ACUTE INFERIOR MYOCARDIAL INFARCTION AND ANYTHING ELSE?. WHAT? AND WHY?

INFARTO DO MIOCÁRDIO AGUDO INFERIOR E ALGO MAIS. O QUE? E PORQUE?

Raimundo Barbosa Barros MD. Nickname: "the Fox" Coronary Center Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza-Ceará-Brazil

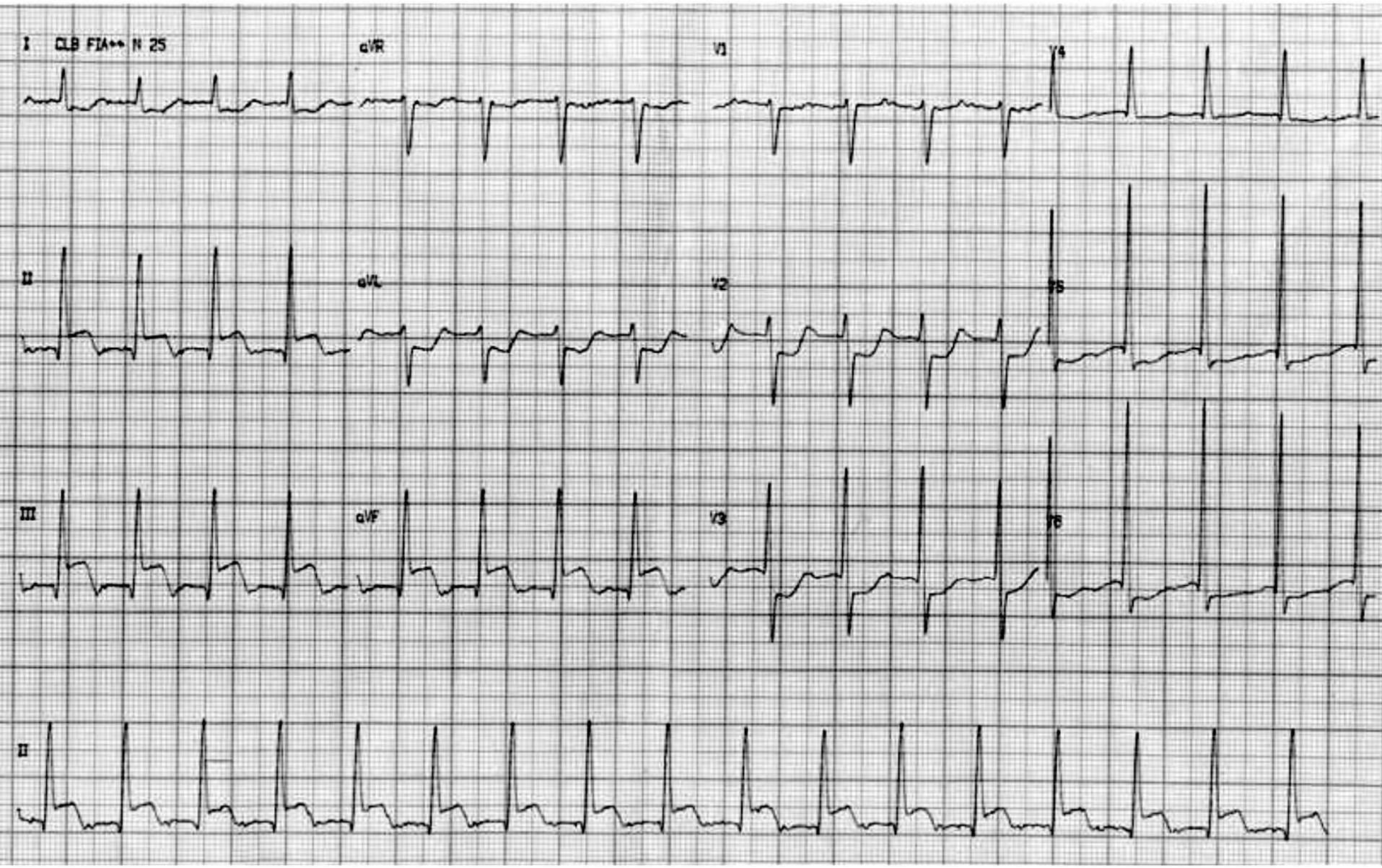
Commentaries Andrés Ricardo Pérez-Riera M.D.Ph.D. Chief of ECG/VCG sector Cardiology discipline ABC Faculty- ABC Foundation-Santo André- São Paulo-Brazil riera@uol.com.br

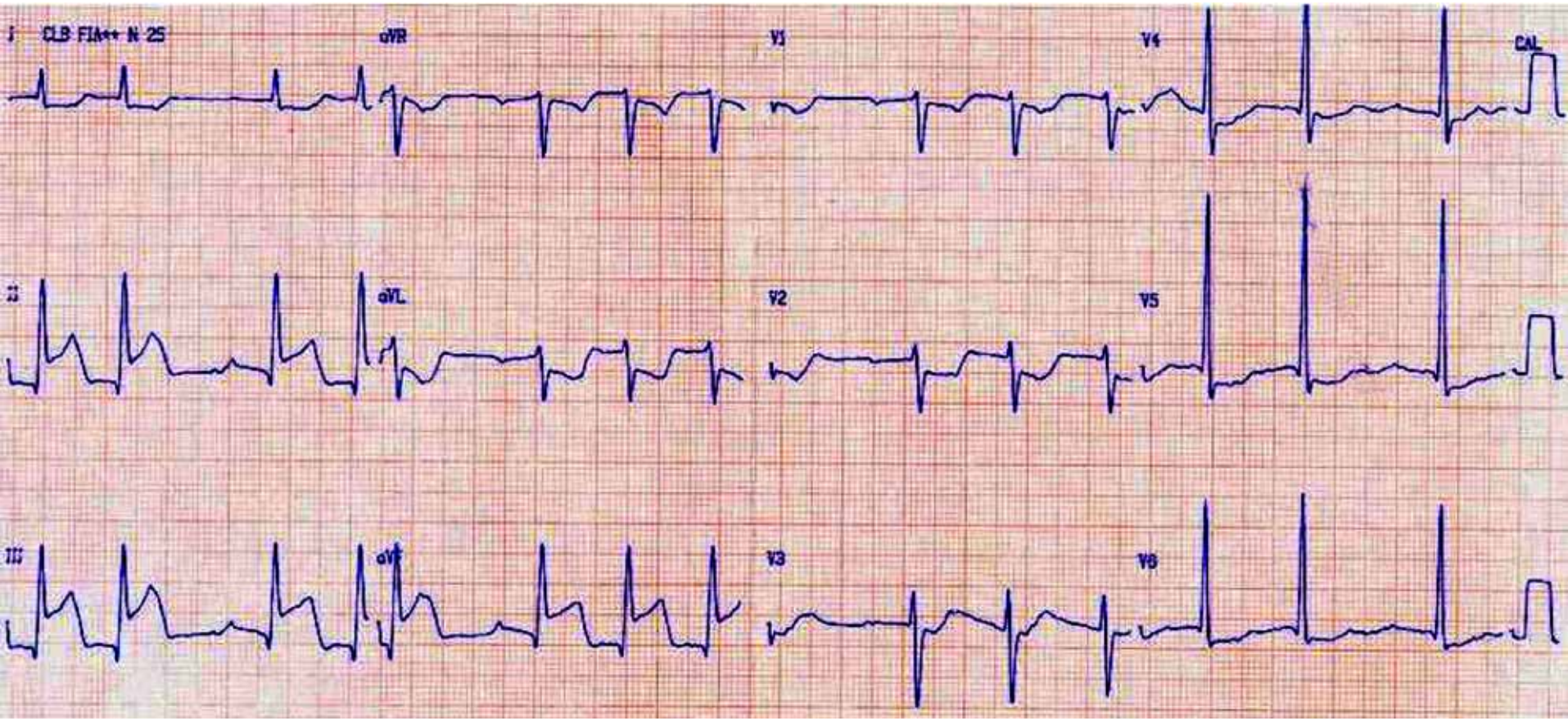
SESSÃO CORONARIANA/ CORONARY SESSION

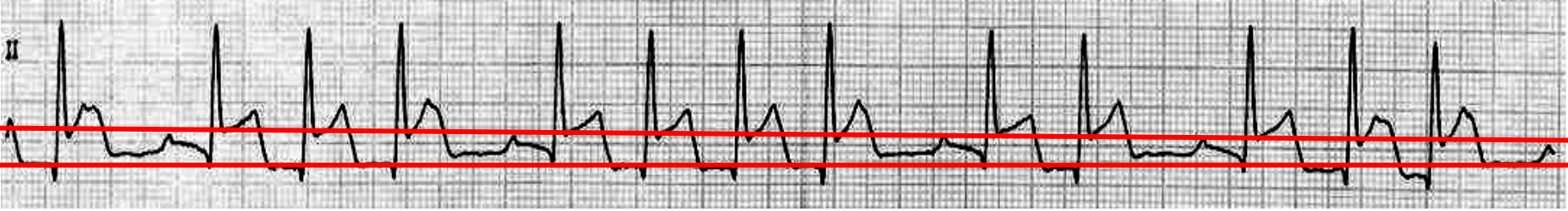
ID: L.M.F.B., 66 anos, natural e residente em Pacatuba-Ceará Brasil.
Queixa principal: “dor no peito e falta de ar”
HDA: paciente informa precordialgia de forte intensidade iniciada há 4horas associada a náuseas e vômitos. Relata dispnéia aos grandes e moderados esforços há 6 meses com piora após o início de dor em precordial.
HPP: hipertensa e tabagista de longa data. AVC em 2009 sem seqüelas. Nega diabetes.
Ex. físico (+ 01 dia) : orientada, Glasgow 15.
ACP: ritmo cardíaco regular, em 2 tempos, bulhas rítmicas normofonéticas, sem sopros. Pressão arterial: 169x78mmHg, Freqüência cardíaca: 104bpm.
MVF sem RAV. Freqüência respiratória 29 irpm.

ID: LMFB, 66 yo, born and resident in Pacatuba, Ceara Brazil.
Main complaint: “chest pain and shortness of breath”
HDA: patient reports intense chest pain started for 4hours before associated with nausea and vomiting. Additionally, dyspnea on moderate exertion and worsening 6 months after the onset of chest pain.
HPP: hypertension and smoking a long time ago. Stroke in 2009 without sequelae. Denies diabetes Mellitus.
Physical (01 days +)-oriented, Glasgow 15. ACP: regular heart rhythm in two times, rhythmic normal sounds without murmurs.
Blood pressure: 169x78mmHg,
Heart rate: 104bpm.
MVF without RAV Respiratory rate 29 incursions/pm.

August 15 2011 11:00h A.M.




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6



Atrial infarction is rarely diagnosed before death because of its characteristically subtle and nonspecific electrocardiographic findings. Atrial infarction is a neglected ECG sign with important clinical implications.

I) MAJOR CRITERIA

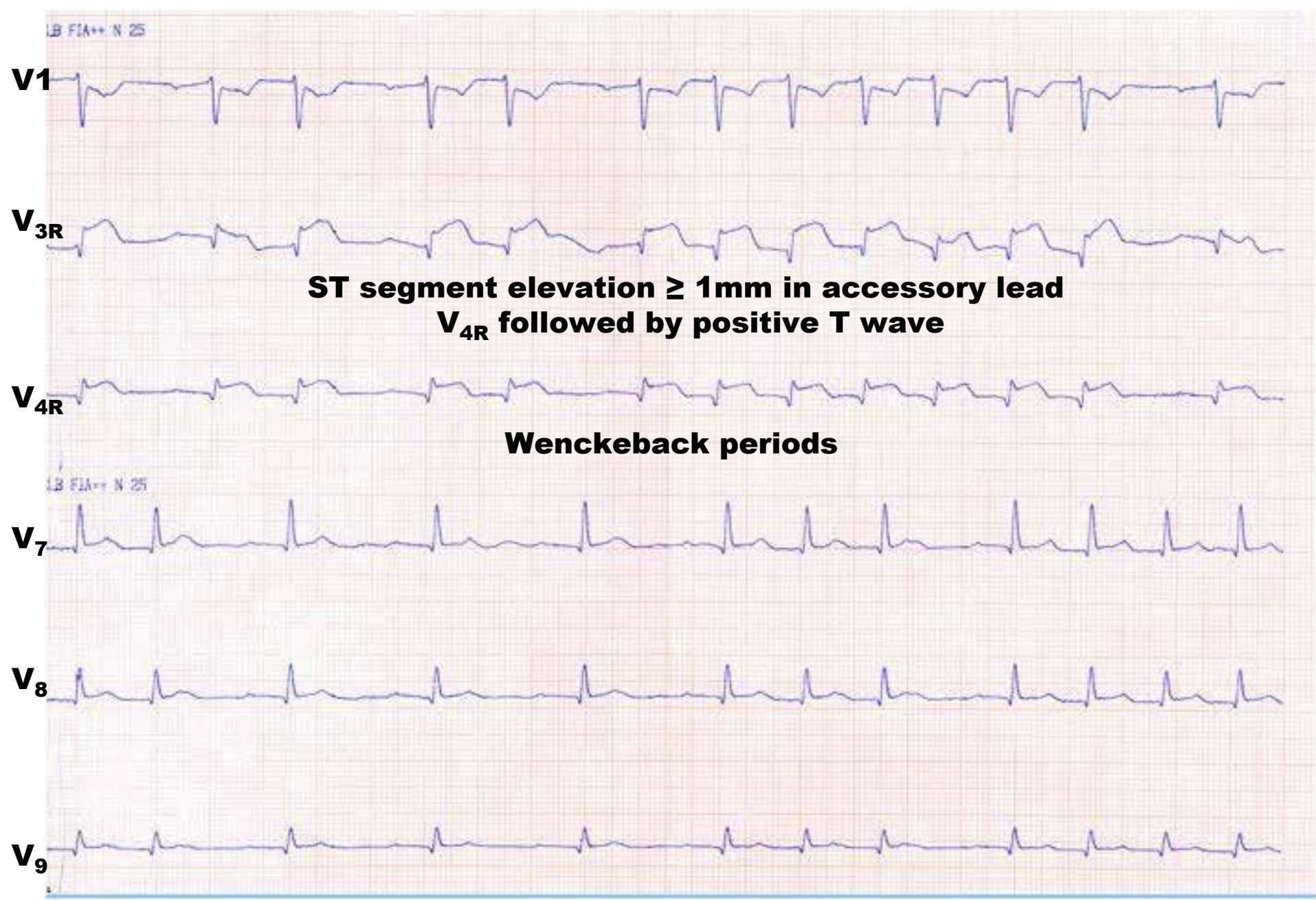
PRs elevation >0.5mm in leads V5 and V6 with reciprocal depression of PRs in V1 and V2 leads.

PRs elevation >0.5mm in leads I with reciprocal depressions in II and III.

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.


