Homem de 55 anos de idade com precordialgia típica de uma hora de duração que desenvolvera colapso.

Male 55 years old with typical chest pain an hour-long that developed collapse.

Case of Raed Abu Sham'a (رائد ابو شمعة) M.D.
Palestine/Israel/Canada
Currently University of Ottawa Heart Institute

Final comments by **Andrés Ricardo Pérez-Riera M.D.Ph.D.**
In charge of electrovectorcardiogram sector – Cardiology Discipline-
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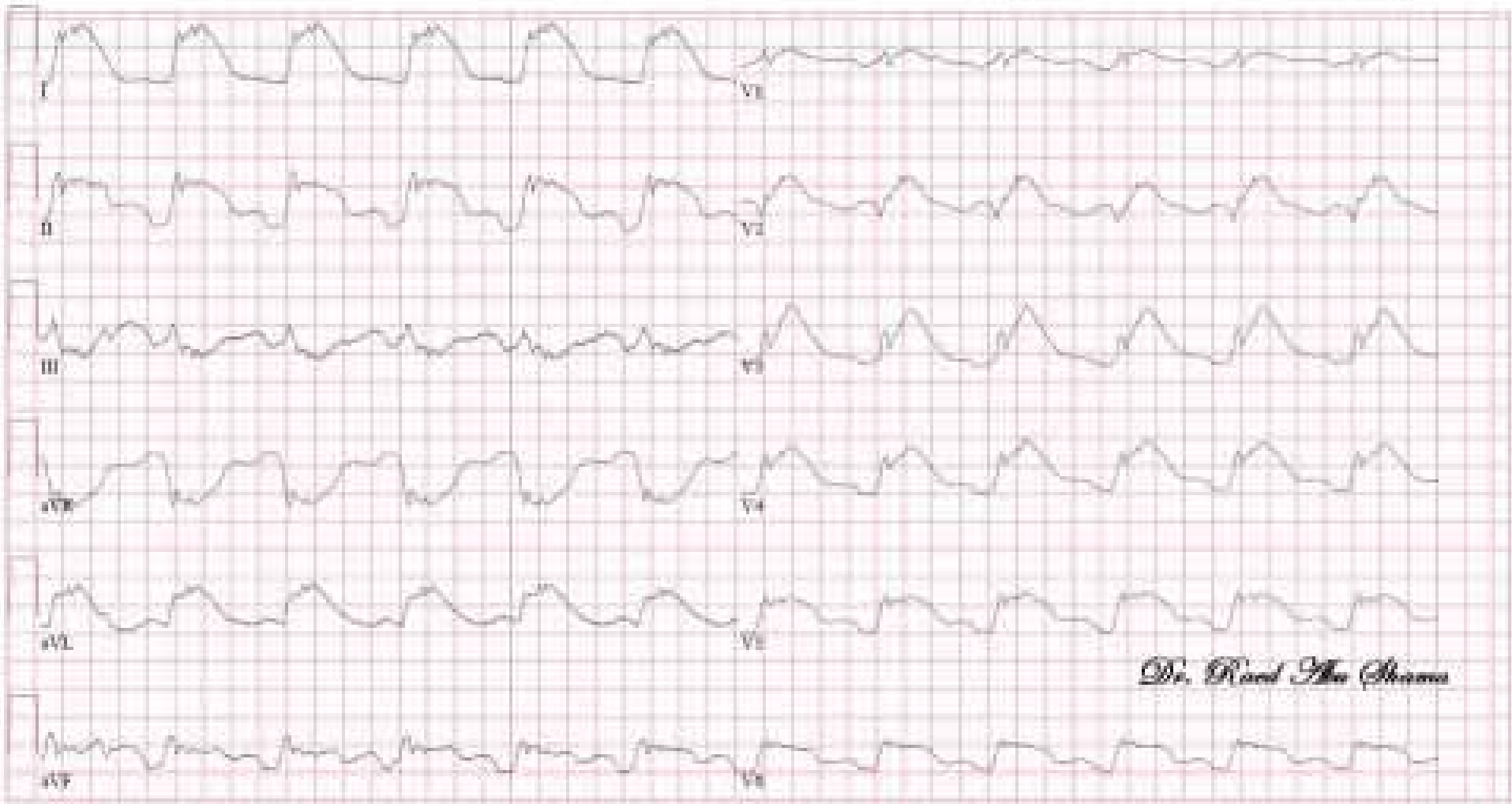
A 55 year old male patient suffered from chest pain one hour before his presentation.
He collapsed at the ER door. CPR was done, no shocks.
Which is the culprit artery?
What electrocardiographic diagnosis? And Why?

Um paciente do sexo masculino de 55 anos sofrera dor no peito iniciado uma hora antes de sua apresentação. Ele apresentou um colapso na porta da sala de emergência. Manobras de ressuscitação cardio-pulmonar foram realizadas sem choque.

Qual é a artéria culpada?
Qual o diagnóstico eletrocardiográfico?


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
Dr. Rinal Mac Sharma

Colleagues opinions

Spanish

Queridos amigos del forum intentaré analizar el caso del dr Raed Abu Sham'a
Se observa isquemia grado 3, es decir miocardio não protegido. La máxima expresión de lesión epicardica se observa en I, II y de V4 a V6 y las derivaciones menos afectadas son V1 y V2
Se observa ligera depresión de segmento ST en III.
El grado de elevación de los segmentos ST y de las ondas T es moderado lo que sugiere que se encuentran atenuadas por la extension de la lesión
El segmento del ST y la onda T se encuentran a la misma altura lo que sugiere acidosis miocárdica severa

Diagnóstico
1. **Isquemia por obstruccion total o súbita del tronco de la artéria coronaria izquierda asociada a lesión periférica severa principalmente por una artéria circunfeja (CX) dominante, pues las derivaciones dependientes de esta son la mas afectadas**

2) Obstruccion subita de una arteria muy dominante proximal I y aVL expresan la primera marginal, y V3 a V6 expresan la segunda marginal y DII la cola posterior de la CX

3) He visto un ECG similar en un paciente joven con un gran derrame pericárdico(> de 2 litros) con taponamiento que viajo a Asia oriental como mochilero.

Algunas veces en este forum fueron presentados ECGs parecidos con obstrucción del tronco de la coronaria izquierda,

Esperemos los estudios complementarios para saber diagnóstico exacto

Samuel

Dear friends of the forum: I will try to analyze the case of Dr. Raed Abu Sham'a
Grade 3 ischemia is observed: UNPROTECTED MYOCARDIUM.
The maximal expression of epicardial lesion is seen in I, II and from V4 to V6, and less affected leads are right precordial leads V1 and V2.
I see a slight ST segment depression in III.
The degree of elevation of the ST segments and T waves are moderate suggesting attenuation by the extension of the lesion.
The ST segment and T wave are at the same height suggesting severe myocardial acidosis.
Diagnosis

1) **Ischemic or sudden total occlusion of the Left Main Coronary Artery (LMCA) associated with severe peripheral ischemia in the territory of the left circumflex artery. (LCx). It is possibly dominant because the leads most affected are dependent of the LCx.**
2) **Sudden occlusion of an proximal artery to I lead. LCx very dominant and express the first marginal aVL, and V3 to V6 and express the second marginal or posterior tail of LCx.**
3) **I've seen a similar ECG in a young patient with a large pericardial effusion (> 2 liters) with Cardiac tamponade in a young that travel to East Asia as a backpacker.**

Sometimes in this forum similar ECGs were presented with obstruction of the LMCA,

Hopefully additional studies to determine accurate diagnosis

Samuel Sclarovsky Israel

Portuguese

Caro Professor. Samuel
Além das observações principalmente nos itens 1 e 2, com as quais concordo, parece-me que há alterações no segmento PTa, com supradesnivelamento em D1, aVL, V1 e V2 e imagem recíproca ou em espelho com infradesnivel do segmento ST em DII, DIII, aVF e V5 e V6, sugerindo infarto atrial associado.
Adail Paixão Almeida M.D. Vitória da Conquista/Bahia/ Brazil

Dear Professor Samuel

Besides the observations mainly in items 1 and 2, with which I agree, it seems to me that there are changes in PRs segment with elevation in I, aVL, V_1 and V_2 and mirror image or reciprocal changes with ST-segment depression in II, III, aVF and V5-V6, suggesting atrial infarction associated.

Adail PaixãoAlmeida M.D. Vitória da Conquista / Bahia / Brazil

Querido amigo Adail poderias explicar-nos em forma clara que é o segmento PTa a que te referes?
Segundo meu entendimento consideram-se critérios maiores de infarto atrial a elevação do PRs >0.5mm em V_5 e V_6 com depressão recíproca do PRs em V_1 e V_2.; ou a elevação do PRs >0.5mm DI com depressão recíproca em II e III.; ou depressão do PRs >1.5mm em qualquer precordial e 1.2mm em I, II, associada com qualquer arritmia.(1)

1) Liu CK, et al. Circulation 1961; 23: 331-338.
Andrés.

Dear Andres,

are we sure it is an acute coronary syndrome? A so diffuse an huge ST segment elevation (without clear "specular" ST depression) can be found in conditions other than ACS: for example acute fulminant myocarditis or epicardial infiltration from metastatic cancers (I had a case with similar ECG presentation of a patient with metastatic melanoma). Another possibility is severe hyperkalemia: the impressive ST elevation in this case could represent a sine-wave pattern (with a STEMI like presentation); moreover P wave are poorly detectable with long PR interval. An urgent coronary angiography should be considered (because ECG and clinical presentation) however, while awaiting, I would first perform an echocardiogram plus blood exams (creatinine, serum electrolytes, white cell count, C reactive protein etc) and possibly a chest X \ray.

Let me know how this history goes on....

Eli de maria Operative Unit of Cardiology, Ramazzini Hospital, Carpi, Modena, Italy.

Division of Cardiology, S. Maria Nuova Hospital, Viale Risorgimento 1, Reggio Emilia, Italy.

e.demaria@inwind.it

Spanish:

Estamos seguros que se trata de un sindrome coronario agudo? Una elevación difusa del segmento ST sin inmagen recíproca clara pueden ser observadas en otras condiciones que no SCA.por ejemplo miocarditis aguda fulminante o infiltracion epicárdica de metástasis cancerosa(yo he tenido un caso semejante con metástasis por melanoma)

Otra posiblidad a ser considerada es la presencia de severa hiperpotasemia por el aspecto en sino de las ondas y dando una elevacion like del ST ; ademas las ondas P son pobremente detectables y el intervalo PR está prolongado.

Una coronarioangiografia de urgencia deberia ser considerada y mientras esperamos yo le haria un ECO y examenes de sangre como creatinina, electrolitos, contaje de blancos, proteina C reactiva etc y posiblemente un Rx de tórax.

Veamos ahora como la história continua.

Eli

Dear Dr. Elia de Maria thank very much for your cleaver opinion.
We are waiting for the final diagnosis in few days
All the best dear colleague, and thank again for your valuable observation
Andrés.

Spanish Estimado Elia; muchas gracias por tu inteligente opinión.
Esperamos por el diagnóstico final en poco dias.
Deseo lo mejor para usted querido colega. y gracias de nuevo por su valiosa contribución
Andrés

Dear Prof. Andrés
Good evening
Unfortunately, no details available about the patient's exact cause of death. He was labeled as SCD. The only available information as follows: He had free past medical history, physically fit and no risk factors for CAD. Almost 60-70 minutes of the onset of the chest pain he collapsed. His pain radiated to the right leg. CPR was done for > 1 hour. This ECG was done 23 minutes after the onset of CPR. The first documented rhythm was asystole followed by pulseless electrical activity (PEA). Bedside echo was done few minutes after the CPR and showed no pericardial effusion or myocardial contractility. The first available Troponin was done 70 minutes after the onset of the chest pain and was completely negative [<0.02 mic/L]. pH was 6.95. No autopsy was done and the family immediately consented for multiorgan donation.
Thank you for your interest.
Caro Prof Andrés
Boa noite
Infelizmente, não temos detalhes disponíveis sobre a causa exata de morte do paciente. Foi rotulado como MCS. As únicas informações disponíveis são as seguintes: Não tinha histórico de enfermidade, fisicamente em boa forma e sem fatores de risco para DAC. ≈ 60-70 minutos após o início da dor torácica entrou em colapso. A dor irradiada para a perna direita. Manobras de ressuscitação cardiopulmonar foram feitas por > 1 hora. Este ECG foi feito 23 minutos após o início das manobras. O ritmo primeiramente documentado foi assistolia seguido por atividade elétrica sem pulso. Um Ecocardiograma na cabeceira do leito foi feito poucos minutos após a parada cardiopulmonar e não mostrou derrame pericárdico ou contratilidade miocárdica. A troponina primeiro disponível foi feita 70 minutos após o início da dor no peito e era completamente negativa [<0,02 mic / L]. pH foi 6,95. Não foi realizada autópsia e a família consentiu imediatamente para a doação de múltiplos órgãos.
Obrigado pelo seu interesse.

Raed Abu Shama, M.D. University of Ottawa Heart Institute. Cardiac Pacing and Electrophysiology 40 Ruskin Street, Ottawa, Ontario, K1Y 4W7. CANADAMobile: 001 613 851 4034. Work: 001 613-761-5000 - Pager 715 8447rabushama@ottawaheart.ca Skype name: raedabushama

Excuse me dear Raed you wrote two think that I did not understand

1. ***Pain radiated to the right leg. Are you sure?*** **Right leg or right arm?**
2. ***Bedside echo was done few minutes after the CPR and showed no pericardial effusion or myocardial contractility. Myocardial contractility affectation?***

Me perdoe prezado Raed voce escreveu duas coisas que eu não entendi:

1. A dor se irradiaba para a perna direita. Está certo? Não será que irradiava para o braço direito?
2. Referes também que foi realizado um ECO na beira do leito pouco minutos após as manobras de ressucitação cardio pulmonar e não mostrou derrame pericárdico o contractilidade miocárdica. Nao será contractilidade miocárdica comprometida?

Hello there
I am in London now for a couple of days and I will check my e-mail infrequently.
Yes it was right leg pain and not right arm.
The echo showed no pericardial effusion and there was Electro-Mechanical Dissociation [PEA] or [no contractility].
Regards
Olá lá
Eu estou em Londres agora, por um par de dias e vou verificar meu e-mail com pouca freqüência.
Sim, foi dor na perna direita e não no braço direito.
O Eco não mostrou derrame pericárdico e havia dissociação eletro-mecánica [DEM] ou [contratilidade não].
Atenciosamente
Raed

Queridos amigos el nivel de tropinina normal se explica por que el paciente no tuvo ningun signo de reperfusión miocárdica, por lo tanto el test es falso negativo. El dramatismo de la evolución y la falta de respuesta a las maniobras de resusitación sugieren obstrucción del tronco de la coronaria izquierda y el ECG obstrucción severa previa de la circunfleja izquierda.
Un fraternal abrazo
Samuel Sclarovsky

Dear friends normal tropinin level is explained because the patient had no signs of myocardial reperfusion. Therefore the test is a false negative. The dramatic clinical picture evolution and the lack of response to resusitación maneuvers suggest LMCA obstruction and the ECG severe prior obstruction of the left circumflex(LCx).

A fraternal hug

Samuel Sclarovsky

Finals comments

by **Andrés Ricardo Pérez-Riera M.D.Ph.D.**
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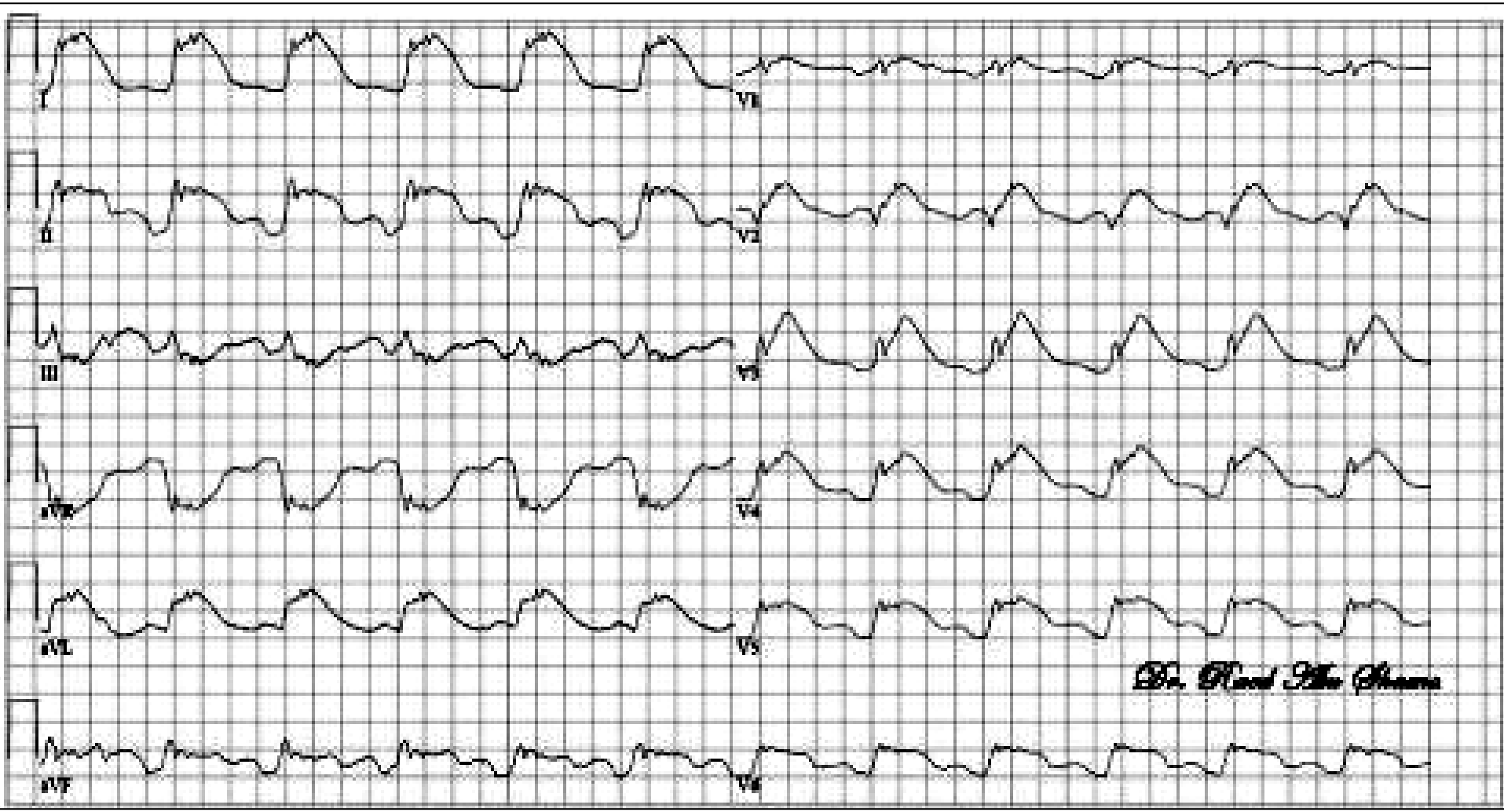
I
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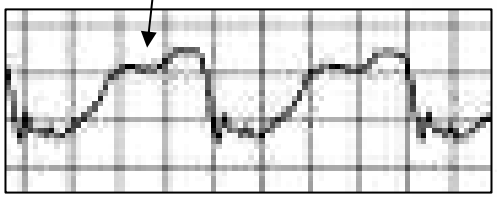
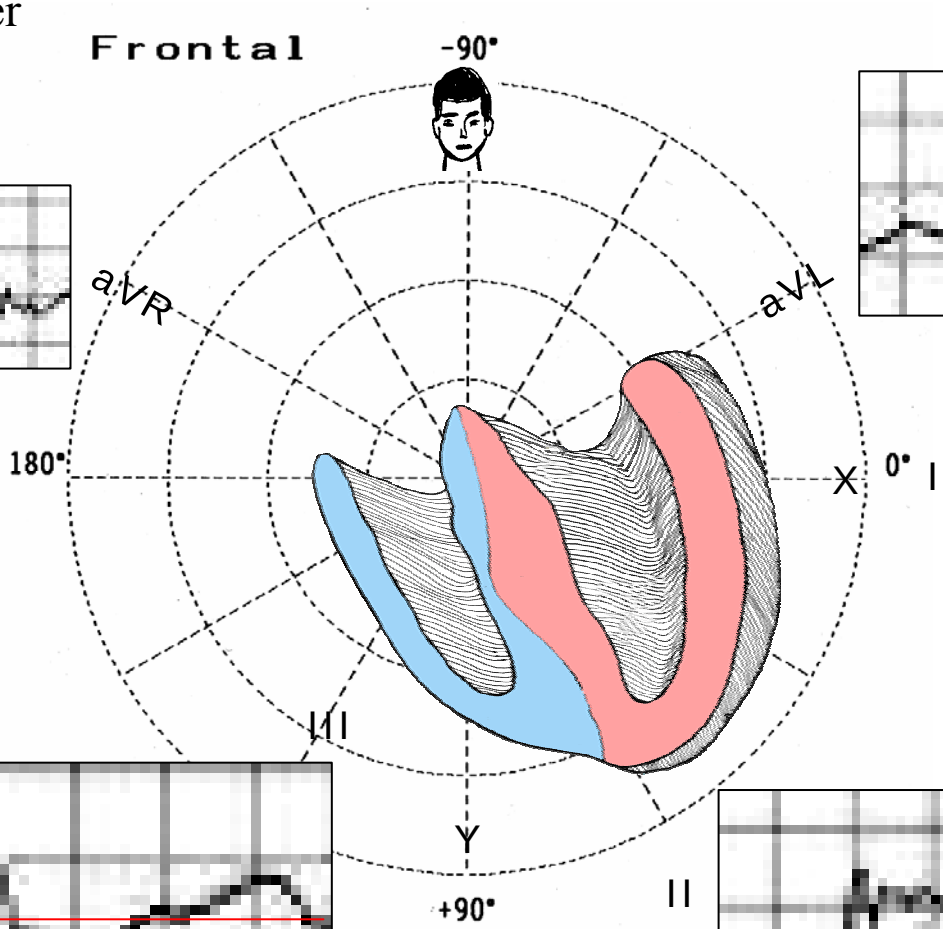
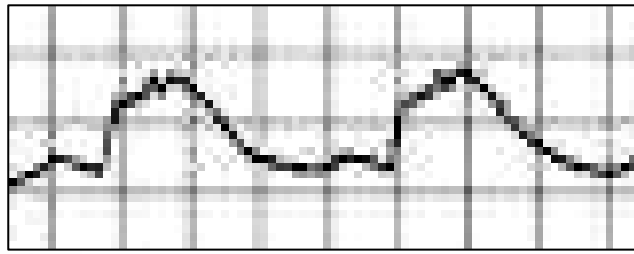
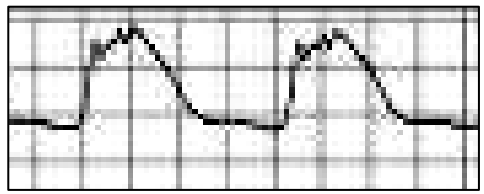
V1
V2
V3
V4
V5
V6

