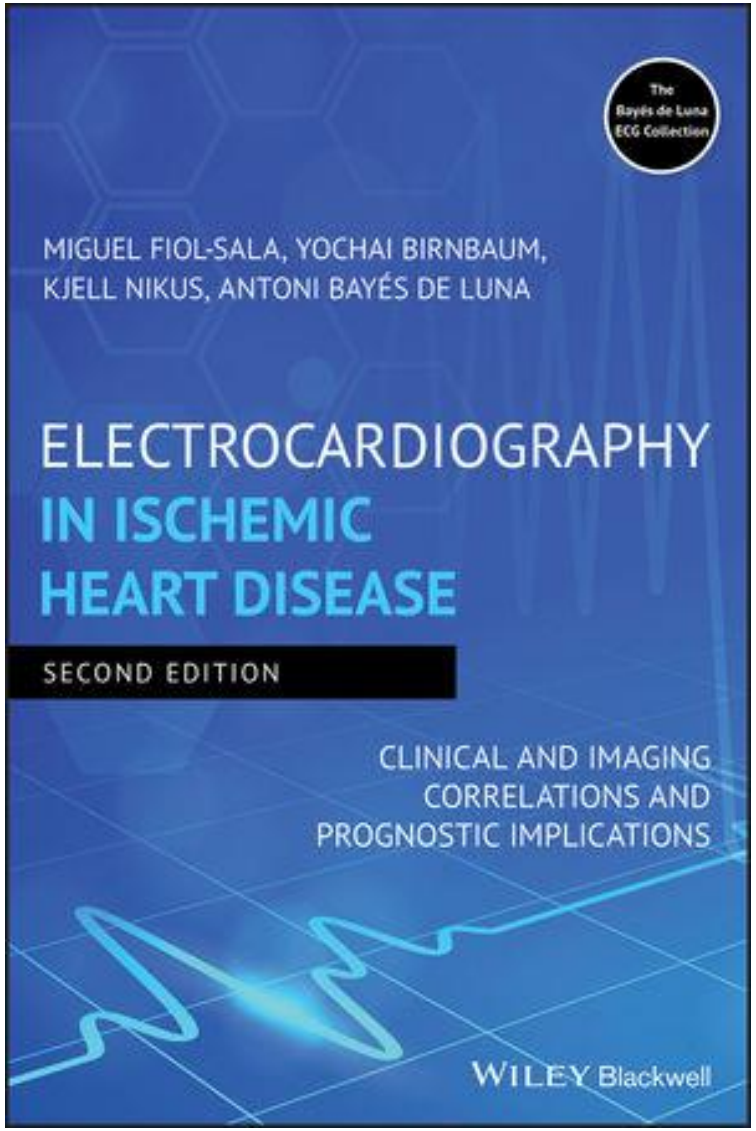
77-year old male Hypertension, Chronic Obstructive Pulmonary Disease, Pulmonary fibrosis Emphysema, Asbestosis

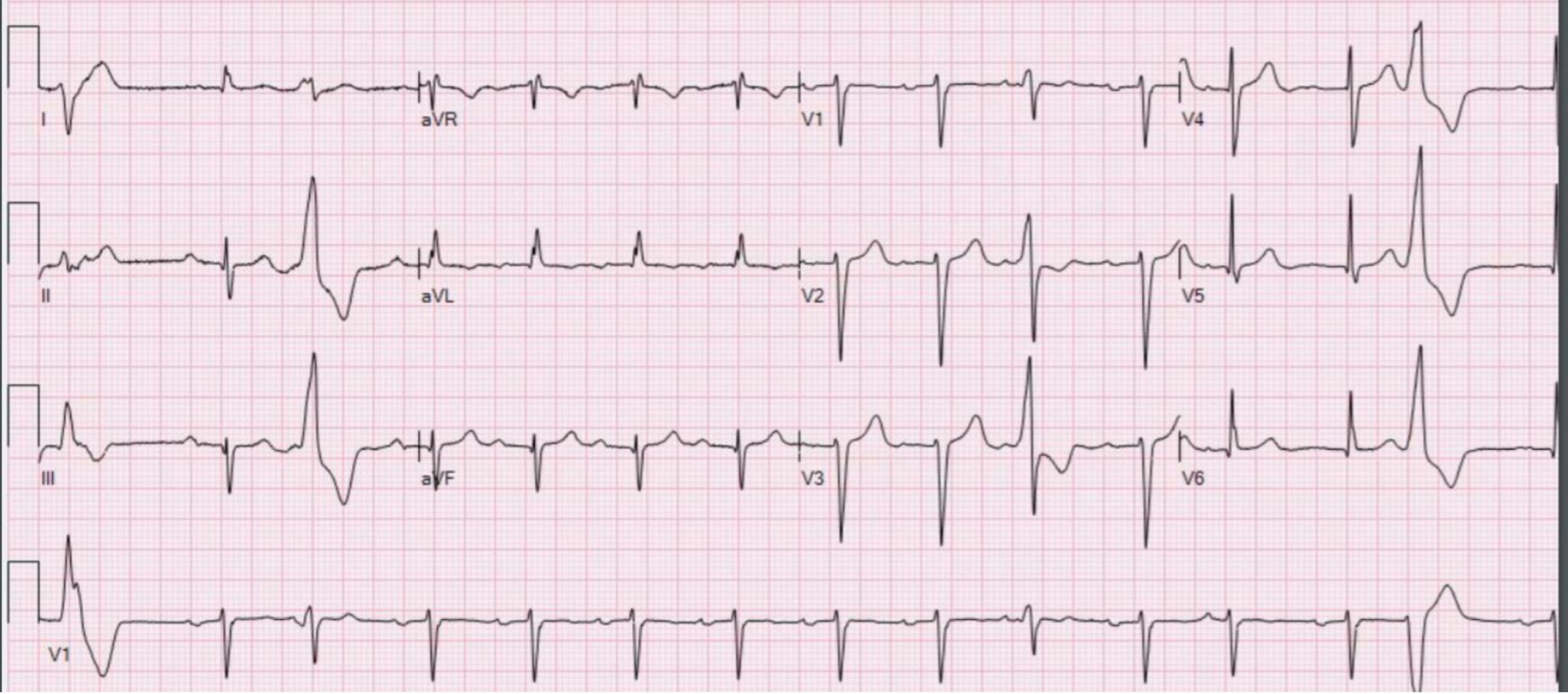
Dear Andrés Do you think that all ECG changes (inferior small Q waves, left axis deviation, and arrhythmias) in this patient is caused by the pulmonary diseases, which also cause a change of the position of the heart?

Regards

Kjell Christer **Nikus MD PhD** Finland Professor of internal medicine/ Professor in Cardiology at Heart Center, Tampere University HospitalTampere, Finland

311 Publications. 40,309. Reads 6,273 Citations




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

Dear Andrés, To me, the ECG in question shows normal sinus rhythm with PVCs, 1st degree AV block, LAFB with q-waves of an old inferior MI. The evidence for LAFB is an axis of ~ -45 degrees with S in III > S in II. The small q in lead I and aVL is part of the LAFB diagnosis. In 1983 in the **Am J Cardiology (1973, 51(5):718-22**), Warner et al published ECG criteria for the diagnosis of combined inferior MI and LAFB. You would like this paper because the authors used a vector approach. The abstract reads as follows:

New electrocardiographic (ECG) criteria for diagnosing the combination of inferior myocardial infarction and left anterior hemiblock are proposed. The proposed criteria are based upon the relations between portions of the vectorcardiographic QRS loop in the frontal plane and the corresponding portions of the QRS complexes recorded by the limb leads. The application of the proposed criteria requires that the tracings be obtained with 3-channel ECG machines. The proposed criteria for the diagnosis of inferior myocardial infarction and left anterior hemiblock are as follows: (1) leads aVR and aVL both end in R waves, with the peak of the terminal R wave in lead aVR occurring later than the peak of the terminal R wave in lead aVL, and (2) a Q wave of any magnitude is present in lead II. The performance of the proposed criteria was superior to that of 10 combinations of traditional ECG criteria for inferior myocardial infarction and left anterior hemiblock.