V1
V3R
ST segment elevation ≥ 1mm in accessory lead
V4R followed by positive T wave
V4R
Wenckeback periods
V7
V8
V9

De acuerdo a los postulados de Samuel, este ECG muestra un infarto inferior, en la etapa de lo que Samuel denomina etapa de preinfarto, con supra de ST mayor en III que en II, por oclusión de la coronaria derecha proximal. Ademas con el concepto de remodelamiento fisiológico, infra ST en I1 y aVL y máximo en V2, con ST in decrescendo hacia V6.
Ademas veo un trastorno de conducción con fenómeno de Wenckebach, frecuente de observar en estos infartos de miocardio agudos inferiores. Si contamos con hemodinamia, anglo y angioplastia, sino gro bonito os.

Saludos Pancho Femenia

Potro y Raimundo: Este no es mi fuerte y por eso quiero probar:

1. Infarto Infero-posterior, con signos indirectos de compromiso de ventriculo derecho (R III> II y depresion ST V2 > ST V1).
2. a.Bloqueo AV 2 grado Mobitz I con diferentes grados de conducción (3:2; 4:3) vs b.ESV bloqueadas, pero parece que el PR se prolonga, en cuyo caso opción a.

Isquemia sobre el nodo AV?

Salud (no me maten!)

AB

Raimundo: IAM hiperagudo inferior por compromiso de la CD. Porque esto? onda R alta monofasica con supra ST y ya no tiene la onda T picuda, probablemente hayan transcurrido entre 2 y menos de 6 horas del inicio del los sintomas.

Porque esto? EL supra ST va decreciendo en amplitud y la onda T comienza a perder la caracteristica de monofasica de las primeras horas y el final de la onda T comienza a negativizarse, ademas comienza a presentar un esbozo de onda q en DII, DIII y aVF. El supra ST DIII/DII mayor de 1 mm validada por Andersen (S (en el infarto inferoposterior S 88%, E 91% en IAM inferior con compromiso del VD), el infradesnivel en DI y aVL > 1 mm (S 82%, E 90%) es otro signo de IAM inferior con extension al VD. El infradesnivel en V3 mayor V2 y supra V1 lesion proximal de CD (86% de los casos). En estos infartos la magnitud el compromiso del VD puede inferirse por el vector medio del segmento ST entre 90 y 120 IAM del VD peque□extension, entre 120 y 180 IAm del VD de gran magnitud. En este caso el vector se encuentra en 150. Los trastornos del ritmo y la descompensación hemodinamica son frecuentes, en este ECG BAV de primer grado en el primer trazado del ECG, en el segundo BAV de diferentes grados con fenómeno de Mobitz, 2:1, #:1 lo que presagia el BAV completo.

Saludos a los queridos amigos Pancho y Adrian.

Martin Ibarrola

Hola TinchoMuy buen analisis, pero disiento en una cosa.
Los trastorno de conducción AV durante infartos inferiores (o inferoposteriores etc) No presagian bloqueo AV.
El reflejo de Bezold-Jarisch se produce por distensión de la CD durante la obstrucción epicardica, y son, si bien lo recuerdo, mecano-receptores sensibles al “stretch” intraluminal. Esto liberan Adenosina, que como sabes es termosensible y permanece solo transitoriamente en la sangre, la cual es responsable de la mayoria de los defectos de conducción AV durante los infartos de CD.
Por eso, cuando Ud era joven, se postulaba dar aminofilina durante el IAM inferior con trastorno de conducción AV. Luego, al reveerse el potencial componente inotropico positivo de dicha medicación (aumenta el consumo de 02) se la dejo de utilizar.
Por lo tanto, los trastorno de conducción AV durante el infarto inferior, NO solo NO presagian bloqueo AV, sino que tampoco presagian la necesidad de MP definitivo.

AB

Gracias Adrian por desasnarme, no estoy plenamente de acuerdo que en este caso en particular sea por reflejo y liberacion de adenosina. Obvio no lo puedo descartar.
Los trastornos agudos de la conducción A-V ocurren en 10-20% de los pacientes con IAM.
IAM inferior se reconocen 3 etapas en relación con el bloqueo A-V:
1) Hiperaguda, en la que el trastorno de conducción es resistente a la atropina y aparentemente sería causado por la isquemia.
2) Aguda, que dura entre 2 y 6 horas y en la cual el mecanismo parecería ser vagal, ya que el trastorno de conducción responde a la atropina.
3) Tardía (desde las 6 horas hasta los 2-3 días). En esta etapa el trastorno de conducción no responde a la atropina y se debería a edema en el nódulo A-V. **(1)**
Muy didactica la explicacion y ademas el Maestro Perez Riera aporto a esto.
En el ECG presentado presenta progresion del BAV de ingreso a BAV de 2do grado, con taquicardia sinusal, mi impresion es por fenomeno isquemico no por reflejo vagal que responda a la atropina.
Esto es simple de averiguar. Dr Raimundo respondio a la administracion de atropina el bloqueo?
Un saludo y obvio nos veremos en noviembre y gracias por la explicacion clara y precisa.
Martin Ibarrola

(1) **Dr. Jorge González Zuelgaray Dr. Edgardo Schapachnik Trastornos de Conducción. Bloqueo A - V completo. Primer Curso de Arritmias por Internet**

Ambos bradiarritmias e distúrbios de condução pode ser vistos em infartos do miocárdio e geralmente estão relacionados à isquemia ou distúrbio autonômico. As características clínicas e manuseio de bradiarritmias e bloqueio de condução depende da localização do infarto. A artéria CD fornece irrigação ao Nó SA em 60 % dos casos e a LCx ao restante 40%. Em mais de 90% das pessoas, a RCA irriga o Nó AV e na sua porção proximal. A porção terminal(NH), feixe de His e os ramos esquerdo e direito são irrigados pelas perfurantes septais da LAD. Bradicardia sinusal, PR prolongado e bloque io tipo Wenkebach e BAV completo são comuns no infarto do miocárdio inferior. Bloqueio AV total ocorre em aproximadamente 10% dos pacientes com IMI. Isso raramente ocorre de repente, na maioria das vezes há prolongamento gradual do intervalo PR progredindo para bloqueio AV completo. O bloqueio AV é do dentro do Nó AV em mai s de 90% dos casos e geralmente resulta em um bloqueio transitório. O complexo de escape é geralmente estreito e raramente exige estimulação. As bradiarritmias que ocorrem nos infartos inferiores são geralmente sensíveis à atropina. Andrés.

Both bradyarrhythmias and conduction disturbances can be seen with myocardial infarctions and are generally related to ischemia or autonomic disturbance. The clinical features and management of bradyarrhythmias and conduction block depends on the location of the infarction. The right coronary artery supplies the SA node in 60 percent of people and the left circumflex the remaining. In over 90 percent of people, the RCA feeds the AV node and proximal His. The terminal portion of the His and main left bundle and right bundle branch are supplied by septal perforators of the LAD. Sinus bradycardia, prolonged PR conduction with Wenkebach and complete heart block are common in inferior myocardial infarctions (IMI). Complete AV block occurs in approximately 10 percent of patients with IMI. This rarely occurs suddenly, most often seen with prolonged PR conduction gradually progressing to complete AV block. AV block occurs within the node in over 90 percent of cases and typically results in a transient block. The escape complex is usually narrow and infrequently requires pacing. Bradyarrhythmias occurring in the setting of inferior infarctions are generally responsive to atropine.

The clinical history is consistent with ischemic heart disease. The age and co-morbidities suggest that this is atherosclerotic in nature rather than coronary vasospasm. With the first ECG, I would diagnose an acute inferior wall MI with hyperacute ST segment elevation in D II, III and aVF. There is impressive ST segment depression in the anterior precordial leads which probably reflects true posterior wall involvement rather than just reciprocal changes. In addition and totally consistent with an acute inferior wall MI is Wenckebach second degree AV block. The underlying sinus rate is fast and the heart rate itself is sufficiently rapid such that I would not place a temporary pacemaker at this time. While atropine could probably reverse the AV block, with the underlying mild sinus tachycardia it might well allow for 1:1 AV conduction with a sinus tach and possible increase the ischemia. On the first PR complex of each Wenckebach cycle, there is also a first degree AV block. The level of AV block is unlikely to cause a clinical problem and will probably totally resolve making the need for a permanent pacemaker highly unlikely. Clearly, this situation requires very close monitoring should the AV block progress to higher grades of 2nd degree (2:1, 3:1) or even complete heart block in which case, he might require a temporary pacing system but I have seen patients with profound complete heart block when due to an acute inferior wall MI have their AV block totally resolve and not require a permanent pacemaker. The clinical history of this severe chest pain starting 4 hours previously makes it unlikely that an acute intervention such as angioplasty and placement of a stent is unlikely to reverse the damage that has already been done. Since he only presented after 4 hours of chest pain and even in the most efficient of hospitals, it may be another 30-60 minutes before he would reach the cath lab and the catheters inserted, I would have to defer to my interventional colleagues as to whether they would want to intervene. Patients with an acute inferior wall MI usually do well although the posterior wall involvement increases the overall size of the infarct and is of concern. I would also obtain another ECG with right precordial leads looking for involvement of the RV. There was no comments on the patient's hemodynamics at the time but if compromised, after looking for evidence for involvement of the right ventricle, this might force my hand to recommend cardiac cath and intervention if an near-total or total occlusion could be identified.

Paul A. Levine MD, FHRS, FACC, CCDS25876 The Old Road #14. Stevenson Ranch, CA 91381Cell: 661 565-5589. Fax: 661 253-2144 Email: paul91321@gmail.com

La história clínica es consistente con enferemdad isquémica del corazon. La edad y las comorbidades sugieren que su naturaleza es arterioesclerótica mas que vasoespástica.
Con el primer ECG yo diagnosticaria IM agudo inferior con elevación hiperaguda del segmento ST en las inferiores. La depresión del ST en las precordiales anteriores probablemente indica extensión dorsal y no cambios recíprocos. Ademas el trazado es totalmente sugestivo de bloqueo de segundo grado tipo Wenckebach. La frecuencia sinusal es rápida lo suficiente como para que no sea necesario el implante de un marcapaso temporário en este momento. La atropina poderia probablemente revertir este bloqueo AV, con moderada taquicardia sinusal y podria mejorar la conducción 1:1 y probalbemente aumentar la isquemia. En el primer complejo el PR revela también bloqueo AV de primer grado. El nivel del bloqueo no debe causar problemas clínicos y se resolverá probablemente en forma total tornando el implante de MP altamente improblable. Claro que esta situación requiere un monitoreo muy próximo pues podria progresar para bloqueos de mas alto grado tipo 2:1 o3:1 o hasta un bloqueo total. En este último caso requerirá un MP temporário. Yo he tenido pacientes con bloqueo AV total secundarios e infarto inferior que resolvieron totalmente sin requerir MP permanente. Como el incio del dolor es de 4 horas de este severo dolor precordial la angioplastia con implante de sten no ocasionará reversión total de los daños ya ocurridos. Desde que el tiene apenas 4 h de dolor aun en los mas eficientes hospitales pasaran mas 30 o 60 minutos antes de estar en la mesa de cateterismo tendré que dejar que mis colegas intervencionistas actuen. Pacientes con infarto agudo inferior con extensión dorsal tiene el tamaño del infarto aumentado.
Me gustaria realizar otro ECG mapeando las precordiales derechas procurando compromiso del VD. No tenemos comentarios del estado hemodinámico del paciente. Si comprometido el VD reforzaria la recomendación del estudio hemodinamico y la intervención si una obstruccion total o casi total podria ser identificada.
Paul Levine

Estoy de acuerdo con ambos si en el contexto de un IM inferior el trastorno de conducción si compromete hemodinamicamente al paciente hay que actuar, desde la atropina, MP transitorio y poco frecuentemente inotropicos.
Martín vos estas planteando dos situaciones juntas que es un IMA inferior con BAV que generalmente obedece a descarga refleja asociado a compromiso del VD, donde para mantener la estabilidad hemodinámica hay que aportar volumen. La repercusión se impone, y lo ideal es contar con hemodinamia ahora si no tenes hay que trombolizar y seguramente no será fácil ya que se sumara a la inestabilidad perse del cuadro planteado, la vasodilatación producida por la estreptokinasa, creo el trombolitico mas utilizado en Argentina por los costos, es asi?
Otra situación que vos planteas es la del shock, y la única forma ademas del soporte clínico, poner el balon, y para los creyentes rezar, ES ABRIR LA ARTERIA,
Pancho

Querido Adrian concuerdo en parte en lo expresado por vos, en este paciente lo reperfundiria no le daria atropina. En eso concuerdo. Tampoco recuerdo haber colocado un marcapasos en un IAM inferior, pero lo teniamos ahi en este tipo de infartos. Si recuerdo le colocabamos un Swan-Ganz para asegurarnos una adecuada precarga, y obviamente no evolucionaban al BAVC por la terapia de reperfusion precoz! Comparto en este paciente que la infusion de atropina aumentaria el consumo de O2, pero si este paciente se encontrara hemodinamicamente inestable con BAV progresivo tiene indicacion de administrarle atropina, al igual que agentes inotropicos, ademas si el centro cuenta con balon de contrapulsacion aortico mejor aun. No olvides que en los pacientes con IAM inferior con compromiso hemodinamico y bradiacardicos, es util la administracion de la misma. Entiendo el concepto de incrementar el consumo de O2, pero lo esencial es compensar al paciente sino dificilmente llege a la sala de hemodinamia, o pensas que el darle inotropicos al IAM en shock esta contraindicado? Como le vas a administrar fibrinoliticos si se encuentra en shock o hemodinamicante inestable. No siempre se cuenta con un balon de contrapulsacion y angioplastia primaria.

La mayoria de los Bloqueos AV de 2 grado tipo Wenckebach, en el contexto de los IAM Inferiores SON TRANSITORIOS.
Que piensa Ud Andrés del reflejo de Bezold Jarisch????
Samuel?
AB
Reflejo de Bezold-Jarisch
El infarto agudo de miocardio inferior con frecuencia induce bradicardia sinusal transitoria a través del aumento de tono vagal, via reflejo de Bezold-Jarisch, que se explica por la distribución preferencial de nervio vago en la pared inferior del corazón.
La deflagración ("trigering") de este reflejo de Bezold Jarisch puede ser observado no apenas en la obstrucción coronária sino también durante el procedimiento de la reperfusión(primary percutaneous coronary intervention (PPCI). Al inyectar el contraste el hemodinamista solicita al paciente que tosa para mantener la conciencia durante el reflejo de BezoldJarisch. Además, es conocido el uso de la tos en la reanimación cardiopulmonar t (tos-RCP) Tos-CPR ha sido reportado como un mecanismo de contemporizar durante la arritmia ventricular previa a la cardioversión eléctrica(2).

1. Kawasaki T, Akakabe Y, Yamano M, et al. Vagal enhancement as evidence of residual ischemia after inferior myocardial infarction. Pacing Clin Electrophysiol. 2009 Jan;32:52-58.
2. Keeble W, Tymchak WJ. Triggering of the Bezold Jarisch Reflex by reperfusion during primary PCI with maintenance of consciousness by cough CPR: a case report and review of pathophysiology. J Invasive Cardiol. 2008 Aug;20:E239-242.