Lead aVR ST elevation accompanied by either anterior ST elevation

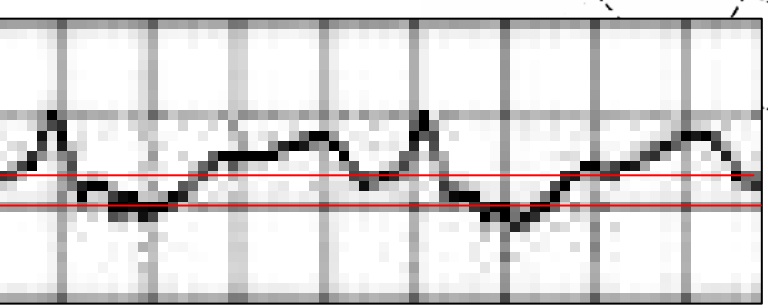






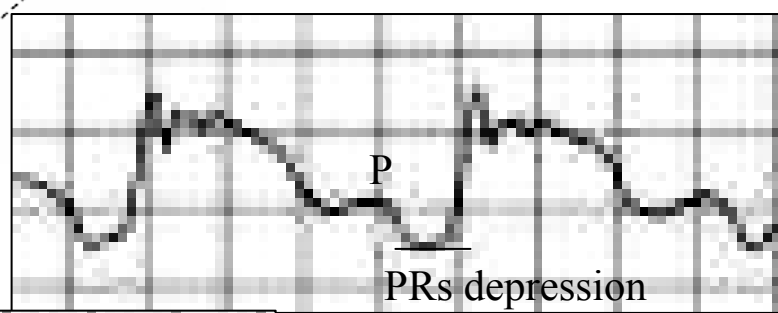


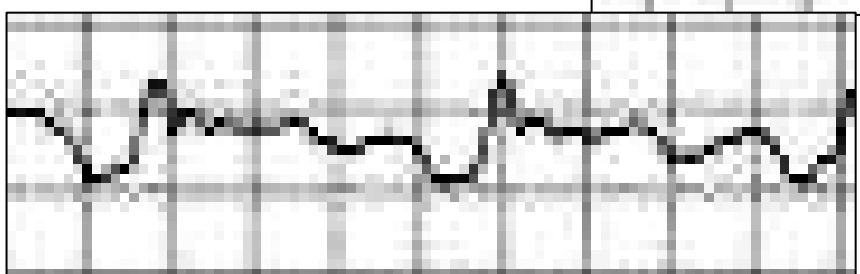
HR = 75lpm



ST depression III=1mm

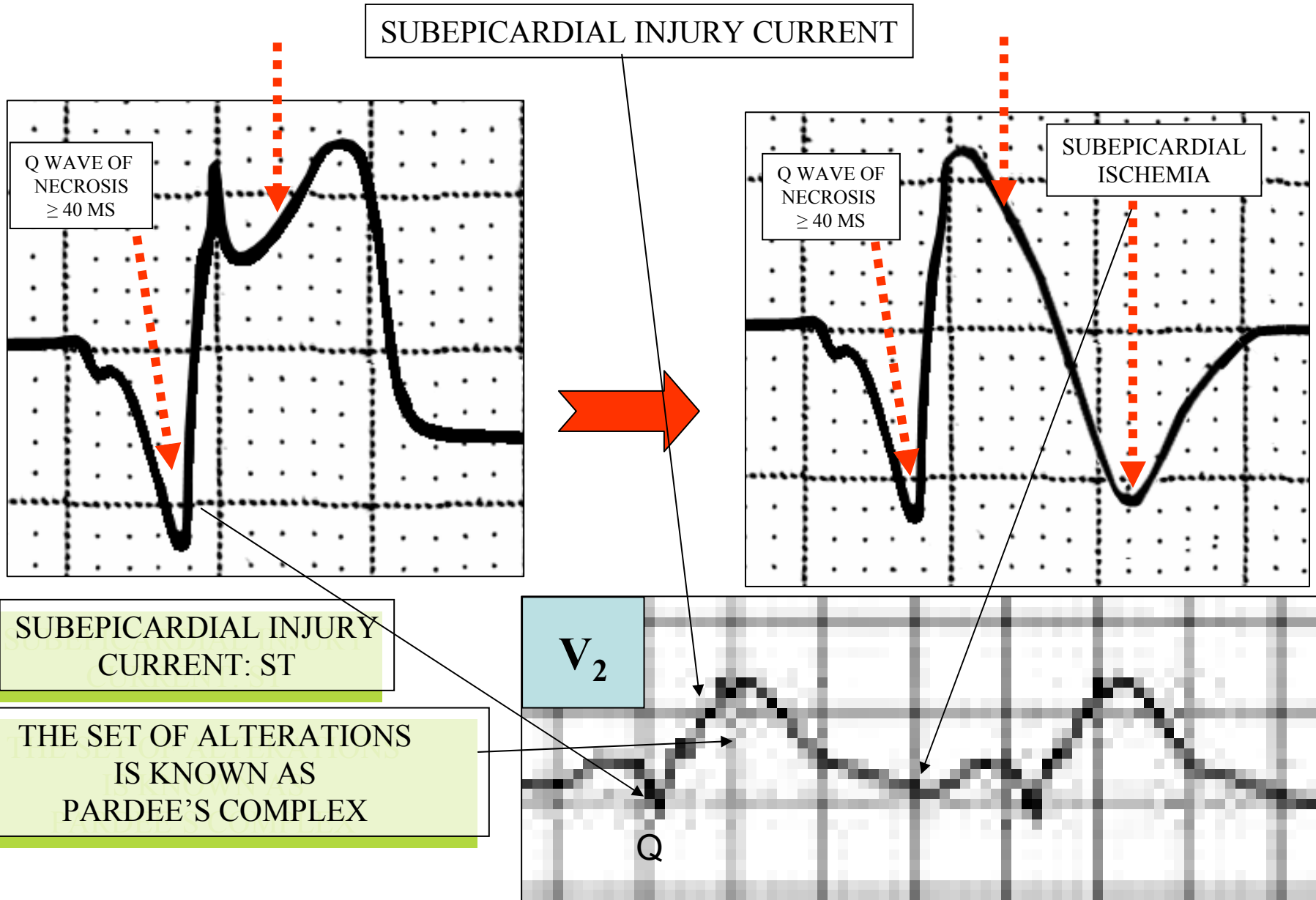
II

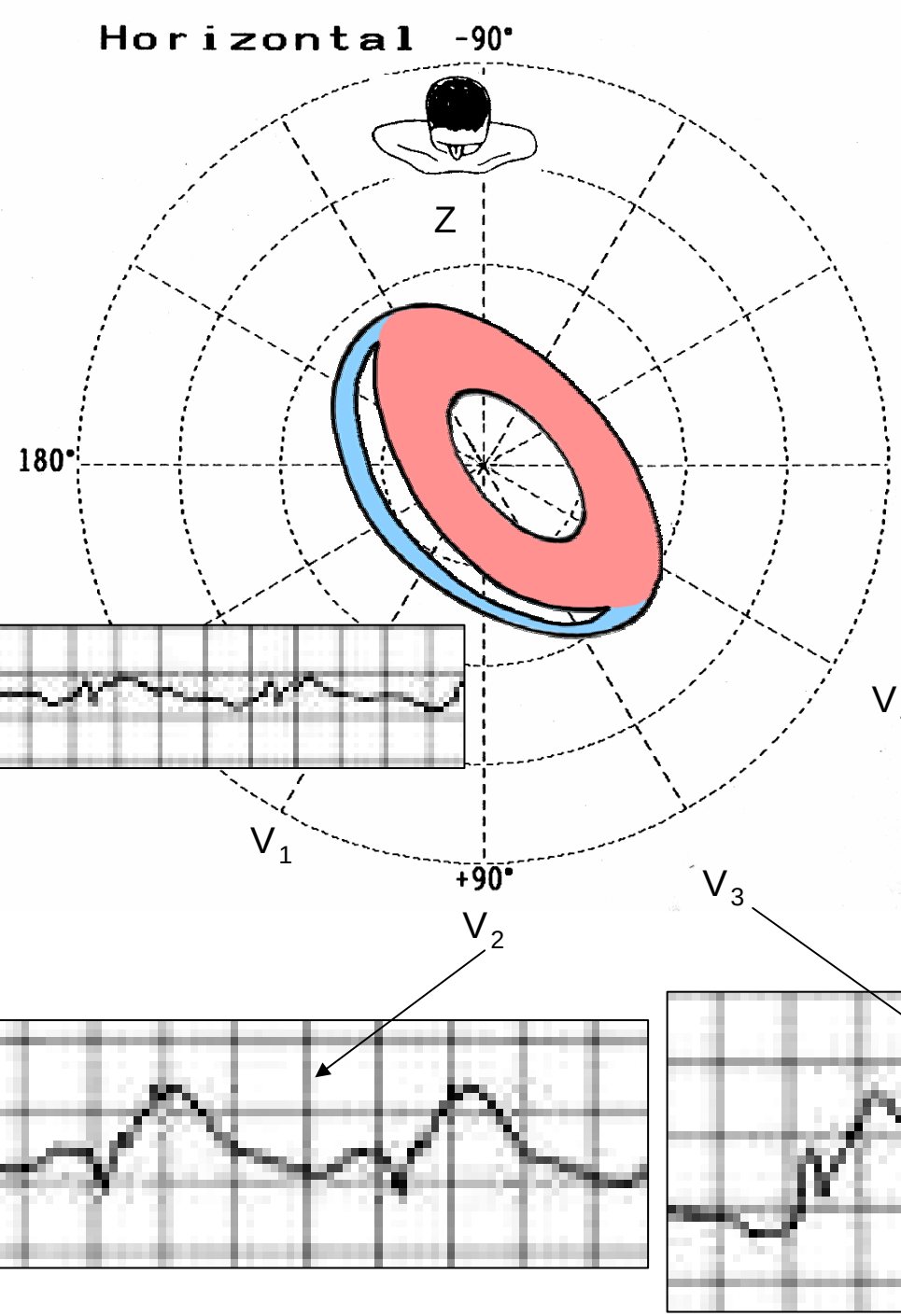




WE OBSERVE THE ECG MODIFICATION AFTER 20 MINUTES OF OBSTRUCTION

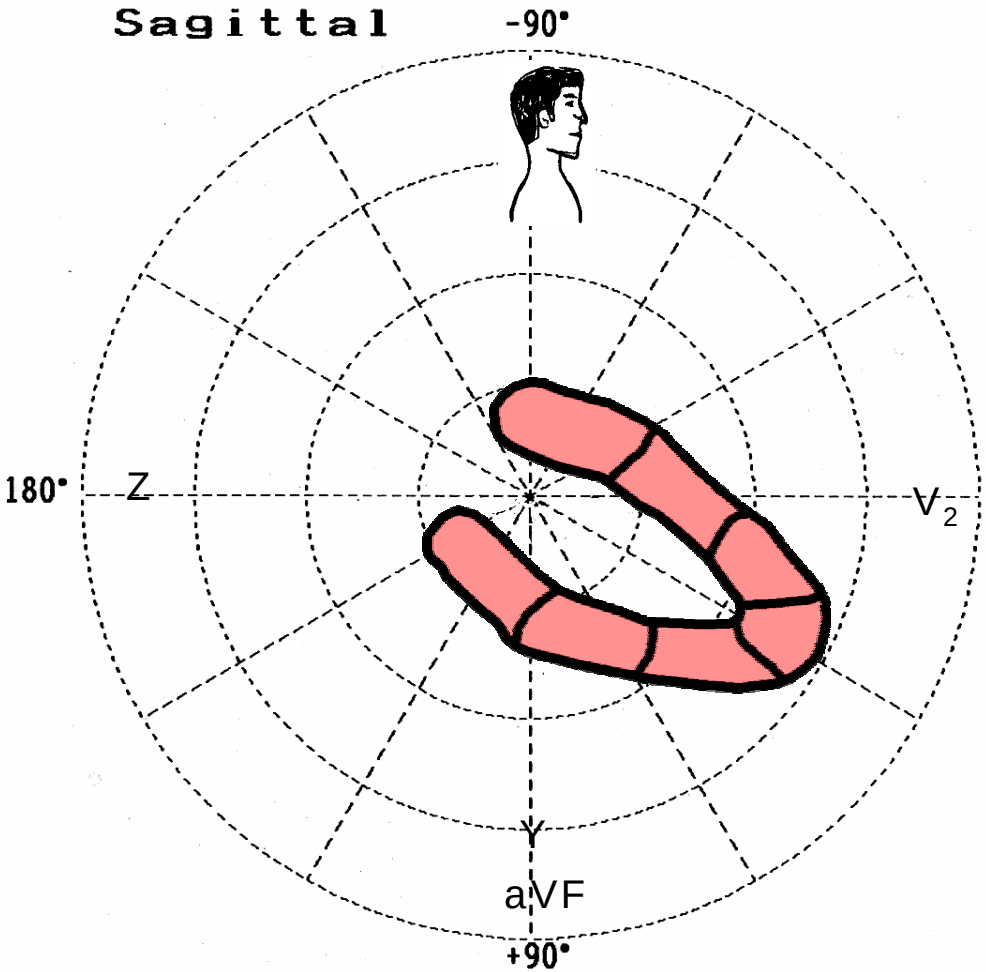
Modifications that occur after 20 minutes of coronary occlusion, necrosis, subepicardial injury and ischemia.

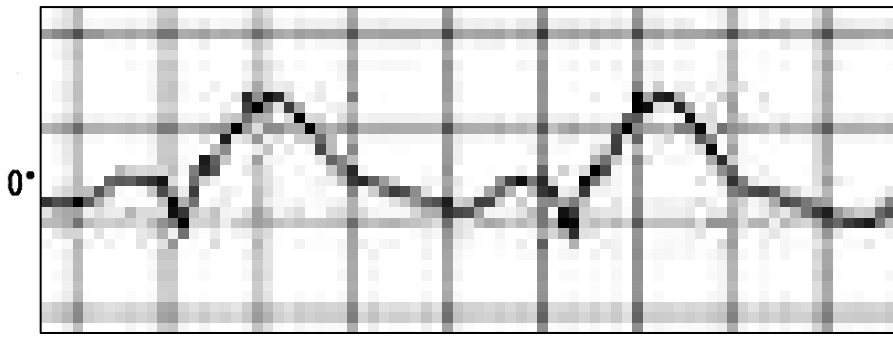


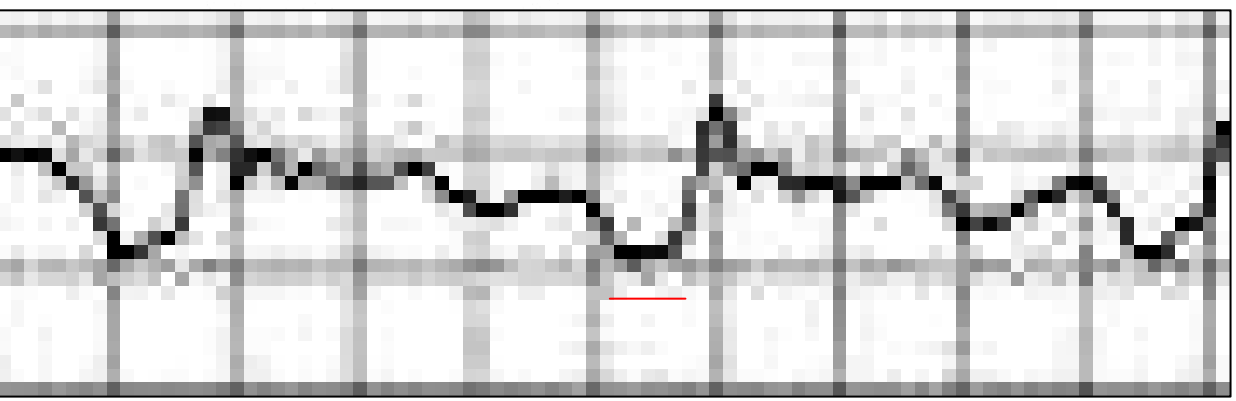


LMCA occlusion should be suspected in severely ill patients with widespread ST-segment depressions, especially in leads V4-V6 with inverted T waves **or ST elevation involving the anterior precordial leads and the lateral extremity leads I and aVL.** In addition, lead aVR ST elevation accompanied by either anterior ST elevation or widespread ST-segment depression may indicate left main occlusion.(1)

1. **Nikus KC, et al. J Electrocardiol. 2008 NovDec; 4: 626-629.**

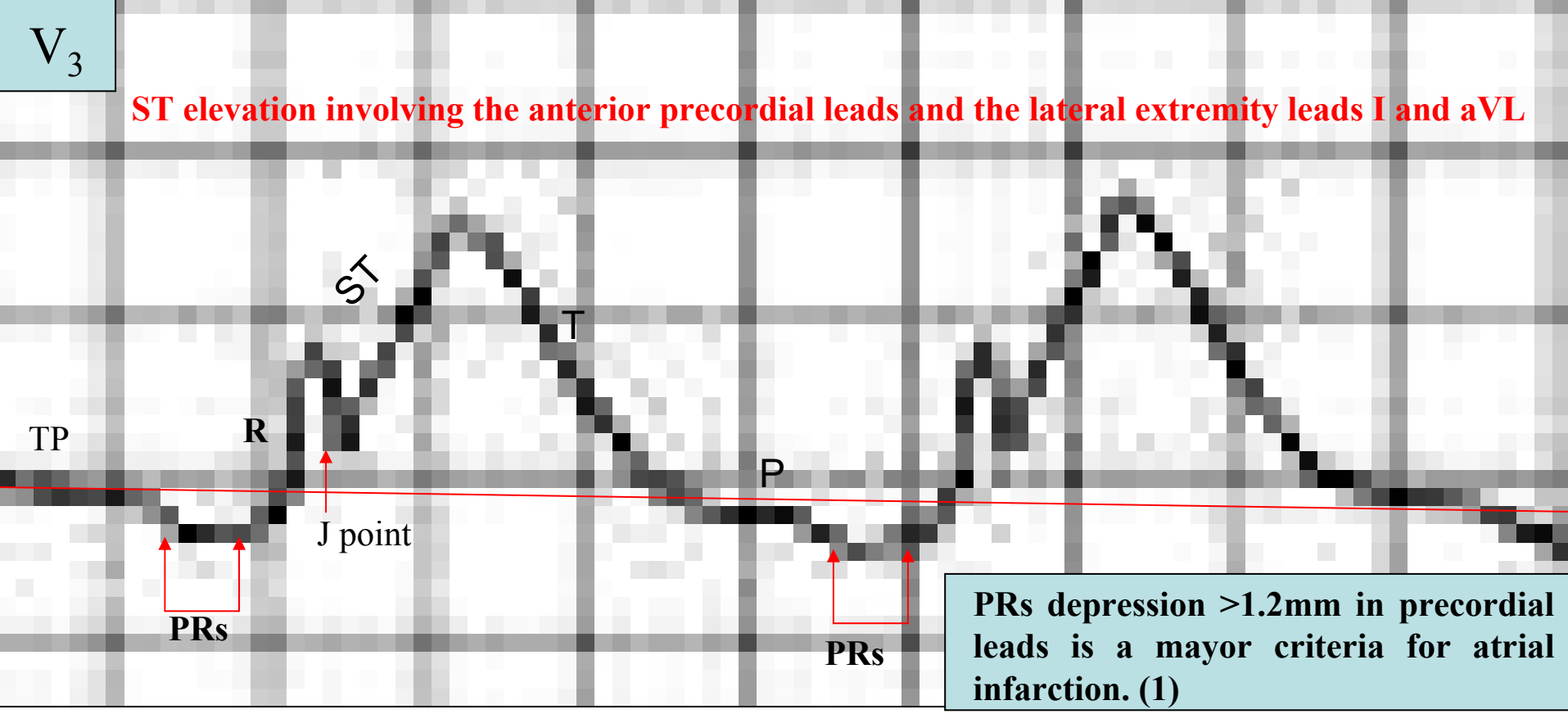

Sagittal
-90°
180°
Z
V2
aVF
+90°


0°

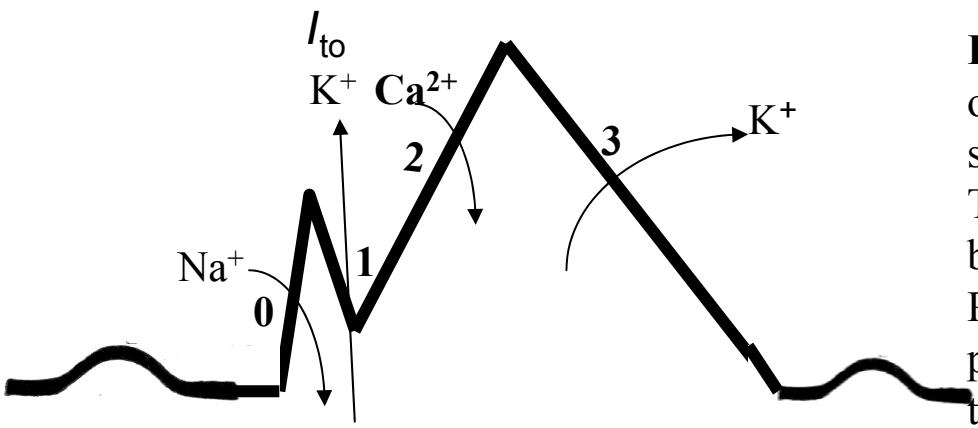


V_3

ST elevation involving the anterior precordial leads and the lateral extremity leads I and aVL



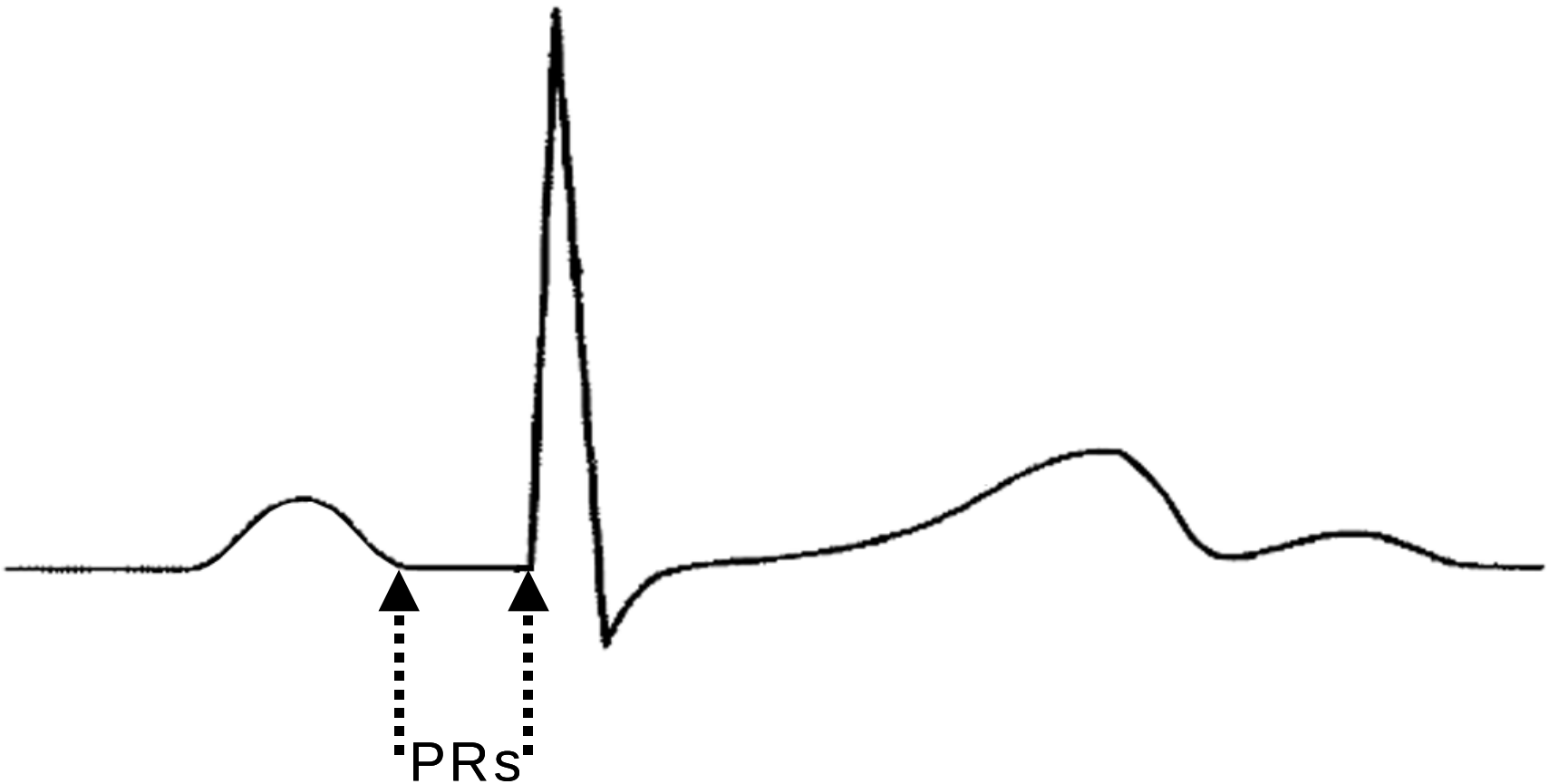
PRs depression >1.2mm in precordial leads is a mayor criteria for atrial infarction. (1)



PR (PRs) or PQ segment: it stretches from the end of P wave to the onset of QRS complex. The PR segment connects the P wave and the QRS complex. The impulse vector is from the AV node to the bundle of His to the bundle branches and then to the Purkinje Fibers. This electrical activity does not produce a contraction directly and is merely traveling down towards the ventricles and this shows up flat on the ECG. The PR interval is more clinically relevant.