I don't have the actual paper, but it came from SUNY Upstate Medical School in Syracuse (NY) where I went to medical school. Although I graduated in 1966 I knew Harold Smulyan, MD, who was the senior author and one of my teachers. It was in medical school when I fell in love with electrocardiography, having worked with Dr. JA Abildskov for 18 months of animal research. (Yanowitz FG, Preston JB, Abildskov JA: Functional distribution of right and left left stellate innervation to the ventricles: Production of neurogenic ECG changes by unilateral alteration of sympathetic tone. Circ Res 1966; 18:416-422.) This patient you sent me brings back a lot of memories. Thanks for sharing.

Regards,

Frank G. Yanowitz, M.D.

Department of Medicine / Cardiology / Geriatrics – University of Utah School of Medicine

Andrés Ricardo Pérez-Riera[1], Raimundo Barbosa-Barros [2], Rodrigo Daminello-Raimundo[1], Luiz Carlos de Abreu[1]

Affiliations

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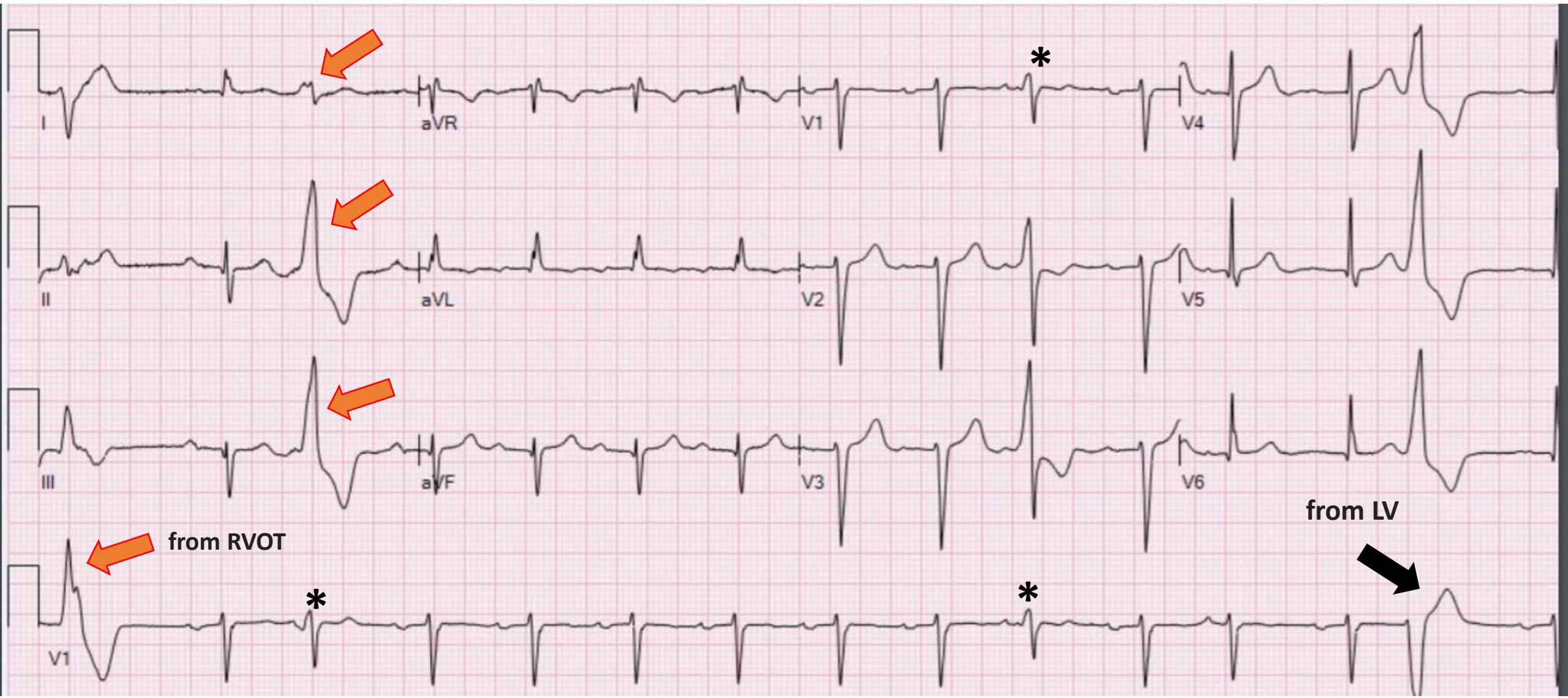
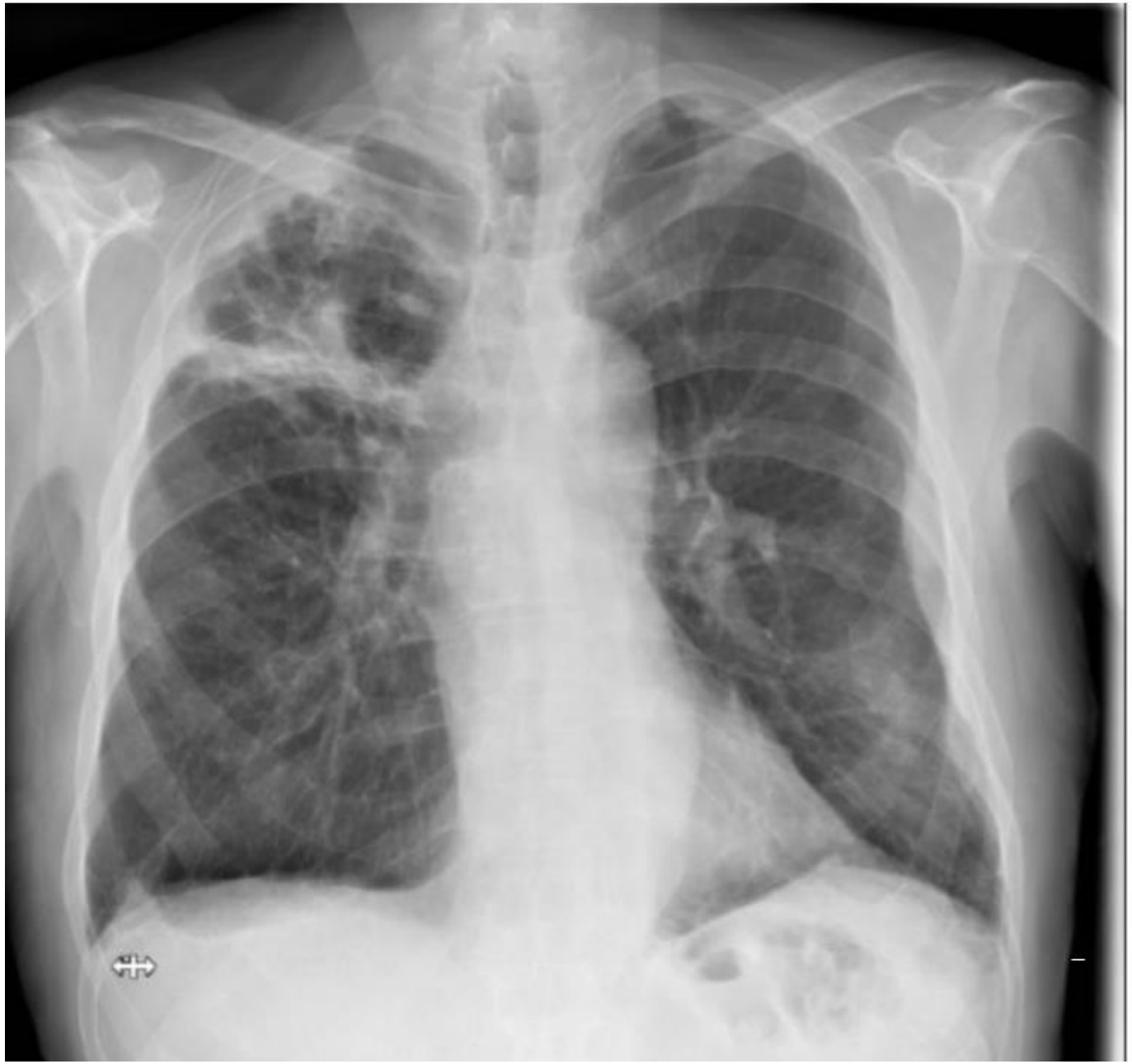
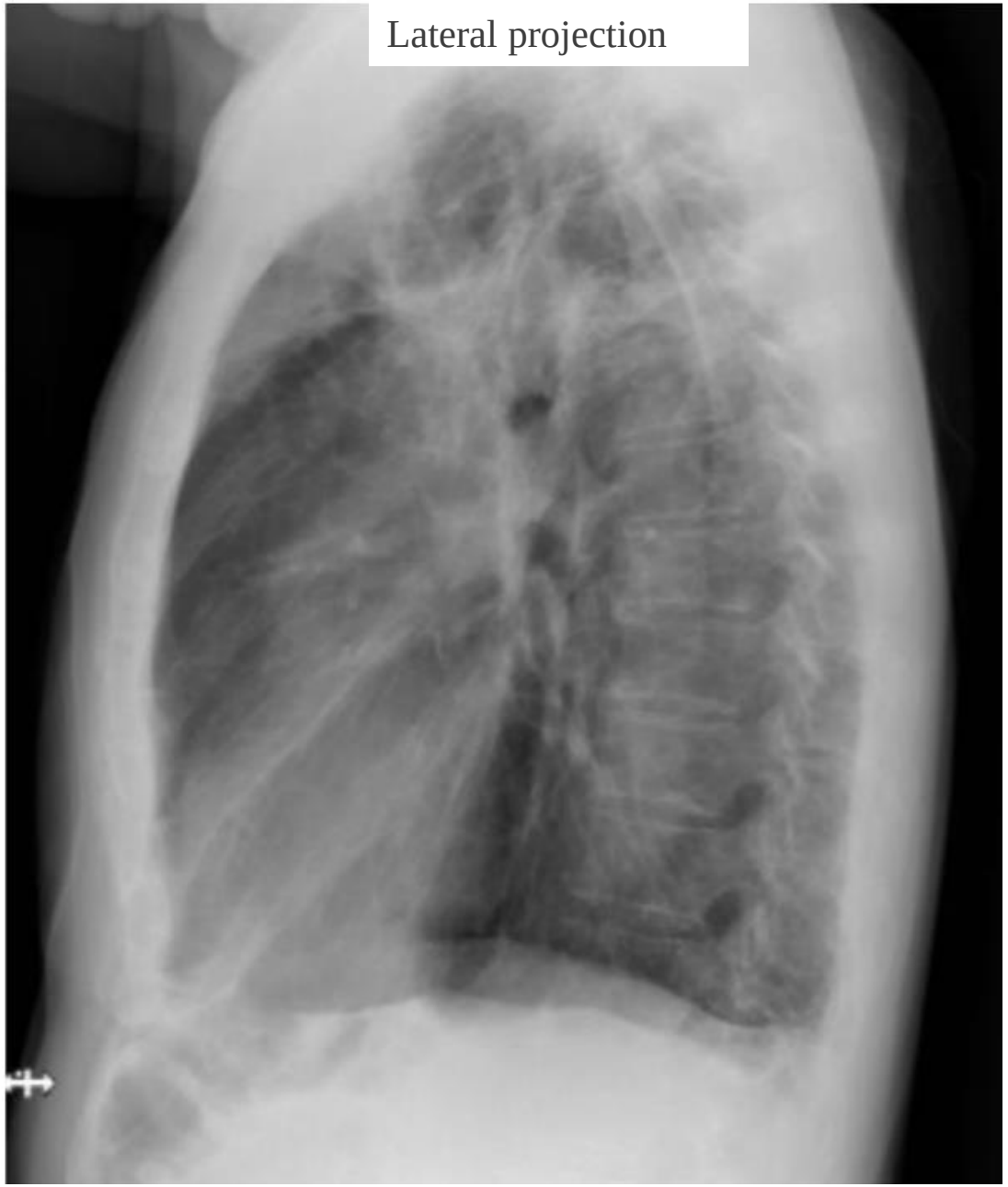
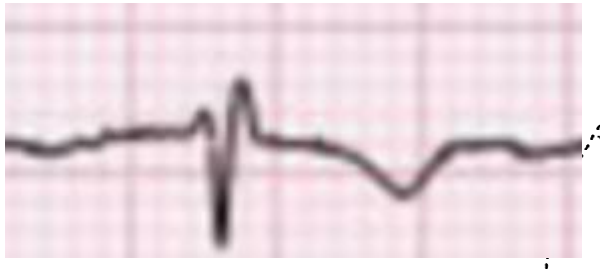
2. Coronary Center of the Hospital de Messejana Dr. Carlos Alberto Studart Gomes, Fortaleza, Ceara, Brazil.

