Meaning of the so-called reciprocal changes on the ECG: Opinions of ECG experts (and non-experts: me)

Dear Andrés and Acácio:

Thank you very much for this interesting case. I have just one additional comment: I am not very happy with using the term “Reciprocal ST depression - Reciprocal effect or mirror image”. This term does not reflect the spatial location of leads and their axes – definitely leads aVF and V1 – V3 are not opposite leads to show “a mirror image”.

Some details can be found in:

1. Bacharova L. Reciprocal ST segment depressions in myocardial infarction which are not reciprocal. J Electrocardiol. 2020;61:61-62.
2. Bacharova L, Szathmary V, Mateasik A. QRS complex and ST segment manifestations of ventricular ischemia: the effect of regional slowing of ventricular activation. J Electrocardiol. 2013;46(6):497-504.

My very best regards,

Ljuba

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Estimados Andrés y Acácio:

Muchas gracias por este interesante caso. Solo tengo un comentario adicional: no estoy muy de acuerdo con el uso del término "depresión recíproca ST: efecto recíproco o imagen especular". Este término no refleja la ubicación espacial de los electrodos y sus ejes: definitivamente los electrodos aVF y V1 - V3 no son electrodos opuestos para mostrar "una imagen especular".

Algunos detalles se pueden encontrar en:

Bacharova L. Depresiones recíprocas del segmento ST en el infarto de miocardio que no son recíprocas. J Electrocardiol. 2020; 61: 61-62.

1. Bacharova L. Reciprocal ST segment depressions in myocardial infarction which are not reciprocal. J Electrocardiol. 2020;61:61-62.
2. Bacharova L, Szathmary V, Mateasik A. QRS complex and ST segment manifestations of ventricular ischemia: the effect of regional slowing of ventricular activation. J Electrocardiol. 2013;46(6):497-504.

Dear colleagues: It would be interesting if Dra Ljuba Bacharova commented on this work below as well as Samuel, Nikus, Fiol o Bayés so that we - the ignorant as the one who writes these lines - can understand the phenomenon of the so-called - at least until now - reciprocal changes or mirror images- . Furthermore, if you agree with this work and why

Prezados colegas: Seria interessante que a Dra Ljuba comentara sobre este trabalho abaixo assim como se o desejarem Samuel, Nikus, Fiol o Bayés para que nós - os ignorantes como o que escreve estas linhas - consigamos entender o fenômeno dos assim denominados -pelo menos até o presente momento - câmbios recíprocos ou imagens em espelho Ademais, se concordam com este trabalho e porque

Estimados colegas: Sería interesante sila Dra Ljuba Bacharova. comentara este trabajo a continuación, así como si lo desean Samuel, Nikus, Fiol o Bayés para que nosotros, los ignorantes como el que escribe estas líneas, podamos entender el fenómeno de los llamados cambios recíprocos, al menos hasta ahora. o imágenes espejo Además, si está de acuerdo con este trabajo y por qué

Andrés Ricardo Pérez-Riera MD PhD

Reciprocal ST-Segment Changes in Myocardial Infarction: Ischemia at Distance Versus Mirror Reflection of ST-Elevation

Gaurang Nandkishor Vaidya 1, Steve Antoine 1, Syed Haider Imam 2, Hani Kozman 2, Harold Smulyan 2, Daniel Villarreal 3 Reciprocal ST-Segment Changes in Myocardial Infarction: Ischemia at Distance Versus Mirror Reflection of ST-Elevation Am J Med Sci. 2018 Feb;355(2):162-167. doi: 10.1016/j.amjms.2017.09.004.

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Abstract: Background: Reciprocal ST-depression in the ECG's of patients with ST-elevation myocardial infarction (STEMI) results from either true ischemia at a distance via collateral circulation diverting blood to the infarcted region or an electrical phenomenon that results from a mirror reflection of ST-elevation. We aimed to identify the role of reciprocal ECG changes in predicting collateral circulation to the infarcted area determined angiographically. Methods: In a retrospective study, ECG and angiography of 53 STEMI patients admitted to SUNY Upstate Medical University in 2014 were reviewed independently by experts blinded to the results of ECG and coronary angiography.

Results: Reciprocal changes (RC) in ECG were present in 41 patients (77%) and on angiography, 14 patients (26%) exhibited collateral vessels to the ischemic areas. No correlation was found between the presence of RC and collateral circulation (p=0.384), or between the depth of reciprocal ST-depression and the degree of the collateral circulation (p=0.195). However, 84% patients without collaterals exhibited resolution of RC after successful coronary intervention(PCI) (p=0.036), suggesting that the ST depressions that resolved after reperfusion were directly caused by the culprit vessel. Patients without RC presented late after symptom onset (9.25 vs 3.83 hours, p=0.004), also suggesting time related resolution.

Conclusions: RC had no relation to or predictive value for collaterals on angiography. Among late presenting patients, RC were less frequent. Thus, reciprocal ST-depression may represent sub-endocardial ischemia from the primary coronary event or simply an electrical phenomenon, rather than ischemia at distance from impaired collateral circulation.

Spanish

Resumen:

Antecedentes: la depresión recíproca del segmento ST en los ECG de pacientes con infarto de agudo de miocardio con elevación del segmento ST (STEMI) resulta de una isquemia verdadera a distancia a través de la circulación colateral que desvía la sangre hacia la región infartada o de un fenómeno eléctrico consecuencia de un reflejo en espejo de la elevación del segmento ST. El objetivo fue identificar el papel de los cambios recíprocos de ECG en la predicción de la circulación colateral al área infartada determinada angiográficamente.

Métodos: en un estudio retrospectivo, expertos en electrocardiografia y angiografía estudiaron 53 pacientes con STEMI ingresados en la Universidad de Medicina Estadual de SUNY "The **State University** of New York" en 2014 fueron revisados los resultados de ECGs y angiografías coronarias de forma independiente por expertos cegados.

Resultados: los cambios recíprocos (RC) en los ECGs estuvieron presentes en 41 pacientes (77%) y en las angiografías, 14 pacientes (26%) exhibieron vasos colaterales en las áreas isquémicas. No se encontró correlación entre la presencia de RC y circulación colateral ($p = 0.384$), o entre la profundidad de la depresión ST recíproca y el grado de circulación colateral ($p = 0.195$). Sin embargo, el 84% de los pacientes sin colaterales mostraron resolución de los cambios recíprocos después de una intervención coronaria exitosa (PCI) ($p = 0.036$), lo que sugiere que las depresiones del ST que se resolvieron después de la reperfusión fueron causadas directamente por el vaso culpable. Los pacientes sin RC presentaron tarde después del inicio de los síntomas (9,25 frente a 3,83 horas, $p = 0,004$), lo que también sugiere una resolución relacionada con el tiempo.

Conclusiones: Cambios recíprocos (RCs) no tuvieron relación o valor predictivo para las garantías en la angiografía. Entre los pacientes de presentación tardía, los RCs fueron menos frecuentes. Por lo tanto, la depresión ST recíproca puede representar isquemia subendocárdica del evento coronario primario o simplemente un fenómeno eléctrico, en lugar de isquemia a distancia de la circulación colateral deteriorada.

Conclusões: Câmbios Recíprocos (RCs) não tiveram relação ou valor preditivo para colaterais na angiografia. Entre os pacientes de apresentação tardia, os RCs foram menos frequentes. Assim, a depressão ST recíproca pode representar isquemia subendocárdica do evento coronariano primário ou simplesmente um fenômeno elétrico, em vez de isquemia à distância da circulação colateral comprometida.

Thanks in advance

Andrés.

Hello. I am well aware that for example leads aVL and III are not absolutely anatomically opposite. However, we use the term “reciprocal” to describe that we don’t consider most of the “mirror-image” ST/T changes (such as ST depression in aVL in the case below with Prinzmetal angina) as ischemic, but as the net effect of “almost opposite” electrical forces during ischemia. One typical case is proximal LAD occlusion with ST elevation and a positive T wave in lead aVL and a “mirror-image”, “reciprocal” ST depression and a negative T wave in lead III. Another one is ST elevation in III in RCA occlusion with concomitant ST depression in aVL. I think that the ST depression in these examples are not caused by ischemia (such as ischemia at a distance). Sometimes ischemia at a distance is evident in 2- or 3-vessel disease with old total occlusion and acute total occlusion of another artery. However, it is always interesting with new knowledge!

