Infarto Agudo do Miocárdio complicado associado a Forças Anteriores Proeminentes

Complicated Acute Myocardial Infarction With Prominent Anterior QRS forces

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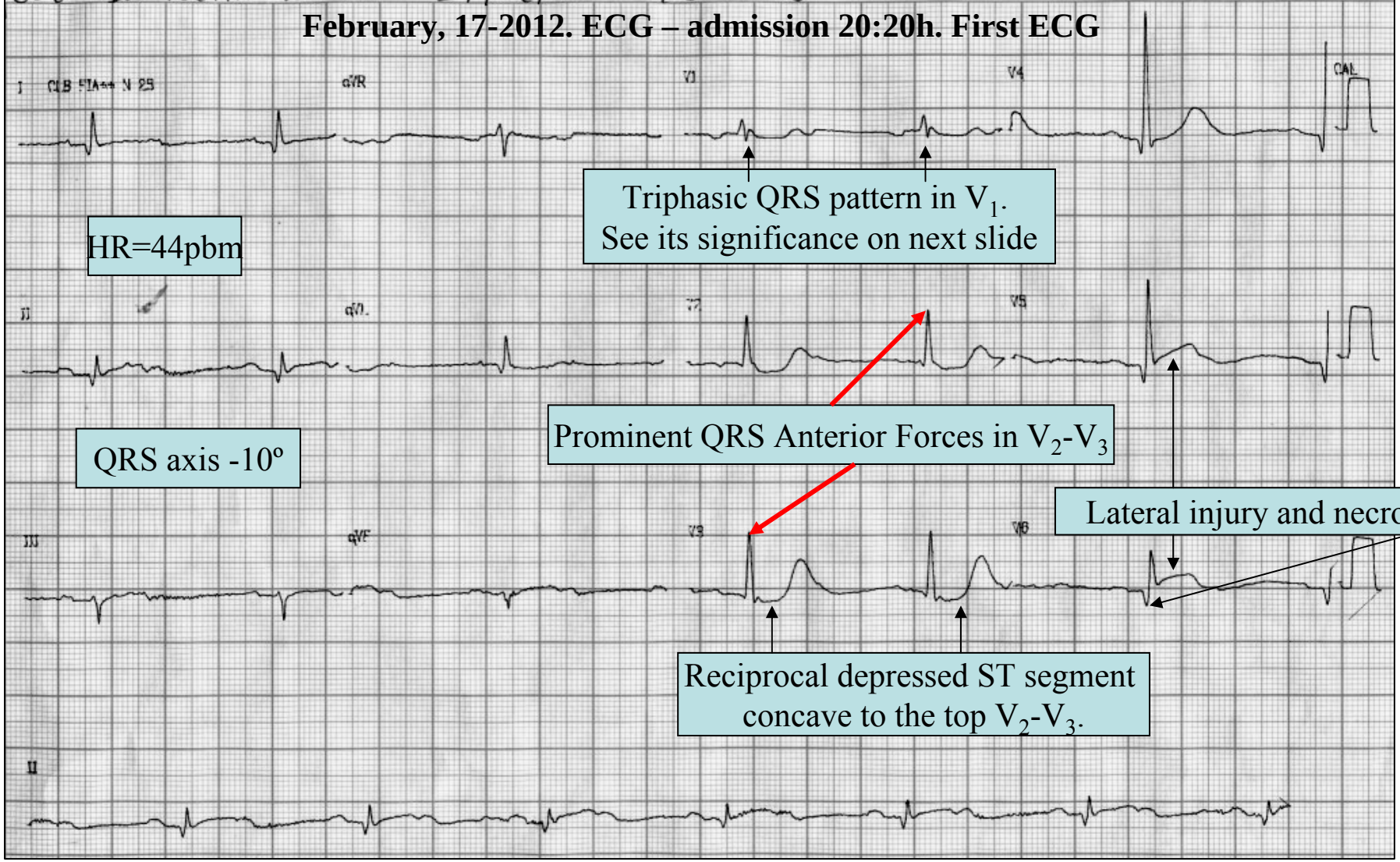
Coronary Center Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza-Ceará-Brazil

Final comments Andrés Ricardo Pérez-Riera M.D. Ph.D.

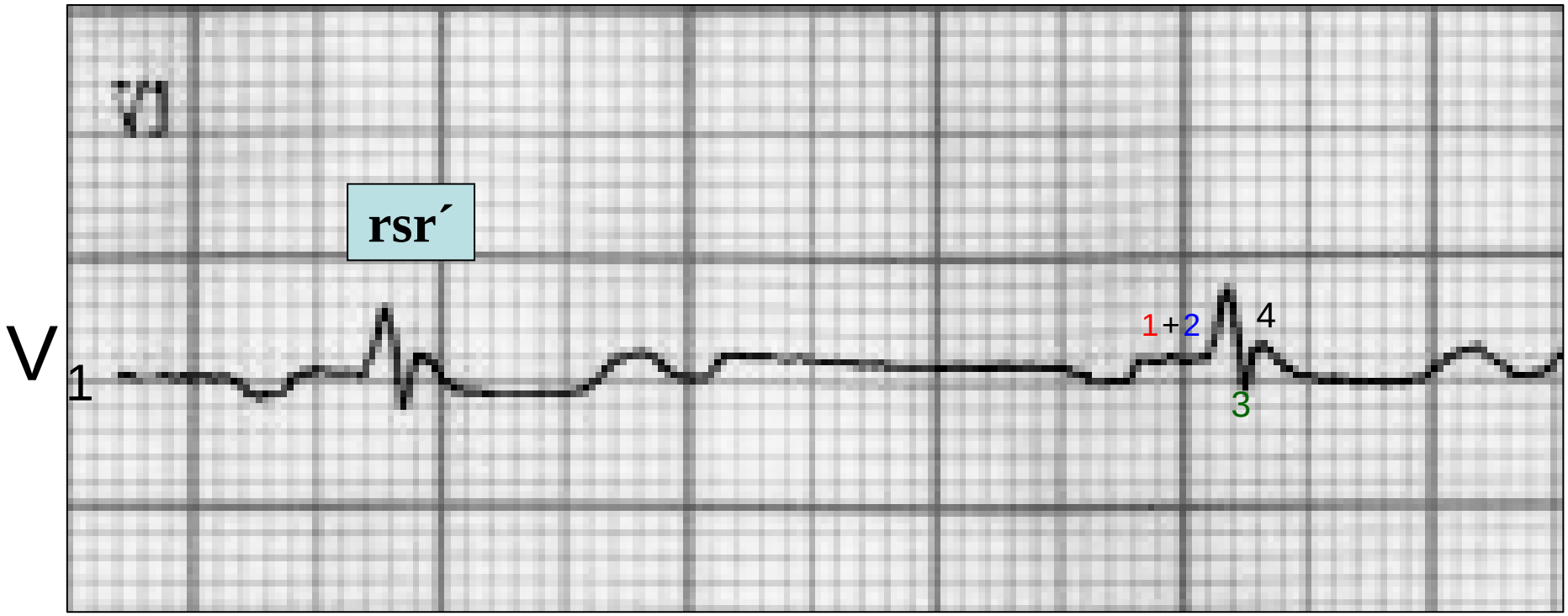
Paciente que foi admitido na nossa sala de emergência com quadro de sindrome coronariana aguda (infarto agudo de miocárdio) de parede ínfero-lateral que complicara inicialmente com bloqueio atrioventricular 2:1 e posteriormente bloqueio AV total .
Encaminhado para o laboratório de hemodinâmica onde implantou-se um marcapassos provisório e colocação de stent para artéria circunflexa esquerdda (artéria culpada).Após alguns dias houve resolução do bloqueio AV com o aparecimento de forças QRS anteriores proeminentes.
O paciente além da oclusão total da artéria circunflexa esquerda também apresentava lesão obstrutiva severa proximal da artéria coronária descendente anterior (está aguardando para realizar intervenção coronária eletiva). Depois mando mais detalhes.
Qual o motivo das forças anteriores prominentes?
O infarto antigamente denominado dorsal (hoje lateral)? ou
é consequecia de um bloqueio das fibras médias do ramo esquerdo do feixe de His pela lesão critica proximal da DA?
Um abraço Raimundo

Patient that was admitted in our emergency room with acute coronary syndrome symptoms (acute myocardial infarction), of inferolateral wall, that initially evolved with 2:1 atrioventricular block and later total AV block. He was submitted to the hemodynamic lab, where a temporary PM was implanted and PTCA with stent for the Left Circumflex Artery (LCx) (culprit artery). After some days, the AV block solved with the appearance of prominent anterior QRS forces (PAF). The patient, besides total occlusion of the LCx, also presented severe proximal obstructive lesions in the left anterior descending artery (LAD) (he is waiting to undergo elective coronary intervention). Later I will send further details.
The question that cannot be held back is this: what is the reason for the prominent anterior QRS forces?
The previously called dorsal wall myocardial infarction? (actually lateral) Or is it just a consequence of the middle fibers block of the His bundle(Left Septal Fascicular Block) by critical proximal lesion of the LAD?
Warm regards,
Raimundo Barbosa-Barros M.D.



The lateral wall necrosis (dorsal or posterior wall necrosis in the antique nomenclature) would only be recorded in the accessory leads V_7, V_8 and V_9. however, V_2-V_3 (leads opposite the lateral wall (antique dorsal wall) record prominent R waves: mirror or reciprocal image of the events occurred in the lateral wall (antique dorsal wall.). Additionally, ST segment elevation in V_5 and V_6 associated with ST segment depression in V_1, V_2 and V_3 are indicative of LCx. occlusion: Sensitivity 83%; Specificity 96%; Positive predictive value 91%; Negative predictive value 93%.

Triphasic QRS pattern in V_1

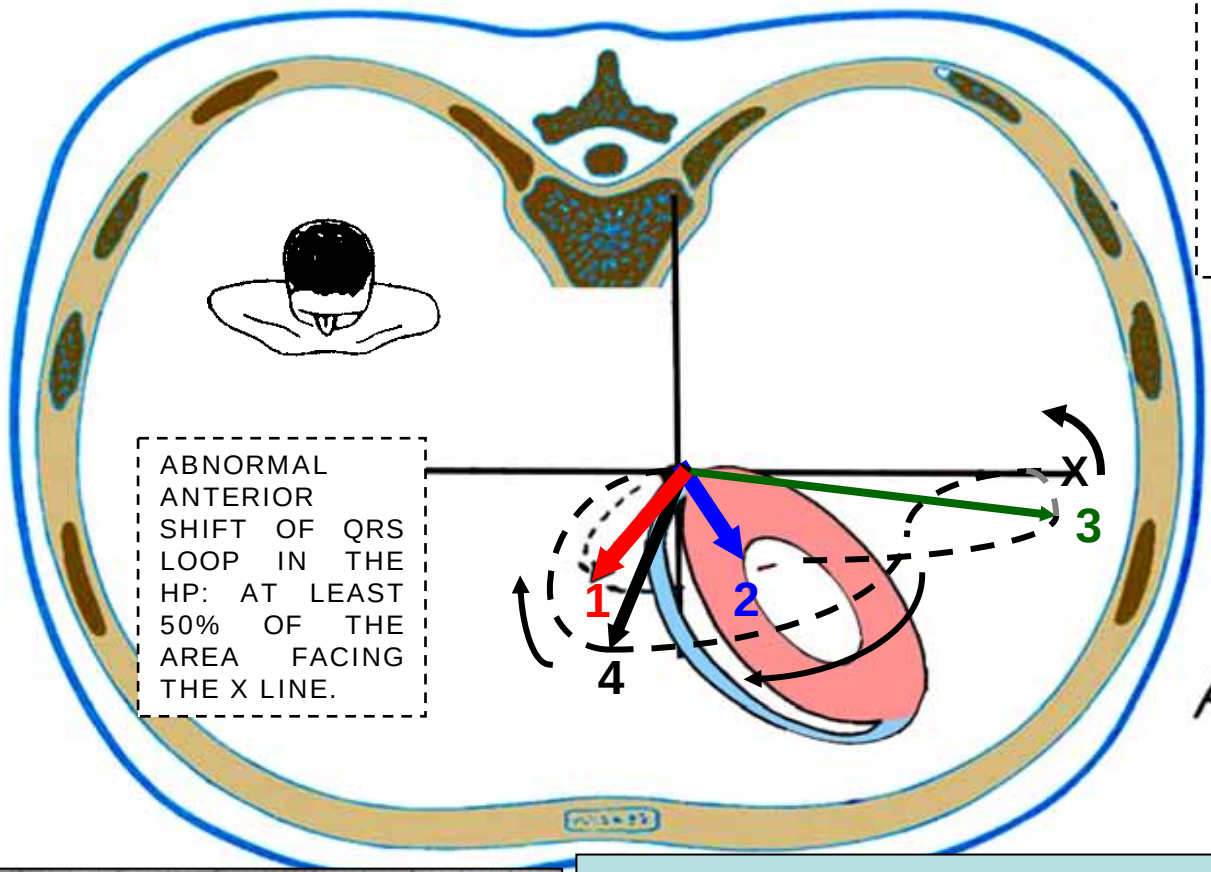


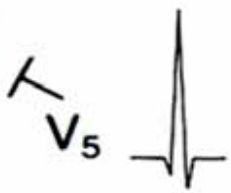
In acute inferobasal MI (antique strictly dorsal MI) an eventual rR', rSr', RSR' or rsR' pattern in V_3R, V_1-V_2: pseudo incomplete right bundle branch block or (pseudo IRBBB) or pseudo complete right bundle branch block pattern (CRBBB pattern) This pattern is present in 40% of cases of inferobasal MI (antique strictly posterior MD) such as observed in the present case. The differential diagnosis is easy because the final r` wave is not broad, differently from truly right bundle branch block.