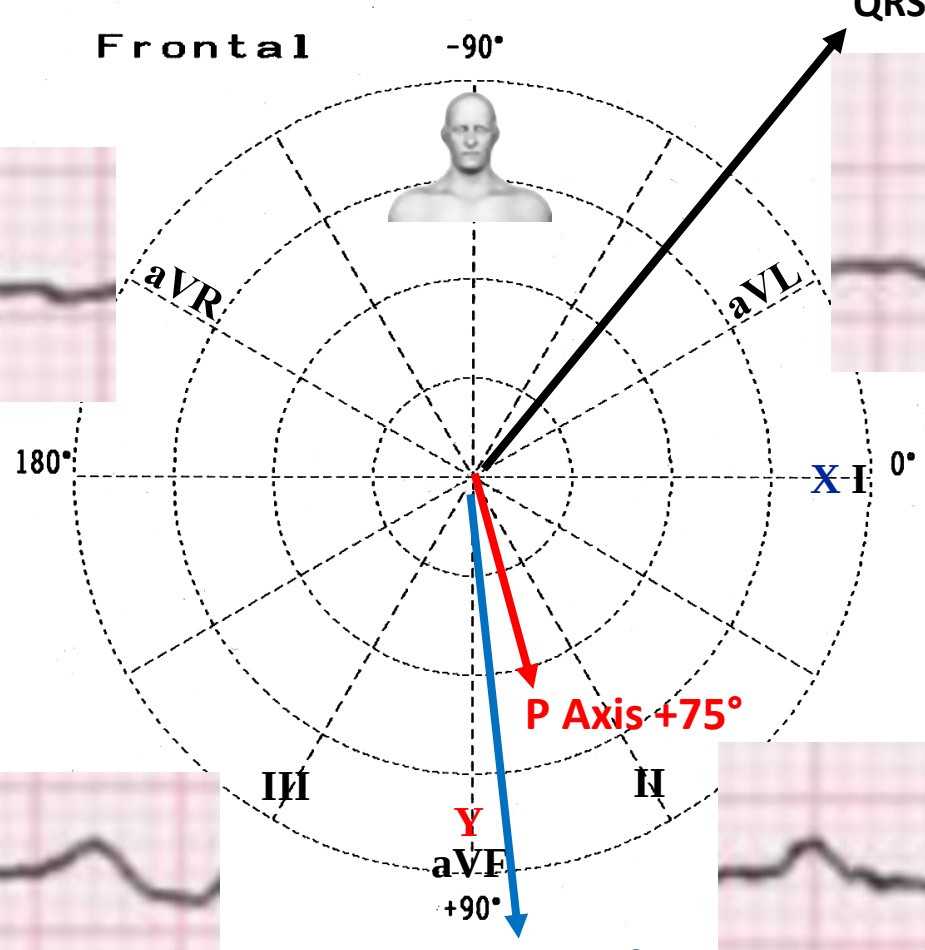
Final comments

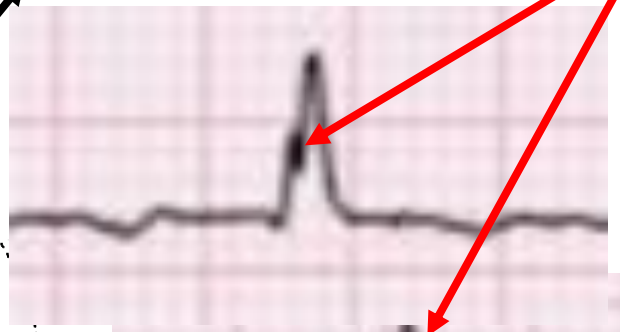

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
from LV
from RVOT
V1

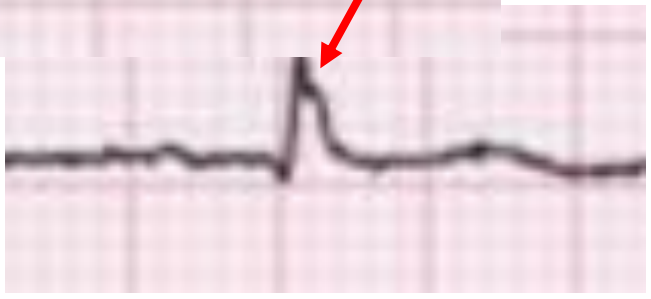



Lateral projection

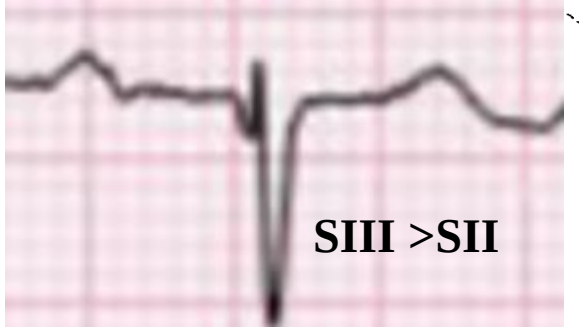




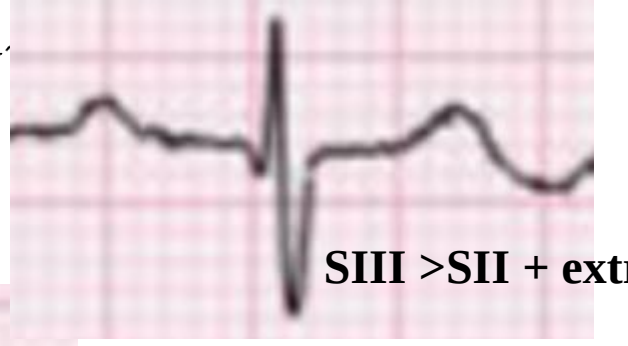




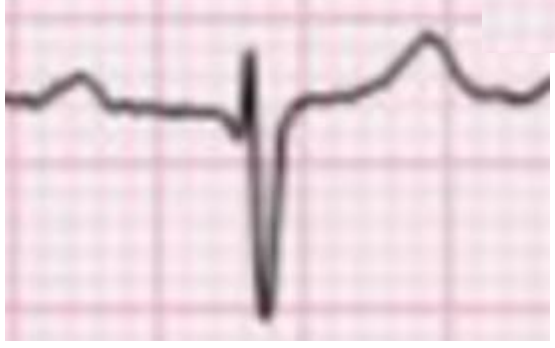
Initial and terminal notching of the QRS complex in the ECG are characteristic in asbestosis (1)



SIII >SII

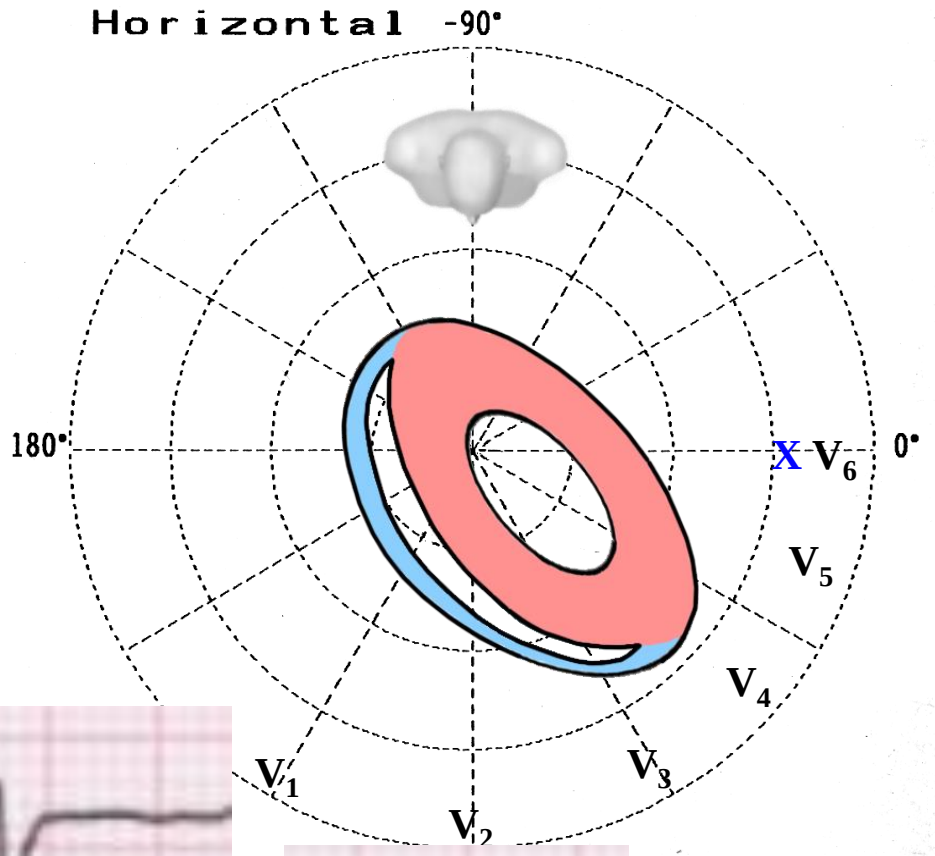


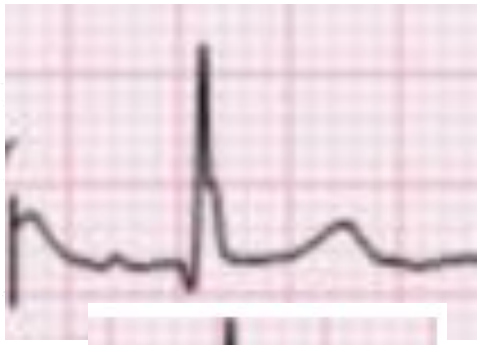
SIII >SII + extreme LAD = LAFB

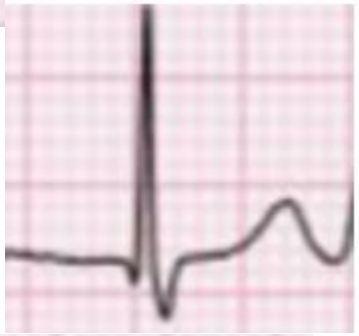


Probable sequalae of inferolateral fibrosis ?