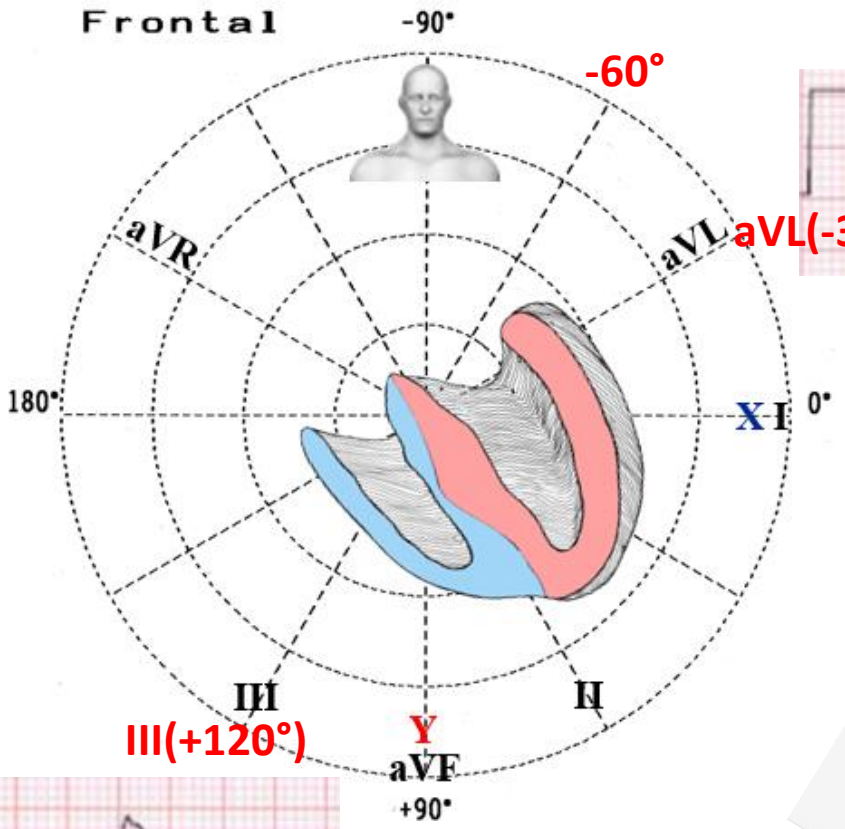
Best regards

Hola. Soy consciente de que los electrodos de aVL (-30°) y III(+120°) no son absolutamente anatómicamente opuestos(ver figura próximo slide) Sin embargo, usamos el término "recíproco" para describir que no consideramos la mayoría de los cambios de ST/T de "imagen especular" (como la depresión del ST en aVL en el caso de la próxima diapositiva que muestra las derivaciones del PF e un paciente portador de angina de Prinzmetal) como isquémicos, sino como efecto neto de fuerzas eléctricas "casi opuestas" durante la isquemia. Un caso típico es la oclusión LAD proximal con elevación del ST y una onda T positiva en la derivación aVL y una "imagen espejo", depresión ST "recíproca" y una onda T negativa en la derivación III. Otra es la elevación del ST en III en la oclusión RCA con depresión ST concomitante en aVL. Creo que la depresión ST en estos ejemplos no es causada por isquemia (como la isquemia a distancia). A veces, la isquemia a distancia es evidente en la enfermedad de 2 o 3 vasos con oclusión total antigua y oclusión total aguda de otra arteria.

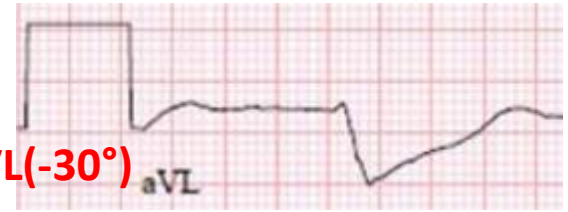
Sin embargo, ¡siempre es interesante con nuevos conocimientos!

Atentamente Kjell Nikus

A steep down-sloping ST-segment elevation, resembling the Greek letter lambda in the II, iii and aCF leads.

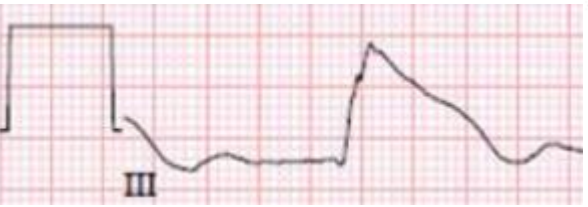


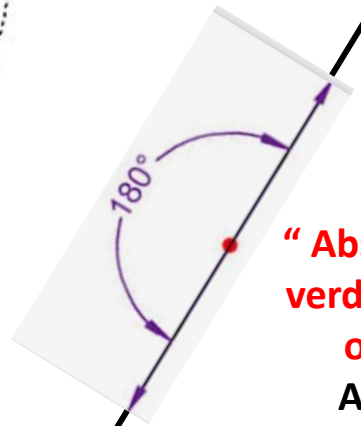
“almost opposite”
"casi opuestas"



aVL(-30°)

III(+120°)