Acute inferior myocardial infarction often induces transient sinus bradycardia through vagal enhancement, known as Bezold-Jarisch reflex, which is explained by preferential distribution of vagal nerve in the inferior wall. Vagal enhancement was associated with residual ischemia in old inferior myocardial infarction as well as inferior angina pectoris. Measurement of coefficient of component variance of high frequency is useful in improving the diagnostic reliability of exercise electrocardiography in patients with old inferior myocardial infarction.(1)

Triggering of the Bezold Jarisch Reflex not by occlusion but reperfusion is possible. In addition, the use of cough cardiopulmonary resuscitation (cough-CPR) to maintain consciousness during the Bezold Jarisch Reflex. Cough-CPR has previously been reported as a temporizing mechanism during ventricular arrhythmia prior to electrical cardioversion.(2)

1. Kawasaki T, Akakabe Y, Yamano M, et al. Vagal enhancement as evidence of residual ischemia after inferior myocardial infarction. Pacing Clin Electrophysiol. 2009 Jan;32(1):52-8.
2. Keeble W, Tymchak WJ. Triggering of the Bezold Jarisch Reflex by reperfusion during primary PCI with maintenance of consciousness by cough CPR: a case report and review of pathophysiology. J Invasive Cardiol. 2008 Aug;20(8):E239-42.

Pensas que aumenta el consumo de O2 de un area que carece de irrigacion? o intentas mejor irrigacion del resto de las coronarias y territorios no afectados en un paciente descompensado? Y ahi si compensandolo voy a poder tirarle los fibrinoliticos.
Lindo tema este, aprecio tus comentarios y comparto algunos de tus conceptos, pero una cosa muy diferentes de los IAM, es el IAM con compromiso severo del VD.
Un abrazo grande
Martin Ibarrola

Finals commentaries

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

ECGs diagnosis

1. **Sinus tachycardia**
2. **Atrial infarction:** See at the end of presentation Slides form 36 to 44;
3. **First degree AV block**
4. **Transient second degree AV block Wenckebacdh type or Mobitz type I**
5. **Left Ventricular enlargement or left ventricular hypertrophy(LVH)** S V_2 + R V_6 = 41mm >35mm positive voltage Sokolow-Lyon criteria.
6. **Hyperacute phase (few hours) of inferolateral MI:** Increase in intrinsicoid deflection time is observed in QRS complexes, significant ST segment elevation of superior concavity, followed by positive symmetrical T wave with increased voltage.ST segment elevation in inferior leads SIII>SII(RCA)
7. **ST segment depression on precordial leads:** indicative of multivessel coronary disease significantly and adversely impacts on patient prognosis.
8. **Right ventricular MI:** additional ST segment elevation ≥ 1mm in accessory lead V_{4R} followed by positive T wave

Approach: Primary percutaneous coronary intervention (PCI). two stent placement Clopidogrel, aspirin, statin, ß-blocker following,...

Several studies have shown that primary PCI performed by experienced operators immediately after patients are admitted to high-volume tertiary care centres with STEMI results in decreased mortality, reinfarction and stroke(1)

1. **Zijlstra F, de Boer MJ, Hoorntje JC, etal. A comparison of immediate coronary angioplasty with intravenous streptokinase in acute myocardial infarction.N Engl J Med. 1993 Mar 11;328(10):680-4.**

CHARACTERISTICS OF INFERIOR INFARCTION IN ACUTE PHASE

1) ST segment elevation in inferior leads II, III and aVF;

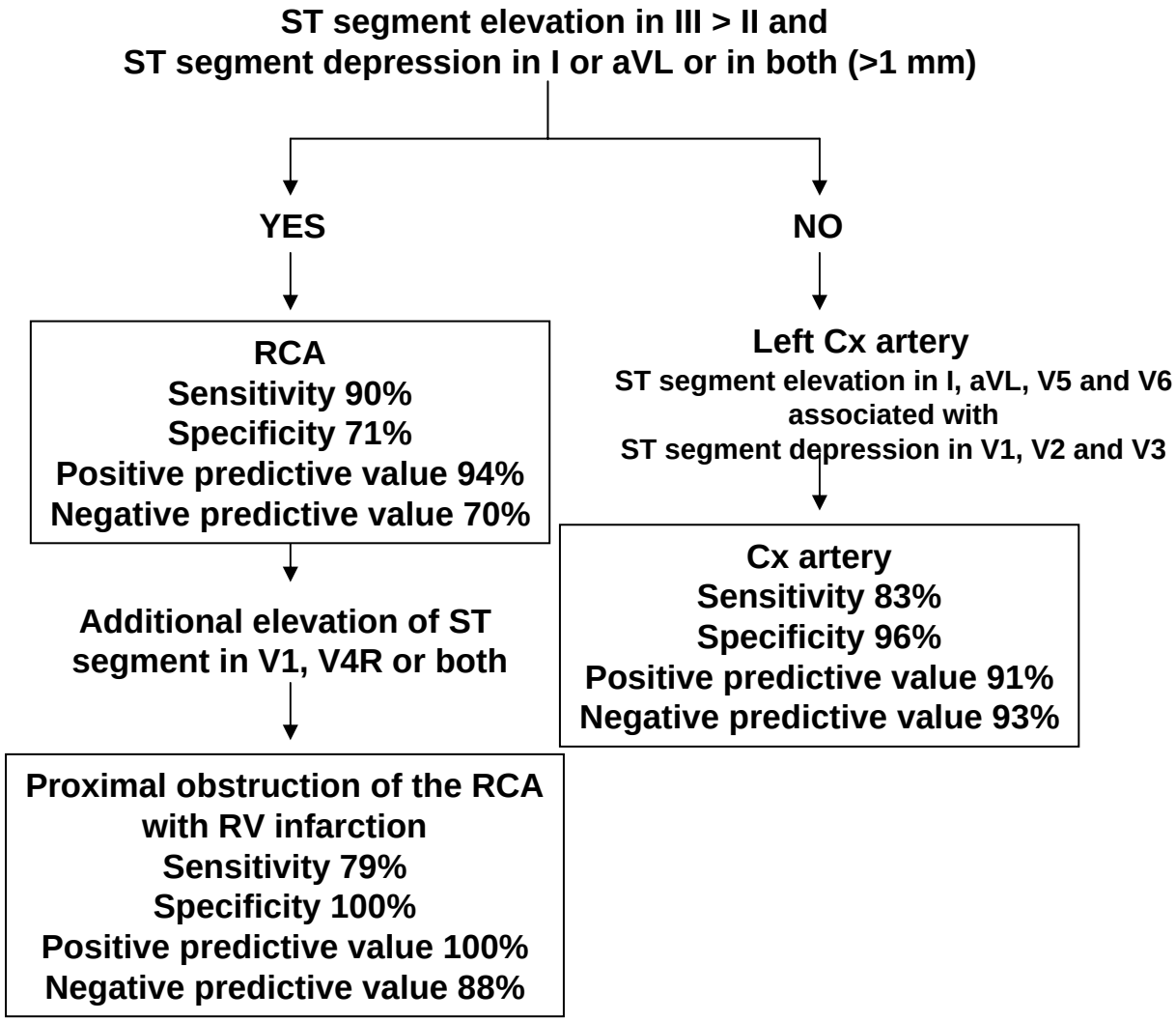
2) Reciprocal ST segment depression in anterior leads. Kosuge et al (1) showed that the degree of ST segment depression in the aVR lead is an independent predictor of reperfusion improvement in patients who suffered **inferior acute myocardial infarction.**.

3) Q wave of any size in II.

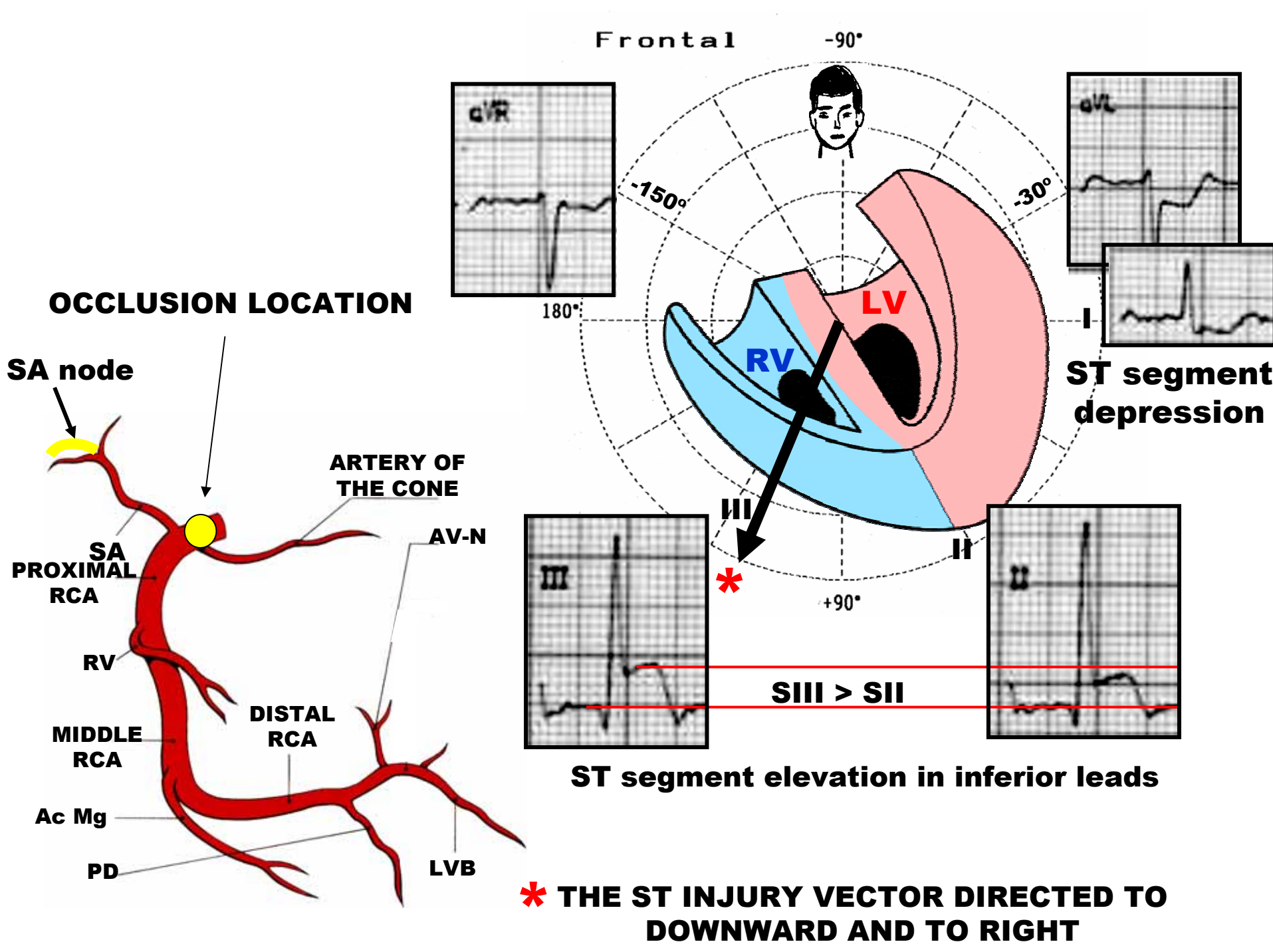
1. **Kosuge M, et al. ST-segment depression in lead aVR: a useful predictor of impaired myocardial reperfusion in patients with inferior acute myocardial infarction. Chest. 2005; 128:780-786.**

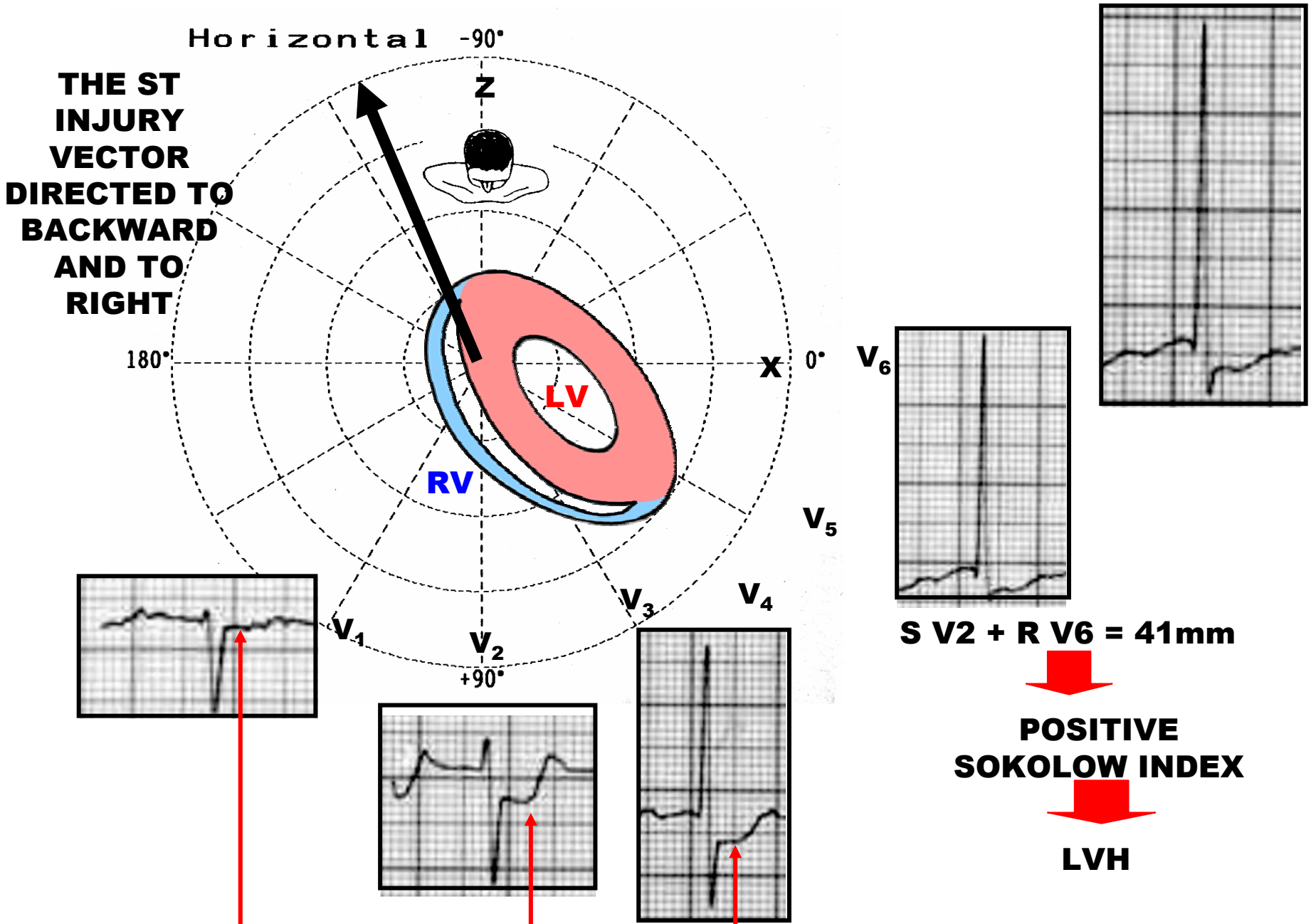
Electrocardiographic characteristics of inferior infarction in the acute phase.

ALGORITHM TO IDENTIFY THE ARTERY INVOLVED WITH INFERIOR INFARCTION BY ECG



Algorithm to identify through ECG the artery involved by inferior infarction.

