

Good morning : My name is Florencia González Amigo, I am a cardiologist from the Bahía Blanca city, Buenos Aires, Argentina. Until now I have behaved as a faithful reader of the messages. Very grateful to allow me to belong to the group.

I want to consult the group on the tracing of an ECG.

This is an 89-year-old male patient with cardiovascular risk factors (ex-smoker, hypertensive) coronary heart disease (previous AMI in 2000 without treatment that evolved with anteroseptal Q)He is currently hospitalized with a diagnosis of non-ST-segment elevation acute coronary syndrome. Transient depression of the ST segment is observed in the anterolateral wall, which reverses (levels) when the precordial pain subsides.

**Positive quantitative troponin.**

Attached is his ECG . It has been difficult for us to define it. We see it as An standard. Maskeranding bundle branch block

I await your opinions regarding the layout. Thank you very much!!Kind regards to the whole group.Florence Gonzalez-Amigo

**Spanish**

**Buenos dias**

**Mi nombre es Florencia González Amigo, soy cardióloga de la ciudad de Bahía Blanca, Buenos Aires, Argentina.**

**Hasta ahora me he comportado como fiel lectora de los mensajes. Muy agradecida de permitirme pertenecer al grupo.**

**Quiero consultar al grupo el trazado de un electrocardiograma.**

**Se trata de un paciente de 89 años, masculino, con factores de riesgo cardiovascular (ex tabaquista, hipertenso) cardiopatía coronaria (IAM anterior en el año 2000 sin tratamiento que evolucionó con Q anteroseptal)**

**Actualmente está internado con diagnóstico de síndrome coronario agudo sin elevación del segmento ST. Se observa depresión transitoria del segmento ST en pared anterolateral, que revierte (nivela) al ceder el dolor precordial. Troponina cuantitativa positiva.**

**Adjunto su electrocardiograma. Nos ha resultado complejo definirlo.**

**Lo vemos como un bloqueo de rama enmascarado en estándar.**

**Aguardo sus opiniones con respecto al trazado.**

**Muchas gracias!!**

**Saludos cordiales a todo el grupo**

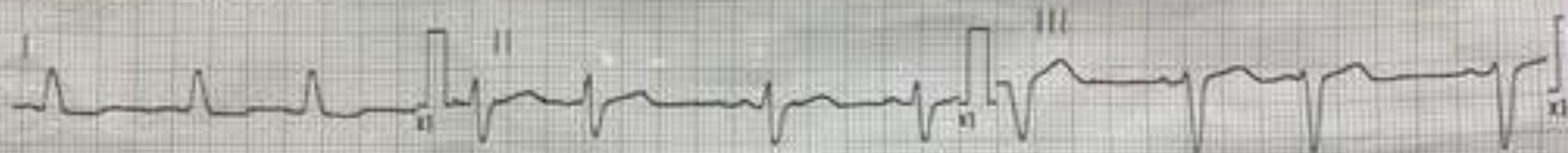
**Florencia González Amigo**

**[flor\\_amigo@HOTMAIL.COM](mailto:flor_amigo@HOTMAIL.COM)**

2022-3-9 18:36:48  
LACARETVA  
RIBI02 7.2016 -

II

FC:77

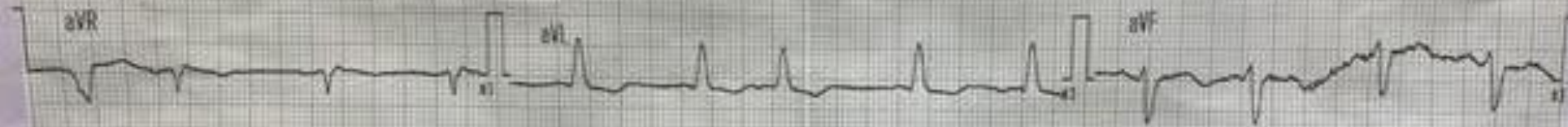


Sex: m  
Age: 65

2022-3-9 18:36:57

III

FC:78



aVR

aVI

aVF

Sex: m  
Age: 65

2022-3-9 18:36:08

IV



V1

V2

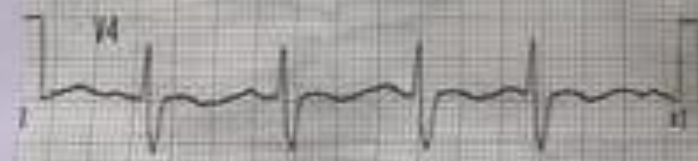
V3

FC:81

3-9 18:36:55

V

2022-3-9 18:36:21



V4

V5

V6



V5

V6

My cardiology sites of scientific interest:

<https://ekgvcg.wordpress.com/>

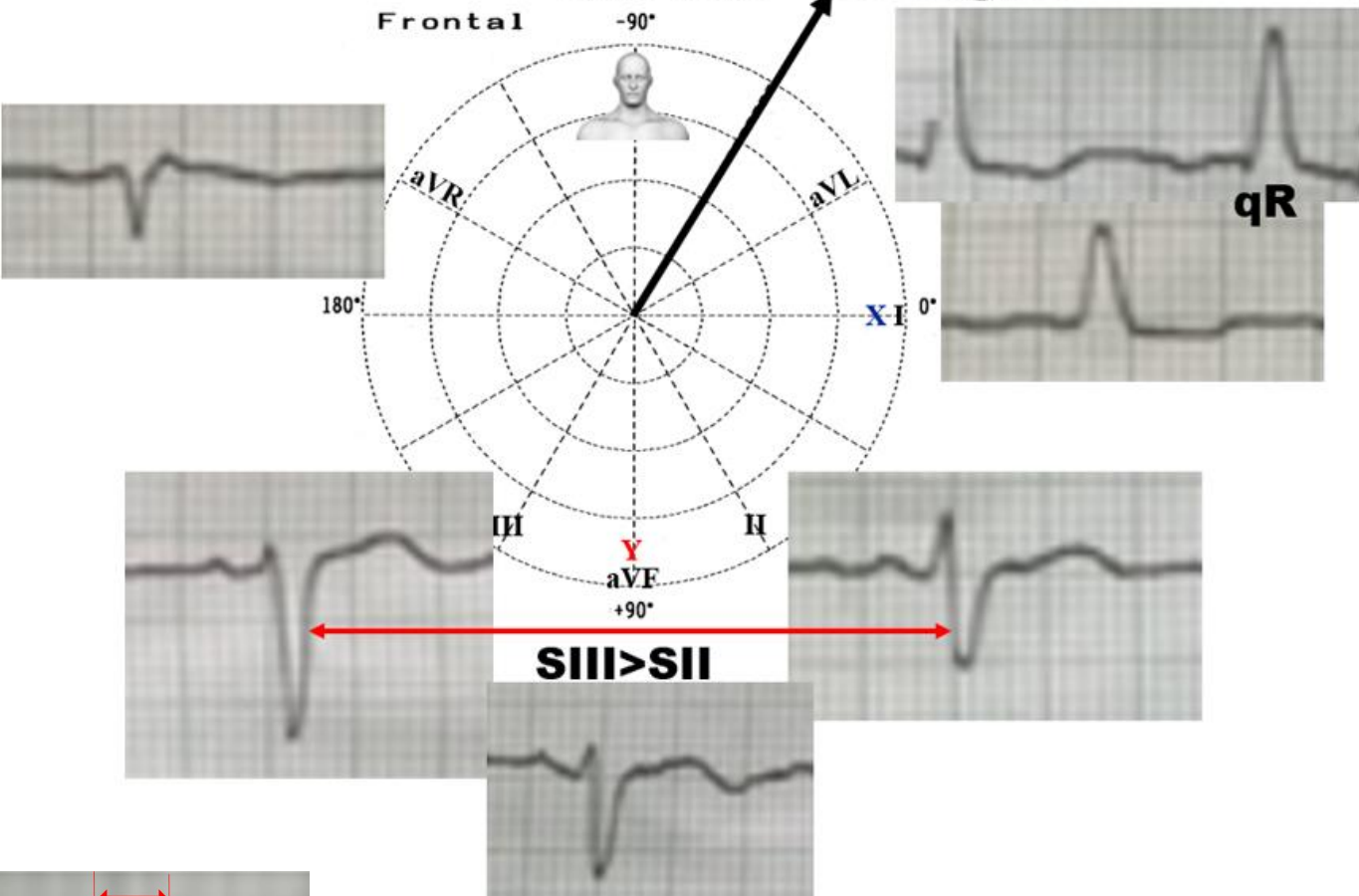
<https://cardiacademy.com/>



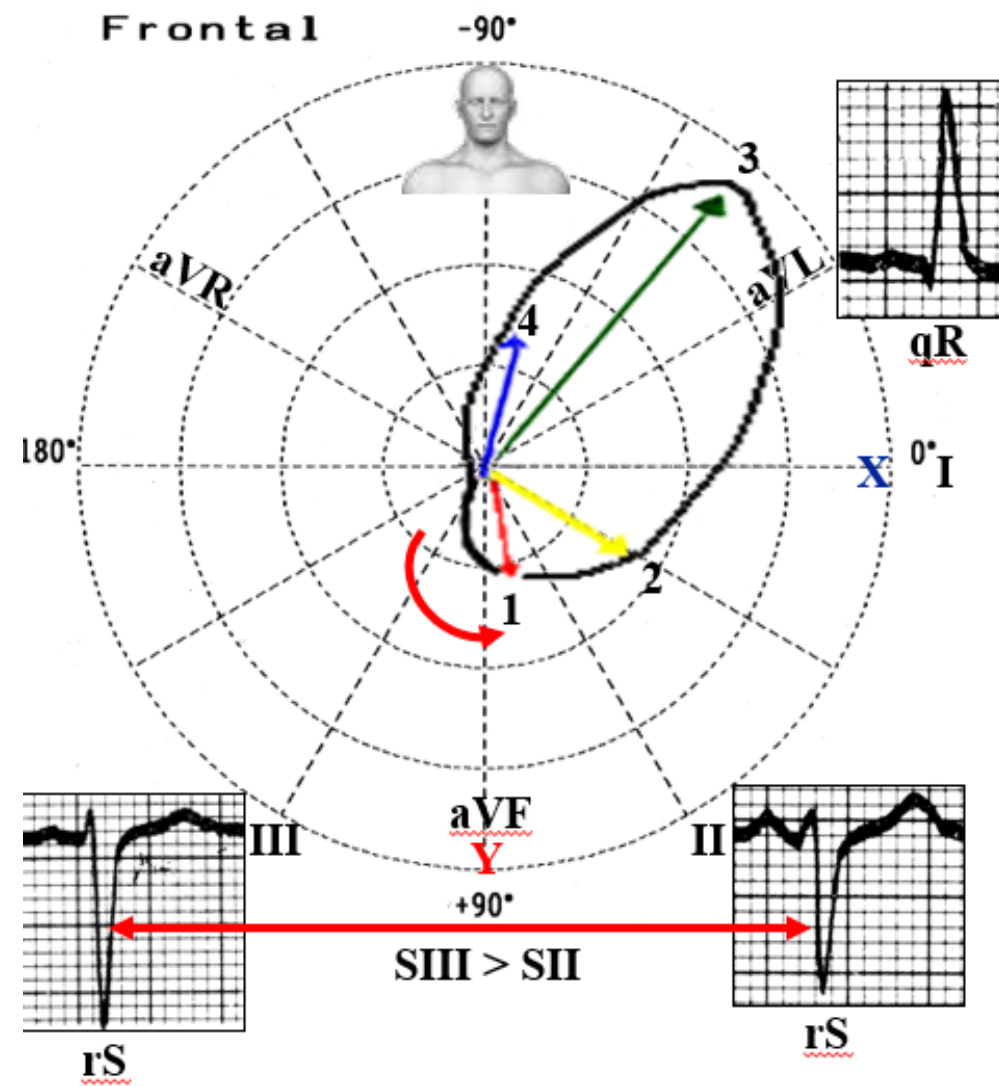
**Prof. Dr Andrés Ricardo Pérez-Riera, MD PhD**  
**Clínica Médica e suas especialidades**  
**Uninove - Universidade Nove de Julho - Campus Mauá**

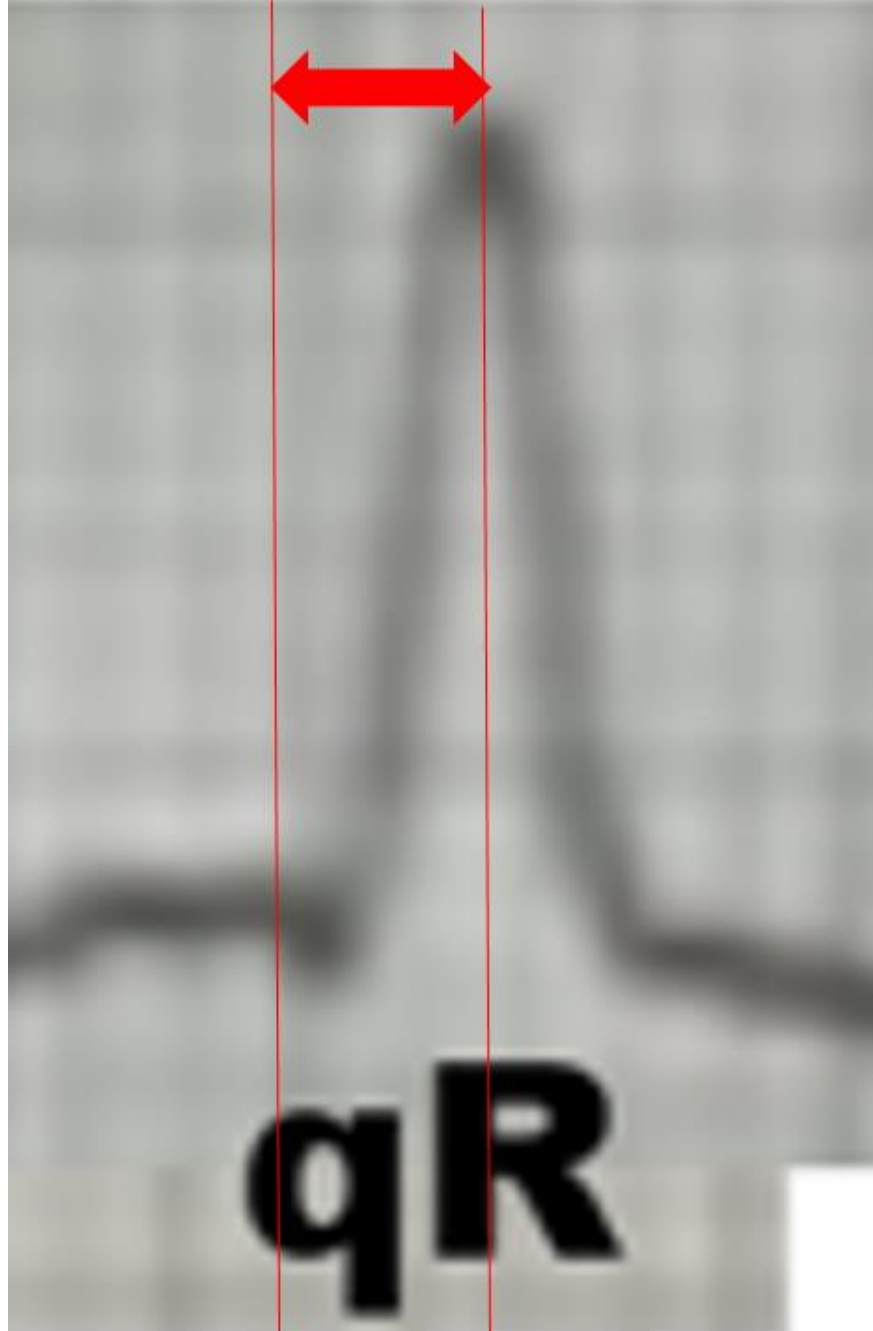
CV Lattes: <http://buscatextual.cnpq.br/buscatextual/visualizacv.do?id=K4244824E7>