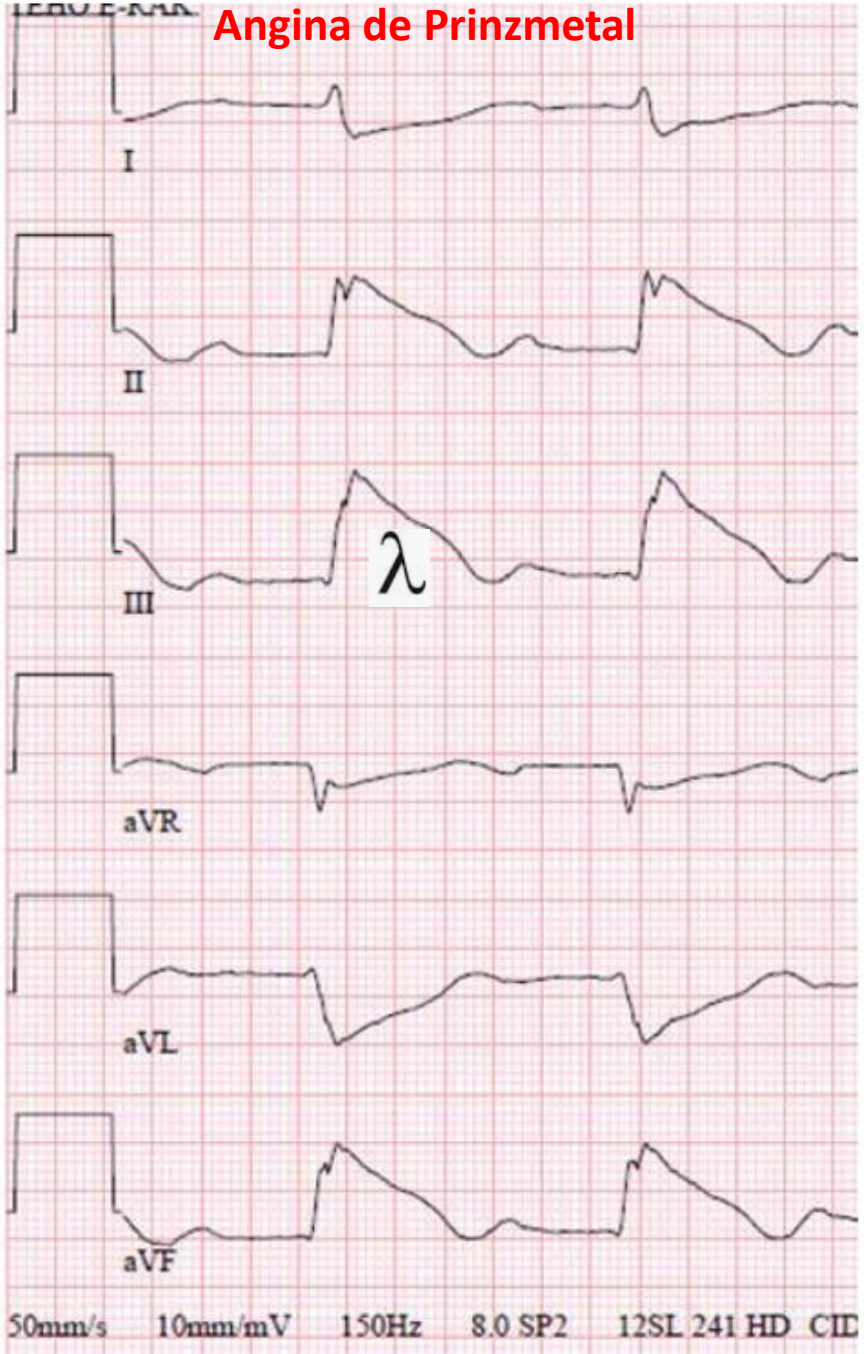


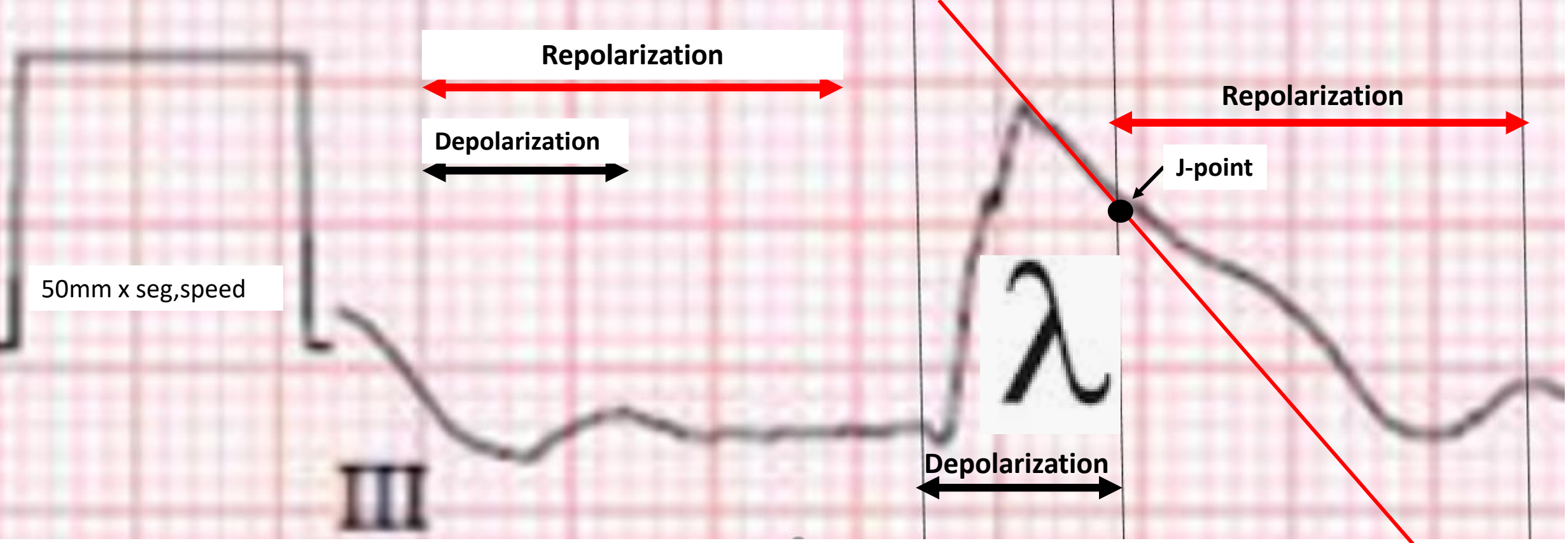


60°

“ Absolutamente verdaderamente opuestas“
Absolutely anatomically opposite

+120°
III





The lambda-like ST-elevation electrocardiography (ECG) pattern is an uncommon pattern in patients with type 2 myocardial infarction (T2MI) triggered by coronary spasm. When this ECG pattern appears, sudden cardiac death (SCD) caused by lethal ventricular arrhythmia may occur because clinicians do not pay sufficient attention to this phenomenon.

La angina de Prinzmetal angina variante, o inversa diferencia de la angina típica, que a menudo se origina por el esfuerzo o el estrés emocional, la angina de Prinzmetal casi siempre aparece cuando la persona está descansando, normalmente entre la medianoche y las primeras horas de la mañana. Estos ataques pueden ser muy dolorosos. Es poco frecuente, (≈ 2 cada100 casos de angina), y normalmente se da en pacientes más jóvenes que los que suelen padecer otros tipos de angina. El dolor de la angina variante se debe a un espasmo en las arterias coronarias y puede o no tener obstruccion coronaria Las arterias coronarias pueden sufrir espasmos debido a lo siguiente:Exposición al frío, estrés, medicamentos que contraen los vasos sanguíneos, tabaquismo, consumo de cocaína. Síntomas de la angina variante (de Prinzmetal): El dolor o la molestia: normalmente aparece en reposo y durante la noche o en las primeras horas de la mañana. Suele ser intensa y se puede aliviar con vasodilatadores y antagonsitas de calcio.

Prinzmetal's or Prinzmetal Angina, Variant angina and angina Inversa. Unlike typical angina – which is often triggered by exertion or emotional stress – Prinzmetal’s angina almost always occurs at rest, usually between midnight and early morning. These attacks can be very painful. Prinzmetal’s angina is rare, representing about two out of 100 cases of angina, and usually occurs in younger patients than those who have other kinds of angina. Causes: The pain from variant angina is caused by a spasm in the coronary arteries.The coronary arteries can spasm as a result of exposure to cold weather, stress, medicines that tighten or narrow blood vessels, smoking, cocaine use

Symptoms : The pain or discomfort: Usually occurs while resting and during the night or early morning hoursAre usually severeCan be relieved bytaking medication Treatment of Variant Angina (Prinzmetal) Angina Medicines can help control the spasms. Drugs such as calcium antagonists and nitrates are the mainstays of treatment. The spasms tend to come in cycles – appearing for a time, then going away. After six to 12 months of treatment,may gradually reduce the medication. It is a chronic condition that will need to be followed by your healthcare provider even though the prognosis is generally good.

To understand electrocardiography, it is essential, as Prof. Antoni Bayes always remembers, to keep in mind the arrangement of the hemifields. Apart from this, and according to Kjell Nikus, in my experience I agree with the conclusion that "... the reciprocal image is simply an electrical phenomenon, rather than ischemia at a distance from impaired collateral circulation." For example, when there is a chronic occlusion of the RCD with collateral circulation from the LAD and the LAD is occluded, a direct image of elevated ST appears in the territory of the RCA but not a descended ST. When there is generalized multivessel disease with arteries not totally occluded, things get a bit complicated. In our recent book *, we remember it this way:"....

The ECG patterns of ischemia and necrosis are of greatest importance in the diagnosis and prognosis of IHD. They are recorded in different leads as direct patterns, according to the affected zones. On the other hand, they may also be recorded in opposite leads as 'mirror patterns' of “reciprocal changes”: a positive T wave instead of a negative T wave, ST-segment depression instead of ST-segment elevation and a tall R wave instead of a Q wave. From the clinical point of view, these mirror patterns should be considered as an evidence of an area of ischemia or necrosis that generates the mirror image pattern in some part of the heart distant from the exploring electrode. Understanding the significance of the presence of direct and mirror patterns is crucial to correctly interpret the ECG patterns “

*** Electrocardiography in Ischemic Heart Disease. Second edition, 2020. Wiley Blackwell. Miguel Fiol-Sala, Yochai Birbaum, Kjell Nikus, Antoni Bayes de Luna.**

