

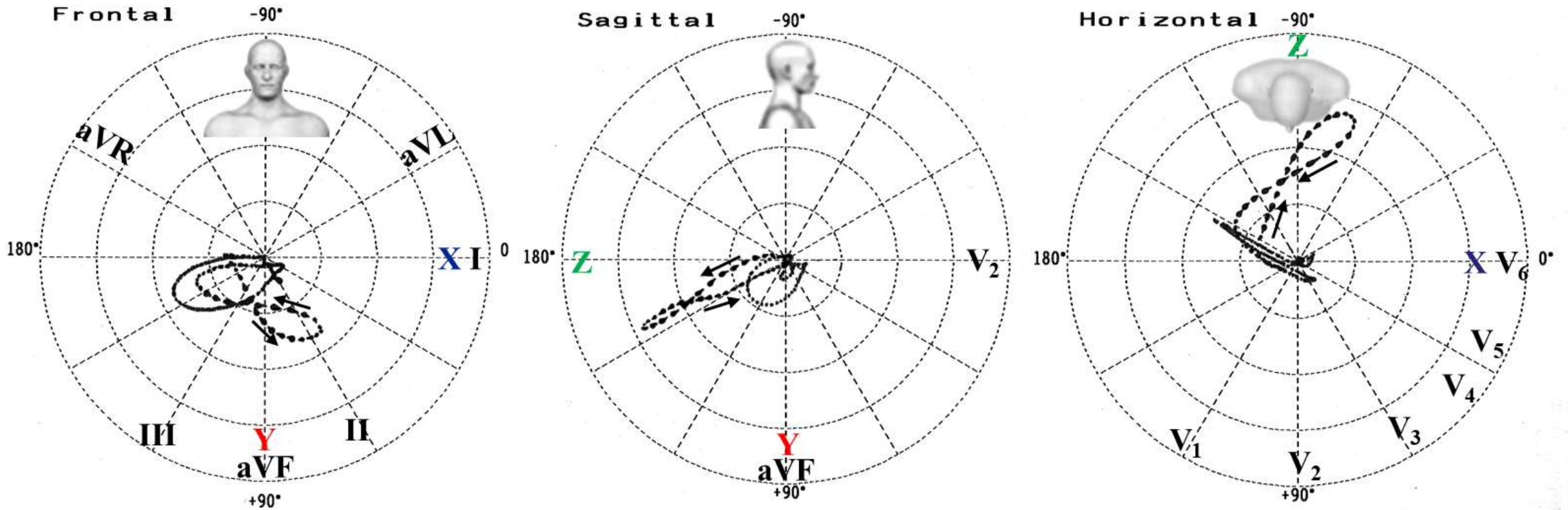
Myocardial infarction picture in an elderly man: VCG exercise



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Portuguese: Trata-se de um homem de 62 anos. Deu entrada em nosso hospital com dor opressiva precordial prolongada retroesternal. Como fator de risco apenas fumante inveterado de longa data. Este VCG foi realizado 15 dias após a internação. Pergunta: Qual o diagnóstico vetorcardiográfico?

English: It is a 62 years old male. He entered our hospital with retrosternal prolonged precordial oppressive pain. As a risk factor only long-time inveterate smoker. This VCG was performed 15 days after admission. Question: What is the vectorcardiographic diagnosis?

Colleagues opinions

Hi all:

In the QRS loop we can see missing anteriorly and rightward oriented electrical forces – my conclusion: an extensive anterolateral myocardial infarction.

Best regards,

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Goal: Left ventricular hypertrophy

Main articles contributions:

1. **Bacharova L**, Estes HE, Schocken DD, Ugander M, Soliman EZ, Hill JA, Bang LE, Schlegel TT. The 4th Report of the Working Group on ECG diagnosis of Left Ventricular Hypertrophy. *J Electrocardiol.* 2017 Jan - Feb;50(1):11-15.
2. Maanja M^{1,2}, Wieslander B¹, Schlegel TT^{1,3}, **Bacharova L^{4,5}**, Abu Daya H², Fridman Y², Wong TC², Schelbert EB², Ugander M⁶. Diffuse Myocardial Fibrosis Reduces Electrocardiographic Voltage Measures of Left Ventricular Hypertrophy Independent of Left Ventricular Mass. *J Am Heart Assoc.* 2017 Jan 22;6(1).
3. **Bacharova L**, Triantafyllou E, Vazaios C, Tomeckova I, Paranicova I, Tkacova R. The effect of obstructive sleep apnea on QRS complex morphology. *J Electrocardiol.* 2015 Mar-Apr;48(2):164-70.



4. **Bacharova L**, Ugander M. Left ventricular hypertrophy: The relationship between the electrocardiogram and cardiovascular magnetic resonance imaging. *Ann Noninvasive Electrocardiol.* 2014 Nov;19(6):524-33.
5. **Bacharova L**, Kudaiberdieva G2, Misak A2, Hakacova N2, Timuralp B2, Wagner GS2. The effect of International Scientific Summer School research training on scientific productivity of trainees. *Int J Cardiol.* 2014 Oct 20;176(3):1142-6.
6. **Bacharova L**, Schocken D, Estes EH, Strauss D1. The role of ECG in the diagnosis of left ventricular hypertrophy. *Curr Cardiol Rev.* 2014 Aug;10(3):257-61.
7. **Bacharova L**, Estes EH Jr, Hill JA, Pahlm O, Schillaci G, Strauss D, Wagner G. Changing role of ECG in the evaluation left ventricular hypertrophy. *J Electrocardiol.* 2012 Nov-Dec;45(6):609-11.
8. **Bacharova L**, Mateasik A, Krause R, Prinzen FW, Auricchio A, Potse M. The effect of reduced intercellular coupling on electrocardiographic signs of left ventricular hypertrophy. *J Electrocardiol.* 2011 Sep-Oct;44(5):571-6
9. **Bacharova L**, Szathmary V, Mateasik A. Secondary and primary repolarization changes in left ventricular hypertrophy: a model study. *J Electrocardiol.* 2010 Nov-Dec;43(6):624-33.
10. **Bacharova L**, Szathmary V, Kovalcik M, Mateasik A. Effect of changes in left ventricular anatomy and conduction velocity on the QRS voltage and morphology in left ventricular hypertrophy: a model study. *J Electrocardiol.* 2010 May-Jun;43(3):200-8.
11. **Bacharova L**; American Heart Association. What is recommended and what remains open in the American Heart Association recommendations for the standardization and interpretation of the electrocardiogram. Part V: electrocardiogram changes associated with cardiac chamber hypertrophy. *J Electrocardiol.* 2009 Sep-Oct;42(5):388-91.
12. **Bacharova L**. The structural and electrical remodeling of myocardium in LVH and its impact on the QRS voltage. *Anadolu Kardiyol Derg.* 2007 Jul;7 Suppl 1:37-42.
13. **Bacharova L**, Michalak K, Kyselovic J, Klimas J. Relation between QRS amplitude and left ventricular mass in the initial stage of exercise-induced left ventricular hypertrophy in rats. *Clin Exp Hypertens.* 2005 Aug;27(6):533-41.
14. **Bachárová L**, Kyselovic J. Role of orthogonal electrocardiography and vectorcardiography in a paradigm shift in diagnosis of left ventricular hypertrophy. *Cesk Fysiol.* 2001 Aug;50(3):134-40.
15. **Bachárová L**. Computer processing of the orthogonal electrocardiogram and vectorcardiogram. *Physiol Res.* 1993;42(2):95-8.
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Spanish

Buenas noches estimados Maestros. Creo que el vectocardiograma muestra necrosis anterior extensa transmural, evolución a 15 días. probable lesión de DA.

Espero la opinión de expertos, los saludo muy cordialmente.

Feliz Pascua!

Dr. Juan Carlos Manzardo

Mendoza Argentina

Good evening Dear Masters. I think the vectocardiogram shows extensive anterior transmural necrosis and T (-) w days. probable LAD obstruction?

I hope the opinion of experts, I greet you very cordially.

Happy easter!

Juan Carlos Manzardo MD Mendoza Argentina

Comments

Dear Juan Carlos: Myocardial infarction(MI) can be subcategorized on the basis of anatomic, morphologic, and diagnostic clinical information. From an anatomic or morphologic standpoint, the two types of MI are transmural and nontransmural. A transmural MI is characterized by ischemic necrosis of the full thickness of the affected muscle segment(s), extending from the endocardium through the myocardium to the epicardium. A nontransmural MI is defined as an area of ischemic necrosis that does not extend through the full thickness of myocardial wall segment(s). In a nontransmural MI, the area of ischemic necrosis is limited to the endocardium or to the endocardium and myocardium. It is the endocardial and subendocardial zones of the myocardial wall segment that are the least perfused regions of the heart and the most vulnerable to conditions of ischemia. An older subclassification of MI, based on clinical diagnostic criteria, is determined by the presence or absence of Q waves on an ECG. However, the presence or absence of Q waves does not distinguish a transmural from a nontransmural MI as determined by pathology(**Rubin 1995**).-A consensus statement was published to give a universal definition of the term MI. The authors stated that MI should be used when there is evidence of myocardial necrosis in a clinical setting consistent with MI. Myocardial infarction was then classified by the clinical scenario into various subtypes:

Type 1 is a spontaneous MI related to ischemia from a primary coronary event (e.g., plaque rupture, thrombotic occlusion);

Type 2 is secondary to ischemia from a supply-and-demand mismatch;



Type 3 is an MI resulting in SCD;

Type 4a is an MI associated with percutaneous coronary intervention;

Type 4b is associated with in-stent thrombosis;

Type 5 is an MI associated with coronary artery bypass surgery (**Thygesen 2007**).

A more common clinical diagnostic classification scheme is also based on ECG findings as a means of distinguishing between two types of MI, one that is marked by ST elevation (STEMI) and one that is not (NSTEMI). Management practice guidelines often distinguish between STEMI and non-STEMI, as do many of the studies on which recommendations are based. The distinction between STEMI and NSTEMI also does not distinguish a transmural from a nontransmural MI (**Phibbs 1983**). The presence of Q waves or ST-segment elevation is associated with higher early mortality and morbidity; however, the absence of these two findings does not confer better long-term mortality and morbidity(**Anderson 2007;2007;2011**).

- I) Abundant experimental and clinical-pathologic evidence now exists to establish permanently and irrefutably the fact that presence or absence of Q waves in the surface ECG does not permit distinction between transmural and subendocardial infarcts. In terms of both sensitivity and accuracy, the Q wave is a useless observation in this setting. (This, of course, must be distinguished from the unquestioned usefulness of the Q wave in diagnosing infarction as such.) Presence or absence of Q waves accompanying MI does not delineate or differentiate any pathologic or clinical subset.
- II) The use of the term "Q wave infarct" or "non-Q wave infarct," which has emerged as a kind of half-way house of the intellect, should be avoided, since it implies a distinction between two varieties of infarction-a distinction that has no basis in pathologic fact.
- III) Editors, research committees and formulators of ECG reading standards should use every effort to drop this misleading and potentially dangerous distinction, deeply embedded in cardiology, from the lexicon of electrocardiographic terminology (**Phibbs 1983**).

