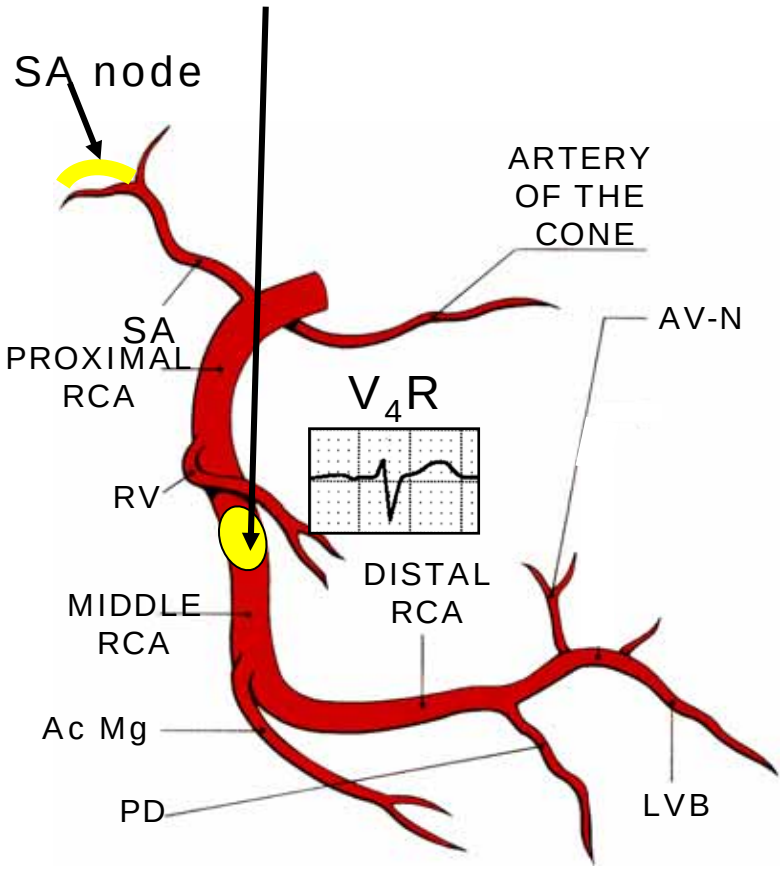


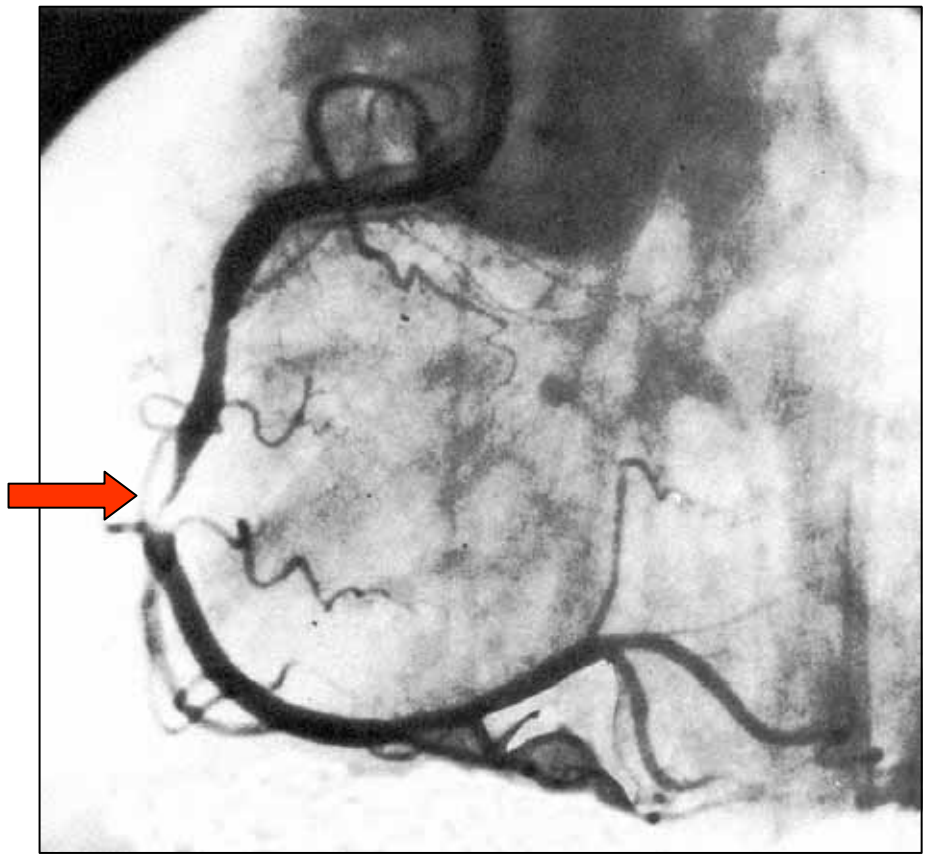
MIDDLE PORTION OCCLUSION
RIGHT CORONARY ARTERY (RCA)


D1
D2
D3
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
ST segment depression
ST DIII > DII
ST segment elevation in inferior leads