Best Regards

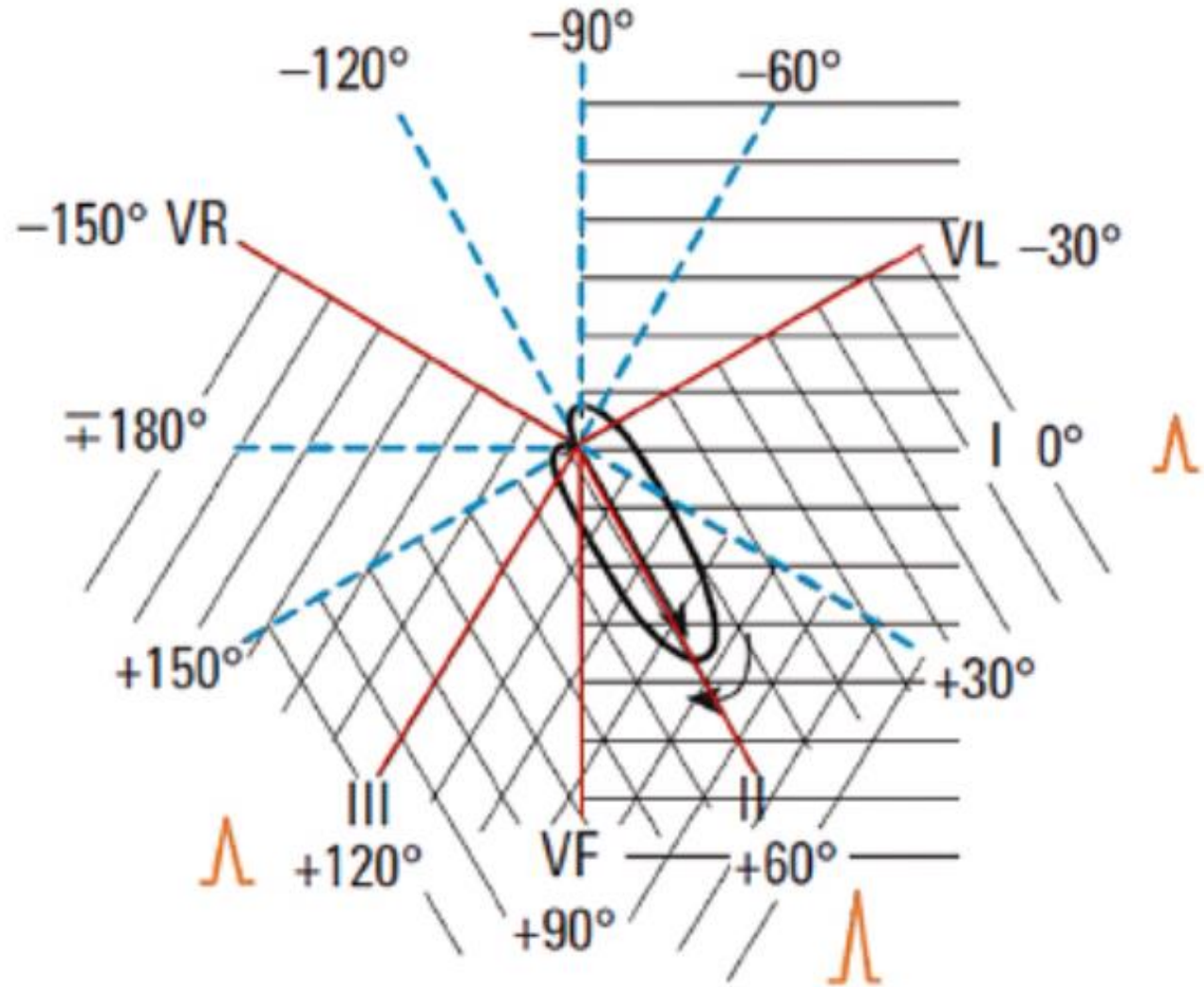
Miquel Fiol Sala director científico del Instituto de Investigación Sanitaria de Mallorca

Para comprender la electrocardiografía, es esencial, como siempre recuerda el profesor Antoni Bayés de Luna, tener en cuenta la disposición de los hemi-campos “hemifields”(Una de las dos mitades de un campo sensorial (a partir de la visión). Las partes de cada hemifield visual que se pueden ver con ambos ojos.) Figura próximo slide) Además, y según Kjell Nikus"... la imagen recíproca **es simplemente un fenómeno eléctrico, en lugar de isquemia a una distancia de la circulación colateral deteriorada".** Por ejemplo, en presencia de una oclusión crónica de la arteria coronaria derecha (**RCA**) con circulación colateral suministrada desde la arteria descendente anterior izquierda (LAD) y cuando esta se encuentra ocluida, aparece una imagen directa de elevación del segmento ST en el territorio de la RCA pero no una depresión del segmento ST.

En el caso de presencia de una enfermedad de múltiples vasos “multiple vessel disease” con arterias parcialmente ocluidas, las cosas se complican un poco.

Los patrones de ECG de isquemia, lesión y necrosis son de gran importancia en el diagnóstico y pronóstico de la insuficiencia coronaria. Se registran patrones directos, en las zonas afectadas manifestadas por ondas **T negativas(isquemia) , segmentos ST elevados(lesión) y ondas Q de necrosis**. Por otro lado, pueden registrarse en las derivaciones opuestas ('patrones espejo’ o "cambios recíprocos“): caracterizados por una onda T positiva en lugar de una onda T negativa, depresión del segmento ST en lugar de elevación del segmento ST y una onda R alta en lugar de una onda Q. Desde el punto de vista clínico, estos patrones de espejo deben considerarse como evidencia de un área de isquemia o necrosis que genera el patrón de imagen de espejo en alguna parte del corazón distante del electrodo explorador. Comprender la importancia de la presencia de patrones directos y recíprocos o en espejo es crucial para interpretar correctamente los patrones de ECG "

* Electrocardiografía en cardiopatía isquémica. Segunda edición, 2020. Wiley Blackwell. Miguel Fiol-Sala, Yochai Birbaum, Kjell Nikus, Antoni Bayés de Luna.



Positive hemifield of III

Positive hemifield of II

Querido amigo profesor Edgardo Schapachnik quisiera discutir el fenómeno de los cambios recíprocos en los síndromes isquémicos y los avanzos en el tema, según la contribución de las ciencias básica los últimos 20 años al tema. El segmento ST infradesnivelado sugiere siempre un aumento de la presión diastólica final, ya sea en las isquemias agudas o crónicas , como en las sobrecargas sistólicas o en mujeres menopaúsicas. Este aumento de la presión diastólica ,sobre el endocardio ,desencadena una "**electrical tension feedback".** Sin presión sobre el endocardio no existe ST deprimido como lo demostró el legendario Louis Nelson Katz (1897-1973) em 1944 Los ST elevados epicárdiocos puros no producen ST deprimidos, como en la repolarización precoz de varones jóvenes, pericarditis agudas o en el Tako Tsubo agudo (primera etapa) En isquemias agudas segmentarias, la lesión miocárdica ejerce en el área opuesta, una presión aumentada(según la ley de Starling) la cual estimula el endocardio opuesto, que produce un reacción eléctrica manifestada en el ECG por un ST deprimido, Esta es una ley biológica: Siempre que el músculo opuesto no esta afectado por alguna lesión previa crónica o aguda en el musculo opuesto se produce una hiperquinesia .con el objetivo de mantener una fracción de eyección eficiente. Como se hace esto? Por un aumento de adrenalina circulante .Pero la adrenalina aumentada es peligrosa para una isquemia aguda. No obstante la madre natura con la evolución de 150 millones de años, descubrió que la forma de eliminar este peligro. De esta forma, la epinefrina estimula los efectores adrenérgicos alfa 2 , que aumenta la contracción muscular sin producir taquicardia

El sistema simpático segrega un neuro polipéptido e cual junto con la molécula arrestina ,bloquean los receptores beta nocivos para el músculo cardiaco. Y los receptores alfa ocasionan la hiperquinesia y las hipertrofia fisiológicas del área opuesta a la lesión, con el fin de mantener la efectividad del ventrículo. Nosotros hemos escrito un trabajo donde discutimos el ST deprimido fisiológico en el que distinguíamos del ST deprimido patológico Me parece que es el anteúltimo trabajo de la lista que mande sobre el ST