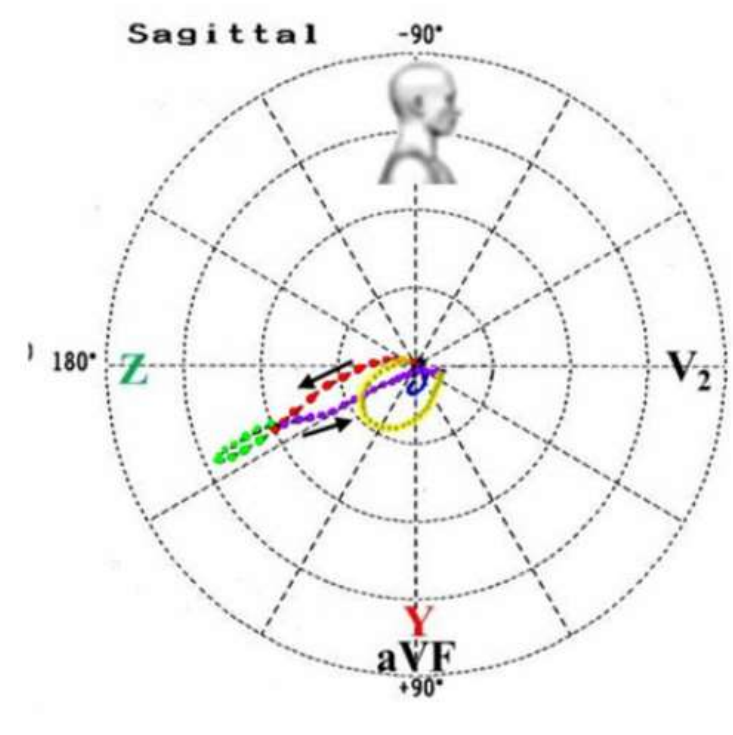
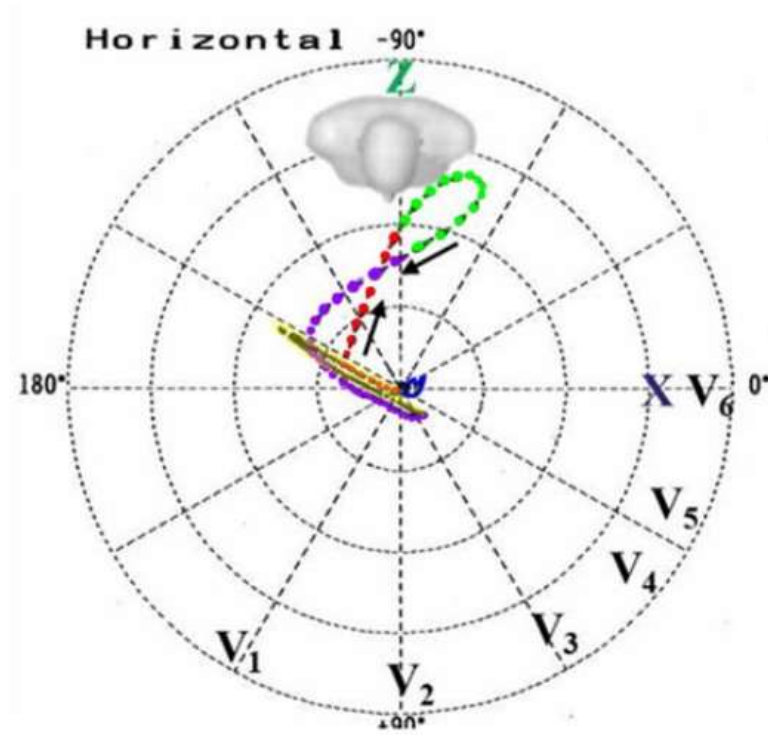
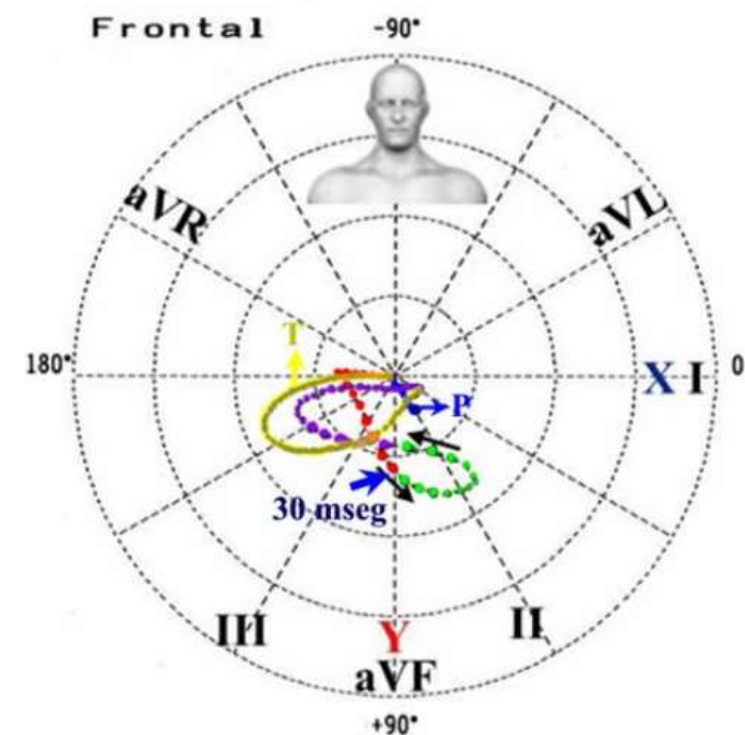
Pipberger and Lopez (**Pipberger 1980**) published a review entitled " 'Silent' Subendocardial Infarcts: Fact or Fiction." They noted that the attempt to distinguish transmural from subendocardial infarcts on the basis of presence or absence of Q waves was not justified by any clinical, pathologic or experimental data. The authors observed that this common misconception arose from a single flawed study, later repudiated by the original investigator and by every credible study since that time. They ended on a despairing note, commenting that this absurdity was so deeply embedded in cardiologic folklore that it would probably never be extirpated.

Andrés & Raimundo.

Spanish

-Buenos días y “Felices Pascuas” a todos los participantes del foro. Adjunto el VCG en donde remarque en color celeste la onda P, en rojo las primeras parte del asa del complejo QRS que corresponde a la activación del VD, en verde cuando pasa a activar el VI, en violeta cuando las fuerzas finales se dirigen hacia el VD ya que las mismas le ganan al del VI; y en un amarillo transparente la onda T, para su mejor entendimiento. En este VCG tengo dudas sobre el ritmo, en el plano frontal la onda P se observa entrecruzada en el plano frontal, en el horizontal impresiona tener rotación horaria si esto es así el ritmo es de la AI. Con respecto al QRS, tiene una duración aparentemente de 128 msec, como bien dijo Juan presenta una necrosis anterior extensa. Llama la atención que recién a los 30 msec active el VI, se despolariza escasas regiones del septum y luego de la pared lateral con entrecruzamiento de las fuerzas medias; esto puede deberse a un BRI atípico y/o trastorno de conducción del VI. Las fuerzas finales son derechas por disminución de la masa del VI y/o agrandamiento del VD. La onda T es abierta y redondeada secundaria a su cardiopatía isquémica.

Afectuosamente Isabel Konopka MD Buenos Aires Argentina.

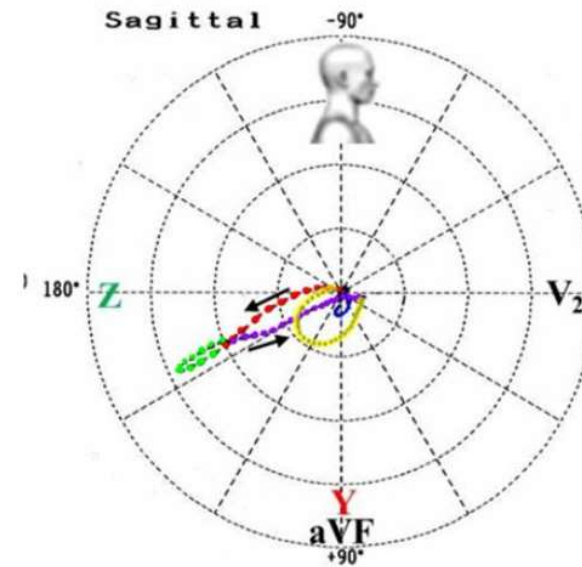
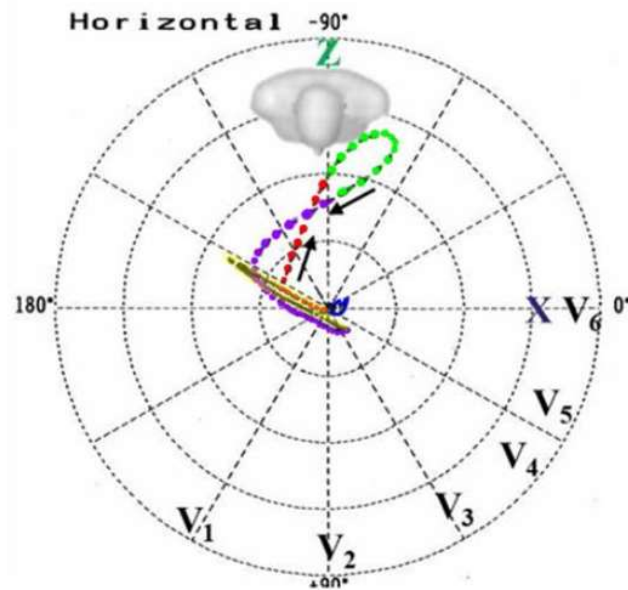
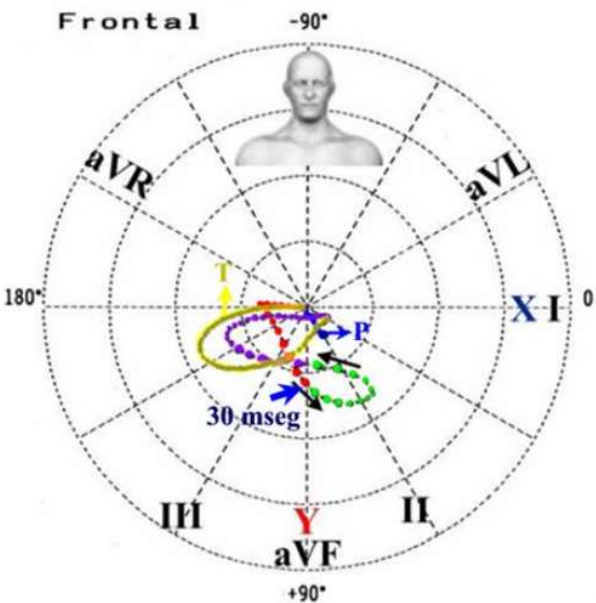


Good morning and “Happy Easter” for all the participants of the forum. I attach the VCG where I highlighted in light blue the P wave, in red the first parts of the QRS complex loop, corresponding to the RV activation, in green when the LV activates, in violet when the final forces go toward the RV, as they are ahead of the LV, and in transparent yellow the T wave, for a better understanding.