V_4R

OCCLUSION LOCATION

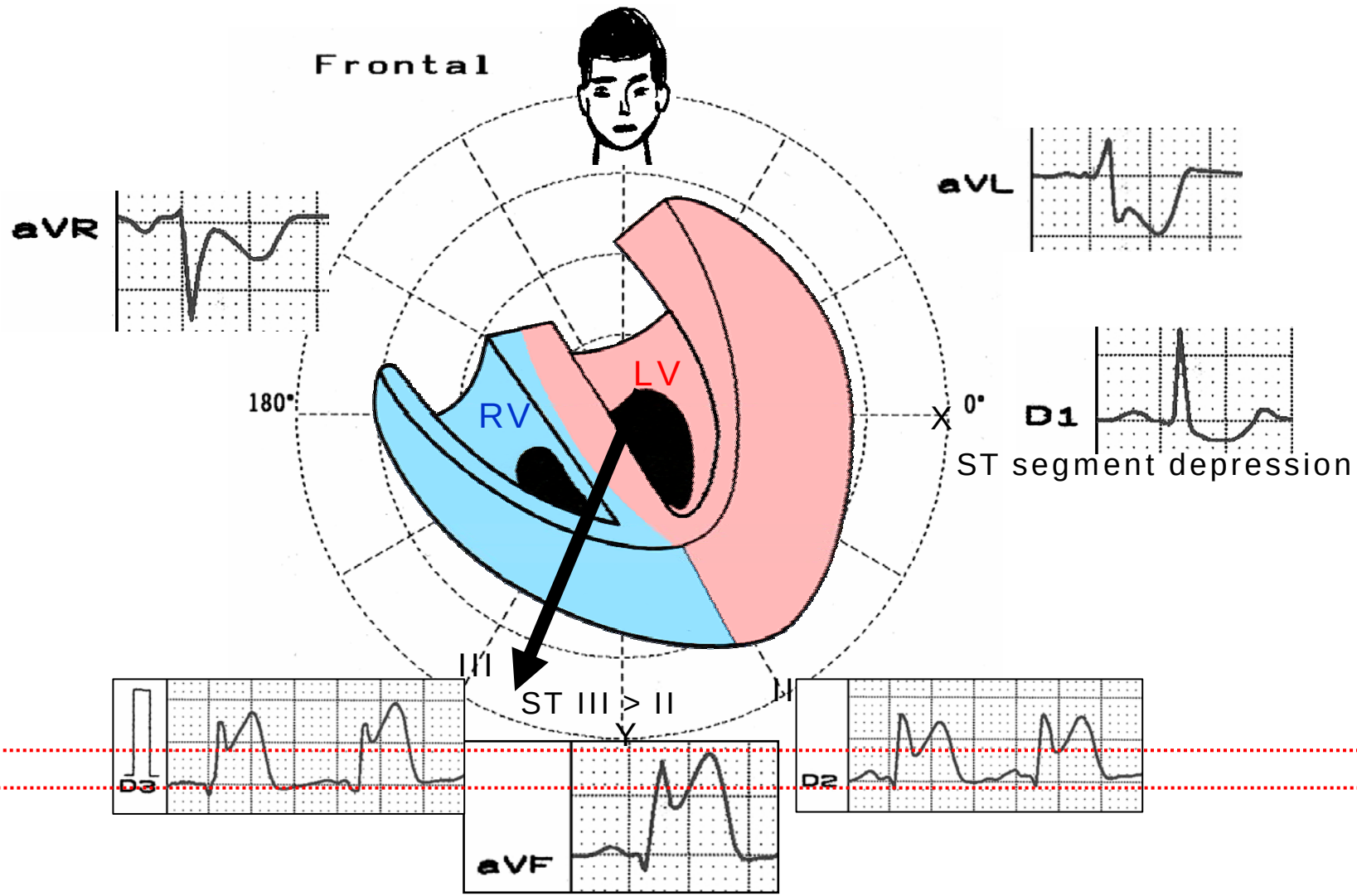




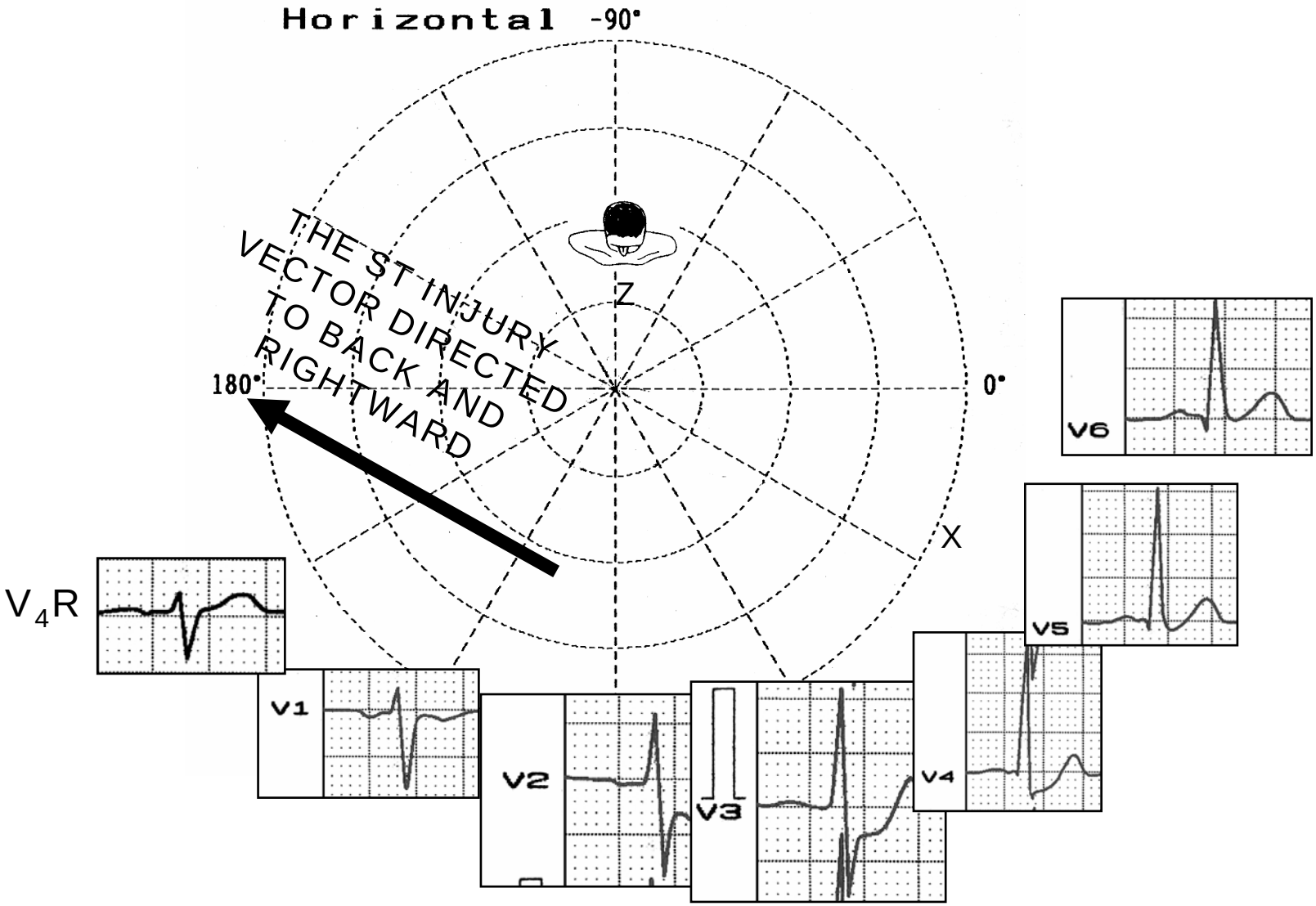
Cineangiography of the previous patient. The red arrow points out the total obstruction in the middle portion of the RCA. The accessory V_4R lead has a isoelectric ST segment, because the RCA obstruction is located distal related to RV artery (without RV Infarction).