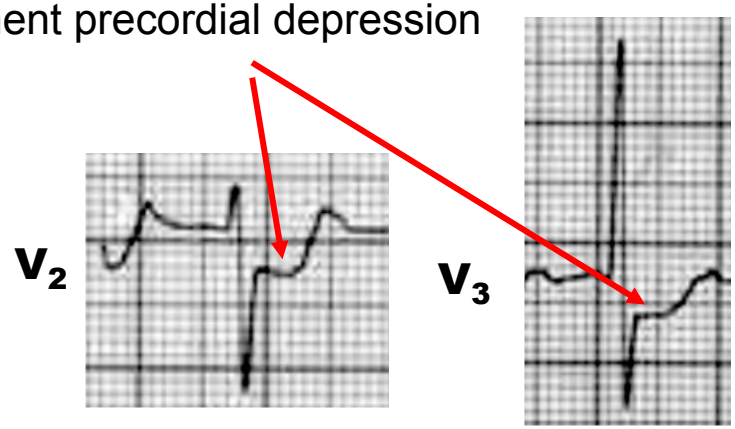

OCCLUSION LOCATION
SA node
ARTERY OF THE CONE
AV-N
SA
PROXIMAL RCA
RV
MIDDLE RCA
DISTAL RCA
Ac Mg
PD
LVB
Frontal
-90°
-150°
-30°
180°
+90°
aVR
aVL
I
LV
RV
III
II
*
SIII > SII
ST segment depression
ST segment elevation in inferior leads
* THE ST INJURY VECTOR DIRECTED TO DOWNWARD AND TO RIGHT


THE ST INJURY VECTOR DIRECTED TO BACKWARD AND TO RIGHT
Horizontal
-90°
Z
180°
X
0°
+90°
LV
RV
V1
V2
V3
V4
V5
V6
S V2 + R V6 = 41mm
POSITIVE SOKOLOW INDEX
LVH
Anterior ST segment precordial depression: multivessels disease!!

FINDING INDICATING HIGH-RISK IN INFERIOR MYOCARDIAL INFARCTION

1) ST segment elevation ≥ 1mm in accessory lead V_{4R}. When present disappear in 10 to 12hours. This signal has high sensitivity for right ventricular MI. Additionally, it is indicative of proximal obstruction of RCA if ST segment elevation is followed by a positive T wave.
2) Ischemic involvement of the RV can complicate the early course of inferior ST elevation IMI has significant management implications but its diagnosis is difficult. Kakouros et al(1) studied assessed RV involvement in the acute and late phase of IMI by pulse-wave tissue Doppler (PW-TDI) and RV myocardial performance index (RVMPI). The authors concluded that Echocardiographic assessment of the RV by PW-TDI of the lateral tricuspid annulus, is a sensitive and specific marker of RV involvement in first IMI and remains so 6 months after the ischemic event. The novel index of S'/MPI appears to have potentially improved diagnostic accuracy in identifying RV involvement and proximal RCA obstruction.
3) Complete AV block
4) Anterior ST segment precordial depression

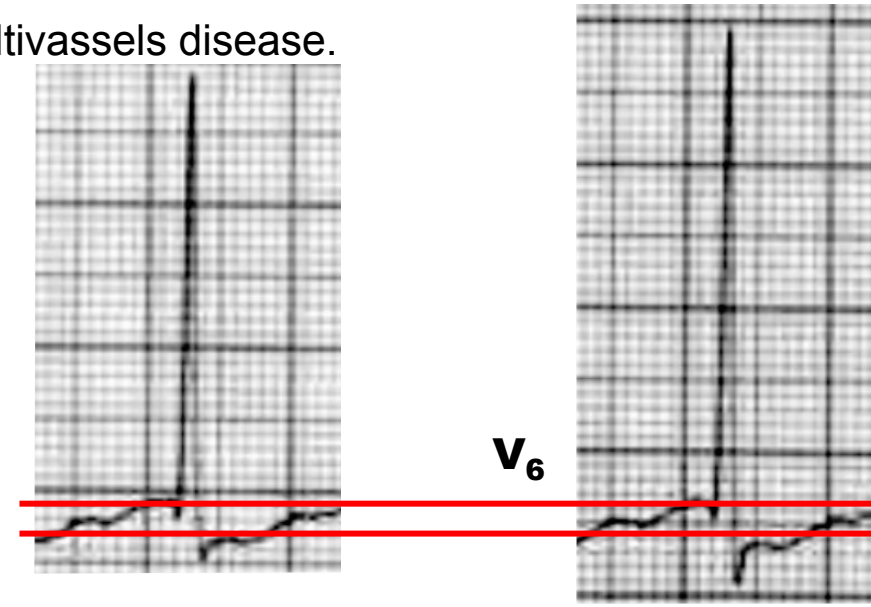
.



1. **Kakouros N, Kakouros S, Lekakis J, et al. Tissue Doppler imaging of the tricuspid annulus and myocardial performance index in the evaluation of right ventricular involvement in the acute and late phase of a first inferior myocardial infarction. Echocardiography. 2011 Mar;28:311-319.**

4) ST depression from V4 to V6 is indicative of multivassels disease.

Multivessel coronary disease significantly and adversely impacts on patient prognosis, yet a culprit-only revascularization strategy should be sought after in most cases, unless patient instability or symptoms/signs of residual myocardial ischemia support nonculprit vessel intervention.(1)



5) ST-segment elevation in V_1 on admission in patients with acute Q-wave inferior wall myocardial infarction indicates a right coronary artery lesion associated with a larger infarct size and a higher incidence of major in-hospital arrhythmias.(2;3)

1. **Biondi-Zoccai G, Lotriionte M, Sheiban I. Management of multivessel coronary disease after ST-elevation** myocardial infarction **treated by primary coronary angioplasty. Am Heart J. 2010 Dec;160:S28-35.**
2. **Tsuka Y, Sugiura T, Hatada K, et al. Clinical significance of ST-segment elevation in lead V1 in patients with acute inferior wall Q-wave myocardial infarction.Am Heart J. 2001 Apr;141:615-620**.
3. **Tsuka Y, Sugiura T, Hatada K. et. al. Clinical characteristics of ST-segment elevation in lead V6 in patients with Q-wave acute inferior wall myocardial infarction. Coron Artery Dis. 1999 Oct;10:465-46**

7) Data from the prethrombolytic era suggest that heart block occurs in approximately 20% of patients with acute inferior MI and is associated with a marked increase in mortality. Among patients receiving thrombolytic therapy mortality remain relatively high. Mortality is augmented in whom AV block developed within 24hours after thrombolitic therapy but not in those who had AV block before treatment.(1)

1. **Berger PB, Ruocco NA Jr, Ryan TJ, Incidence and prognostic implications of heart block complicating inferior myocardial infarction treated with thrombolytic therapy: results from TIMI II. J Am Coll Cardiol. 1992 Sep;20:533-540.**

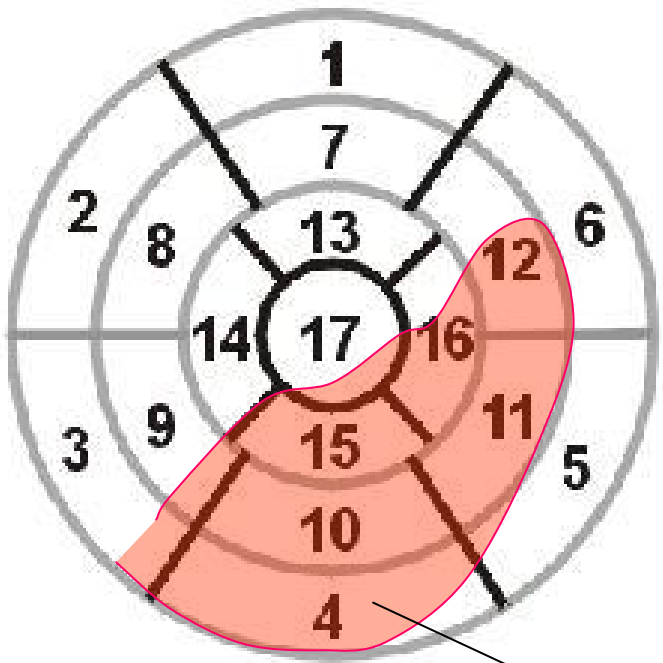
NEW ELECTROCARDIOGRAPHIC TERMINOLOGY FOR Q-WAVE INFARCTIONS BASED ON THE CORRELATION WITH CE-CMR

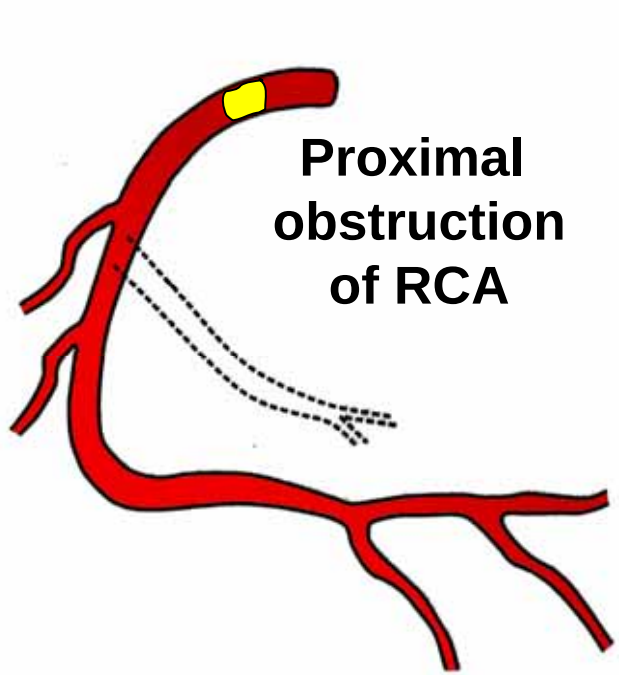
2) INFEROLATERAL ZONE

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

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

LATERAL INFARCTION
B-3





Basal inferior wall (formerly called dorsal)

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

INFERIOR OR DIAPHRAGMATIC MYOCARDIAL INFARCTION

Concept: it is the infarction where necrosis is located in the LV inferior wall and the low and dorsal regions of the interventricular septum, the electrocardiographic translation of which is recorded in II, III and VF in the VCG by the modifications in the frontal (FP) and left (LSP) or right sagittal (RSP) planes.

Irrigation of LV inferior wall

In 84% of the cases it is a consequence of occlusion in the right coronary artery (RCA) or its posterior descending (PD) branch.

In 10% of the cases, by occlusion of the circumflex artery of the left coronary (Cx) or of the PD branch that may be the branch of the Cx.

In just 4% of the cases, the inferior wall has two PD arteries, one dependent on the RCA and the other one on the Cx.

The PD artery (ramus interventriculares) originates in the RCA in 86% of the cases and in the LCx artery in 14%, or in both in 4%.

Concept of inferior wall irrigation.

INFERIOR OR DIAPHRAGMATIC INFARCTION

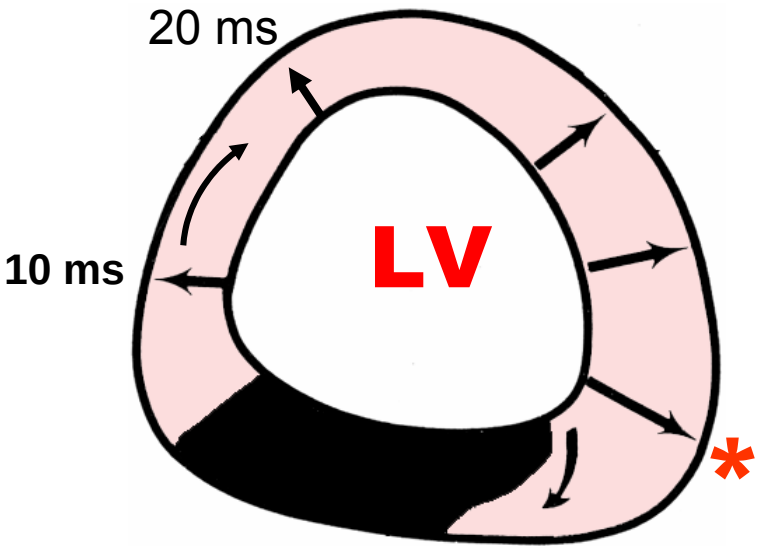
The right coronary artery (RCA) provides blood supply to the SA Node by this branch, to the right atrium (RA), part of the left atrium (LA), right ventricle (RV), AV Node, inferior wall and low and dorsal region of the left ventricle (LV).

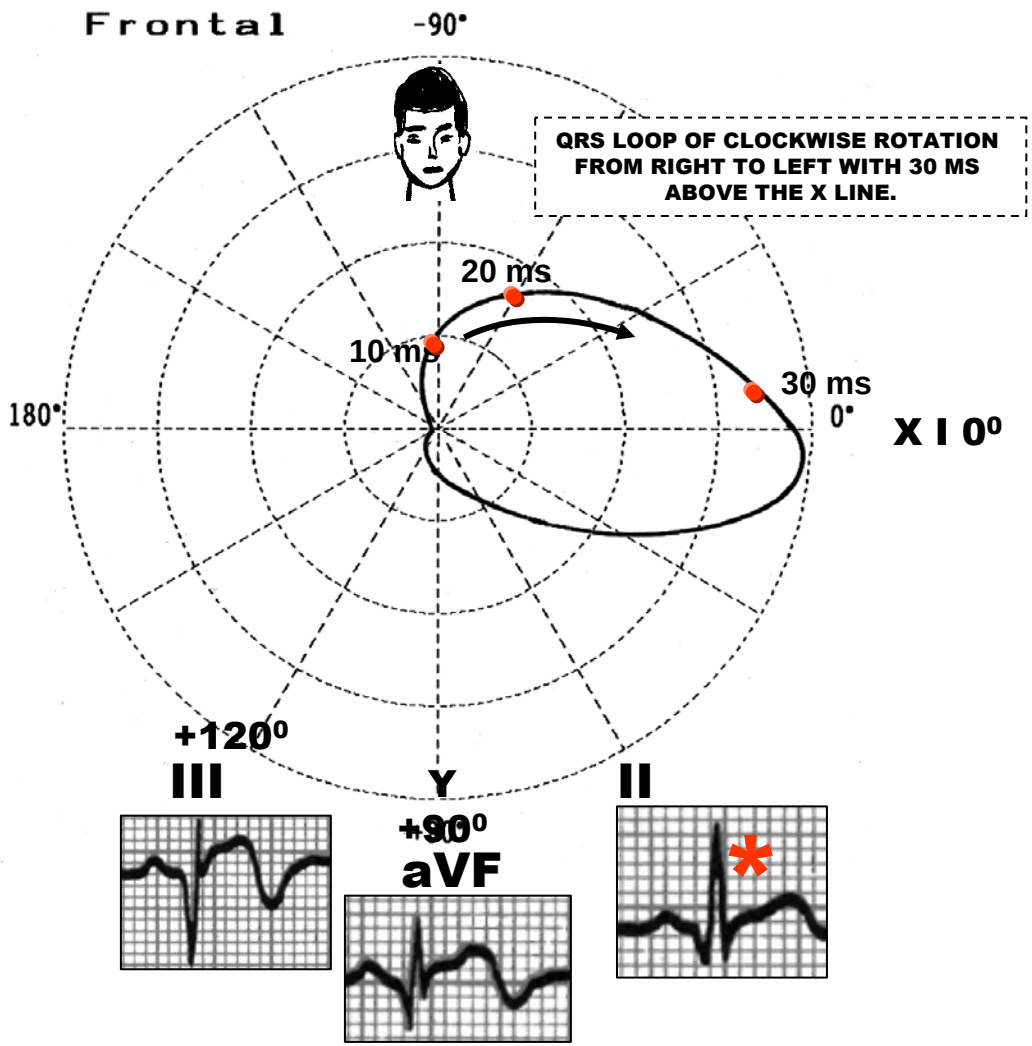
The branches of the RCA that irrigate the inferior wall are:
1) Posterior descending artery (PD);
2) Left ventricular artery (LV);
3) Postero-lateral artery (PL) that originates in the RCA in≈ 20% of the cases.