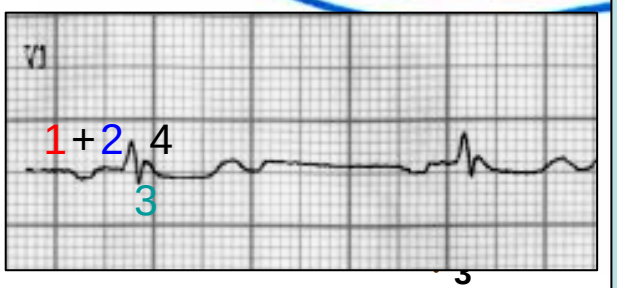
Vector 1 or septal vector and vector 2 or vector from of the low portion of the septum are not affected.(1+2). mild anterior dislocation of the vector 3 (vector of the free wall of ventricles) and finally is observed a significative anterior dislocation of vector basal 4. See explanation next slide

VECTORIAL REPRESENTATION OF VENTRICULAR ACTIVATION IN BASAL INFERIOR MI (ANTIQUE STRICTLY POSTERIOR MYOCARDIAL INFARCTION IN THE HP

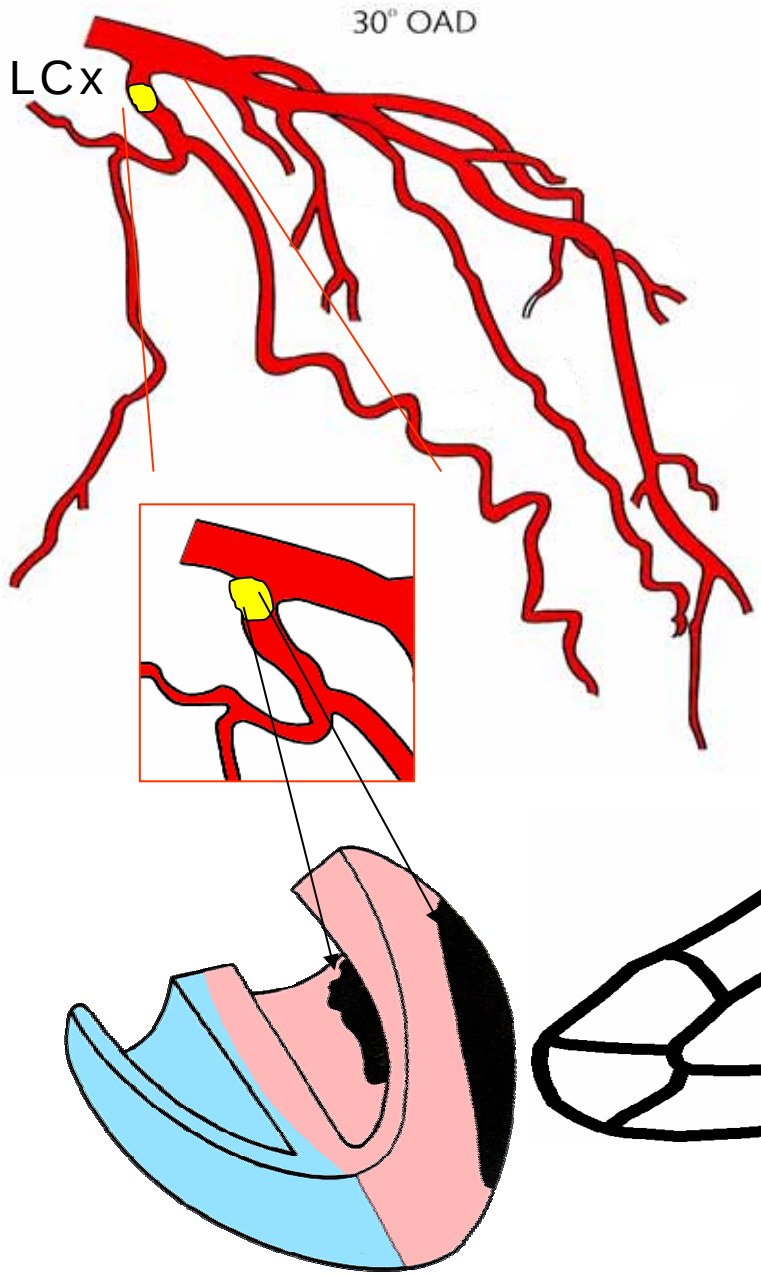
Basal inferior MI or antique strictly dorsal infarction affects only the middle and final portions of QRS loop between 30ms and 100 ms: The final second half of QRS loop;

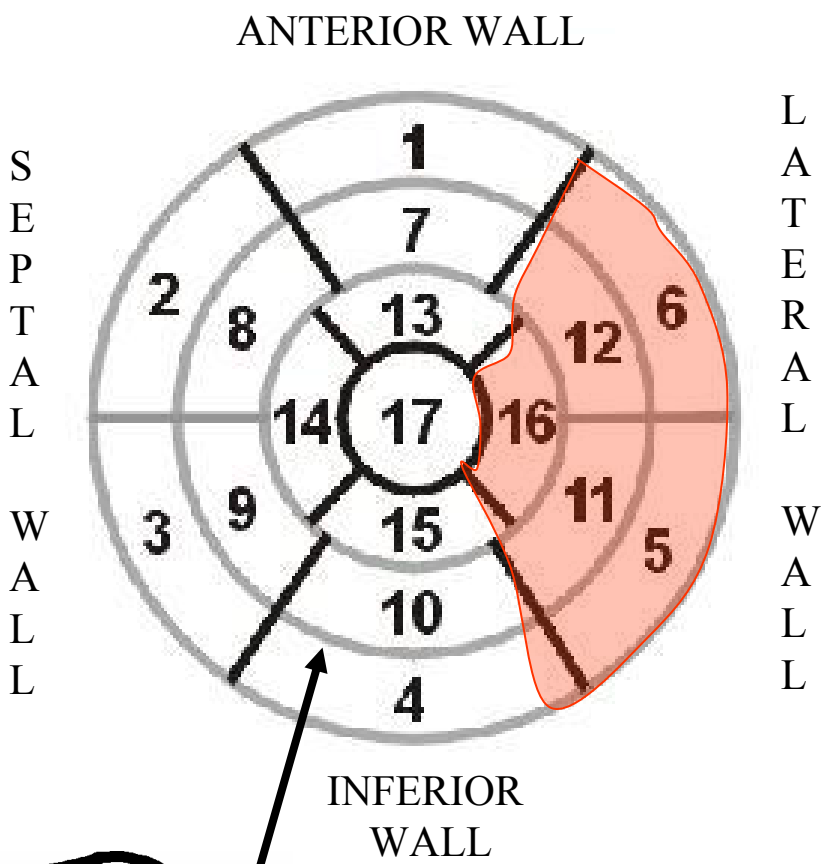






Vector 1, 1_{AM} vector or septal vector **and** vector 2 **or vector from of the low portion of the septum are not affected. (**1+2**) We observe a mild anterior dislocation of** vector 3 **(vector of the free wall of ventricles) and finally, a singnificative anterior dislocation of** vector 4 basal **is observed**

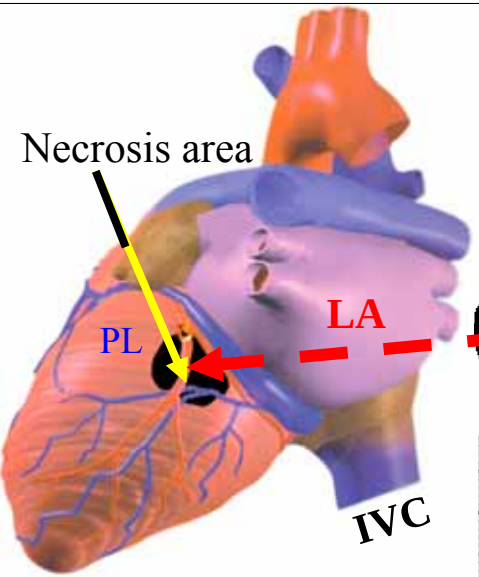


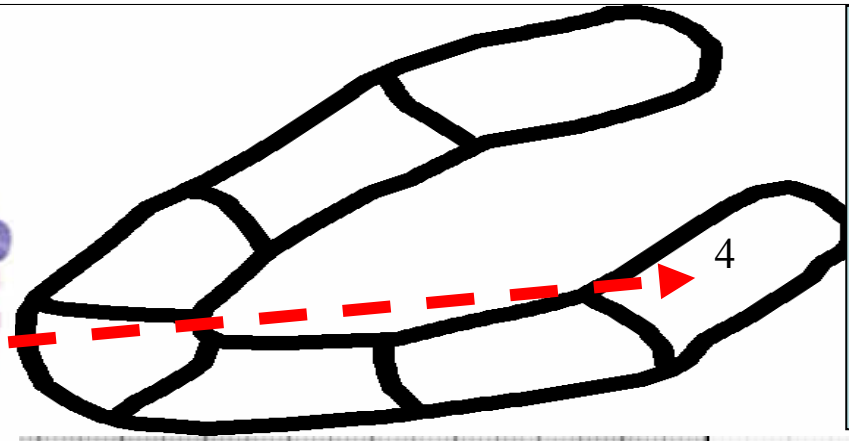


Segment 4 : Inferobasal segment

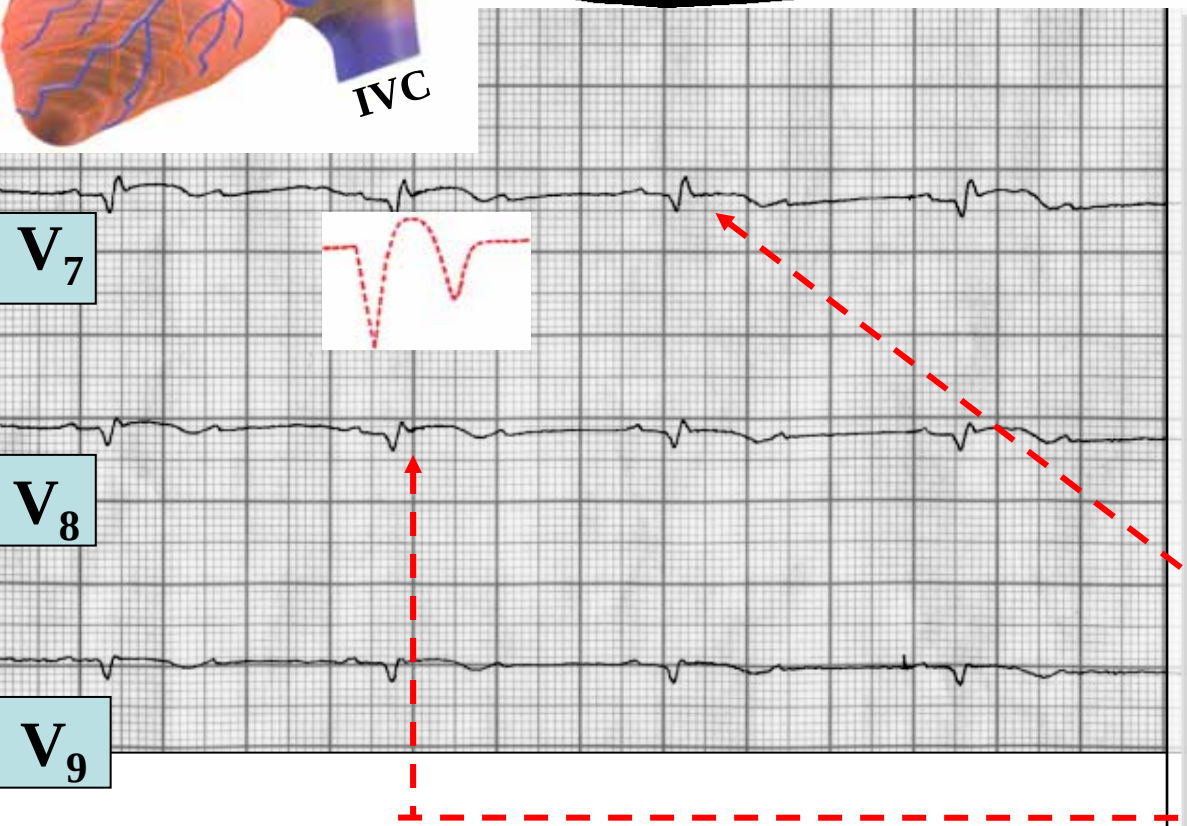
Old nomenclature:
strictly dorsal, posterior
myocardial infarction

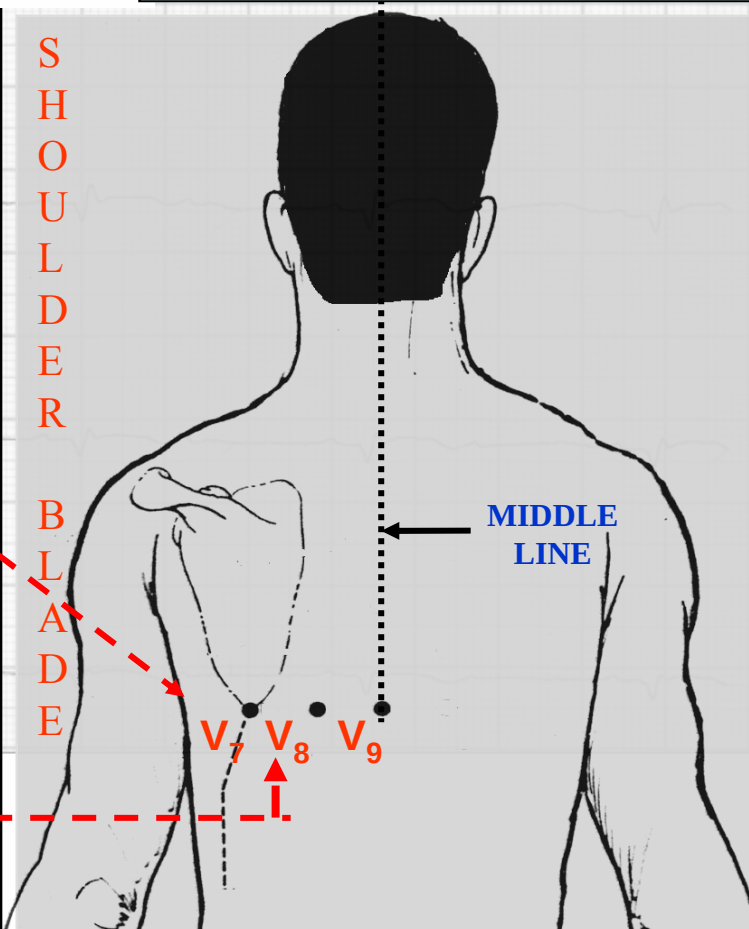
February 17/2012 20:20h First ECG dorsal accessory leads