ST segment elevation in inferior leads. III>II because the ST injury vector pointed to III

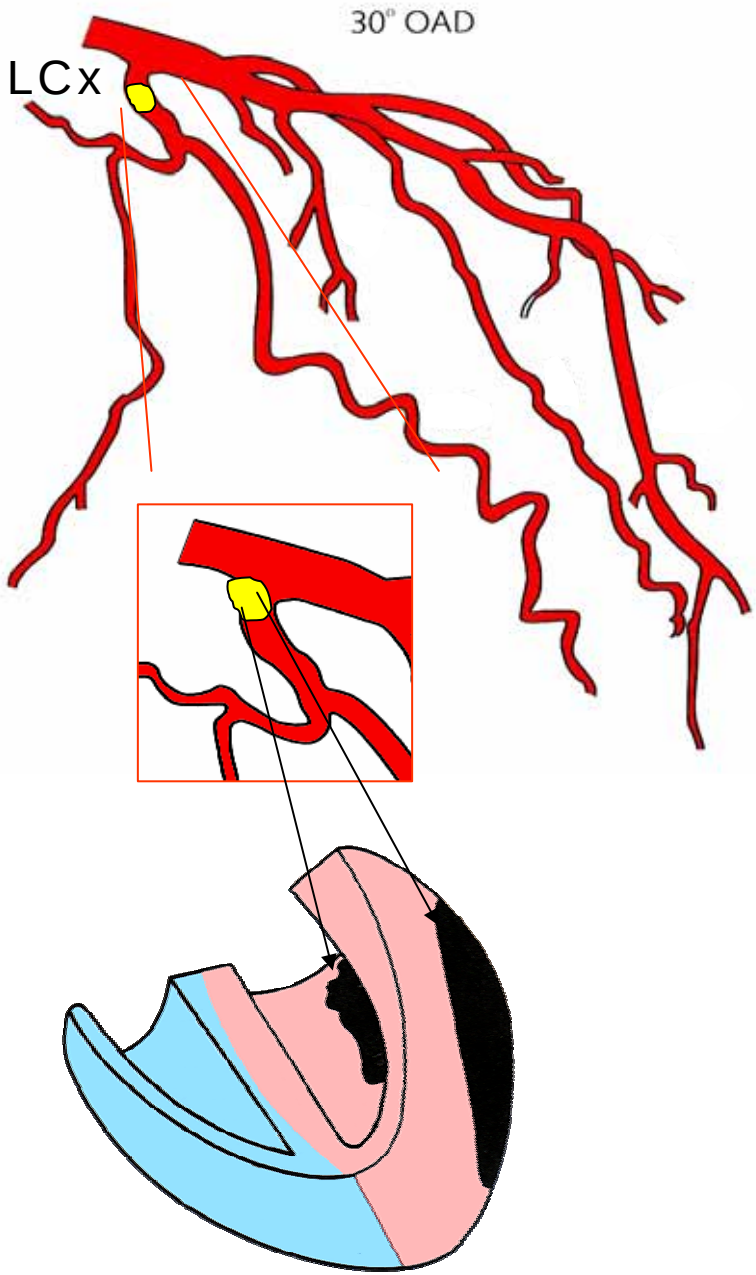
THE ST INJURY VECTOR DIRECTED TO DOWNWARD AND TO RIGHT(1)