Un fraternal abrazo y será interesante una discusión en el fórum sobre este tema

Samuel Sclarovsky

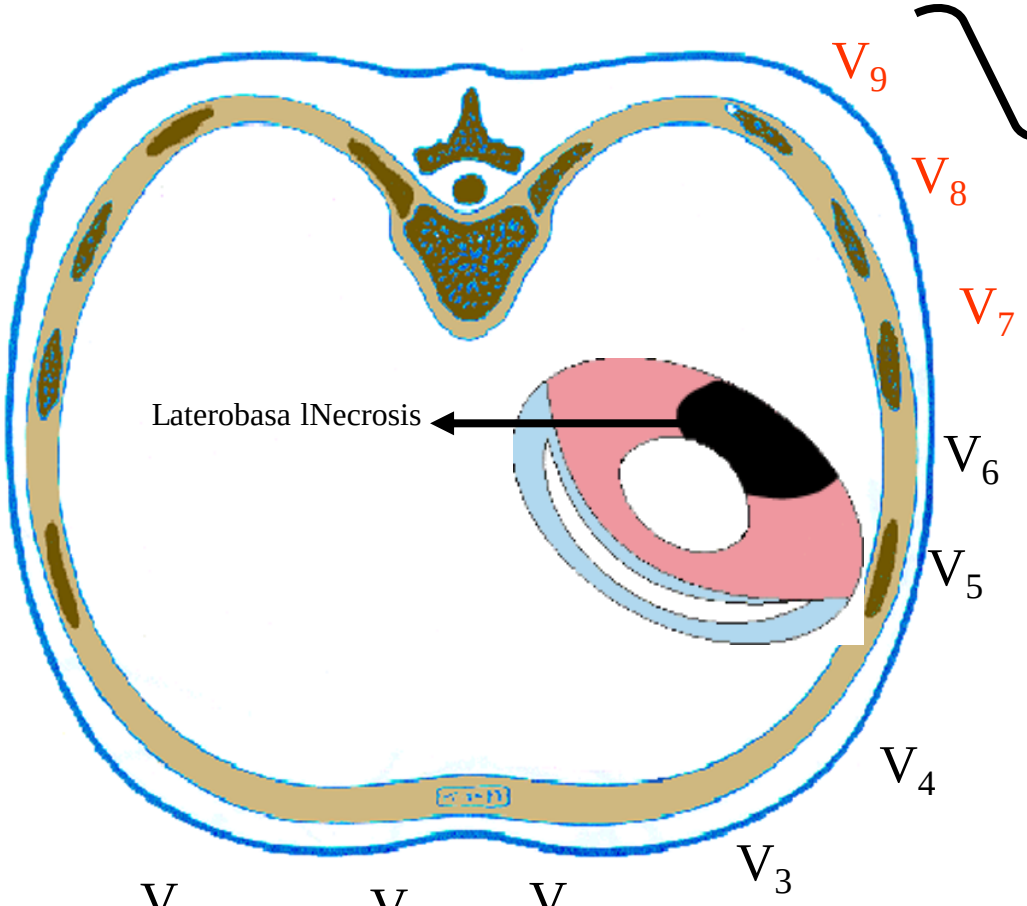
Dear friends, I would like to discuss the phenomenon of reciprocal changes in ischemic syndromes and advances in the subject, according to the contribution of the basic sciences in the last 20 years to the subject. The under-leveled ST segment always suggests an increase in the final diastolic pressure, either in acute or chronic ischemia, as in systolic overloads or in menopausal women. This increase in diastolic pressure on the endocardium triggers an "electrical tension feedback". Without pressure on the endocardium, there is no depressed ST as demonstrated by the legendary Louis Nelson Katz (1897-1973) in 1944. Pure elevated epicardial STs do not produce depressed ST, as in the early repolarization of young men, acute pericarditis or in the acute phase of Tako Tsubo (first stage) In acute segmental ischemia, the myocardial injury exerts in the opposite area, an increased pressure (according to Starling's law) which stimulates the opposite endocardium, which produces an electrical reaction manifested on the ECG by a depressed ST, This is a biological law: Whenever the opposite muscle is not affected by any previous chronic or acute injury to the opposite muscle, hyperkinesia occurs, with the aim of maintaining an efficient LV ejection fraction. How do you do this? Due to an increase in circulating epinephrine. But increased epinephrine is dangerous for acute ischemia. However mother nature with the evolution of 150 million years, discovered that the way to eliminate this danger. In this way, epinephrine stimulates alpha 2 adrenergic effectors, which increases muscle contraction without producing tachycardia. The sympathetic system secretes a neuro polypeptide which, together with the arrestin molecule, blocks beta receptors that are harmful to the heart muscle. And alpha receptors cause hyperkinesis and physiological hypertrophy of the area opposite the injury, in order to maintain the effectiveness of the ventricle. We have written a paper where we discussed physiological depressed TS in which we distinguished from pathological depressed ST. It seems to me that it is the last last work on the list that I sent about ST.

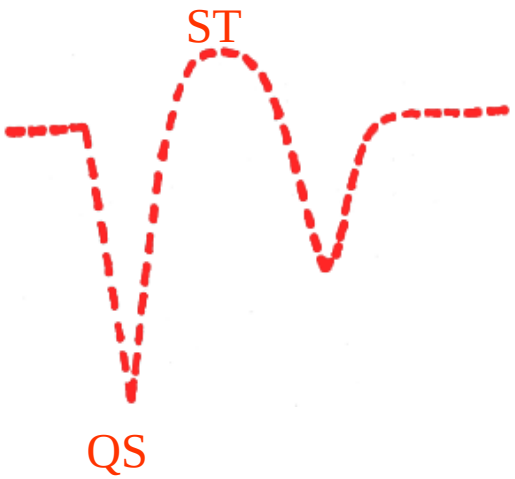
A fraternal hug and it will be interesting a discussion in the forum on this topic

Samuel Sclarovsky

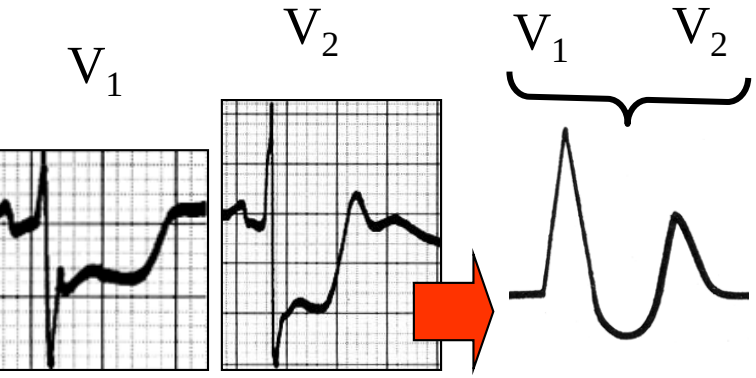
I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

SITE	FACING	RECIPROCAL
SEPTAL	V1, V2	NONE
ANTERIOR	V3, V4	NONE
ANTEROSEPTAL	V1, V2, V3, V4	NONE
LATERAL	I, aVL, V5, V6	II, III, aVF V1, V2
ANTEROLATERAL	I, aVL, V3, V4, V5, V6	II, III, aVF
INFERIOR	II. III. aVF	I. aVL





A lateral (V5, V6, aVL) ST-SEMI is likely to cause a reciprocal inferior ST-depression (II, III, aVF and in anteroseptal leads V1-V2.) V_1 $V_{2\ l}$ are opposite the lateral wall. T