1) Liu CK, et al. Circulation 1961;23:331-338.

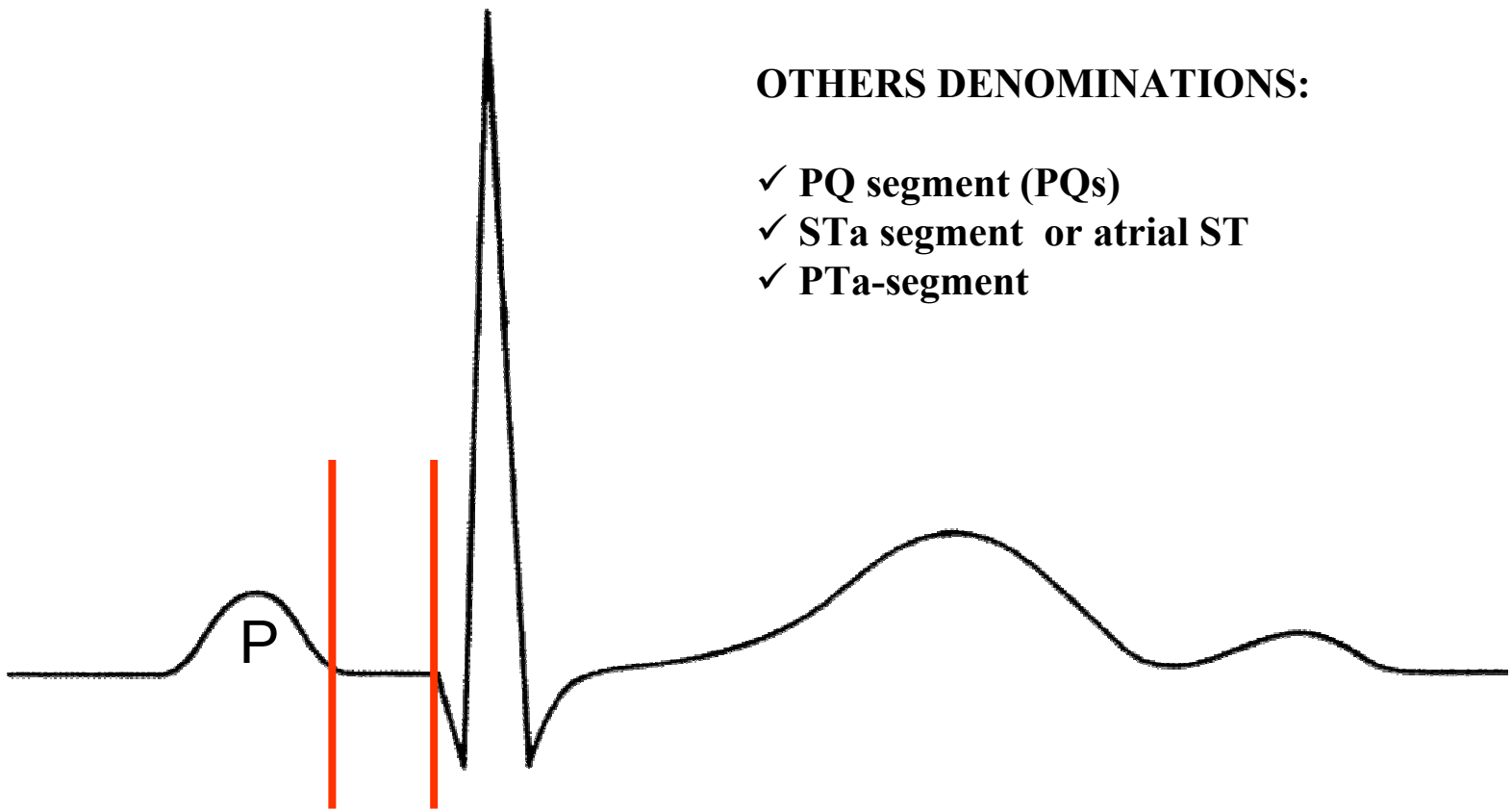
PR segment or PQ segment (PRs-PQ)



It stretches from the end of P wave until the onset of QRS

Representation of the PR segment from the end of the P wave to the onset of the QRS complex (onset with q or r wave).

PR segment (PRs)



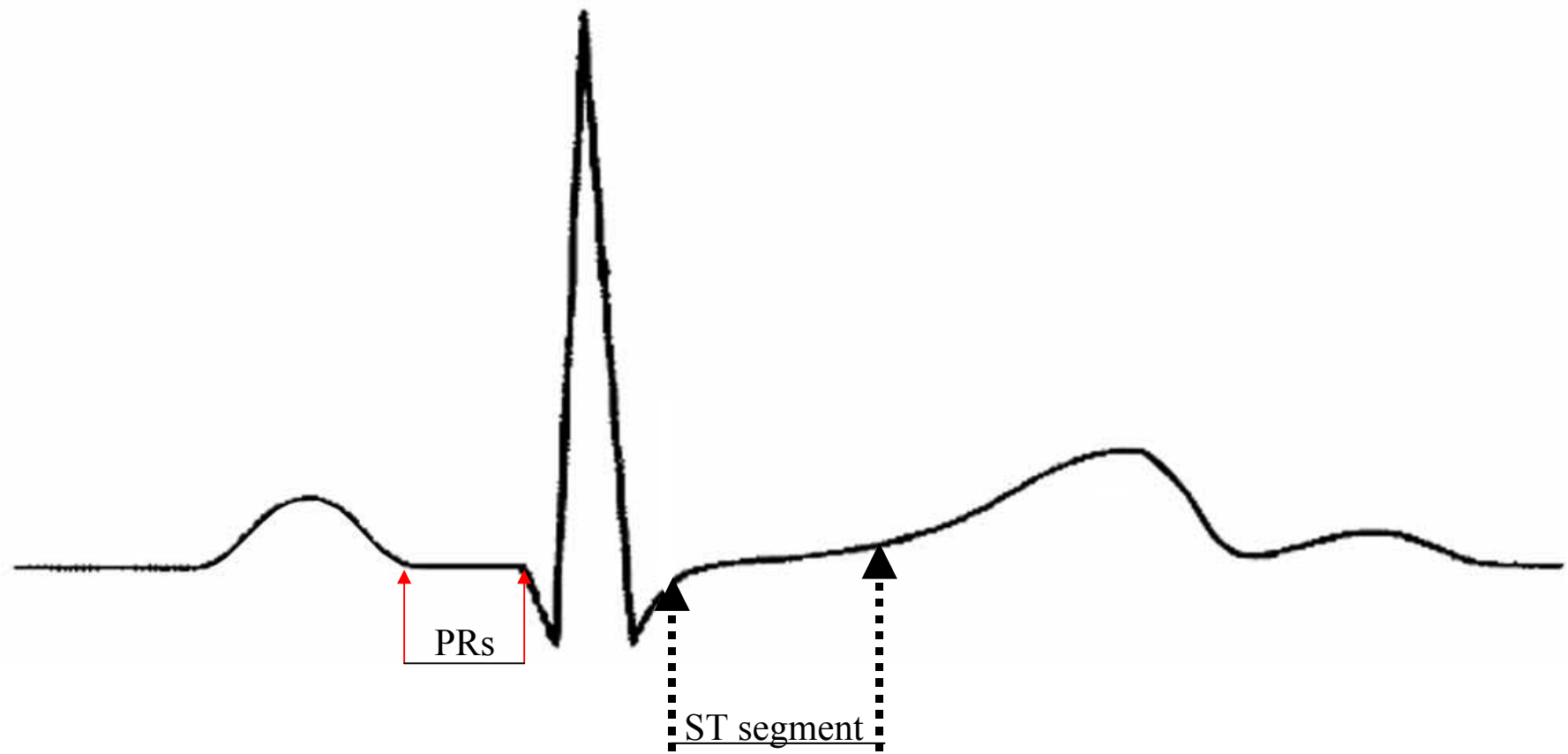
OTHERS DENOMINATIONS:

- ✓ **PQ segment (PQs)**
- ✓ **STa segment or atrial ST**
- ✓ **PTa-segment**

PR (PRs), PQ segment (PQs), STa segment or PTa-segment

PRs: from the end of P wave to the onset of QRS complex.

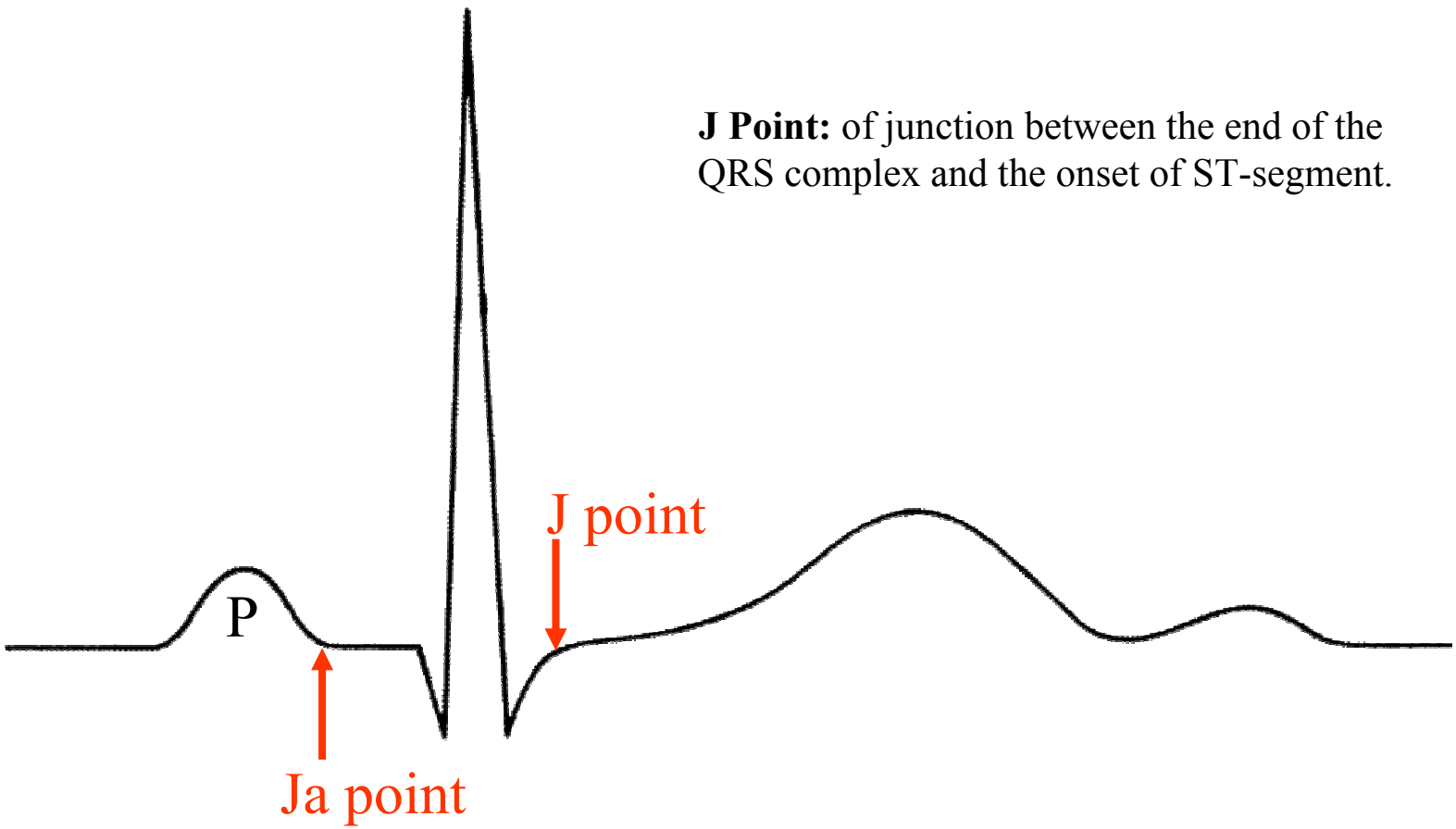
ST- Segment



It stretches from the J point (union of the ST segment with the end of QRS) until the onset of the T wave, which is usually hard to determine.

Concept of ST segment.

Ja point & J point



Ja Point: junction between the end of the P wave and the onset of PRs.

Ta OR TP WAVE

Normal location of atrial repolarization (Ta or TP wave). It coincides with ventricular depolarization (QRS complex), what explains its absence for being concealed by the ventricular phenomenon. Ta wave usually not visible. It is concealed by QRS. It represents atrial repolarization.

Its polarity is opposite to the P wave and its magnitude is 100 to 200 mμV.

Sometimes it may appear in the PR segment, ST segment and the T wave.

During exercise, it may in theory, cause ST segment depression and resemble myocardial ischemia (1)

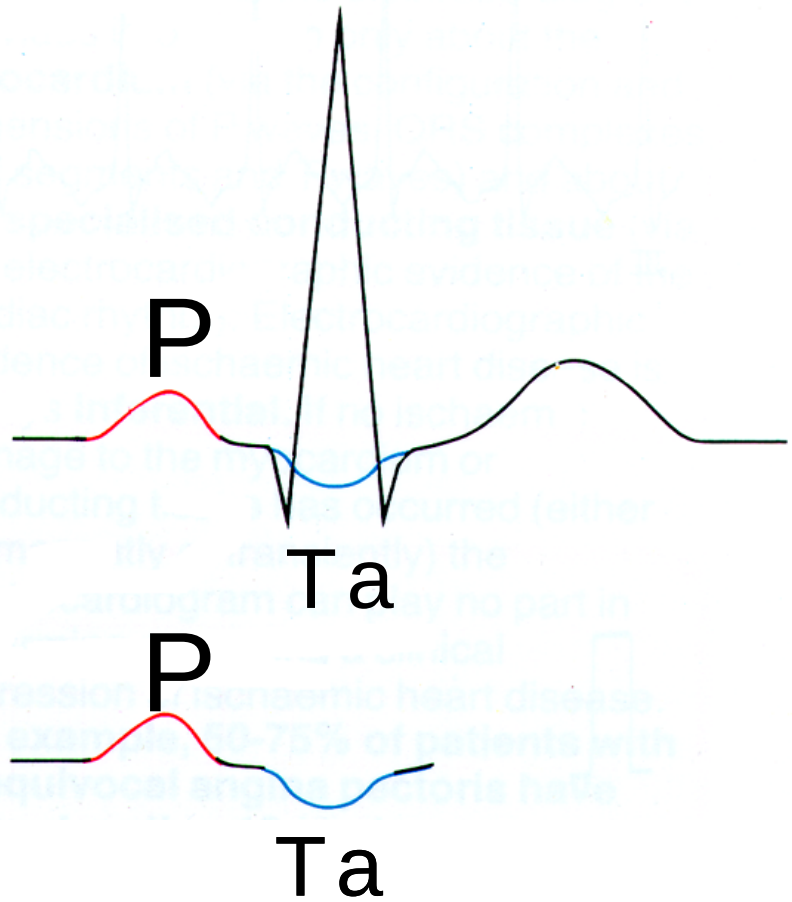
False positive must be suspected in the presence of:

Important PR segment depression in maximal strain;

Longer time of exercise and maximal strain faster than those truly positive;

Absence of effort-induced pain;

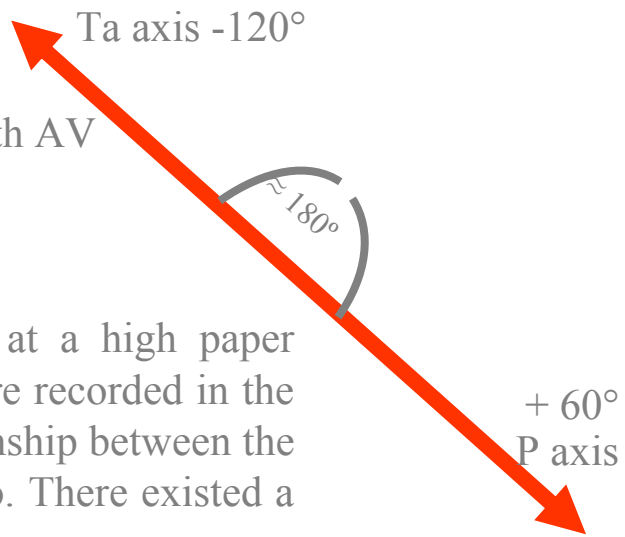
P wave of voltage higher in maximal strain.



1. Sapin PM, et al. J Am Coll Cardiol. 1991; 18: 127-135.

Ta wave has a saucer-like shape

Normal Ta wave axis is near **-120° ≈ 180° opposite to P axis**



Hayashi et al (1) studied the P and the Ta waves of two patient groups with AV block:
Group A: patients minimal clinical evidence of heart disease
Group B: patients with more severe disease.
Waves were magnified with a direct-current amplifier and recorded at a high paper speed. The authors verified that in Group A the P and the Ta waves were recorded in the opposite direction (near 180º) in every lead and there was a linear relationship between the amplitude of the P and the Ta waves. The atrial gradient was nearly zero. There existed a positive correlation between the P + Ta time and the PP interval

In Group B patients there were significant differences between P wave and Ta wave with respect to form, polarity, amplitude, duration, and the relationship between the Ta and the P waves. The atrial gradient was markedly large. Ta loop may be very useful in separating normal from diseased atria in individuals with AV block. There are some frequency differences between the Ta wave and the QRS complex. If the Ta wave could be extracted from the QRS complex by the use of some kind of filter when A-V block dose not exist, most of the Ta wave could be visualized. This, along with high fidelity recording techniques, may help detect atrial abnormalities in patients without AV block. Attention should be paid to the deviation of the PR segment caused by the Ta wave in daily ECG's to detect atrial abnormalities. The Ta wave extends into the ST segment and, while describing the deviation of the ST segment, the influence of the Ta wave should be kept in mind.

1. Hayashi H, Okajima M, Yamada K.Atrial T(Ta) wave and atrial gradient in patients with A-V block. Am Heart J. 1976 Jun;91:689-98. Am Heart J. 1976 Apr;91:492-500.

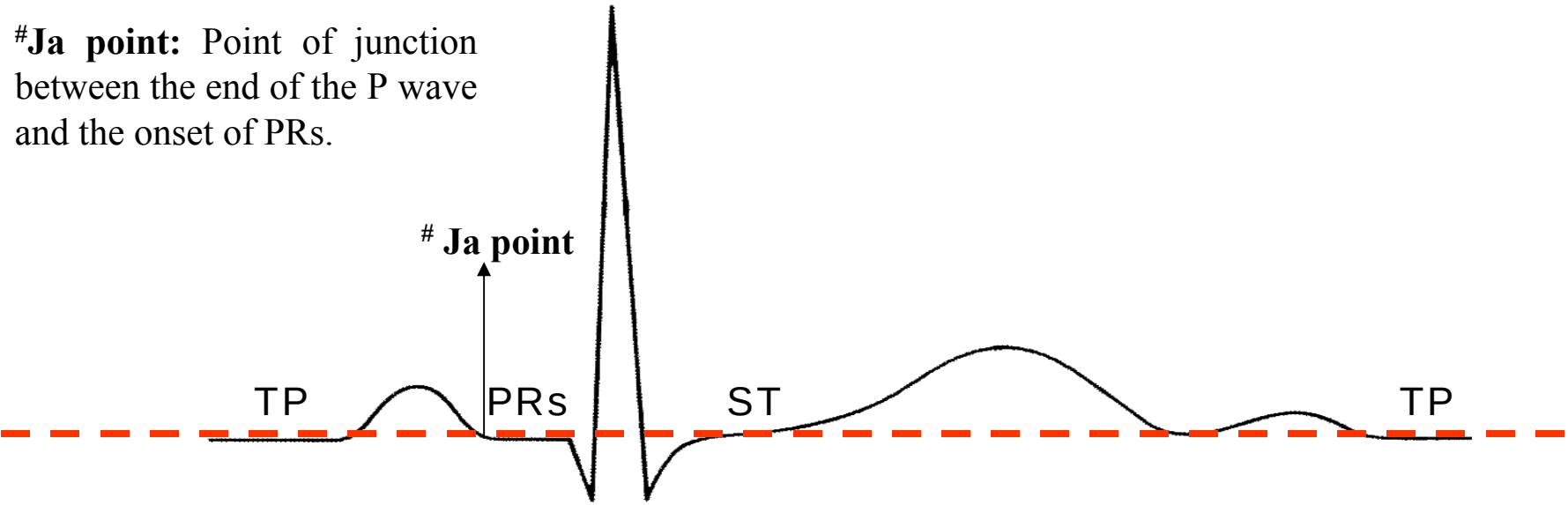
Holmqvist et al(1) studied, 40 consecutive patients with third-degree AV block to better analyze the Ta wave.