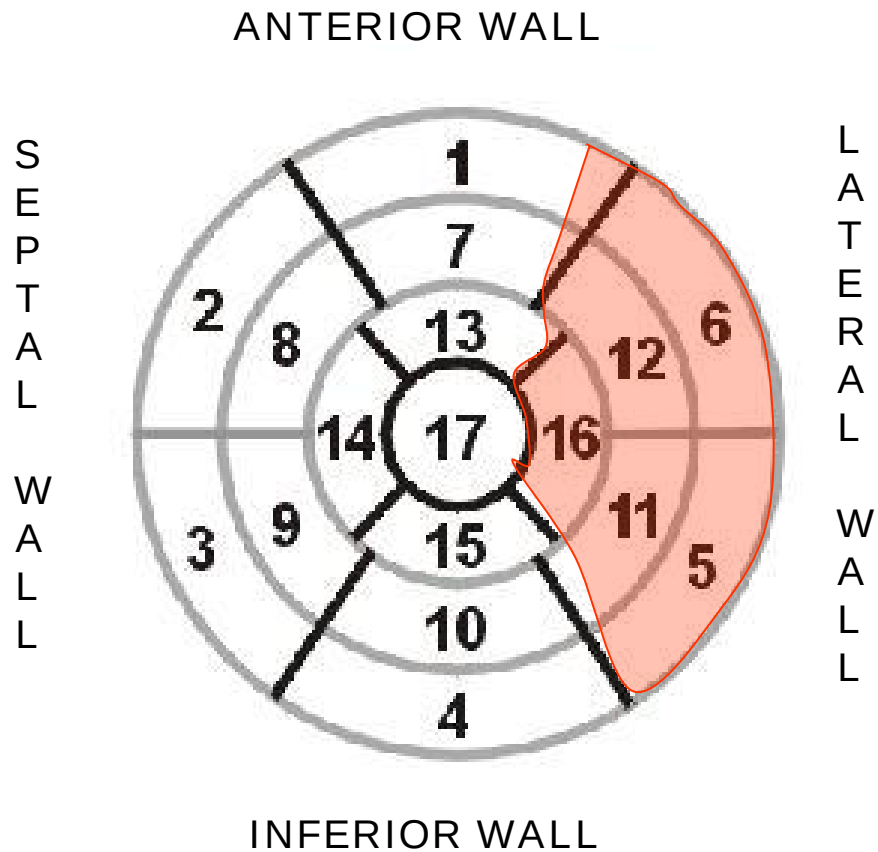


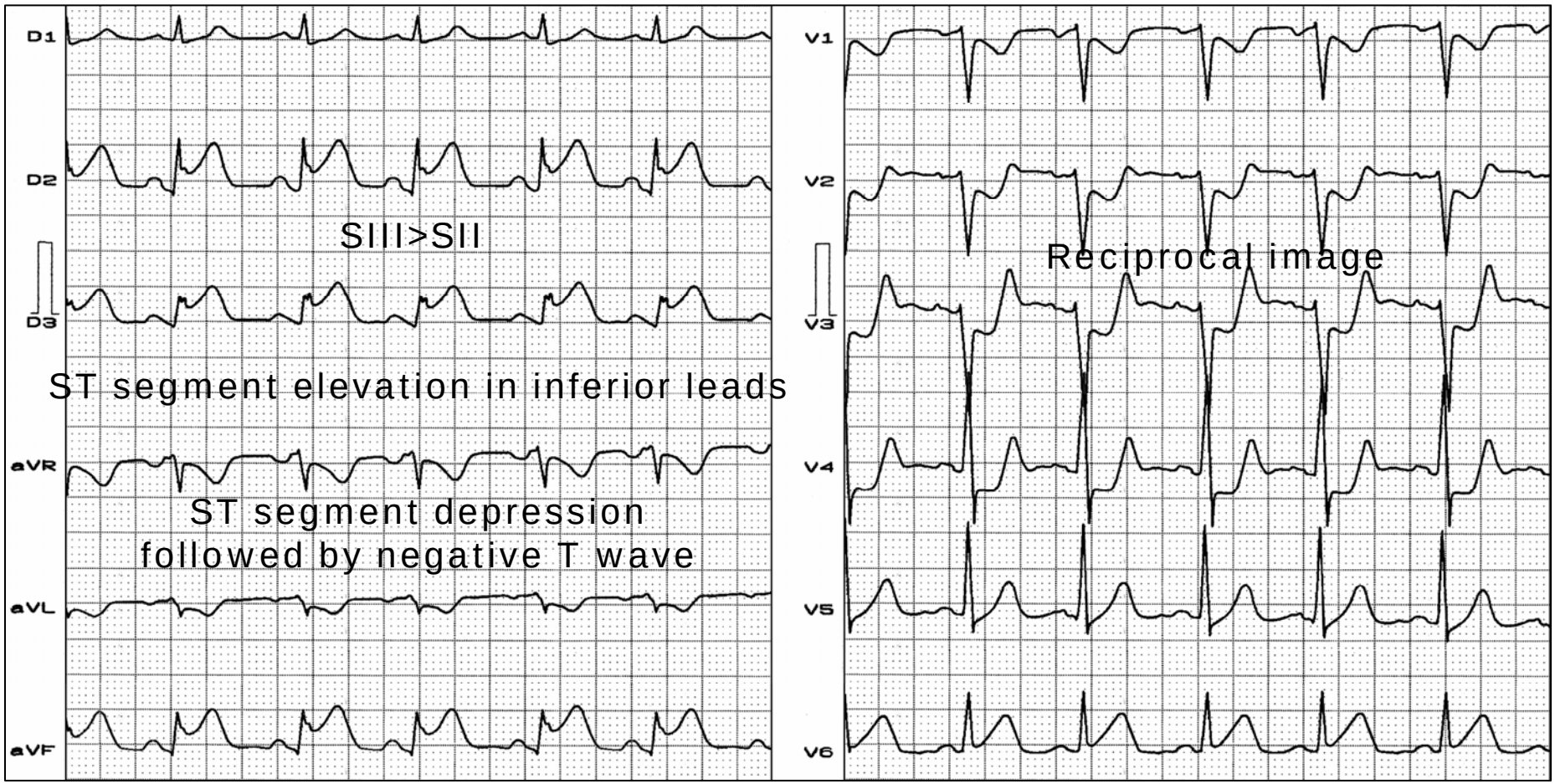
1. Fiol M, Cygankiewicz I, Carrillo A, et alValue of electrocardiographic algorithm based on "ups and downs" of ST in assessment of a culprit artery in evolving inferior wall acute myocardial infarction. Am J Cardiol. 2004 Sep 15;94:709-714.


Horizontal
-90°
180°
0°
Z
X
THE ST INJURY VECTOR DIRECTED TO BACK AND RIGHTWARD
V4R
V1
V2
V3
V4
V5
V6

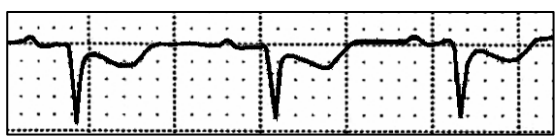
OCCLUSION OF
LEFT CIRCUNFLEX ARTERY (LCX)