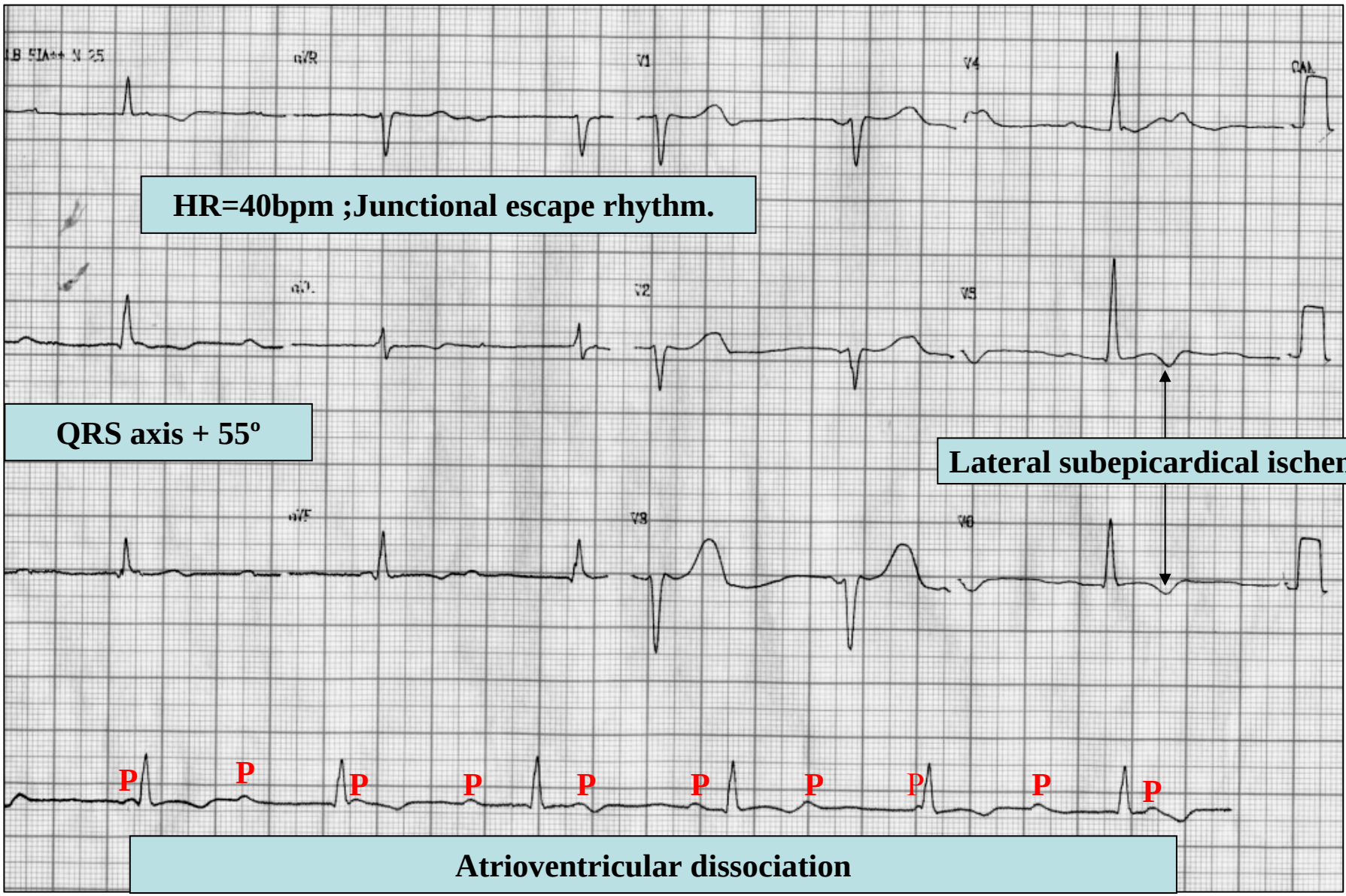


The lateral wall necrosis (dorsal or posterior necrosis in the antique nomenclature) would only be recorded in the accessory leads V_7, V_8 and V_9. however, V_2 V_3 (leads opposite the lateral wall (antique dorsal wall) record prominent R waves: mirror or reciprocal image of the events occurred in the lateral wall (antique dorsal wall.)

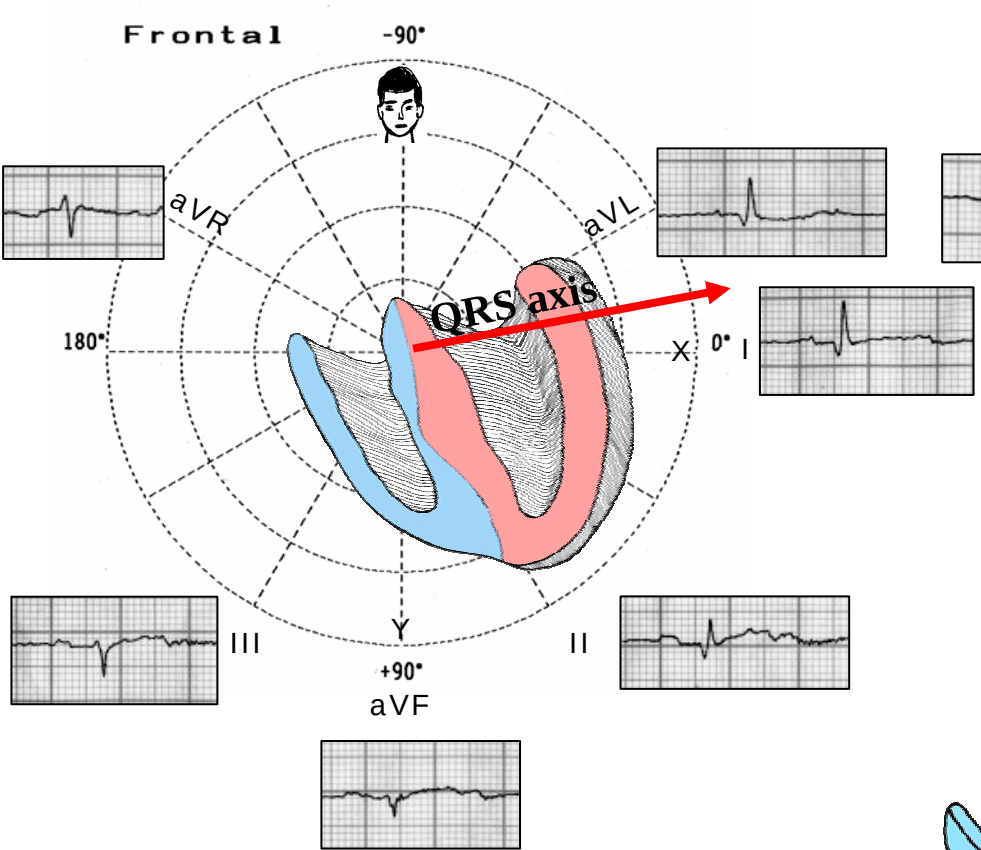




Second ECG preformed three hours later February 17/2012 23:00h

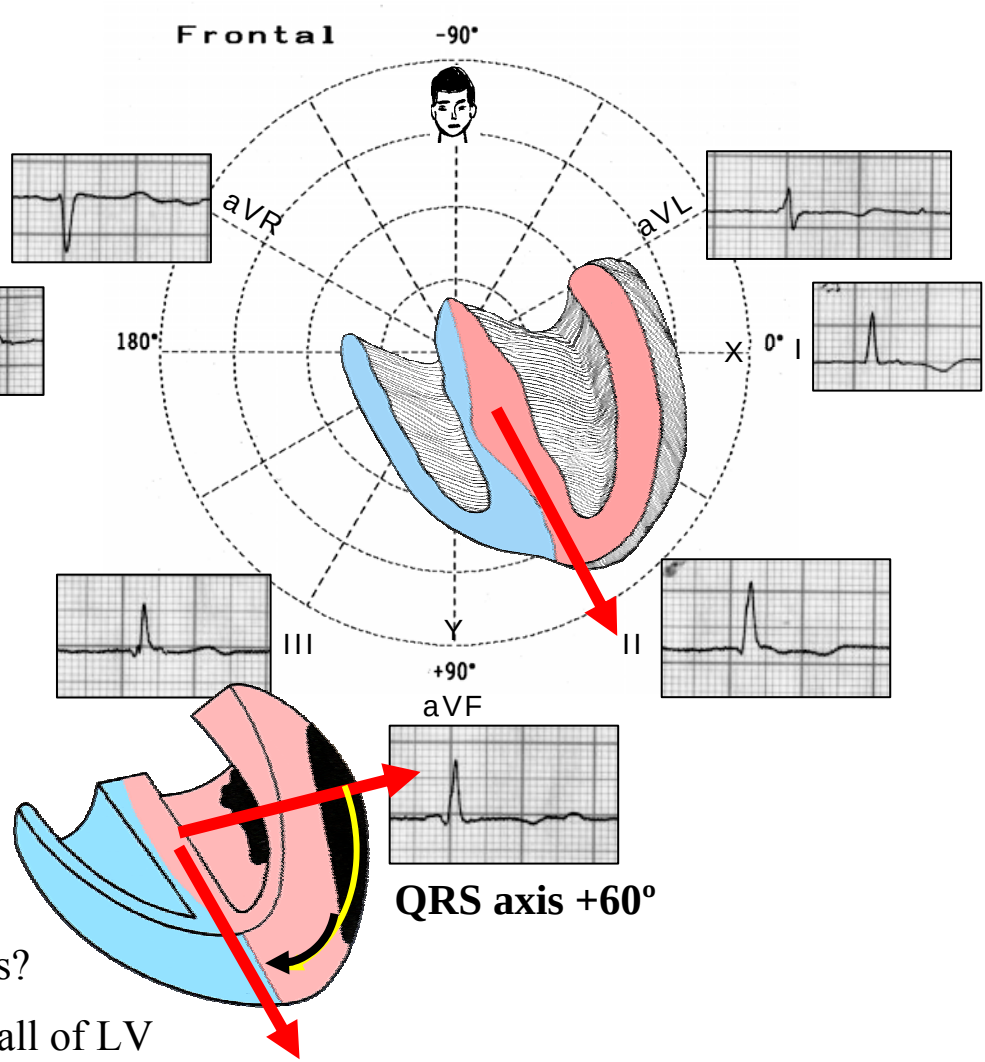


First admission ECG
February, 17-2012.
20:20h.



QRS axis -10º

Second ECG preformed three hours later February 17/2012 23:00h



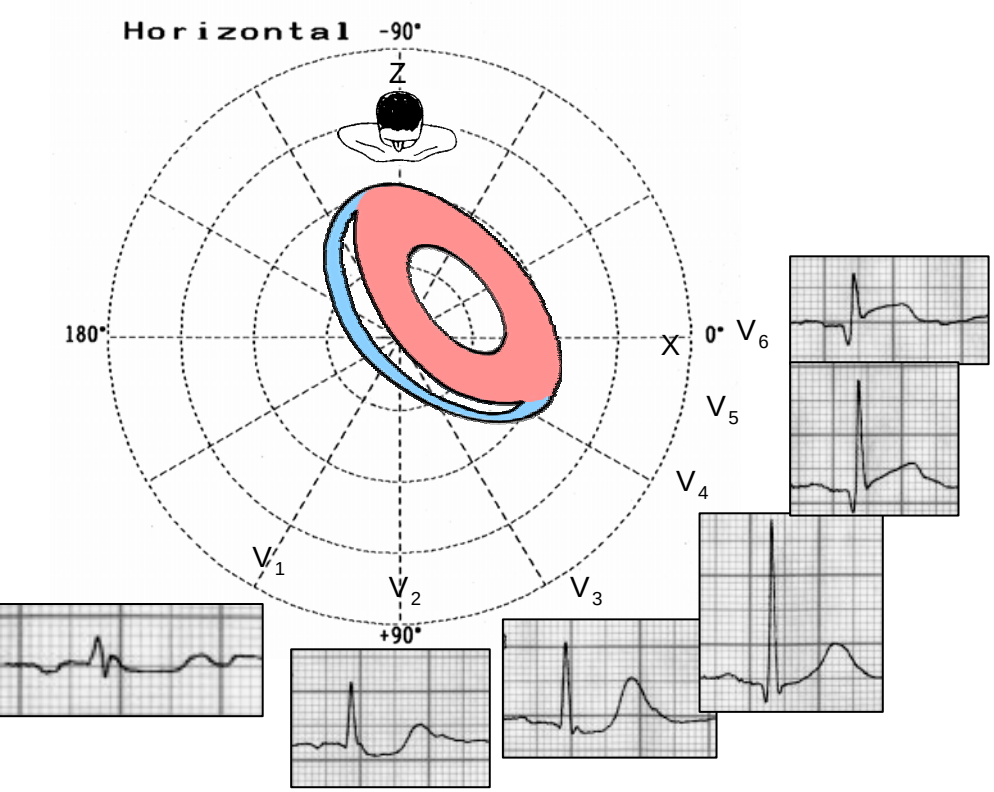
QRS axis +60º

Why QRS axis shift from -10º to +60° in only 3 hours?

Answer: because materialization of loss left lateral wall of LV

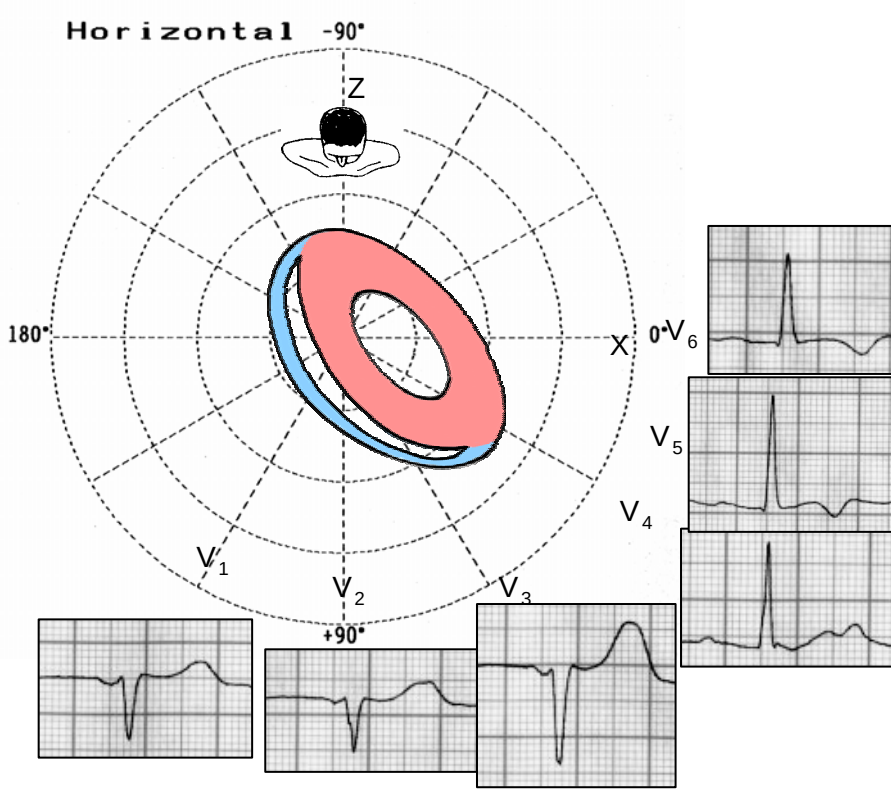
First ECG

February, 17-2012. ECG admission 20:20h.