In this population the Ta wave had the opposite polarity, a duration two to three times that, of the P wave and Ta peak may occasionally be located in the PR interval during normal AV conduction, it is unlikely that enough information can be obtained from analysis of this segment to differentiate normal from abnormal atrial repolarization. Hence, an algorithm for QRST cancellation during sinus rhythm is needed to further improve analysis.

1. Holmqvist F, Carlson J, Platonov PG.Detailed ECG analysis of atrial repolarization in humans.Ann Noninvasiv e Electrocardiol. 2009 Jan;14:13-18.

CORRELATION OF LEVEL BETWEEN PRs, ST & TP

#Ja point: Point of junction between the end of the P wave and the onset of PRs.



The PR segment (PRs) is leveled when it is at the same level of the PR segment of the beat being studied.

Usually, PRs (end of P wave up to QRS complex onset), ST segment (from J point or the end of QRS up to the beginning of the T wave) and TP (from the end of the T wave up to the P wave of the following cycle) segments are at the same level. The figure shows a normal ECG and a line of dots pointing out the level of the three segments: **PR, ST** and **TP**.

1. **Riera AR.** Correlation of levels between PRS or PQ, ST segment and TP. Cardiol J. 2008; 15:204-205.

ATRIAL INFARCTION ECG CRITERIA

1) Depression of the STa segment alone is not a reliable sigh unless the degree of depression is marked.
2) P shape with M or W morphology during the acute MI episode.
3) Frequently atrial arrhythmias (35% of cases): Higher incidence of supraventricular arrhythmias in acute atrial fibrillation compared with ventricular infarction, atrial flutter, supraventricular tachycardia, changing pacemaker, junctional rhythm, sinus bradycardia, and AV conduction disturbances. Ischemia of the sinus node due to coronary occlusion proximal to the origin of the sinus node artery is a likely cause of arrhythmias[1].
4) Atrial arrhythmias (present in 35% of cases): ischemia of the sinus node due to coronary occlusion proximal to the origin of the sinus node artery is a likely cause of arrhythmias[1].
5) Pump failure of the right and left ventricle
6) Atrial wall rupture
7) Thromboembolization[2]

1) Kyriakidis M. Chest. 1992;101:944-947.
2) Neven K, et al. J Cardiovasc Electrophysiol. 2003;14:306-308.

ATRIAL INFARCTION CRITERIA[1]

I) MAJOR CRITERIA

1) PRs elevation >0.5mm in leads V5 and V6 with reciprocal depression of PRs in V1 and V2 leads.
2) PRs elevation >0.5mm in leads I with reciprocal depressions in II and III.
3) PRs depression >1.5mm in precordial leads an 1.2mm in I, II, associated with any atrial arrhtymia.

II) MINOR CRITERIA

1) Abnormal P waves, flattening of P-wave in M, flattening of P-wave in W, irregular or notched P wave.

First conclusion: In the present case there are clear PRs depression >1.5mm in precordial leads; consequently atrial infartion diagnose is made, consequently fulminant myocarditis, metastatic cancers with epicardia infiltration and severe hyperkalemia now they get more distant

1) Liu CK, et al. Circulation 1961;23:331-338.

Dr Raed noticied that the first documented rhythm was asystole followed by **pulseless electrical activity** PEA. What's the Difference Between Ventricular Fibrillation and asystole or cardiac Arrest?

Answer: Cardiac arrest is the term that describes when a heart stops pumping blood around. When a victim's heart stops pumping blood and he or she stops breathing (which usually happens within a few seconds of the heart stopping), the victim is considered clinically dead. If the victim's heart doesn't start again or CPR isn't started within 4 minutes of cardiac arrest, brain damage is almost guaranteed.

VF is a form of heart rhythm disturbance that causes cardiac arrest. During VF, the heart stops beating normally and simply begins quivering. No blood is pushed through because there is no squeezing action.

If the patient to go into cardiac arrest, VF is the best case scenario. VF responds very well to electric shock, which stops the quivering and lets the heart's normal electrical activity start over. That's why we call those shock boxes *de*fibrillators.

VF causes cardiac arrest, but not all or cardiac arrest is caused by VF. Asystole or mechanic electro dissociation is the other possibility. Based on a large-scaled population-based cohort of out-of-hospital cardiac arrest , subsequent VF with defibrillation was associated with better outcomes among patients with an initial non-shockable rhythm(1). In cardiac arrest, pulseless electrical activity (PEA) such as the present case is a challenging clinical syndrome. Epinefrine has notable clinical effects during advanced life support in patients with initial PEA. The drug extends the time window for return of spontaneous circulation to develop, but also renders the patient more unstable. Further research should investigate the optimal dose, timing and mode of adrenaline administration during advanced life support.(2)

1. **Kajino K, Iwami T, Daya M, et al. Subsequent ventricular fibrillation and survival in out-of-hospital cardiac arrests presenting with PEA or asystole. Resuscitation. 2008 Oct;79:34-40.**
2. **Nordseth T, Olasveengen TM, Kvaløy JT, et al. Dynamic effects of adrenaline (epinephrine) in out-of-hospital cardiac arrest with initial pulseless electrical activity (PEA). Resuscitation. 2012 Aug;83:946-952.**

ECG classical patterns of Left Main Coronary Artery (LMCA) occlusion:

1. **ST segment elevation in aVR, and V1**
2. ST segment elevation in aVR > V1(2) However, ST elevation in aVR is not entirely specific to LMCA occlusion. It may also be seen with proximal LAD occlusion and severe triple-vessel disease The mechanism of ST segment elevation in aVR is electrically opposite to the left-sided leads I, II, aVL and V4-6; therefore ST depression in these leads will produce reciprocal ST elevation in aVR. Lead aVR also directly records electrical activity from the right upper portion of the heart, including the RVOT and the basal portion of the interventricular septum; infarction in this area could theoretically produce ST elevation in aVR. ST elevation in aVR result from two possible mechanisms: Diffuse subendocardial ischaemia (producing reciprocal change in aVR) or transmural ischaemia / infarction of the basal interventricular septum (e.g. due to a proximal occlusion within the left coronary system) Magnitude of ST elevation in aVR is correlated with mortality in patients with ACSs:

   STE in aVR ≥ 0.5mm is associated with a 4-fold increase in mortality

   STE in aVR ≥ 1mm is associated with a 6- to 7-fold increase in mortality(4)

   STE in aVR ≥ 1.5mm is associated with mortalities ranging from 20-75%
3. The absence of aVR STE appears to exclude LMCA as the underlying cause in NSTEMI; in the context of anterior STEMI, its presence indicates a culprit lesion in the proximal segment of LAD(6).
4. Ischemic evidences in inferobasal* wall: depression of the ST segment in II and from V4 to V6
5. ST segment depression in II or in inferior leads II>III
6. Depression of ST segment in V6 > ST segment elevation in V1
7. Diffuse ST segment depression in the inferolateral leads with inverted T waves
8. Eventually observation of life-threatening tachyarrhythmias and conduction disturbances such as RBBB, LAFB and/or LSFB.

Severe3-vessel disease (LM/3VD) is defined as ≥75% stenosis of LM and/or 3VD with ≥90% stenosis in ≥2 proximal lesions of the LAD and other major epicardial arteries. Kosuge et al (4) studied 3 groups of patients according to angiographic findings:

1) No LM/3VD (n = 460),
2) LM/3VD but not severe LM/3VD (n = 57), and
3) Severe LM/3VD (n = 55).

Severe LM/3VD was associated with a higher rate of urgent CABG compared to no LM/3VD and LM/3VD but not severe LM/3VD (46%, 2%, and 2%, p <0.001). On multivariate analysis, degree of ST-segment elevation in lead aVR was the strongest predictor of severe LM/3VD, followed by positive troponin T level.

ST-segment elevation ≥1.0 mm in lead aVR best identified severe LM/3VD with 80% sensitivity, 93% specificity, 56% positive predictive value, and 98% negative predictive value.

In conclusion, ST-segment elevation ≥1.0 mm in lead aVR on admission ECG is highly suggestive of severe LM/3VD in patients with NSTE-ACS. Selected patients with this finding might benefit from promptly undergoing angiography, withholding clopidogrel to allow early CABG.

1. **Nikus KC. Acute total occlusion of the left main coronary artery with emphasis on electrocardiographic manifestations. Timely Top Med Cardiovasc Dis. 2007;11:E22**
2. **Nikus KC, Eskola MJ. Electrocardiogram patterns in acute left main coronary artery occlusion. J Electrocardiol. 2008 Nov-Dec;4:626-629.**
3. **Gaitonde RS, Sharma N, Ali-Hasan S, et al. Prediction of significant left main coronary artery stenosis by the 12-lead electrocardiogram in patients with rest angina pectoris and the withholding of clopidogrel therapy. Am J Cardiol. 2003;92:846.**
4. **Kosuge M, Ebina T, Hibi K, et al. An early and simple predictor of severe left main and/or three-vessel disease in patients with non-ST-segment elevation acute coronary syndrome. Am J Cardiol 2011 Feb 15;107:495-500.**
5. **Dwyer N, Kanani R. Images in clinical medicine. Left main coronary artery thrombosis. N Engl J Med. 2012 Apr 5;366(14):e21.**
6. **Kühl JT, Berg RM. Utility of lead aVR for identifying the culprit lesion in acute myocardial infarction. Ann Noninvasive Electrocardiol. 2009 Jul;14:219-225.**

Despite current guideline-based ECG criteria, challenges remain in optimizing the rate of appropriate catheterization laboratory activation.
The HORIZONS-AMI trial(1) enrolled 3,602 patients with chest pain consistent with AMI ECG and angiographic core laboratory databases were analyzed for correlation between the qualifying study ECG and the baseline coronary angiogram.

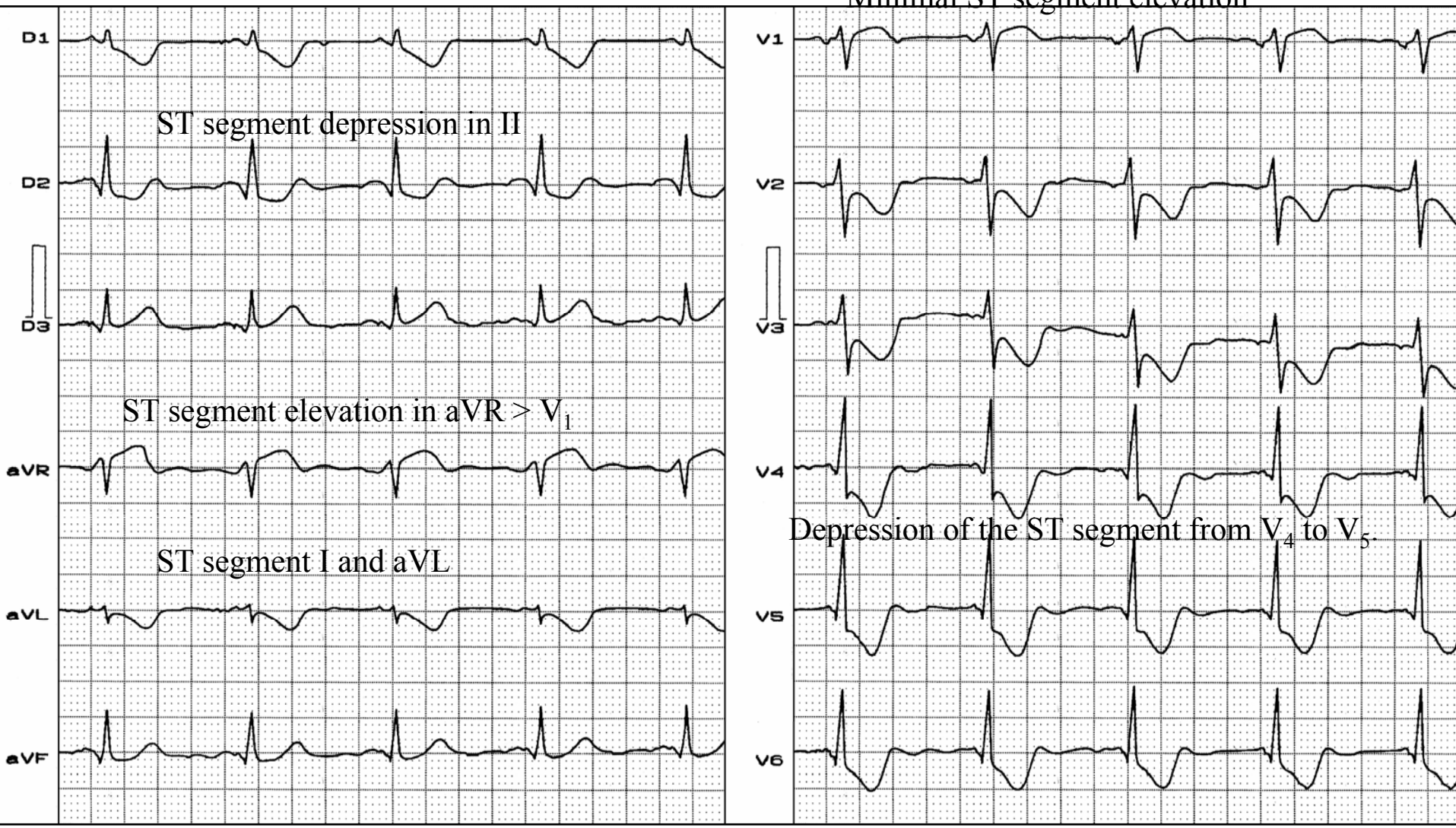
1. LAD occlusion manifested in >80% of cases as ST-segment elevation in leads V2 and V3.
2. The culprit vessel was the RCA and LCx in 75 and 25% of cases, respectively, for inferior MI ECG patterns.
3. The study threshold of ≥ 1.0 mm ST-segment elevation in ≥ 2 contiguous ECG leads was not met in 189 (5.3%) patients.
4. When stratified by culprit artery, the prevalence of reciprocal ST-segment depression ranged from 24 to 88%, being least common for lesions in the mid- and distal LAD.. Despite study eligibility, no posterior MIs were enrolled.
5. Only 36 LBBB cases were identified (25% of whom did not undergo PCI), and
6. 5 of 11 LMCA occlusions (45%) had ST-segment elevation in lead aVR.

The HORIZONS-AMI trial confirms prior ischemic ECG findings predicted by vectorcardiography, validates certain ECG patterns as reliable surrogate markers for acute coronary occlusion, and provides novel insights correlating index ECG ischemic changes and pre-intervention coronary angiography. These results may enhance the rate of appropriate catheterization laboratory activation.

1. **Rokos IC, Farkouh ME, Reiffel J, et al. Correlation between index electrocardiographic patterns and pre-intervention angiographic findings: insights from the HORIZONS-AMI trial. Catheter Cardiovasc Interv. 2012 Jun 1;79:1092-1098.**

TYPICAL ECG PATTERN OF LMCA OCCLUSION

Diffuse ST segment depression in the inferolateral leads



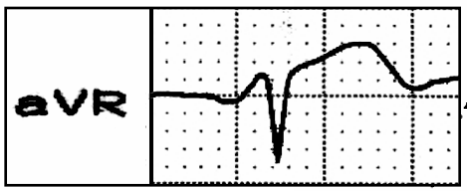
Why this pattern is observed?

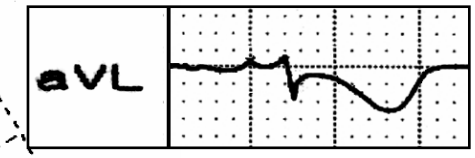
ST segment depression in V_6 > ST segment elevation in V_1.

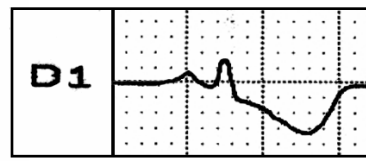
The ST vector lesion pointing to aVR

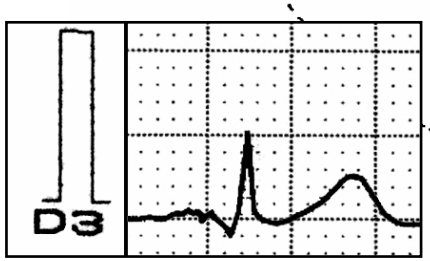
Frontal

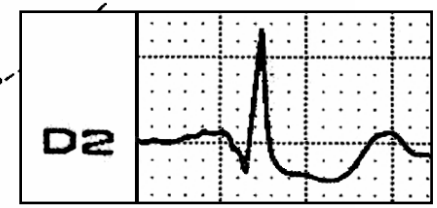
ST segment elevation

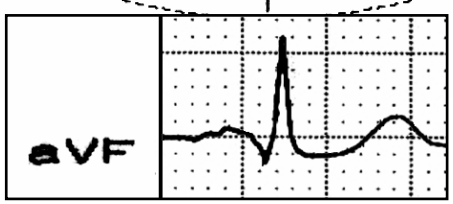


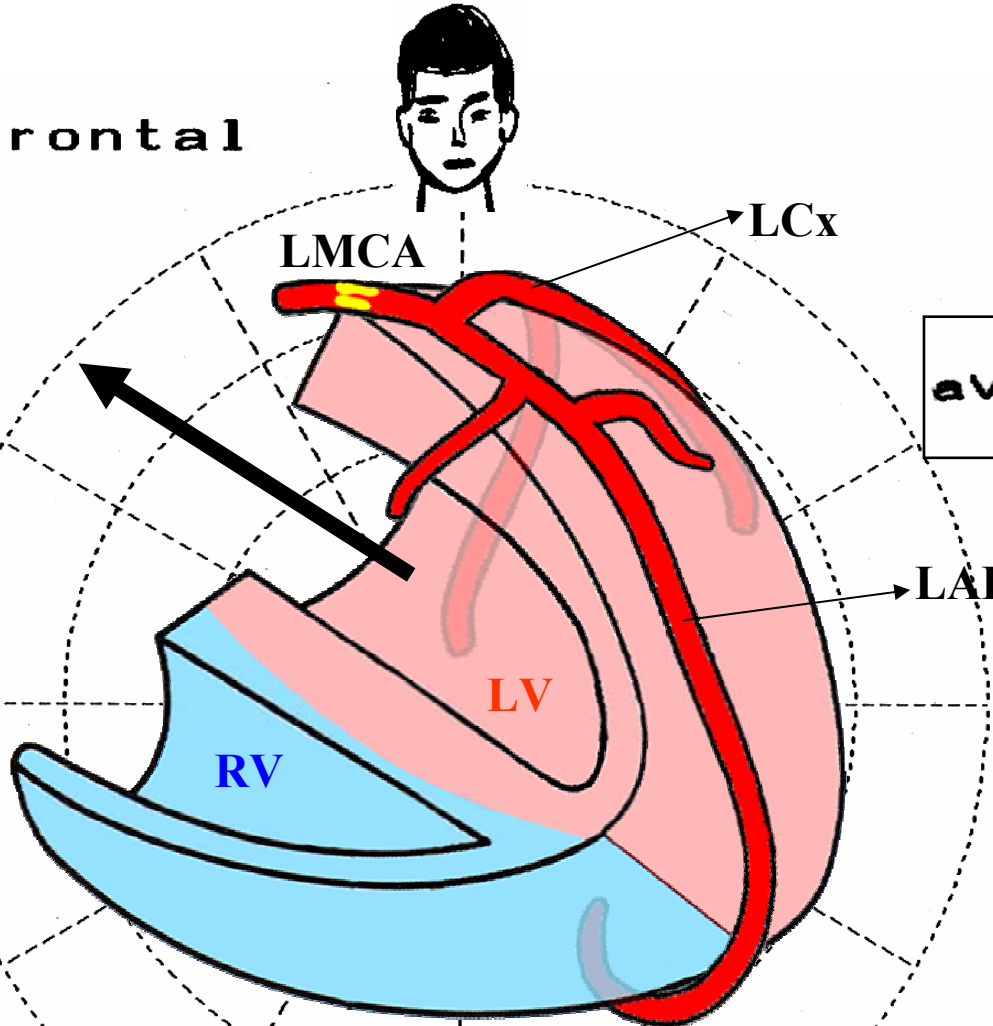






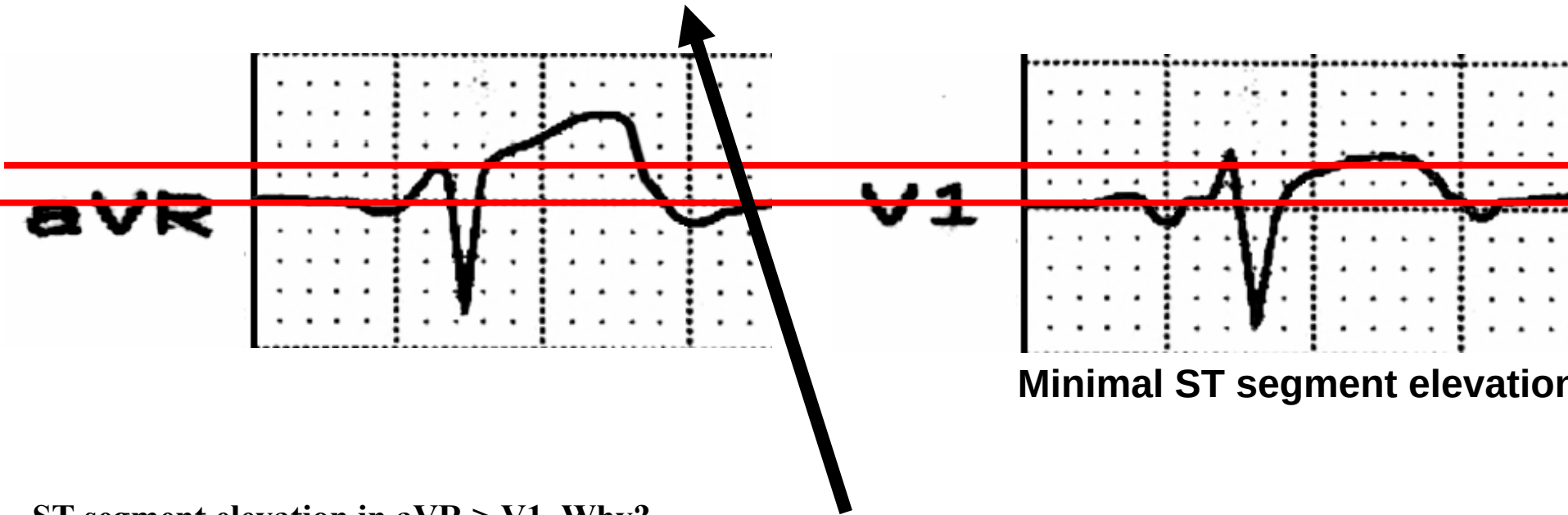






ST segment depression in II I and aVL

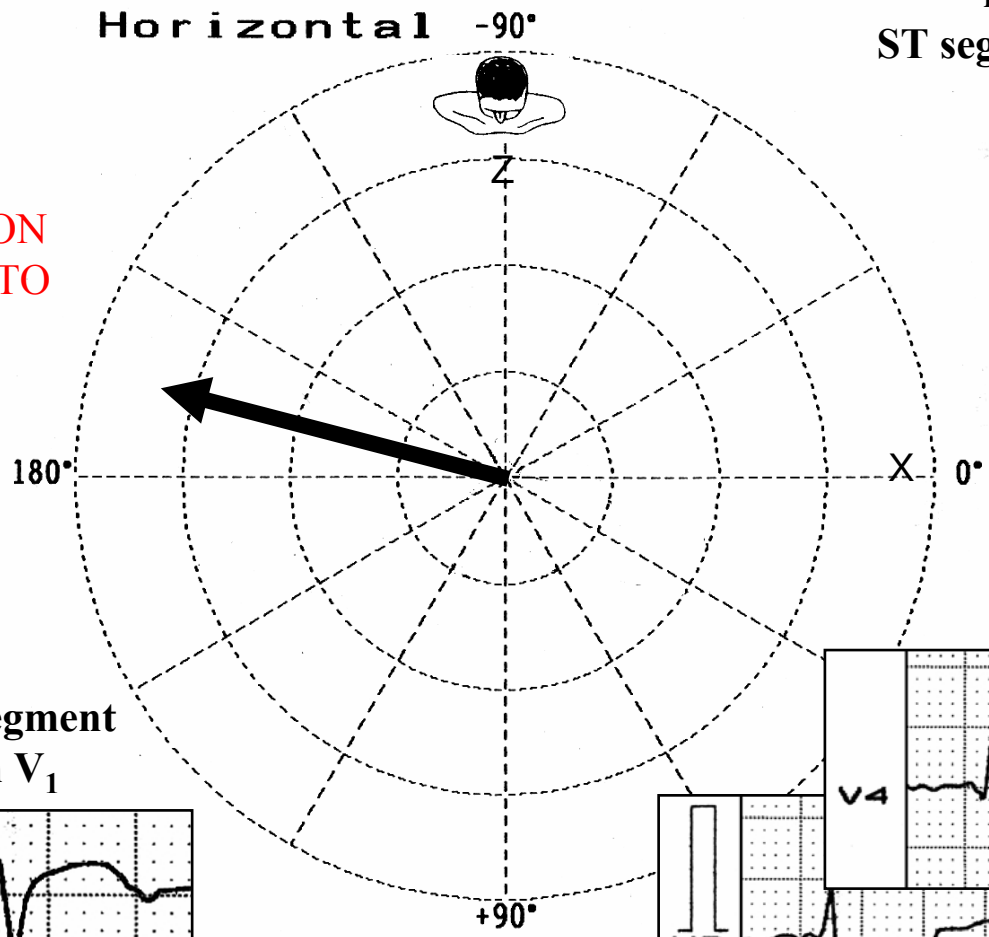
aVR STSE > V_1 STSE



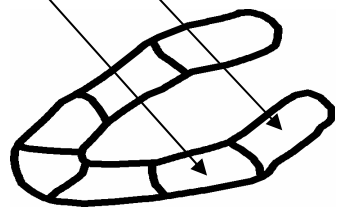
ST segment elevation in aVR > V1. Why?
Because ST segment lesion vector is directed to upward and rightward, pointing to aVR lead(RVOT)