The left ventricular branch (LV) originates in the RCA in 80% of the cases and in the Left circumflex artery (LCX) in the remaining 20%.
Finally, the postero-lateral branch (PL) originates in the LCX in 80% of the cases and RCA in the remaining 20%.

Inferior wall irrigation by the branch of the right coronary and circumflex arteries.

NON-EXTENSIVE DIAPHRAGMATIC INFARCTION

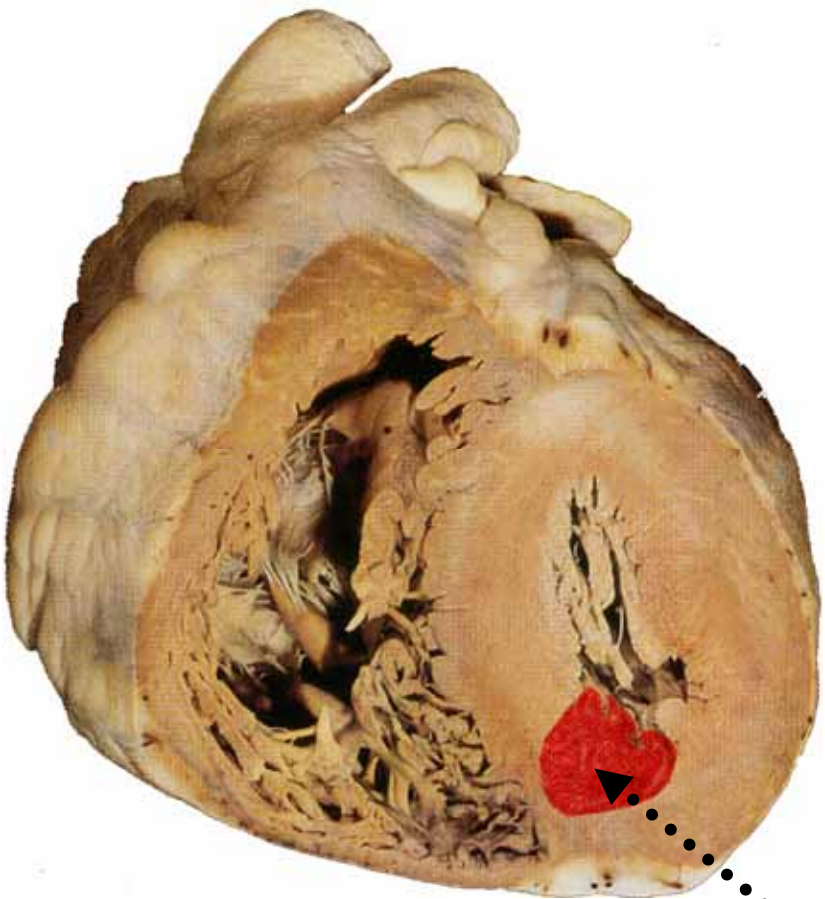


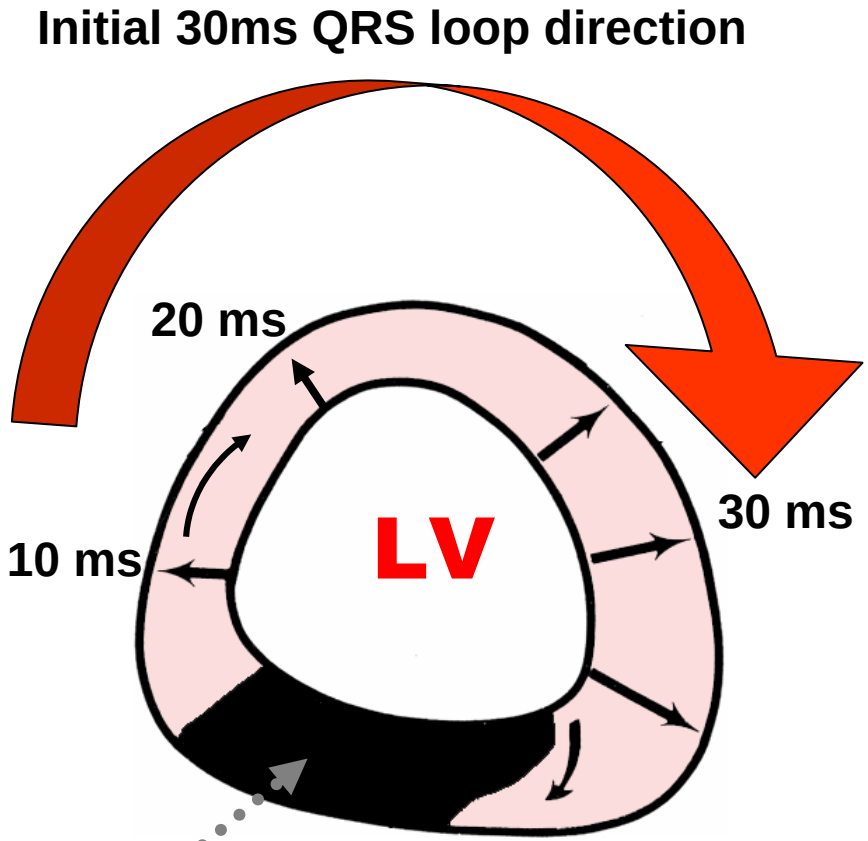


* **THIS VECTOR IS RESPONSIBLE FOR THE FINAL R WAVE IN DII, SINCE THE LEFT PART OF THE INFERIOR WALL WAS NOT AFFECTED.**

Outline of ventricular activation in the frontal plane in non-extensive diaphragmatic infarction.

NON-EXTENSIVE DIAPHRAGMATIC INFARCTION

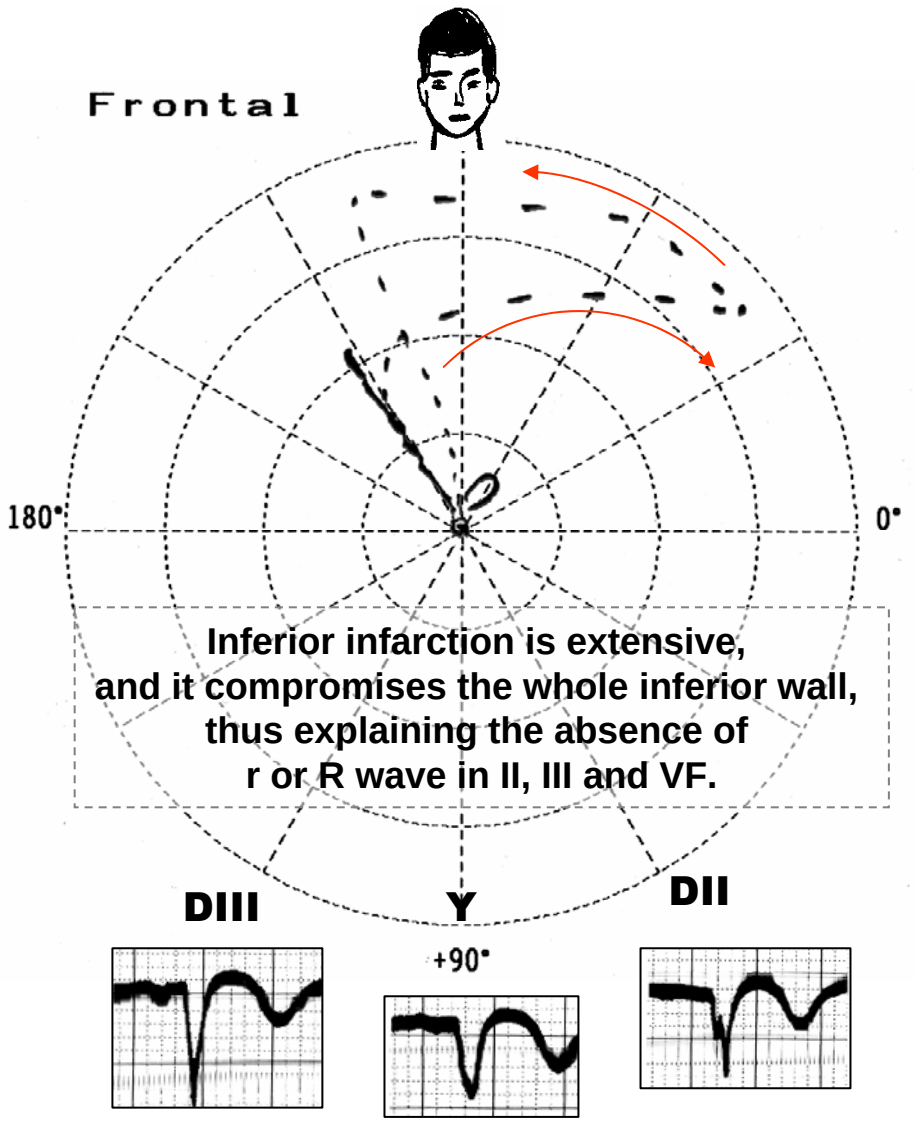




NECROTIC AREA

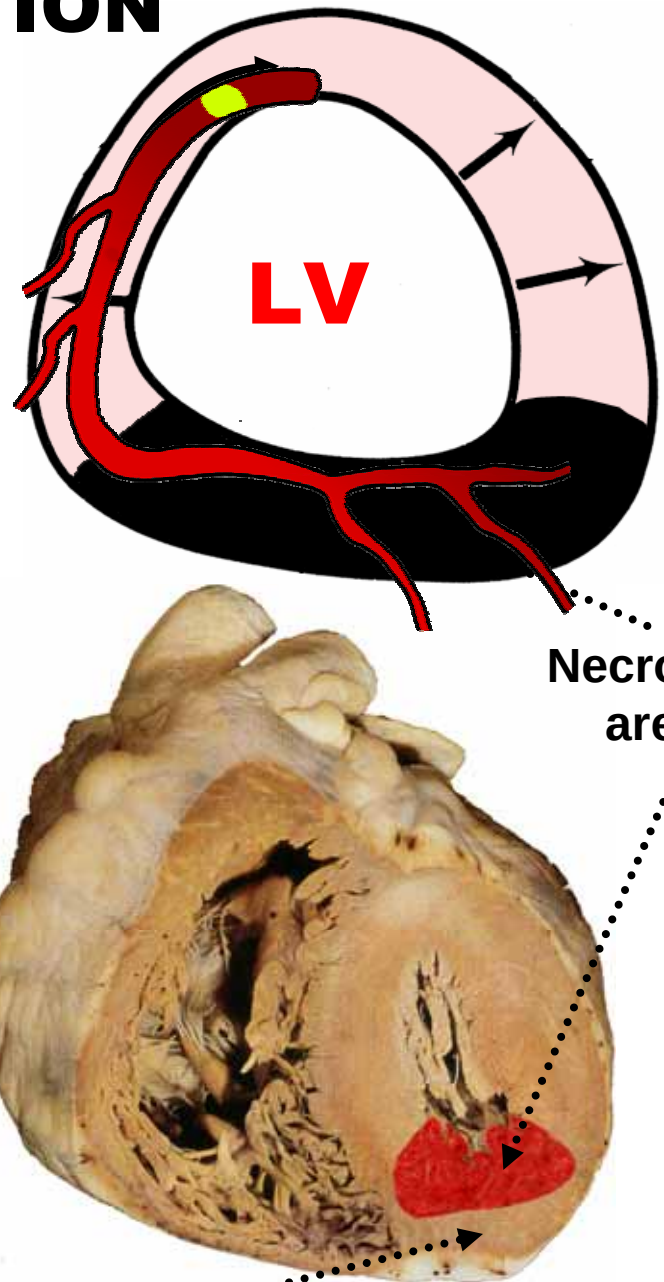
Anatomical element that displays the short axis view of the LV and the site affected in non-extensive diaphragmatic infarction and activation mode from right to left, convex to the top in the initial 30 ms.

INFEROLATERAL INFARCTION

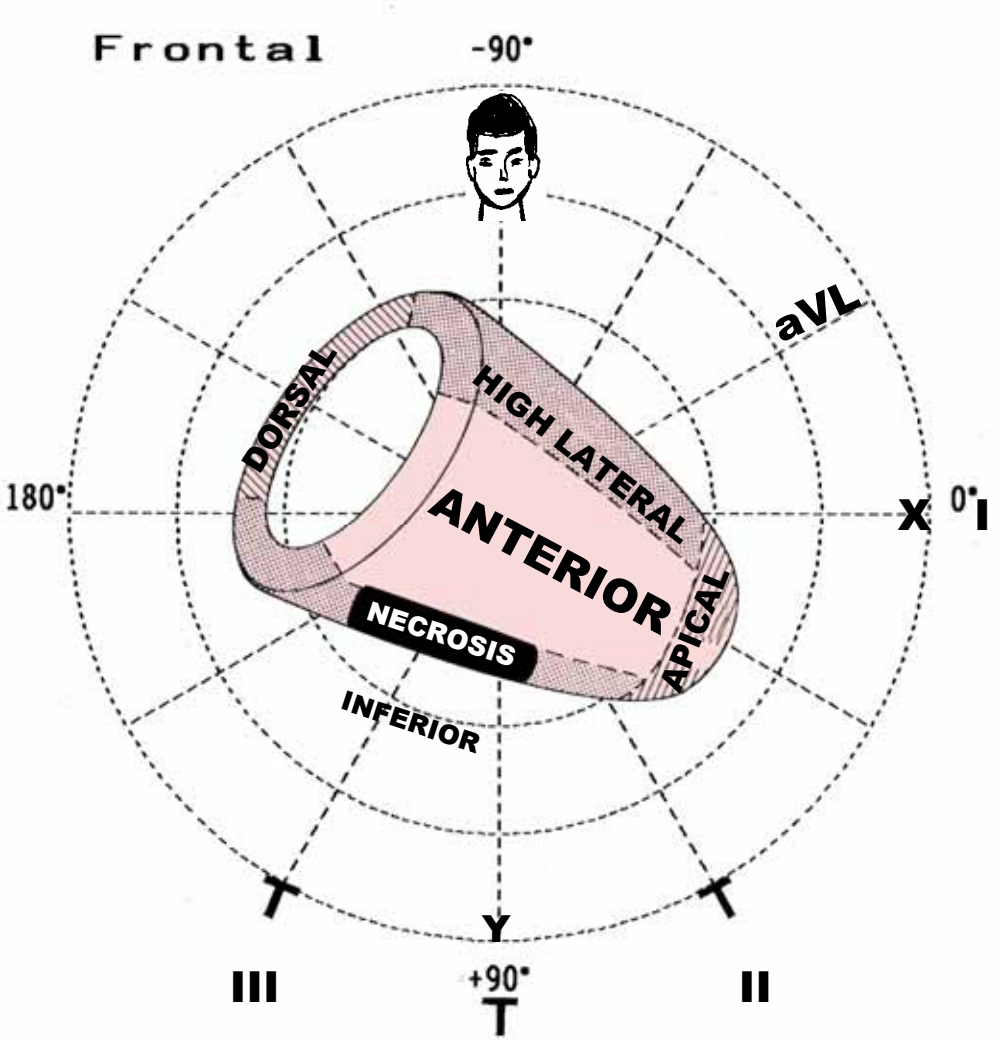


Inferior infarction is extensive, and it compromises the whole inferior wall, thus explaining the absence of r or R wave in II, III and VF.