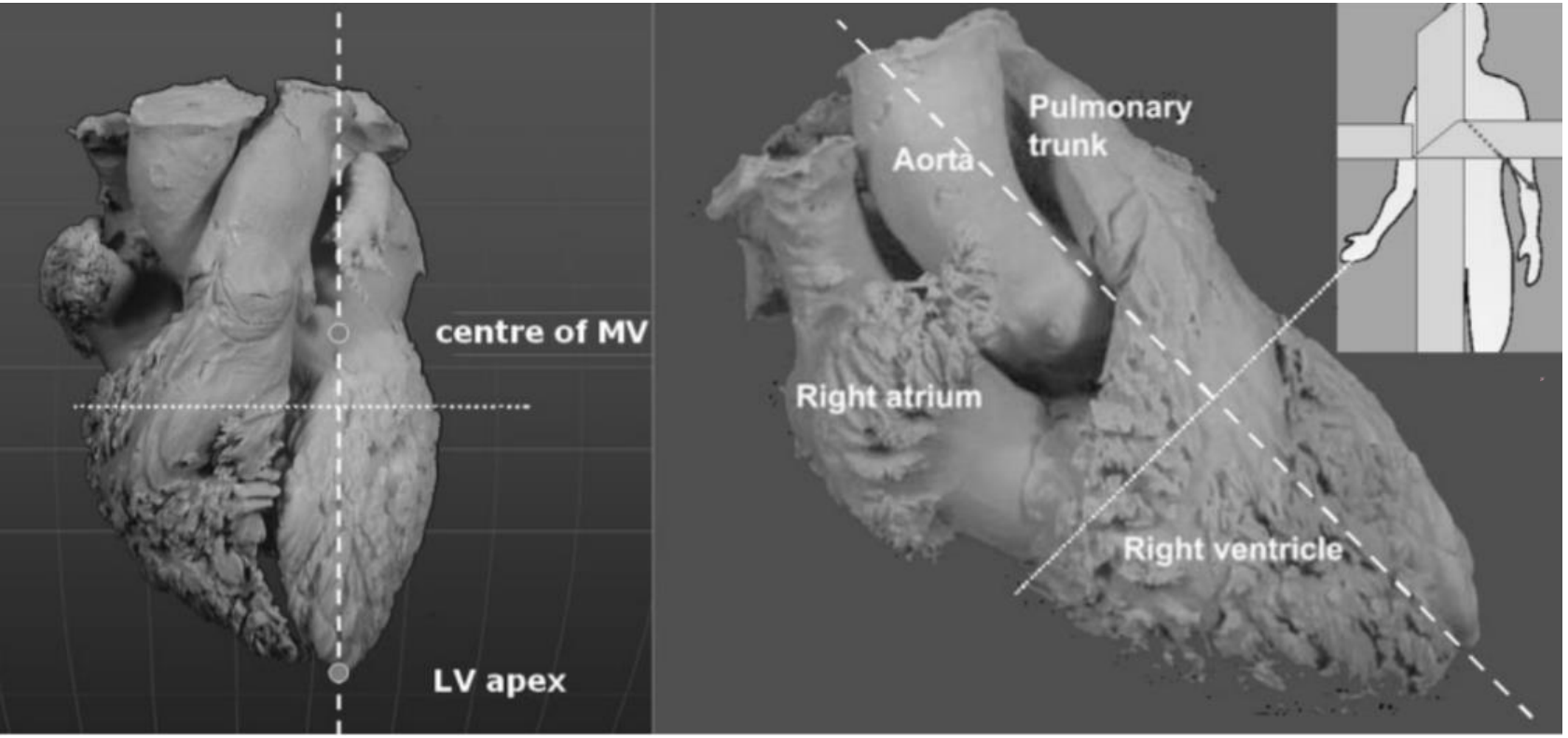


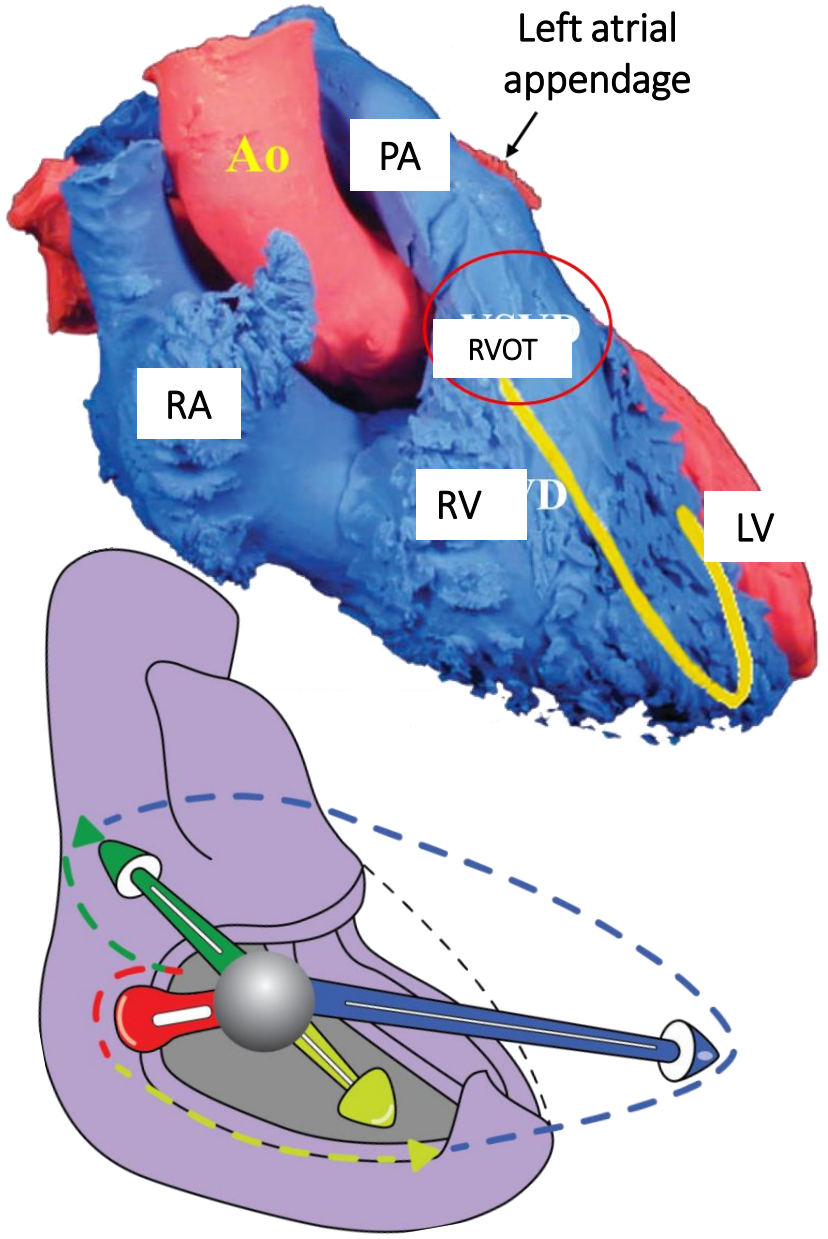
The presence of prominent the R wave in V1 is due to the lateral MI and not to the involvement of inferobasal segment of inferior wall (old posterior wall: posterior wall does not exist!!!!). Lateral MI explains the presence of prominent R wave (R ≥ S) in V1(**Diego Goldwasser et al.Ann Noninvasive Electrocardiol. 2015 Nov;20(6):570-7. doi: 10.1111/anec.12260.**

Expert consensus recommendations

The terms *true* and *strictly posterior* MI have been applied when the basal part of the LV wall that lies on the diaphragm was involved. However, although in echocardiography the term *posterior* is still used in reference to other segments of the LV, it is the consensus of this report to recommend that the term *posterior* be abandoned and that the term *inferior* be applied to the entire LV wall that lies on the diaphragm.(**Antoni Bayés de Luna 1, Galen Wagner, Yochai Birnbaum, Kjell Nikus, Miguel Fiol, Anton Gorgels, Juan Cinca, Peter M Clemmensen, Olle Pahlm, Samuel Sclarovsky, Shlomo Stern, Hein Wellens, Wojciech Zareba, International Society for Holter and Noninvasive A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography. A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography Circulation. 2006 Oct 17;114(16):1755-60. doi:10.1161/CIRCULATIONAHA.106.624924.**)

Current nomenclature of heart wall segmentation with Contrast-Enhanced Cardiovascular Magnetic Resonance