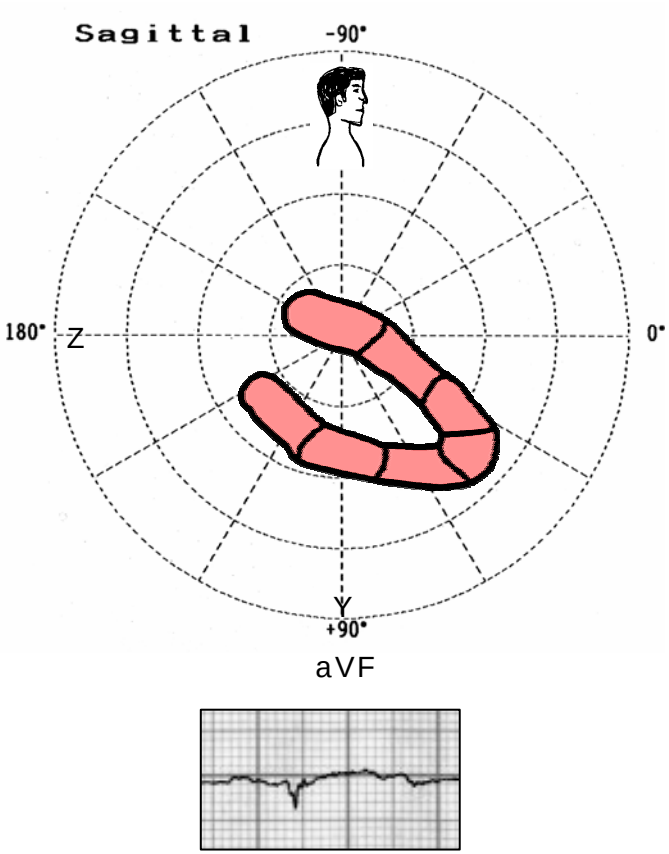
Second ECG

February 17/2012 preformed three hours later 23:00h



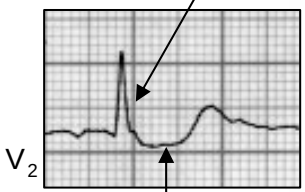
Heart rate **Heart rhythm** **QRS axis**	**HR = 44pbm** **Sinus** **-10º**	**HR=40bpm** **Junction escape rhythm. AV dissociation** **+60º**
PAF in V_2-V_3	**Present**	**Absent**
QRS pattern in V_1	**Pseudo IRBBB: tryphasic rsr´**	**rS**
Repolarization V_5-V_6	**Subepicardial injury**	**Subepicardial ischemia**
Reciprocal depressed ST segment	**Concave to the top V_2-V_3.**	**Absent**

First ECG admission
February, 17-2012. 20:20h.



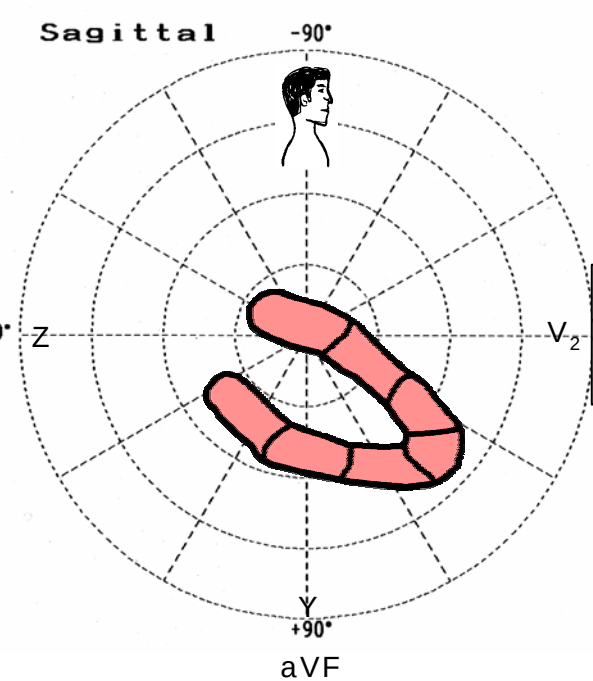
QRS axis -10º : negative QRS in aVF

PAF in V_2-V_3

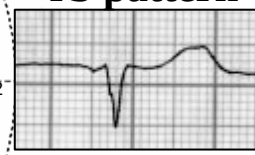


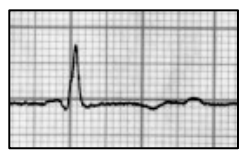
Reciprocal depressed ST segment

Second ECG
preformed three hours later
February 17/2012 23:00h



rS pattern





QRS axis +55º : positive QRS in aVF

The myocardial infarction consequence of obstruction of the LCx, involving primarily the basal segment of inferior wall (segment 4) and the lateral wall of the LV (segments 6, 5, 11, 12 and 16).
The electrocardiographic diagnosis of MI in this area is difficult and remains elusive. The sensitivity of standard ECG leads is less than 50% in angiographically documented occlusion of the LCx (antique strictly posterior infarction).
The accessory posterior chest leads (V_7- V_8 -V_9) improve sensitivity.
The electrocardiographic criteria of myocardial infarction consequence of obstruction of the LCx, lateral and basal inferior MI (antique posterior myocardial infarction) are:

1. Abnormally tall or broad initial R waves, or both, in lead V_1-V_2 and V_3R
2. R-wave in lead V_1-V_2 width ≥ 40 ms or 0.04 s (broad);
3. R wave ≥ 7 mm in lead V_1;
4. R/S ratio ≥ 1 in lead V_1 (present in 35% of cases);
5. R/S ratio ≥ 1.5 in lead V_2 or R/S ratio ≥ 1 in V_2.
6. Frequent slurring of the descending limb of R wave in lead V_1;
7. Eventual rR', rSr', RSR' or rsR' pattens in V_3R, V_1-V_2: pseudo incomplete right bundle branch block or (pseudo IRBBB) or pseudo complete right bundle branch block pattern (CRBBB patterns) (present in 40% of cases); Such as observed in the present case.
8. Abnormal Q waves (≥ 40 ms or 0.04 s) in accessory additional dorsal leads V_7-V_9 (paraspinal leads). Routine recording of leads – V_7, V_8 and V_9 has been recommended in patients with suspected IM, but with nondiagnostic 12-lead ECG. Basal inferior MI (antique dorsal MI) may be recognized directly just by the accessory leads located between the left shoulder blade and the spine: leads V_7, V_8 and V_9. Dr. Zalenski, Professor of Emergency Medicine of the Wayne State University of Detroit, published that using ECG with 15 leads with V_4R, V_8 and V_9 routinely, significantly increases sensitivity to diagnose coronary syndromes, especially to detect dorsal MI (*actual basal inferior MI*).(1)

1. Zalenski RJ, Cooke D, Rydman R, et al. Assessing the diagnostic value of an ECG containing leads V4R, V8, and V9: the 15-lead ECG. Ann Emerg Med 1993; 22:786-793.

9. Reciprocal depressed ST segment (concave to the top) in acute phase in lead V_1 through V_3.
10. Isolated ST-segment depression or nonsignificant ST-segment depression in the standard leads is observed in 26% and 28% respectively. The infarct size – as measured by maximum CK-values – did not differ among the respective groups (*1;2*).
11. Isolated ST elevation in posterior chest leads V_7 through V_9 identifies patients with acute basal inferior MI wall (antique posterior wall MI.). Early identification of these patients is important for adequate triage and treatment of patients with ischemic chest pain without ST on standard 12-lead ECG (*3*).
12. Posterior chest leads should be routinely recorded in patients with suspected MI and nondiagnostic, routine 12 lead-ECG. This simple bedside technique may help proper treatment of some of these patients now classified as having unstable angina or non-Q-wave MI, because criteria for reperfusion therapy in AMI require the presence of ST elevation in 2 contiguous leads (*4*).
13. Positive, tall, and symmetrical T waves in right precordial leads. Abnormal T-wave shift is present in over 70% of the patients with inferobasal MI (antique posterior MI) and is clearly discernible from the 12-lead

1. Sclarovsky S, Topaz O, Rechavia E, et al.: Ischemic ST segment depression in leads V2 -V3 as the presenting electrocardiographic feature of posterolateral wall myocardial infarction. Am Heart J 1987; 113:1085-1090.
2. Schmitt C, Lehmann G, Wailersbacher M, et al. Problems of electrocardiographic diagnosis of occlusion of the left circumflex coronary artery Dtsch Med Wochenschr. 2001; 126:1257-1260.
3. Matetzky S, Freimark D, Feinberg MS, et al. Acute myocardial infarction with isolated ST-segment elevation in posterior chest leads V7-9: "hidden" ST-segment elevations revealing acute posterior infarction. J Am Coll Cardiol. 1999; 34:748-753.
4. Agarwal JB, Khaw K, Aurignac F, et al. Importance of posterior chest leads in patients with suspected myocardial infarction, but nondiagnostic, routine 12-lead electrocardiogram. Am J Cardiol. 1999; 83:323-326.

ECG, as manifested by tall T waves in lead V_2 and flat T waves in lead V_6. T2-T6 index: it is estimated by subtracting the amplitude of T wave in lead V_6 from its amplitude in lead V_2. An index of 0.38 mV or more yielded a sensitivity of 81% and a specificity of 75% for inferobasal or posterior IM; however, this is not as discriminating as the VCG, where a T angle of 60º or more in the HP yielded a sensitivity of 70% and a specificity of 97% (*1*).Combining the T-wave index with lead V_9 further enhanced the diagnostic yield: the sensitivity for detecting posterior MI by at least one of these criteria was 78%, and when both criteria were positive, specificity was 98.5%. A single, unipolar posterior lead in the V_9 position is superior to standard 12-lead ECG criteria in diagnosing remote inferobasal or posterior myocardial infarction, and combining V_9 with the T-wave index maximizes the diagnostic yield. Posterior paraspinal lead V_9 provided the best overall predictive accuracy (94%), positive predictive value (58%), and ability to differentiate patients with and without inferobasal or posterior MI of any single criterion (*2*);

Kanemoto et al(3) criteria for patients with LCx-related MI in AMI without tented T waves or definite ST elevation: 1) ST segment depression $\geq$ 1 mm in 2 consecutive chest leads, 2) prominent positive U wave $\geq$ 1 mm in leads V_2 or V_3, 3) T/U ratio in leads V_2 or $V_3 \geq 4$. Considering two of the above criteria as positive, the sensitivity is 71.9%, the specificity 97.0%, and the diagnostic accuracy 88.8%. In 85.2% of the patients, ST depression returned to the baseline by 24 hours. As the amplitude of the U waves decrease gradually, the T/U ratio increased. The R/S ratio in leads V_1 or V_2 became $\geq$ 1 mm by 24 hours in 46.4% and the amplitude of R wave in lead V_1 increased gradually. T waves in the right precordial leads increased with time. These findings are consistent with isolated basal inferior or strictly posterior MI. From these results the authors identified new ECG criteria: R/S ratio in leads V_1 or $V_2 \geq 1$; R wave $\geq$ 7 mm in lead V_1;T wave $\geq$ 0.5 mV in lead V_1.

1. Eisenstein I, Sanmarco ME, Madrid WL, et al. Electrocardiographic and vectorcardiographic diagnosis of posterior wall myocardial infarction. Significance of the T wave. Chest. 1985; 88:409-416.
2. Rich MW, Imburgia M, King TR, et al. Electrocardiographic diagnosis of remote posterior wall myocardial infarction using unipolar posterior lead V9. Chest. 1989; 96:489-493.
3. Kanemoto N, Wang Y, Fukushi H, et al. Electrocardiographic characteristics of patients with left circumflex-related myocardial infarction in the acute phase without tented T waves or definite ST elevation. J Cardiol. 1995 Sep;26:149-158.

Considering any of the above criteria as positive, the sensitivity is 72.0%, specificity 87.9%, and diagnostic accuracy 86.7% on the 14th day. These ECG criteria of lateral MI (antique strictly posterior MI) with the LCx as an infarct-related coronary artery apply at less than 6 hours or at 24 hours from the onset of the symptoms

Eventually QRS complex of low voltage in the frontal plane (< 5 mm). This criteria is very important for the differential diagnosis with left septal fascicular block (LSFB).

Non shifted QRS axis in the frontal plane (normal). This criterion is important for the differential diagnosis with right ventricular hypertrophy/enlargement.

Absence of Q wave, increase of R wave in V_1 and V_2, and ST segment depression from V_1 to V_4 (anterior wall) reflect the mirror or reciprocal image of the events of the basal inferior wall (antique dorsal wall.)

ST segment elevation in V_6 and in the inferior wall point out apex-inferior subepicardial injury.

Vectocardiographic criteria of the lateral MI (antique dorsal infarction)

1. Anteriorly shifted QRS loop in the horizontal plane (HP): at least ½ (≥50%) of QRS loop area located on anterior quadrants *

2. Affectation only the middle and final portions of QRS loop between 30 and 100 ms, i.e. the second half of QRS loop.

3. The first septal vector, 1_{AM} vector or septal vector (corresponds to the activation of the middle third of the left septal surface) and the second vector or vector 2 (vector of the low portion of the septum) are not affected.

4. Mild anterior shift of the vector of the free wall (vector 3) observed and important anterior shift of vector 4 (basal). This explains the triphasic pattern (pseudo IRBBB pattern) observed in V1 and V2.in 40% of cases.