**QRS axis  $\approx -60$  degree: Extreme QRS left axis deviation**



**LAFB Right Frontal Plane  
Hypothetical depolarization in the FP**



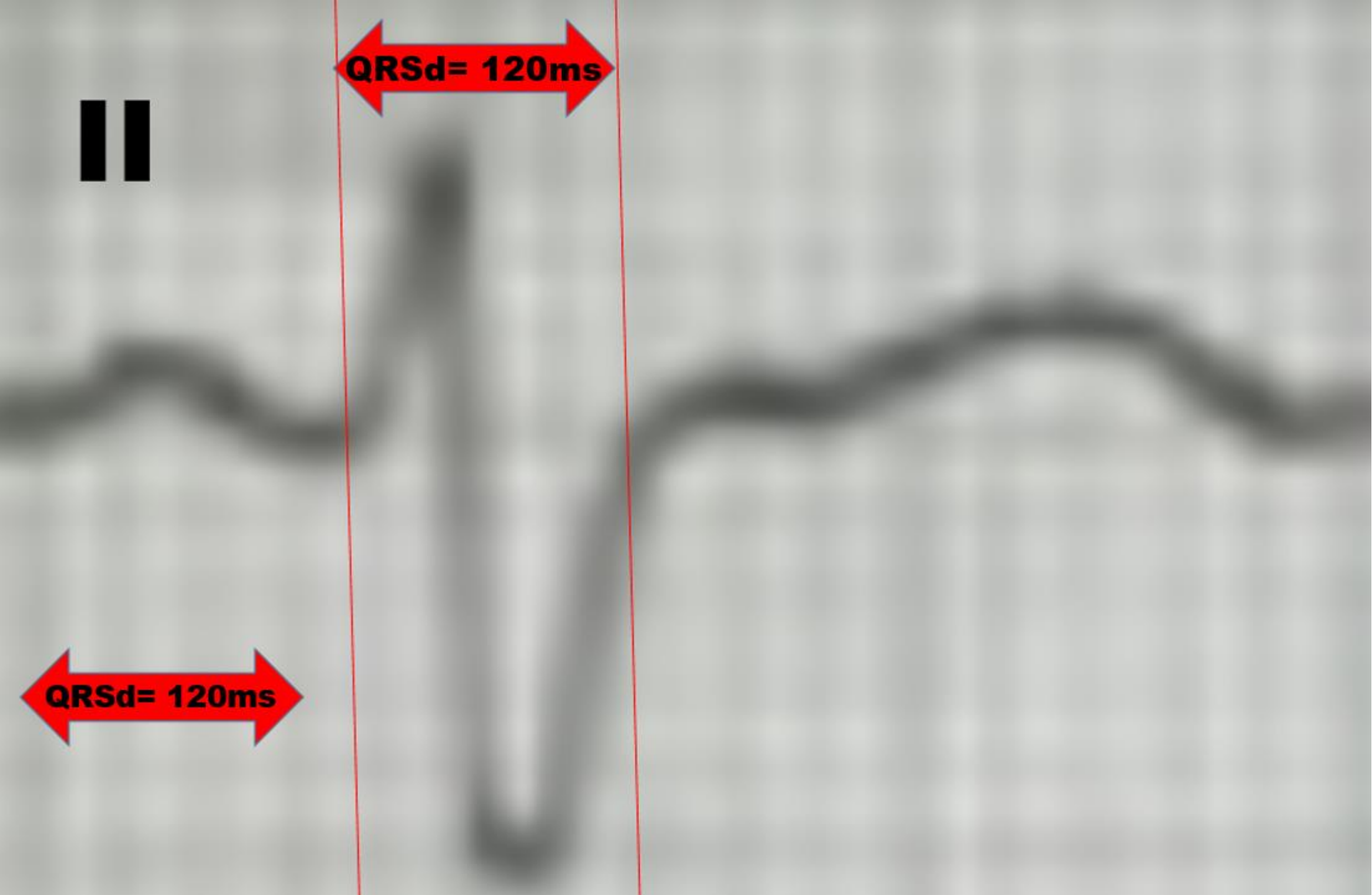


**Prolonged R- Wave Peak Time,  
Ventricular Activation Time or  
intrinsicoid deflection in aVL  $\geq 45$  ms: LAFB;**

II

QRSd= 120ms

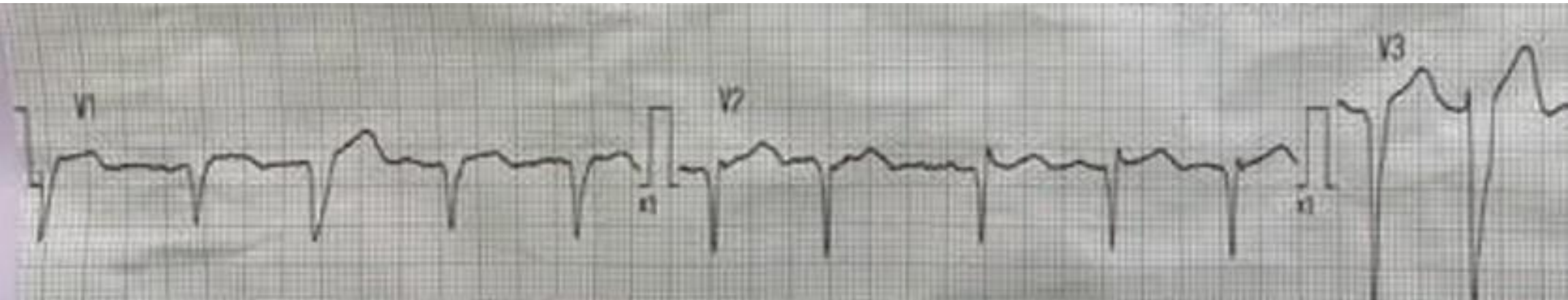
QRSd= 120ms



**Clinical diagnosis: Acute coronary syndrome: Non-ST Segment elevation MI: Dynamics ST segment depression**

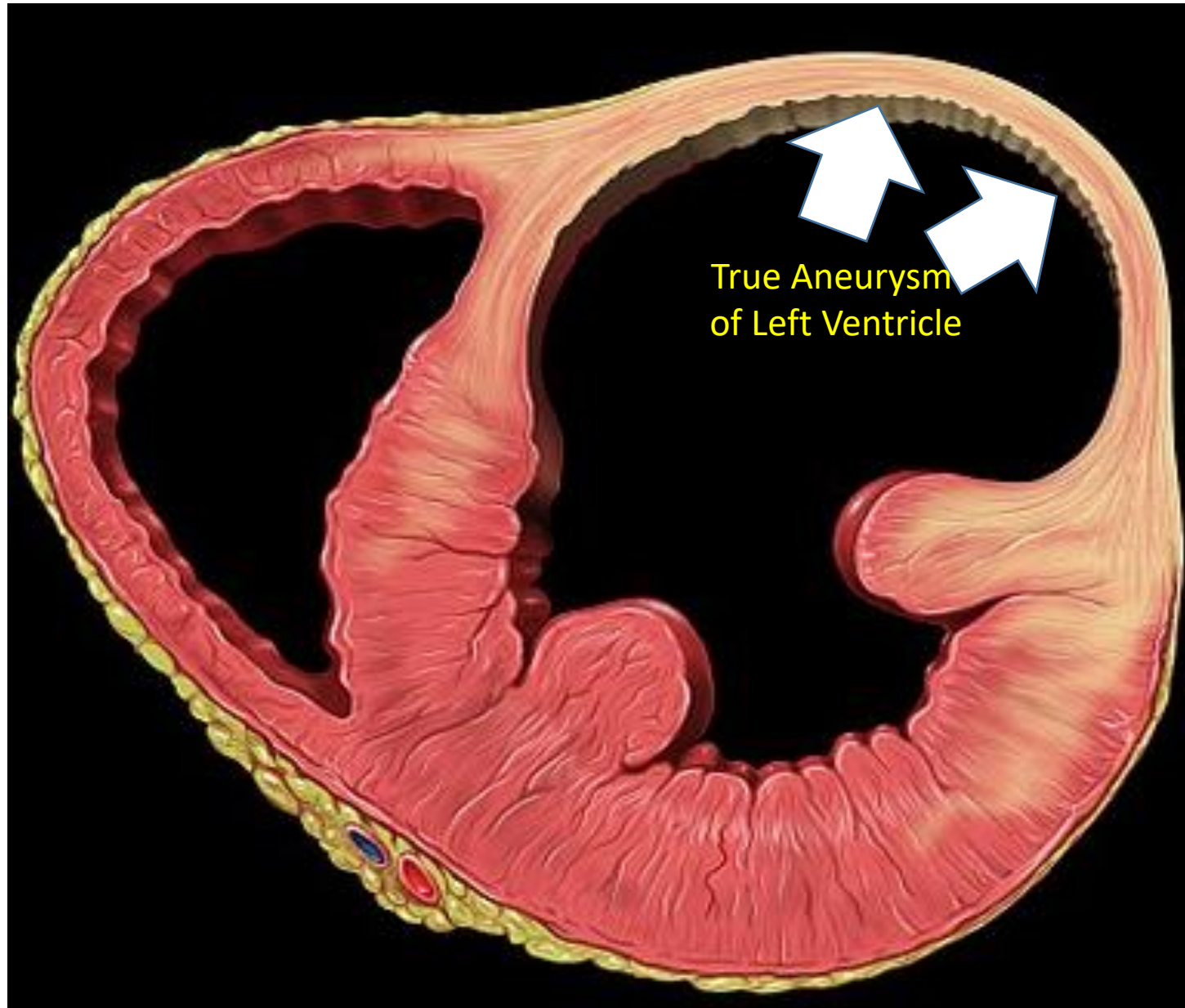
## **ECG diagnoses**

- LAFB +**
- Standard Type” or Standard Masquerading Bundle-Branch Block: consisting of the pattern of left bundle-branch block (LBBB) in the limb leads and right bundle-branch block (RBBB) in the unipolar precordial leads.+***
- Old Anteroseptal MI +**
- Non-ST-segment elevation acute coronary syndrome. Transient depression of the ST segment+**
- Premature supraventricular contractions+**
- Probable true ventricular aneurysm: why?**





## True Left Ventricle Aneurysm after Acute Myocardial Infarction



**View of the minor axis of an aneurysm of the left ventricle of the heart**

Ventricular aneurysms are one of the late mechanical complications of acute myocardial infarction. What can we do after an AMI? The word aneurysm refers to a bulge or 'pocket' in the wall or lining of a vessel that commonly occurs in blood vessels at the base of the septum or within the aorta. No heart, they usually arise from a wall of the VE with weakened tissue, which distends during systole, forming a large ball of blood. (**"What is an Aneurysm?". [www.heart.org](http://www.heart.org). Retrieved 14 March 2017.**). A ventricular aneurysm is a section of defective wall that is projected to occur during systole or a paradoxical local expansive movement of the wall, "paradoxal contracture". A ventricular aneurysm is a defect in the left ventricle (95%) or right (5%) of the heart, usually caused by transmural infarction. The LV wall aneurysm, in turn, decreased the LV ejection fraction, leading to a lack of systemic blood flow. Ventricular aneurysms can be fatal. Generally they will not break because they will be covered by scar tissue. Characteristically, a non-ECG LV aneurysm is associated with ST-segment **elevation.****Victor F. Froelicher; Jonathan Myers (2006). Exercise and the heart. Elsevier Health Sciences. p. 138. ISBN 978-1-4160-0311-3.**

The two most common symptoms are dyspnea and angina alone or in combination. In 15-20% there are ventricular arrhythmias and systemic embolism. Areas of scar tissue around these foci for the origin of ventricular arrhythmias: sustained ventricular tachycardia, syncope or sudden death. Arrhythmias occur in 20% of patients with large aneurysms and 3% with small ones. The most valuable physical sign is a double, diffuse, or dislocated apical impulse. Either the diagnosis was made with an echocardiographic study by radioventriculography or, at the time of cardiac catheterization, by left ventriculography. A computerized tomography and nuclear magnetic resonance also are important.

Predisposition to an increased risk of: Ventricular arrhythmias and sudden cardiac death (arrhythmogenic myocardial scar tissue), Congestive heart failure (CHF) and Formation of mural thrombus and subsequent embolization

# Characteristics of the ECG with aneurysm of the left ventricle

1. ST elevation observed > 2 weeks after AMI: **The present case**;
2. Most commonly recorded in the precordial leads **The present case**;
3. The ST segments can be concave or convex **The present case**;
4. Generally associated with Q- or QS waves **The present case**;
5. The T waves have a relatively small amplitude compared to the corresponding QRS complex (unlike the hyperacute T waves of acute STEMI). **The present case**: T-wave/QRS ratio < 0.36 in all precordial leads favors LV aneurysm, T-wave/QRS ratio > 0.36 in *any* precordial lead favours anterior STEMI

## Masquerading Right Bundle Branch Block concept

The ECG complex coined since Richman as “masquerading bundle-branch block”(Richman 1954) to day we know that is essentially a complete RBBB and high degree LAFB, with further modifications of the initial and final QRS vectors, so that standard leads, and at times the left precordial leads, resemble left bundle-branch block (Schamroth 1975). Masquerading BBB is not a specific entity but is an electrocardiographic complex the result of RBBB with varying combinations of LAFB, intramural left ventricular block, left ventricular enlargement/hypertrophy and anterior myocardial infarction or fibrosis. Since the pioneer Rosembaum’s et al studies (Rosembaum 1968; Rosembaum 1973) we know two ECG types of “Masquerading” Bundle-Branch Block. There are a third type that is the association of both:

- I. The “Standard Type” or Standard Masquerading Bundle-Branch Block: consisting of the pattern of left bundle-branch block (LBBB) in the limb leads and right bundle-branch block (RBBB) in the unipolar precordial leads.*
- II. The “Precordial Type” or Precordial Masquerading Bundle-Branch Block*
- III. The Standard and Precordial Masquerading Bundle-Branch Block in Association.*

## The four main developmental ECG patterns of standard Masquerading type

	aVL	I	II	III
1. Uncomplicated LAFB: QRS duration <120ms	qR	qR	rS	Rs (SIII>SII)
2. LAFB with CRBBB: QRS duration ≥120ms	qRS	qRS	rS with notch on ascending ramp of S	Rs with notch on ascending ramp of S
3. LAFB with CRBBB and diminution of the final QRS vectors. QRS duration ≥120ms	qR	qR	rS	rS
4. LAFB with CRBBB and diminution of the final QRS vectors and diminution of the initial QRS vectors	R	R	QS	QS

## I. The “standard type” (“*standard masquerading right bundle-branch block*”) *The present case*

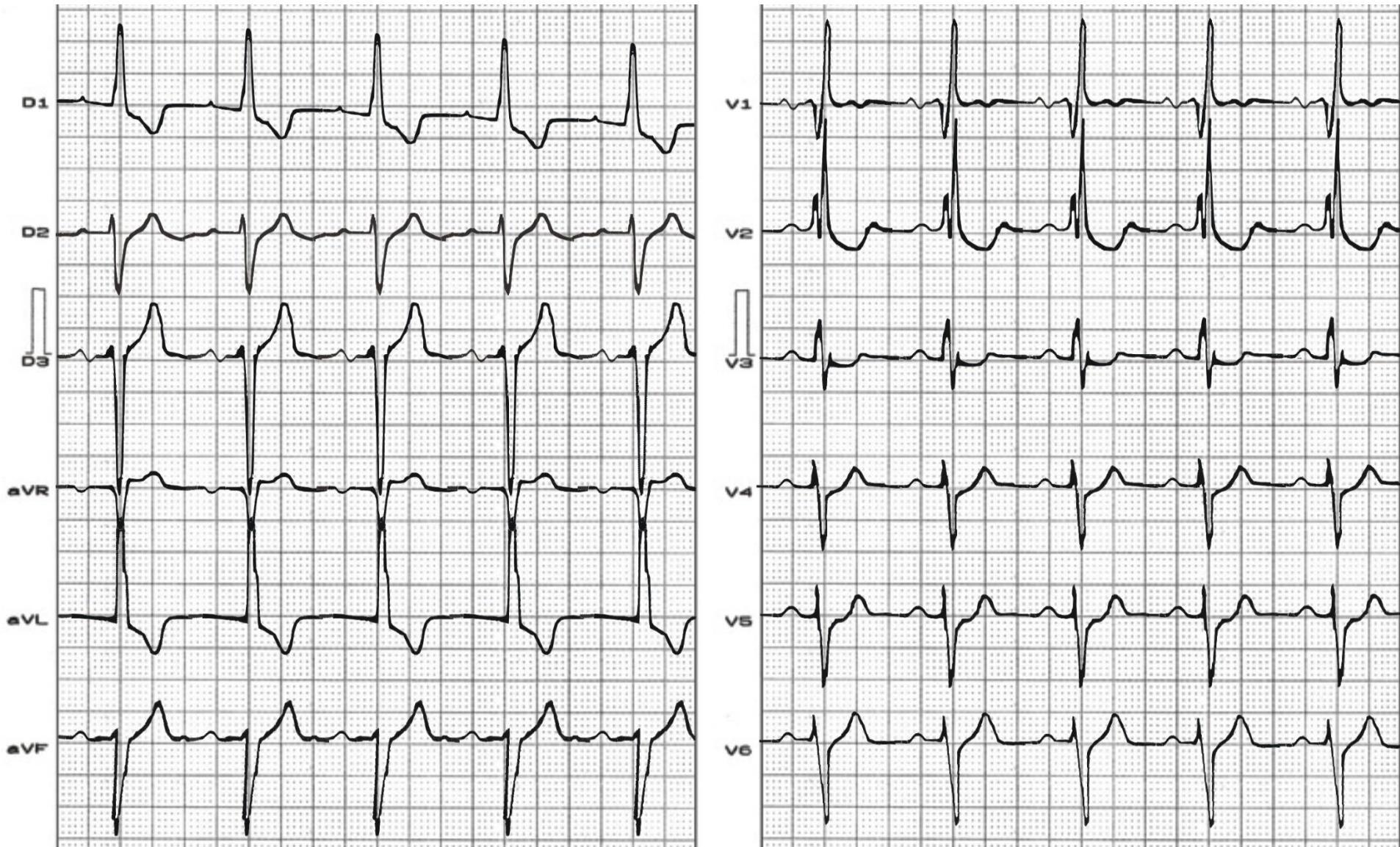
In “*standard masquerading right bundle-branch block*” the presence of a high degree left anterior fascicular block (LAFB) obscured totally or partially the diagnosis of right bundle branch block (RBBB) only on frontal plane by abolishing (or becomes very small) the final broad S wave in the leads I and aVL (**Ortega-Carnicer 1986**). Consequently, the limb leads may resemble left bundle branch-block (LBBB) although the precordial ECG remain typical for CRBBB. The precordial leads reflect the feature of RBBB.

### **Conditions necessary for the presence of standard Masquerading Right Bundle –Branch Block**

1. High degree of left anterior fascicular block ,(the present case)
2. Right Bundle-Branch Block ,(the present case)
3. Bilateral bundle-branch lesions of considerable intensity, which do not completely disrupt the continuity of the branches (**Unger 1958**) ,(the present case)
4. Left Ventricular Enlargement or Hypertrophy (LVE/LVH) ,(the present case) and marked biventricular hypertrophy
5. Localized block in the left ventricle.
6. Frequent severe fibrosis, or truly massive myocardial infarction mainly in anterior wall.

**Etiologies:** **Coronary heart disease**,(the present case) Long standing **systemic hypertension**. ,(the present case) Cardiomyopathy Ex. Chronic Chagasic myocarditis. Lev’s disease and Association of previous one. **Prognosis:** always poor.

## Example of “Standard Masquerading Right Bundle-branch Block”



*Extreme QRS left axis deviation ( $\hat{S}\hat{A}QRS -50^\circ$ ),  $S_{III} > S_{II}$ : LAFB. The limb leads show a LBBB-like pattern, but the precordial leads show a RBBB.  $S_{III} > 15\text{mm}$ : Type IV Rosebaum LAFB: association of LAFB + LVE or LVH.*



## **Case report (similar)**

### **Symptomatic elderly man (syncope) with critical coronary obstruction on LAD treated 6 Months ago with stent implantation and current dynamic dromotropic disorder on his successive ECGs**

Patient of 67 years carrying Coronary Artery Disease (CAD), with the antecedent of stent implantation on the LAD artery 6 months ago.

The first ECG was performed on admission at 10.14.02 08: 21' AM.

The second one was recorded shortly after the syncopal episode occurred approximately two hours after admission (02.10.14 10: 38').

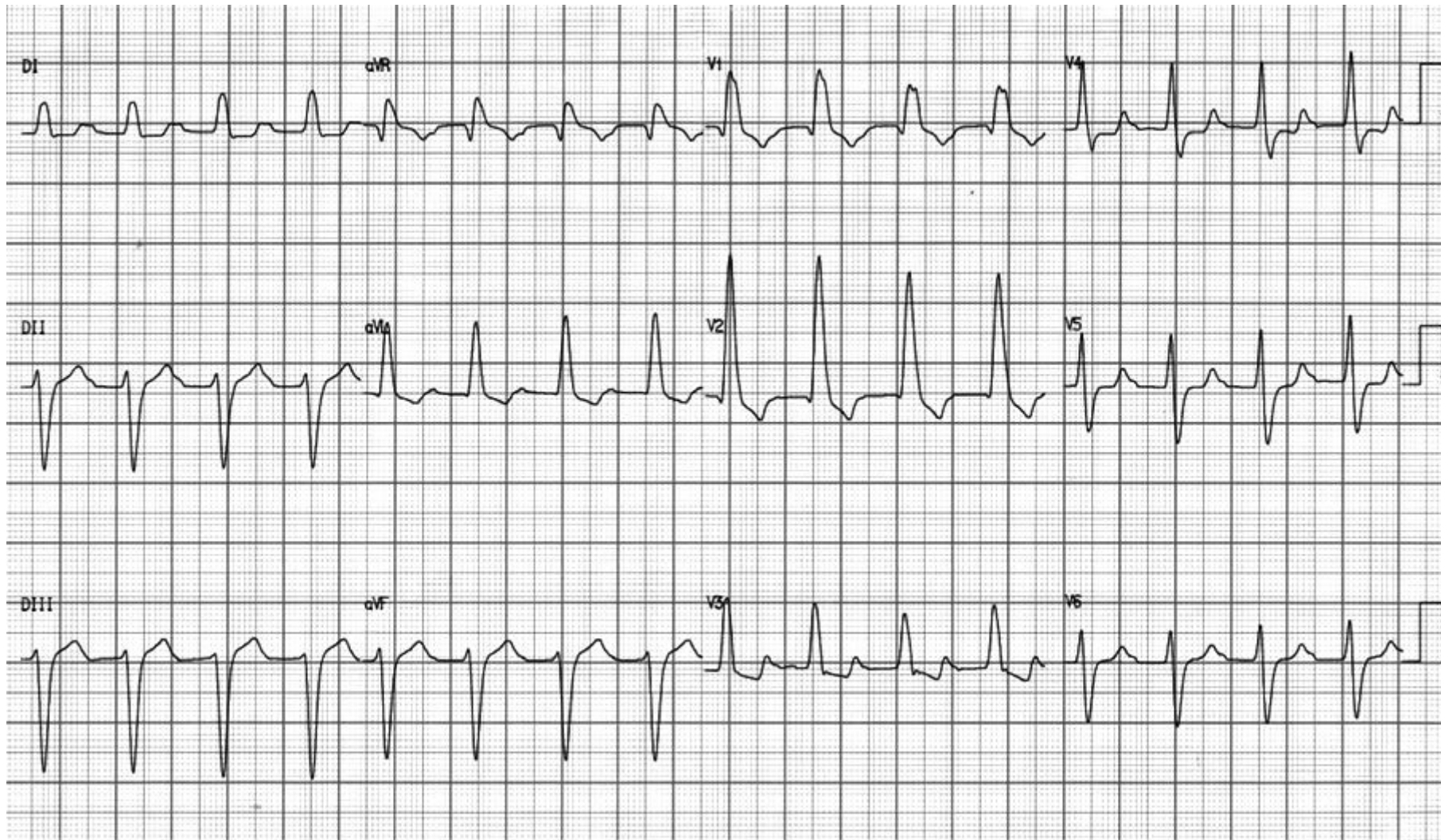
The Holter monitoring 24h revealed an intermittent LBBB pattern. Later we will send the Holter study and the VCG.