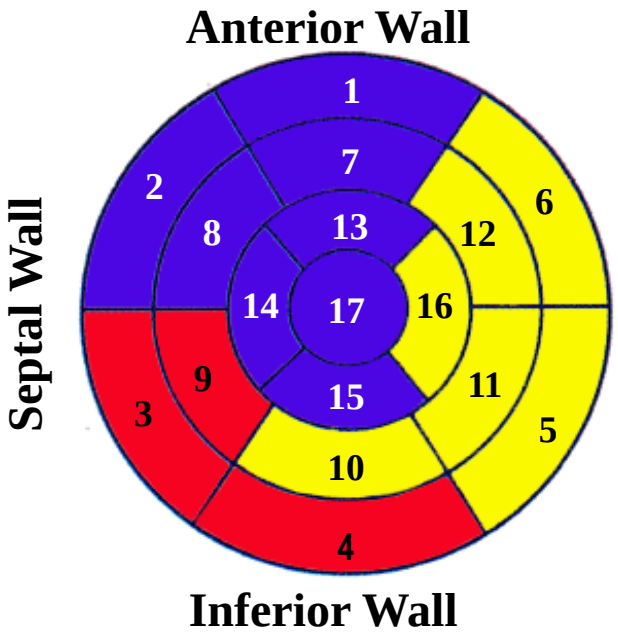


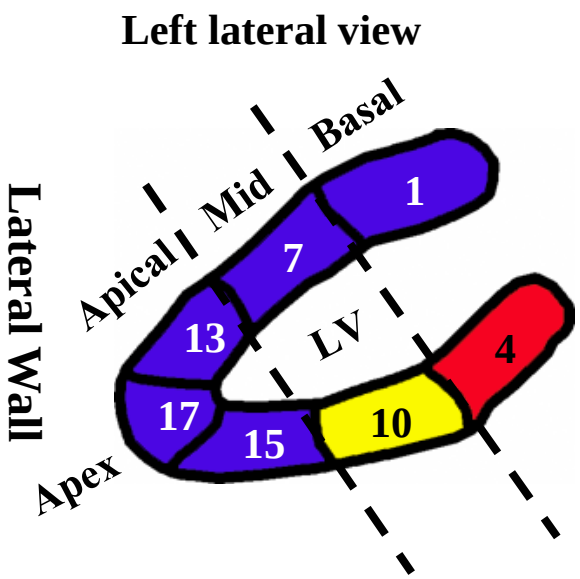


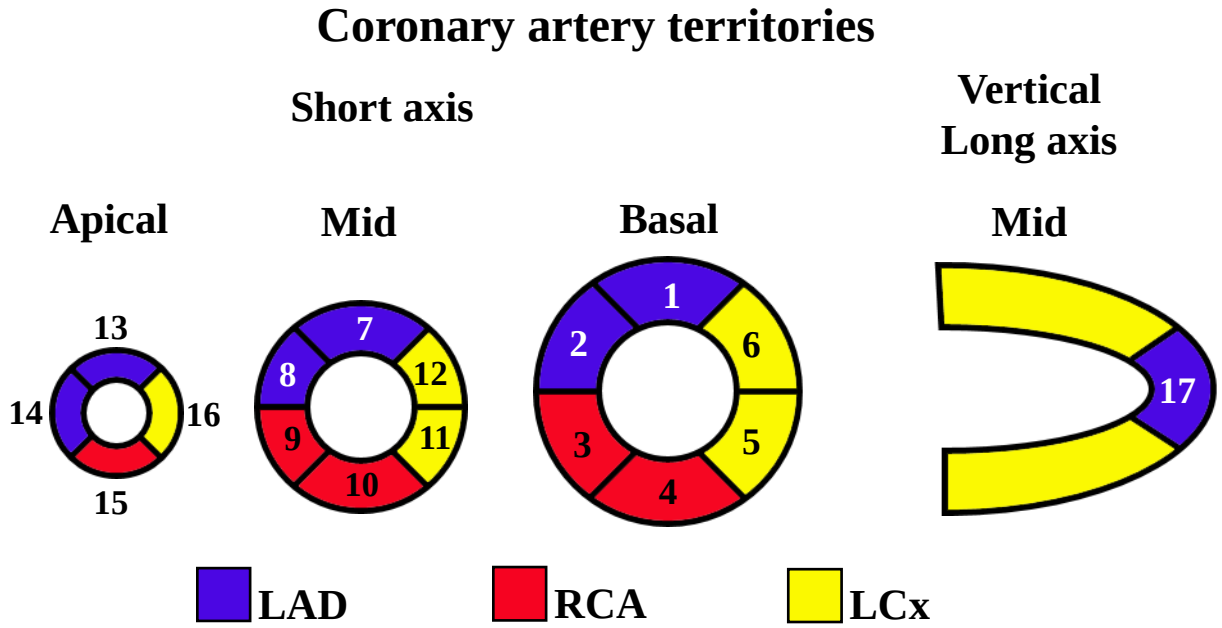
The left panel shows the heart in its ‘‘Valentine’’ position, with the long axis of the left ventricle and its defining points (dashed line) and a short axis (dotted line). In the right panel, we have positioned the heart in attitudinally appropriate fashion, showing the angulation of the ventricular axes relative to the axes of the body.

Ventricular segmentation heart walls with Contrast-Enhanced Cardiovascular Magnetic Resonance (CE-CMR)

Polar map short axis in “bull’s-eye”



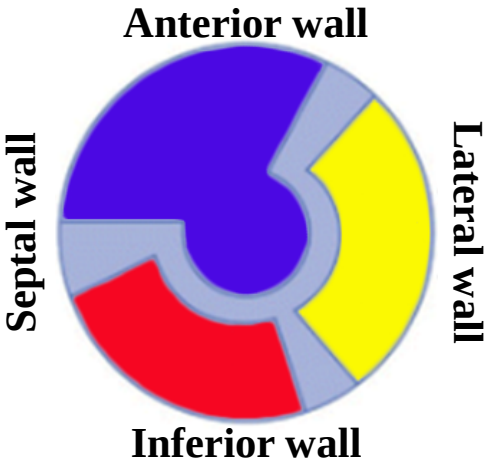




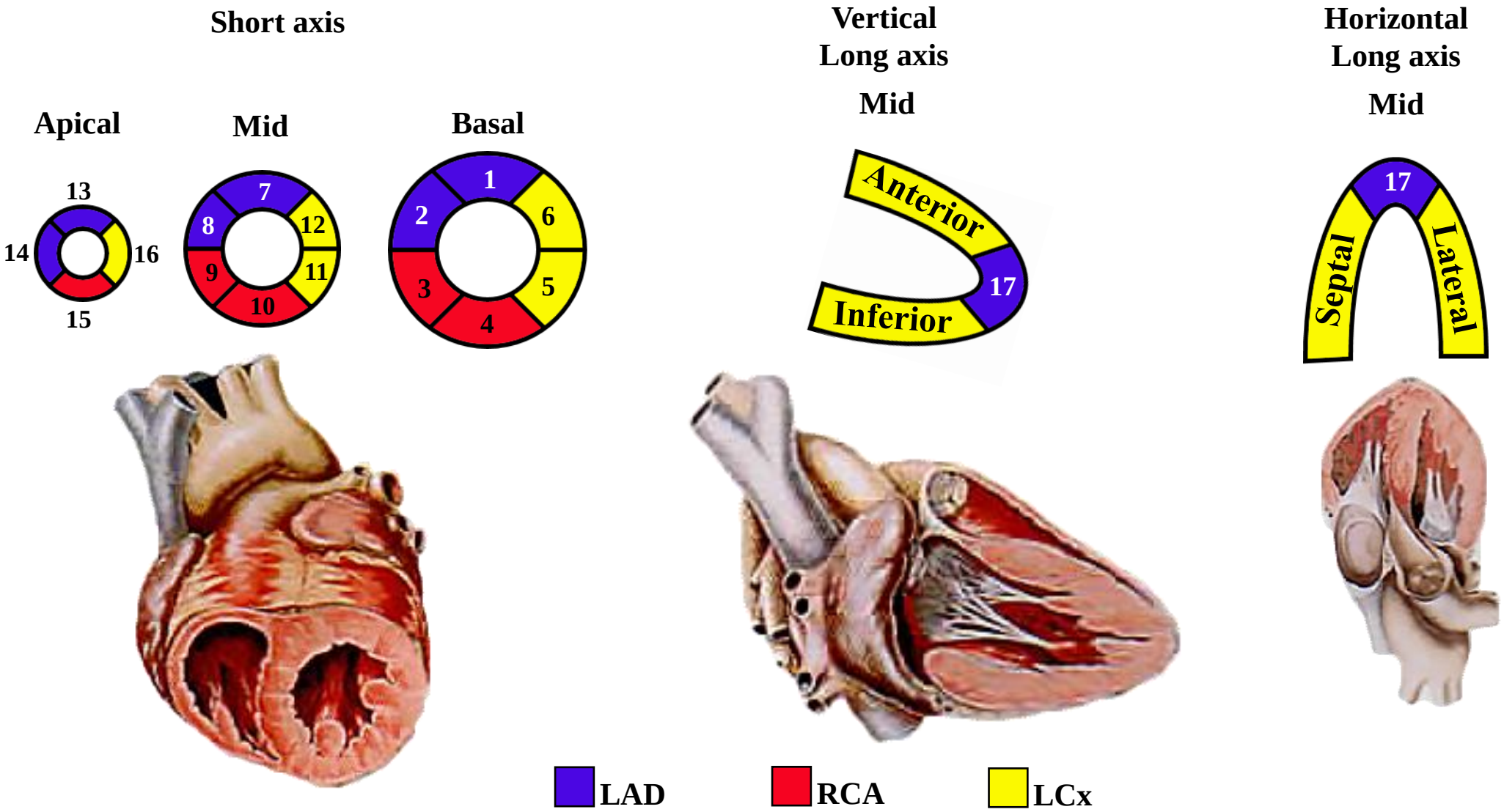
17 myocardial segments and the recommended nomenclature for tomographic imaging of the heart. Data from the individual short-axis tomograms can be combined to create a polar map plot, representing a 2D compilation of all the 3D short-axis perfusion data. Standard nomenclature for the 17 segments is outlined.