5. Presence of a QRS loop mostly anterior to point E (end of QRS loop)

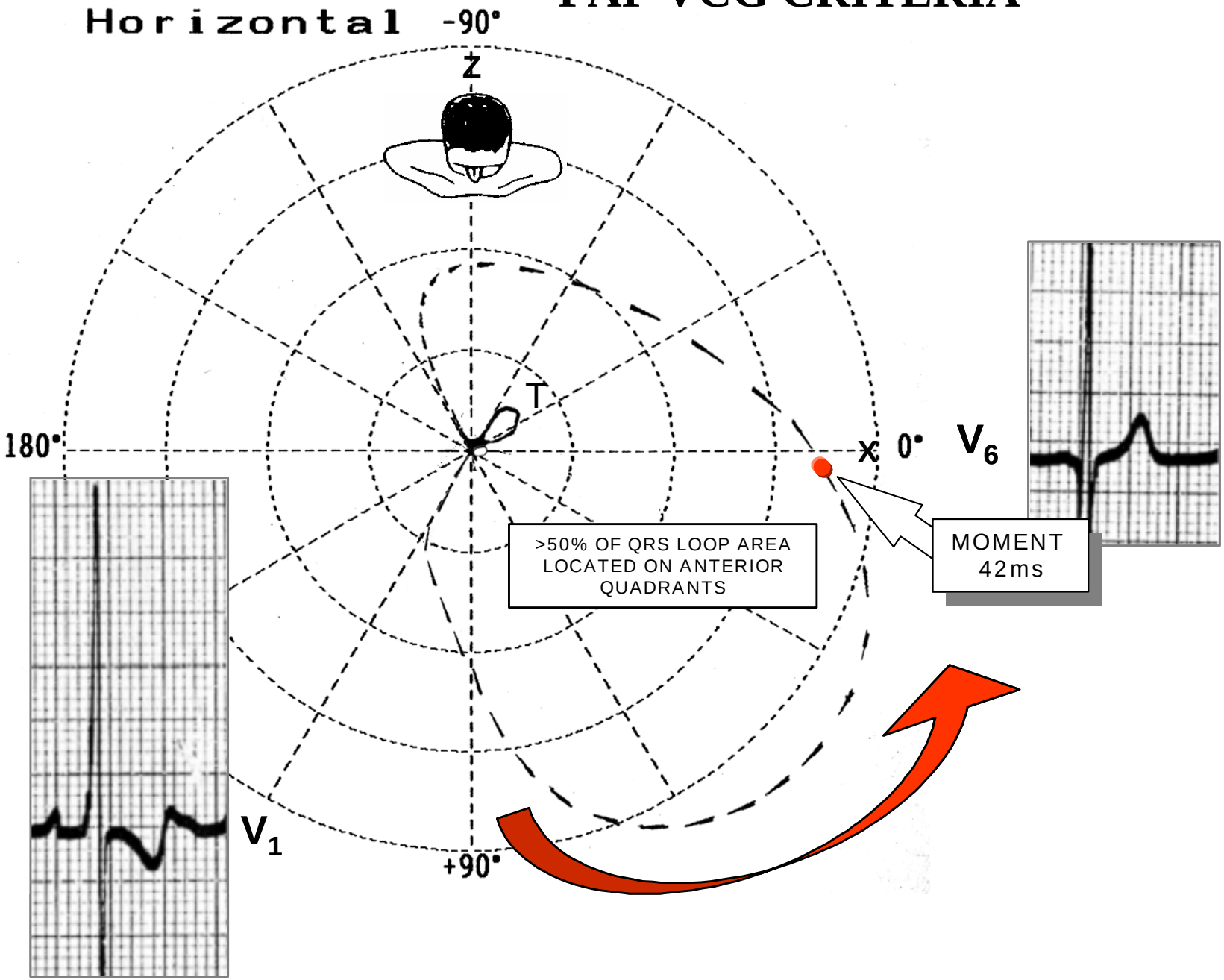
6. Total duration of QRS anterior forces ≥42 ms*

7. Location of the half-area vector at +10º or more anterior orthogonal X lead (0º to ±180º)

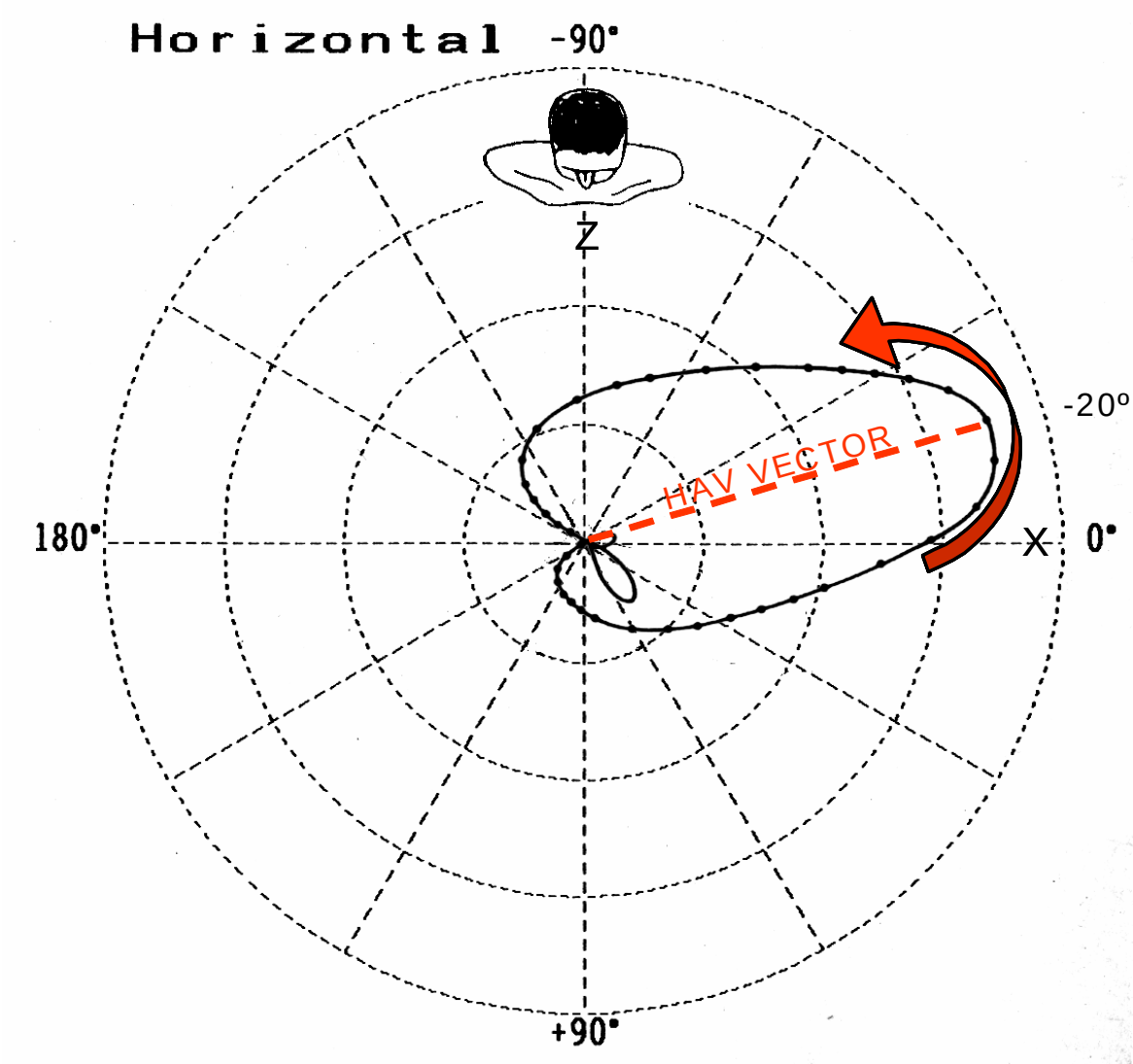
8. Time required for the peak anterior voltage to be recorded (anterior accession time AAT) ≥30 ms

* Observation: Vectocardiographically we consider that there is prominent anterior forces (PAF) when the vector of the 42 ms moment of the QRS loop of the HP, is located in the anterior quadrants, or when ≥50% of the area of the QRS loop is in the anterior quadrants (to the front of the orthogonal X lead) (0º to ±180º)

PAF VCG CRITERIA

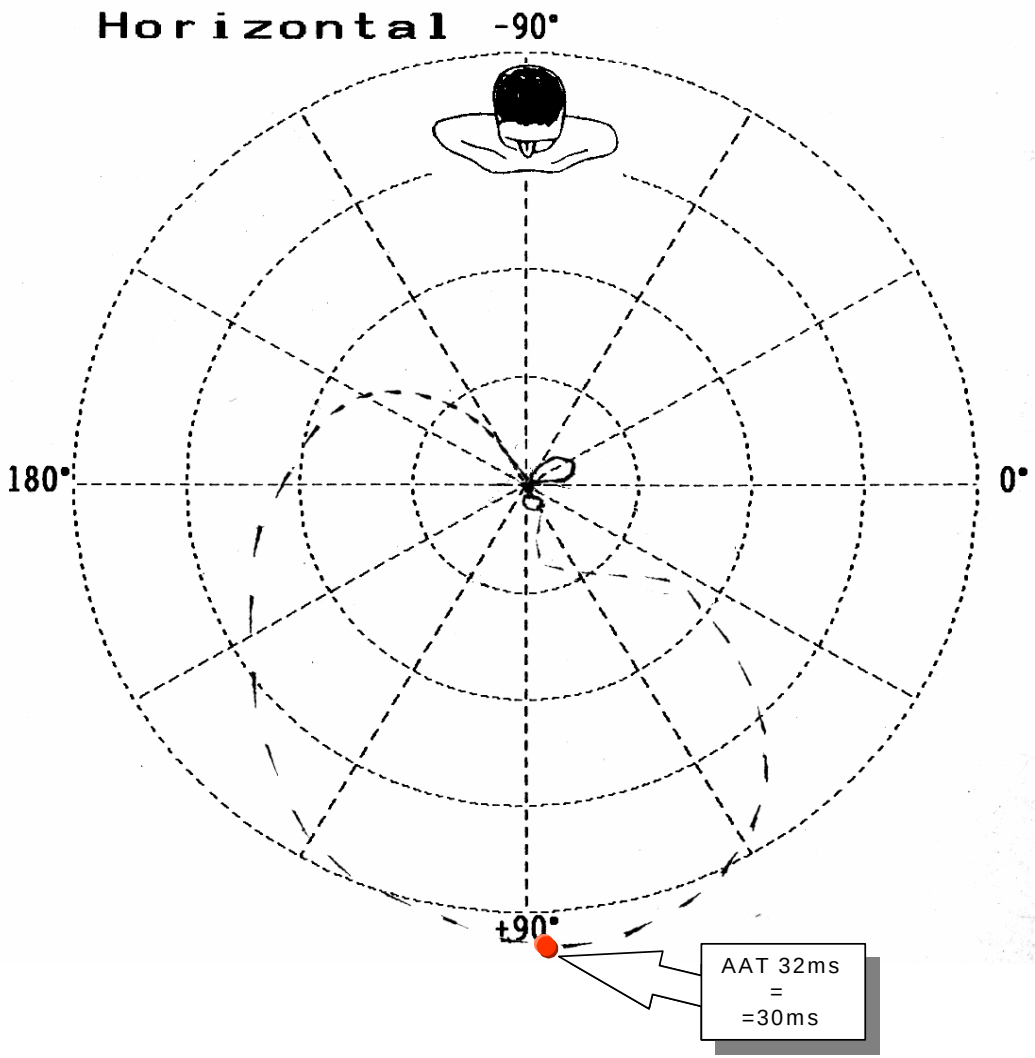


HALF-AREA VECTOR: HAV



We consider the presence of PAF when **HAV** is located anteriorly to +10º. In normal conditions, HAV is located in the left posterior quadrant, around -20º; however, it may be in the left anterior quadrant in normal individuals too.

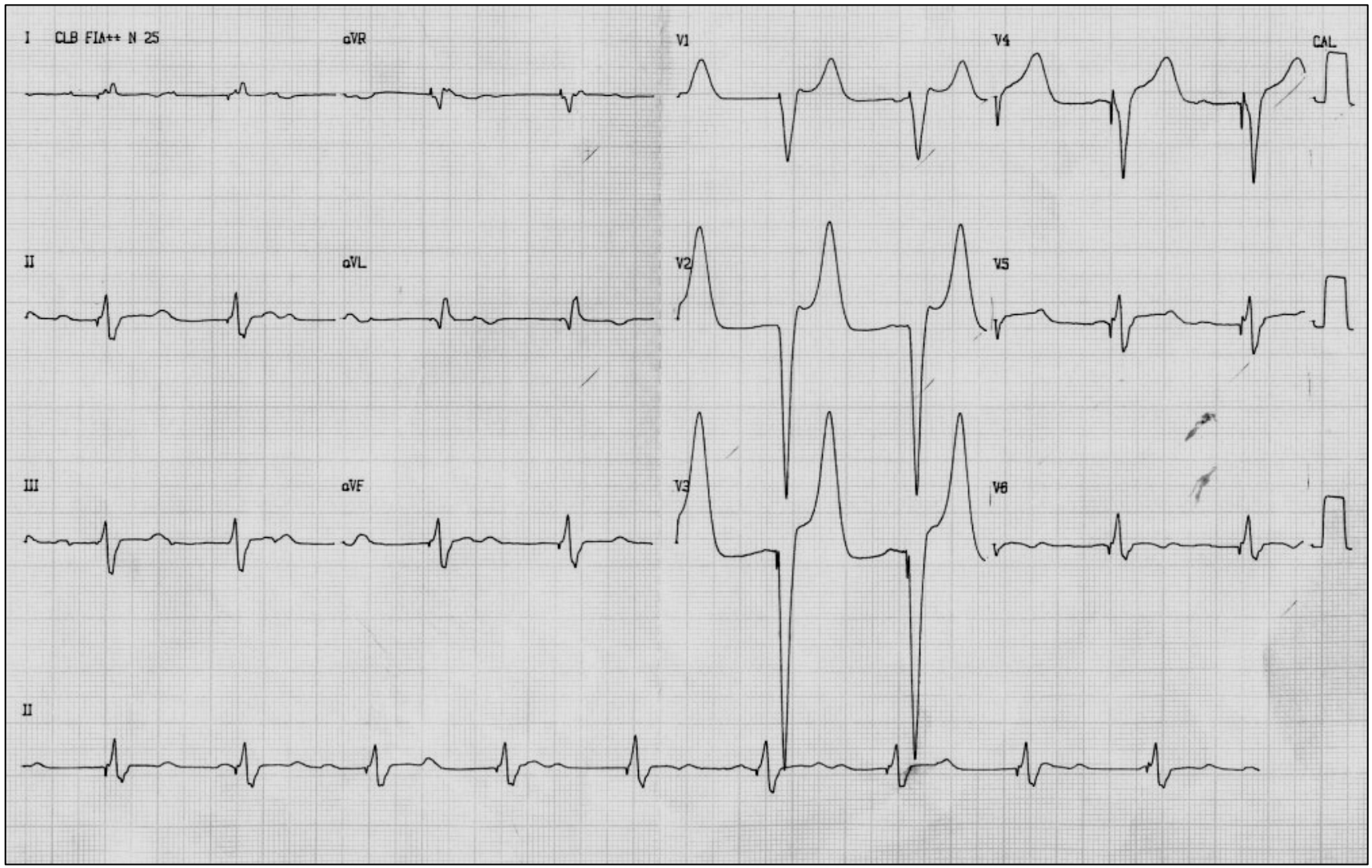
ANTERIOR ACCESSION TIME (AAT)