QS in the three inferior leads



EXPLANATION OF THE REASON OF THE POSSIBLE ABSENCE OF THE Q WAVE IN II IN NON-EXTENSIVE INFERIOR INFARCTION



In the figure we observe that the II lead faces the inferior wall and part of the apical region. In the cases of not so extensive infarctions, the II lead may not show the pathological Q wave of necrosis, because it does not face the “dead” area.

Ventricular cone and necrosis area in non-extensive diaphragmatic infarction. The II lead does not face the necrosis area.

ATRIAL INFARCTION

Infarction of the cardiac atria occurs more frequently than is commonly considered. Ischemic damage to the atrial myocardium is usually associated with infarction of cardiac ventricles, but isolated infarction of an atrium can occur and may be of clinical significance[1].

Atrial infarction is rarely diagnosed before death because of its characteristically subtle and nonspecific electrocardiographic findings.

Atrial infarction is a neglected electrocardiographic sign with important clinical implications.

These findings may be overshadowed by changes associated with concomitant ventricular infarction[2].

1) Cunningham KS, Cardiovasc Pathol. 2008; 17:183-185.
2) Shakir DK.et al.Can J Cardiol. 2007;23:995-997.

ATRIAL INFARCTION

ECG DIAGNOSIS CRITERIA

PR (PRs), PQ segment (PQs), STa segment or PTa-segment: it stretches from the end of P wave to the onset of QRS complex. Displacement of this segment (depression or elevation), which represents part of the atrial ST (STa) segment only ostensive when associate with AV block as a consequence of atrial infarction (Figure 1).

Ja point: Point of junction between the end of the P wave and the onset of PRs.
(Figure 2)

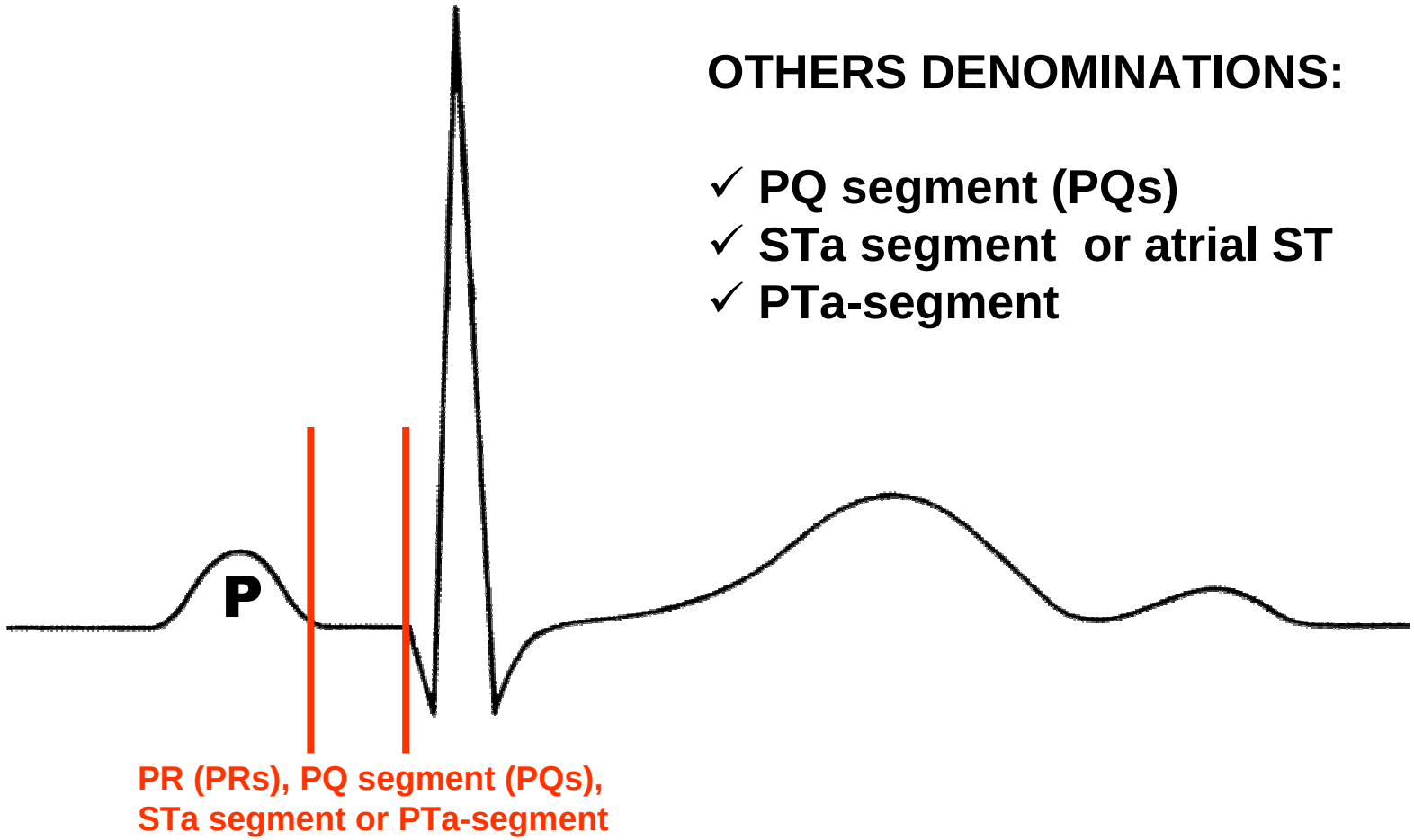
Normal location of atrial repolarization (Ta or TP wave). It coincides with ventricular depolarization (QRS complex), what explains its absence for being concealed by the ventricular phenomenon (Figure 3).

The 3 segments **PRs, ST** and **TPs** normally are at the same level (Figure 4).
Ta wave may cause falsely positive strain tests 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 and P wave of voltage higher in maximal strain.

In acute right ventricular MI high degree AV block is present in almost half of the cases.

STa segment elevation may produce a diagnostic monophasic pattern during the early stage of ventricular ischemia.

Figure 1
PR segment (PRs)

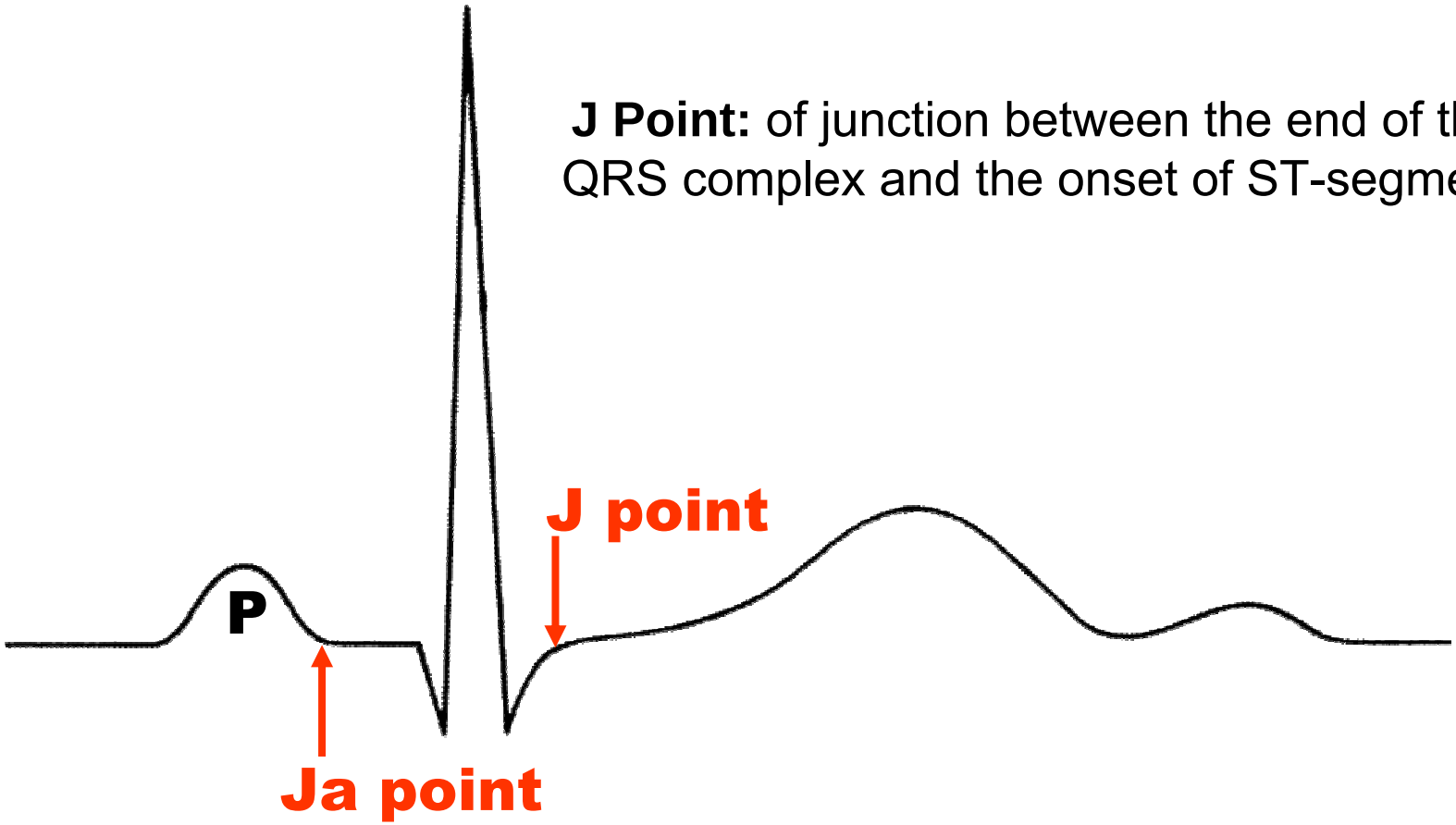


OTHERS DENOMINATIONS:

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

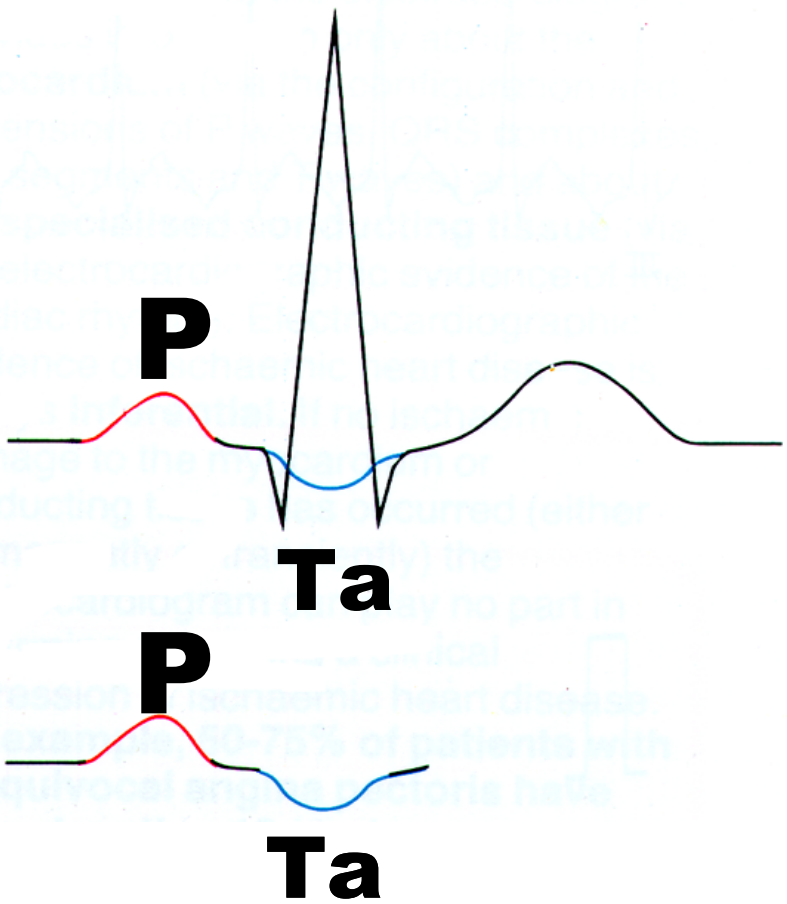
PRs: from the end of P wave to the onset of QRS complex.

Figure 2
Ja point & J point



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

Figure 3
Ta or TP wave



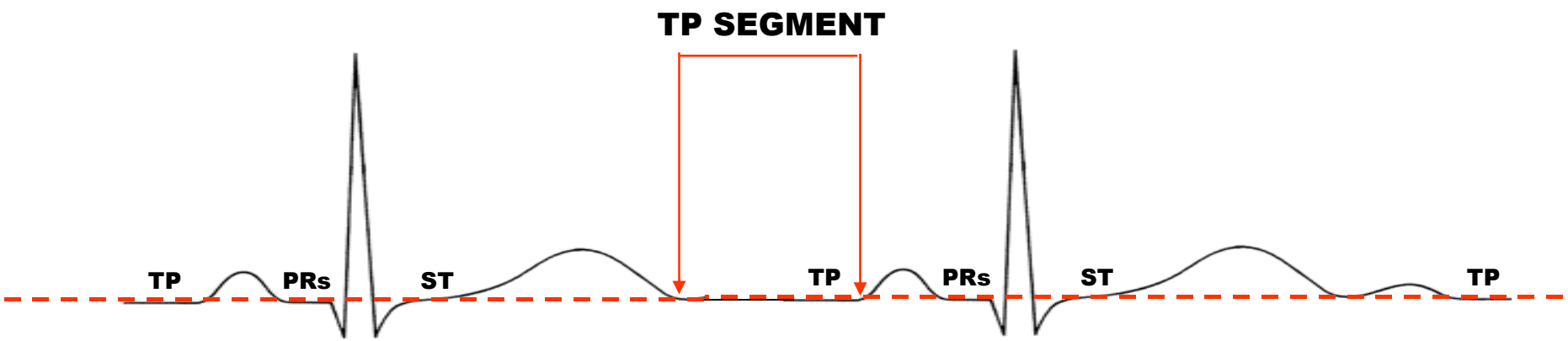
Normal location of atrial repolarization (Ta or TP wave). It coincides with ventricular depolarization (QRS complex), what explains its absence for being concealed by the ventricular phenomenon.