In this VCG I have doubts about the rhythm; in the frontal plane the P wave is observed intertwined in the frontal plane, in the horizontal plane it seems there is clockwise rotation, and if so, the rhythm is from the LA. With regard to QRS, it has a duration apparently of 128 ms, and as Juan said very properly, the patient presents an extensive anterior necrosis. It is remarkable that only at 30 ms the LV activates, scant regions of the septum depolarize, and then regions of the lateral wall with intertwining middle forces; this could be due to atypical LBBB and/or LV conduction disorder. The final forces are right by decrease of the LV mass and/or RV enlargement. T wave is open and rounded, secondary to ischemic heart disease.

Affectionately, Isabel **Konopka MD**

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Final comments



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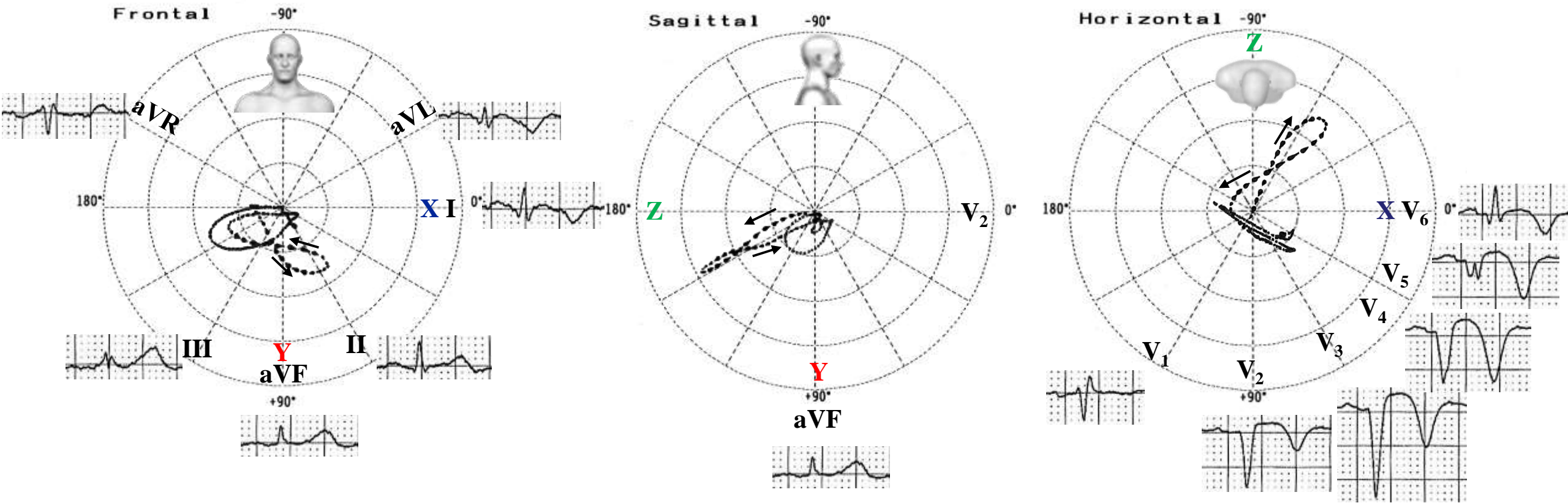
Raimundo **Barbosa-Barros**, MD

Centro Coronariano do Hospital de Messejana Dr. Carlos Alberto Studart Gomes, Fortaleza – CE- Brazil

“Of all the instruments of man, the most amazing is, without a doubt, the book. The rest are extensions of your body. The microscope, the telescope, are extensions of your sight; the telephone is extension of the voice; then we have the plow and the sword, extensions of the arm. But the book is something else: the book is an extension of memory and imagination.” – Jorge Luis Borges

“De todos los instrumentos del hombre, el más asombroso es, sin duda, el libro. Los demás son extensiones de su cuerpo. El microscopio, el telescopio, son extensiones de su vista; el teléfono es extensión de la voz; luego tenemos el arado y la espada, extensiones del brazo. Pero el libro es otra cosa: el libro es una extensión de la memoria y la imaginación.” – Jorge Luis Borges

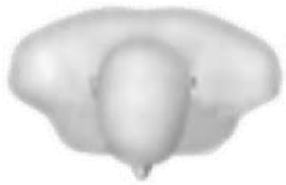
ECG / VCG correlation of a typical anterolateral wall infarction with possible anterior wall aneurysm



Laterobasal MI (old dorsal) is one of the more difficult ECG or VCG diagnoses. The basal wall of the left ventricle is said to be depolarized late in the QRS cycle. In some cases of laterobasal infarct the late part of the QRS forces may be located anteriorly with a pseudo RBBB pattern with triphasic pattern in V₁ lead such as the present case. Following an acute STEMI, the ST segments return towards baseline over a period of 14 days, while the Q waves persist and the T waves usually become flattened or inverted. However, some degree of ST elevation remains in 60% of patients with anterior STEMI and 5% of patients with inferior STEMI. The mechanism is thought to be related to incomplete reperfusion and scar formation following an acute MI. This VCG/ECG pattern is associated with paradoxical movement of the ventricular wall on echocardiography (ventricular aneurysm). The wringing motion of the myocardium might be an important mechanism involved in maintaining normal cardiac function with minimal expenditure of energy. This mechanism no longer operates in patients with LV aneurysms and operates significantly less than normal in those with anterolateral hypokinesia. Diastolic untwisting is significantly delayed and prolonged in patients with anterolateral infarction, which could explain the (Nagel 2000) occurrence of diastolic dysfunction in these patients.

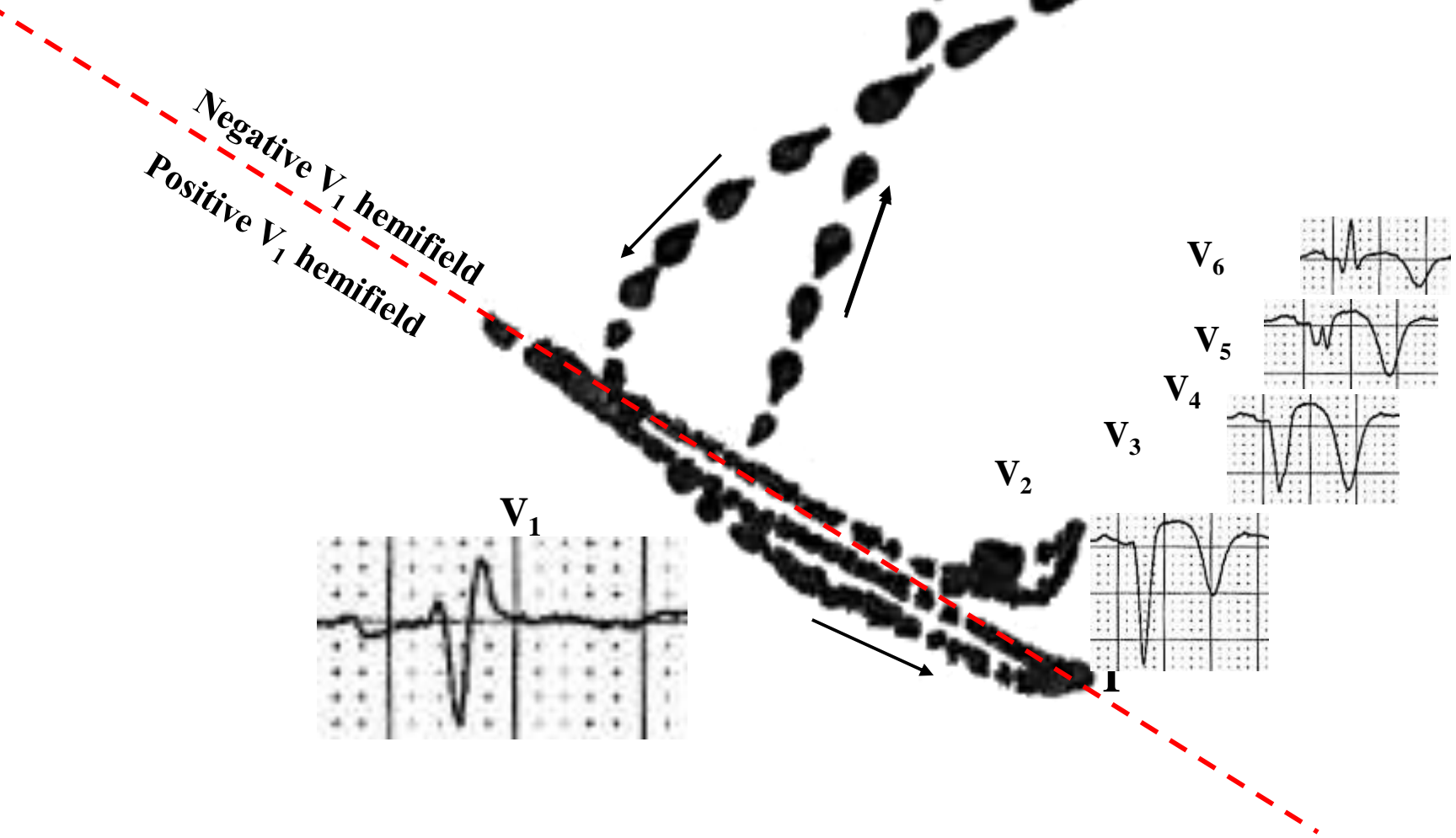
LV aneurysm is a well-known sequela of MI. It was apparently recognized centuries ago (**Dolly 1951**); however, its association with coronary occlusion and MI was not definitively noted until the early 1900s (**Hall 1903**). A LV aneurysm can be diagnosed on ECG when there is persistent ST segment elevation occurring 6 weeks after a known MI (usually an anterior or anterolateral MI). Without knowing the past medical history, the ECG changes of an aneurysm may mimic an acute anterior MI. With an anterior or apical aneurysm, the persistent ST elevation is in lead V1 and V2 with associated Q waves indicating the old anterior MI. In an inferior aneurysm it would be in lead II, III and aVF, although this is less common. The only way to be sure that the ECG changes present are from an LV aneurysm (not ST elevation from an acute MI) is to have the patient's history of a prior MI and cardiac imaging to document the presence of an aneurysm. The shape of the ST elevation is also relatively unique and has been described as "coving". A large mostly anterior MI without adequate and timely reperfusion therapy can lead to extensive loss of myocardial tissue. As this weakens the anterior ventricular wall, this can result in local bulging of the anterior wall. If profound this is called a cardiac aneurysm. Cardiac aneurysms often have typical ECG characteristics: anterior Q waves and persistent ST elevation. Patients with cardiac aneurysms have a bad prognosis due to the severely reduced LVEF, they often have HF and risk SCD due to VF. LV aneurysm, which can impair systolic function, has a reported incidence of 10% to 35% in patients after MI (**Henry 2014**). In the current era of early revascularization, the overall prevalence of this complication might be decreasing. LV aneurysm, true or pseudo, is a potential complication of MI; the establishment of a patent infarct artery reduces the risk of aneurysm formation. Ventricular aneurysm may be seen in other conditions such as hypertrophic cardiomyopathy, cardiac sarcoidosis, myocarditis (example: Chagasic myocarditis), congenital disease, after cardiac surgery, and in blunt trauma. A true ventricular aneurysm differs from a pseudoaneurysm in terms of morphology and risk of rupture.⁹ A true ventricular aneurysm is thinning of the ventricular wall in an area void of muscle and is caused by fibrosis or scar formation; it is akinetic and bulges during systole and diastole. A true aneurysm has a wide neck, and the original wall of the myocardium is present within the aneurysmal wall. A pseudoaneurysm has a narrow neck and involves a rupture of the free wall of the LV that is contained by pericardial adhesions. No elements of the myocardium are present in the wall of the pseudoaneurysm. Rupture is more common with a pseudoaneurysm than with a true aneurysm. A true aneurysm is not likely to rupture once fibrosis has occurred, whereas a pseudoaneurysm can rupture after the development of fibrosis. Large pseudo-aneurysms are more likely to rupture than smaller ones. Rupture of the free wall of the ventricle occurs in 4% of patients who experience a MI and in 23% of patients who die of a MI. Rupture of the free wall is more common than septal rupture and is typically a fatal event unless the rupture is contained by adhesions in the pericardium, such as in pseudoaneurysm. Pericardial adhesions can be present before or can develop during a rupture. True ventricular aneurysms and pseudoaneurysms can cause chest pain. However, chest pain after MI has many differentials, including pericarditis, stent thrombosis or restenosis, ACS from another lesion, and residual angina from small vessel disease not amenable to revascularization. A pseudoaneurysm can result in dyspnea, with HF being the most common presentation, or it can be clinically silent.