1. **Nikus KC, Eskola MJ. Electrocardiogram patterns in acute left main coronary artery occlusion. J Electrocardiol. 2008 Nov-Dec;4:626-629.**

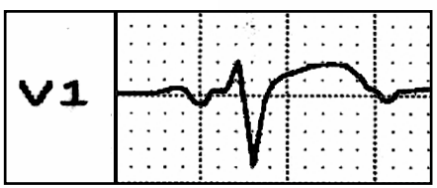
ST SEGMENT LESION VECTOR POINTED TO RIGHT AND BACKWARD

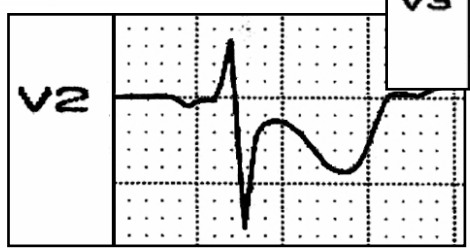


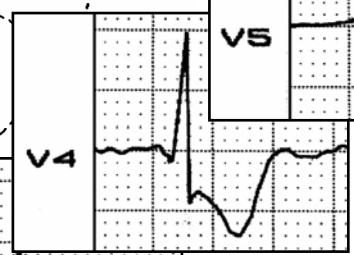
Ischemic evidences on inferobasal wall: ST segment depression from V_4 to V_5

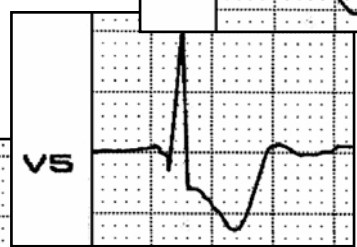


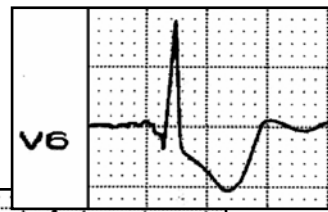
Minimal ST segment elevation in V_1



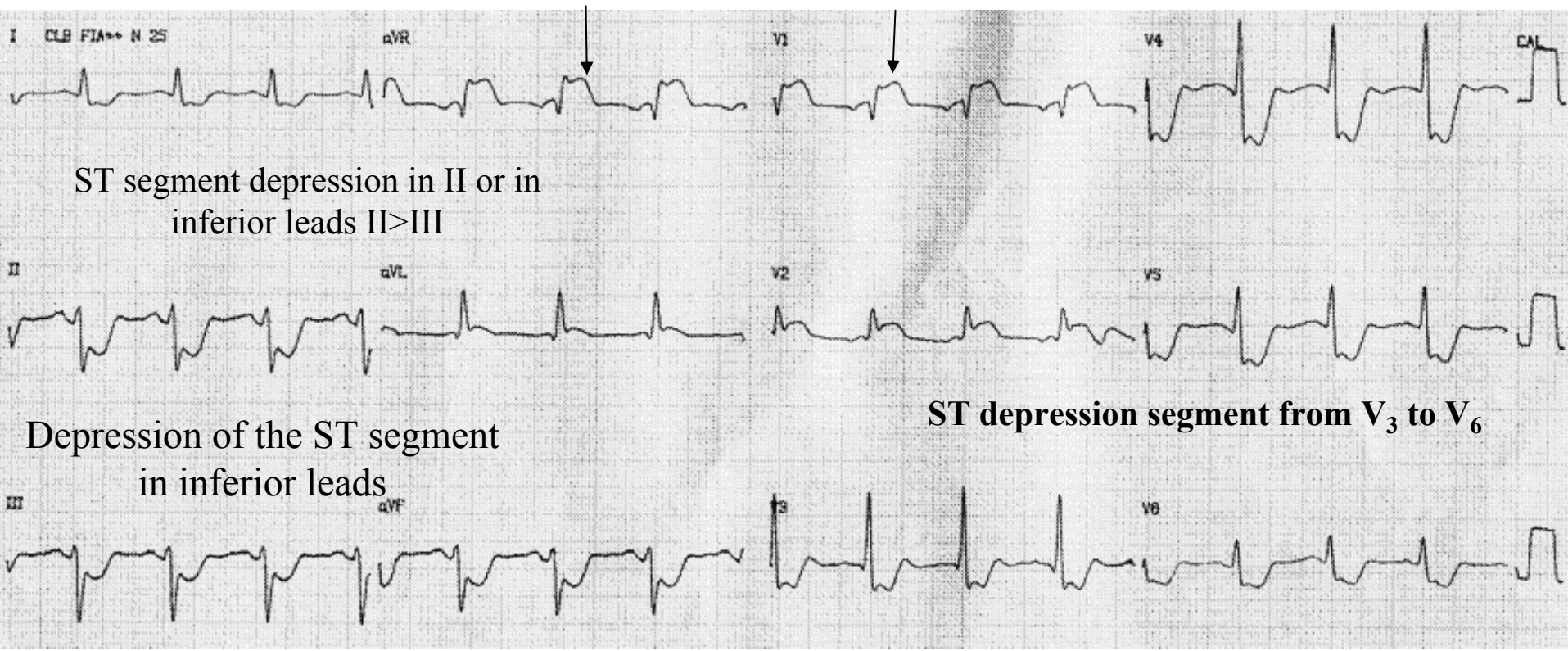






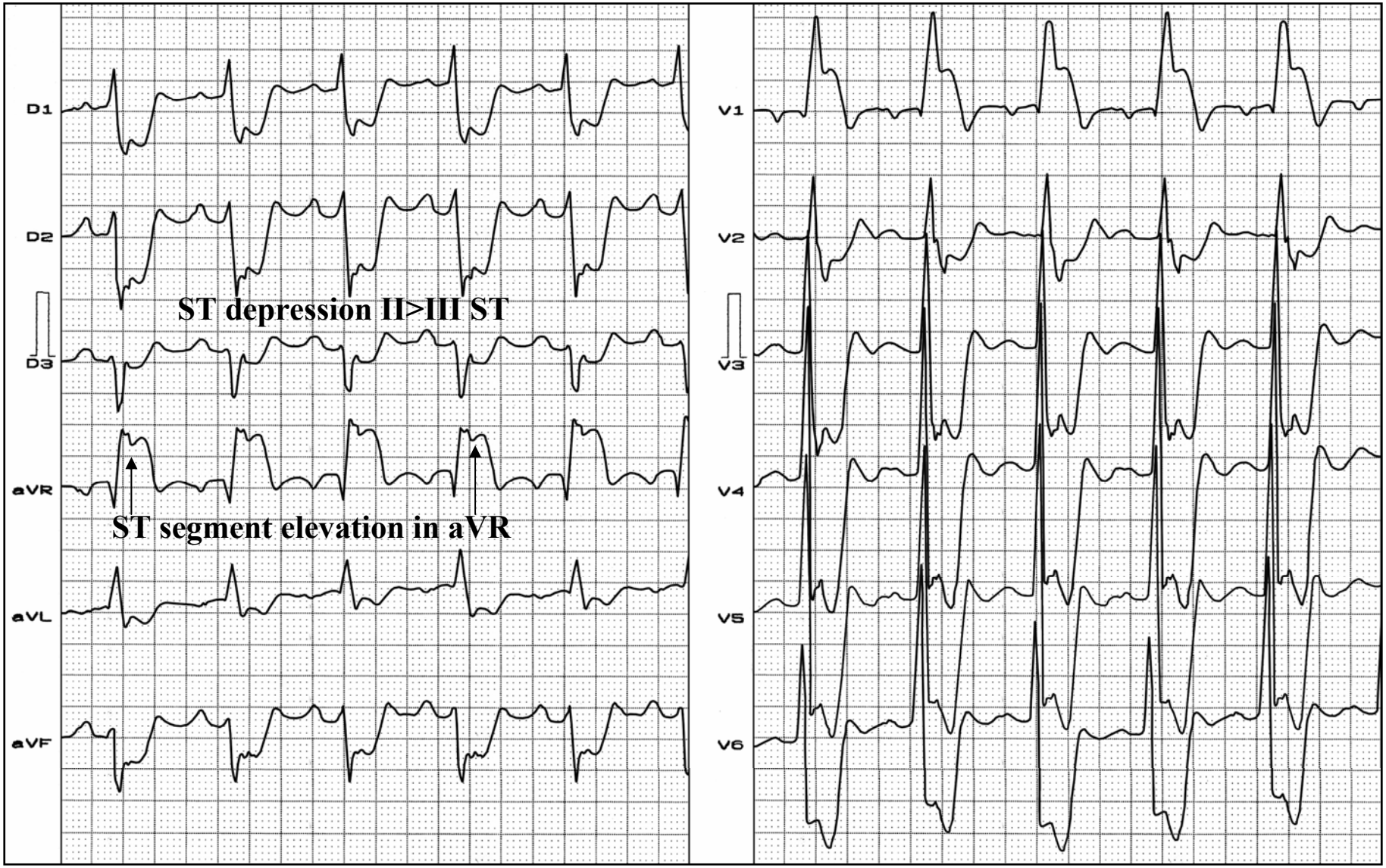


Depression of the ST segment from V_2 to V_6



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

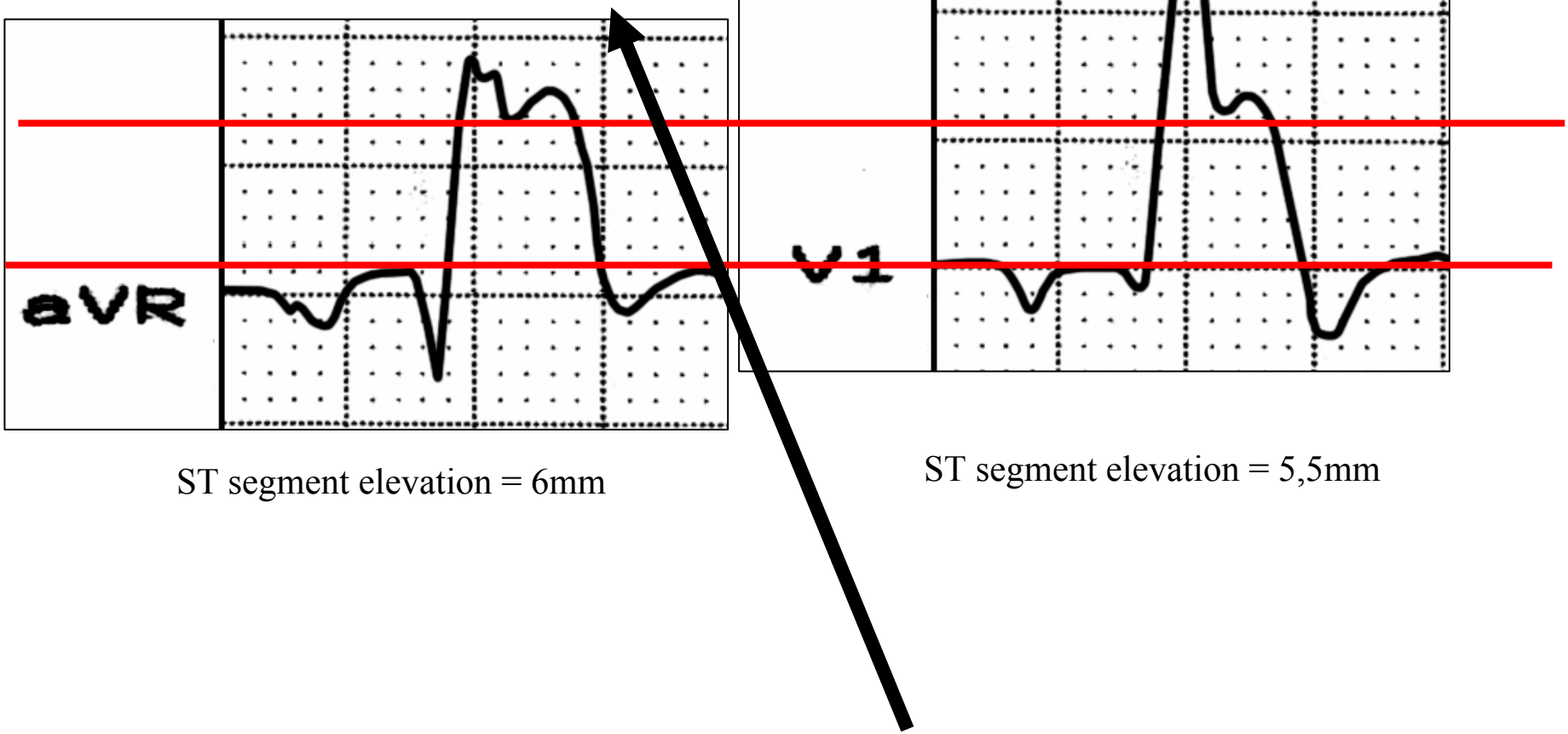
LMCA Occlusion complicated with Complete RBBB.



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

ST segment elevation in aVR > V_1

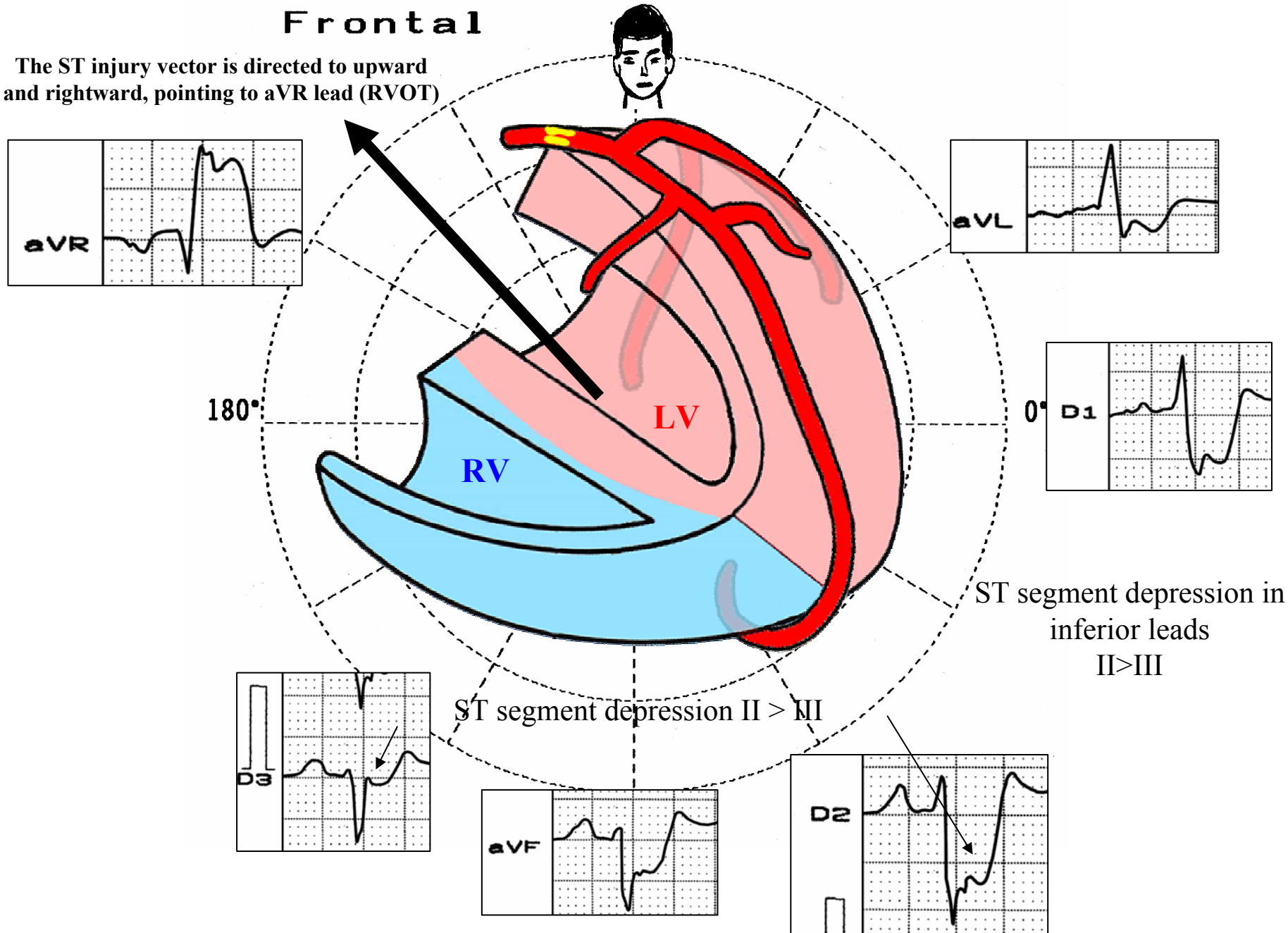
The ST injury vector is directed to upward and rightward, pointing to aVR lead (RVOT)



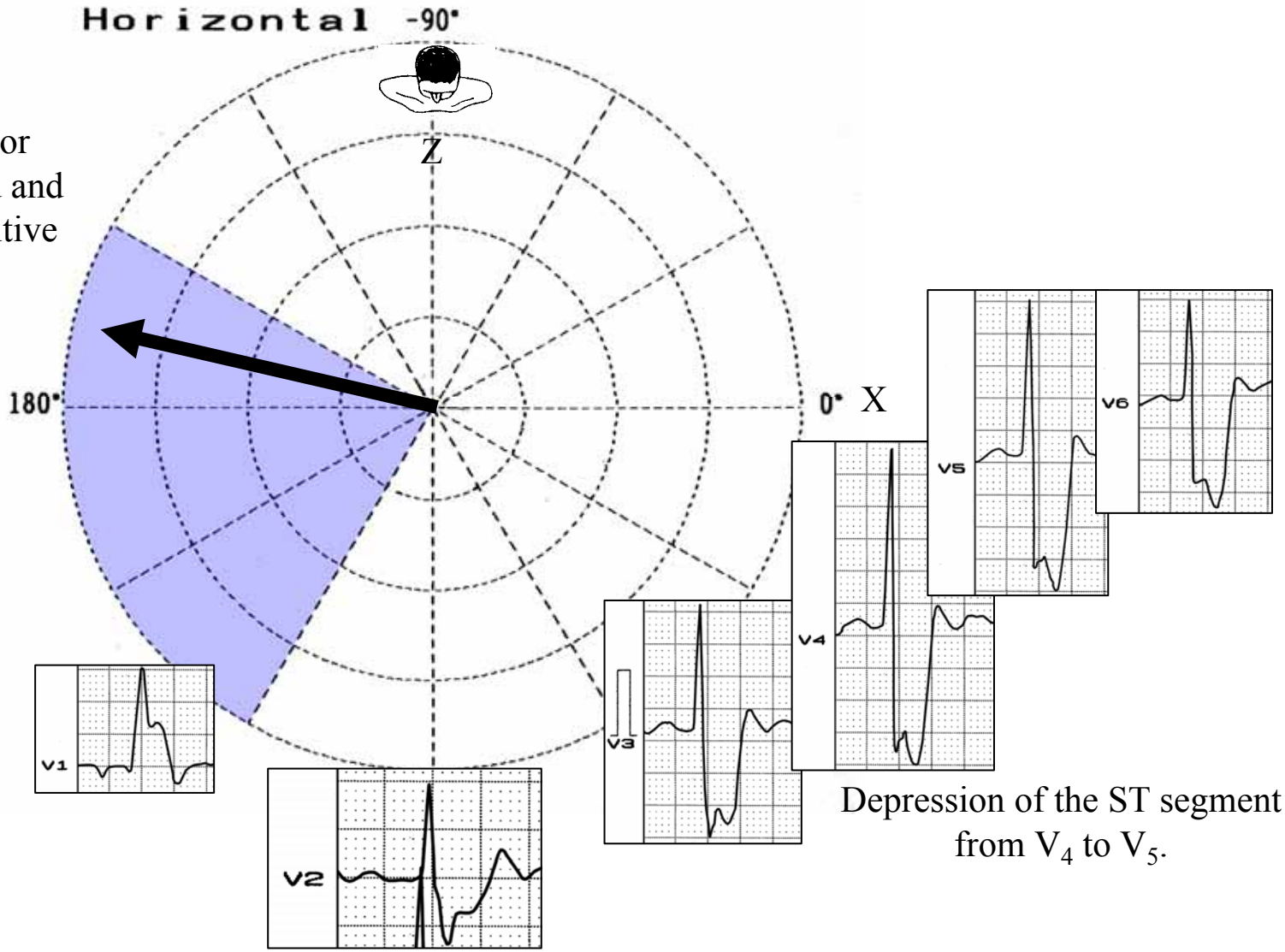
ST segment elevation = 6mm

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.**

Acute Coronary Syndrome (ACS)

Acute coronary syndrome (ACS) covers the spectrum of clinical conditions:
Unstable angina: not associated with heart muscle damage.