The electrophysiological study (EPS) revealed HV interval prolongation = 84 ms (normal 55 ms).

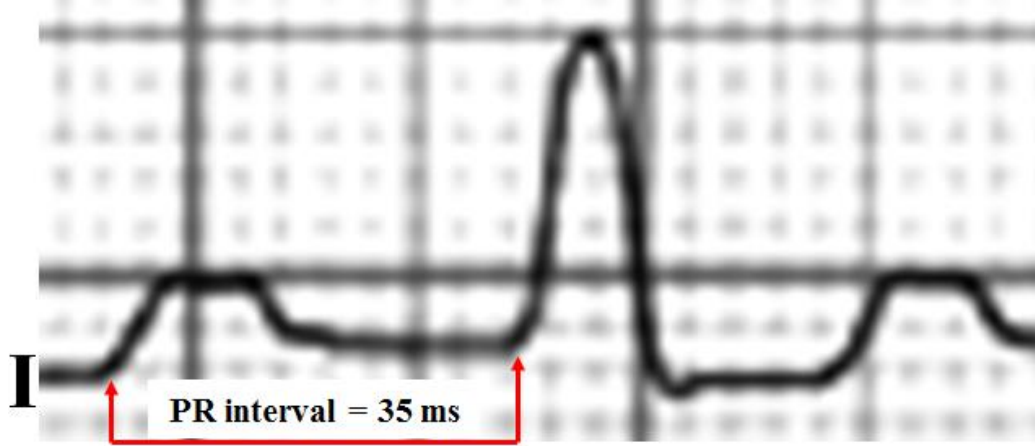
Concomitantly, the AH interval also was prolonged.

We would like to hear your valuable opinions.

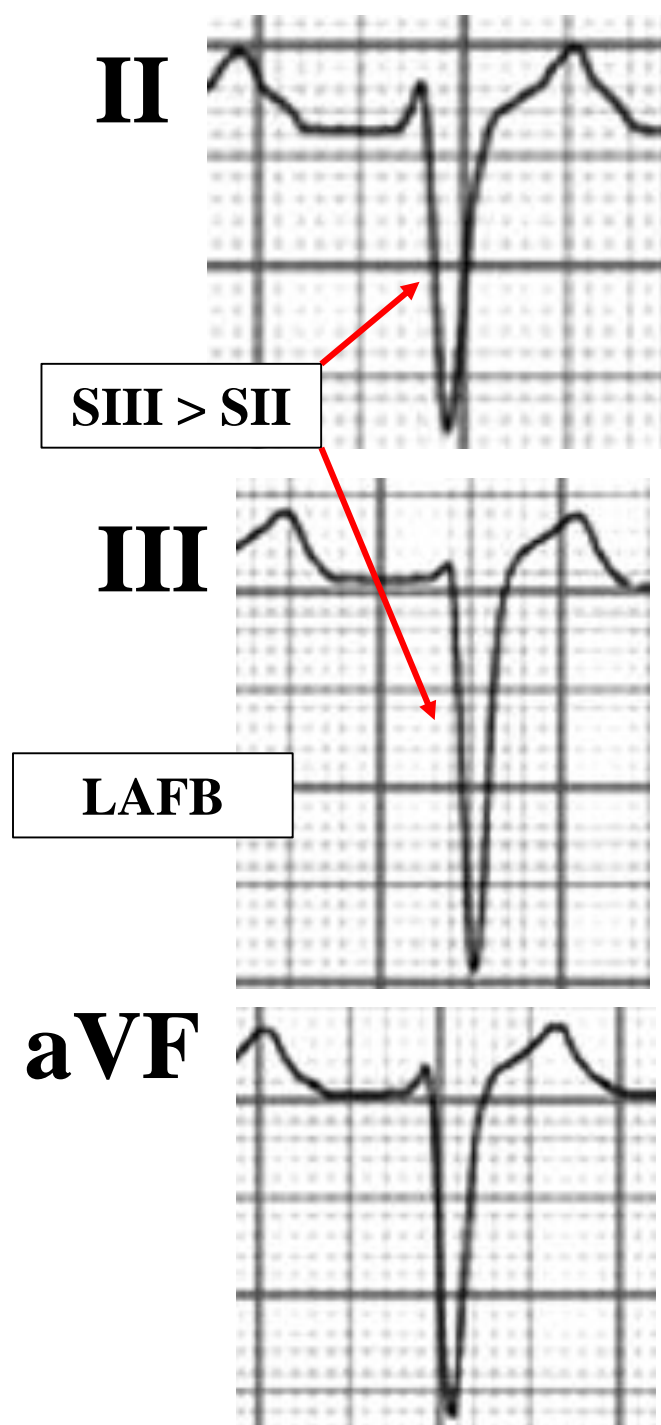
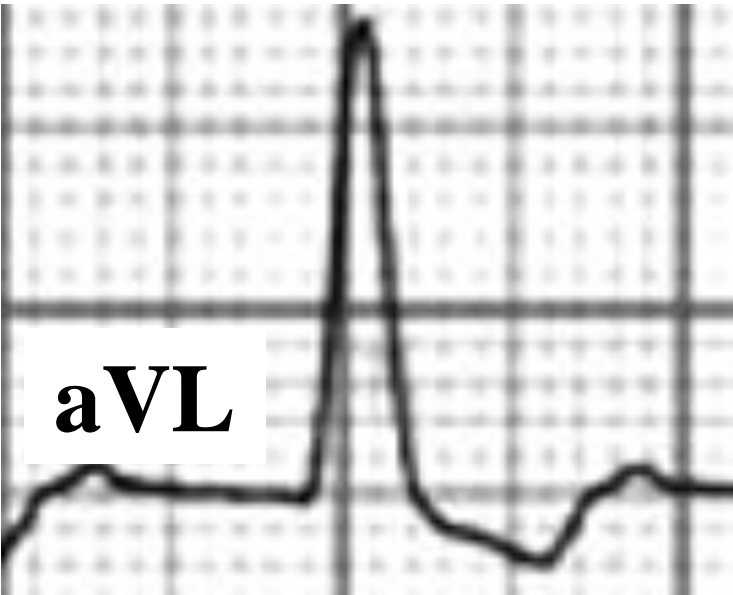
ECG1=02-10-14 08:21' admissão



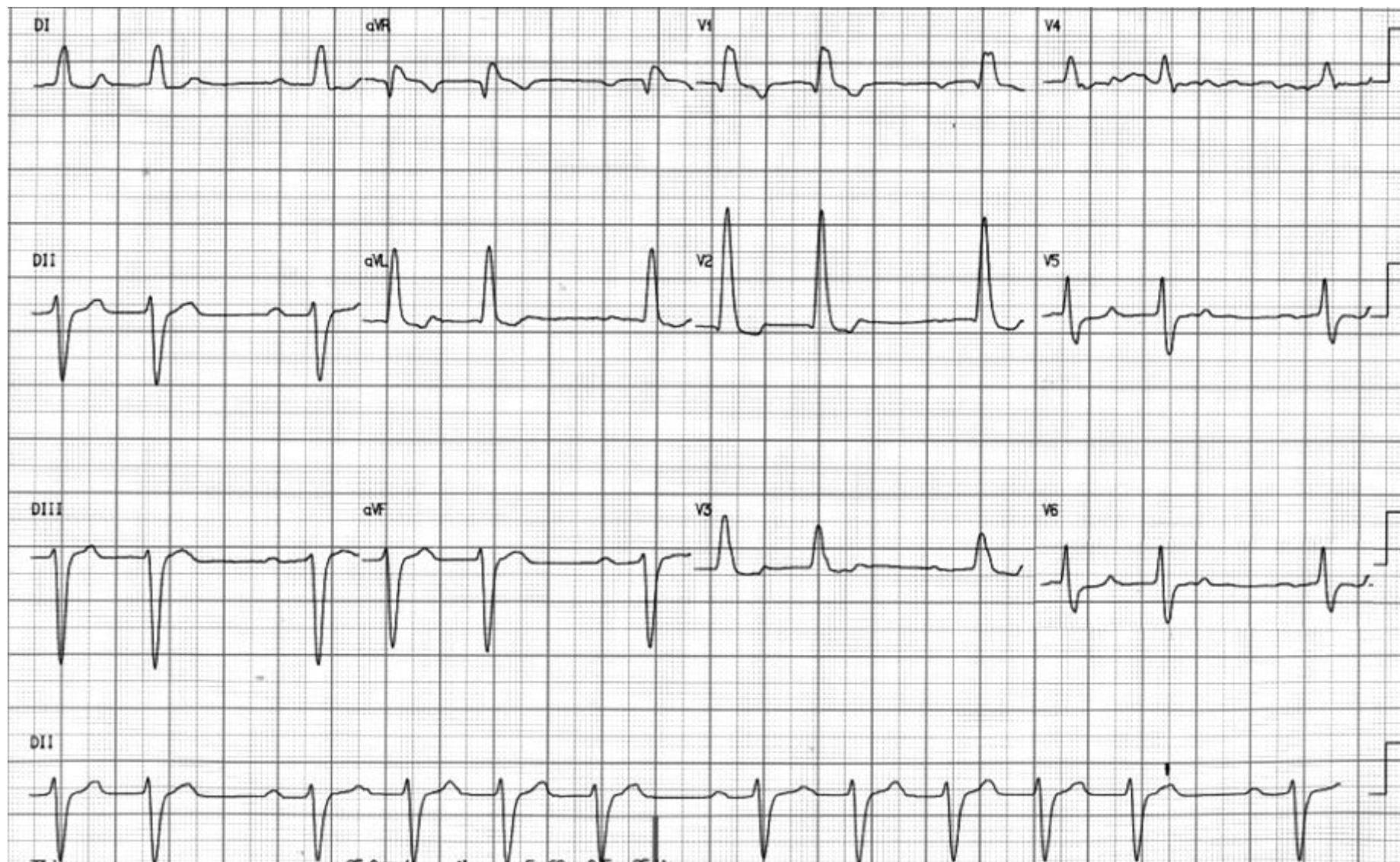
**ECG diagnosis:** Standard Masquerading Right Bundle-Branch Block +LAFB+LSFB+prolonged PR interval: probable claudication of posteroinferior fascicle suggesting tetrafascicular block. Why ? See next slide



In “*standard masquerading right bundle-branch block*” the presence of a high degree LAFB obscured totally or partially the diagnosis of RBBB only on FP by abolishing (or becomes very small) the final broad S wave in the leads I and aVL. Consequently, these limb leads may resemble LBBB although the precordial ECG remain typical for CRBBB.



ECG2= 02-10-14 10:38'



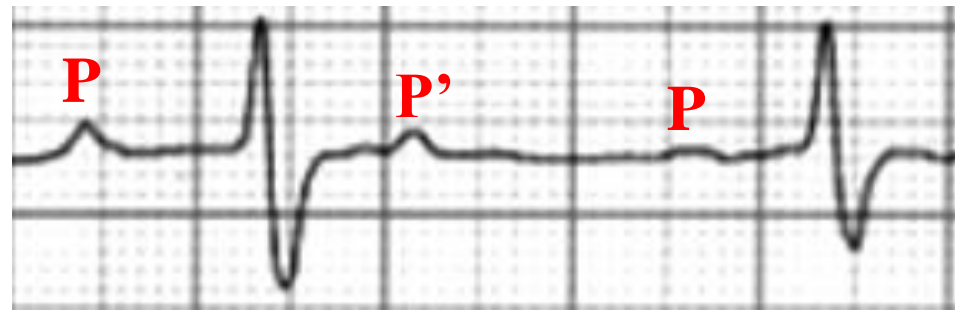
V1



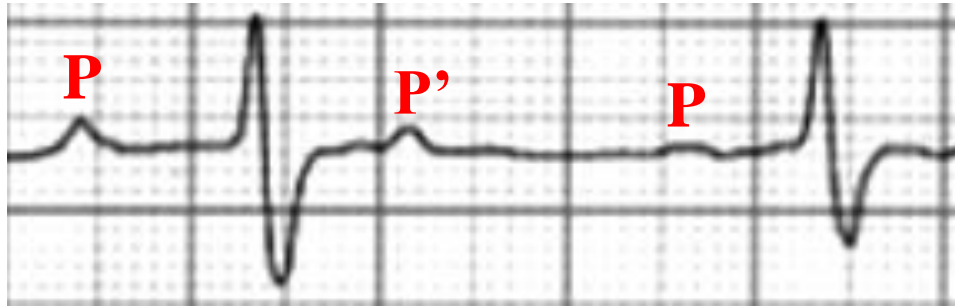
V2



V5



V6

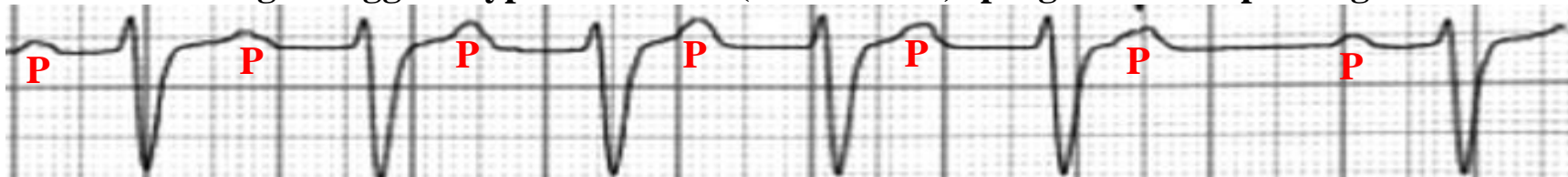


2:1 2nd degree AV block or pseudo 2:1 2nd degree AV block, the second (non conducted) and third P waves (P') have a different morphology from the conducted P wave, being ectopic, premature and not conducted: sinus rhythm with blocked atrial extrasystoles. P' waves are atrial extrasystoles.

**V1:** qR pattern RBBB with septal myocardial infarction.

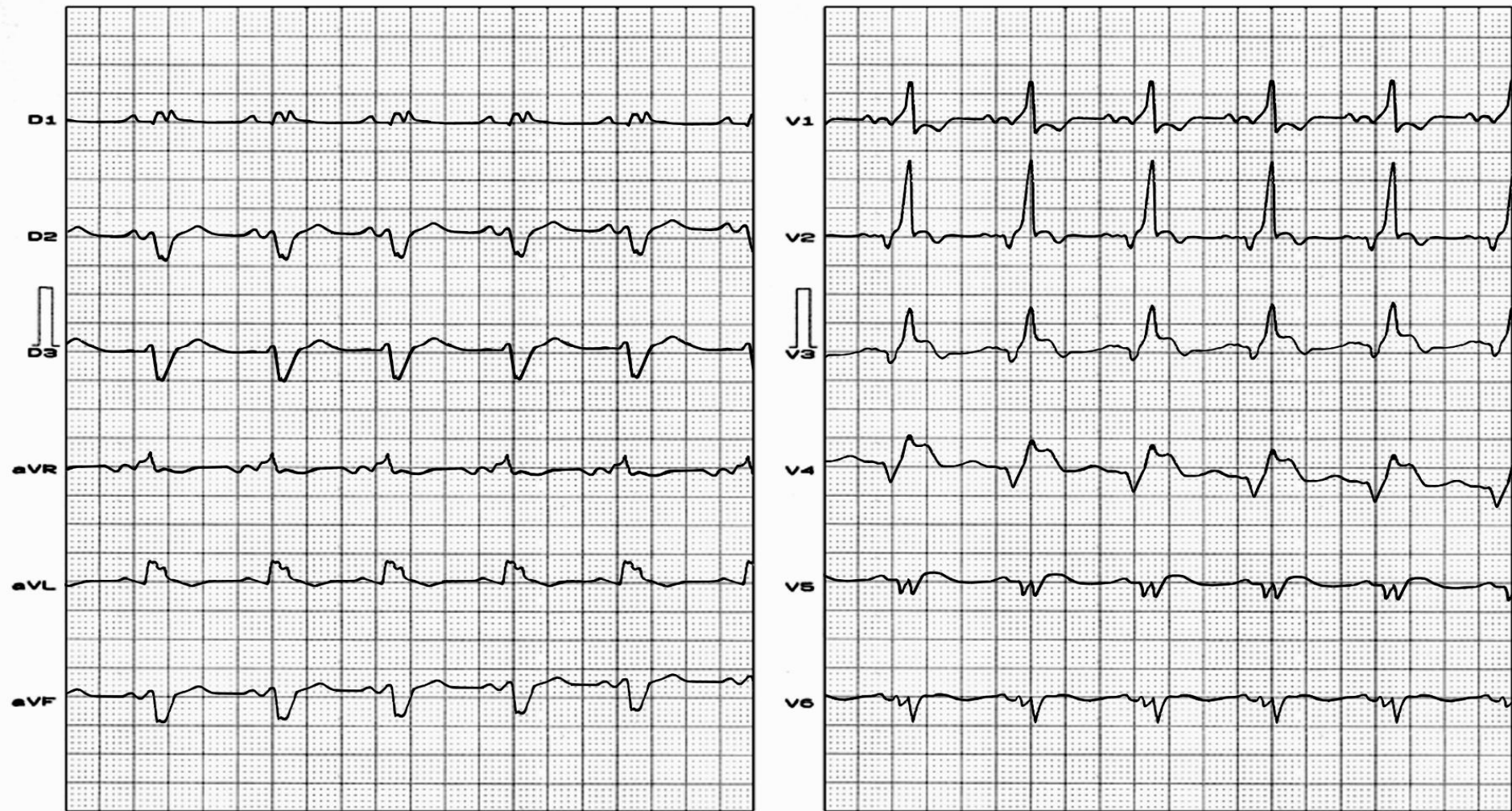
**V1-V2:** qR pattern, Increased intrinsicoid deflection of V<sub>1</sub> and V<sub>2</sub>, R wave “in crescendo”, R wave voltage of V<sub>1</sub> ≥ 5 mm, small q wave in V2 or V1 and V2, R wave of V2 > 15 mm, absence of q wave in V5, V6 and I (by absence of the first septal vector 1<sub>AM</sub>): Left Septal Fascicular Block.