1. H Raunio, V M Anttonen. Initial and terminal notching of the QRS complex in the conventional electrocardiogram. Am Heart J. 1972 May;83(5):717-9. doi: 10.1016/0002-8703(72)90415-2.

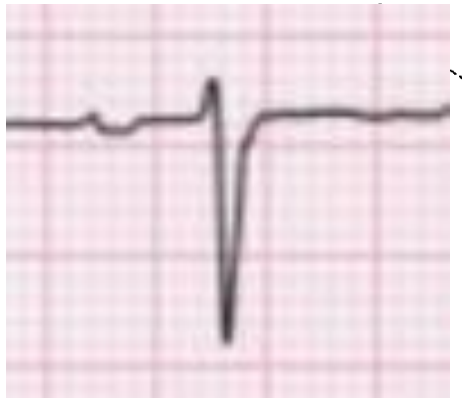


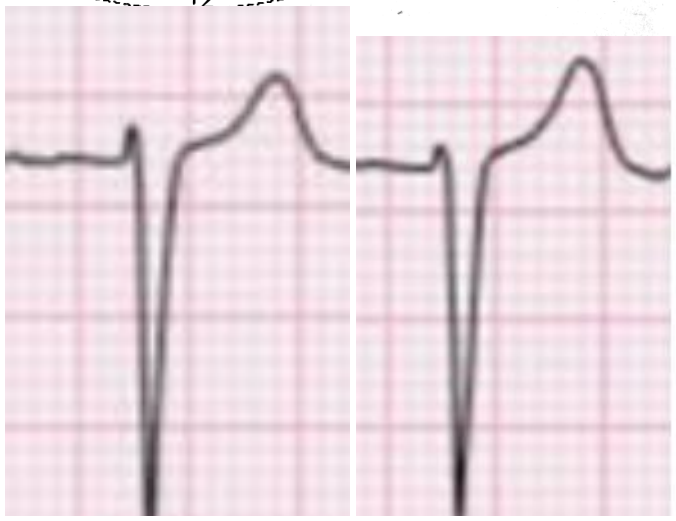


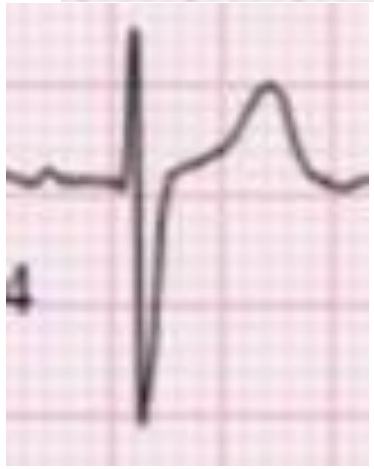


Delayed precordial transition zone located in V5

Clockwise rotation



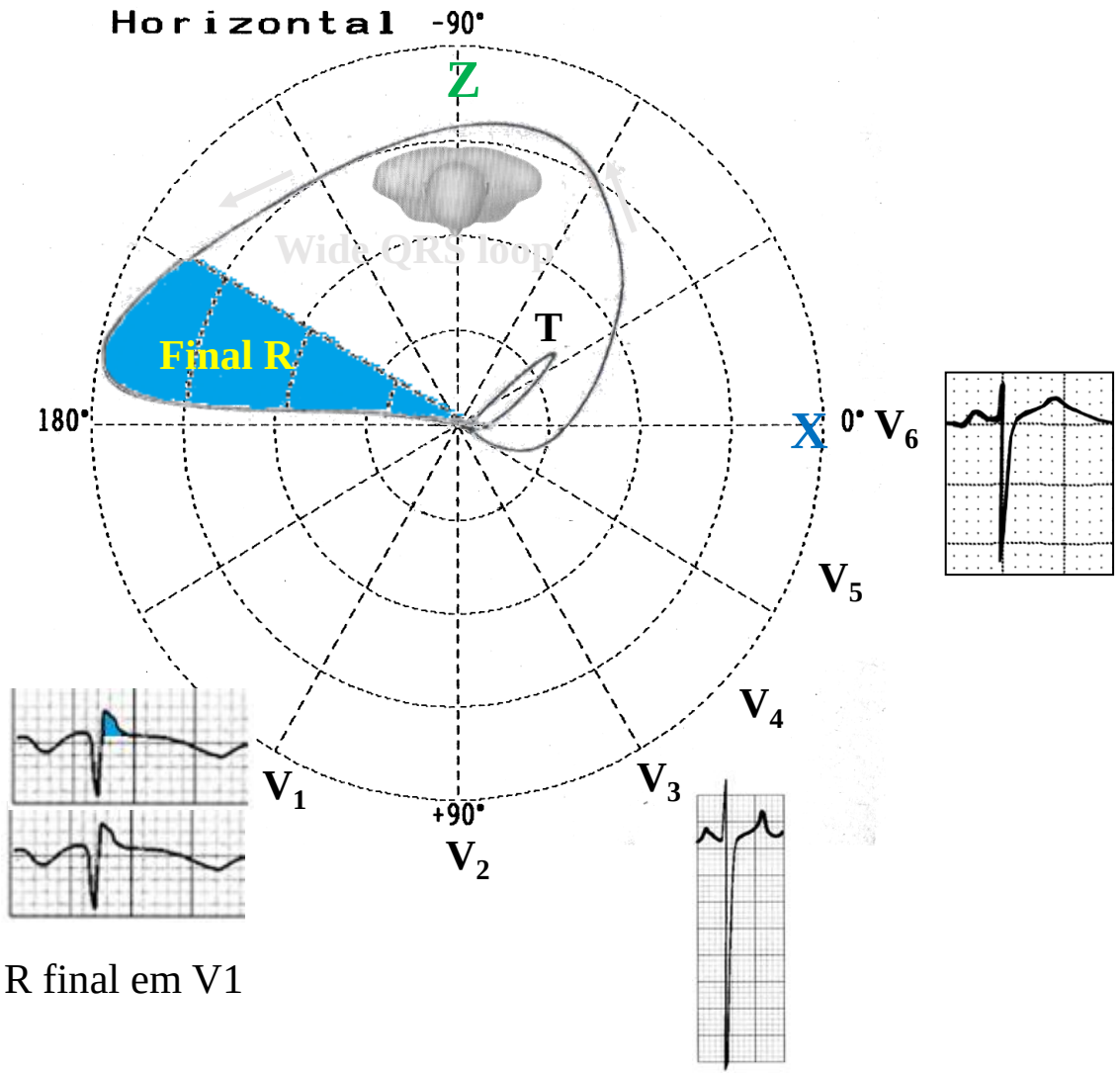