Its polarity is opposite to the P wave and its magnitude is 100 to 200 mμV. Sometimes it may appear in the ST segment and the T wave.
During exercise, it may in theory, cause ST segment depression and resemble myocardial ischemia [1]

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

Figure 4
CORRELATION OF LEVEL BETWEEN PRs, ST & TP

From the end of the T wave up to the onset P wave of the following cycle



The PR segment 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 (from J point or the end of QRS up to the beginning of the T wave) and TPs (from the end of the T wave up to the onset 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: PRs, ST and TPs.

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].

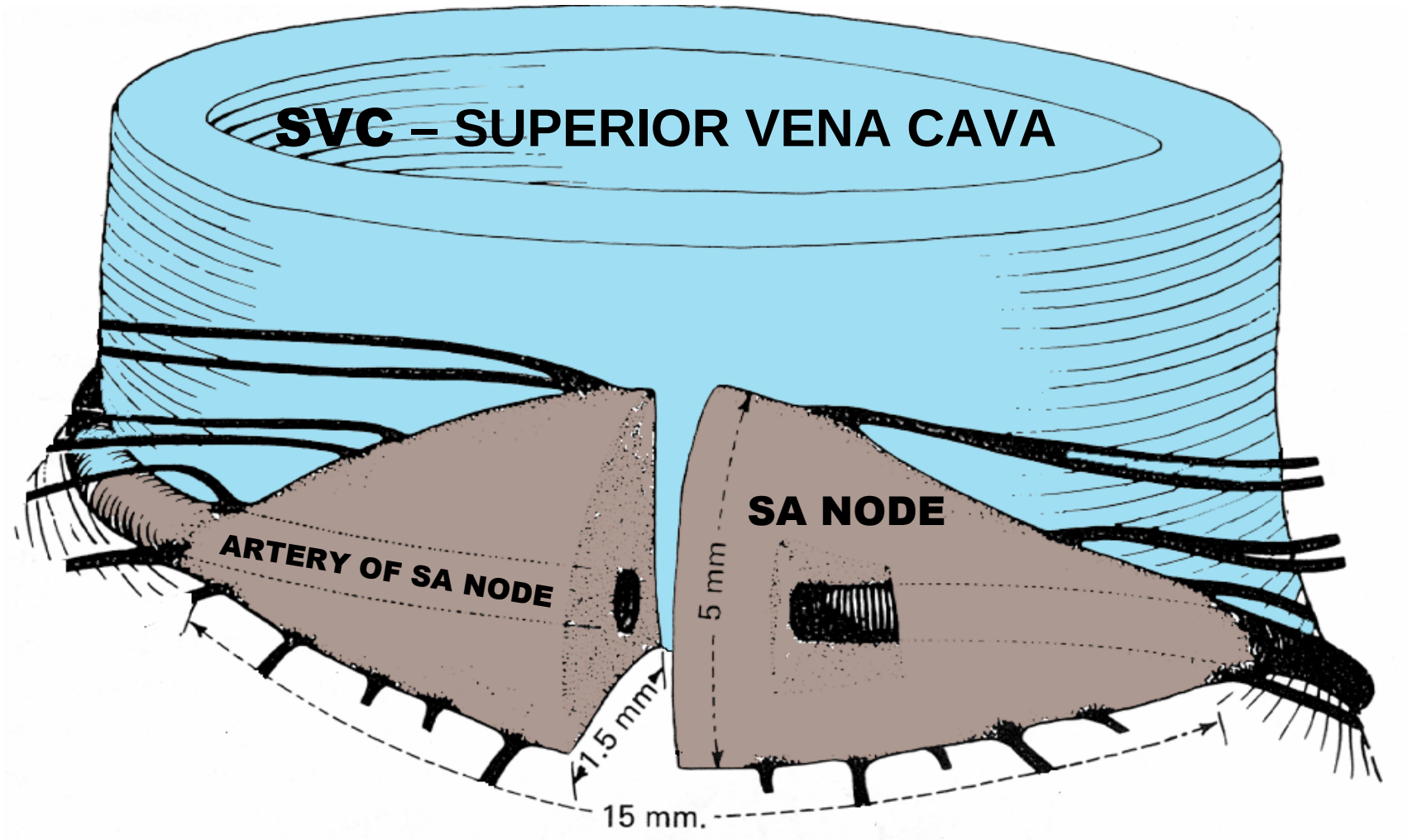
1) Kyriakidis M. Chest. 1992;101:944-947.

ATRIAL INFARCTION
COMPLICATIONS

1) 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]. (Figure 5)
2) Pump failure of the right and left ventricle
3) Atrial wall rupture
4) Thromboembolization[2]

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

FIGURE 5
RIGHT CORONARY ARTERY ORIGIN



RA – RIGHT ATRIUM

The Right coronary artery supplies the SA node artery in 60% of patients. The other 40% of the time, the SA nodal artery is supplied by the left circumflex artery.

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.

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

RIGHT VENTRICULAR MYOCARDIAL INFARCTION (RVMI) CLINICAL CHARACTERIZATION

Typical precordial pain that lasts, associated with characteristic signs of right ventricular failure:

1) Low blood pressure (BP below 90 mmHg) or shock;
2) Jugular vein and liver congestion with clean lungs;
3) Fourth noise with right ventricular gallop that increases with inspiration;
4) Possible presence of Kussmaul's sign: jugular distension at deep inspiration;
5) Paradoxical pulse.

It is seen in patients who suffered acute infarction in the inferior, (24%) infero-dorsal, infero-latero-dorsal or strictly posterior wall, and when present, it increases hospital mortality very much. Thus, hospital mortality by inferior infarction associated to RVMI is 27%; while when isolated is only 7%. There are rare cases of severe isolated RVMI by proximal injury of the RCA with no signs of infarction in the LV inferior wall.

Clinical characteristics of right ventricle infarction.

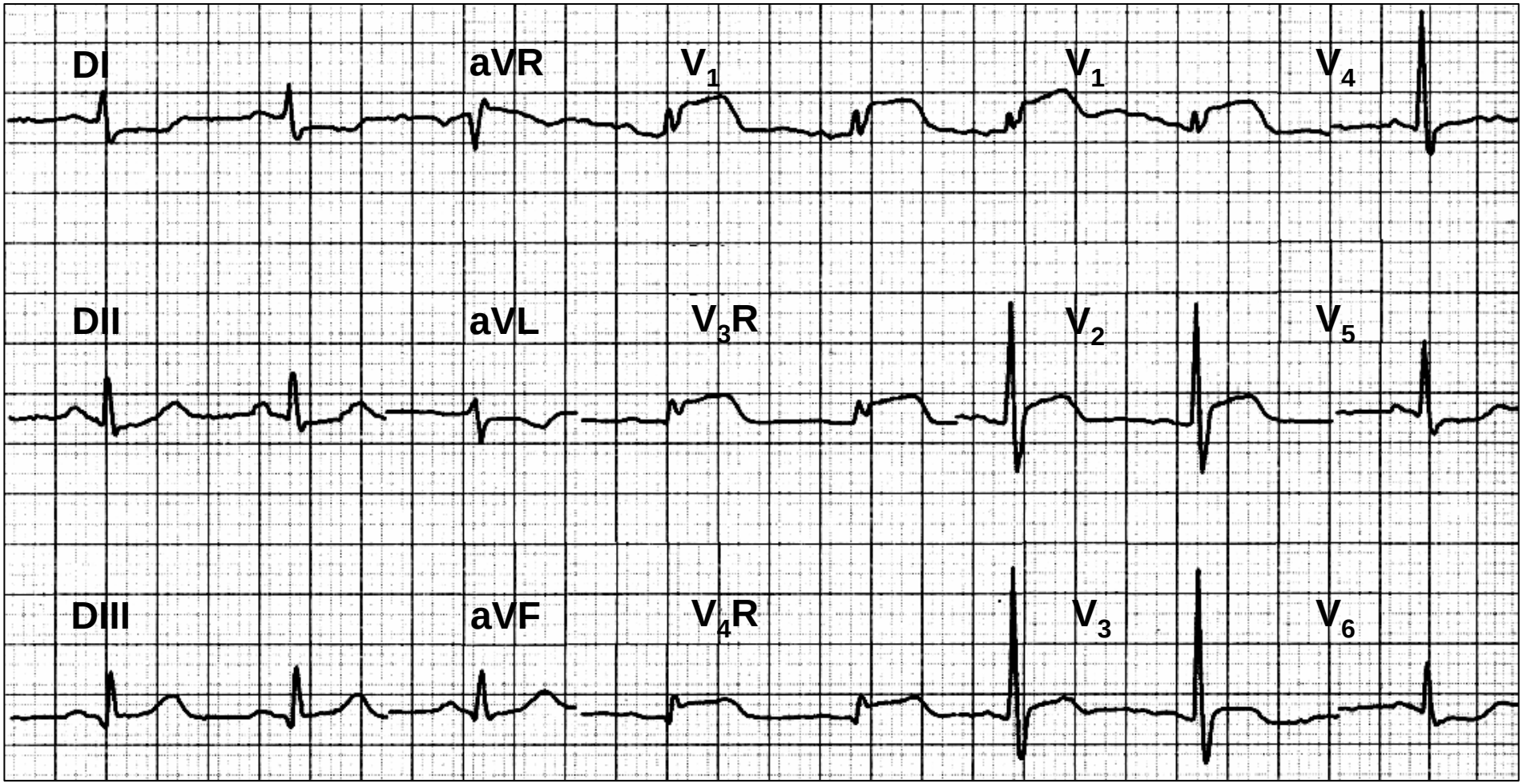
RV IRRIGATION

The different segments of the right ventricular chamber are irrigated in this way:

1) **RV free wall:** RCA truncus, except the anterior edge;
2) **RV lateral wall:** acute marginal branch (Ac Mg) or ramus marginalis dexter;
3) **RV anterior surface:** right ventricular branch of the RCA;
4) **RCA cone branch:** part of the septum;
5) **Posterior descending:** (in 86% of the cases, RCA branch): RV posterior wall. In 14% of the cases, the PD is the branch of the Cx.

Note: in a small percentage of cases, the branches of the anterior descending artery (ADA) irrigate part of the RV. This happens with prolonged ADA, called type IV ADA, which surround the tip.

ISOLATED RIGHT VENTRICULAR INFARCTION

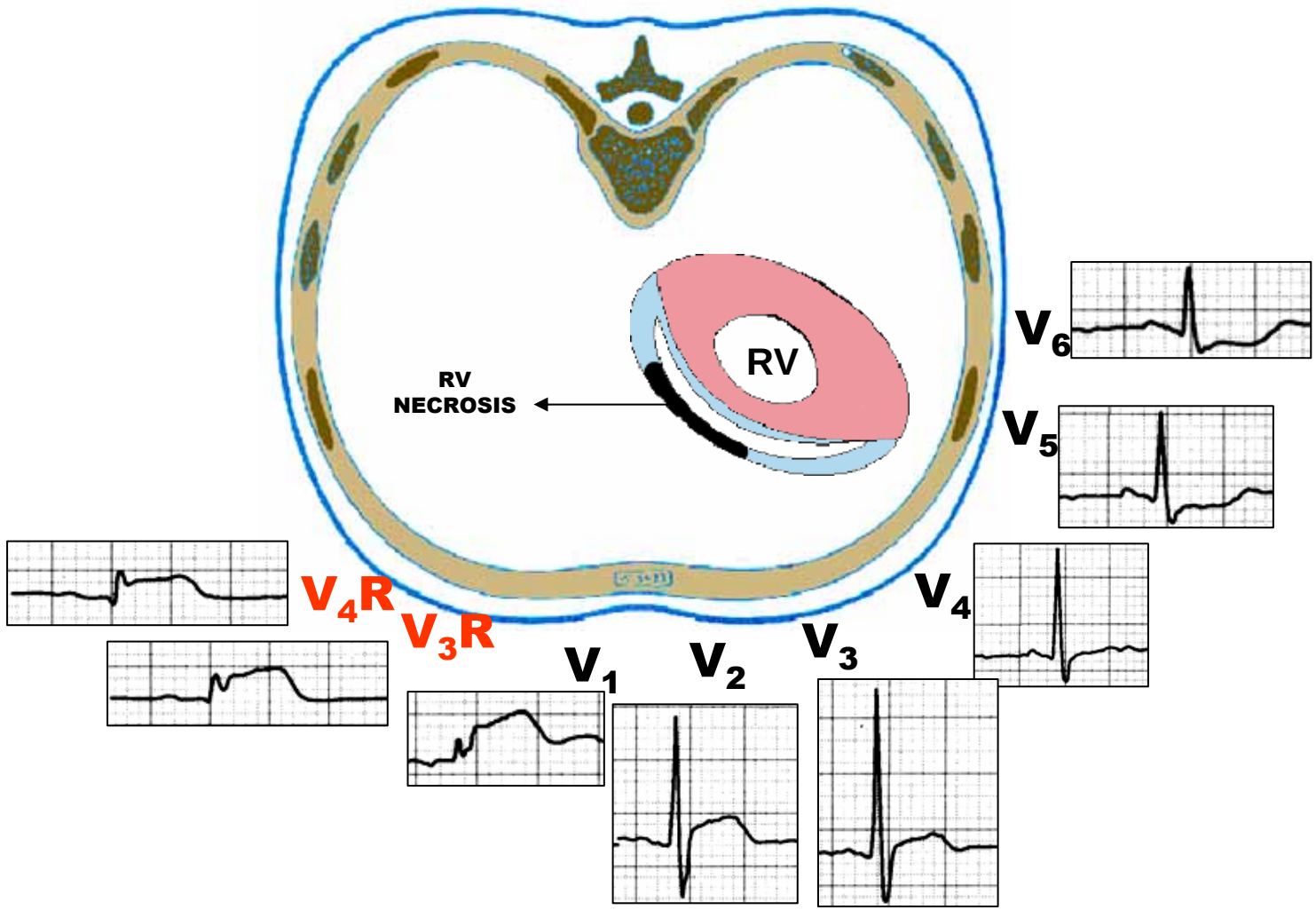


Isolated right ventricular infarction without left ventricle involvement, subepicardial injury current recorded in V_1,V_3R and V_4R.

Typical ECG of right ventricle infarction (very rare and isolated).