**ECG 2: Long II suggests type I AV block (Wenkebach): progressive PR prolongation**



## Conclusions:

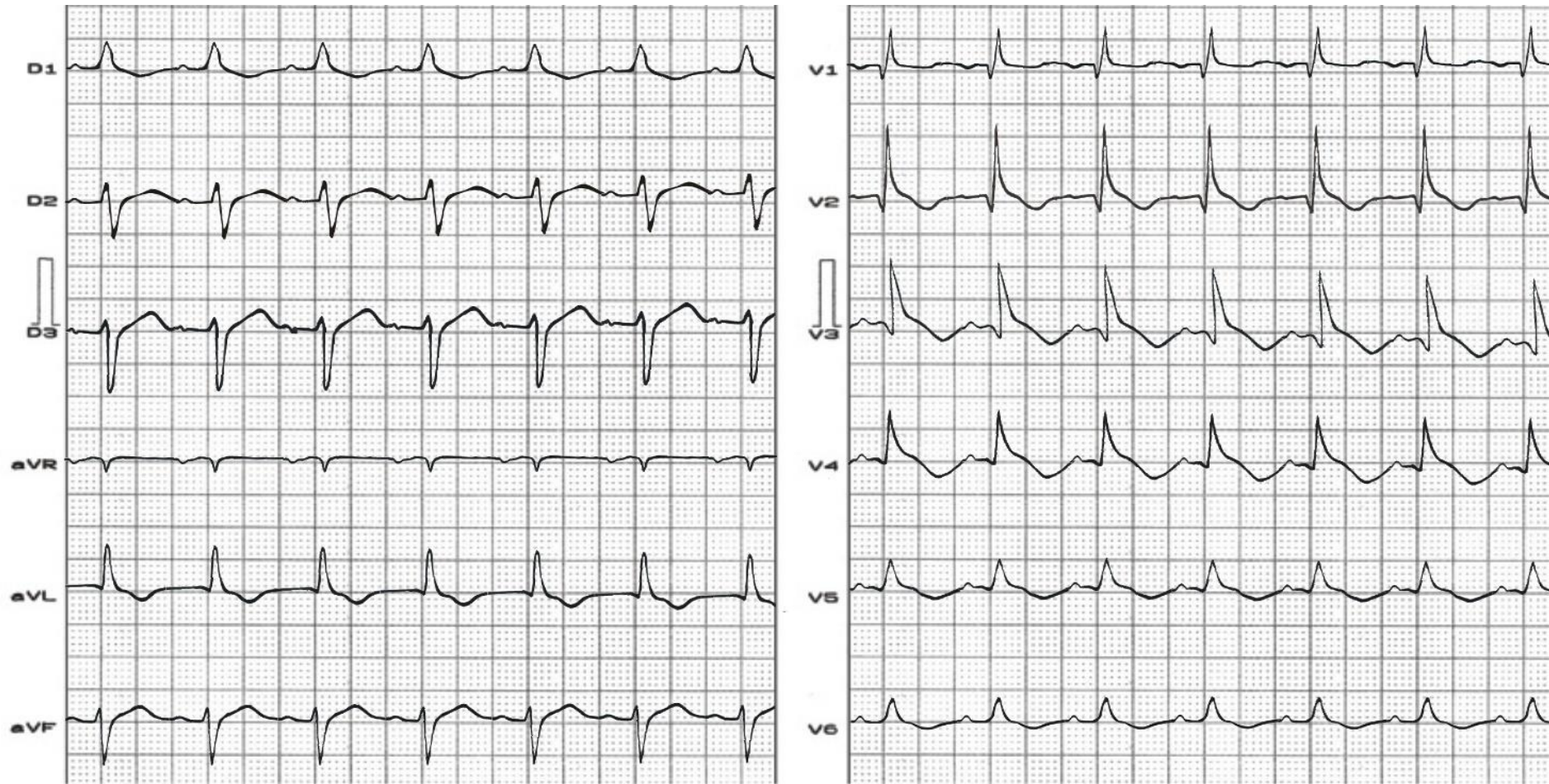
1. First degree AV block
2. Second degree AV block Mobitz type I (Wenkebach) in the second ECG and pseudo 2:1 2nd degree AV block?)
3. LAFB: extreme left axis deviation  $S_{III} > S_{II}$
4. LSFB: qR pattern, Increased intrinsicoid deflection of  $V_1$  and  $V_2$ , R wave “in crescendo”, R wave voltage of  $V_1 \geq 5$  mm, small q wave in  $V_2$  or  $V_1$  and  $V_2$ , R wave of  $V_2 > 15$  mm, absence of q wave in  $V_5$ ,  $V_6$  and I (by absence of the first septal vector  $1_{AM}$ ).
5. Standard masquerading RBBB
6. RBBB with septal MI: qR pattern in  $V_1$
7. First degree AV block + bifascicular left fascicular block + RBBB = tetrafascicular block (this is a new nomenclature used by us.)
8. LBBB on Holter monitoring is indicative of alternanting BBB, such as the following next three slides.
9. EPF study shows HV and AH prolongation and Holter monitoring transiente LBBB pattern.



*Acute extensive anterior myocardial infarction associated with standard masquerading bundle branch block: LAFB associated with RBBB. Extreme QRS left axis deviation ( $S\hat{A}QRS -70^\circ$ ),  $SIII > SII$ : LAFB. The limb leads show an atypical LBBB-like pattern (isolated r wave in I and aVL), but the right precordial leads show a RBBB.*

## II. The precordial type (“*precordial masquerading right bundle-branch block*”)

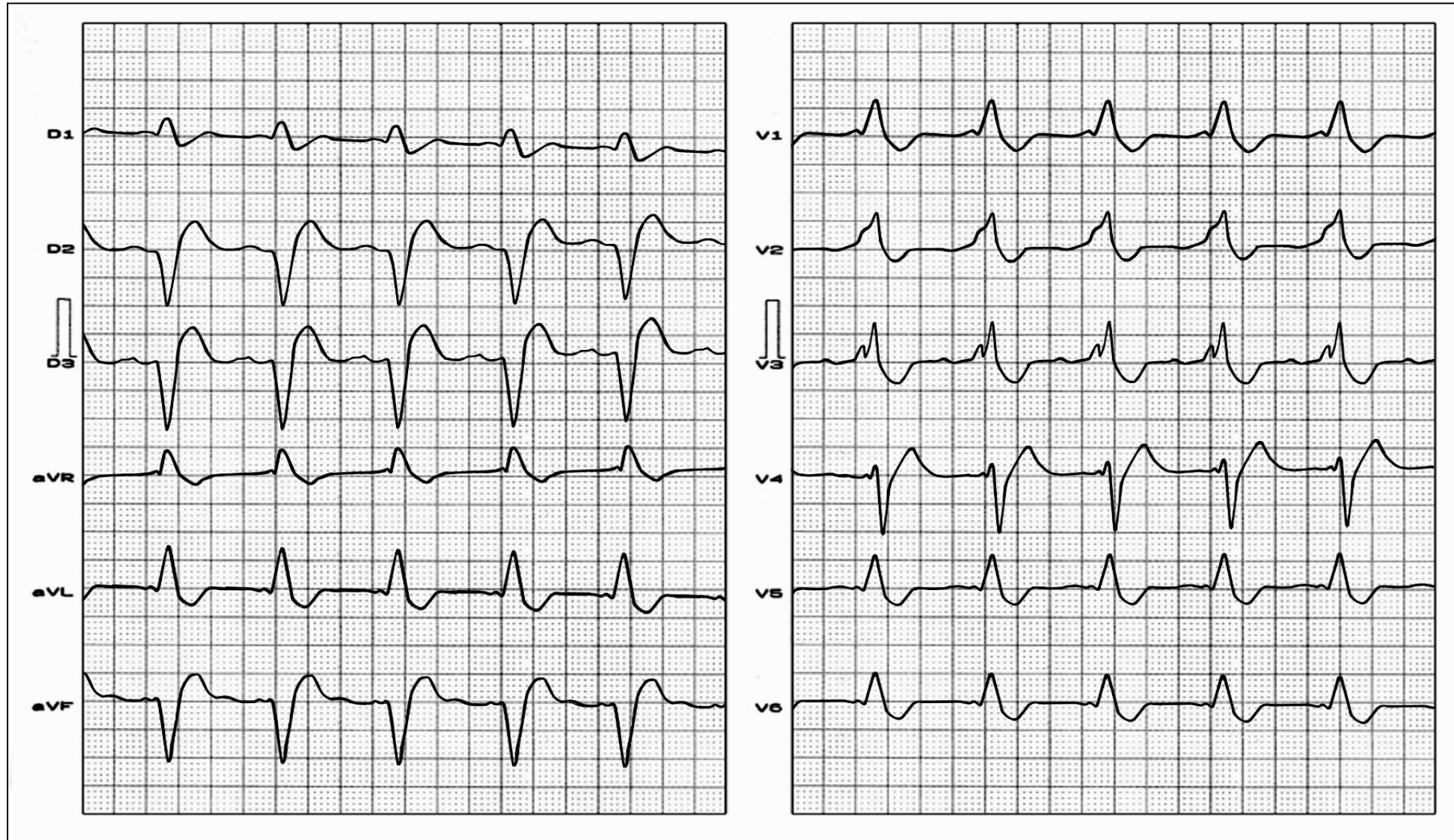
This type shows the pattern of CRBBB in the right precordial leads and complete left branch block pattern (CLBBB) in the left-side precordial leads. This result from CRBBB associated with severe left ventricular hypertrophy/enlargement (LVH/LVE), a localized block in the anterolateral wall of the left ventricle often due to myocardial infarction, and usually LAFB. Presumably, the intramural left ventricular block, together with the LVH or the LAFB, or both, produce predominant leftward forces which tend to cancel out the late rightward forces of the RBBB in the left precordial leads. Finally, masquerading bundle-branch block can be associated with severe and diffuse conduction system disease, and that patients with this finding may require permanent pacemaker implantation, especially if they are symptomatic (**Kowey 1989**).





### III. The Standard and Precordial masquerading bundle-branch block in association

In this case the limb leads show an apparent Left bundle-branch block pattern with extreme left axis deviation (LAFB) and the precordial leads exhibit the pattern of CRBBB in the right precordial leads and LBBB pattern in left precordial leads V5-V6. Additionally, an abnormal Q waves are frequently present on right precordial leads



## Case report

RMC female patient, 36 years old, Caucasian, married, housewife, basic education, born and raised in Cametá, Pará (PA) Brazil.

**Main complaint:** tiredness on mild exertion and leg swelling for four months.

For the past four months she began displaying symptoms of chest discomfort not related to exertion, epigastric pain and fatigue with great efforts initially, progressing rapidly to moderate and slight, followed by the appearance of swelling in the legs and abdomen. One day before the consultation she had been notified by the municipality sanitary authorities as a carrier of Chagas disease and due to complaints was referred to the local tertiary referral Hospital for cardiological evaluation.

At the time when seeking medical attention she was diagnosed with congestive cardiac heart failure and treated with association of furosemide 80mg/daily, spironolactone 25 mg, enalapril maleate 20 mg 2 x daily, and carvedilol 25 mg 2 x day. Enalapril maleate was suspended in a few days due to presenting very low blood pressure. At the time of consultation the patient was in New York Heart Association (NYHA) functional class III even on optimized medication. (Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m). Comfortable only at rest.).

**Personal history:** denied hypertension, diabetes mellitus, dyslipidemia, smoking, or any other addiction.

**Family history:** Nothing of note.

**Epidemiological background:** In spite of residing in an urban area, her house was made of wood and mud roofed and with no backyard. Food habits: red meat, poultry and fish. She frequently ate the “açai” fruit (*Euterpe oleracea*) bought in street booths.

# Physical Examination

**Vital signs:** blood pressure 84/56 mmHg, heart rate 96 lpm, dyspnea at rest +++/4, no fever, pale skin and mucous membranes ++/4, acyanotic.

**Neck:** Distended neck veins, JVD to 12 cm. Carotids without bruits.

**Lungs:** pulmonary auscultation: bilateral vesicular murmur. Absence of adventitial sounds.

**Heart:** visible and palpable ictus cordis in the sixth intercostal space on anterior axillary line, not covered with two fingertips. Arrhythmic heart sounds, holosystolic murmur Grade 3/6 in mitral focus, radiated to the armpit; protodiastolic murmur audible in the aortic and accessory aortic focus. Systolic murmur of tricuspid regurgitation and third heart sound with gallop cadence.

**Abdomen:** Liver palpable five centimeters below right costal margin and slightly tender. HJR+. Non-tender to palpation, +Bowel sounds 4 quadrants.

**Extremities:** 4+ pitting edema of lower limbs up to the knees. Nail beds minimally cyanotic, no clubbing. Pulses present, arrhythmic and filiform.

**Positive Chagas antibody tests**

**ECG**

**Figure 1**

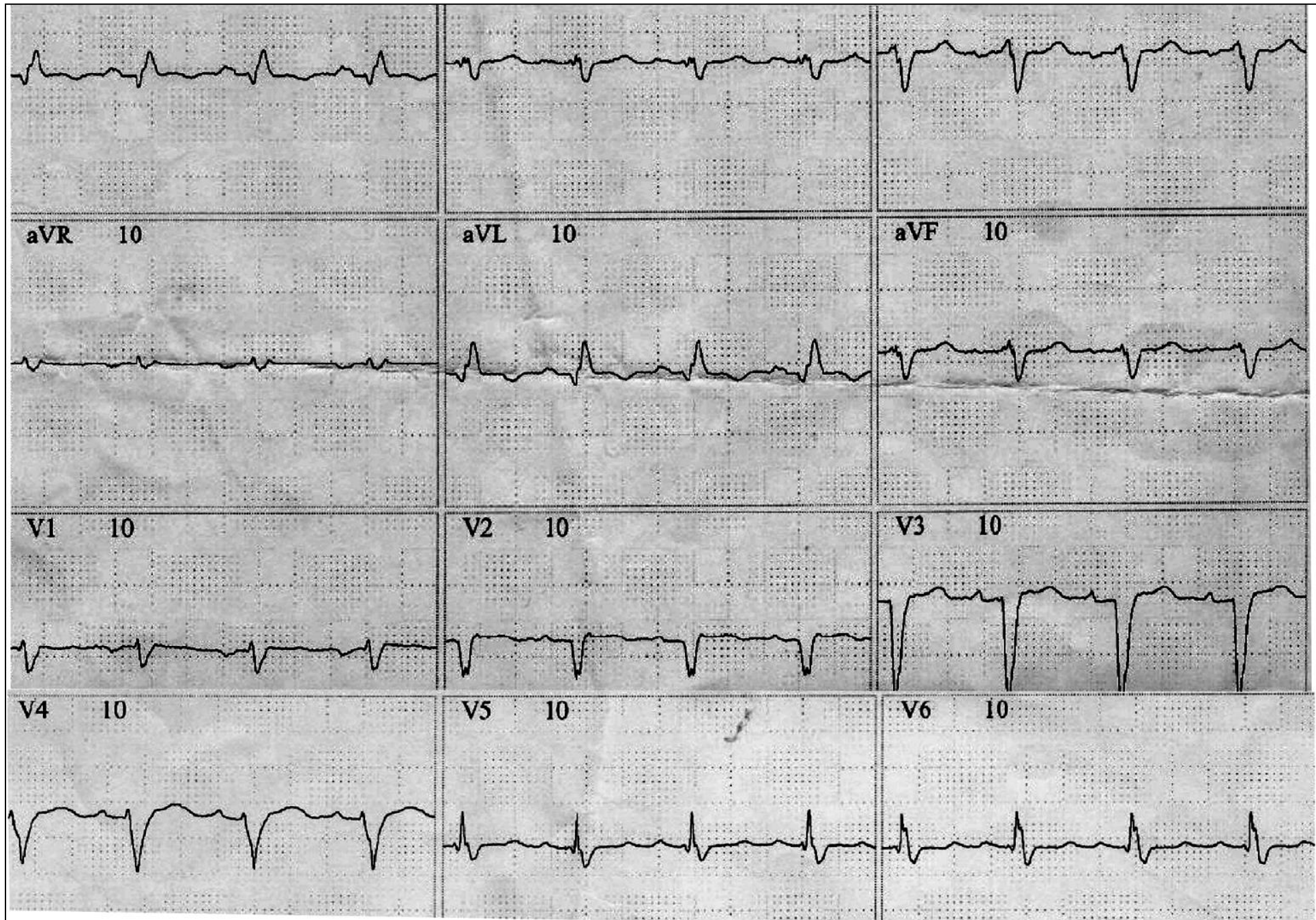
**Echo:** significant increase in diameter of the left ventricle and left atrium, severe diffuse hypokinesis, significant degree of mitral regurgitation, mild aortic reflux, minimal pericardial effusion and pulmonary artery systolic pressure estimated at 56 mmHg

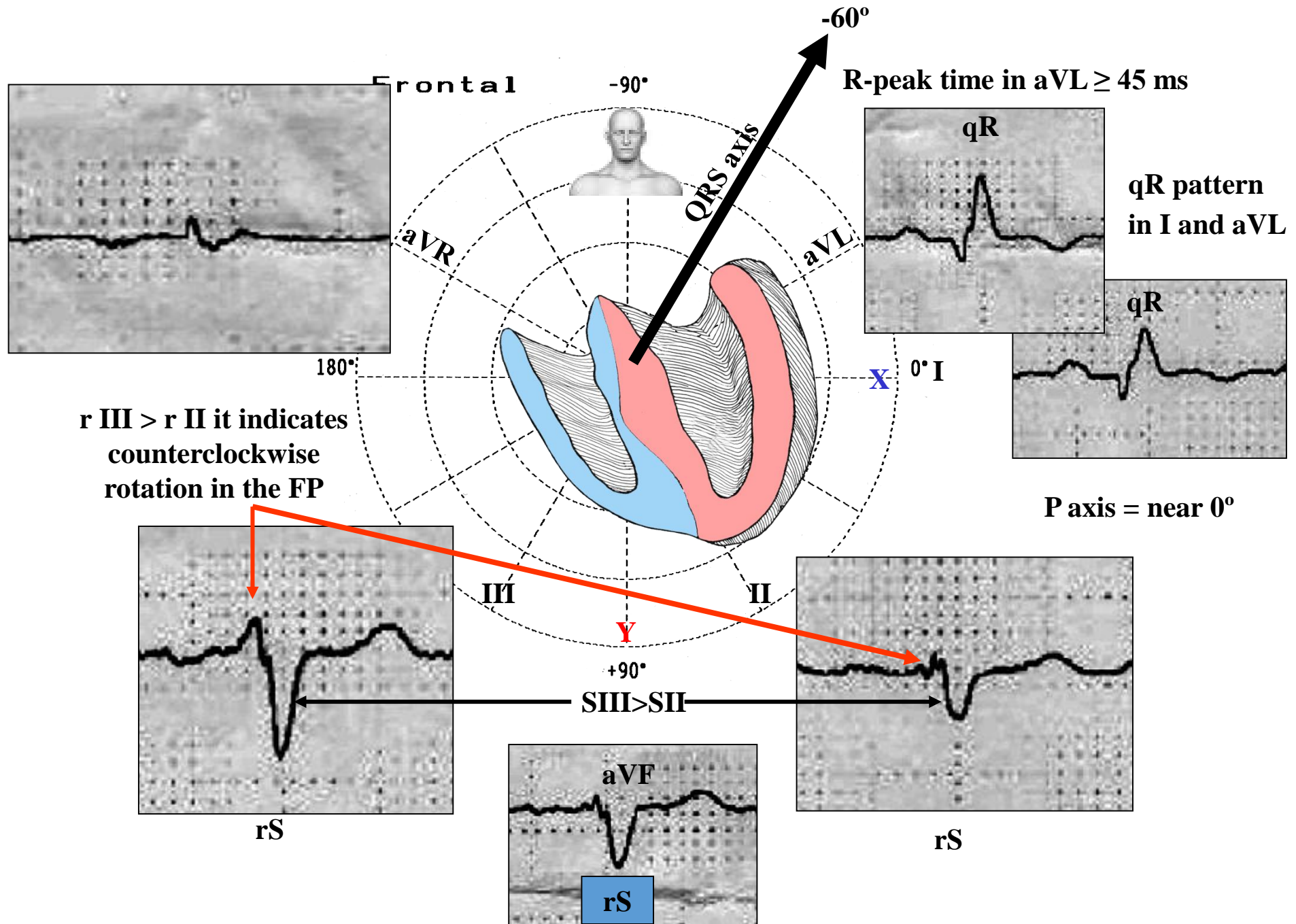
**Holter Monitoring:** 676 polymorphic PVCs, short non-sustained VT, 359 supraventricular premature contractions and wide permanent QRS duration complexes.

**Management:** Optimization of medication and repeated the echocardiogram Doppler, which revealed a thrombus in the apex of the left ventricle without changing other parameters. The anticoagulant warfarin 5mg/day was added.