30° OAD
LCx

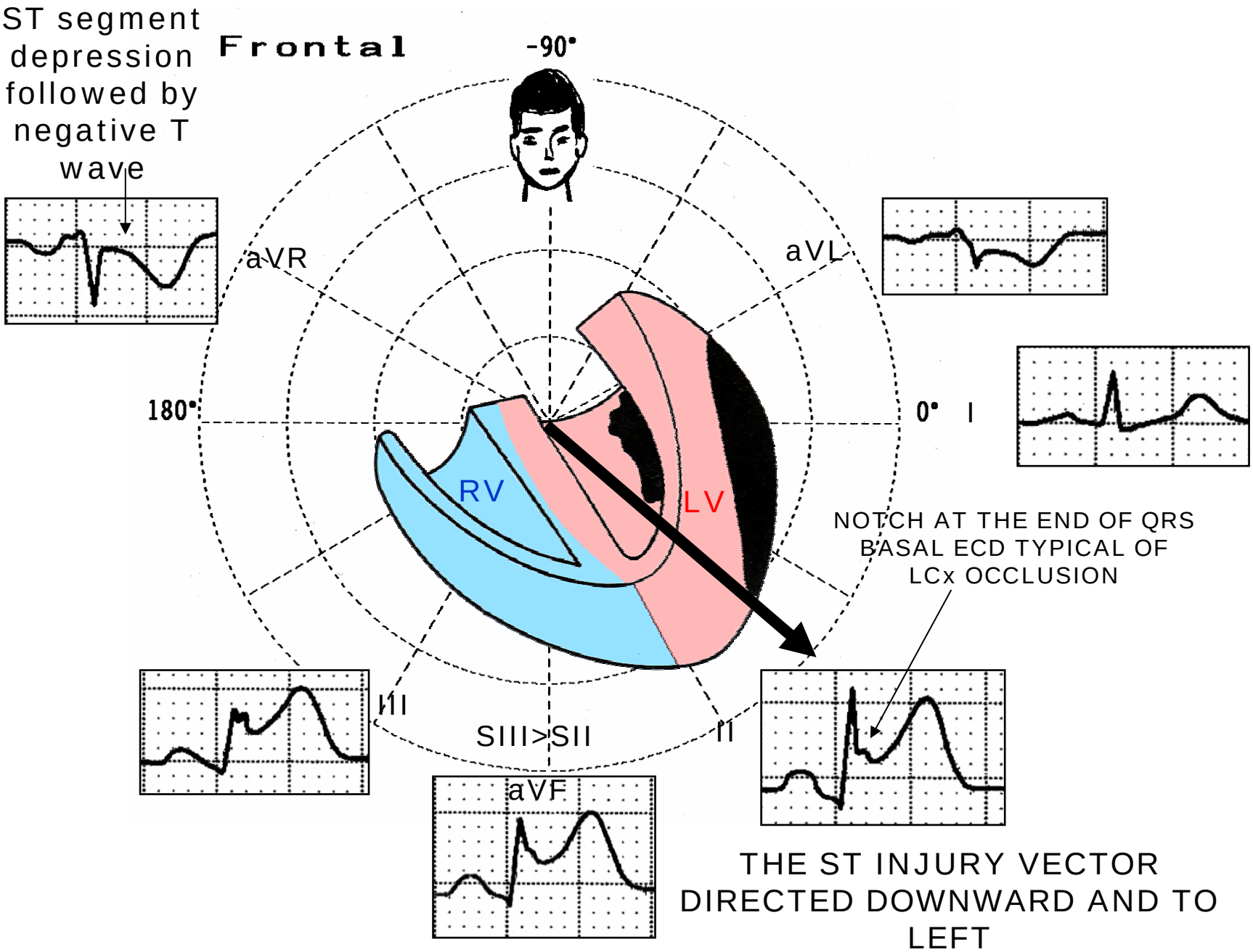

ANTERIOR WALL
SEPTAL WALL
LATERAL WALL
INFERIOR WALL
1
7
2
8
13
6
12
14
17
16
3
9
15
11
5
10
4



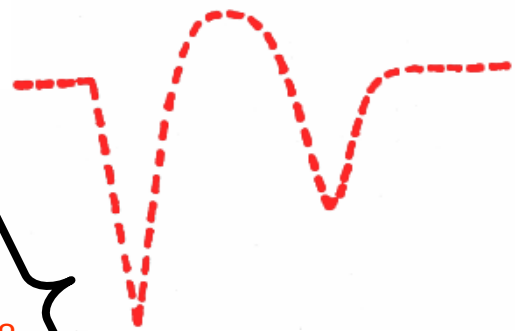
V_4R



ST segment depression in V_4R followed by negative T wave

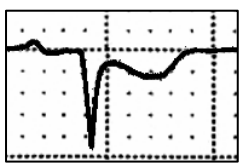

ST segment depression followed by negative T wave
Frontal
-90°
aVR
aVL
180°
0°
I
RV
LV
NOTCH AT THE END OF QRS
BASAL ECD TYPICAL OF
LCx OCCLUSION
III
SIII>SII
II
aVF
THE ST INJURY VECTOR
DIRECTED DOWNWARD AND TO
LEFT

ST segment elevation from V_7 to V_9



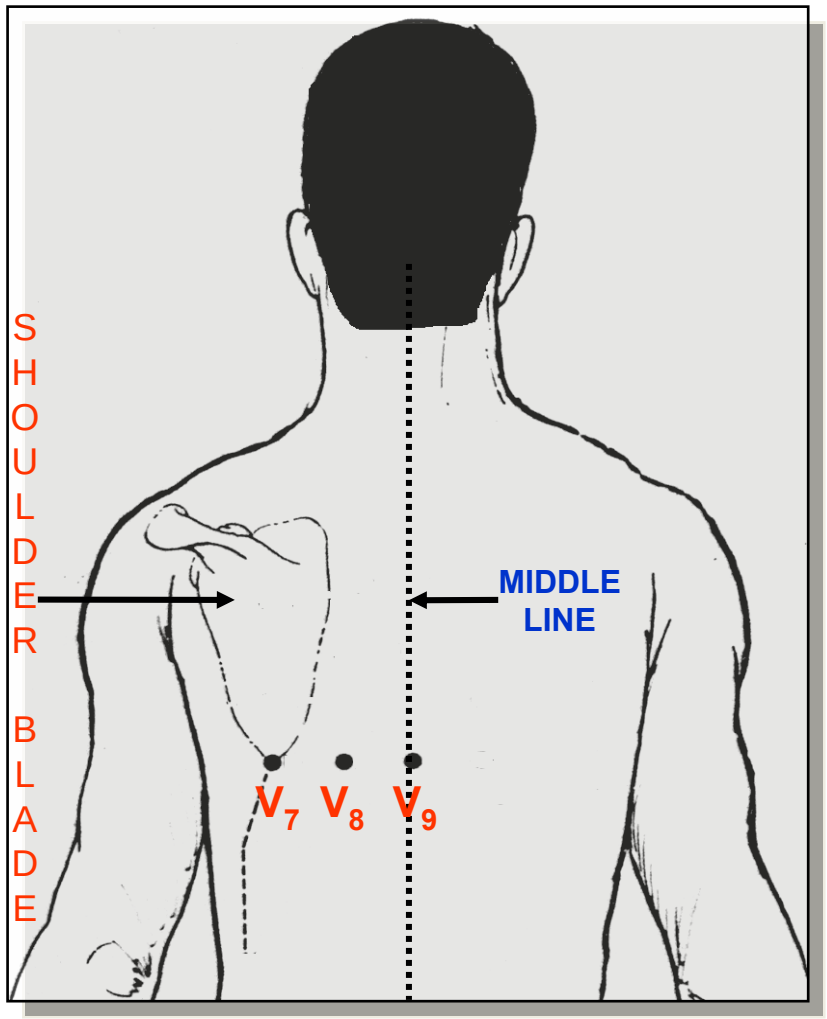
ST segment depression followed by negative T wave indicative of LCx occlusion