ISOLATED RIGHT VENTRICULAR INFARCTION

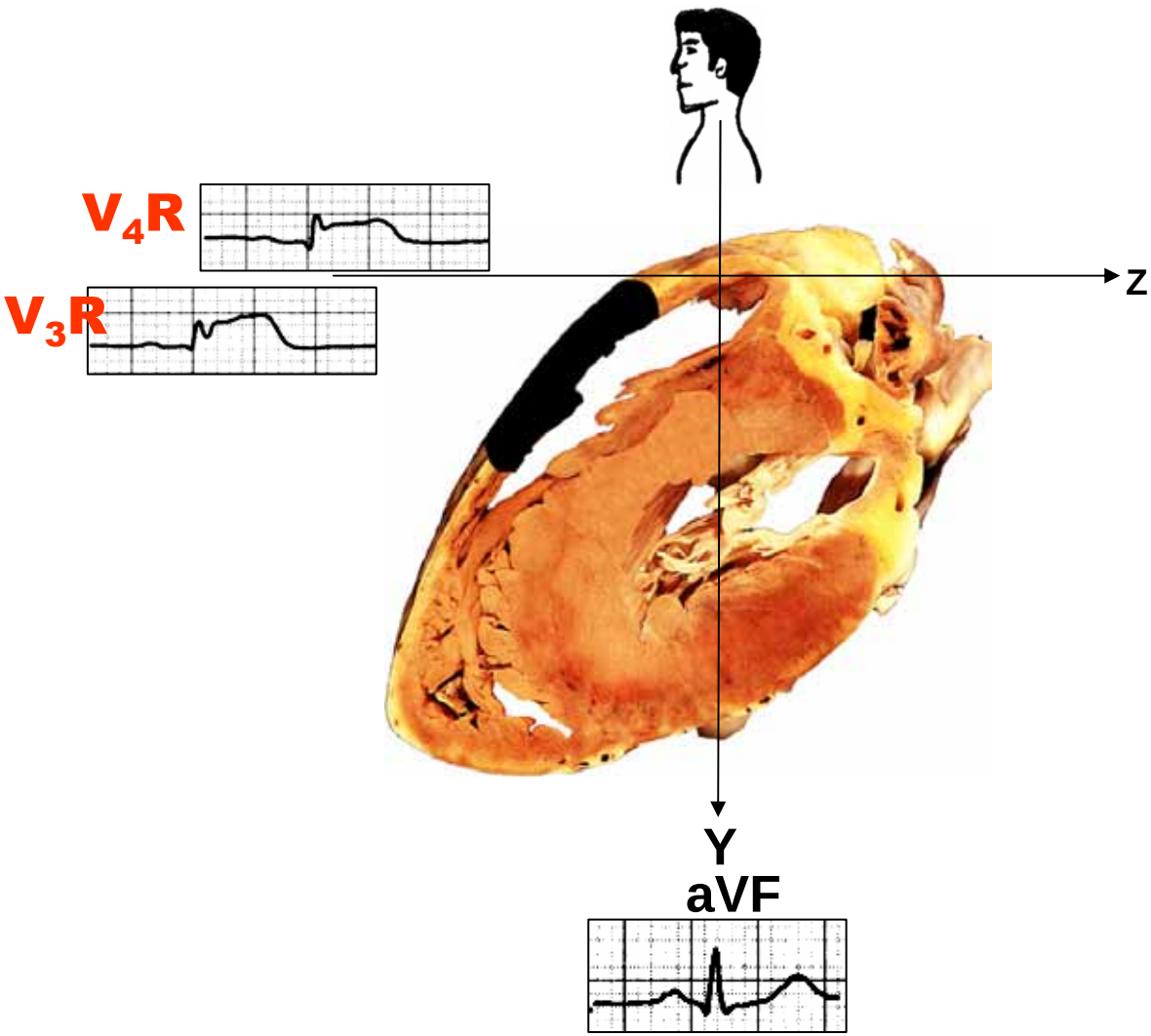
MODIFICATIONS IN ACCESSORY RIGHT PRECORDIAL LEADS



Precordial leads in right ventricle infarction. Value of accessory precordial leads (V_{3R} and V_{4R}).

ISOLATED RIGHT VENTRICULAR INFARCTION

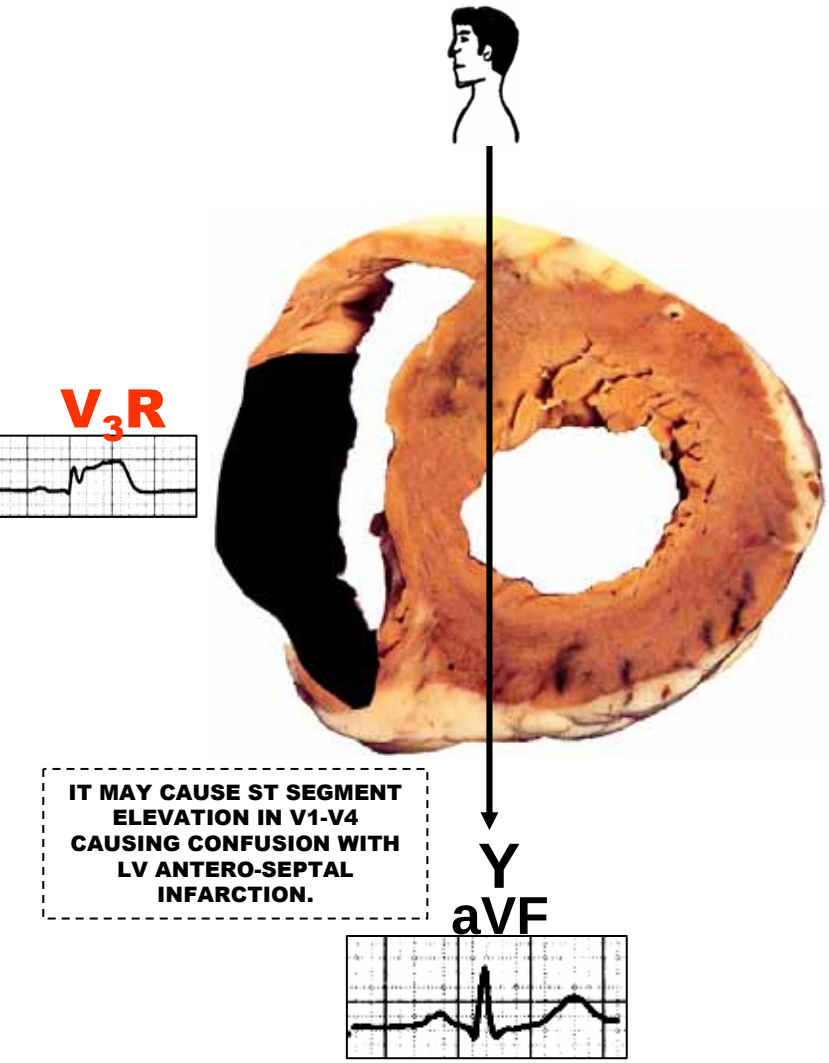
LEFT SAGITTAL VIEW



Electro-anatomical correlation of an isolated right ventricle infarction, in a left sagittal view.

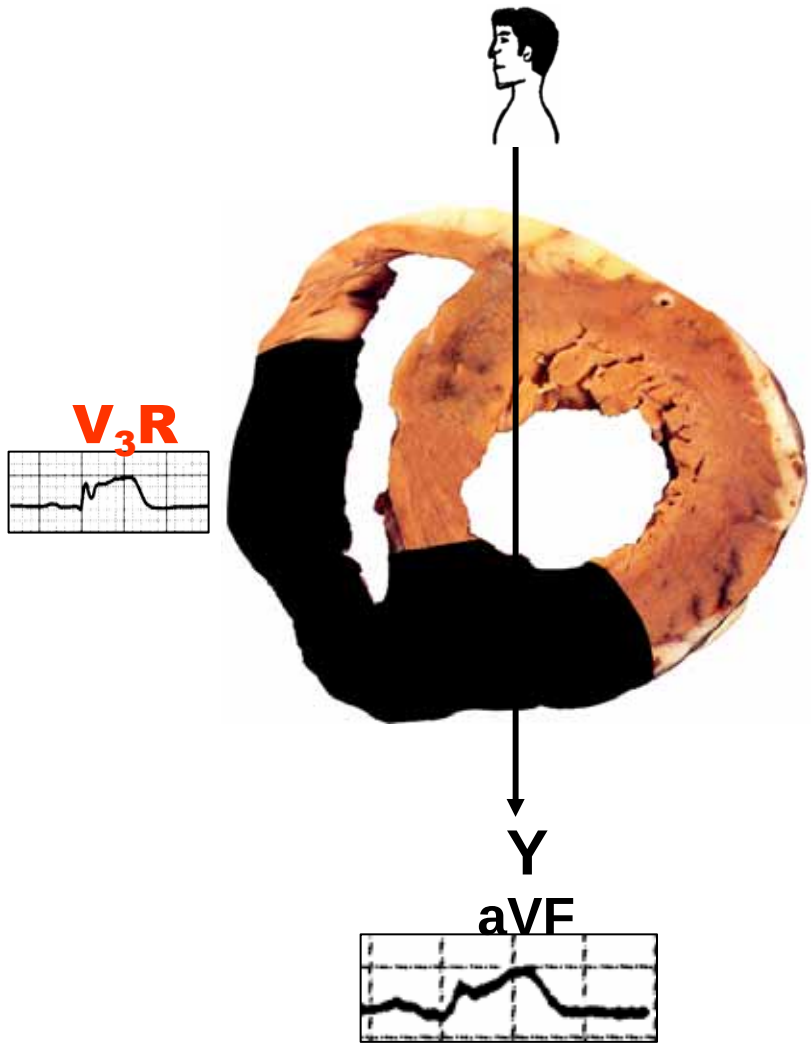
ISOLATED RV INFARCTION (EXCEPTIONAL)

VIEW IN THE MINOR AXIS



RV INFARCTION ASSOCIATED TO INFERIOR INFARCTION

VIEW IN THE MINOR AXIS



Electro-anatomical differences in isolated RV infarction and associated with diaphragmatic infarction of the left ventricle

DIFFERENCES BETWEEN ISOLATED INFERIOR INFARCTION AND ASSOCIATED TO RIGHT VENTRICULAR INFARCTION

	ISOLATED INFERIOR INFARCTION	INFERIOR INFARCTION ASSOCIATED TO RIGHT VENTRICULAR INFARCTION
Signs of right heart failure	No	Frequent
Kussmaul's sign	Negative	Positive
High degree of AV block	13%	48%
ST segment elevation from V4R to V6R	Absent	Present

Braat SH, et al. Am Heart J. 1984;107: 1183-1187

Comparative table between isolated inferior infarction and the one associated with RVMI

ECG CRITERIA FOR RV ACUTE MYOCARDIAL INFARCTION DIAGNOSIS

1. **Rhythm:** frequent atrial fibrillation, flutter, changing pacemaker, and junctional rhythm by associated atrial infarction (1/3 of patients present concomitant atrial infarction).

2. **P wave:** there may be right atrial enlargement pattern as a consequence of increase of right atrial pressure by increase of RV final diastolic pressure.

3. **PR or PQ interval:** displacement of this interval (depression or elevation), which represents par to the atrial ST segment (STa) only ostensive when associate with AV block as a consequence of atrial infarction.

4. **ST segment**: transitory ST segment elevation of 1 mm (0.1 mV) or more in at least one of the right precordial leads V3R, V4R V5R V6R. The sensitivity of the ST elevation sign in V4R is 100% and specificity is 70%. The right precordial leads should always be mapped in patients with diaphragmatic infarction and suggestive clinical symptoms. Generally, this infarction is associated with LVIMI.

5. ST elevation of 1 mm or more in the CR lead (fifth intercostal space at the right from the clavicular middle line).

6. Occasional ST segment elevation in V1-V2 especially when the LV injury is minimal.

7. ST elevation usually disappears in average in 10h.

8. ST segment depression with negative, symmetrical, deep, and wide-based T wave from V1 to V3.

9. Q or QR waves associated to ST segment elevation in right precordial leads.

10. Q wave appears in lateral dorsal necrosis of the RV, but not so in RV anterior wall necrosis. (Sensitivity 100% in V4R- low specificity).

11. QS complexes may be normal in V1 especially in elderly people. In V3R and V4R this type of complex occurs even more.

12. ST segment elevation of 1.5 mV in the intracavitary unipolar lead of the RV with the electrode located in the RV apex.

13. Right bundle branch block has been experimentally observed in dogs in all the cases of isolated RVMI and this dromotropic disorder has been found clinically, too. As RBBB is rare in inferior infarction, its presence may indicate associated involvement of the RV.

14. Total AV block: of high mortality.

15. Greater incidence if thrombolytic agents are not used early.

16. High degree AV block: present in almost half of the cases.

17. In the chronic phase of RV infarction, it cannot be diagnosed by ECG.

SENSITIVITY AND SPECIFICITY OF ST SEGMENT ELEVATION >1 mm IN V_1, V_3R AND V_{4R} IN RV INFARCTION

LEAD	SENSITIVITY	SPECIFICITY
V_1	28%	92%
V_3R	69%	97%
V_4R	93%	95%

ST segment elevation $\geq$1 mm or 0.1 mV in one or more leads from V4 to V6 presents a high sensitivity (90%) and specificity to identify RV acute myocardial infarction[1].

1) Croft, CH. et al. Am J Cardiol.2001;50:3421-427

Electrocardiographic criteria of right ventricle infarction.

INFERIOR OR DIAPHRAGMATIC INFARCTION

Electrocardiographic manifestations of inferior infarction occur mainly in inferior leads II, III and VF, influencing according to the evolutionary moment, the QRS complex (necrosis), the ST segment (injury) and the T wave (ischemia).

1) **HYPERACUTE PHASE** (few hours): an increase in intrinsicoid deflection time is observed in QRS, a significant ST segment elevation of superior concavity, followed by positive, symmetrical T wave with increased voltage;
2) **ACUTE PHASE** (first days): appearance of pathological Q wave (40 ms), ST segment elevation of superior concavity followed by inverted, symmetrical or wide-based T wave;
3) **CHRONIC OR ESTABLISHED PHASE:** it is characterized by residual pathological Q wave, present in 65% to 70% of the cases.
   In the three leads in >15 % of the cases; only in II in >25 % of the cases; only in VF in 5% to 10%; and in III and aVF in 25% to 30%.

The three chronological phases of inferior infarction and electrocardiographic manifestations: hyperacute, acute and chronic.

INFERIOR OR DIAPHRAGMATIC INFARCTION

It may be observed in any of these phases with concomitant ST segment elevation and T wave with greater voltage and symmetrical in the anterior wall, which is known as reciprocal or mirror image.

Diaphragmatic infarctions that present reciprocal image have a worse prognosis because they have less ejection fraction and are more extensive infarctions. (like the present case)

Catheterization has shown that 50% of diaphragmatic infarctions present obstructive lesion of the LAD concomitantly.

Meaning of the "mirror" or reciprocal image

INFERIOR OR DIAPHRAGMATIC INFARCTION

The patients with acute anterior infarction and Q waves in the inferior leads are carriers of minor infarctions, with a greater incidence of middle-distal occlusion of the LAD and relatively preserved ventricular function.

The patients with acute anterior infarction without reciprocal alterations in the inferior leads are carriers of better ventricular function.

Patients of acute anterior wall MI with Q waves in inferior leads indicate a smaller infarct with higher incidence of mid/distal LAD occlusion and a relatively preserved LV function. acute anterior wall MI patients without reciprocal changes in inferior leads have a better LVEF. Patients of acute anterior wall MI with ST depression in apicolateral leads have more occurrence of multivessel disease with significant LV dysfunction. Reciprocal ST depression in I, aVL suggests a possibility of RCA lesion.(1)

1. **Parale GP, Kulkarni PM, Khade SK, et al. Importance of reciprocal leads in acute myocardial infarction. J Assoc Physicians India. 2004 May;52:376-379.**

Meaning of acute anterior infarction with Q wave in inferior leads.