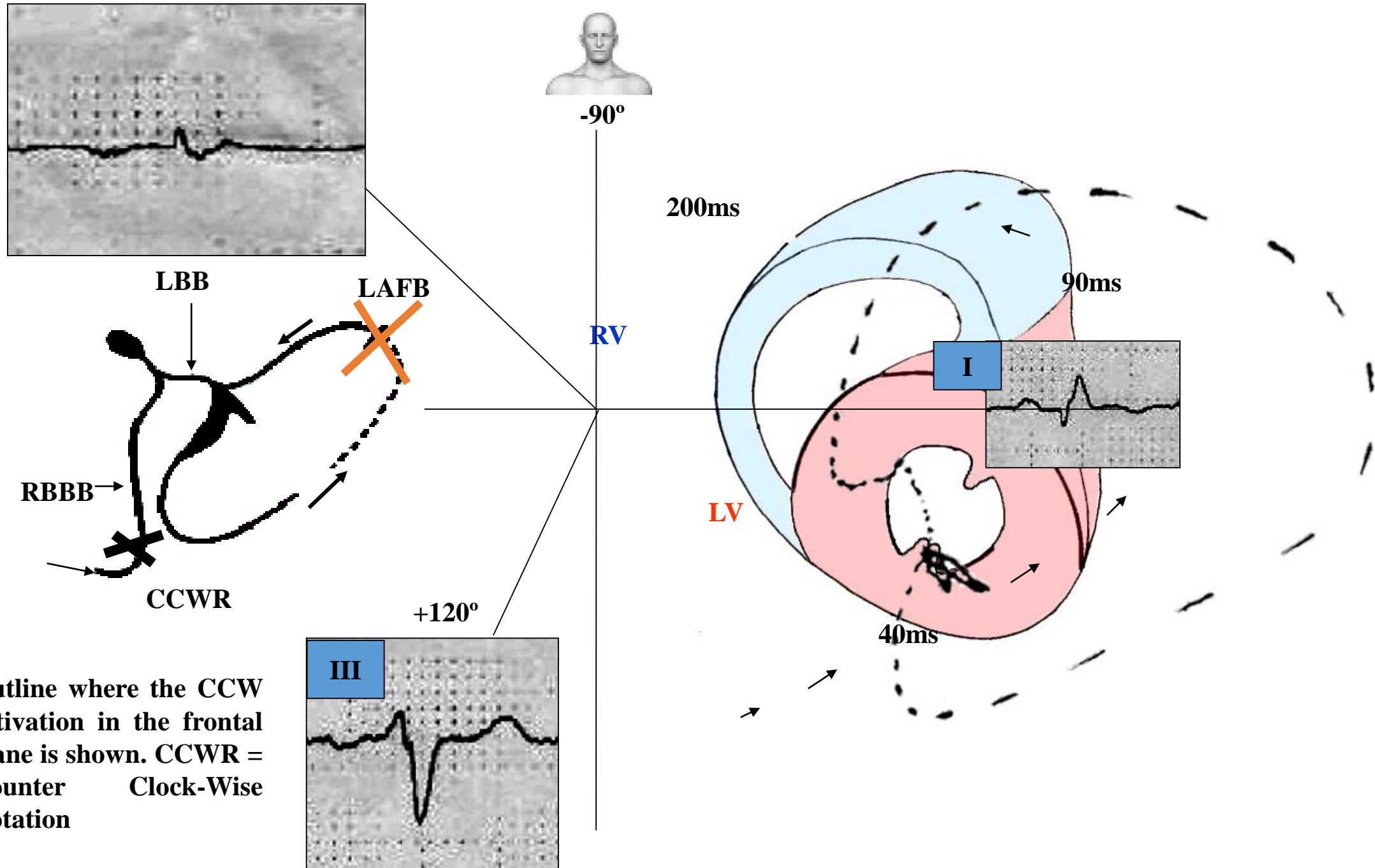
**Evolution:** Refractory Cardiac heart failure with anasarca, jaundice, low output and death from ventricular arrhythmias within a few days.

**Figure 1** Which is the ECG diagnosis?

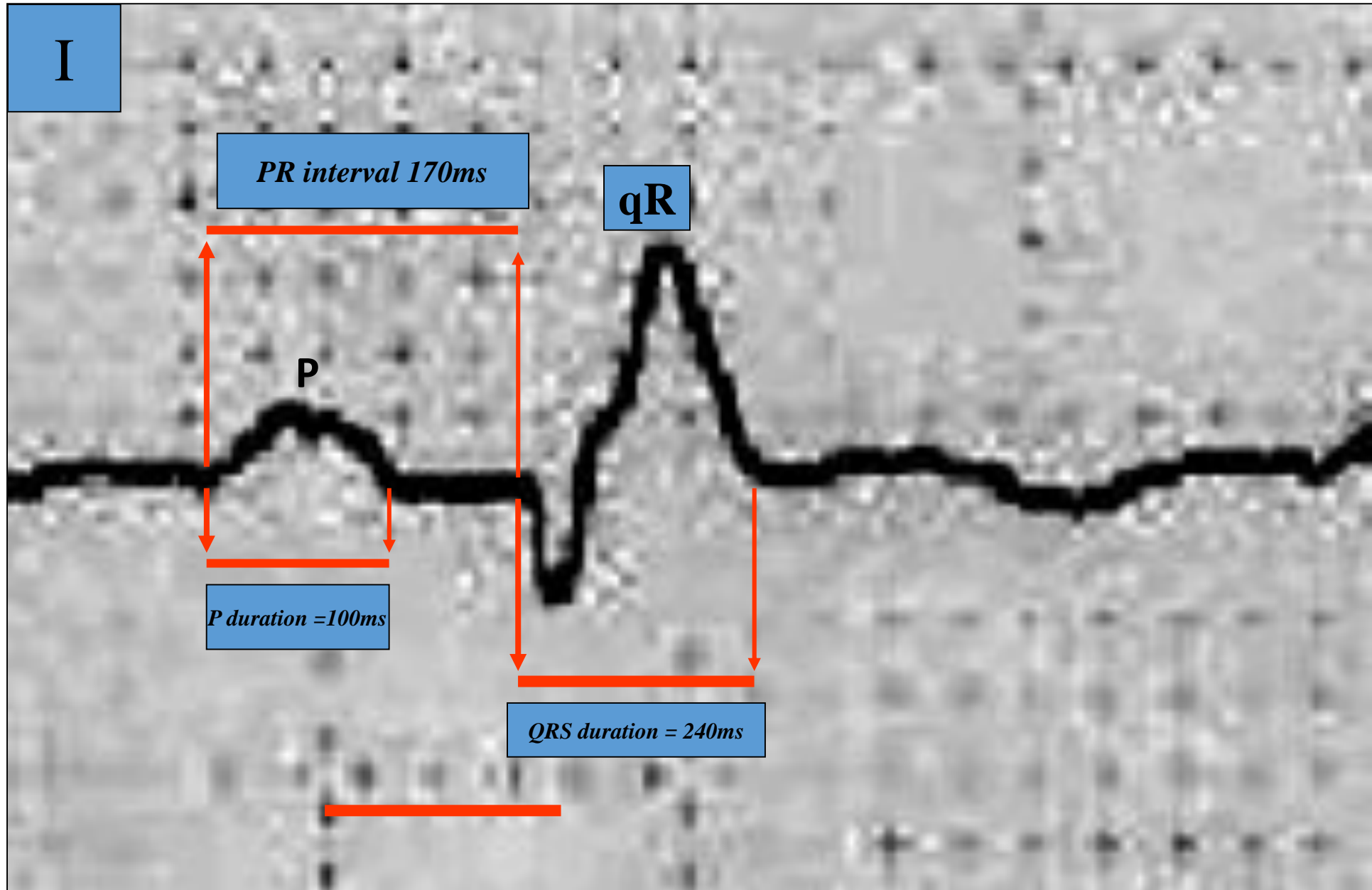


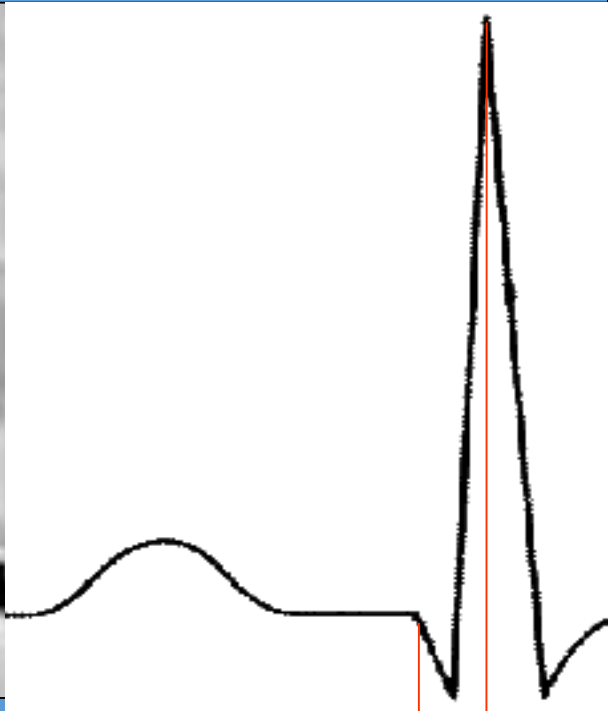
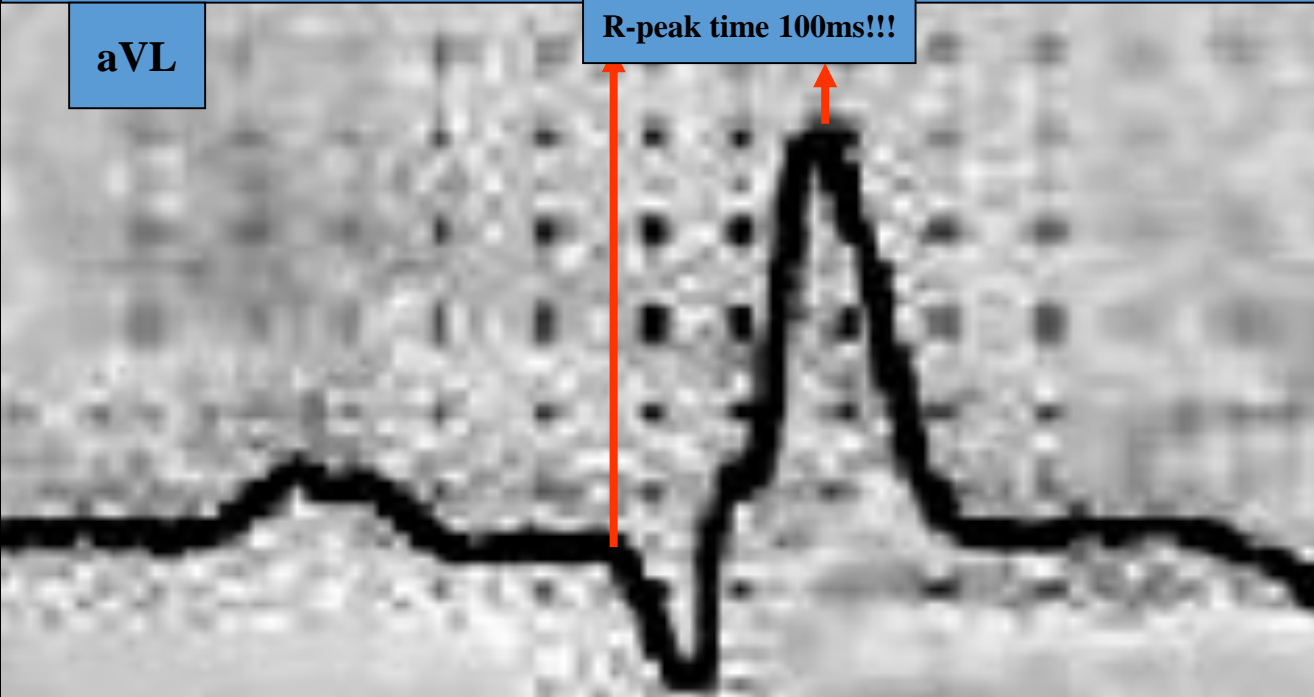


# Hypothetical ventricular activation in FP



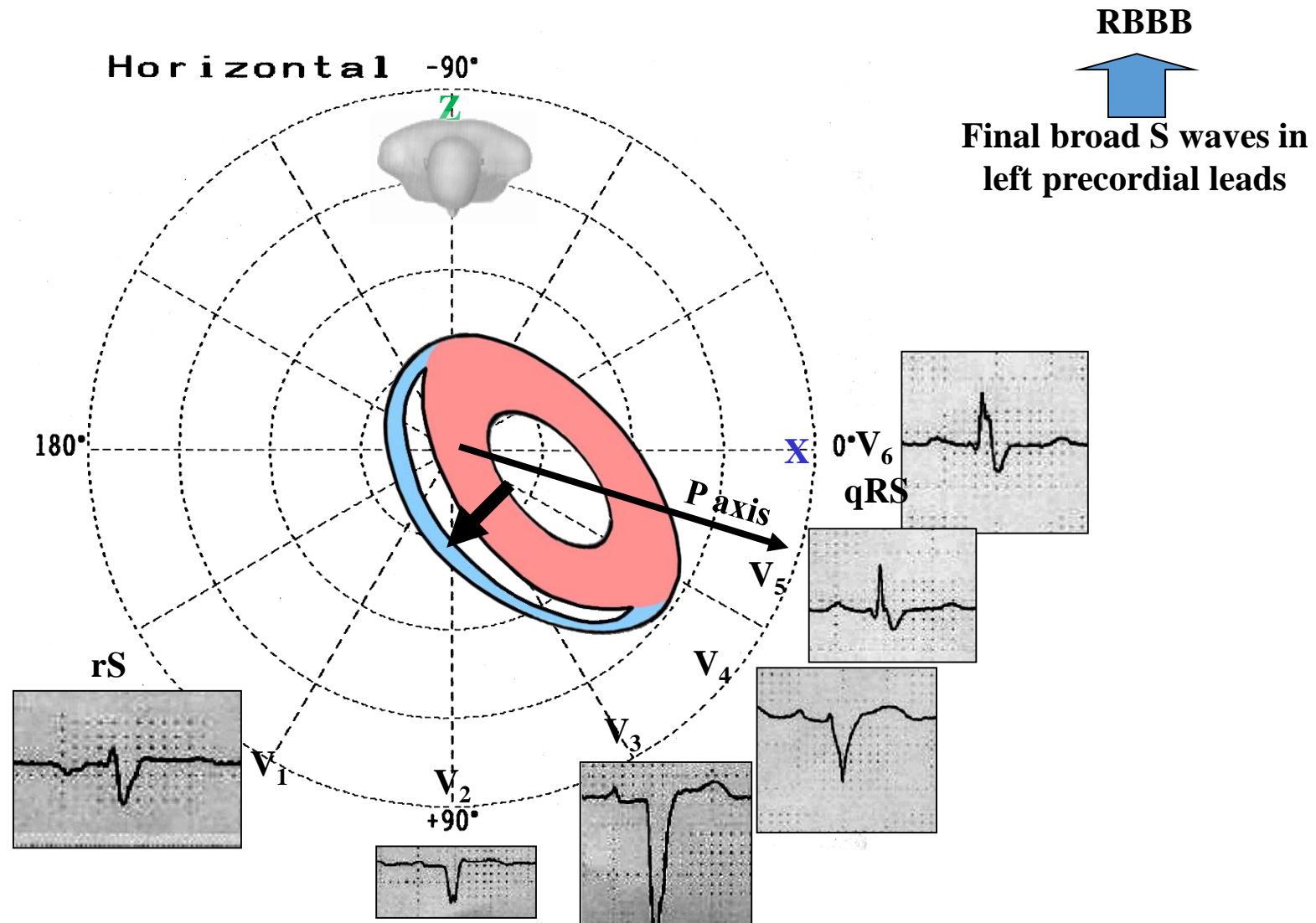
# P wave, PR interval and QRS complex duration





*R-peak time, ventricular activation time (VAT) or Intrinsicoid deflection in aVL  $\geq 45$  ms*

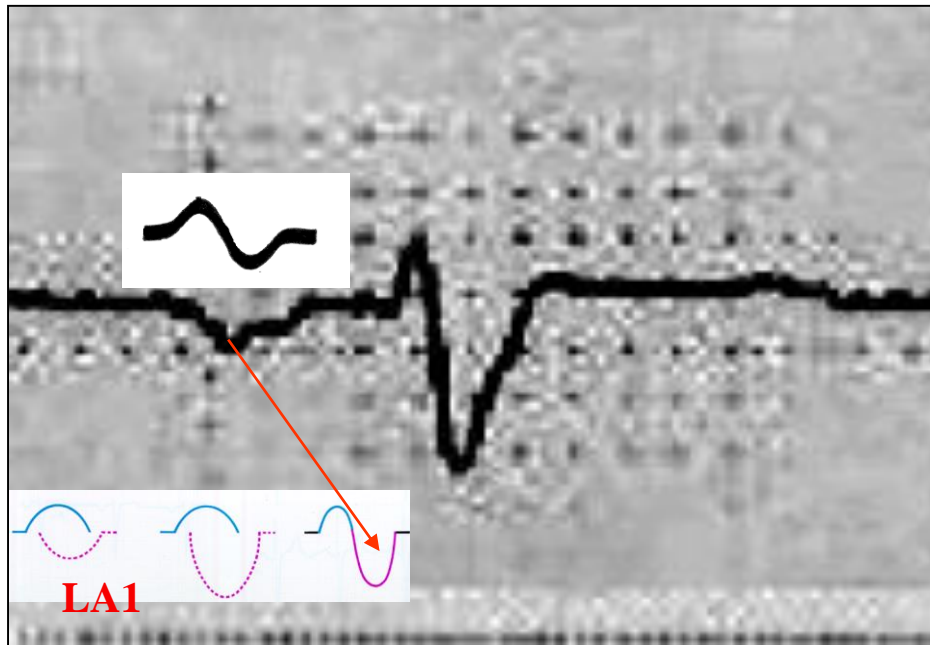




Impossible CRBBB diagnosis on right precordial leads.