V_4R



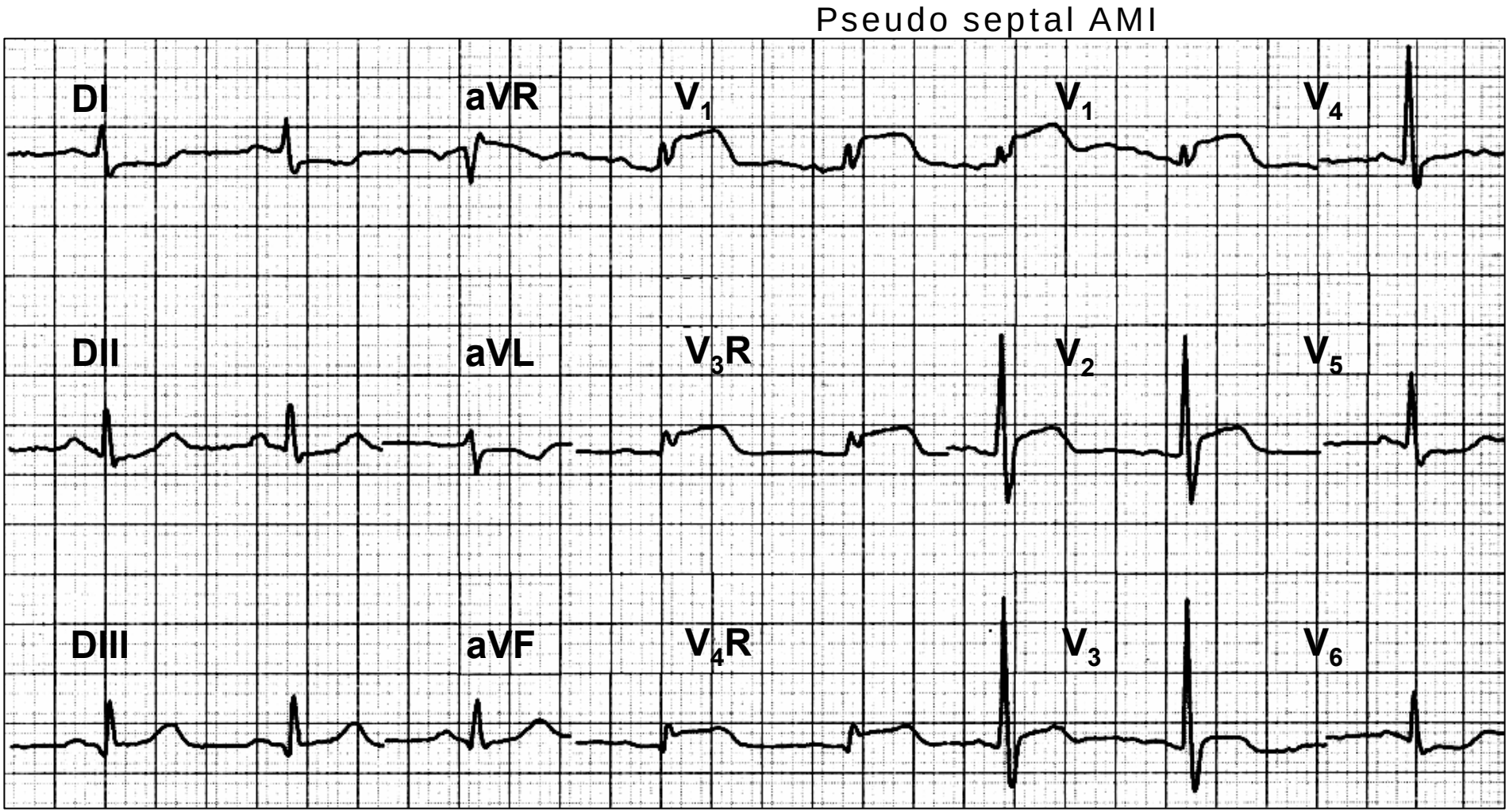
Reciprocal image of basal features

ACCESSORY DORSAL LEADS



The accessory leads are located between the left shoulder blade and the spine V_7, V_8 and V_9 leads.

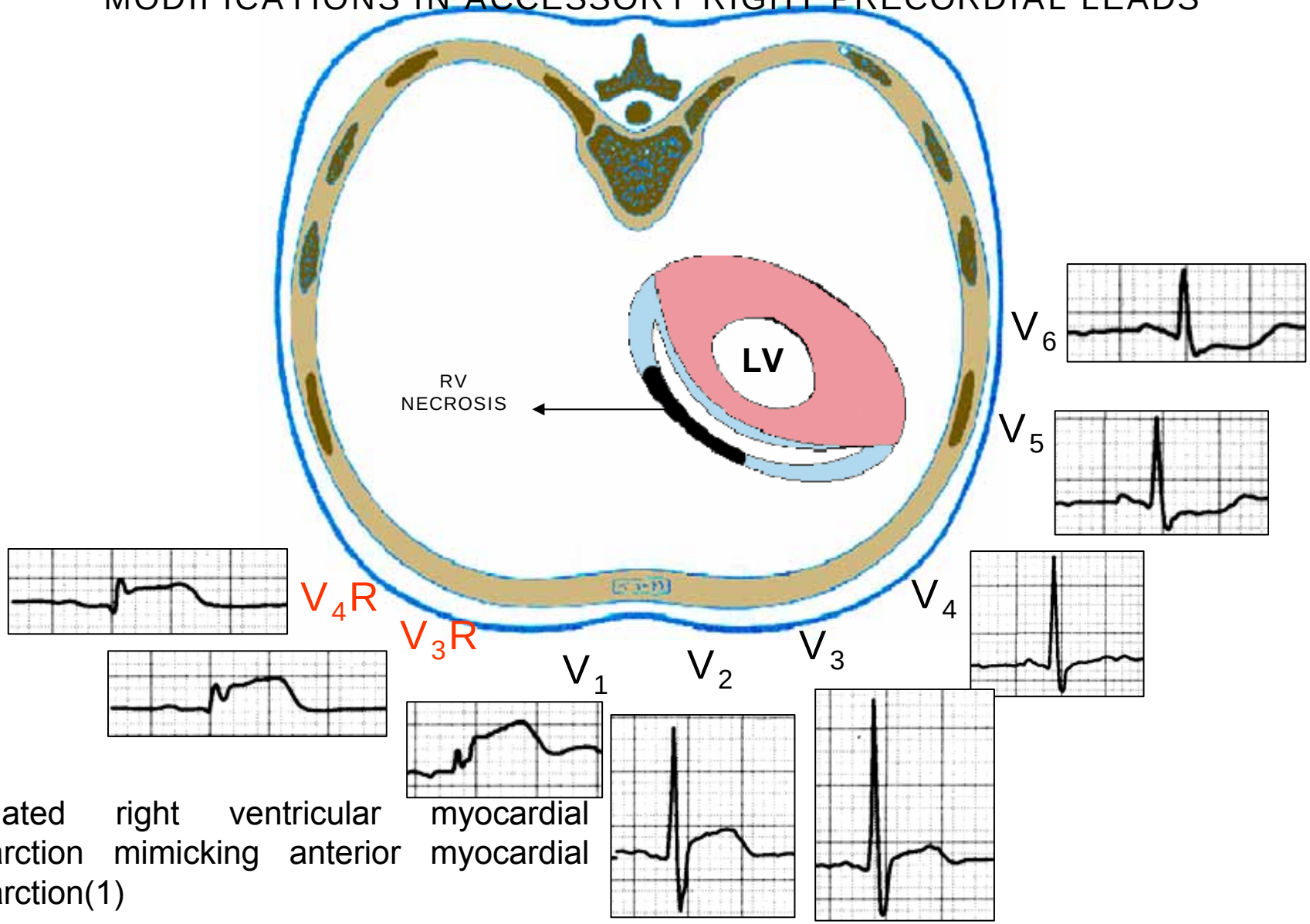
ISOLATED RIGHT VENTRICULAR INFARCTION



Isolated right ventricular infarction without left ventricle involvement, subepicardial injury current recorded in V_1,V_3R and V_4R.