Non-ST segment elevation myocardial infarction **(NSTEMI)** if the ECG does not show typical changes, the term "non-ST segment elevation ACS" is applied. The patient may still have suffered a "non-ST elevation MI" (NSTEMI). The accepted management of unstable angina and acute coronary syndrome is therefore empirical treatment with aspirin, heparin (usually a low-molecular weight heparin such as enoxaparin) and clopidogrel, with intravenous glyceryl trinitrate and opioids if the pain persists.A blood test is generally performed for cardiac troponins twelve hours after onset of the pain. If this is positive, coronary angiography is typically performed on an urgent basis, as this is highly predictive of a heart attack in the near-future. If the troponin is negative, a treadmill exercise test or a thallium scintigram may be requested.

ST segment elevation myocardial infarction **(STEMI).** or new left bundle branch block or a true posterior MI pattern Treatment for a heart attack in the form of primary angioplasty(PCI) or thrombolysis with thrombolytics is indicated immediately. If the ECG confirms changes suggestive of MI (ST elevations in specific leads, a new left bundle branch block or a true posterior MI pattern or basal inferior in new nomenclature), thrombolytics may be administered or primary coronary angioplasty may be performed. In the former, medication is injected that stimulates fibrinolysis, destroying blood clots obstructing the coronary arteries. In the latter, a flexible catheter is passed via the femoral or radial arteries and advanced to the heart to identify blockages in the coronaries. When occlusions are found, they can be intervened upon mechanically with angioplasty and perhaps stent deployment if a lesion, termed the *culprit* lesion, is thought to be causing myocardial damage.

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.**

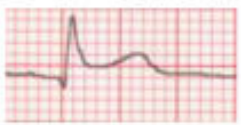
The current guidelines for the ECG diagnosis of acute myocardial infarction (AMI) require at least 1 mm (0.1 mV) of ST segment elevation in the limb leads, and at least 2 mm elevation in the precordial leads. These elevations must be present in anatomically contiguous leads.(1)
(I, aVL, V5, V6 correspond to the lateral wall; V1-V4 correspond to the anterior wall; II, III, aVF correspond to the inferior wall.) This criterion is problematic, however, as acute MI is not the most common cause of ST segment elevation in chest pain patients.(2) Over 90% of healthy men have at least 1 mm (0.1 mV) of ST segment elevation in at least one precordial lead.(3) The clinician must therefore be well versed in recognizing the so-called ECG mimics of acute MI, which include LVH, LBBB, paced rhythm, early repolarization, pericarditis, hyperkalemia, and ventricular aneurysm. LBBB and pacing interferes with the ECG diagnosis of acute MI. The GUSTO investigators Sgarbossa et al. .(4) developed a set of criteria for identifying AMI in the presence of LBBB and paced rhythm. They include concordant ST segment elevation > 1 mm (0.1 mV), discordant ST segment elevation > 5 mm (0.5 mV), and concordant ST segment depression in the left precordial leads. The presence of reciprocal changes on the 12 lead ECG may help distinguish true AMI from the mimics of AMI. The contour of the ST segment may also be helpful, with a straight or upwardly convex (non-concave) ST segment favoring the diagnosis of AMI.(5)
The constellation of leads with ST segment elevation enables the clinician to identify what area of the heart is injured, which in turn helps predict the culprit artery.

1. **2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care - Part 8: Stabilization of the Patient With Acute Coronary Syndromes" (2005). *Circulation* 112: IV–89–IV–110.**
2. **Brady WJ, Perron AD, Martin ML, Beagle C, Aufderheide TP (January 2001). "Cause of ST segment abnormality in ED chest pain patients". *Am J Emerg Med* 19 (1): 25–8.]**
3. **Wang K, Asinger RW, Marriott HJ (November 2003). "ST-segment elevation in conditions other than acute myocardial infarction". *N. Engl. J. Med.* 349 (22): 2128–35**
4. **Sgarbossa EB, Pinski SL, Barbagelata A, *et al* (February 1996). "Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-1 (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators". *N. Engl. J. Med.* 334 : 481–487.**
5. **Brady WJ, Syverud SA, Beagle C, *et al* (October 2001). "Electrocardiographic ST-segment elevation: the diagnosis of acute myocardial infarction by morphologic analysis of the ST segment". *Acad Emerg Med* 8: 961–967.**

Acute Coronary Syndrome

Electrocardiogram

ST-elevation



No ST-elevation

Cardiac markers

negative

Unstable angina

positive

Myocardial infarction

STEMI

NSTEMI

Q-wave MI

non-Q-wave MI

Clinical risk scores

Calculating clinical risk scores can help to estimate the risk of morbidity and mortality in patients with suspected ACAS.. Widely employed systems include the GRACE (Global Registry of Acute Coronary Event) risk score, which evaluates the risk of death or myocardial infarction (MI) in patients with ACS, and the Thrombolysis In Myocardial Infarction(TIMI) risk score for STEMI and NSTEMI. These scores are based on the following risk factors:

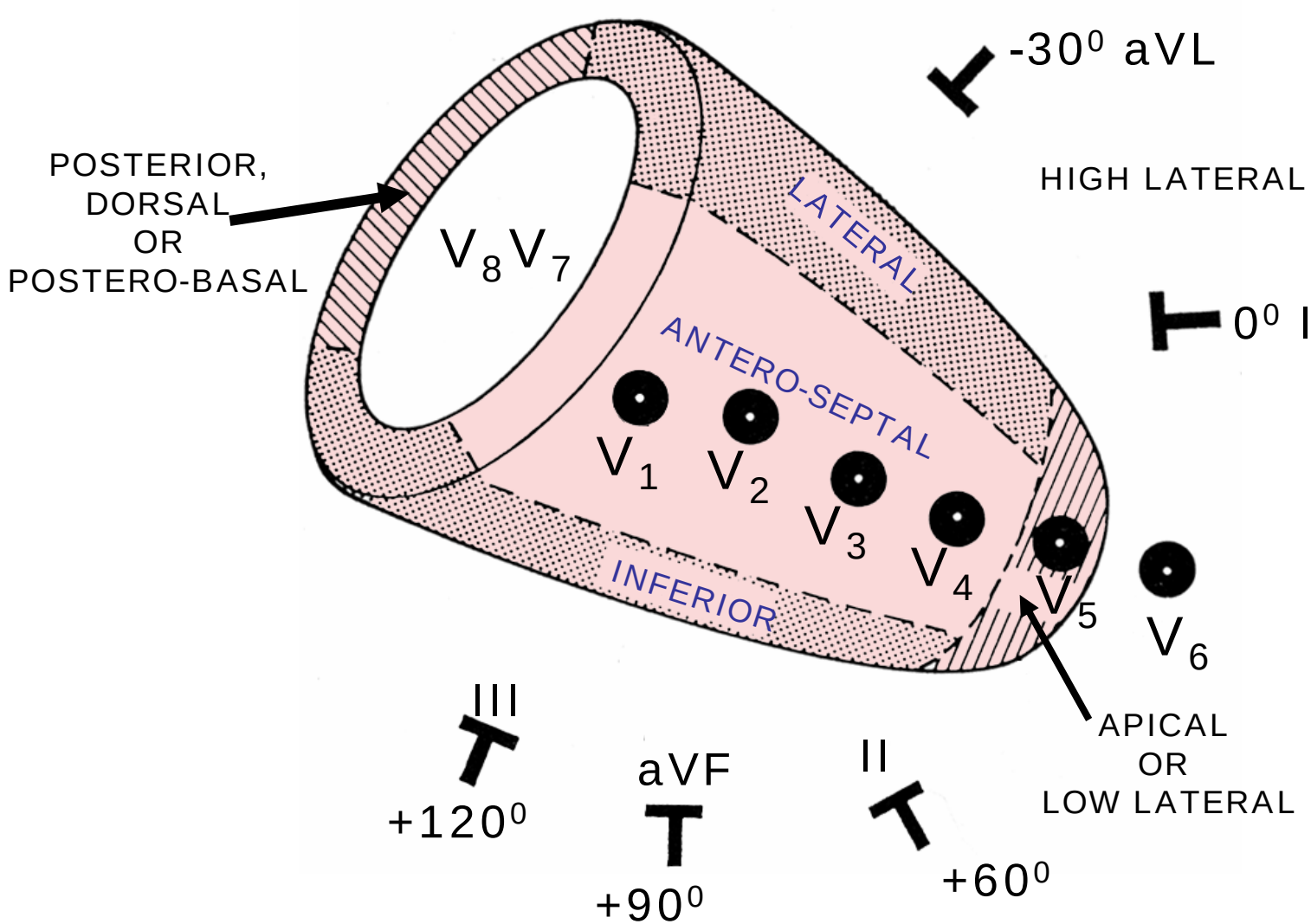
TIMI Risk Score Factors	GRACE Risk Score Factors
Age ≥ 65 years	Age
Over 3 risk factors for ischaemic heart disease (family history, hypertension, hypercholesterolaemia, diabetes, smoking)	Heart rate
Known ischaemic heart disease	Systolic blood pressure
Aspirin use in the past 7 days	Killip class
2 episodes of severe angina in 24 hours	Cardiac arrests
ST segment changes ≥ 0.5 mm	ST segment deviation
Positive troponin T	Cardiac biomarker status

While the TIMI score is designed to be used acutely, the GRACE risk model provides prognostic estimates both at the time of initial presentation to the hospital and for a period of up to 6 months following hospital discharge.Patients with complete occlusion of a coronary artery can often be identified by ST-segment elevation on ECG. This group, representing approximately one in three patients presenting with **ACS**, should receive prompt reperfusion treatment with fibrinolytic therapy or percutaneous coronary intervention (PCI). The remaining two thirds of **ACS** patients do not have ST-segment elevation on initial ECG and require further risk stratification.

Wall Affected	Leads Showing ST Segment Elevation	Leads Showing Reciprocal ST Segment Depression	Suspected Culprit Artery
Septal	V_1, V_2	None	Left Anterior Descending (LAD)
Anterior	V3, V4	None	Left Anterior Descending (LAD)
Anteroseptal	V1, V2, V3, V4	None	Left Anterior Descending (LAD)
Anterolateral	V3, V4, V5, V6, I, aVL	II, III, aVF	Left Anterior Descending (LAD), Circumflex (LCX), or Obtuse Marginal
Extensive anterior (Sometimes called Anteroseptal with Lateral extension)	V1,V2,V3, V4, V5, V6, I, aVL	II, III, aVF	Left main coronary artery (LCA)
Inferior	II, III, aVF	I, aVL	Right Coronary Artery (RCA) or Circumflex (LCX)
Lateral	I, aVL, V5, V6	II, III, aVF	Circumflex (LCX) or Obtuse Marginal
Posterior (Usually associated with Inferior or Lateral but can be isolated) Actual nomenclature basal inferior	V7, V8, V9	V1,V2,V3, V4	Posterior Descending (PDA) (branch of the RCA or Circumflex (LCX))
Right ventricular (Usually associated with Inferior)	II, III, aVF, V1, V_{4R}	I, aVL	Right Coronary Artery (RCA)

As the myocardial infarction evolves, there may be loss of R wave height and development of pathological Q waves (defined as Q waves deeper than 1 mm and wider than 1 mm.) T wave inversion may persist for months or even permanently following acute myocardial infarction. Typically, however, the T wave recovers, leaving a pathological Q wave as the only remaining evidence that an acute myocardial infarction has occurred.

ELECTROCARDIOGRAPHIC TOPOGRAPHIC CLASSIFICATION OF MYOCARDIAL INFARCTIONS (OLD NOMENCLATURE)



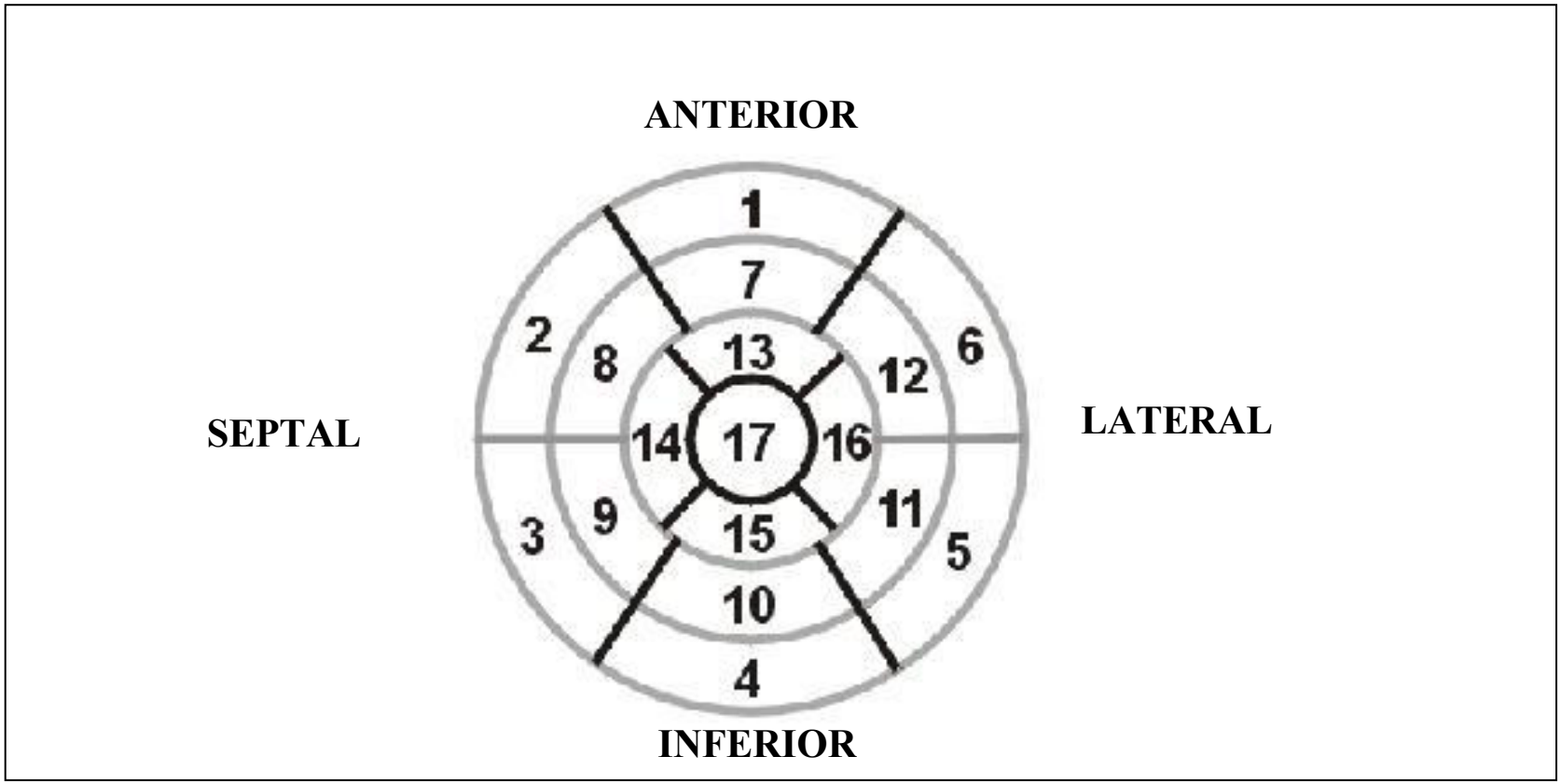
The cardiac cone, its sides and corresponding leads: Antero-septal: V1 to V4; Apical or low lateral: V5 and V6; High lateral: I and aVL; Inferior or diaphragmatic: II, III and aVF; Dorsal, posterior or postero-basal: V7 and V8.

ELECTROCARDIOGRAPHIC CLASSIFICATION OF Q-WAVE MYOCARDIAL INFARCTION

1) ***Anteroseptal, "strictly anterior" or of the middle and low region of the septum: V1 to V3 or V4. Anteriorly located;***
   - ✓ ***Anterior middle or middle third of the septum: V1 and V2.***
   - ✓ ***Inferior third of the septum: V3 and V4.***
2) ***Inferior or diaphragmatic: II, III and aVF***
3) ***Apical or low lateral: V5 and V6.***
4) ***High or superior lateral: aVL. Some authors add I.***
5) ***Dorsal, postero-basal or strictly posterior: V7, V8 and V9 and mirror or reciprocal image from V1 to V2 or V3.***
6) ***Extensive anterior V1 to V6 + I and aVL.***
7) ***Anterolateral: V4 to V6 + I and aVL.***
8) ***Deep septal or inferoseptal: II, III and aVF + V1 to V3.***
9) ***Inferolateral: II, III and aVF + V5 and V6 possibly I and aVL.***
10) ***Posterolateral: V7, V8 and V9 + V5 and V6 (possibly I and aVL)***
11) ***Posteroinferior: V7, V8 and V9 + II, III and aVF.***
12) ***Inferolaterodorsal (II, III and aVF + V5 and V6 + V7, V8 and V9).***
13) ***Right ventricle infarction: V3R, V4R V5R V6R + CR + V1 to V3 + DI.***
14) ***Atrial infarctions.***

Observation: To day, the terms posterior and high lateral infarction are considered incorrect and should be changed to lateral wall and limited anterolateral wall MI.

LEFT VENTRICULAR SEGMENTATION SHORT AXIS NEW NOMENCLATURE

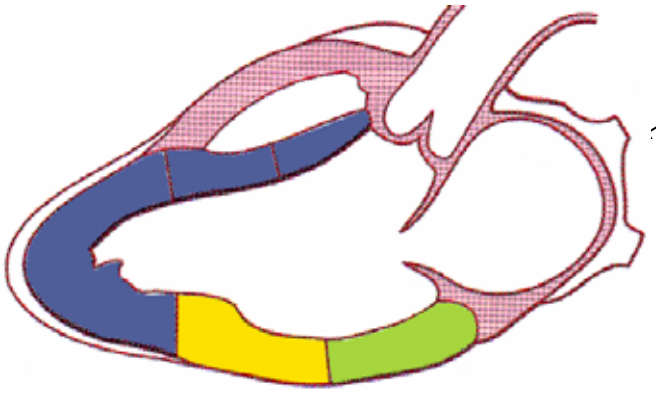


1 basal anterior
2 basal anteroseptal
3 basal inferior septal
4 basal inferior
5 basal inferolateral
6 basal anterolateral

7 mid anterior
8 mid anteroseptal
9 mid inferoseptal
10 mid inferior
11 mid inferolateral
12 mid anterolateral

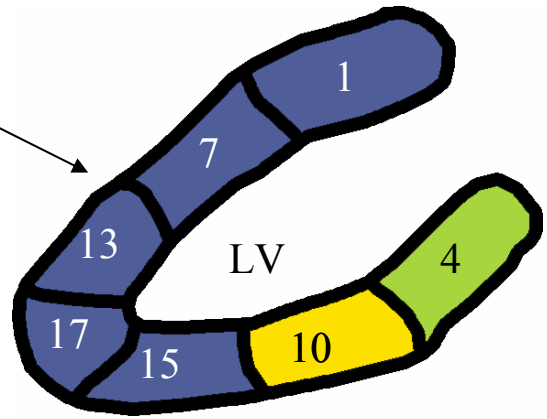
13 apical anterior
14 apical septal
15 apical inferior
16 apical lateral
17 apex

CURRENT MODEL OF VENTRICULAR SEGMENTATION AND WALL/ARTERY IRRIGATION



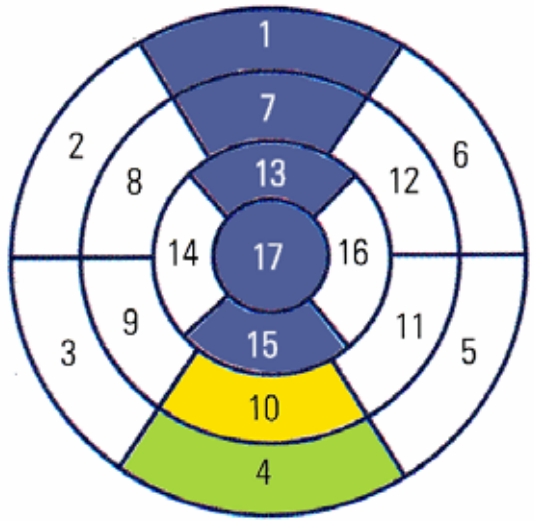
Apical four chambers

Longitudinal paraesternal axis



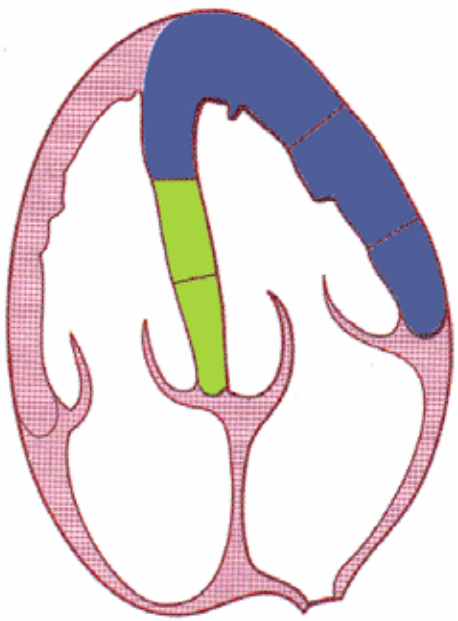
Apical two chambers

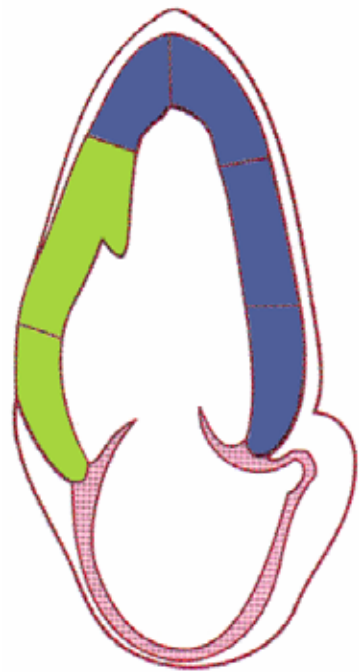
Bulls eye (short axis)



Septal Wall

Lateral Wall





Left Anterior Descending (LAD)

Left Circunflex (LCx)