Deep S wave in V2-3 type C RVH characteristic of Enfisema See next slide

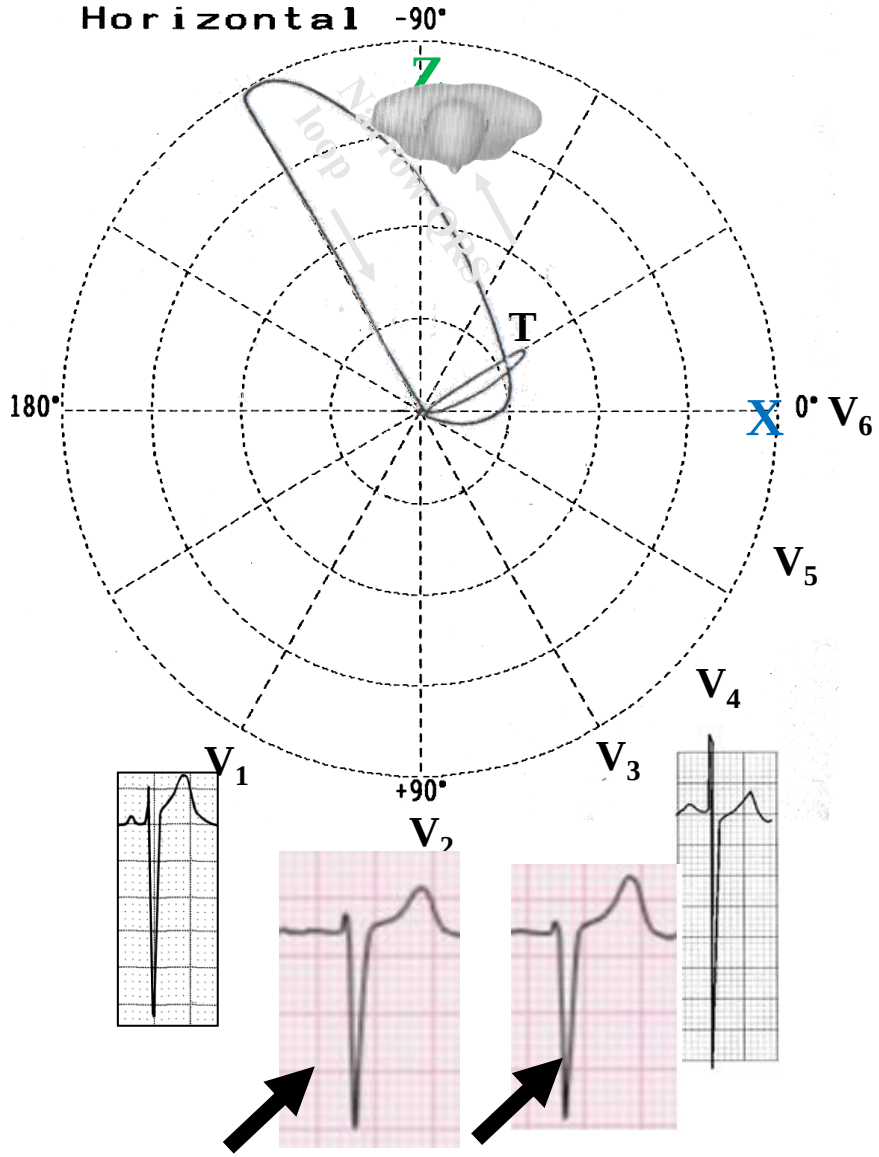
Type C or special RVH classification by VCG criteria in the HP

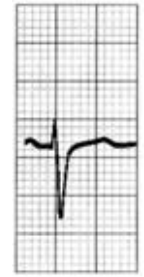
Type C-I



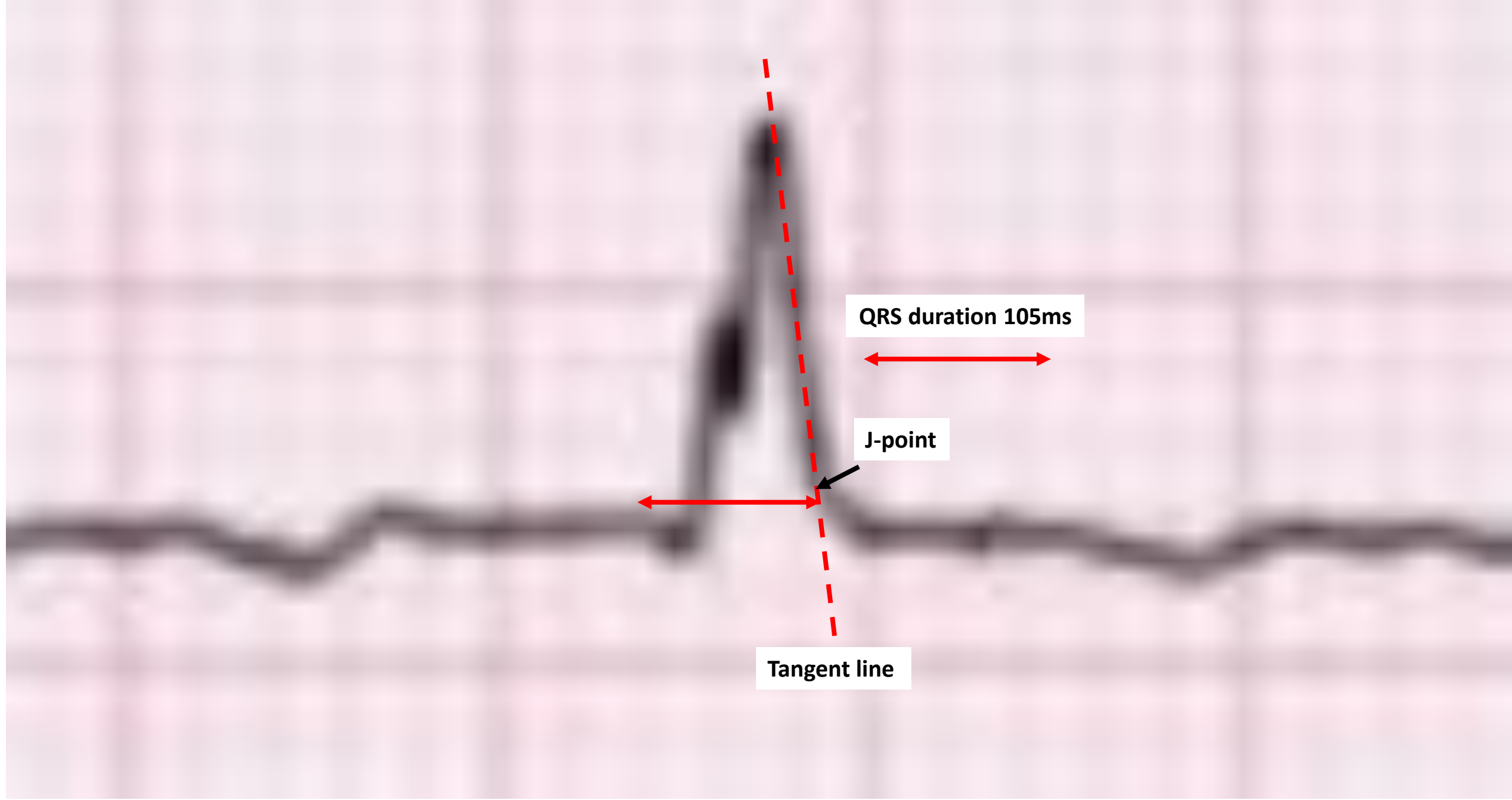
R final em V1

Type C-II





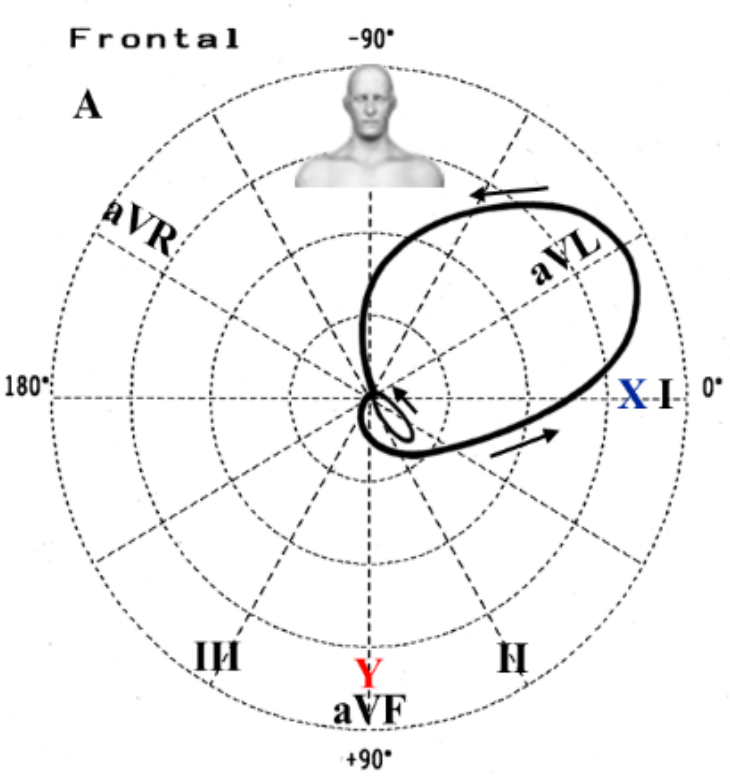
The presente case Deep S wave in V2-3 type C RVH characteristic of Enfisema