ISOLATED RIGHT VENTRICULAR INFARCTION

MODIFICATIONS IN ACCESSORY RIGHT PRECORDIAL LEADS



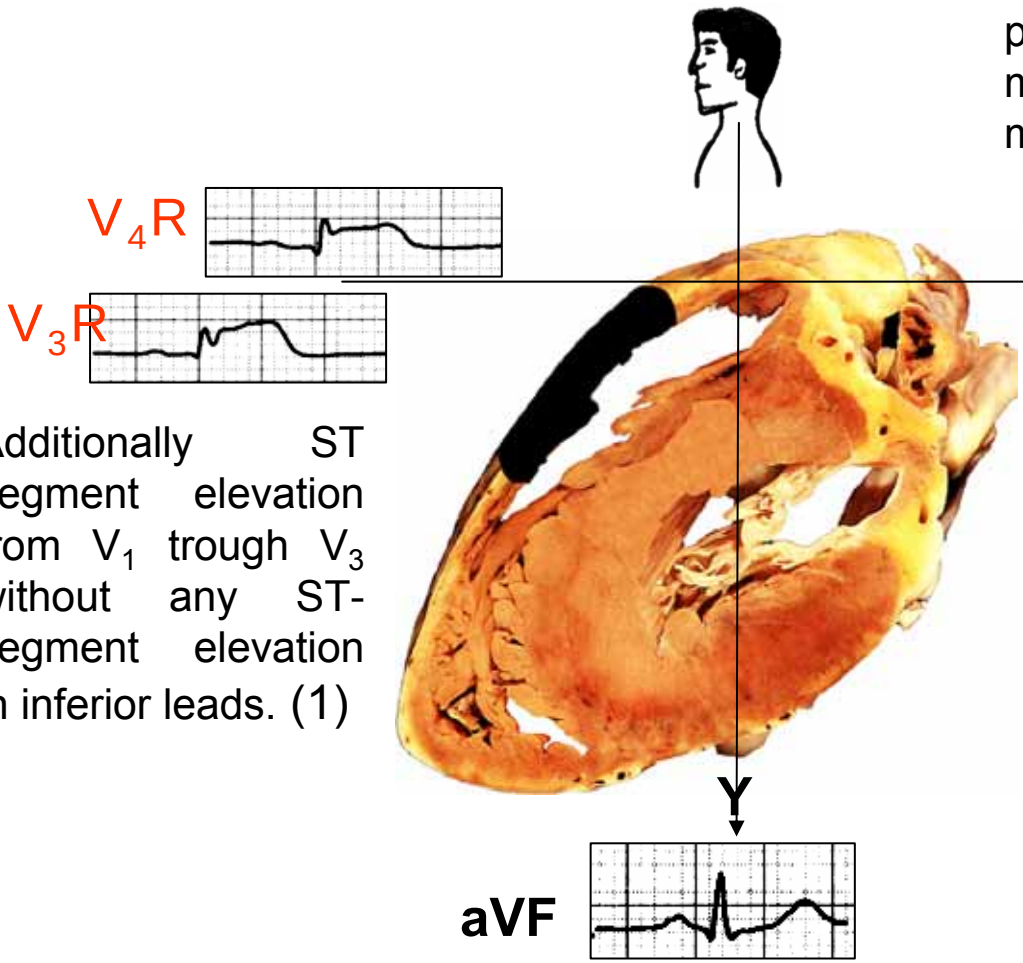
Isolated right ventricular myocardial infarction mimicking anterior myocardial infarction(1)

1. **Cetin M, Ucar O, Canbay A, et al. Isolated right ventricular myocardial infarction mimicking anterior myocardial infarction in a patient with coronary artery. Tex bypass grafts. Heart Inst J. 2011;38:598-9.**

ISOLATED RIGHT VENTRICULAR INFARCTION

LEFT SAGITTAL VIEW

Isolated right ventricular infarction is an extremely rare phenomenon. Its ECG features may be misinterpreted or even missed if not suspected.(1)



Additionally ST segment elevation from V_1 trough V_3 without any ST-segment elevation in inferior leads. (1)

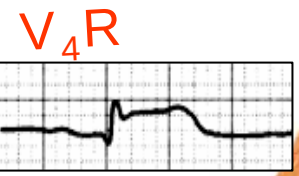
Anterior ST-segment elevation has also been described in patients with RCA occlusion without inferior ST-segment elevation. In these cases the inferior left ventricular wall is protected by left-to-right collaterals, with resultant isolated right ventricular infarction upon proximal RCA occlusion. an isolated right ventricular infarction(2)

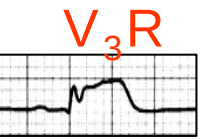
1. **Carroll R, Sharma N, Butt A, Hussain KM. Unusual electrocardiographic presentation of an isolated right ventricular myocardial infarction secondary to thrombotic occlusion of a non-dominant right coronary artery--a case report and brief review of literature. Angiology. 2003 Jan; 54:119-124.**
2. **Muhammad KI, Kapadia SR. Anterior ST-segment elevation with right coronary artery occlusion: a unique case of isolated right ventricular infarction. Angiology 2008 Oct-Nov;59:622-614.**

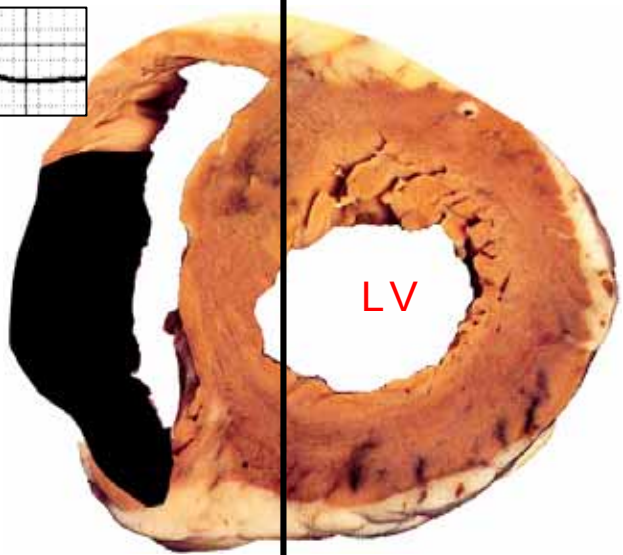
ISOLATED RV INFARCTION (EXCEPTIONAL)

VIEW IN THE MINOR AXIS

Rarely isolated right ventricular MI shows a tombostone anterior ST elevation (1)



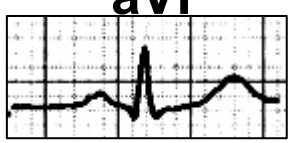




IT MAY CAUSE ST SEGMENT ELEVATION IN V1-V4 CAUSING CONFUSION WITH LV ANTEROSEPTAL INFARCTION.