Pseudo RBBB in lateral MI

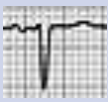






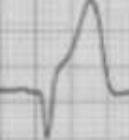




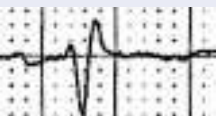


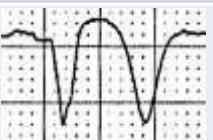
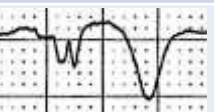
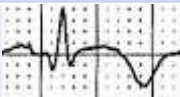


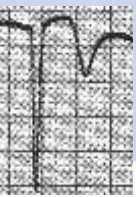
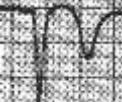
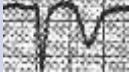
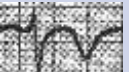


Triphasic QRS complexes that resemble IRBBB in V_1 , appear in 40% of the cases in lateral MI (old dorsal): pseudo RBBB

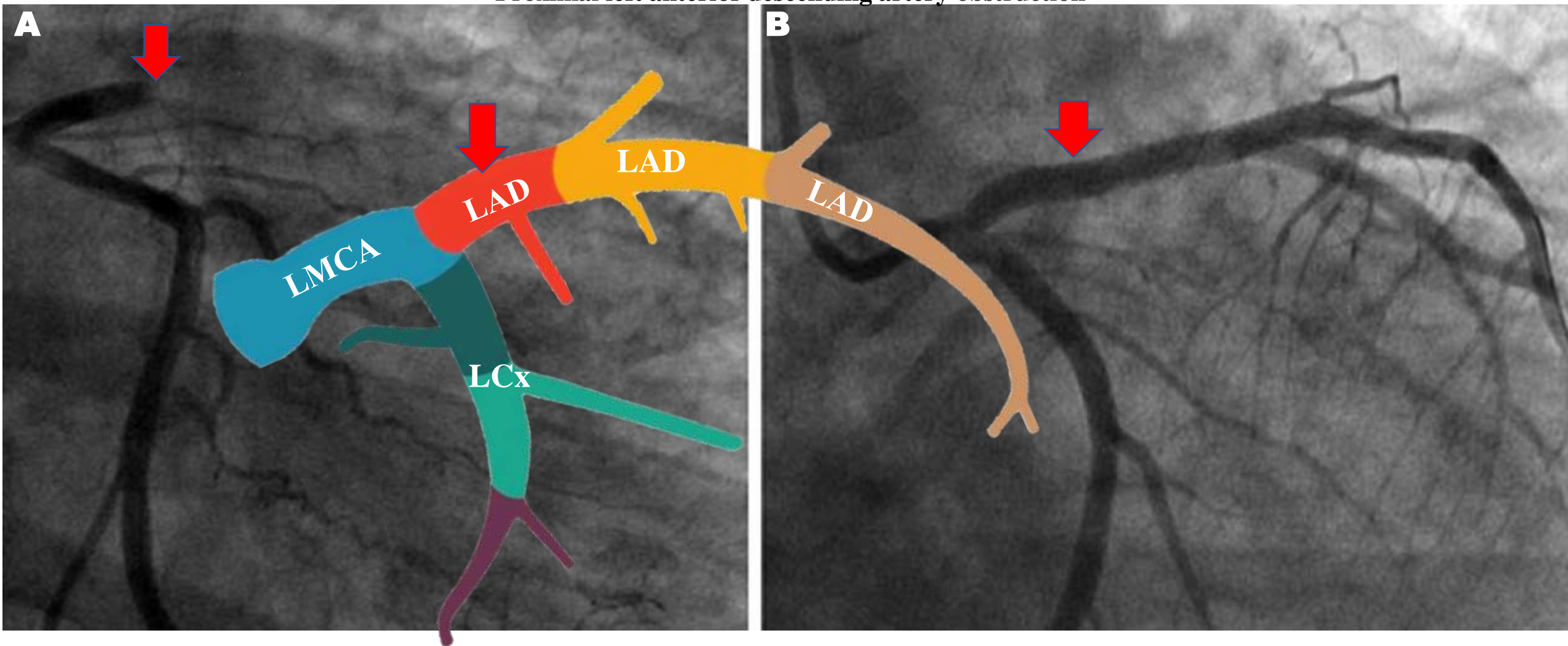
In many of these patients the direction of inscription may also be definitely abnormal with marked figure-of-eight or even completely clockwise QRS-loop rotation.



Effective electrical site of injury(ST) and ischemia(T-loop-wave)	Direction of ST vector	Orientation of T sÊ loop
Anteroseptal to the left	Right, anterior	Left , posterior
Anterior	Anterior and slightly	Posterior and slight to the right
Anterolateral	Left anterior	Right, posterior
Extensive anterior	Anterior or left and anterior	Posterior, right

	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Normal						
Acute anteroseptal MI						
Acute anterior MI						
Acute anterolateral MI	 rS or rSr'					
Acute extensive Anterior MI	 QS					

Proximal left anterior descending artery obstruction



Angiography (right anterior oblique cranial projection) of the left anterior descending artery. (A) Initial angiogram demonstrating total occlusion of the proximal left anterior descending artery(LAD) (red arrow). (B) In the same projection as in A, after deployment of a stent in the proximal LAD (arrow). Thrombolysis in myocardial infarction (TIMI) III flow was demonstrated. LMCA: left main coronary artery; LAD: proximal, intermediate and distal left anterior descending coronary artery; LCx - Left circumflex artery.

The LAD supplies blood to a large part of the myocardium. However, the amount of myocardium supplied varies depending on the length of the LAD and as a result, occlusion of its proximal portion may influence outcome.

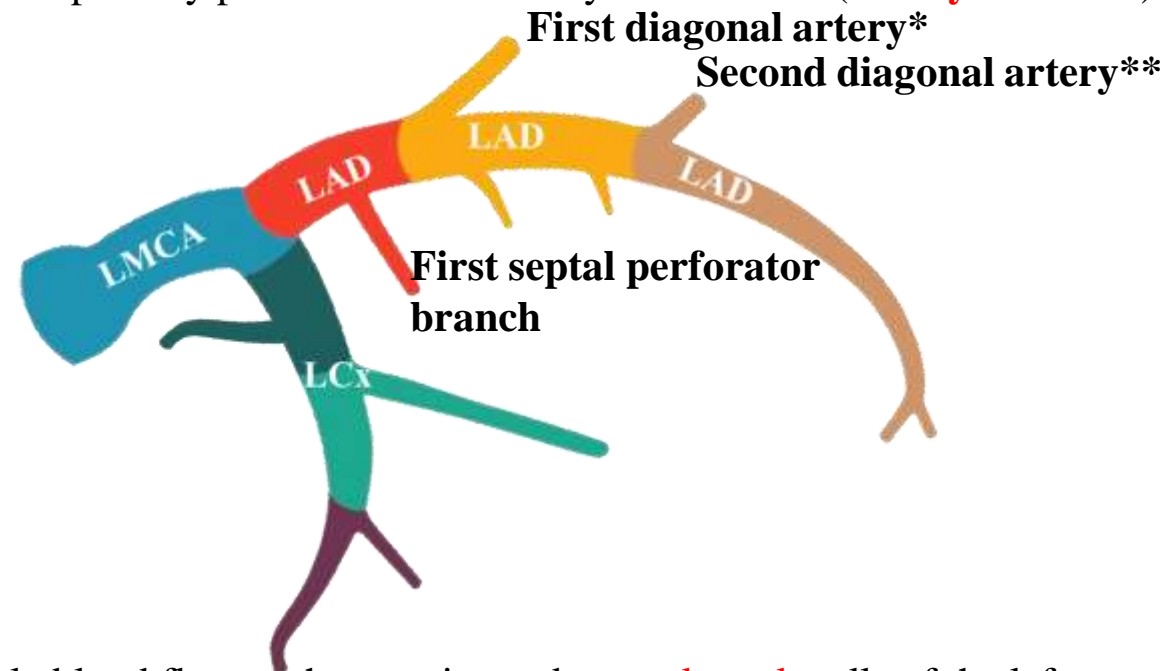
Left anterior descending artery (LAD) – approx. 130 mm in length and proximal diameter of about 3-4 mm.

This one goes through the anterior interventricular groove to the apex and is surrounding it and continues with 1-2 cm in the posterior interventricular groove (recurrent artery). It gives the following branches:

- I. Diagonal arteries: irrigate the anterior wall of the left ventricle and a part of the antero-lateral papillary muscle;
- II. anterior septal arteries: irrigate two-thirds of anterior interventricular septum, the right and left branch of the His bundle;
- III. Posterior septal branches (from recurrent artery): irrigates one-third of the posterior interventricular septum;
- IV. Anterior right ventricular branches (left artery of the pulmonary cone).