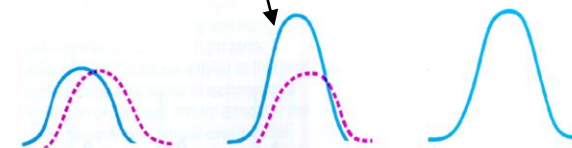
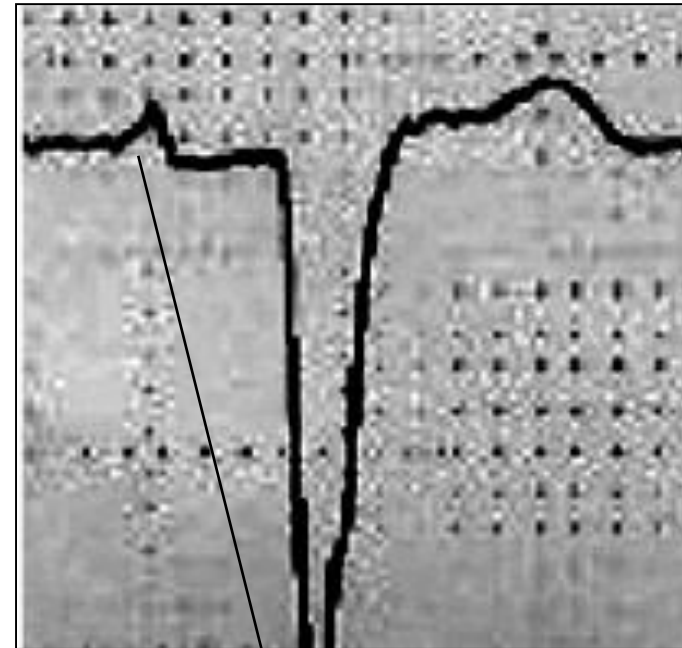
Preserved first vector. Fibrosis low septal and free lateral wall

1. Association P axis in  $0^\circ$  in FP with P axis to front (left anterior quadrant) in HP = biatrial enlargement
2. Significant negative final P wave component in V1+ positive 1mm P wave in V3 is suggestive of biatrial enlargement

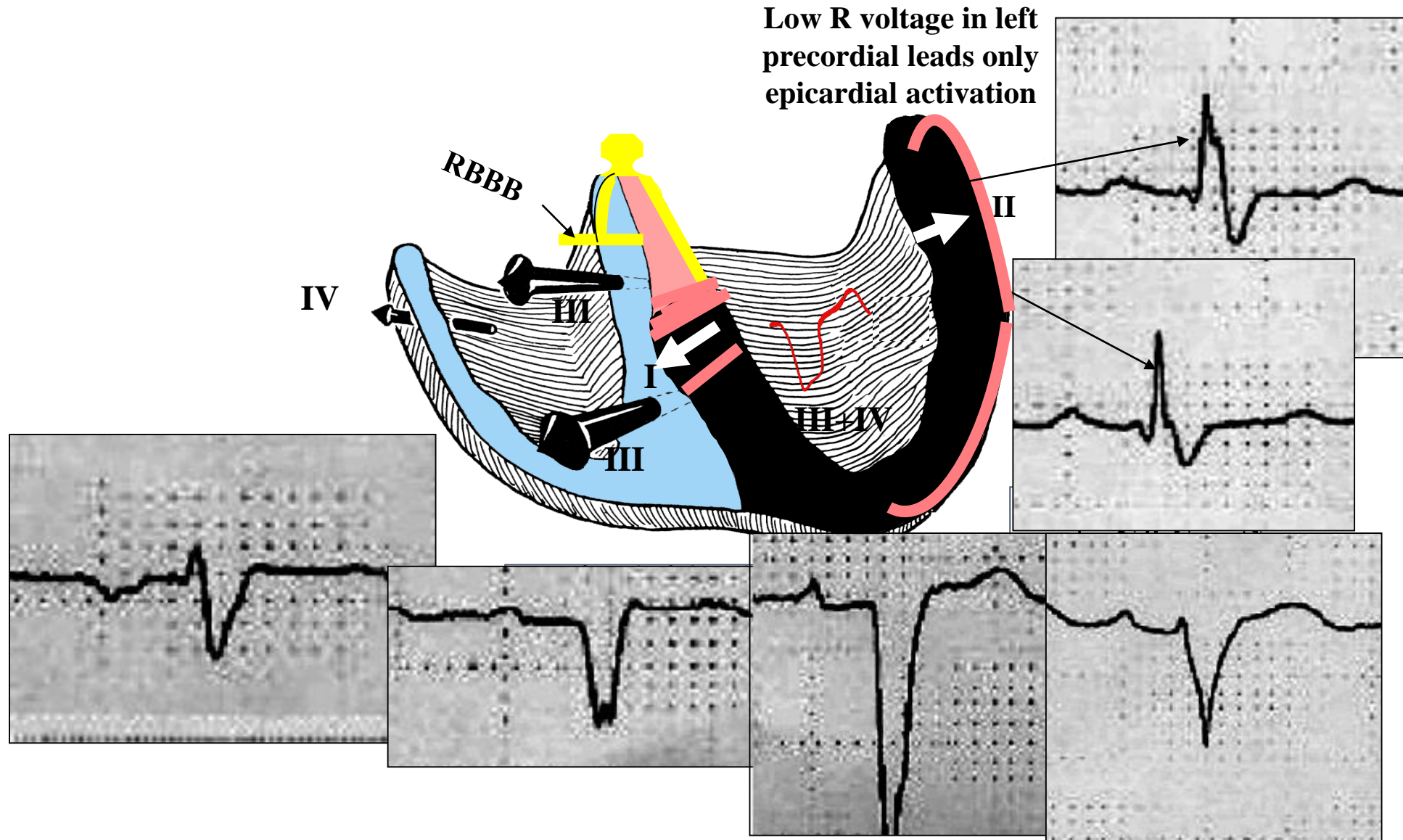


**LA2/:** final deep and slow component: **LAE**

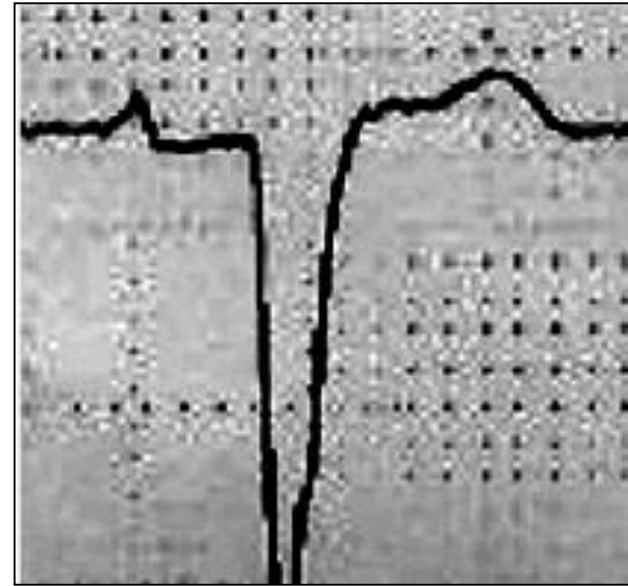
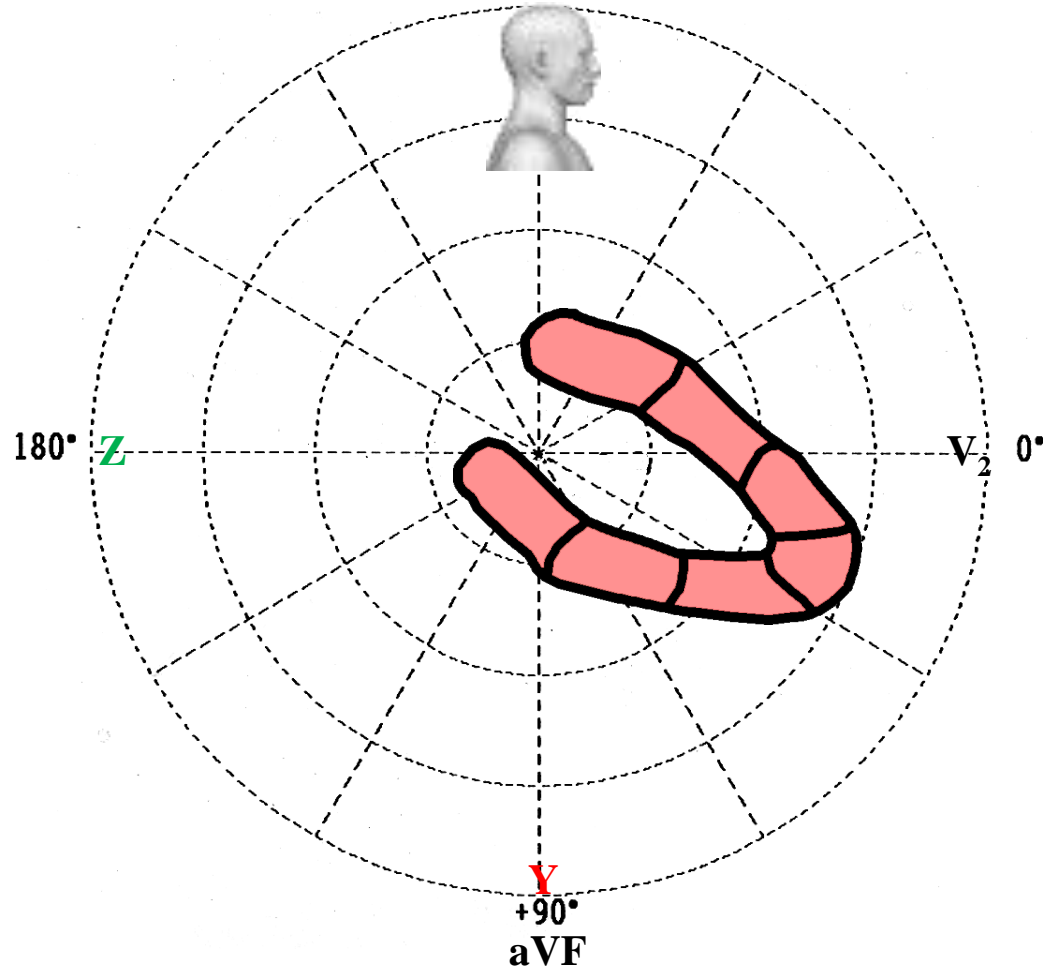
$\geq$  the area of one small square the final minus portion indicates left atrial enlargement, abnormality or advanced interatrial block



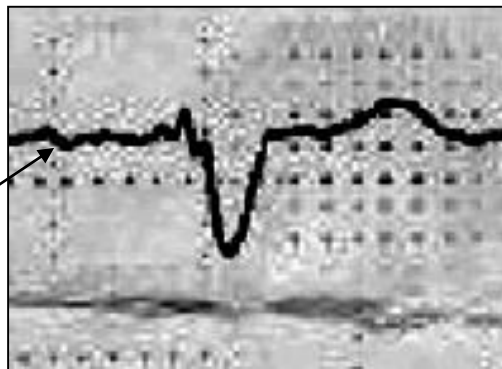
# Complete RBBB complicated with low septum and free wall extensive fibrosis



Sagittal -90°



P wave axis perpendicular to +90°



# ECG analysis Figure 1

**Rhythm:** Sinus rhythm

**Heart rate:** 82bpm.

**P wave:** P axis near  $0^\circ$  (LAE), P duration = 100ms, notched P wave in II and peaked P wave in V3 (RAE) = Suggestive of biatrial enlargement?

**PR interval:** 170ms. Normal.

**QRS axis:** axis  $-60^\circ$  Extreme left axis deviation. QRS duration (QRSd) = 240ms very broad.

**QRS voltage:** Low QRS voltage complexes in the frontal plane (FP) no wave exceeds 5 mm (one large square or 5 small squares, vertically). In the FP it is considered low voltage. In the horizontal plane no wave exceeds 10 mm: QRS low voltage un both planes. Why? consequence of severe universal fibrosis.

**Left Anterior Fascicular Block (LAFB):** QRS axis  $-60^\circ$ , isodyphasic QRS complexes in aVR (QRS perpendicular to aVR), negative QRS complexes in inferior leads with rS pattern,  $r_{III} > r_{II}$ ,  $S_{III} > S_{II}$ , and qR pattern in I and aVL, prolonged R peak time

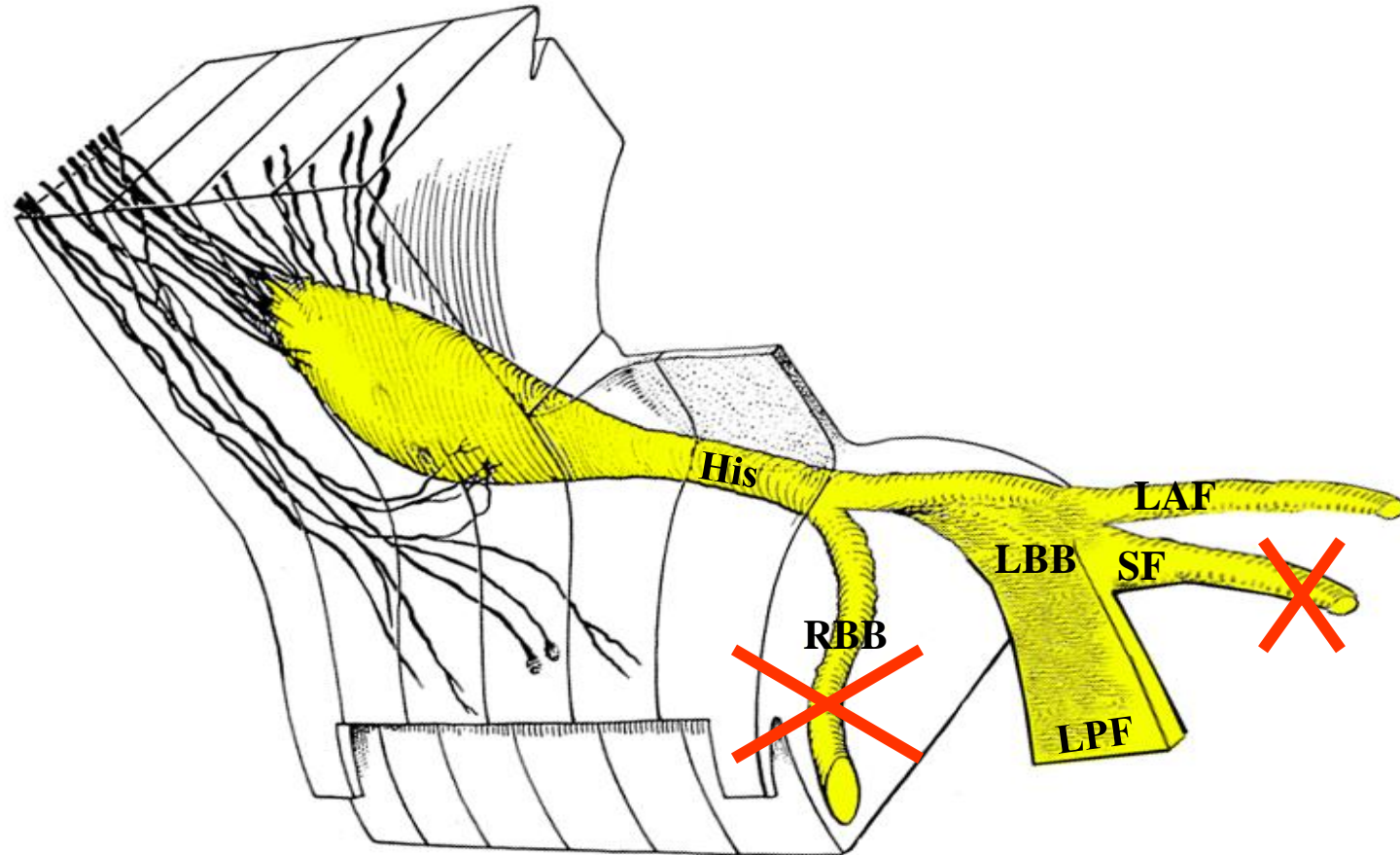
*Electrically inactive low and inferior septum with lateral wall extension( Low R voltage waves V5-V6).*

*“Standard Masquerading Right Bundle Branch Block”* this is an atypical form of RBBB with LAFB where S wave in I and aVL becomes very small or disappears, the limb leads may resemble LBBB.

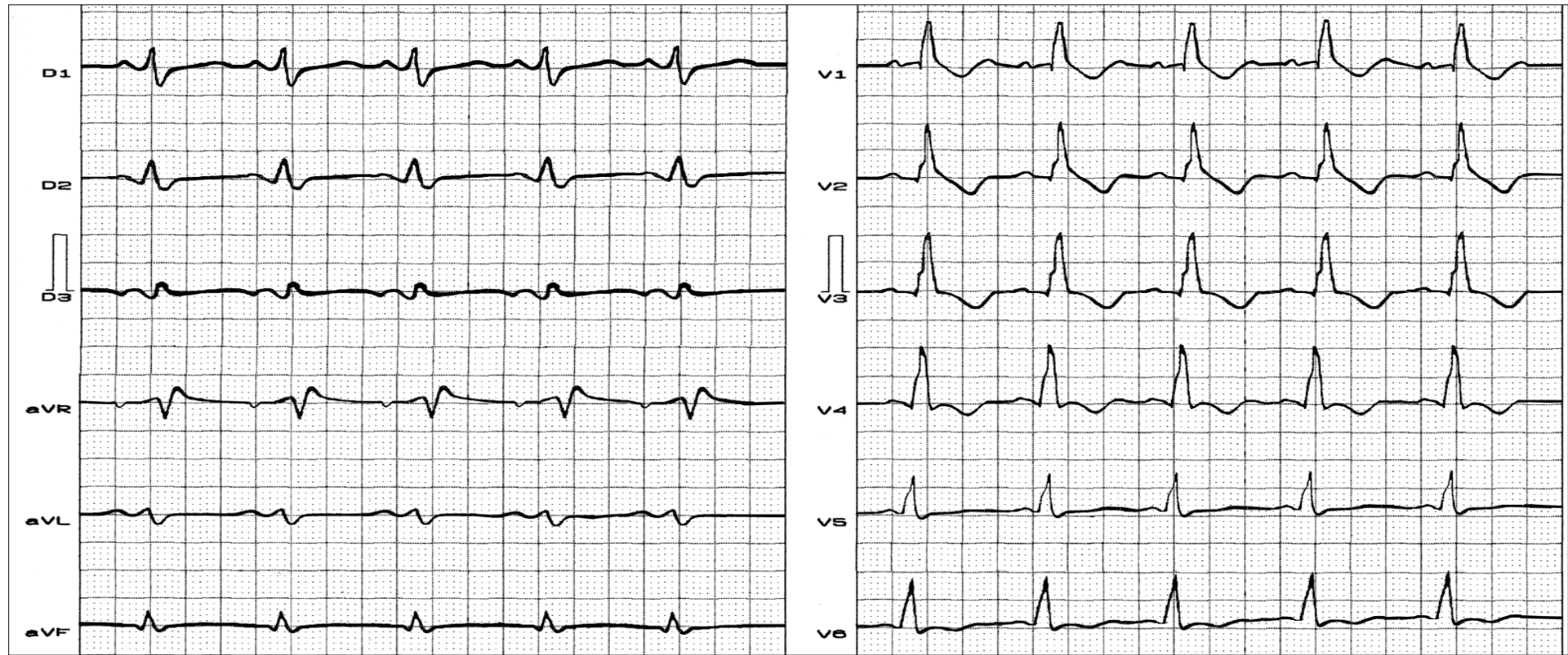
In other words, LAFB obscured totally the diagnosis of CRBBB only in FP by abolishing the broad S final wave in left standard leads I and aVL.

In the unipolar anterior precordial wall leads from V1 to V4 as a consequence of low septum anterior electrically inactive area the Complete RBBB is almost totally hidden and is reflected by a Rs, QS or Qr pattern in these leads. The presence of Complete RBBB could be register by recording high right-sided chest leads (**Sclarovsky 1979**). The final wide S waves in left precordial leads denounce the presence of Complete RBBB.

# CRBBB associated to SFB



**Name:** AB; **Date:** 07/10/1988; **Age:** 45 yo.; **Gender:** M.; **Race:** W.; **Weight:** 70 Kg.; **Height:** 1.70 m.;  
**Biotype:** athletic; **Medication in use:** nothing stated.