1. Basal anterior
2. Basal anteroseptal
3. Basal inferoseptal
4. Basal inferior
5. Basal inforolateral
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

The 17 myocardial segments to the territories of the LAD, RCA, and LCx. The 2D compilation of perfusion data can then easily be assigned to specific vascular territories.



Coronary artery territories

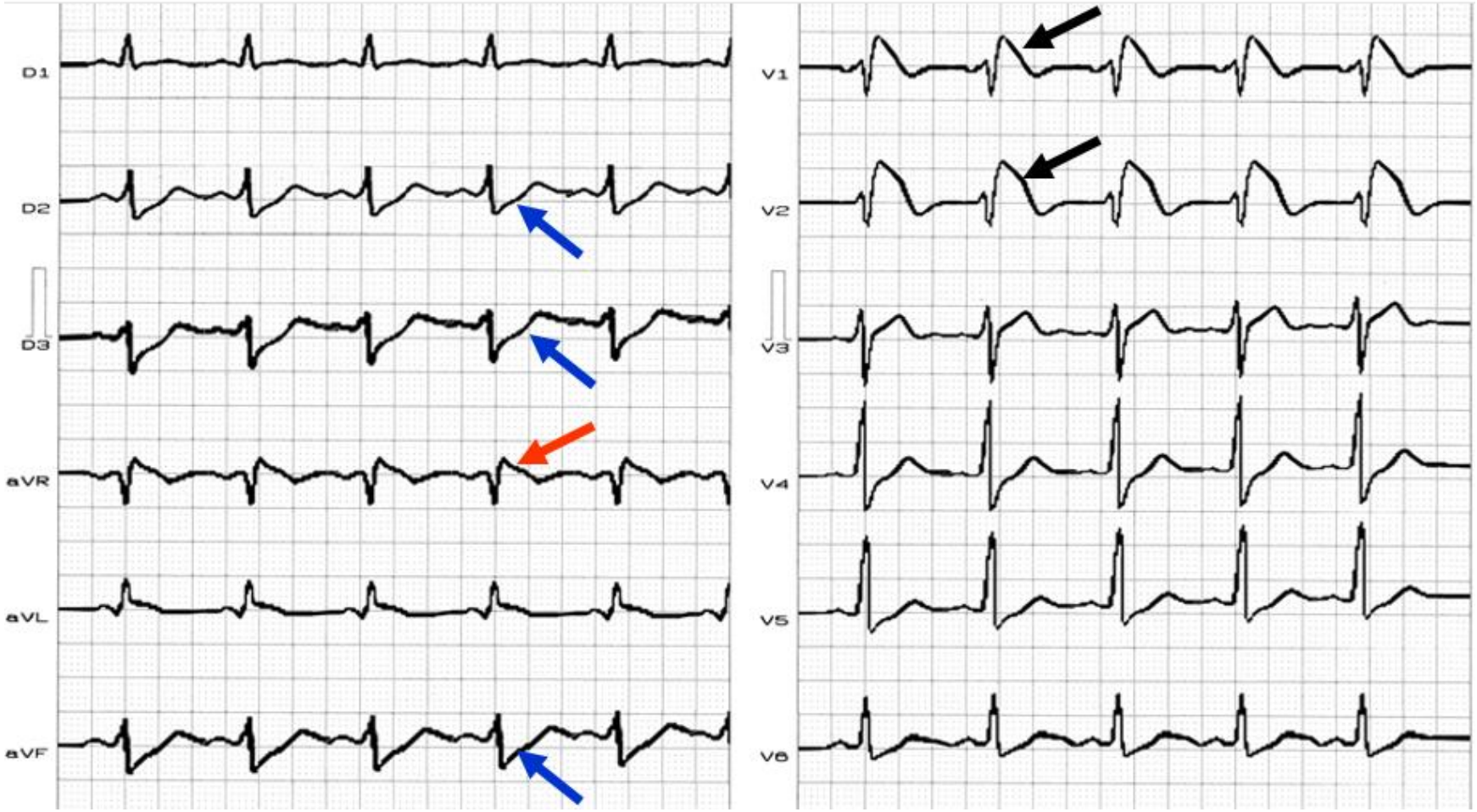


Cerqueira MD, Weissman NJ, Dilsizian V, Jacobs AK, Kaul S, Laskey WK, Pennell DJ, Rumberger JA, Ryan T, Verani MS; American Heart Association Writing Group on Myocardial Segmentation and Registration for Cardiac Imaging. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. Circulation. 2002 Jan 29;105(4):539-42.

The presence of reciprocal change supports the diagnosis of STEMI and also is a sign of a high-risk patient. Reciprocal ST-segment depression, also known as reciprocal change, is defined as ST-segment depression in leads separate and distinct from leads that reflect ST-SE**;** in other words, the concept of reciprocal change cannot be used if ST-SE is not present. The concept of reciprocal change cannot be used in patients with abnormal intraventricular conduction, including LBBB, ventricular paced rhythm and, to a lesser extent, LVH via voltage criteria. The morphology of the depressed ST segment is either horizontal or downsloping; it need only be present in a single lead, but frequently is present in several leads. Reciprocal change is seen in approximately 75% of patients with inferior wall AMIs; in 30% of patients with anterior wall MI, and frequently in lateral STEMI. Reciprocal change is an important ECG concept to consider for two reasons. First, it identifies patients with a high-risk ACS presentation. Reciprocal change in the setting of STEMI identifies a patient with an increased likelihood of cardiovascular complication (heart block, malignant ventricular dysrhythmia, cardiogenic shock) and poor outcome (significant LV dysfunction, death). Second, the presence of reciprocal change is strong confirmatory evidence that STEMI is present and has both very high specificity and a positive predictive value greater than 90%. (**Brady WJ, Perron AD, Martin ML, et al. Electrocardiographic ST segment elevation in emergency department chest pain center patients: etiology responsible for the ST segment abnormality.Am J Emerg Med. 2001;19:25-28**.)(**Otto LA, Aufderheide TP. Evaluation of ST segment elevation criteria for the prehospital electrocardiographic diagnosis of acute myocardial infarction. Ann Emerg Med. 1994; 23: 17-24.1994;23:17-24.)** With ECG presentations that are straightforward, reciprocal changes does not assist with ECG diagnosis; in more subtle presentations, can aid in making the ECG diagnosis. ECG leads are electrical “sensors”. Imagine 2 sensors facing one another and measuring their distance from an object in between them. If the object moves toward lead1, you expect the distance “lead1-object” to decreased but in a RECIPROCAL manner you expect the distance “lead2-object” to increase. Basically a reciprocal change is an expected/logical change to occur and if it doesn’t then it means your initial setup is not as expected. On an ECG, we have multiple sensors, creating a 3D-plan to represent the

heart electrical activity. For every change, you'd expect a reciprocal counterchange in certain leads. Finally, the ECG reciprocal changes are not pathognomonic for STEMI because we identify them in some cases of Brugada syndrome with type 1 electrocardiographic pattern.

Typical spontaneous type 1 ECG Brugada pattern in a patient with Brugada syndrome



J point and ST segment elevation, convex to the top, in the right precordial leads from V_1 through V_2 (black arrows): Brugada sign or type 1 Brugada pattern . Unipolar aVR lead that heads toward the RV epicardium above the RVOT, which shows subtle ST segment and J point elevation (red arrows). Inferior leads show reciprocal or mirror images (blue arrows).