LAD is divided into three segments:

- **First Segment** (proximal red): between the left coronary artery trunk and the first diagonal (when this is missing until the first septal).
- **Second Segment** (medium yellow): between the first and the second diagonal.
- **Third Segment** (distal. brown): after the second diagonal artery. Wrap-around LAD is a strong predictor of prognosis in patients with anterior wall MI undergoing PPCI to isolated proximal LAD occlusion. In addition, those with a shorter LAD have an excellent prognosis (Iliia 2014). In patients with an anterior STEMI. In conclusion, a wrap-around LAD predicted adverse clinical outcomes at 3 years in patients with anterior STEMI who underwent primary percutaneous coronary intervention (Kobayashi 2015).



* and **: Diagonal branches of the LAD supply blood flow to the anterior and anterolateral walls of the left ventricle.

Acute anterolateral MI is recognized by ST segment elevation in leads I, aVL and the precordial leads overlying the anterior and lateral surfaces of the heart. Generally speaking, the more significant the ST elevation, the more severe the infarction. There is also a loss of general R wave progression across the precordial leads and there may be symmetric T wave inversion as well.

Anterolateral myocardial infarctions frequently are caused by occlusion of the proximal left anterior descending coronary artery, or combined occlusions of the LAD together with the right coronary artery or left circumflex artery. Arrhythmias which commonly preclude the diagnosis of anterolateral MI on ECG and therefore possibly identify high risk patients include right and left bundle branch blocks, fascicular block and Mobitz type II second degree atrioventricular conduction blocks.

Other important ECG patterns to be aware of:

Anterior-inferior STEMI due to occlusion of a “wraparound” LAD: simultaneous ST elevation in the precordial and inferior leads due to occlusion of a variant (“type III”) LAD that wraps around the cardiac apex to supply both the anterior and inferior walls of the left ventricle.

Left main coronary artery occlusion/multivessel obstruction:

widespread ST depression with ST elevation in aVR \geq V1. When the resting ECG reveals ST-segment depression greater than 0.1 mV (1 mm) in 8 or more body surface leads coupled with ST-segment elevation in aVR and/or V1 but is otherwise unremarkable, the automated interpretation should suggest ischemia due to multivessel or left main coronary artery obstruction (**Wagner 2009**)

Wellens’ syndrome, LAD coronary T-wave syndrome or the “widow maker”, is a pre-infarction syndrome with non-classical features. Diagnostic criteria include a history of anginal chest pain, little or no cardiac enzyme elevation (negative cardiac biomarkers), little or no ST-segment elevation (<1 mm), no loss of precordial R waves, no pathologic precordial Q waves and typical T-wave changes: Deep precordial T wave inversions or biphasic T waves in V2-3, indicating critical proximal LAD stenosis (a warning sign of imminent anterior MI). Urgent cardiac catheterization is vital to prevent myocardial necrosis (**de Zwaan 1982; de Zwaan 1989**). This syndrome continues to be a 'can't miss' for the clinician as delay in urgent angiography and intervention can result in anterior MI left ventricular dysfunction, arrhythmias, and death.

De Winter’s T waves:

Upsloping ST depression with symmetrically peaked T waves in the precordial leads; a “STEMI equivalent” indicating acute LAD occlusion. The de Winter ECG pattern is an anterior STEMI equivalent that presents *without* obvious ST segment elevation.

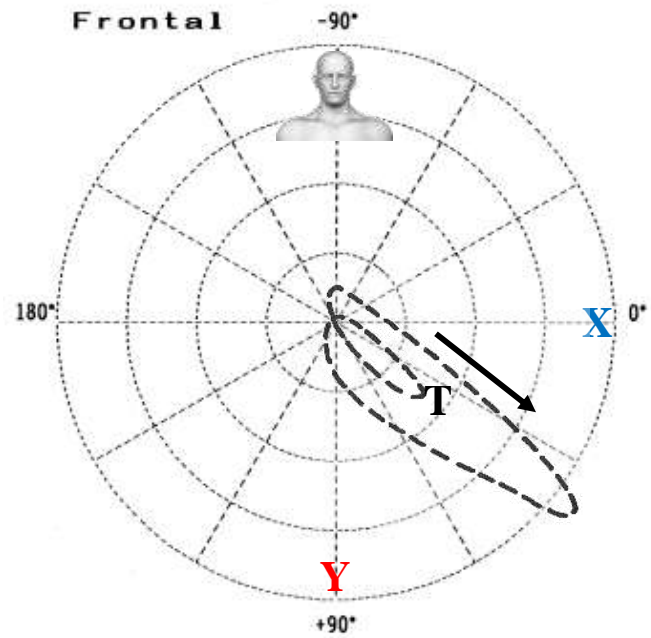
Key diagnostic features include ST depression and peaked T waves in the precordial leads.

The de Winter pattern is seen in \cong 2% of acute LAD occlusions and is under-recognized by clinicians.

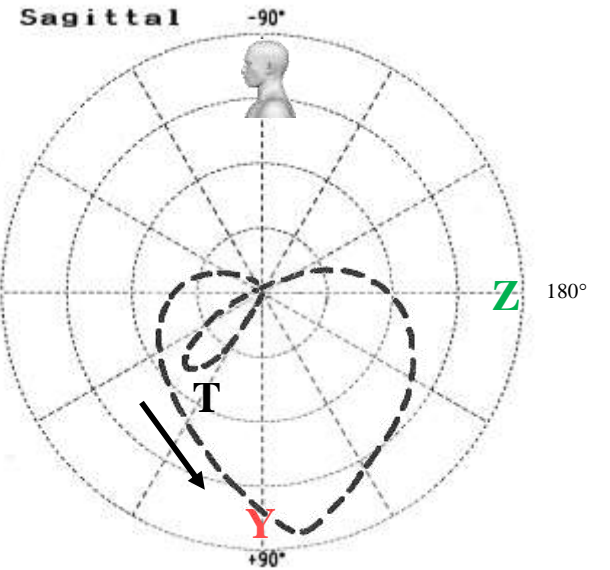
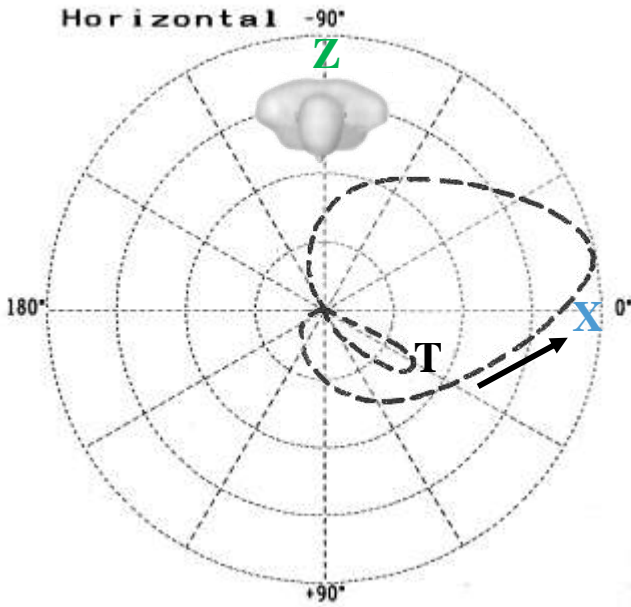
Unfamiliarity with this high-risk ECG pattern may lead to under-treatment (e.g. failure of cath lab activation), with attendant negative effects on morbidity and mortality (**de Winter 2008**).

Direction of the normal T-loop and in the ischemia in the various walls (Phibbs 1983)