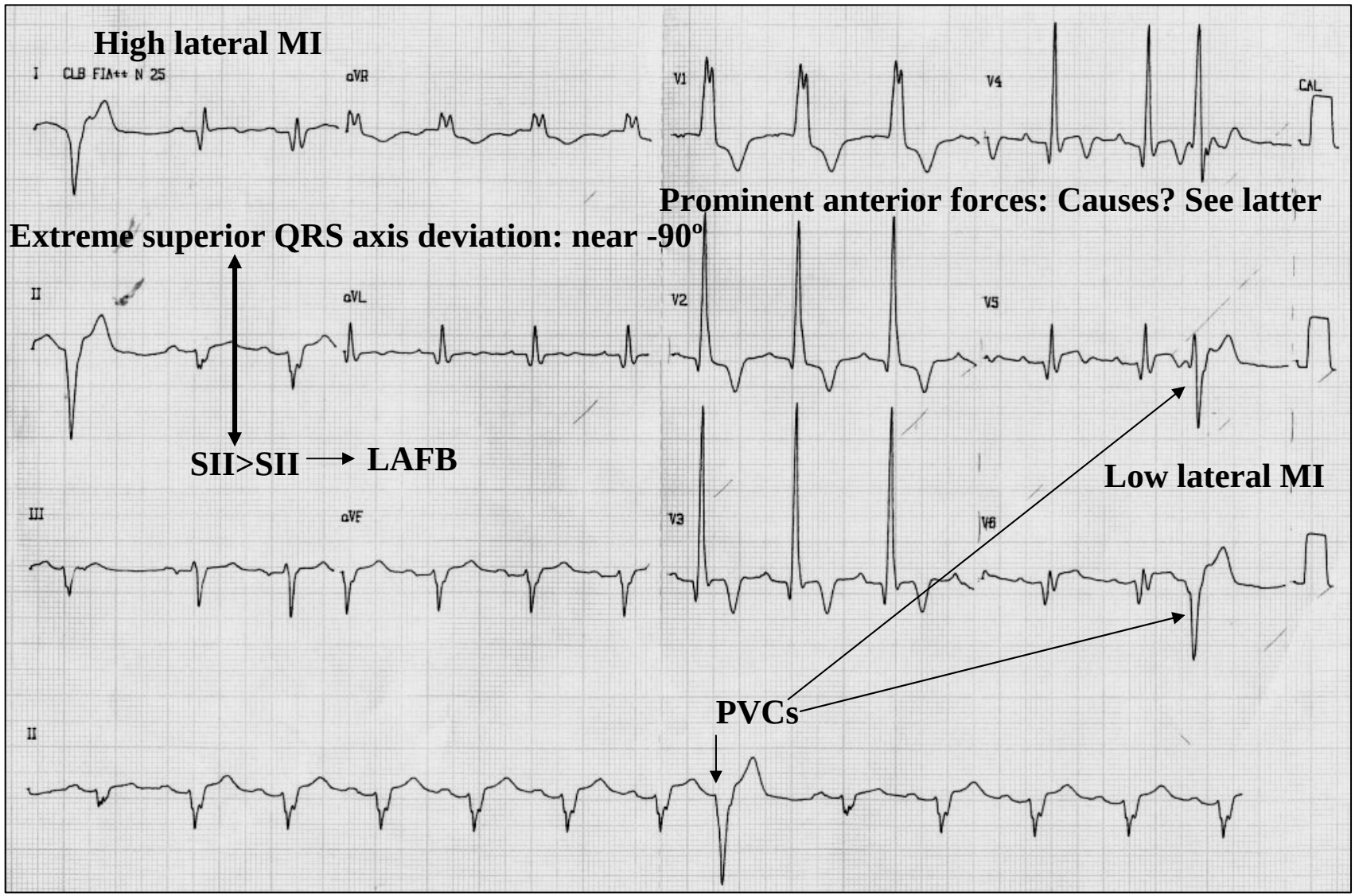
The time required for the peak anterior voltage to be recorded (Anterior Accession Time). AAT ≥30 ms = PAF. It is defined as the time elapsed between point 0* and the most anterior point of the QRS loop. In normal conditions, it is <30 ms. Times ≥30 ms are a criterion for PAF.

AAT ≥30 ms may be observed in antique strict posterior MI.(Atual lateral MI)

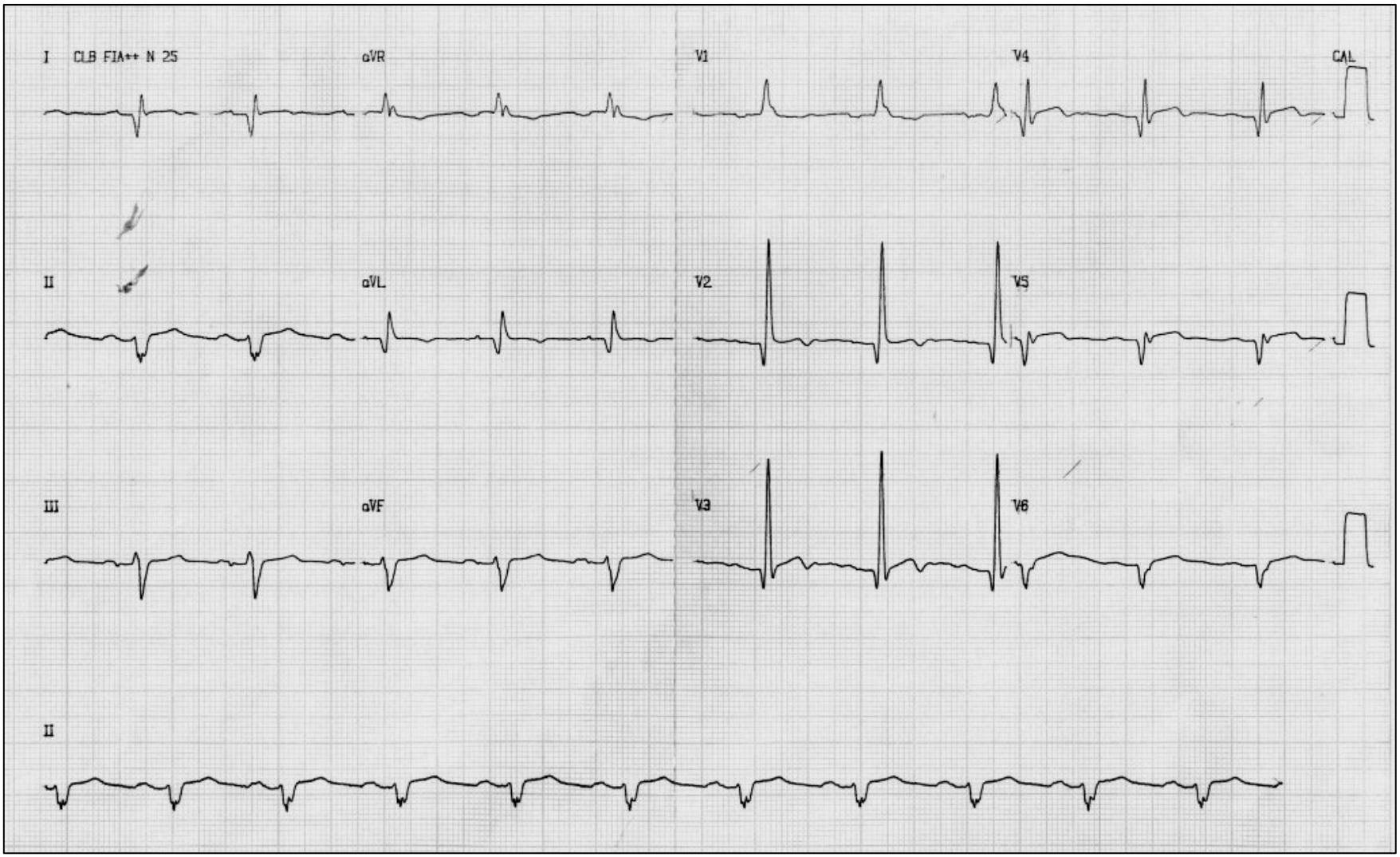
Third ECG February 18/2012 (01:15 h)immediately Pacing implantation associated Primary Percutaneous Coronary Intervention (PCI) on left circumflex artery (culprit artery)



February 23/12 08:00h. With the pacemaker off. Fifth ECG



February 25/12 09:00h Sixth ECG



Prezados Andrés e Raimundo e demais queridos companheiros do Foro:

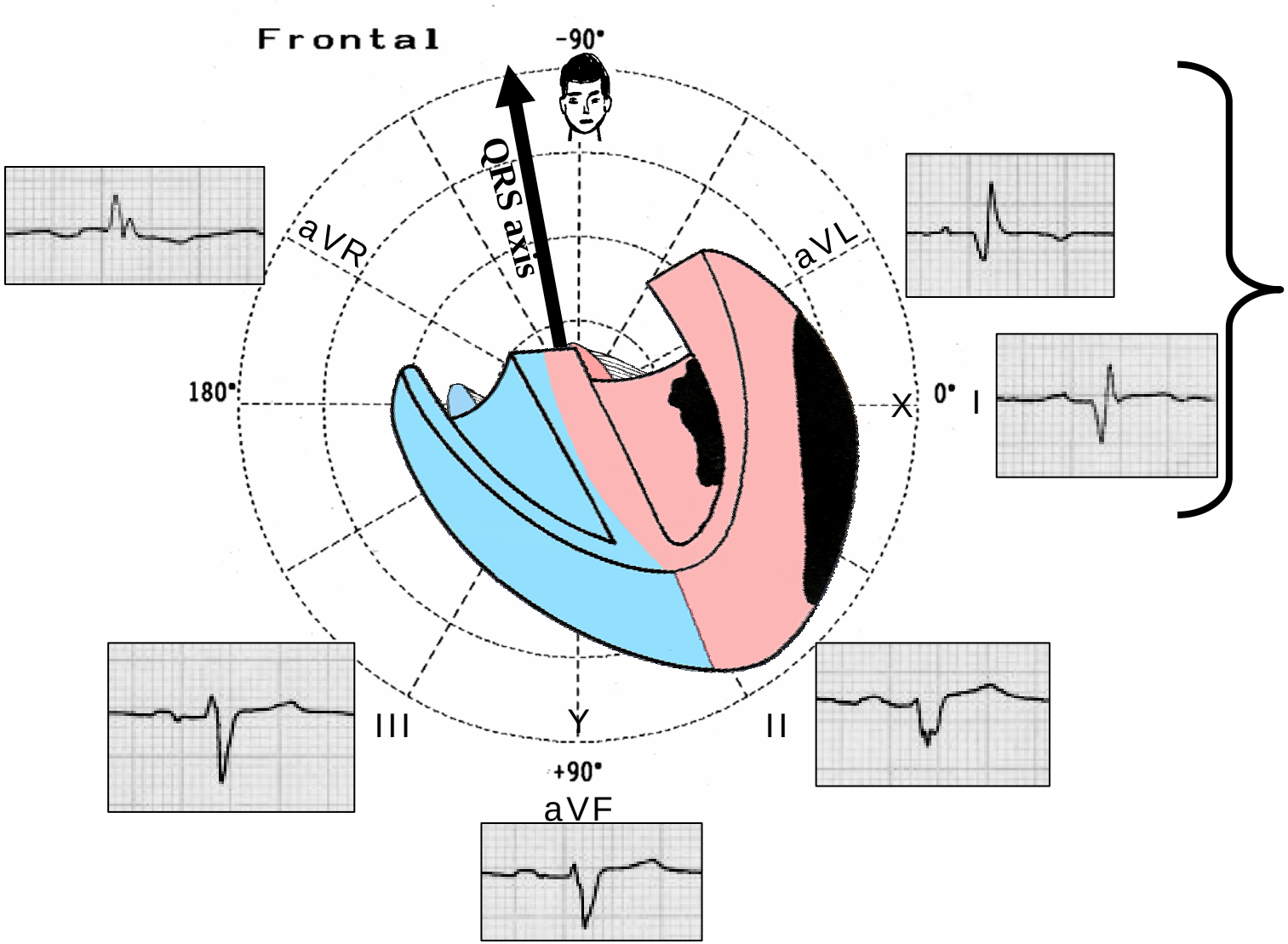
1º ECG = 1. Bradicardia Sinusal(44bpm) 2. Infarto Lateral (antigo dorsal estrito: qR V5,V6 ST supra V5,V6 confirmado com o 2º ECG 3. Bloqueio de Ramo Direito (V1 = RSr´0,12" V2 = rsR) 4.ST supra V5,V6 - 4. ST infra V3, V4 com T alta pontiaguda)
2º ECG = qr V7-V9 c/ supra de ST - Infarto lateral(antigo dorsal)
3º ECG = BAV total Ritmo Hissiano c/ Tinvertida D1, aVL, V5, V6
4º ECG = Sob Estimulação artificial padrão esperado
5º ECG = Ritmo sinusal s/ marca-passo. Bloqueio do Ramo Direito com novo padrão (RR´ em)V1. Infarto lateral: qRr V5, aVL Qrs V6, D1. Extra-sístoles ventriculares de origem no VE Extra-sistoles supraventriculares.(Reperfusão incompleta)
6º ECG = 1. BRD. 2 . Infarto lateral. 3. FAP

Comentários:

1. Forças anteriores prominentes são devidas ao infarto lateral. Bayés de Luna acredita que existe suficiente corpo de doutrina para concluir que a onda R de voltagem aumentada em V_1 se origina por infarto da parede lateral e não da posterior, e que na maioria das instâncias esta última parede no existe
2. Que o chamado infarto de parede lateral alta (Q em DI e a VL), obedece a infarto de parede médio-anterior.
3. Que a denominação de infarto apical (apical-anterior) é muito mais adequada que infarto ântero-septal para aqueles infartos com padrão QS de V1–V4..