QRS duration 105ms
J-point
Tangent line

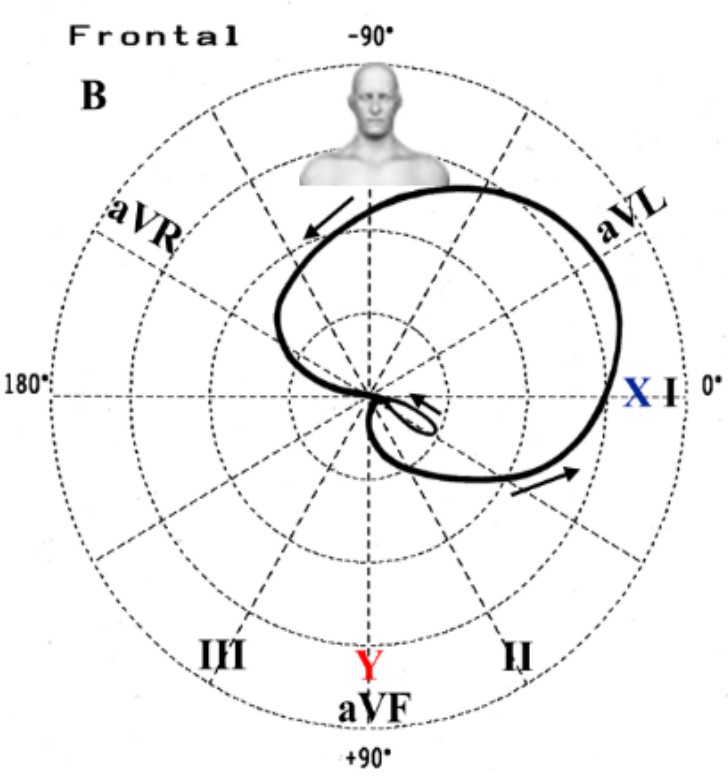
ECG diagnosis

1) **Sinus rhytm**
2) **P- Axis +75°, P-duration 120ms Left Atrial Enlargement. Asociation prolonged P-wave + P axis to the right: biatrial enlargement**
3) **PR interval = 238ms: Firth Degree AV block**
4) **QRS axis-45°, QRS duration 80ms, SIII >SII, q in I and aVL: Left Anterior Fascicular Block**
5) **Broad QRS-T angle = 135°: A wide QRS-T angle greater than 90° is associated with an increased risk of SCA independent of the left ventricular ejection fraction. Kelvin C.M, et al. Wide QRS-T angle on the 12-lead ECG as a Predictor of Sudden Death beyond the LV Ejection Fraction. J Cardiovasc Electrophysiol. 2016 Jul; 27(7): 833–839.. doi: 10.1111/jce.12989**
6) **Delayed precordial transition zone located in V5**. It is defined as the precordial lead in which the R wave equals or exceeds the S wave in amplitude. delayed QRS transition, or clockwise rotation of the heart, carries prognostic implications and predicts sudden cardiac death (SCD) Delayed QRS transition in the precordial leads of an ECG seems to be a novel ECG risk marker for SCD. In particular, markedly delayed transition was associated with significantly increased risk of SCD, independent of confounding factors.(**Aapo L Aro, et al. Delayed QRS transition in the precordial leads of an electrocardiogram as a predictor of sudden cardiac death in the general population. Heart Rhythm. 2014 Dec;11(12):2254-60. doi: 10.1016/j.hrthm.2014.08.014**.)
7) **Premature Ventricular Contractions with two focus: from RVOT (Left Bundle Branch morphology) with inferior axis: and RBBB pattern (LV);**
8) **Supraventricular Premature Ventricuar Contractions** *
9) **Probable sequalae of inferolateral fibrosis ?** Necessary Cardiovascular magnetic resonance (CMR) with late gadolinium enhancement (LGE)

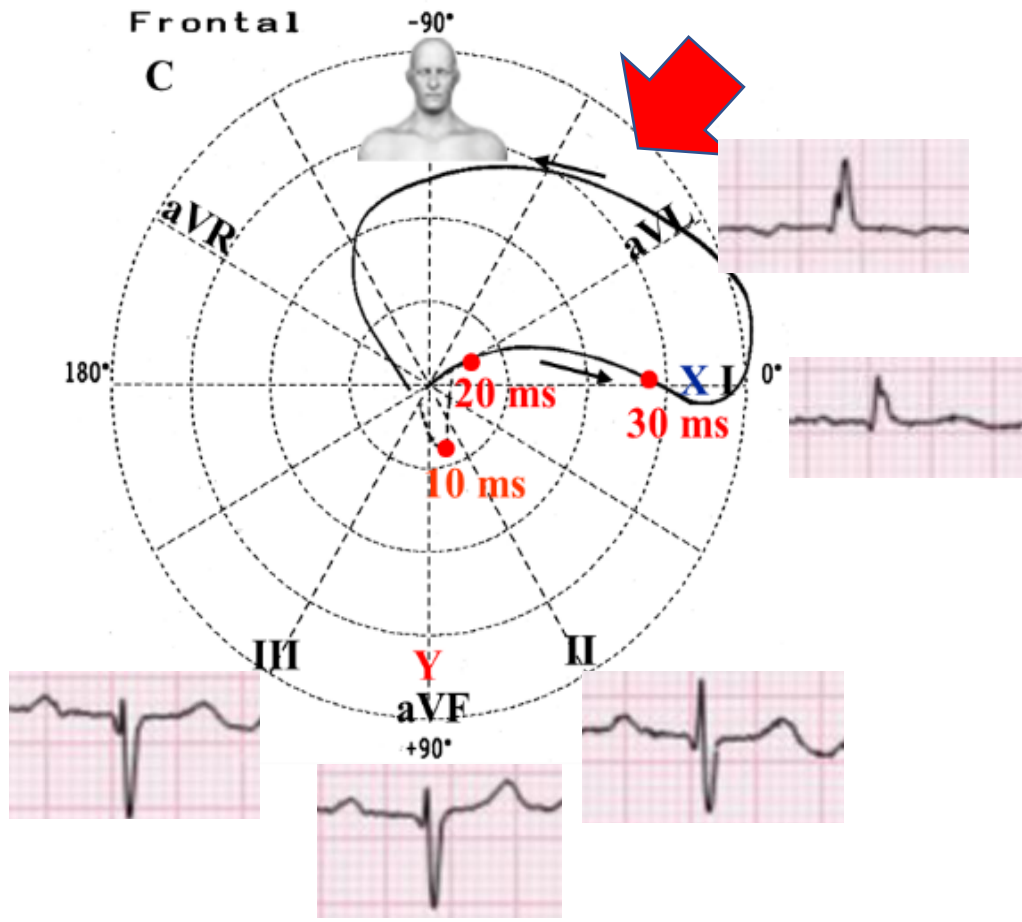
QRS loops in isolated Left Anterior Fascicular Block (A); LAFB associated with RBBB (B); and LAFB + inferior myocardial infarction in the frontal plane (C)