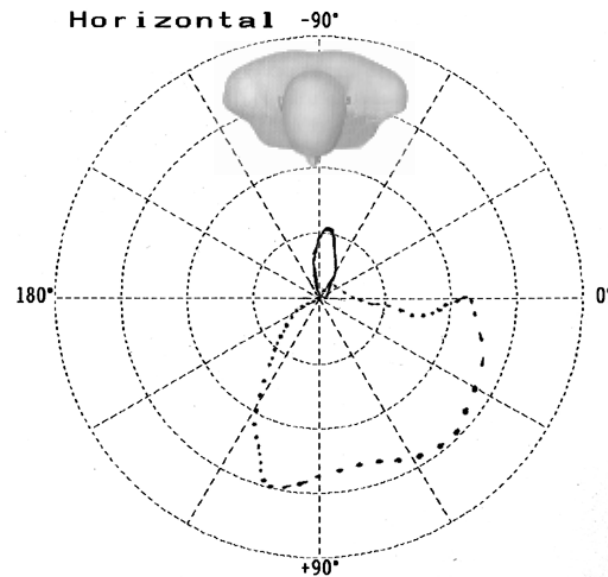
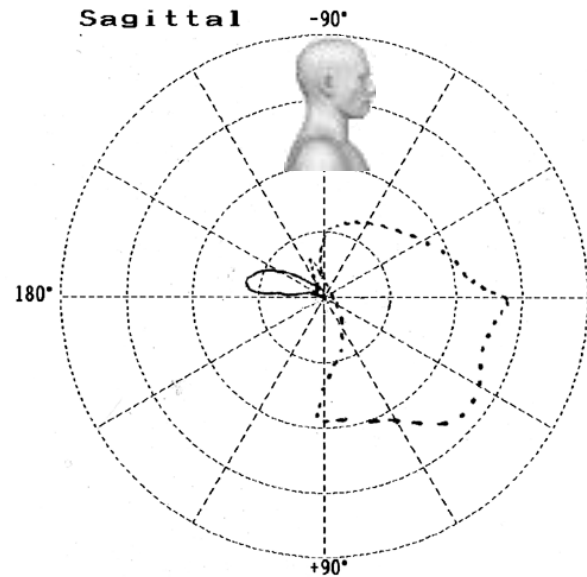
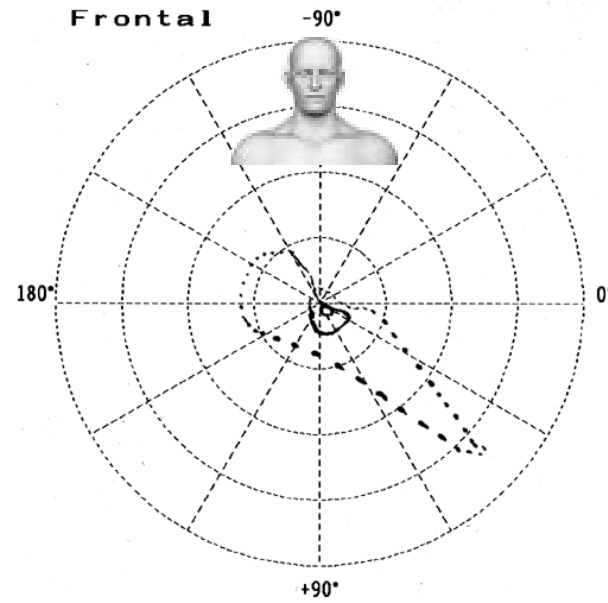


**Clinical diagnosis:** chronic chagasic cardiomyopathy, dromotropic form.

**ECG diagnosis:** CRBBB + SFB = bifascicular block. Sinus Rhythm; HR: 79 bpm; P WAVE:  $\hat{S}\hat{A}P$  close to  $0^\circ$  and to the front; PR interval: 170 ms;  $\hat{S}\hat{A}QRS$ : perpendicular to the frontal plane, duration: 220 ms, morphology: broad S from I and aVL, qR from  $V_1$  to  $V_3$  with peaked R waves and without the plateau proper of CRBBB. Broad descending branch of  $V_2$  and  $V_3$ . intrinsicoid deflection in  $V_2$  < 50% of total duration of QRS.

Rs waves from  $V_4$  to  $V_6$ , voltage of R grows from  $V_1$  to  $V_2$  and  $V_3$  and decreases from  $V_4$  to  $V_6$ , absence of q in  $V_5$  and  $V_6$  and s wave a little broadened and with small depth in these leads, as it would be in CRBBB in isolation.

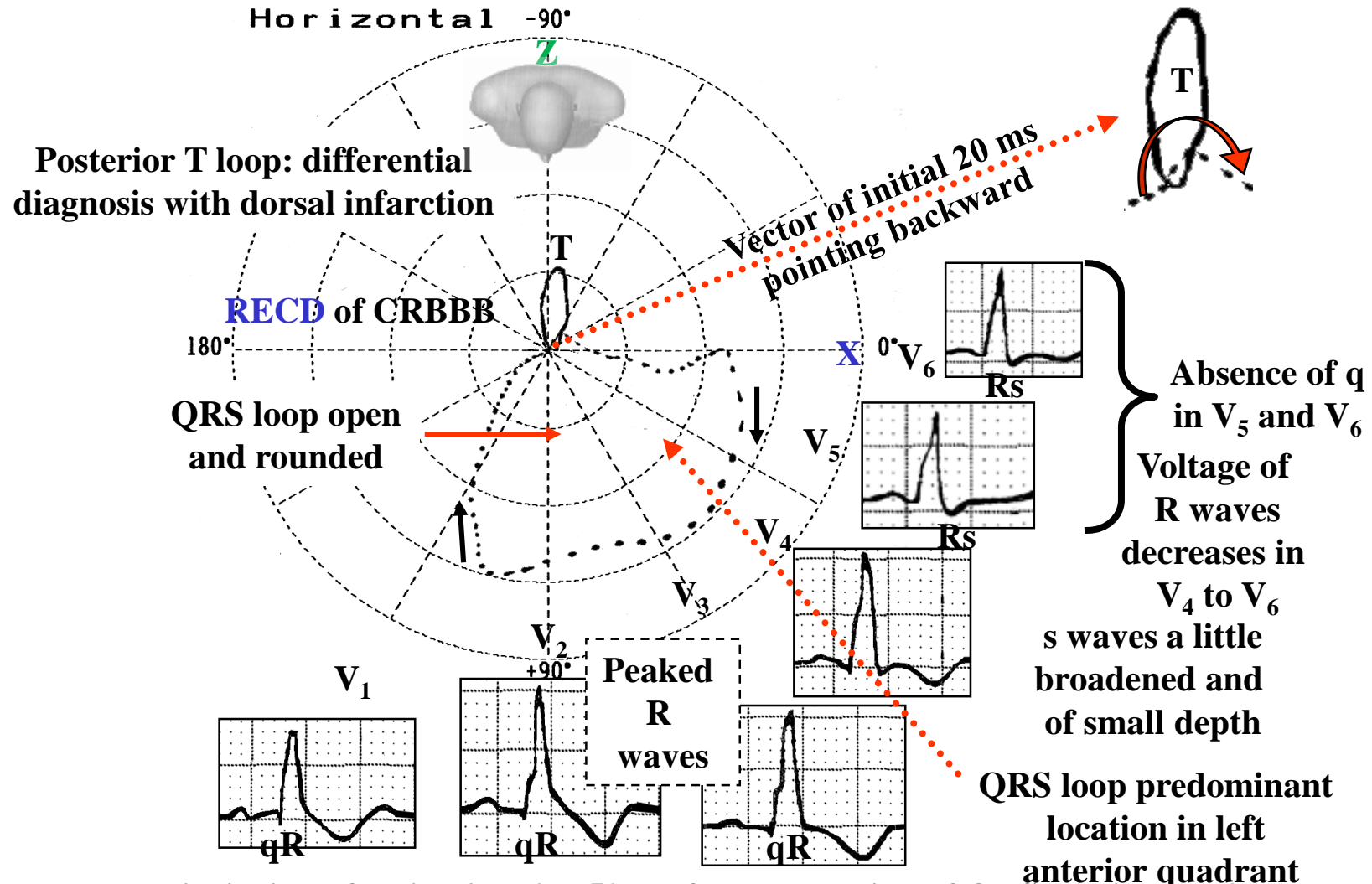
**Sensi.**        2  
**Timer**        2    msec  
**Loop**         All Loop  
**Sagittal**      Right  
**Z Axis**        Back  
**Filter**        Hum  
                   Muscle  
                   Drift



VCG in the three planes, where in the horizontal plane we observe typical CRBBB associated to SFB. Vectors of initial 10 ms pointing backward; loop almost completely located in the left anterior quadrant. The frontal plane shows ECD in the right superior quadrant.



# ECG/VCG correlation on HP (SFB + CRBBB)

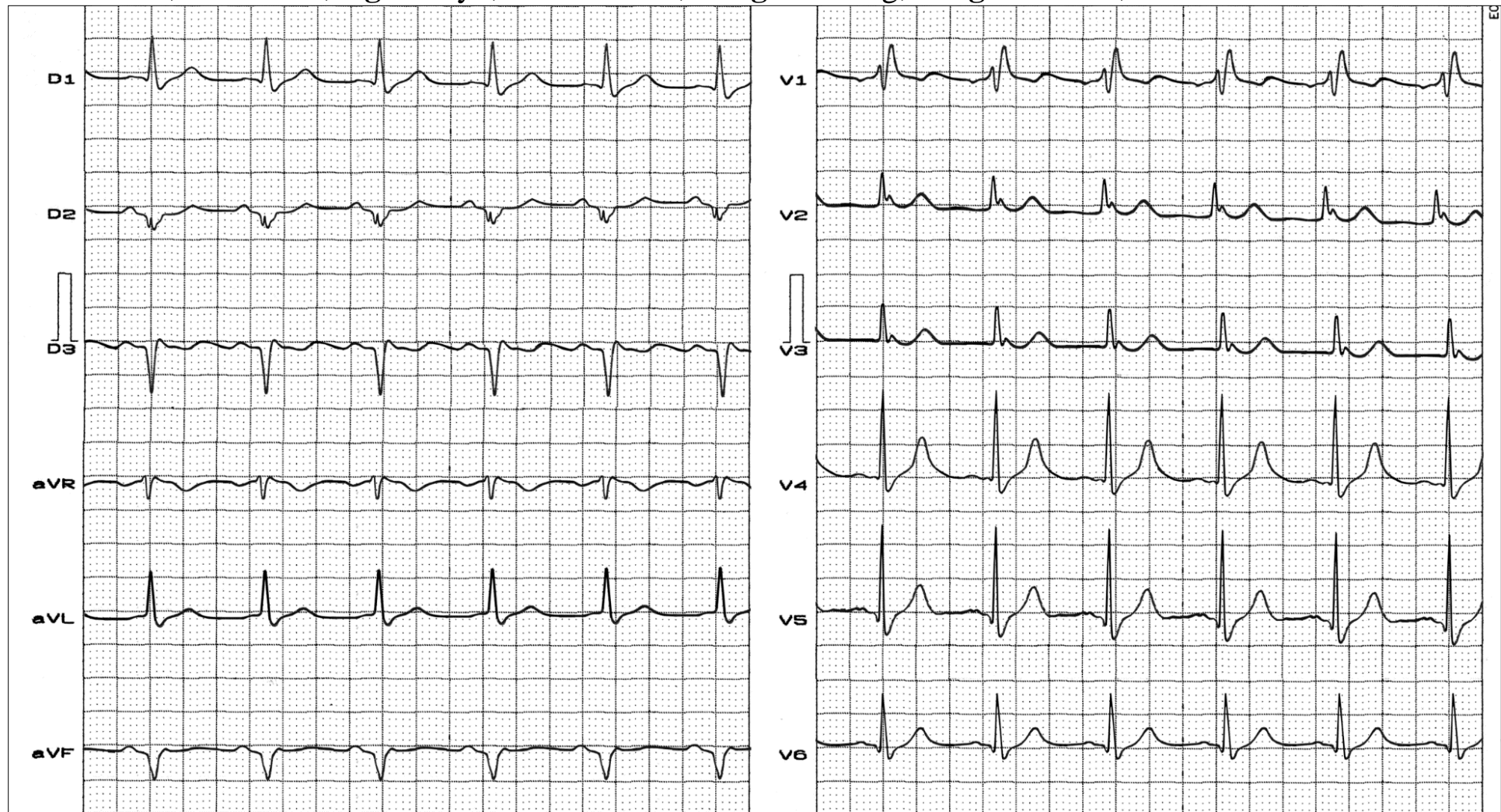


Intrinsicoid deflection in V2 < 50% of total duration of QRS R V2 > R V3

ECG/VCG correlation in the horizontal plane that shows association of CRBBB and SFB: vectors of initial 20 ms heading backward, QRS loop open and rounded of clockwise rotation and predominantly located in the left anterior quadrant. Leads V1, V2 and V3, qR pattern with peaked R waves. Absence of q wave in V5 and V6, voltage of R waves decreasing from V4 to V6 and S waves a little broadened.

**CRBBB associated to inferior electrically inactive area and LAFB**

**Name:** DS; **Sex:** Male; **Age:** 65 yo; **Race:** White; **Weight:** 80 Kg; **Height:** 1.72m; **Date:** 19/09/1994

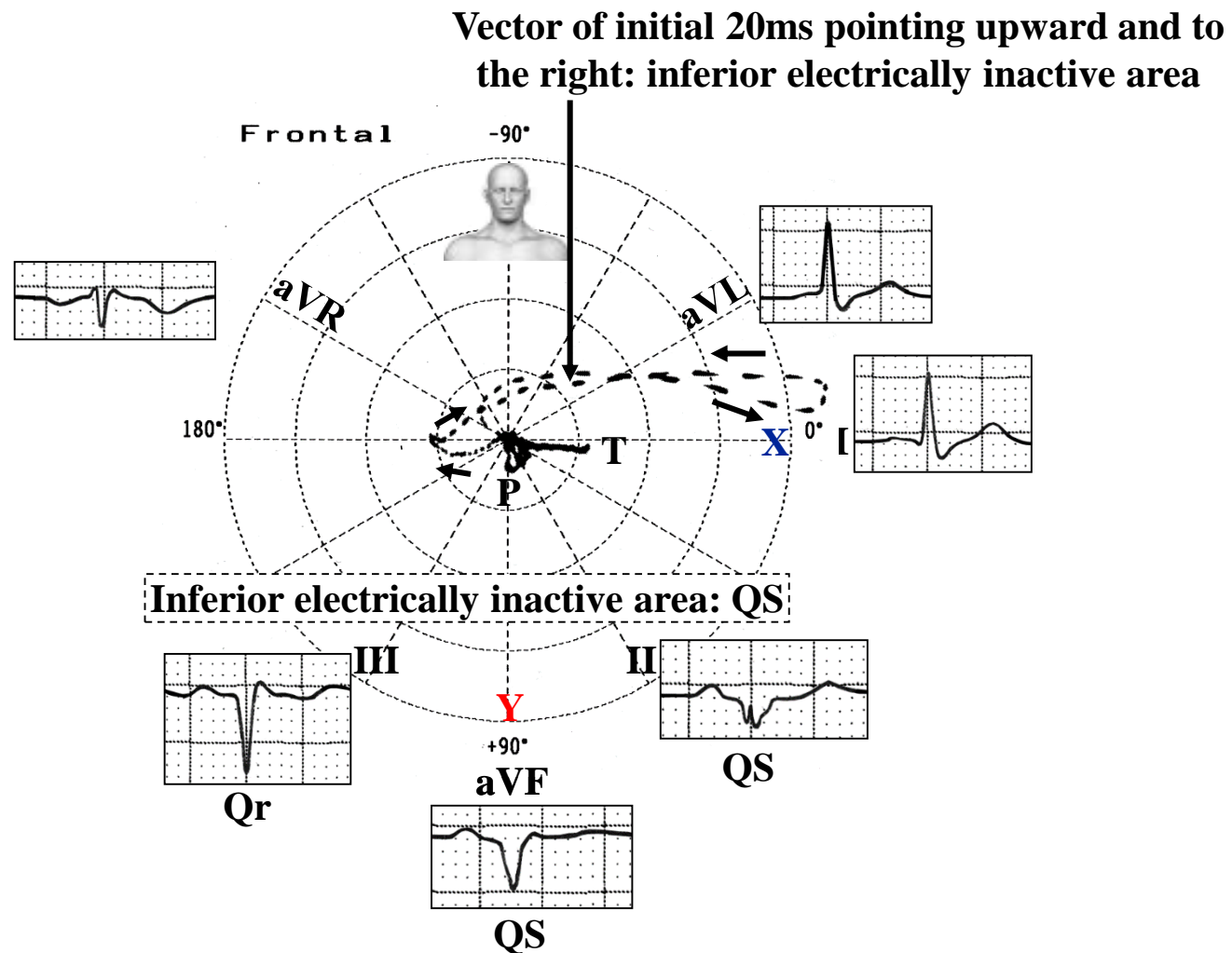


**Clinical diagnosis:** Coronary insufficiency; myocardial infarction two years ago.

**ECG diagnosis:** electrically inactive area in inferior wall: abnormal Q wave (II, III and aVF) associated to Complete RBBB. rsr' in V1 with broad final S wave in left leads.

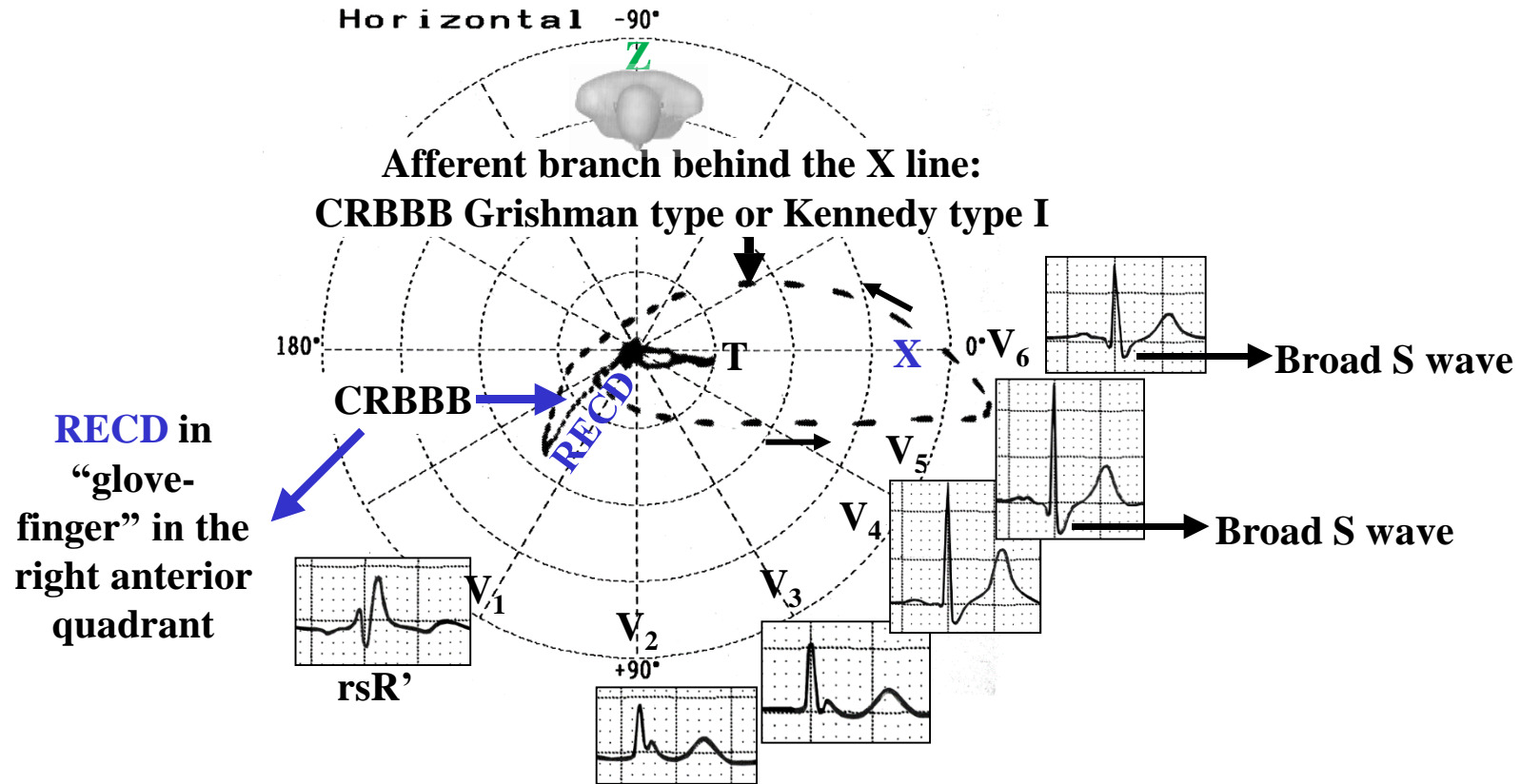
ECG of patient with coronary disease that shows CRBBB and inferior electrically inactive area with extreme deviation of the axis in superior quadrants, indicating the possibility of associated LAFB.