Bloqueio AV e os Bloqueios de Ramo D: Irrigação do sistema de condução(A porção proximal do ramo direito e o feixe de His estão irrigados pela artéria do Nó A-V da coronária direita (CD) e pela primeira perfurante septal da descendente anterior (DA). Eventualmente, o ramo direito na sua porção média está irrigado por: ramos septais da artéria descendente posterior (DP), da segunda perfurante septal da DA e a artéria de Kugel, ramo da circunflexa (Cx). A porção média e distal do ramo direito estão irrigadas pelo "ramus limbi dextri" ramo da segunda perfurante septal da DA.
Adail - Bahia - Brasil

February 25/12 09:00h Sixth ECG Frontal Plane

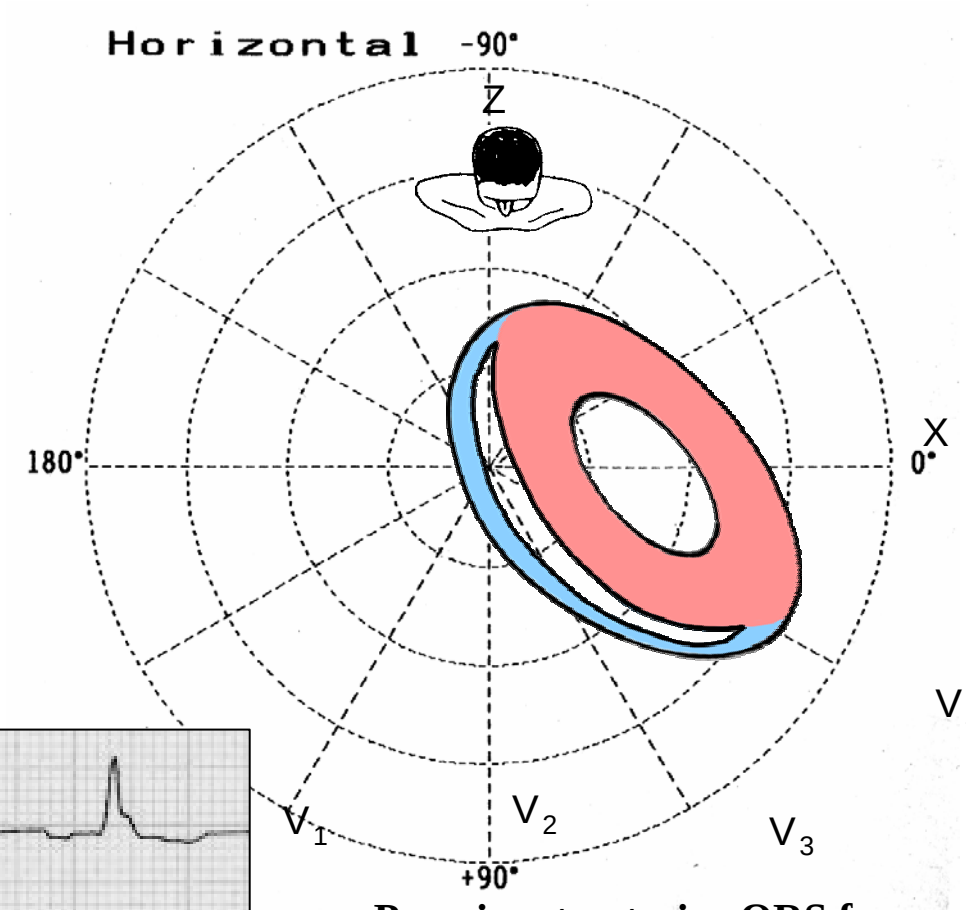


The QR morphology in lead aVL is due to lateral MI. (Antique high lateral MI)

rS pattern in inferior leads, SIII>SII, notched in ascending ramp on II S wave, extreme superior axis QRS deviation (QRS axis on right superior quadrant) QR or Qr pattern in I and aVL

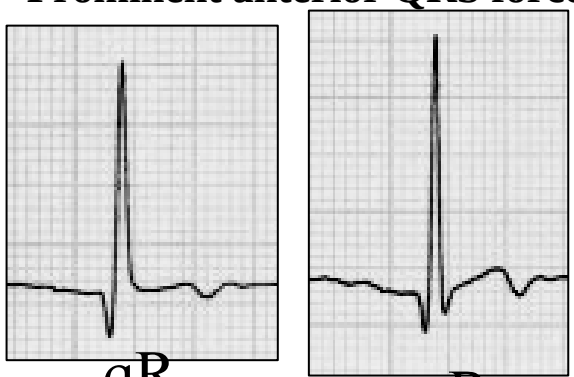
Conclusion: Left anterior fascicular block associated with lateral myocardial infarction

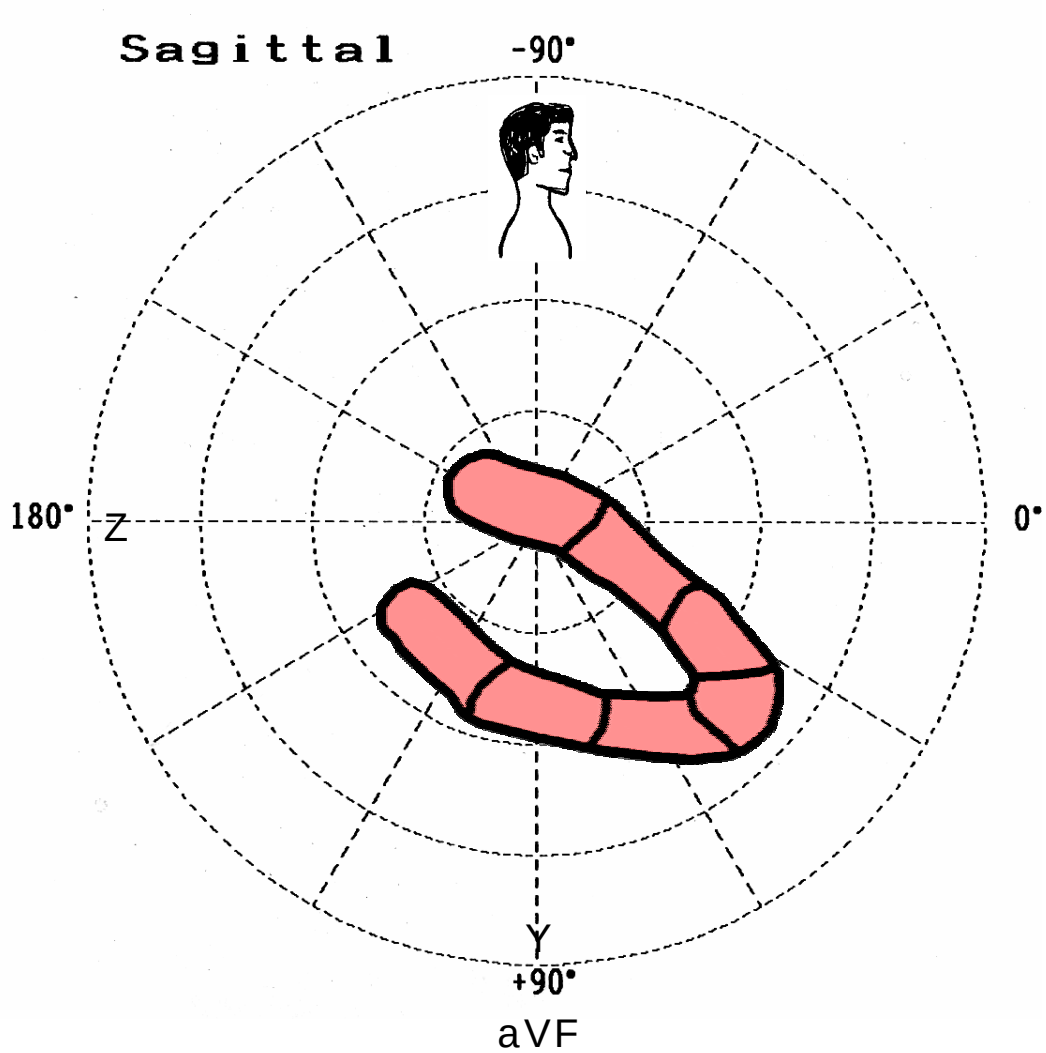
February 25/12 09:00h Sixth ECG Horizontal Plane



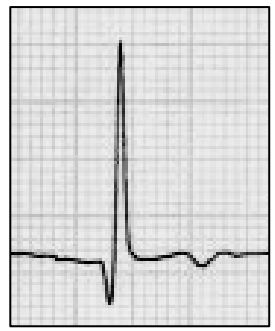
Low lateral MI

Prominent anterior QRS forces





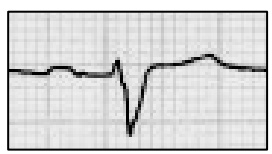
PAF



qR pattern
with narrow final R wave

Rule out RBBB

RBBB has broad
final R or R´ wave.



Final diagnosis of sixth ECG

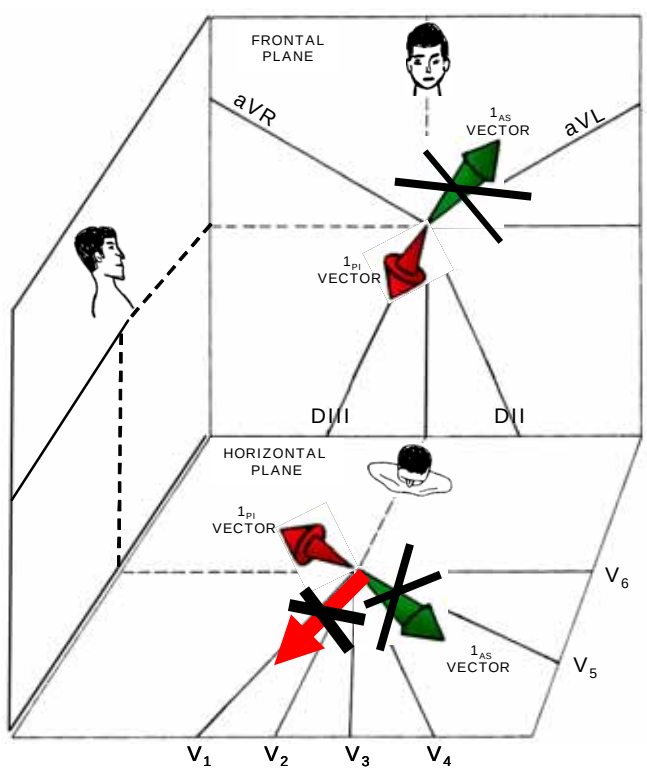
1) Left Anterior Fascicular Block (LAFB): because extreme superior axis deviation, SIII>SII. LAFB was inexistent in the first and second ECGs.
2) Lateral Myocardial infarction: QR, Qr or QS pattern in left leads I, aVL, V_5-V_6 and PAF.
3) Prominent Anterior QRS Forces (PAF): Cause? or Causes?

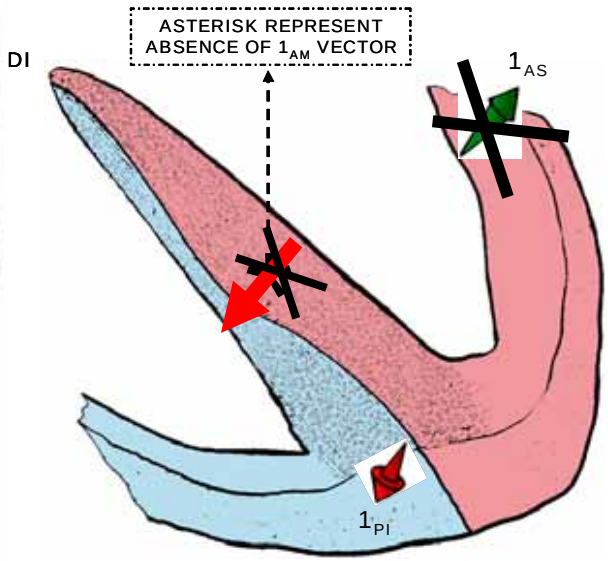
Which is the cause or causes of PAF in this ECG?
Answer: as demonstrated Professor Bayés de LunaQ-wave myocardial infarction based on correlations with contrast-enhanced cardiovascular magnetic resonance (CE-CMR), the presence of the RS morphology in lead V1 PAF is consequence of lateral myocardial infarction. Therefore, the terms posterior and high lateral infarction are incorrect and should be changed to lateral wall and limited anterolateral wall MI.(**1;2;3;4**)

1. **Bayés de Luna A. New heart wall terminology and new electrocardiographic classification of Q-wave myocardial infarction based on correlations with magnetic resonance imaging. Rev Esp Cardiol. 2007 Jul; 60:683-689.**
2. **Bayés de Luna A, Zareba W. New terminology of the cardiac walls and new classification of Q-wave M infarction based on cardiac magnetic resonance correlations. Ann Noninvasive Electrocardiol. 2007 Jan;12:1-4.**
3. **Bayés de Luna A, Wagner G, Birnbaum Y, et al. 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;114:1755-1760.**
4. **Bayés de Luna A, Cino JM, Pujadas S, et al. Concordance of electrocardiographic patterns and healed myocardial infarction location detected by cardiovascular magnetic resonance. Am J Cardiol. 2006 Feb 15;97:443-451.**

In the present case we speculate that PAF are atypical for to impute exclusively to lateral MI.
Why?
Answer: because this pattern of PAF have a clear **qR/qRs** morphology from V_2 to V_4 indicative of affectation of the initial portions of QRS complex/loop. In truly lateral myocardial infarction (antique dorsal infarction) the QRS complex/loop affectation is exclusively of the middle and final portions of QRS complex/loop between 30ms and 100 ms, i.e. the second half of QRS complex/loop. Initial q wave in anterior leads followed by tall narrow R wave is characteristic of left septal fascicular block (LSFB). Additionally, the appearance of left anterior fascicular block (LAFB) is another strong argument in favor of bifascicular left bundle branch block(LAFB+LSFB). In this situation the supraventricular stimulus reaches the LV endocardium (vector from 0 to 20 ms) by the unique non blocked fascicle: the left posterior fascicle (represented by the 1_{PI} vector) directed to back: initial q wave in V_2-V_3.

Vector 1_{AS} **blocked:** Left Anterior Fascicular block
Vector 1_{AM} blocked: Left Septal Fascicular block





Vector 1_{PI} non blocked directed to back: initial q wave V_2-V_3

Vector 1_{PI} or first vector dependent on left posterior fascicle (LPF) heading backward, below and to the right. The LSF ends at about one third of the distance from the apex to the base, near the base of the posteromedial papillary muscle of mitral valve. The vector 1PI directed to back explains the frequent appearance of small initial q waves in one or more leads of anterior wall, mainly V_2 or V_3 in the presence of LSFB.

The vector dependent on the left septal fascicle LSF (1_{AM} vector) normally heading to the front and the right, does not manifest by block, which is translated by absence of initial q wave in left leads. In this particular case Q waves in left leads are consequence of lateral MI

Following, during a middle moment (from 20 to 40 ms or intermediate initial forces) activation of free wall is completed: As consequence of concomitant LAFB the superior portions of the free wall shift the QRS axis to upward (LAFB) and rightward on right superior quadrant (lateral infarction)

During the final intermediate moments (from 40 to 60 ms.) the forces activate the blocked **septum central apical area.** The vector is heading from back to front and mildly to the left, originating prominent great voltage R waves in V_2 and V_3 that obligatorily grow from V_1 to V_2 or V_3 and decrease from V_4 to V_6.

Finally, basal final vectors from 60 to 100 ms or 110 ms. It corresponds to activation of final basal portions of both ventricles heading from down to top and backward, and discretely rightward or leftward.