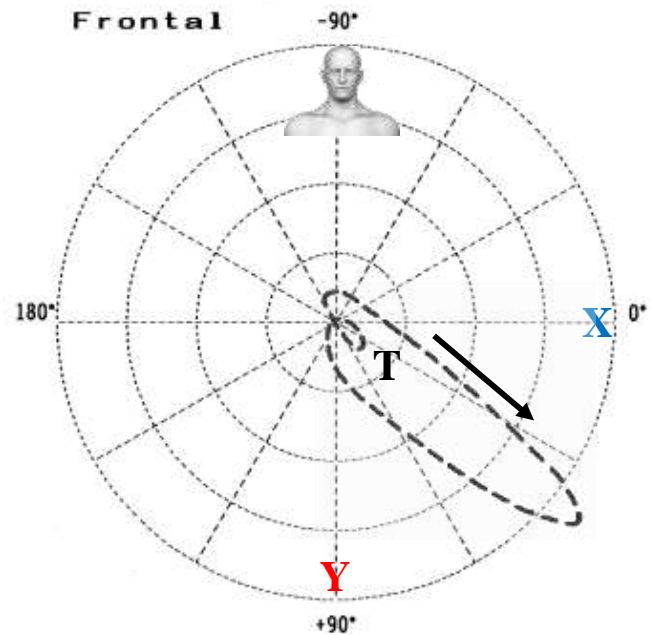
Normal T-loop on the 3 planes



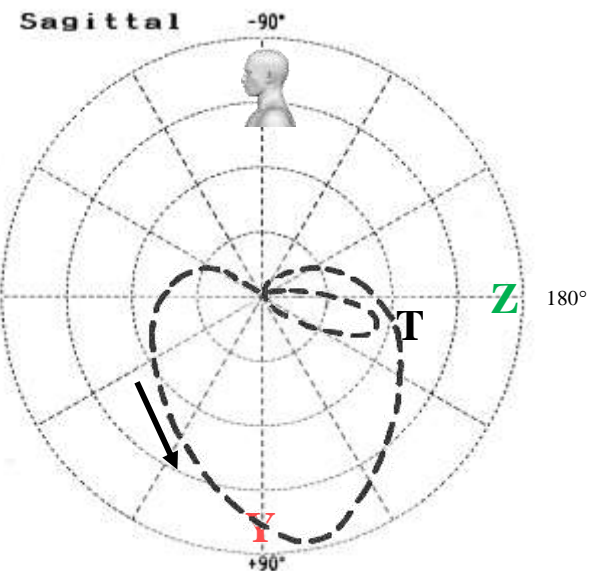
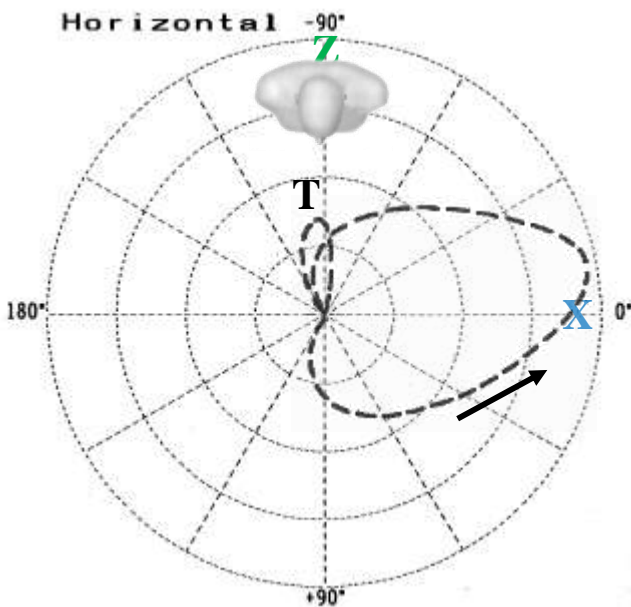
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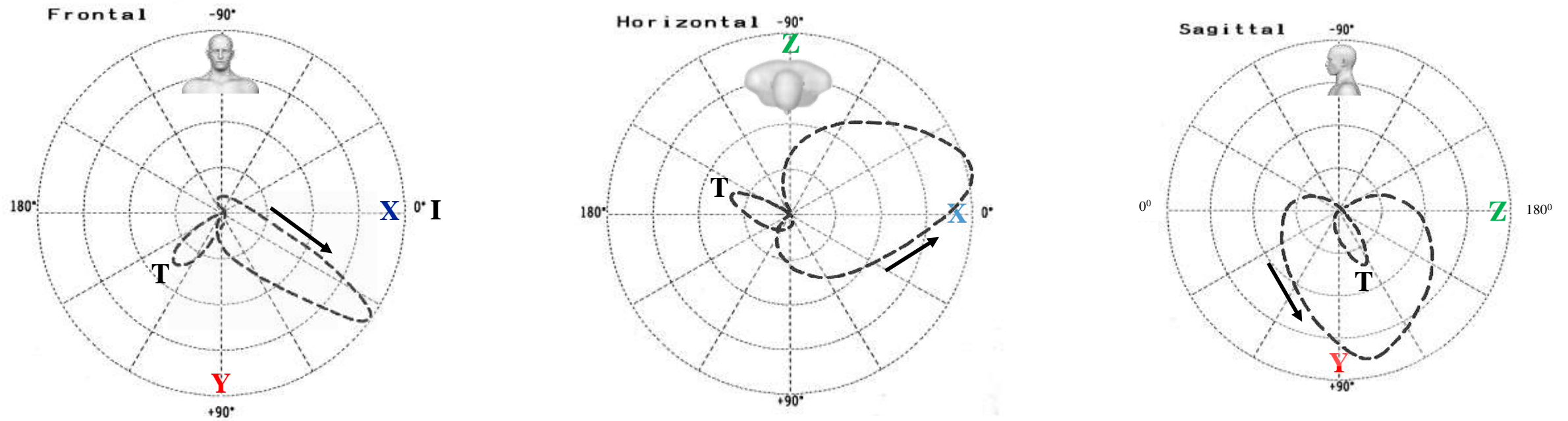
T-loop in the 3 planes in anterior wall ischemia



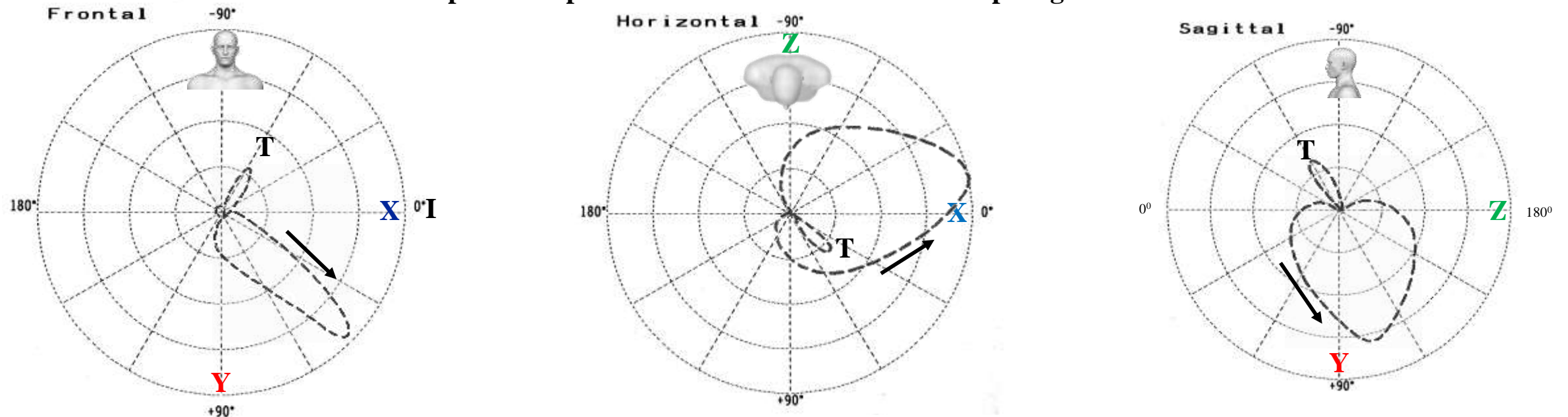
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T-loop in the 3 planes in the ischemia of the anterolateral wall

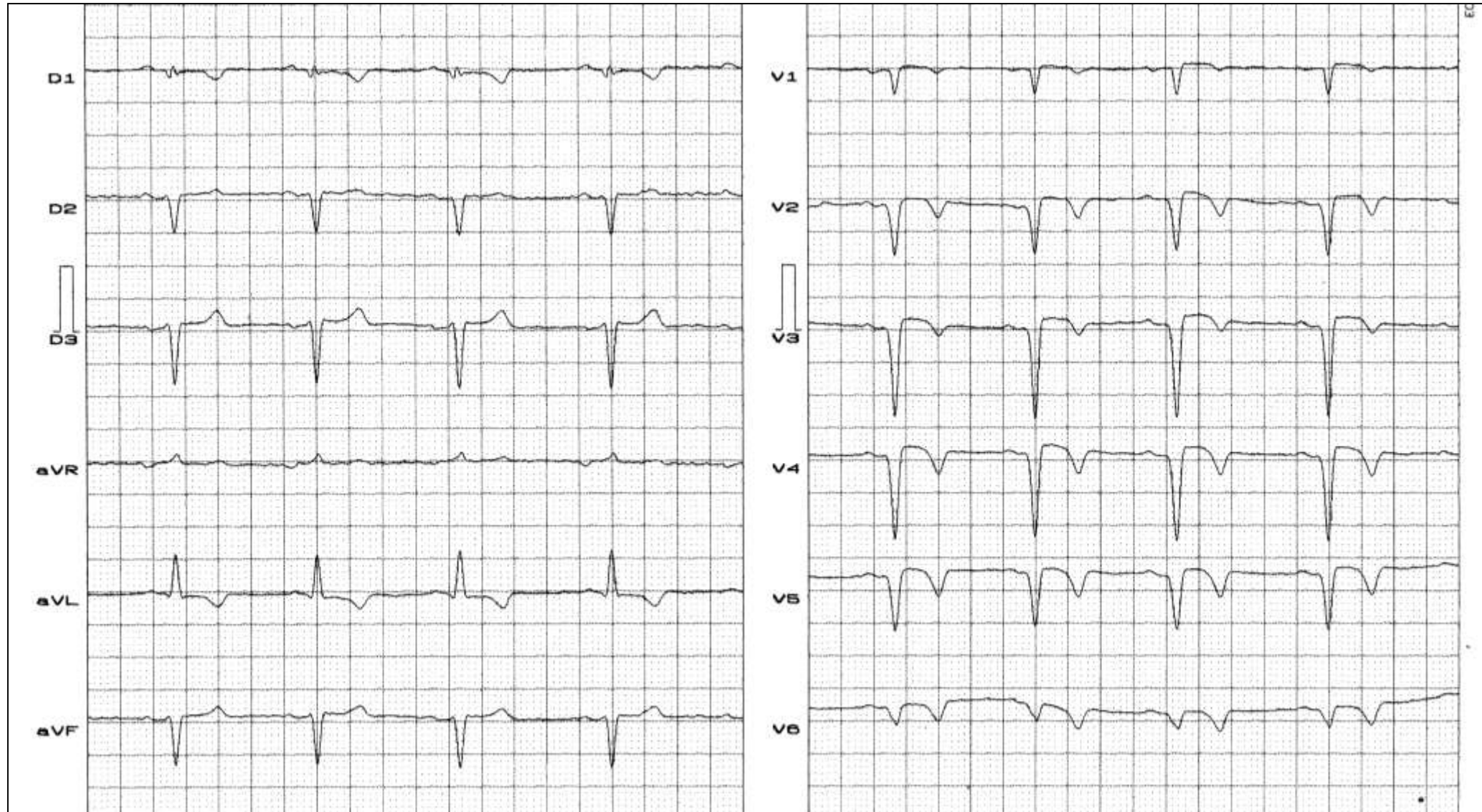


T-loop in the 3 planes in ischemia of inferior or diaphragmatic wall



Another example of ECG/VCG with extensive anterolateral MI

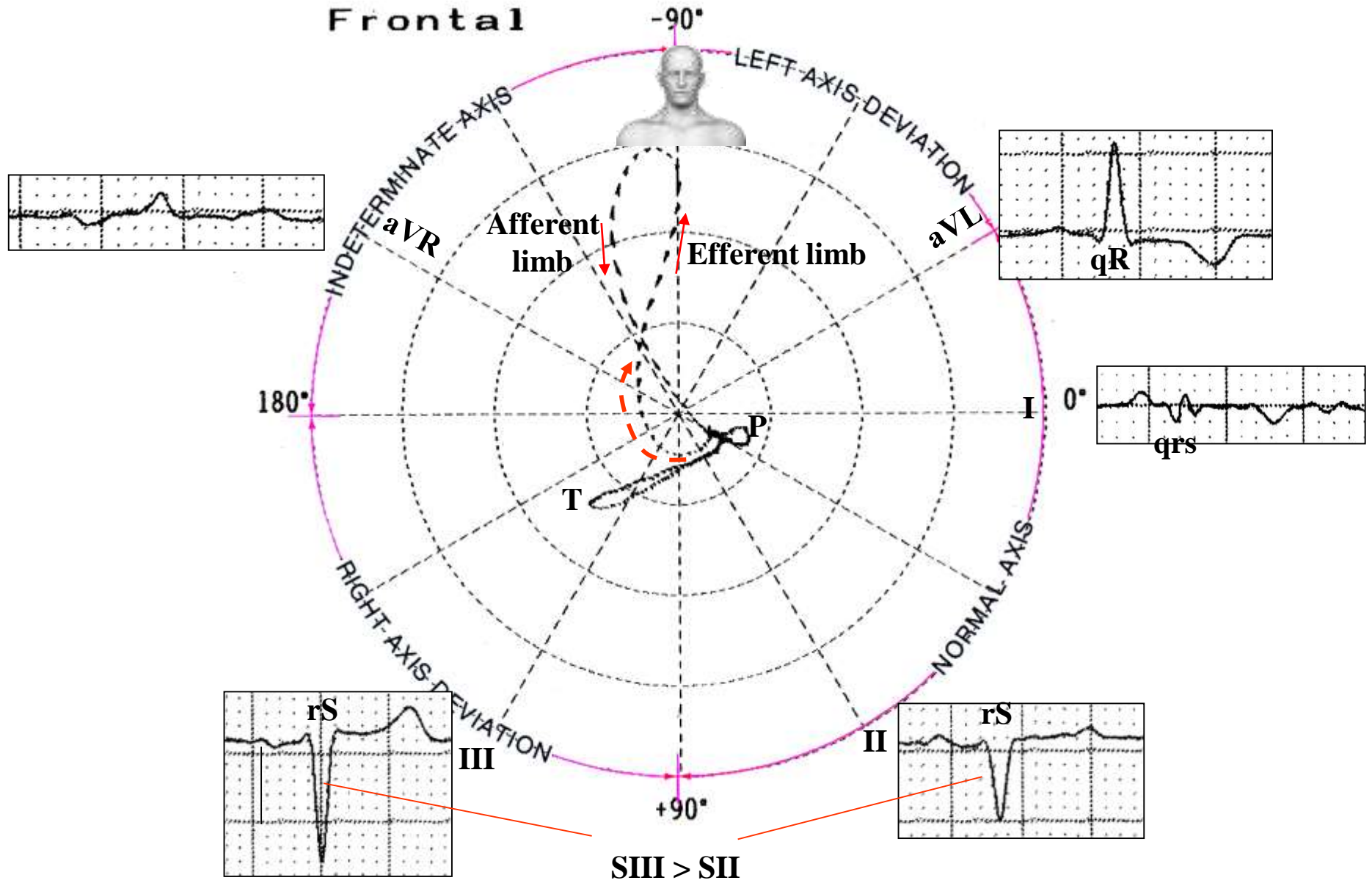
Name: JBSN; **Date:** 18/08/2007; **Age:** 46y; **Sex:** M; **Race:** Caucasian; **Weight:** 72 Kg; **Height:** 1.62 m.