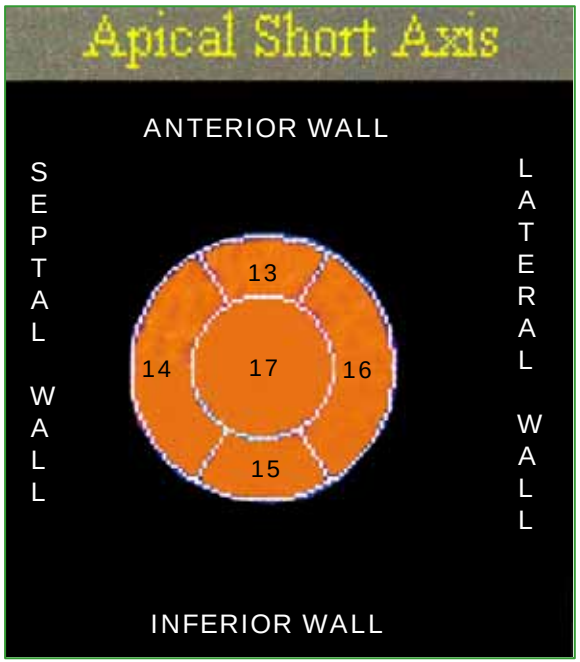
Posterior Descending (RCA or LCx)

CURRENT TERMINOLOGY OF CARDIAC WALLS FOR INFARCTION WITH Q WAVE BASED ON THE CORRELATION CONTRAST-ENHANCED CARDIOVASCULAR MAGNETIC RESONANCE (CE-CMR): A NEW PARADIGM

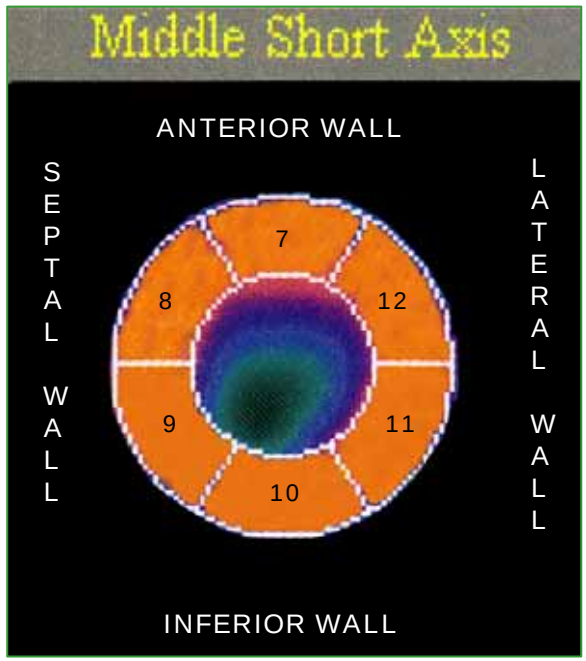
Bayés de Luna concept

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

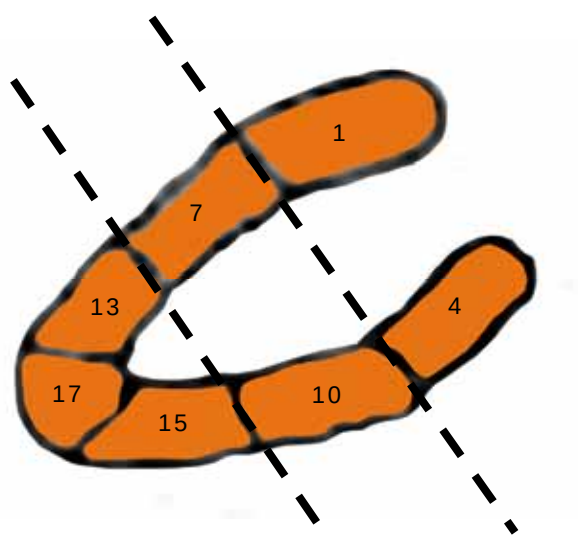
APICAL SHORT AXIS

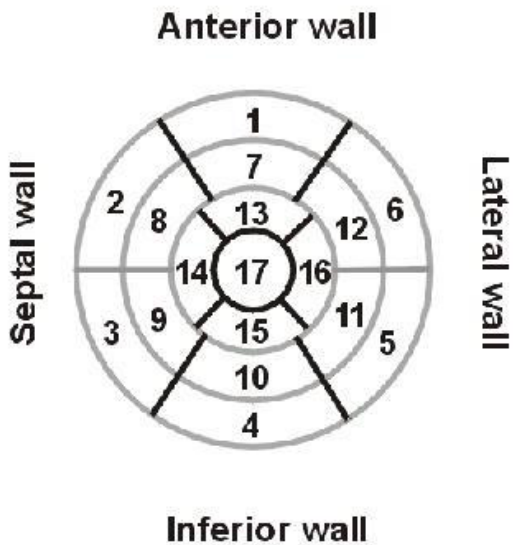


MIDDLE SHORT AXIS



BASAL SHORT AXIS





POLAR MAP SHORT AXIS IN “BULL’S-EYE”

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

1) ANTEROSEPTAL ZONE

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

2) INFEROLATERAL ZONE

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

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

1) ANTEROSEPTAL ZONE

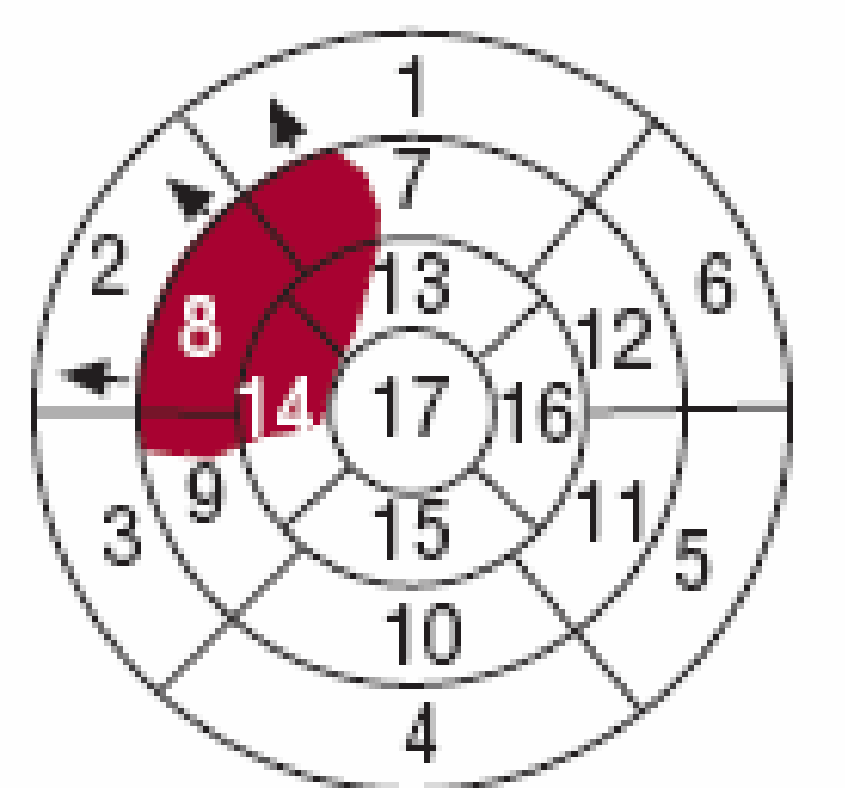
- **Septal myocardial infarction**
- **Type:** A-1.
- **Most likely site of occlusion**: perforating branch (S1) of LAD.
- **ECG pattern**: Q in V_1-V_2.
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **Sensitivity (SE):** 100%
- **Specificity (SP):** 97%.

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

SEPTAL INFARCTION
A-1

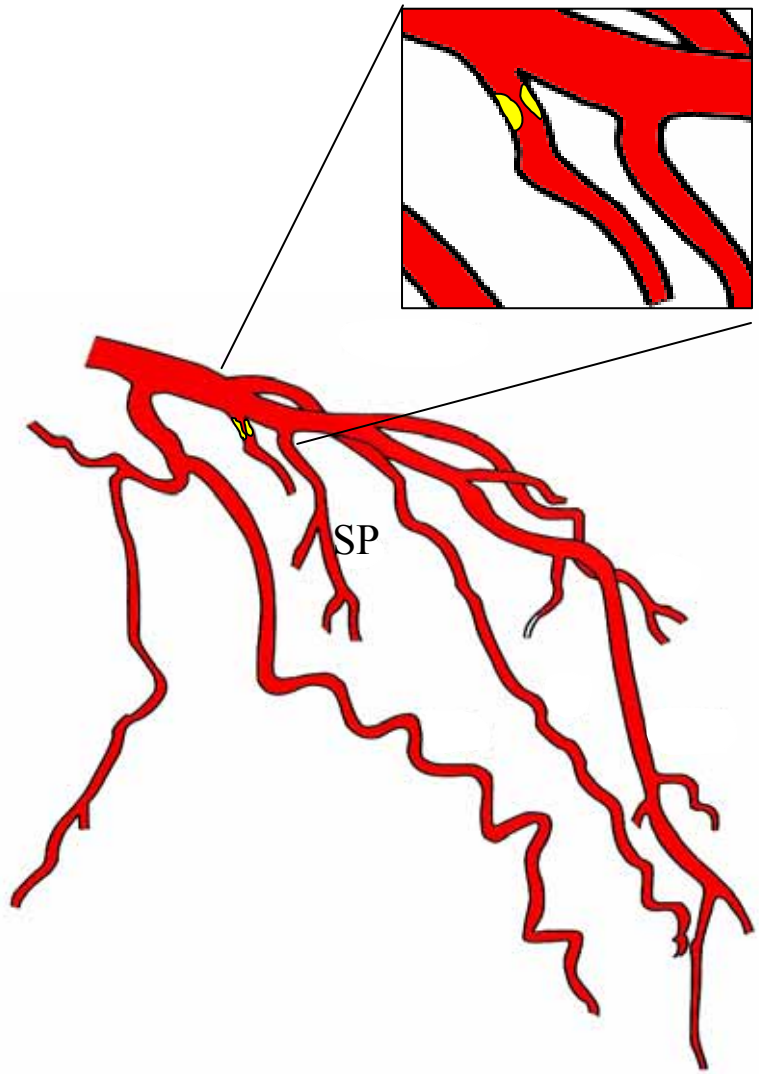
ANTERIOR WALL

SEPTAL WALL



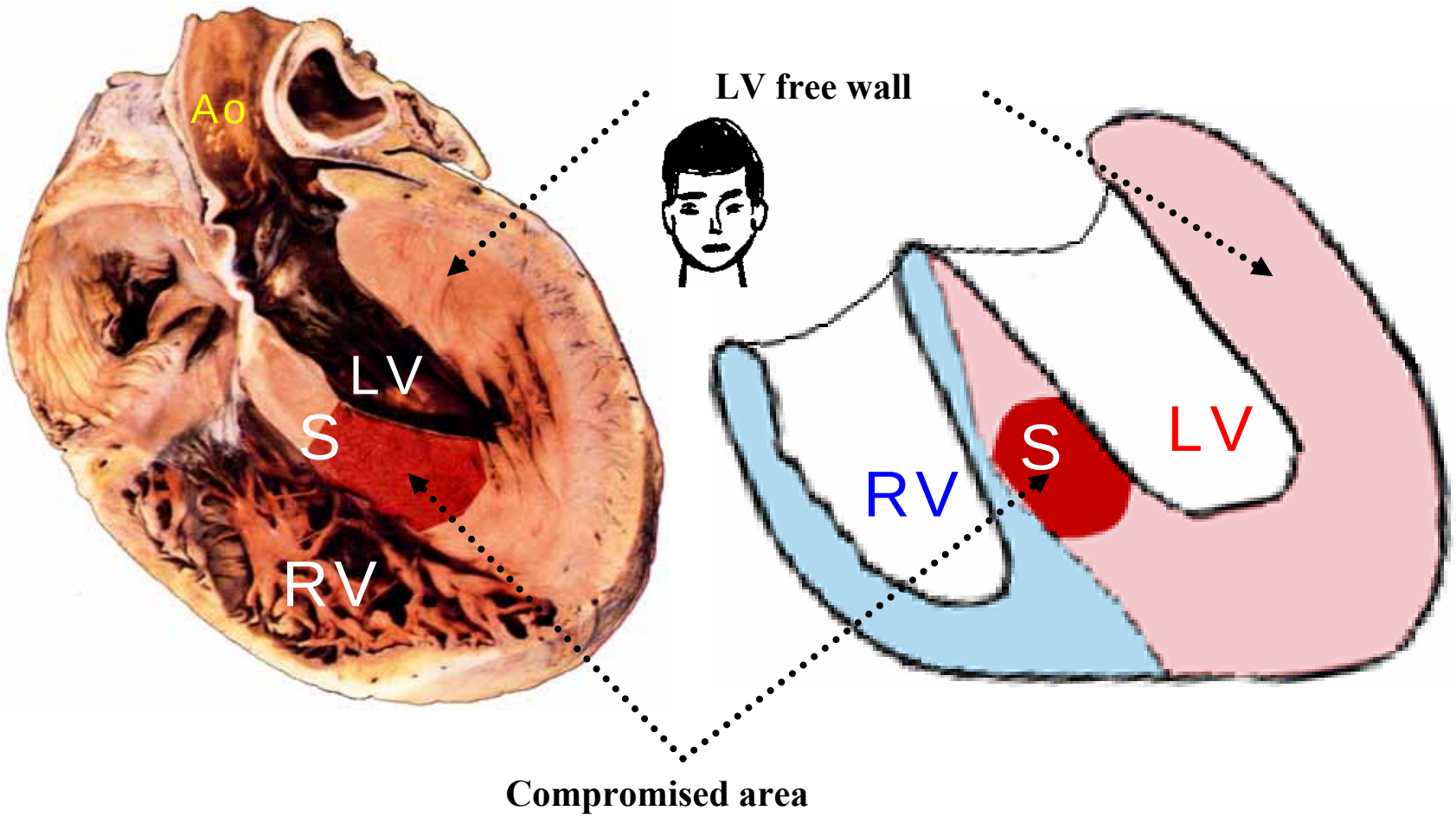
LATERAL WALL

INFERIOR WALL



ECG pattern: Q in V1-V2

FRONTAL VIEW OF SEPTAL OR
ANTEROSEPTAL INFARCTION



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

1) ANTEROSEPTAL ZONE

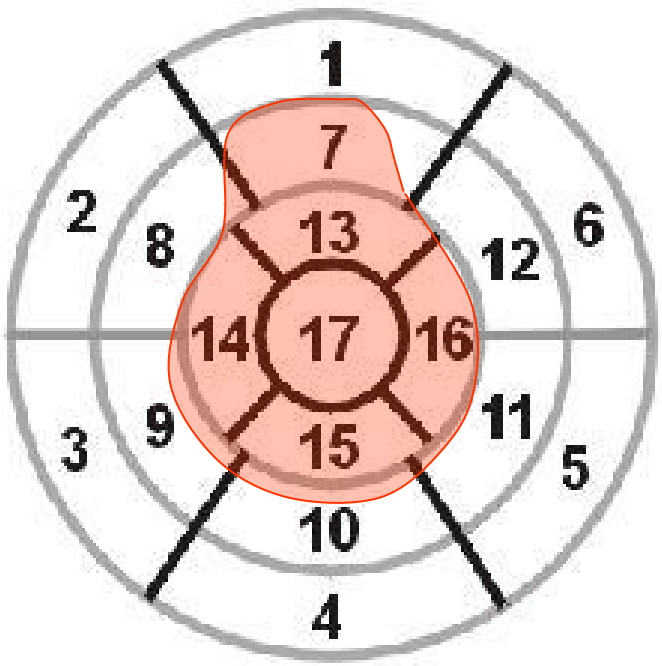
- **Apico-anterior myocardial infarction**
- **Type:** A-2
- **Most likely site of occlusion:** distal portion of LAD
- **ECG pattern:** Q in V_1 through V_6
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **SE:** 85%
- **SP:** 98%.

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

APICOANTERIOR INFARCTION
A-2

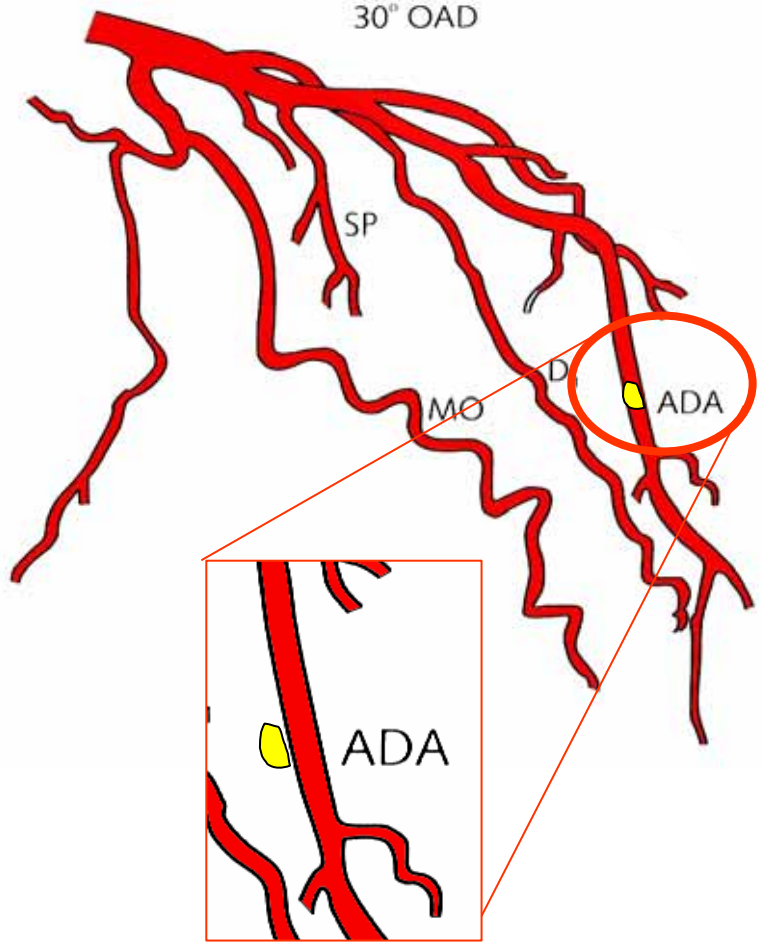
ANTERIOR WALL

SEPTAL WALL



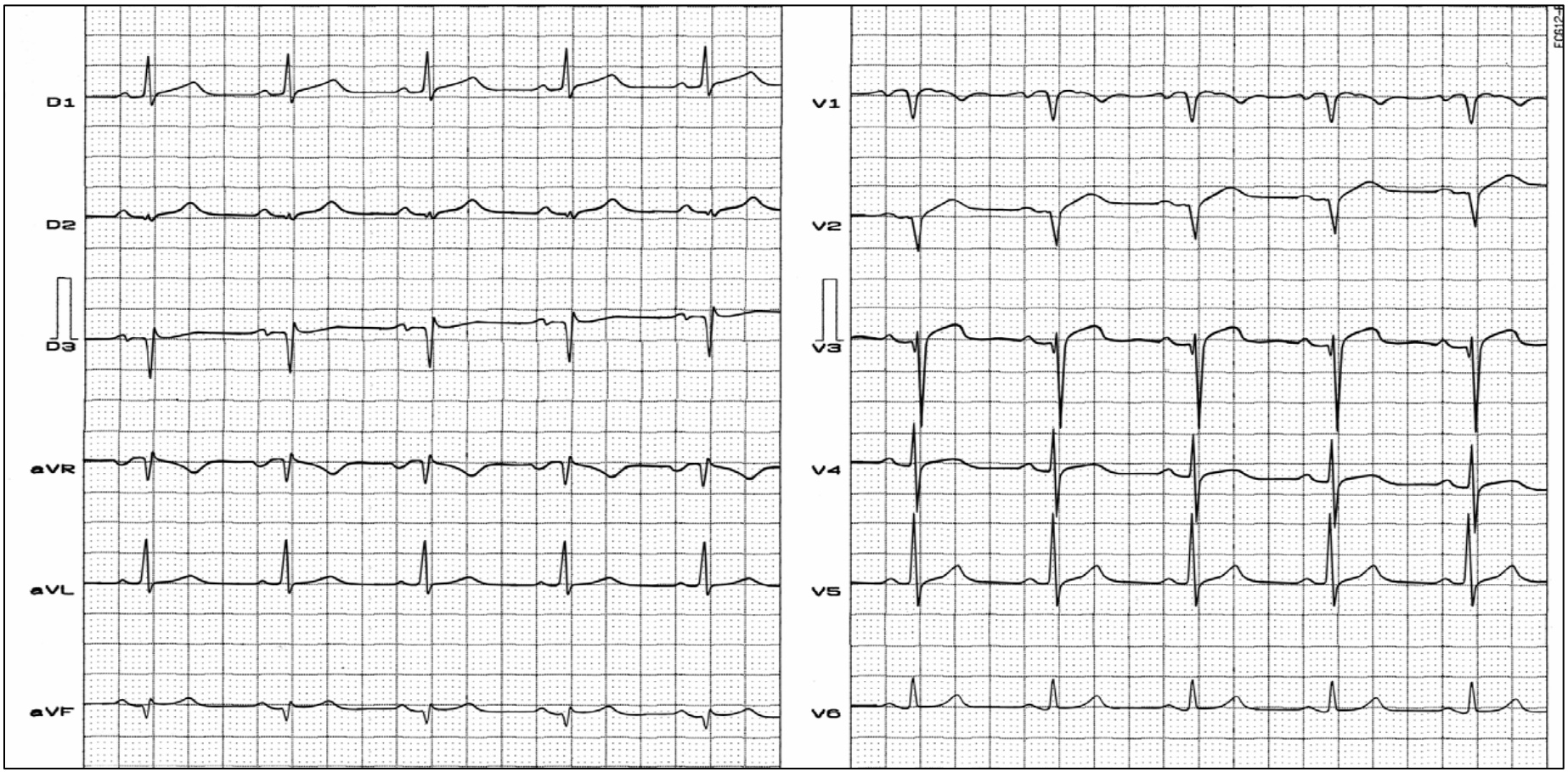
INFERIOR WALL

LATERAL WALL



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

NAME: TA; SEX: MALE; AGE: 65 y.o.; RACE: ASIAN.; WEIGHT: 65 Kg.;HEIGHT: 1.67 m
DATE: 03/17/2004.; MEDICATION IN USE: Enalapril 10 mg 2X + Atenolol 50mg + ASA 2X



Clinical diagnosis: Coronary insufficiency. AMI in 2002.
ECG diagnosis: QS in V1 and V2. In V3 qrS. Doubtful Q waves in III and VF.

CONCLUSION: Electrically inactive apical-anterior area. Doubtful electrically inactive inferior area.