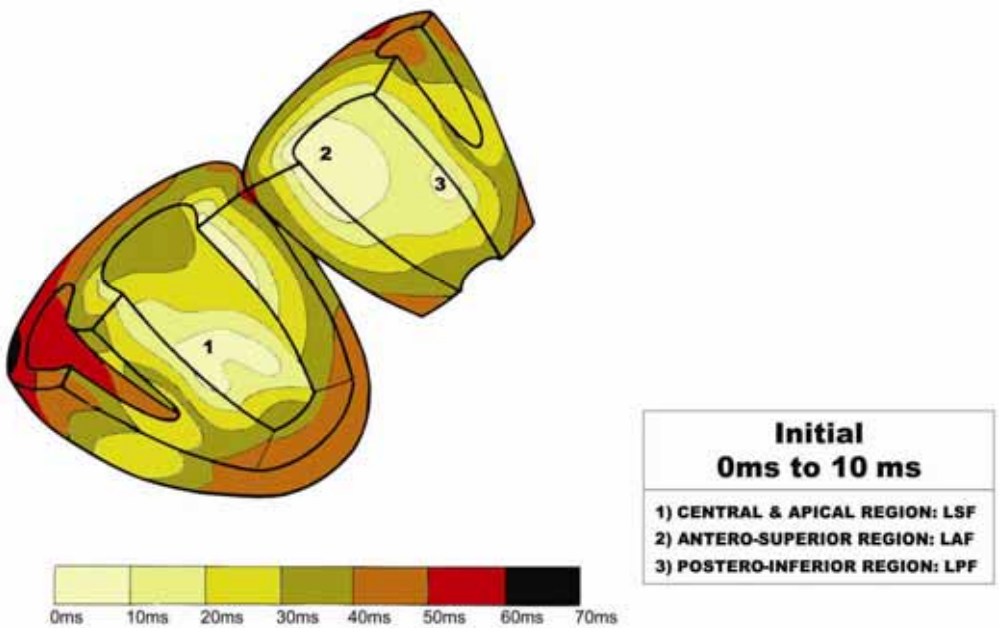
The Durrer concept

In 1970, Dr. Dirk Durren et al from the University Department of Cardiology and Clinical Physiology, Wilhelmina-Gasthuis, Amsterdam, The Netherlands, demonstrated in a classical manuscript using 870 intramural terminals in isolated human hearts, that three endocardial areas are synchronously excited from 0 to 5 ms after the start of left ventricle (LV) activity potential. To obtain information concerning the time course and instantaneous distribution of the excitatory process of the normal human heart, the authors studied on isolated human hearts from seven individuals who died from various cerebral conditions, but who had no history of cardiac disease.

The first LV areas excited were:

1. High on the anterior paraseptal wall just below the attachment of the anterolateral papillary muscle (ALPM) where the left anterior fascicle (LAF) ends;
2. Central on the left surface of the interventricular septum (IVS) where the left septal fascicle (LSF) ends. Septal activation started in the middle third of the left side of the IVS, somewhat anteriorly and the lower third at the junction of the IVS and posterior wall. The normally functioning LSF, the left middle septum surface and the inferior two-thirds of the septum originate the first vector, vector 1 or first anteromedial (1_{AM}) vector and left inferior two-thirds of the IVS (second vector or vector of the inferior $^{2}/_{3}$ of IVS)
3. Posterior paraseptal about one third of the distance from the apex to the base near the base of posteromedial papillary muscle (PMPM) where the left posterior fascicle (LPF) ends. The posterobasal area is the last part of the LV to activate.(1).

FIGURE 17. SEQUENCE OF VENTRICULAR ACTIVATION



1. Durrer D, van Dam RT, Freud GE, et al. Total excitation of the isolated human heart. Circulation 1970; 44: 899-912.

The possible electro-vectorcardiographic expression of block of the middle fibers of the left bundle branch, generally described as "*left septal fascicular block*" (LSFB), remains one of the few mysteries of the century-old ECG. The last consensus for the standardization and interpretation of ECG, part III, about intraventricular conduction disorders, does not advise using the term "left septal fascicular block", because of the "lack of universally accepted criteria" **(1).** This rationale contains the implicit idea that the members of the consensus agreed on the existence of the above mentioned middle fibers and its electrocardiographic expression, but did not advise including it in the guidelines due to the non-existence of universally agreed electrocardiographic criteria. In the USA, there is a single investigator that suggested ECG criteria for LSFB **(2).** In Latin America the School of Buenos Aires created by legendary Professor Mauricio B. Rosenbaum, from the Cardiology Division of the Ramos Mejía Hospital, currently led by his main disciple, Dr. Marcelo V. Elizari, in a recent review in Circulation(3). agreed on the anatomical existence of the middle fascicle, when stating that in spite of the fact that conduction disorders that involve the anterior and posterior fascicles of the left branch described as hemiblocks, were accepted by the cardiology community, some anatomical and electrocardiographic studies have proposed that, besides the anterior and posterior fascicles, a middle or septal fascicle may be found in the left ventricular conduction system, which could have clinical and functional significance (4)..

1. Surawicz B, Childers R, Deal BJ,AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. 2009 Mar 17; 53: 976-981.
2. MacAlpin, R N Left Septal Fascicular Block: Myth Or Reality? Indian Pacing Electrophysiol J. 2003 Jul-Sep; 3: 157–177.
3. Elizari MV, Acunzo RS, Ferreiro M. Hemiblocks revisited. Circulation. 2007 Mar 6; 115:1154-1163.
4. *Tawara S. The Conduction System of the Mammalian Heart. An Anatomic-Histological Study of the Atrioventricular Bundle and the Purkinje Fibers. Suma K, Shimada M, trans; Anderson RN, ed. London: National Heart and Lung Institute; 2000: 45–62.*

The Argentine authors end their commentary about left septal fascicular block (LSFB) stating: "In fact, the existence of middle-septal fibers cannot be dismissed, and as such, the functional, and clinical significance of the middle or septal fascicle cannot be completely ignored" **(1).** Finally, they confirmed the anatomical existence when they wrote: "*in the left branch anatomy, the fibers of the middle fascicle originate in most cases, in the posteroinferior fascicle, and less often, in the anterosuperior fascicle or both, and in rare cases, it has an independent origin from the central area of the left branch trunk, located in its bifurcation.*" Clearly, the Buenos Aires school does not deny the anatomical existence of middle fibers, and suggests that its block may have a clinic-functional expression. Most authors today use the terms *anterosuperior* and *posteroinferior* fascicular blocks rather than the older term hemiblock.

The Brazilian school, started by late Professor João Tranchesi, has published several papers proving the existence of the block in the middle fascicule, using the methods of ECG, CXG, body surface mapping, and electrical endocardial mapping. These studies were conducted in patients with chronic Chagasic cardiomyopathy and coronary insufficiency. Cases of LSFB, both permanent and intermittent, have been describe with electro-vectorcardiographic criteria **(2-7).**

1.Nakaya Y, Hiasa Y, Murayama Y, et al.. Prominent anterior QRS forces as a manifestation of left septal fascicular block. J Electrocardiol. 1978; 11: 39–46.

2.Tranchesi J, Grinberg M, Moffa PJ, et al. The block of the division of the left branch (hemiblock). Current concepts . Arq Bras Cardiol. 1971; 24:77-90.

3.Tranchesi J, Moffa PJ, Pastore CA, et al. Block of the antero-medial division of the left bundle branch of His in coronary diseases. Vectrocardiographic characterization. Arq Bras Cardiol 1979;32: 355-360.

4.Moffa PJ, Del Nero E, Tobias NM, Serro Azul LG, Pileggi F, Decourt LV.: The left anterior septal block in Chagas' disease. Jap Heart J. 1982; 23:163-165.

5.Moffa PJ, Pastore CA, Sanches PCR et al. The left-middle (septal) fascicular block and coronary heart disease. In Liebman J, ed. Electrocardiology'96 –From the cell to body surface. Cleveland, Ohio, Word Scientific, 1996; 547-550.)

6.Moffa PJ, Ferreira BM, Sanches PC, Tobias NM, Pastore CA, Bellotti G. Intermittent antero-medial divisional block in patients with coronary disease Arq Bras Cardiol 1997; 68:293-296.

7.Sanches PCR, Moffa PJ, Sosa E, et al. ELECTRICAL ENDOCARDIAL MAPPING OF FIVE PATIENTS WITH TYPICAL ECG OF LEFT-MIDDLE(SEPTAL) FASCICULAR BLOCK. In Proceeeding of The XXVIII International Congress on Electrocardiology Guarujá SP Brazil. 2001 pp89-95..

More recently, our group reported on cases showing the existence of LSFB. In one patient, LSFB was induced by exercise as an expression of severe ischemia resulting from critical proximal obstruction of the anterior descending artery (LAD) **(1).** We also showed a transient isolated LSFB in a patient with Wellens syndrome, an entity that indicates critical proximal lesion of the LAD.**(2)** Finally, we described LSFB in sequential electrocardiograms and vectorcardiograms from a patient with a rare neuromyopathy associated with mitochondrial alterations (Kearns-Sayre syndrome), characterized by the triad of external ophthalmoplegia, atypical pigmentary retinopathy, and progressive muscular disorder including the intraventricular conduction system. **(3)**.

The last Brazilian Guidelines for Interpreting Rest Electrocardiogram **(4)** provided us with the following criteria for ECG diagnosis of LSFB, also called "*anteromedial divisional block*":

1.Uchida AH, Moffa PJ, Riera AR, Ferreira BM. Exercise-induced left septal fascicular block: an expression of severe myocardial ischemia. Indian Pacing Electrophysiol J. 2006 Apr 1;6:135-138.

2.Riera AR, Ferreira C, Ferreira Filho C, Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block? J Electrocardiol. 2008 Nov-Dec;41:671-674.

3.Riera AR, Kaiser E, Levine P, et al Kearns-Sayre syndrome: electro-vectorcardiographic evolution for left septal fascicular block of the his bundle. J Electrocardiol. 2008 Nov-Dec; 41: 675-678.

4.Pastore CA, Pinho C, Germiniani H, Samesima N, Mano R, et al. Sociedade Brasileira de Cardiologia. Diretrizes da Sociedade Brasileira de Cardiologia sobre Análise e Emissão de Laudos Eletrocardiográficos (2009). Arq Bras Cardiol 2009;93(3 supl.2):1-19. Brazilian Society of Cardiology. Cardiology Brazilian Society guidelines about Analysis and Emission Eletrocardiographic reports (2009).

QRS duration <120 ms, in general, close to 100 ms. Left fascicular blocks do not increase QRS duration (QRSD) by more than 25 ms, due to multiple interconnections between the fascicles of the left bundle branch ("passage way zone" of Rosenbaum.) The QRS complex is slightly prolonged, between 100 ms and 115 ms. Thus, LSFB patterns with more prolonged QRSD indicate the presence of additional conduction disturbances such as: other fascicular blocks, right bundle branch block, myocardial infarction, focal block, or a combination of these.

R waves voltage ≥ 15 mm in V2 and V3 or from V1; Increasing voltage in intermediary precordial leads and decreasing from V5 to V6; "r" wave jump may occur from V1 to V2 ("rS" in V1 for R in V2); an embryonic initial q in V2-V3 may be seen as a consequence of absence of the first vector.

Absence of SAQRS shift or no more than -30º in the frontal plane.

Absence of q wave on left precordial leads, as a consequence of absence of the first vector, from the centroseptal surface of the interventricular septum.

It is important to emphasize that all the above criteria are only valid in absence of right ventricular hypertrophy, septal hypertrophy or lateral myocardial infarction (antique dorsal nomenclature), and other causes of PAF. The ECG example in Figure 1A-B shows all the criteria for isolated LSFB suggested in our consensus.

Like left posterior fascicular block (LPFB), isolated LSFB is rarely identified as an isolated finding except when seen intermittently. Most cases are associated with left anterior fascicular block (LAFB), right bundle branch block, or LPFB. Fernando de Padua **(1)** wrote that it is uncertain whether this represents the normal variant of LSFB.

1. de Padua F. Intraventricular conduction defects-What future? In: de Pádua F: Macfarlene PW, eds. New Frontiers of Electrocardiology. Chichester: Research Studies Press. 1981: 181-185.

In isolated LSFB, the sequence of ventricular activation begins simultaneously at two points:
The base of the anterolateral papillary muscle (ALPM) of mitral valve, dependent on the left anterior fascicle (LAF) in the anterior paraseptal wall, just below the attachment of ALPM (1_{AS} vector);
The base of the posteromedial papillary muscle (PMPM) of mitral valve, dependent on the Left Posterior Fascicle (LPF). It is located on the posterior paraseptal wall, about one third of the distance from apex to base (1_{PI} vector).
These initial two vectors ($\mathbf{1_{AS}}$ and $\mathbf{1_{PI}}$) have opposite directions, and they cancel each other except for minimal predominance of vector $\mathbf{1_{PI}}$ directed backward (initial q waves in V2-V3). Next, the activation sequence moves to the middle-septal or left paraseptal region, blocked by numerous Purkinje areas of "Rosenbaum passage areas", thus shifting the forces to the front and left. This results in prominent anterior QRS forces (PAF). It is extremely important to take into account that like LPFB, LSFB is a diagnosis of exclusion, since it can only be made when other clinical causes of PAF on the electrocardiogram can be excluded. How do we define PAF?

Prominent Anterior Forces Definition by Electrocardiographic Parameters

In electrocardiography, prominent anterior forces (PAF) is defined when the R wave voltage in any anterior precordial lead from V_1 (+115º) through V_4 (+47º) is greater than the normal maximal limit for gender and age. Electro-vectorcardiographic criteria of PAF should be age-related and gender-related.
We think that the criterion used by some authors to consider the presence of PAF regarding the R/S ratio in V1 is not appropriate because V_2 to V_4 are not considered. Thus, an R/S ratio in $V_1 \geq 1$ is usually considered abnormal in adults. **(1)**. In our view, this criterion cannot be considered as valid, since in 1% of normal individuals have an R/S ratio in $V_1 \geq 1$ as a normal variant. In lead V_2, approximately in 25% of men and 12% of women have an R/S ratio is 1.

1. Mattu A, Brady WJ, Perron AD, et al. Prominent R wave in lead V1: electrocardiographic differential diagnosis. Am J Emerg Med. 2001; 19: 504-513.

Conclusion

There is no controversy on the anatomical and functional existence of middle fibers in the left bundle branch. The anatomy of these fibers is variable and complex, unlike the anterosuperior and posteroinferior fascicles. There are specific proofs by electrocardiography, vectorcardiography, body surface mapping, invasive endocardial electrophysiological, and atrial extra-stimulus techniques for the existence of LSFB. There is more than enough evidence about the existence of intermittent and transient forms of LSFB and it's evolutionary character over time, which is an essential element to characterize the block. Thus, there is a need for an international consensus to unify diagnostic criteria. The diagnosis should be obligatorily clinic-electro-vectorcardiographic for exclusion, ruling out the numerous other causes for prominent anterior forces. Considering PAF seen in LSFB as a possible conduction disorder involving the right bundle branch has no foundation, since the electro-vectorcardiographic pattern is characteristic and absolutely different from the dromotropic disorder in this branch.