Clinical Diagnosis: This patient has suffered from extensive anterolateral MI associated with cardiogenic shock and congestive heart failure six months previously. ischemic T waves. It's a combination of anteroseptal, anterior and anterolateral infarctions. Extreme superior axis deviation. Atypical left anterior fascicular Block (LAFB). See comments in ECG/VCG correlation in the FP.

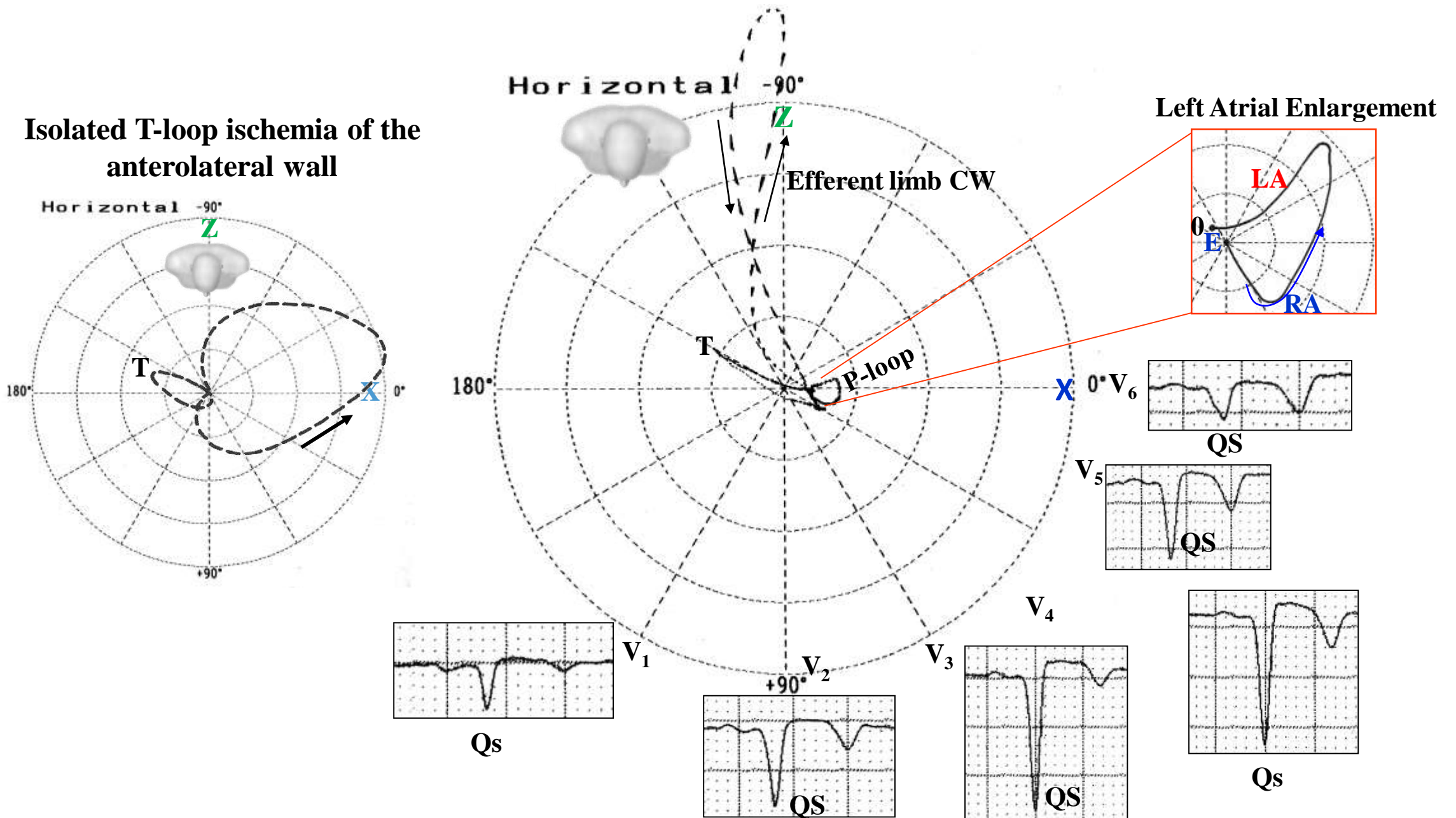
ECG diagnosis: anterolateral myocardial infarction and ischemia + atypical LAFB.

ECG/VCG correlation in the frontal plane



QRS axis on right superior quadrant (AKA “Northwest Axis” or No man's land), the 10 to 20ms vector directed to down and rightward, QRS loop-rotation in eight with the main of QRS-loop with CCW rotation. T-loop directed to back and downward: ischemia of the anterolateral wall.

ECG/VCG Horizontal Plane correlation



Left atrial enlargement + extensive anterolateral myocardial infarction (abnormal QS across all precordial leads associated with T-loop directed to back and rightward: ischemia. Efferent limb of the QRS loop is inscribed clockwise (CW) in the HP.

Vectorcardiographic features in anterolateral myocardial infarction

Several studies have show that vectorcardiographic criteria for anterior myocardial infarction can be significantly more sensitive than traditional electrocardiographic criteria (**McConahay 1970: Levine 1972**).

Horizontal plane

- The initial rightward QRS forces may show early rightward forces exceeding 222ms in duration (**Chou 1967**). This initial deflection of the QRS loop usually encloses a relatively large area.
- The body of the horizontal QRS loop is written far posteriorly and slightly to the left or right of the middle line. The long axis of the QRS loop, or the maximal instantaneous QRS vector in the HP ordinarily is situated between -90° and -50° in the horizontal reference frame
- The mean 20ms instantaneous QRS vector in this plane usually lies and either anteriorly or posteriorly, in contrast with its normal orientation to the left and anteriorly. Occasionally this vector is located to the left and far posteriorly.
- The QRS loop rotation may have clockwise, counterclockwise or figure in eight configuration depending apparently on the medial displacement of the **efferent limb**.
- Efferent limb of the QRS loop is inscribed clockwise (CW).
- Occasionally moderate conduction delay is observed in the **afferent limb** in all three planes.
- The T-loop oriented rightward and posterior

Sagittal plane (left or right)

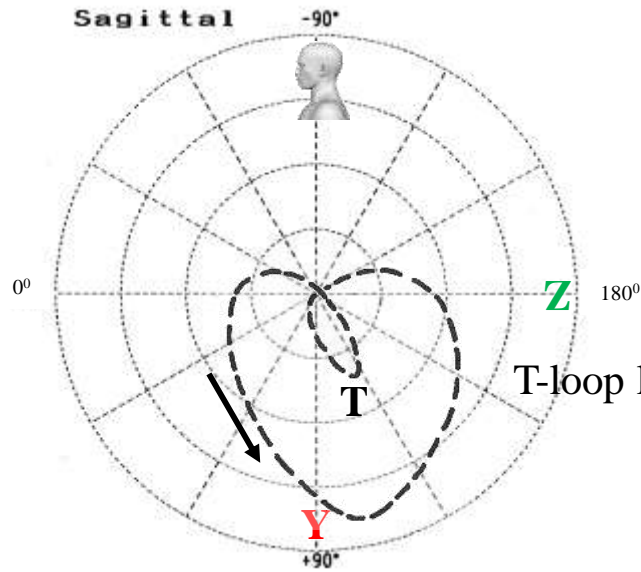
- Marked posterior orientation
- The inscription may be either clockwise or counterclockwise
- T-loop directed to back and downward