# ECG/VCG correlation on Frontal Plane



ECG/VCG correlation in the frontal plane. The QRS loop shows clockwise initial rotation and final counterclockwise rotation proper of LAFB associated to inferior electrically inactive area and ECD to the right, configuring the triple association of CRBBB + LAFB + inferior electrically inactive area.

# ECG/VCG correlation on Horizontal Plane



## References:

1. Adams DA, Kellner CH, Aloysi AS, et al. Case report: Transient left bundle branch block associated with ECT. *Int J Psychiatry Med* 2014;48(2):147–153-7.
2. Ardoin KB, Moodie DS, Snyder CS. Rate-dependent left bundle-branch block in a child with propionic aciduria. *Ochsner J* 2009;9(2):65–67.
3. Barker JM, Valencia F. The precordial electrocardiogram in incomplete right bundle branch block. *Am Heart J*. 1949 Sep;38(3):376-406.
4. Bhatt A, Menon AA, Bhat R, Ramamoorthi K. Myocarditis along with acute ischaemic cerebellar, pontine and lacunar infarction following viper bite. *BMJ Case Rep* 2013;2013.pii:bcr201300336.
5. Baydar ID, Walsh TJ, Massie E. A vectorcardiographic study of right bundle branch block with the Frank lead system. clinical correlation in ventricular hypertrophy and chronic pulmonary disease. *Am J Cardiol*. 1965 Feb;15:185-94.
6. Bayés de Luna A, Torner P, Oter R, Oca F, Guindo J, Rivera I et al. Study of the evolution of masked bifascicular block. *PACE* 1988; 11: 1.517-1.521.
7. Bayés de Luna A. *Clinical Electrocardiography: A Text Books*. II Edition. New York: Futura, 1999.
8. Bayés de Luna A, Brugada J, Baranchuk A, Borggreffe M, Breithardt G, Goldwasser D, Lambiase P, Pérez-Riera AR, Garcia Niebla J, Pastore CA, Oreto Giuseppe, McKenna William, Zareba W, Brugada R, Brugada P. Current electrocardiographic criteria for diagnosis of Brugada pattern: a consensus report. *Journal of Electrocardiol*. 2012; 45:433-
9. Bussink BE, Holst AG, Jespersen L, et al. Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study. *Eur Heart J*. 2013 Jan;34(2):138-46.
10. Bussink BE, Holst AG, Jespersen L, et al. Right bundle branch block: prevalence, risk factors, and outcome in the general population: results from the Copenhagen City Heart Study. *Eur Heart J*. 2013 Jan;34(2):138-46.

11. Caird FI, Wilcken DE. The electrocardiogram in chronic bronchitis with generalized obstructive lung disease. Its relation to ventilatory function. *Am J Cardiol.* 1962 Jul;10:5-13.
12. Caird FI, Wilcken DE, Williams RS. The electrocardiogram in diffuse interstitial disease of the lung and its relation to pulmonary function. *Am J Cardiol.* 1962 Jul;10:14-9.
13. Chapman JH. Intermittent left bundle branch block in the athletic heart syndrome. *Chest* 1977;71(6):776–9.
14. Chevallier S, Forclaz A, Tenkorang J, et al. New electrocardiographic criteria for discriminating between Brugada types 2 and 3 patterns and incomplete right bundle branch block. *J Am Coll Cardiol.* 2011 Nov 22;58: 2290-2298.
15. de Micheli A, Medrano GA, Martínez Rios MA. [Right blocks in interauricular communication]. *Arch Inst Cardiol Mex.* 1978 Nov-Dec;48(6):1091-113.
16. Di Cori A, Gemignani C, Lazzari M, et al. New-onset left bundle branch block as an early electrocardiographic feature of takotsubo cardiomyopathy]. *G Ital Cardiol (Rome).* 2010;11(5):442-5.
17. Draeger HT, Assassi S, Sharif R, et al. Right bundle branch block: a predictor of mortality in early systemic sclerosis. *PLoS One.* 2013 Oct 31;8(10):e78808.
18. Dubner S, Schapachnik E, Riera AR, Valero E. Chagas disease: state-of-the-art of diagnosis and management. *Cardiol J.* 2008;15:493-504.
19. Elizari MV. [Chagasic myocardopathy: historical perspective]. *Medicina (B Aires).* 1999;59 Suppl 2:25-40.
20. Fedor JM, Walston A 2nd, Wagner GS, Starr J. The vectorcardiogram in right bundle branch block: correlation with cardiac failure and pulmonary disease. *Circulation.* 1976 Jun;53(6):926-30.
21. Gómez Barrado JJ, Turégano Albarrán S, et al. Clinical and electrocardiographic characteristics of masquerading bifascicular block. *Rev Esp Cardiol.* 1997 Feb;50:92-97. Massing GK, James TN. Conduction and block in the right bundle branch, real and imagined. *Circulation.* 1972 Jan;45(1):1-3.
22. Gelband H, Waldo AL, Kaiser GA, et al. Etiology of right bundle-branch block in patients undergoing total correction of tetralogy of Fallot. *Circulation.* 1971 Dec;44(6):1022-33.
23. Hébert JL, Duthoit G, Hidden-Lucet F, et al. Images in cardiovascular medicine. Fortuitous discovery of partial Uhl anomaly in a male adult. *Circulation.* 2010 Jun 8;121(22):e426-9.
24. Hiss RG, Lamb LE. Electrocardiographic findings in 122,043 individuals. *Circulation.* 1962 Jun;25:947-61.
25. Holtzman D, Aronow WS, Mellana WM, et al. Electrocardiographic abnormalities in patients with severe versus mild or moderate chronic obstructive pulmonary disease followed in an academic outpatient pulmonary clinic. *Ann Noninvasive Electrocardiol.* 2011 Jan;16(1):30-2.

26. Ishikawa K, Ohsaka H, Omori K, Yanagawa Y. A case of transient left bundle branch block after a cervical wound. *J Emerg, Trauma, Shock* 2014;7(3):247–8.
27. Izumi K. Optimal control of intermittent normal conduction in a tachycardia-dependent right bundle branch block. *Mater Med Pol.* 1996;28(4):141-8.
28. Janse MJ, van Dam RT. Effect of sudden changes in heart rate on refractory periods of ventricular myocardium and specialized conducting system in dog's heart. *Br Heart J.* 1971 Jan;33(1):148.
29. Juraschek SP, Kovell LC, Childers RE, Chow GV, Hirsch GA. Heart failure with transient left bundle branch block in the setting of left coronary fistula. *Cardiol Res Pract* 2011;2011:786287.
30. Kasmani R, Okoli K, Mohan G, Casey K, Ledrick D. Transient left bundle branch block: An unusual electrocardiogram in acute pulmonary embolism. *Am J Med Sci* 2009;337(5):381–2.
31. Kinoshita S, Katoh T, Tsujimura Y, et al. Apparent bradycardia-dependent right bundle branch block associated with atypical atrioventricular Wenckebach periodicity as a possible mechanism. *J Electrocardiol.* 2003 Oct;36(4):355-61.
32. Kleemann T, Juenger C, Gitt AK, et al. Incidence and clinical impact of right bundle branch block in patients with acute myocardial infarction: ST elevation myocardial infarction versus non-ST elevation myocardial infarction. *Am Heart J.* 2008 Aug;156(2):256-61.
33. Koos R, Mahnken AH, Aktug O, et al. Electrocardiographic and imaging predictors for permanent pacemaker requirement after transcatheter aortic valve implantation. *J Heart Valve Dis.* 2011 Jan;20(1):83-90.
34. Kounis NG, Zavras GM, Papadaki PJ, et al. Electrocardiographic changes in elderly patients during endoscopic retrograde cholangiopancreatography. *Can J Gastroenterol* 2003;17(9):539–44.
35. Kowey PR, Koslow M, Marinchak RA. Masquerading Bundle-branch block – Electrophysiological correlation *J electrophysiol.* 1989; 3:156-159.
36. Krongrad E, Hefler SE, Bowman FO Jr, et al. Further observations on the etiology of the right bundle branch block pattern following right ventriculotomy. *Circulation.* 1974 Dec;50(6):1105-13.
37. Kusumoto S, Kawano H, Makita N, et al. Right bundle branch block without overt heart disease predicts higher risk of pacemaker implantation: the study of atomic-bomb survivors. *Int J Cardiol.* 2014 Jun 1;174(1):77-82.
38. Kumpuris AG, Casale TB, Mokotoff DM, et al. Right bundle-branch block. Occurrence following nonpenetrating chest trauma without evidence of cardiac contusion. *JAMA.* 1979 Jul 13;242(2):172-3.



39. Lancaster MC, Schechter E, Massing GK. Acquired complete right bundle branch block without overt cardiac disease. Clinical and hemodynamic study of 37 patients. *Am J Cardiol.* 1972 Jul 11;30(1):32-6.
40. Lerecouvreur M, Perrier E, Leduc PA, et al. Right bundle branch block: electrocardiographic and prognostic features]. *Arch Mal Coeur Vaiss.* 2005 Dec;98(12):1232-8.
41. Lev M. Anatomic basis for atrioventricular block. *Am J Med.* 1964 Nov;37:742-8.
42. Lev M, Bharati S. Atrioventricular and intraventricular conduction disease. *Arch Intern Med.* 1975 Mar;135(3):405-10.
43. Lewinter C, Torp-Pedersen C, Cleland JG, Køber L. Right and left bundle branch block as predictors of long-term mortality following myocardial infarction. *Eur J Heart Fail.* 2011 Dec;13(12):1349-54.
44. Lubczynska-Kowalska W, Zagrobelny Z, Bielicki F, Budzynska A, Burdzinska-Golowin J. [Transient left bundle-branch block in Graves-Basedow disease in a child]. *Pol Tyg Lek* 1971;26(46):1781-3
45. Luna Filho B, Bocanegra JA, Pfeferman A, Andrade JL, Martinez Filho EE. [Fascicular block of the His bundle: critical approach for its identification]. *Arq Bras Cardiol.* 1989 Nov;53(5):261-5.
46. Marin-Neto JA, Simões MV, Maciel BC. Specific diseases: cardiomyopathies and pericardial diseases. Other cardiomyopathies. In: Yusuf S, Cairns J, Camm J, Fallen E, Gersh BJ, eds. - Evidence Based Cardiology. London, GB: BMJ Books, Brit Med Association, 1998: 744-61.
47. Martynov Iu S, Krishna Kumar O, Shuvakhina NA, et al. [Cerebro-cardial disorders in hemorrhagic stroke]. *Ter Arkh* 2004;76(2):44-9.
48. Massing GK, James TN. Conduction and block in the right bundle branch, real and imagined. *Circulation.* 1972 Jan;45(1):1-3.
49. Mavrogeni S, Sfikakis PP, Karabela G, et al. Cardiovascular magnetic resonance imaging in asymptomatic patients with connective tissue disease and recent onset left bundle branch block. *Int J Cardiol.* 2014 Jan 15;171(1):82-7.
50. Melgarejo-Moreno A, Galcerá-Tomás J, Garcíá-Alberola A, et al. Incidence, clinical characteristics, and prognostic significance of right bundle-branch block in acute myocardial infarction: a study in the thrombolytic era. *Circulation.* 1997 Aug 19;96(4):1139-44.
51. gs in pulmonary embolism in relation to vascular obstruction. *Cardiology.* 1989;76(4):274-84.
52. Miquel C, Sodi-Pallares D, Cisneros F, et al. Right bundle branch block and right ventricular hypertrophy; electrocardiographic and vectorcardiographic diagnosis. *Am J Cardiol.* 1958 Jan;1(1):57-67
53. Mueller C, Laule-Kilian K, Klima T, et al. Right bundle branch block and long-term mortality in patients with acute congestive heart failure. *J Intern Med.* 2006 Nov;260(5):421-8. Rodriguez MI, Sodi-Pallares D. The mechanism of complete and incomplete bundle branch block. *Am Heart J.* 1952 Nov;44(5):715-46.

54. Nielsen TT, Lund O, Rønne K, et al. Changing electrocardiographic findings. Okajima S, Okumura M, Sotabata I. Comparison of Frank-vectorcardiograms of normal conduction and right bundle branch block in patients with intermittent or transient right bundle branch block. *Jpn Heart J*. 1980 Mar;21(2):257-71.
55. Ortega-Carnicer J, Malillos M, Muñoz L, Rodriguez-Garcia J. Left anterior hemiblock masking the diagnosis of right bundle branch block. *J Electrocardiol*. 1986 Jan; 19: 97-98. Penaloza D, Gamboa R, Sime F. Experimental right bundle branch block in the normal human heart. Electrocardiographic, vectorcardiographic and hemodynamic observations. *Am J Cardiol*. 1961 Dec;8:767-79.
56. Parharidis G, Nouskas J, Efthimiadis G, et al. Complete left bundle branch block with left QRS axis deviation: defining its clinical importance. *Acta Cardiol*. 1997;52(3):295-303.
57. Pastore CA, Moffa PJ, Spiritus MO, et al. [Fascicular blocks of the right branch. Standardization of vectorelectrocardiographic findings]. *Arq Bras Cardiol*. 1983 Sep;41(3):161-6.
58. Pezzilli R, Barakat B, Billi P, Bertaccini B. Electrocardiographic abnormalities in acute pancreatitis. *Eur J Emerg Med* 1999;6(1):27-9.
59. Pizzo VR, Beer I, de Cleve R, Zilberstein B. Intermittent left bundle branch block (LBBB) as a clinical manifestation of myocardial contusion after blunt chest trauma. *Emerg Med J* 2005;22(4):300-1.
60. Prabha A, Mohanan, Pereira P, Raghuvver CV. Myocarditis in enteric fever. *Indian J Med Sci*. 1995;49(2):28-31.
61. Pratila MG, Pratilas V, Dimich I. Transient left bundle-branch block during anesthesia. *Anesthesiology* 1979;51(5):461-3.
62. Préda I. Results of randomized studies on cardiac resynchronization therapy and the reevaluation of cardiac ventricular activation in left bundle branch block. *Orv Hetil*. 2013;154(18):688-93.
63. Richman JL, Wolff L. Left bundle branch block masquerading as right bundle branch block. *Am Heart J*. 1954 Mar; 47: 383-393.

44. Riera AR, de Cano SJ, Cano MN, et al. Vector electrocardiographic alterations after percutaneous septal ablation in obstructive hypertrophic cardiomyopathy. Possible anatomic causes. *Arq Bras Cardiol.* 2002 Nov;79(5):466-75.
45. Rodriguez MI, Sodi-Pallares D. The mechanism of complete and incomplete bundle branch block. *Am Heart J.* 1952 Nov;44(5):715-46.
46. Rosenbaum MB, Elizari MV, Lazzari JO. *Los hemibloqueos.* Buenos Aires; Paidós 1968.
47. Rosenbaum MB, Yesuron J, Lazzari JO, Elizari MV. Left anterior hemiblock obscuring the diagnosis of right bundle branch block. *Circulation.* 1973 Aug; 48: 298-303.
48. Rotman M, Triebwasser JH. A clinical and follow-up study of right and left bundle branch block. *Circulation.* 1975 Mar;51(3):477-84.
49. Rusconi L, Nava A, Sermasi S, Antonioli GE. The left posterior fascicular block: is the diagnosis possible only by ECG? *G Ital Cardiol.* 1980;10:1129-1134.
50. Sayin MR, Karabag T, Dogan SM, Akpinar I, Aydin M. Transient ST segment elevation and left bundle branch block caused by mad-honey poisoning. *Wien Klin Wochenschr* 2012;124(7-8):278–81.
51. Schamroth L, Dekock J. The concept of 'masquerading' bundle-branch block. *S Afr Med J.* 1975 Mar 15; 49: 399-400.
55. Schamroth L, Myburgh DP, Schamroth CL. The early signs of right bundle branch block. *Chest.* 1985; 87:180-5. Ocal A, Yildirim N, Ozbakir C, et al. Right bundle branch block: a new parameter revealing the progression rate of mitral stenosis. *Cardiology.* 2006;105(4):219-22.
56. Schmunis GA, Zicker F, Moncayo A. Interruption of Chagas' disease transmission through vector elimination. *Lancet* 1996; 348: 117.
57. Schneider JF, Thomas HE, Kreger BE, et al. Newly acquired right bundle-branch block: The Framingham Study. *Ann Intern Med.* 1980 Jan;92(1):37-44.
58. Sclarovsky S, Lewin RF, Strasberg B, Agmon J. Left anterior hemiblock obscuring the diagnosis of right bundle branch block in acute myocardial infarction. *Circulation.* 1979 Jul; 60: 26-32.

55. Senoo K, Otsuka T, Suzuki S, Sagara K, Yamashita T. Impact of pulmonary vein isolation on left bundle branch block following tachycardia-induced cardiomyopathy in a patient with persistent atrial fibrillation. *Intern Med* 2014;53(7):721–4.
56. Serrano Junior CV, De Cleve R, Mancini MC, Tranchesi Junior B, Scaff M, Ramires JA. Intermittent left branch block as a complication in Guillain-Barre syndrome. Report of a case. *Arquivos Brasileiros de Cardiologia* 1987;49(5):299–301.
57. Shimamoto T, Nakata Y, Sumiyoshi M, et al. Transient left bundle branch block induced by left-sided cardiac catheterization in patients without pre-existing conduction abnormalities. *Jpn Circ J* 1998;62(2):146–9.
58. Sodi D, Bisteni A, Medrano G. *Electrocardiografía y vectorcardiografía deductivas*. Vol. 1 Mexico, DF: La Prensa Médica Mexicana, 1964.
59. Strauss DG, Selvester RH, Wagner GS. Defining left bundle branch block in the era of cardiac resynchronization therapy. *Am J Cardiol*. 2011;107(6):927-34.
60. Surawicz B, Childers R, Deal BJ, et al. American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society. 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(11):976-81.
61. Tabatabaei N, Katanyuwong P, Breen JF, et al. Images in cardiovascular medicine. Uncommon variant of Ebstein anomaly with tricuspid stenosis. *Circulation*. 2009 Jul 7;120(1):e1-2.
62. Tian Y, Zhang P, Li X, Gao Y, Zhu T, Wang L, Li D, Wang J, Yuan C, Guo J. True complete left bundle branch block morphology strongly predicts good response to cardiac resynchronization therapy. *Europace*. 2013;15(10):1499-506.

55. Tobias NM, Pastore CA, Moffa PJ, et al. [Divisional blocks of the right branch in Chagas' cardiomyopathy]. *Arq Bras Cardiol.* 1986 Dec;47(6):387-91.
56. Tomita M, Kitazawa H, Sato M, et al. A complete right bundle-branch block masking Brugada syndrome. *J Electrocardiol.* 2012 Nov-Dec;45(6):780-2.
65. Unger PN, Lesser ME, Kugel VH, Lev M, The Concept of "Masquerading" Bundle-Branch Block An Electrocardiographic-Pathologic Correlation *Circulation.* 1958;17:397-409.
66. Varriale P, Kennedy RJ. Right bundle branch block and left posterior fascicular block. Vectorcardiographic and clinical features. *Am J Cardiol.* 1972 Apr;29(4):459-65.
67. Vasic N, Stevic R, Pesut D, Jovanovic D. Acute left bundle branch block as a complication of brachytherapy for lung cancer. *Respir Med* 2011;105(Suppl 1):S78–S80.
68. Wanderley DMV, Corrêa FMA. Epidemiology of Chagas' heart disease. *S, o Paulo Med J* 1995; 113: 742-9.
69. Willems JL, Robles de Medina EO, Bernard R, et al. Criteria for intraventricular conduction disturbances and pre-excitation. World Health Organizational/International Society and Federation for Cardiology Task Force Ad Hoc. *J Am Coll Cardiol.* 1985 Jun;5(6):1261-75.
70. Zhong-Qun Z, Bo Y, Nikus KC, et al. Correlation between ST-segment elevation and negative T waves in the precordial leads in acute pulmonary embolism: insights into serial electrocardiogram changes. *Ann Noninvasive Electrocardiol.* 2014 Jul;19(4):398-405.