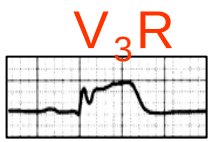
Y

aVF



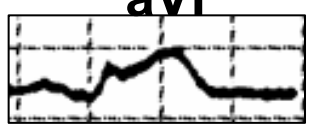
RV INFARCTION ASSOCIATED TO INFERIOR INFARCTION

VIEW IN THE MINOR AXIS



Y

aVF



1. **Collins N, Elliott V, Seidelin P. True isolated right ventricular infarction with tombstone anterior ST elevation. Heart. 2007Mar;93(3):374**.

LIMITATIONS OF THE ST INJURY VECTOR AND THE LOCATION OF MYOCARDIAL ISCHEMIA

Specificity: high[1]
Predictive accuracy: high
Sensitivity: quite low

Clinical situations where the deviation of the ST segment is limited

1. Presence of a previous infarction
2. Preexisting abnormalities of the ST segment
3. Left Bundle Brach Block/Right Bundle Branch Block
4. Ventricular Preexcitation
5. Multivessel disease
6. Abnormal site of origin of a coronary artery
7. Dominance or underdevelopment of the coronary arteries.
8. Left coronary artery dominance, multivessel disease, and absence of ECG signs of proximal culprit lesion are associated with failure to predict the culprit artery of inferior myocardial infarction by the 12-lead ECG.(2) The present case!!!!

1. **Andersen MP, Terkelsen CJ, Sørensen JT, Kaltoft AK, Nielsen SS, Struijk JJ, The ST injury vector: electrocardiogram-based estimation of location and extent of myocardial ischemia. J Electrocardiol. 2010 Mar-Apr;43:121-131.**
2. **Tahvanainen M, Nikus KC, Holmvang L, et al. Factors associated with failure to identify the culprit** artery **by the electrocardiogram in inferior ST-elevation myocardial infarction. J Electrocardiol. 2011 Sep-Oct;44:495-501.**

Vectorcardiography

Einthoven's Nobel prize-winning illustration of the cardiac electromagnetic current was based on a single vector (dipole) in the middle of an isosceles triangle. The electrical and geometrical requirements for his hypothesis were a spherical body surface with a homogeneous volume conductor and only one source for the dipole in the middle. Because a single human heart cycle does not quantitatively fulfill these conditions, a method for analyzing 3D electrocardiography data, known as vectorcardiography (VCG) was developed the late 1930s and many different VCG lead methods advanced since then. (1;2;3)The most common is the 7-lead method developed by Frank.(4). VCG was especially popular from the 1950s to 1980s. A widely published method, it was mainly used for ischemia diagnosis and has proved its potential in principle (5;6;6;7;8;9). For example, Mengden et al. retrospectively showed that VCG using 5 parameters for discriminant analysis has a sensitivity of 77.8% and a specificity of 78.4% in diagnosing a coronary condition compared to coronary angiography.(10) Difficult to interpret, VCG never became established as a routine method and, despite new approaches for use in ischemia, has lost importance.(11)

1. **Burch GE. The history of vectorcardiography. *Med Hist Suppl.* 1985;5:103–107.**
2. **Wilson FN, Johnston FD. The Vectorcardiogram. *Am Heart J.* 1938;16(1):14–28..**
3. **Chou TC, Helm RA, Kaplan S.*Clinical Vectorcardiography.* 2nd edition. New York: Grune and Stratton; 1974.**
4. **Frank E. An accurate, clinically practical system for spatial vectorcardiography. *Circulation.* 1956;13:737–749.**
5. **von Mengden HJ, Mayet W, Lippold K, Just H. Quantifizierung des coronaren Befallsmusters mit Hilfe einer computergestützten Analyse multipler EKG-Parameter. [Pattern quantification of coronary artery stenosis by computerized analysis of multiple ECG parameters (author's transl)]. *Klin Wochenschr.* 1981;59(12):629–637.**
6. **Erikssen J, Müller C. Comparison between scalar and corrected orthogonal electrocardiogram in diagnosis of acute myocardial infarcts. *Br Heart J.* 1972;34(1):81–86.**
7. **Gray W, Corbin M, King J, Dunn M. Diagnostic value of vectorcardiogram in strictly posterior infarction. *Br Heart J.* 1972;34(11):1163–1169. doi: 10.1136/hrt.34.11.1163.**
8. **Howard PF, Benchimol A, Desser KB, Reich FD, Graves C. Correlation of electrocardiogram and vectorcardiogram with coronary occlusion and myocardial contraction abnormality. *Am J Cardiol.* 1976;38(5):582–587. doi: 10.1016/S0002-9149(76)80006-9.**
9. **McConahay DR, McCallister BD, Hallermann FJ, Smith RE. Comparative quantitative analysis of the electrocardiogram and the vectorcardiogram. Correlations with the coronary arteriogram. *Circulation.* 1970;42(2):245–259.**
10. **Mehta J, Hoffman I, Smedresman P, Hilsenrath J, Hamby R. Vectorcardiographic, electrocardiographic, and angiographic correlations in apparently isolated inferior wall myocardial infarction. *Am Heart J.* 1976;91(6):699–704.**
11. **Murray RG, Lorimer AR, Dunn FG, Macfarlane PW, Hutton I, Lawrie TD. Comparison of 12-lead and computer-analysed 3 orthogonal lead electocardiogram in coronary artery disease. *Br Heart J.* 1976;38:773–778.**

Rubulis has summarized the results of VCG studies and publications from the Karolinska Institute, Stockholm on the analysis of T-vectors and T-loop morphology in myocardial ischemia. He showed significant differences in ventricular repolarization in patients with CAD(1) compared to the healthy control group, even in the absence of major co-morbidities. At rest, the areas under the T-loop and its shape and roundness significantly differed between CAD patients and healthy controls. Mostly, acute ischemia consistently reduced T-loop planarity and increased its roundness and area under T-loop (1). Rubulis further investigated the relationship between the size and location of myocardium at risk and the ventricular repolarization response during ischemia (during elective PCI and Tc-99m-sestamibi administration). Ventricular repolarization measures during maximum ischemia were compared with baseline measurements and the changes were related to the myocardium at risk and the occluded artery. He found significant correlations between the size of myocardium at risk and ST-segment alterations and changes of T-loop planarity, shape and roundness. In a longitudinal cohort study, Rubulis followed 187 CAD patients for 8±1 years. Cardiovascular death was independently predicted by a prolonged QRS duration and a widened QRS-T angle (spatial angle between maximum vectors of R-loop and T-loop). Myocardial infarction was most consistently predicted by increased T-loop planarity.

1. Rubulis A, Jensen SM, Näslund U, Lundahl G, Jensen J, Bergfeldt L.Ischemia-induced repolarization response in relation to the size and location of the ischemic myocardium during short-lasting coronary occlusion in humans.J Electrocardiol. 2010 Mar-Apr;43:104-112.