Frontal plane

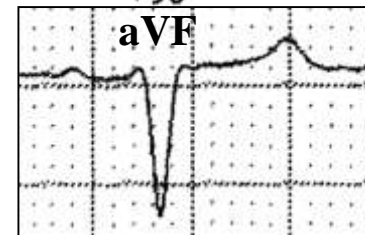
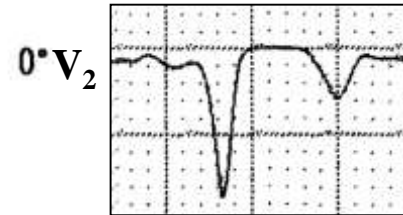
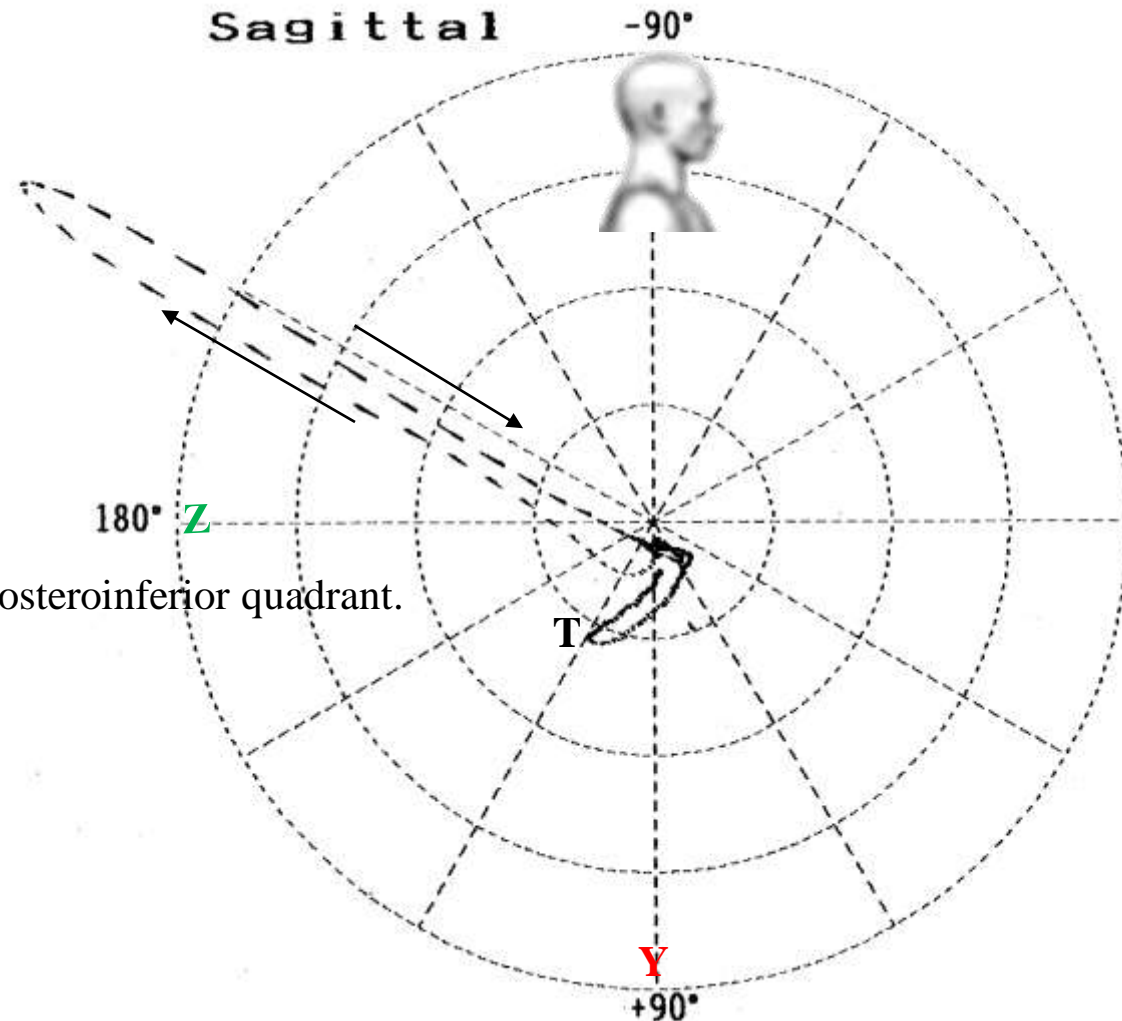
- The initial portion of the QRS loop is written faster to the right than normal at least 200 ms.

ECG/VCG correlation in the right sagittal plane

Isolated ischemic T-loop in the LSP

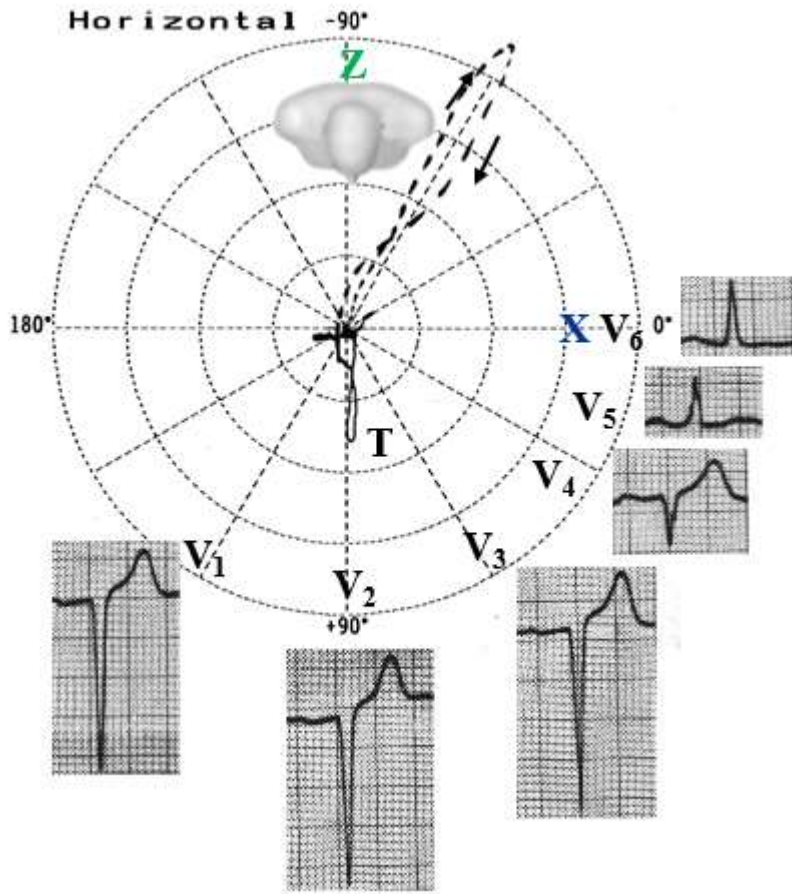


T-loop located in the posteroinferior quadrant.



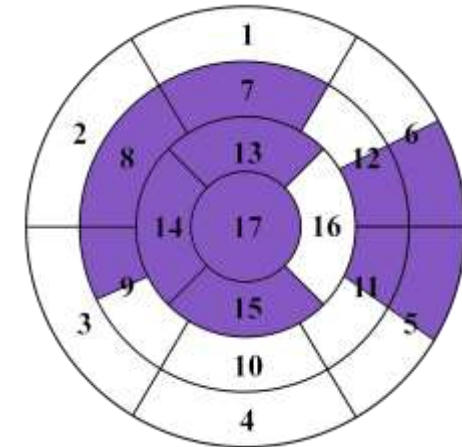
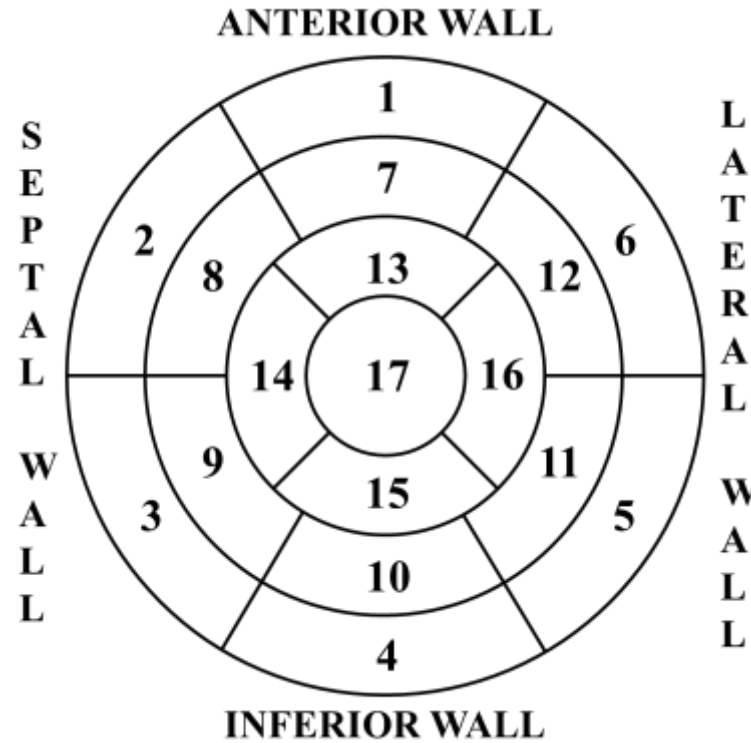
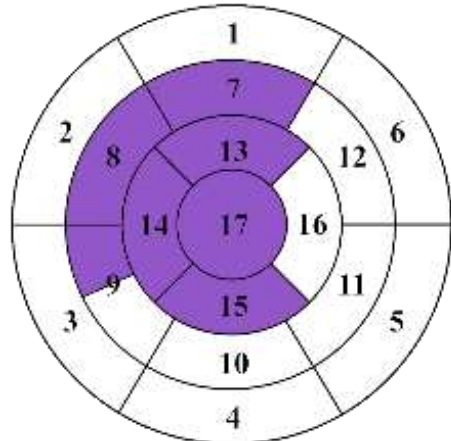
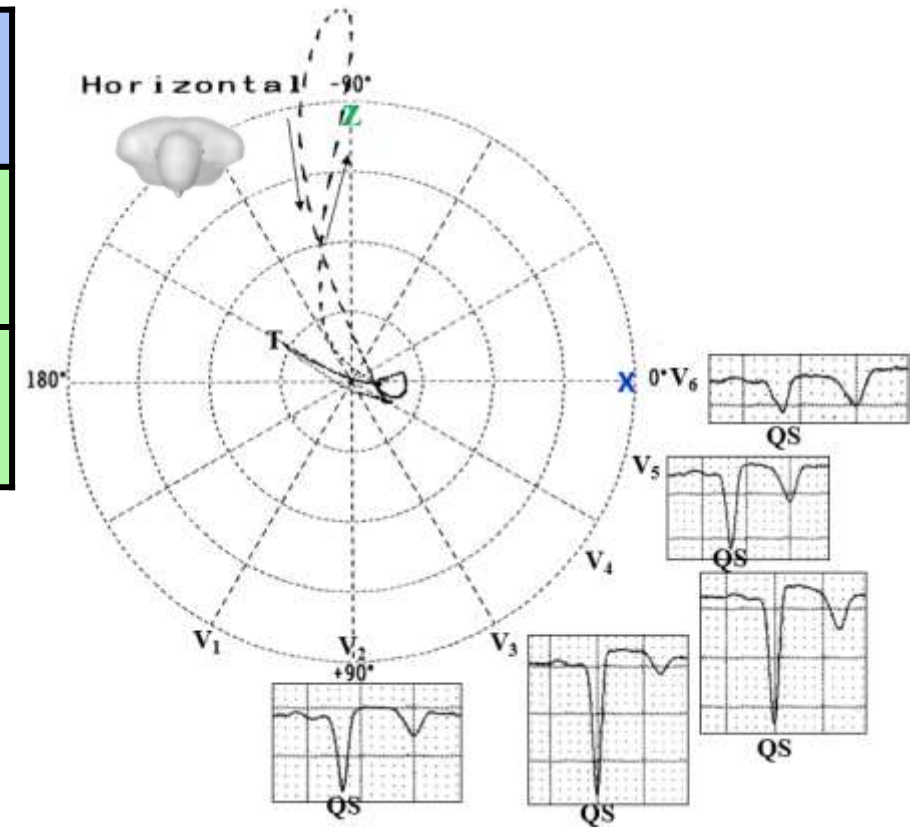
The QRS loop is located in superior-posterior quadrant. The T-loop is directed to back and downward: ischemia of the anterolateral wall

Extensive anterior MI in the HP



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

Extensive anterolateral MI in the HP



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