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

1) ANTEROSEPTAL ZONE

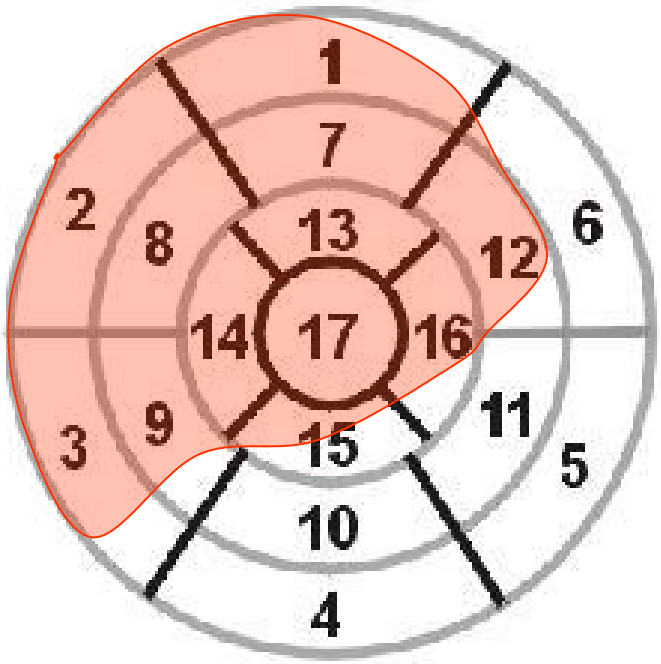
- **Extensive anterior myocardial infarction**
- **Type:** A-3
- **Most likely site of occlusion:** proximal LAD
- **ECG pattern:** Q from V_1 through V_6, I and VL
- **Segments compromised by infarction CE-CMR:** image in the next slide
- **SE:** 83%
- **SP:** 100%.

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

EXTENSIVE ANTERIOR INFARCTION
A-3

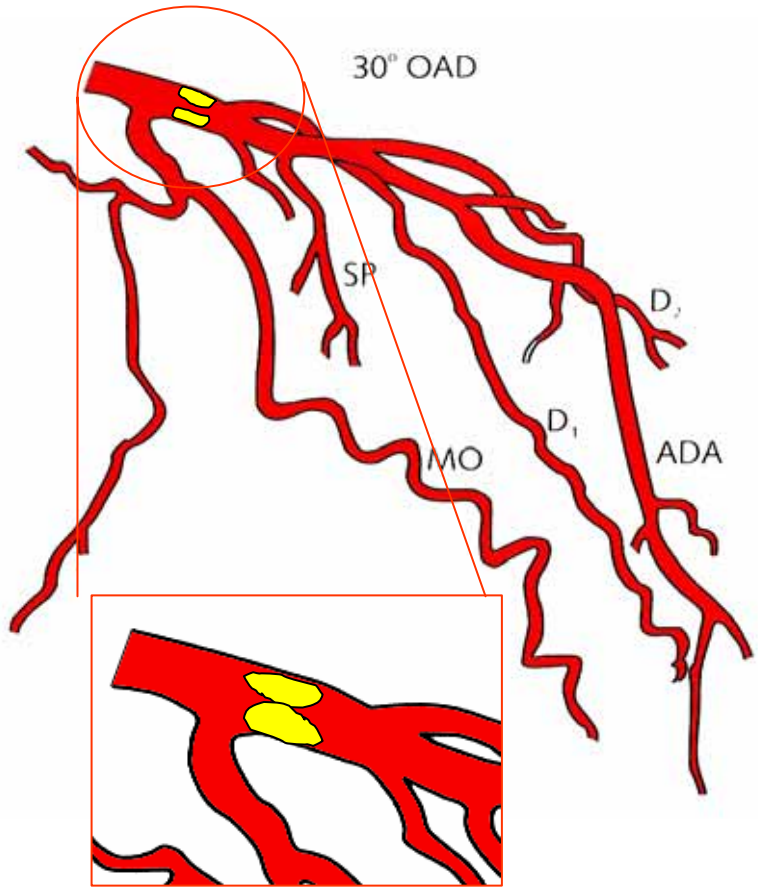
ANTERIOR WALL

SEPTAL WALL



LATERAL WALL

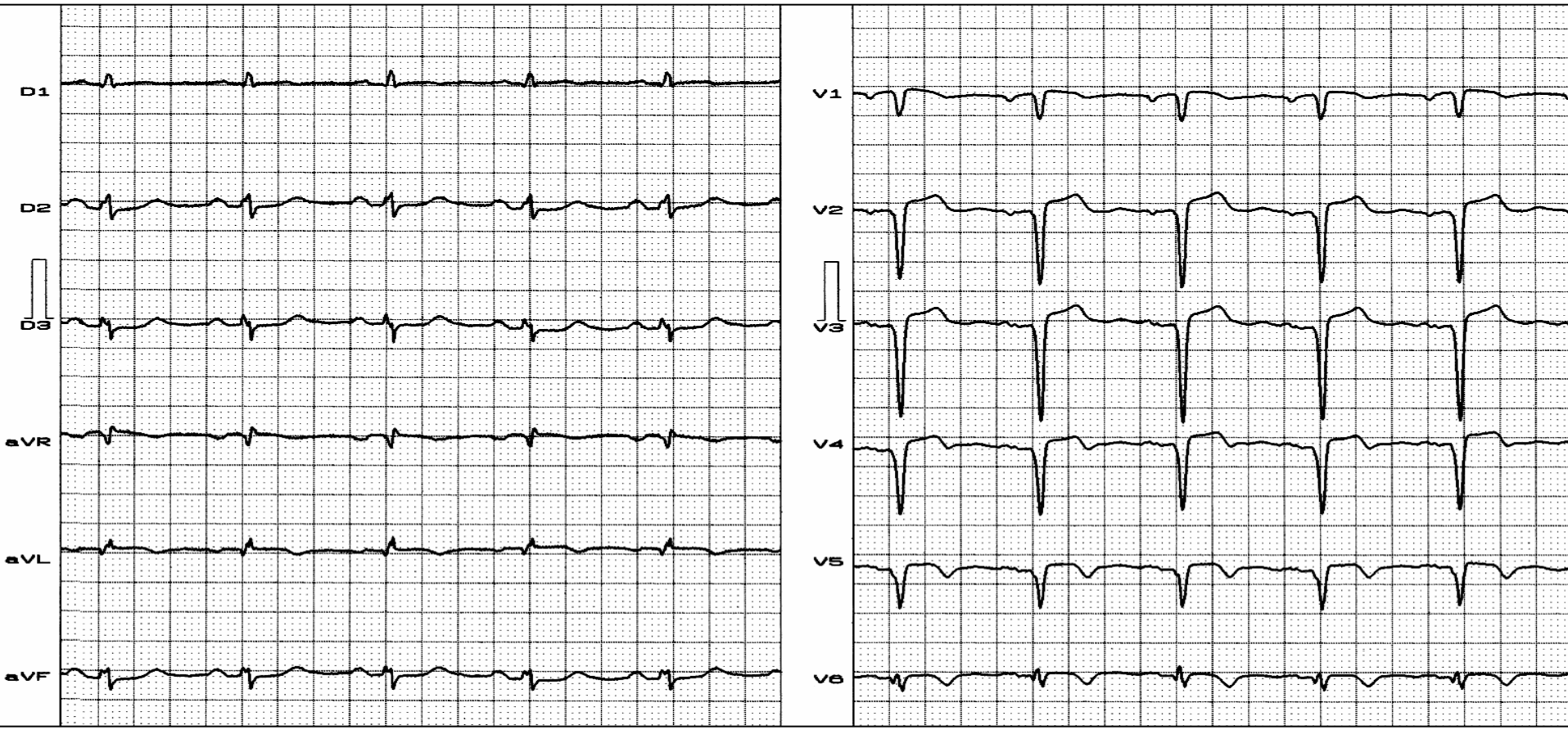
INFERIOR WALL



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

EXTENSIVE ANTERIOR INFARCTION V_1 THROUGH V_6 + I AND aVL.

Male, Asian, 57 y.o., clinical symptoms of infarctions three years ago.



QS from V_1 through V_5, qrs in V_6, qr in VL, and r in I. Electrically inactive transmural inactive area; extensive apical subepicardial (V_5 and V_6) and lateral (VL) ischemia;
ST segment elevation from V_2 through V_4 that suggests anterior aneurysm.

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

1) ANTEROSEPTAL ZONE

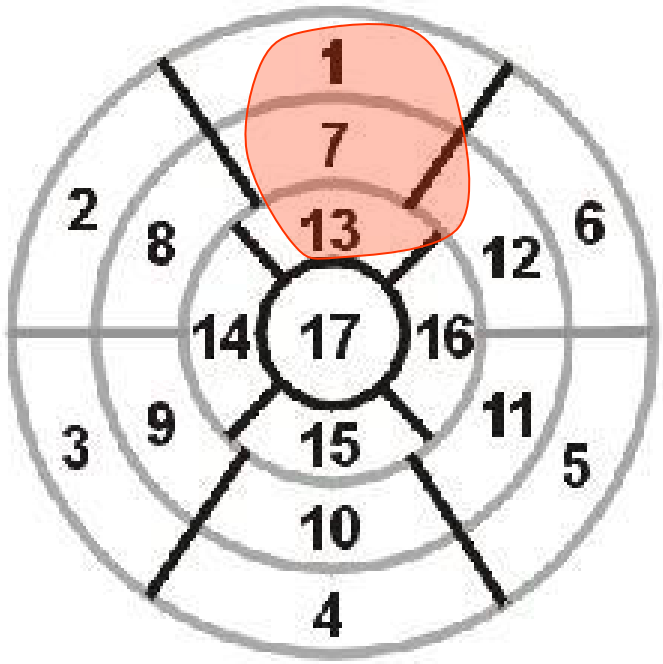
- **Mid-anterior myocardial infarction**
- **Type:** A-4
- **Most likely site of occlusion:** First Diagonal.
- **ECG pattern:**
- **In acute phase[1]:** ST segment elevation in VL and V2
- **In Chronic phase:** QS in VL, sometimes Q in I, without Q wave in V5-V6. Possibly q in V2-V3.
- **Segments compromised by infarction CE-CMR:** segments 1, 7 and 13. (next slide)
- **SE:** 67%
- **SP:** 100%.

1) **Birnbaum Y et al. Am Heart J. 1996;131:38-42**
2) **Bayés de Luna A, et al.Am J Cardiol. 2006;97:443-451.**
3) **Bayés de Luna A, et al. Circulation 2006; 114:1755-1760.**
4) **Bayés de Luna A, et al. J Electrocardiol. 2006; 39 (4 Suppl):S79-81.**
5) **Bayés de Luna A, et al. J Electrocardiol. 2007;40:69-71.**
6) **Bayés de Luna A,et al. Ann Noninvasive Electrocardiol. 2007; 12:1-4.**
7) **Bayés de Luna A, et al. Cardiology Journal 2007;14 : 417-419.**
8) **Cino JM, et al. J Cardiovasc Magn Reson. 2006;8:335-44.**
9) **Pons-Lladó G, et al. J Cardiovasc Magn Reson. 2006;8:325-6.**

MID-ANTERIOR INFARCTION
A-4

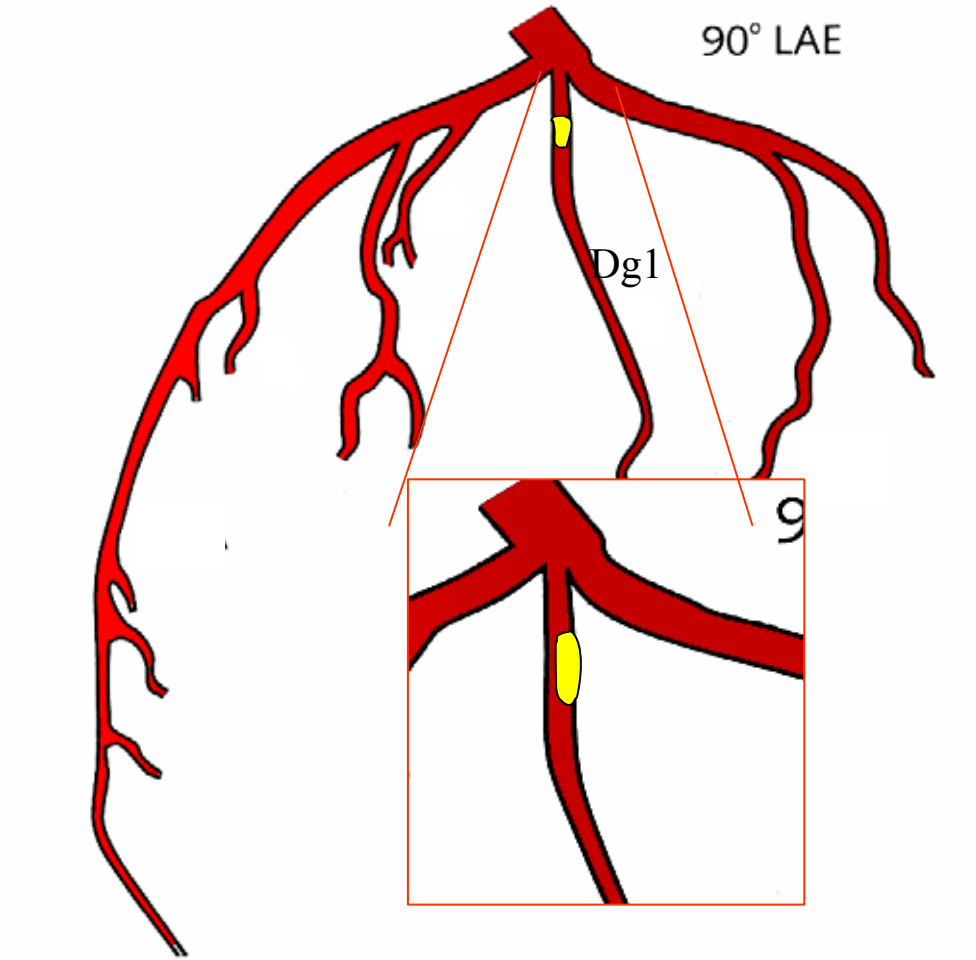
ANTERIOR WALL

SEPTAL WALL



INFERIOR WALL

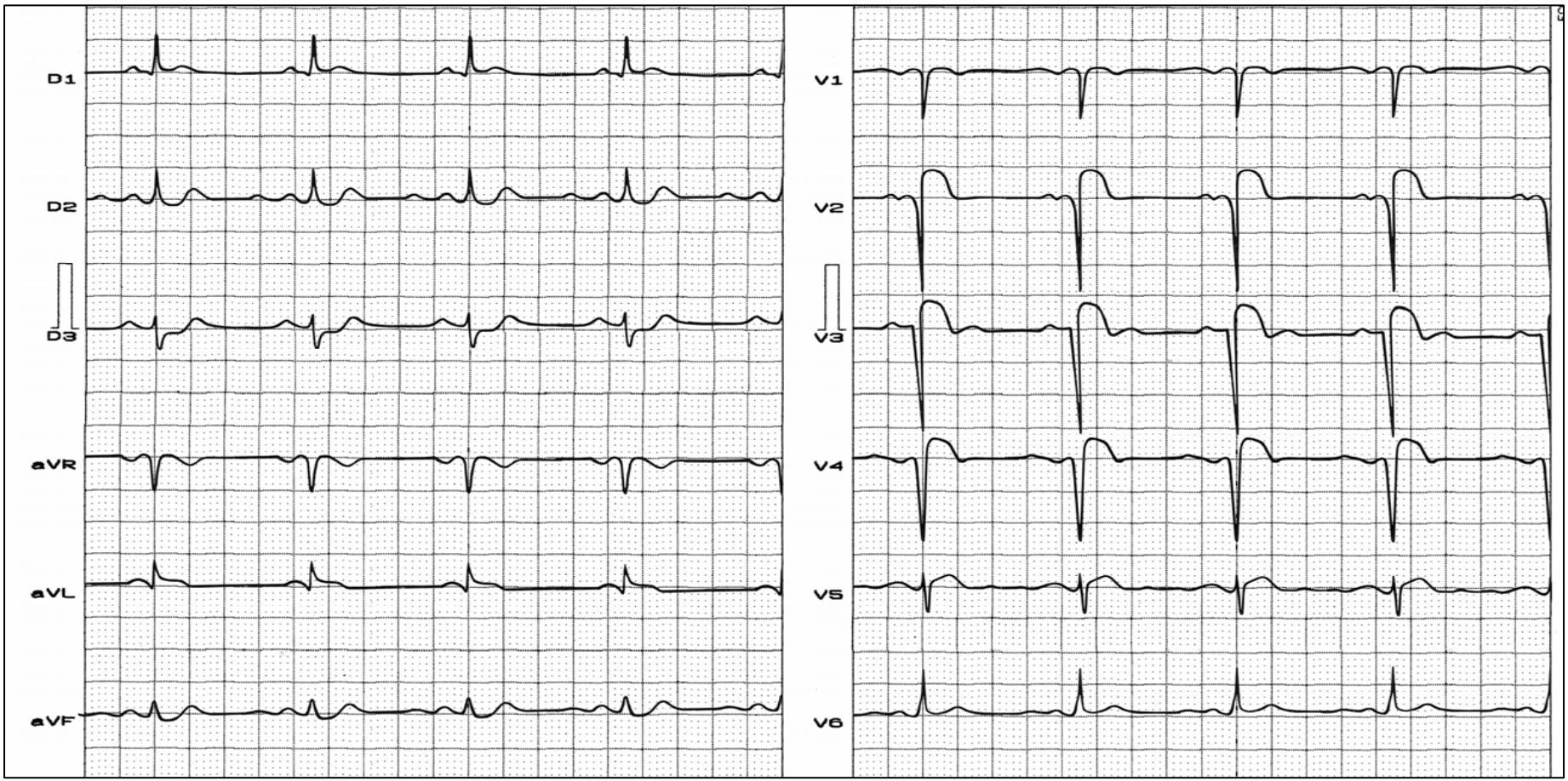
LATERAL WALL



ECG in acute phase
ST segment elevation
in VL and V2

ECG pattern in chronic phase:
QS in VL, sometimes Q in I, without Q wave in V5-V6. Possible q in V2-V3

Male, white, 52 y.o., obese, hypertensive, diabetic type 2, dyslipidemic type 2, smoker, and sedentary.
Infarction after 10 days of evolution.



QS from V_1 through V_4: Electrically inactive area in anterior wall and subepicardial lesion current in the same wall.

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

2) INFEROLATERAL ZONE

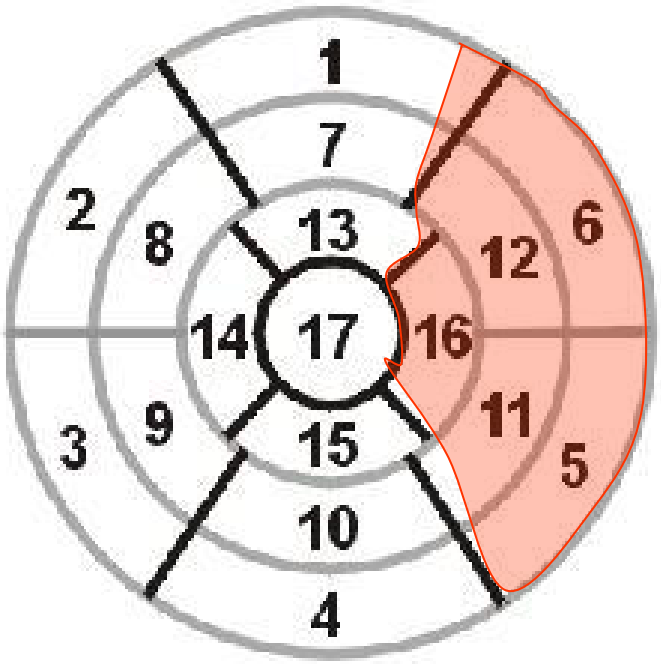
- **Lateral**
- **Type:** B-1
- **Most likely site of occlusion:** LCx artery or its oblique marginal branch (OM)
- **ECG pattern:** RS in V_1-V_2 and/or Q in I, VL, V_5-V_6. Voltage of R wave in V_6 of less amplitude
- **Segments compromised by infarction in CE-CMR:** image in the next slide.
- **SE:**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

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

LATERAL INFARCTION
B-1

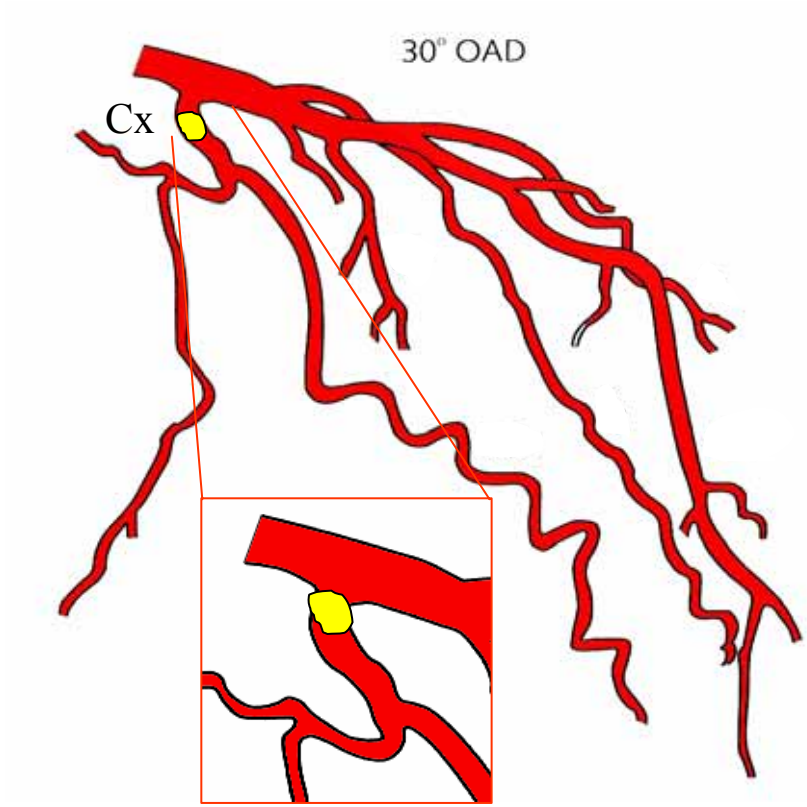
ANTERIOR WALL

SEPTAL WALL



LATERAL WALL

INFERIOR WALL



ECG pattern:
RS in V1-V2 and/or Q in I, VL, V5-V6.
Voltage of R in V6 of less amplitude

ECG Changes Mimics Myocardial Infarction

1) Vagotonia
2) Left ventricular enlargement
3) Afro-descendents
4) Early repolarization variant or pattern
5) Juvenile pattern
6) Asthenic habitus
7) Artifact caused by excessive inertia of the needle of the device
8) LBBB
9) Paced rhythm
10) Early repolarization pattern
11) Acute Pericarditis
12) Hyperkalemia
13) Myocarditis
14) Cardiac tumors
15) Ventricular aneurysm
16) Brugada Syndrome
17) Acute Pulmonary Embolism(1)
18) Tako-tsubo (stress) cardiomyopathy(2)
19) Acute dysfunction of mechanical aortic valve(3)

1. Ciliberti P, Rapezzi C, Villani C, Boriani G. Massive Pulmonary Embolism with Acute Coronary Syndrome-like Electrocardiogram Mimicking Acute Left Main Coronary Artery Obstruction.J Emerg Med. 2011 Dec 3. [Epub ahead of print]
2. Sharkey SW. Electrocardiogram mimics of acute ST-segment elevation myocardial infarction: insights from cardiac magnetic resonance imaging in patients with tako-tsubo (stress) cardiomyopathy. J Electrocardiol. 2008 Nov-Dec;41(6):621-5.
3. Shan P, Huang W, Li S, et al. Acute dysfunction of mechanical aortic valve as electrocardiographic mimic of acute left main coronary artery occlusion. Ann Thorac Surg. 2012 Apr;93:1307-1309.