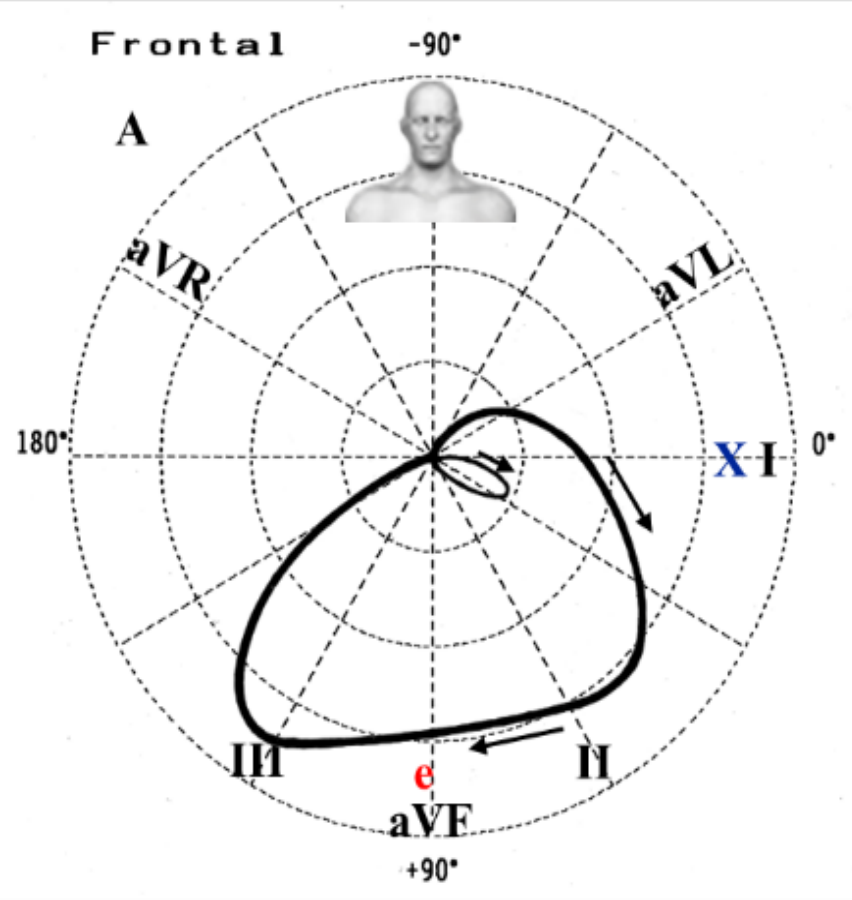
Isolated Left Anterior Fascicular Block

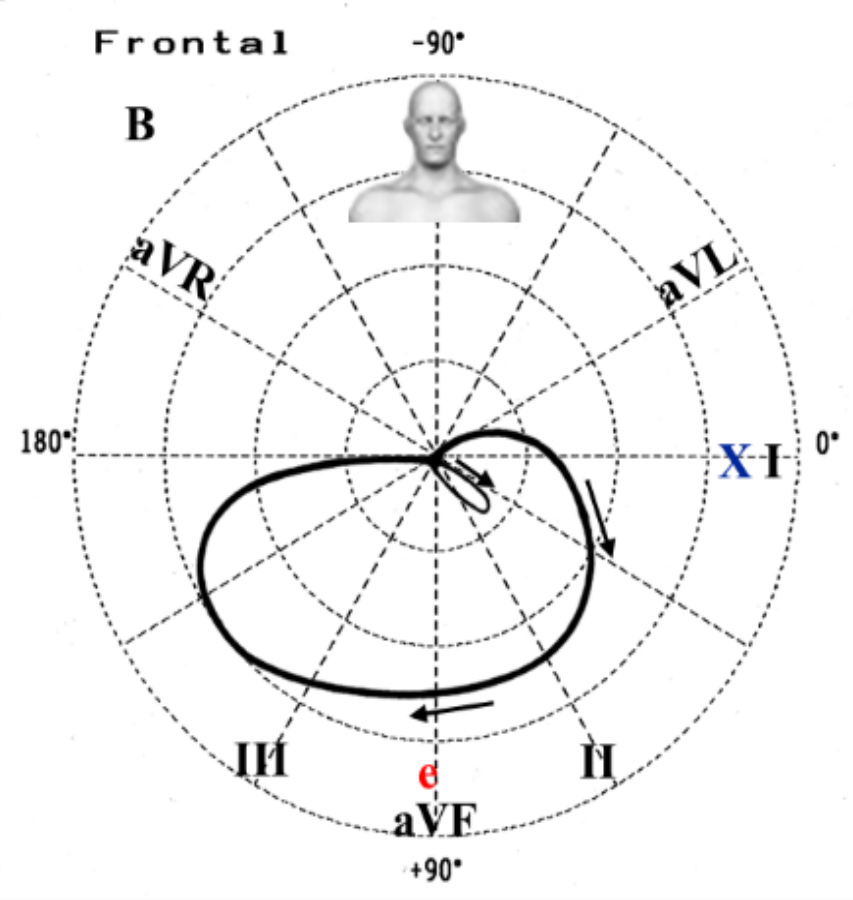


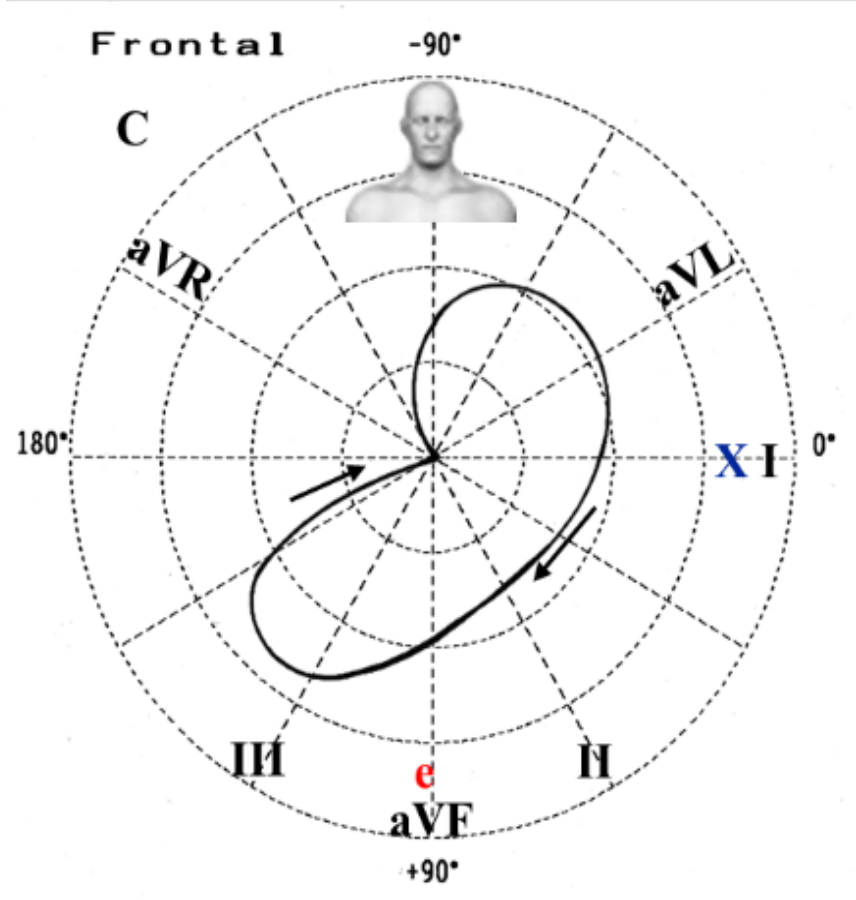
LAFB+ RBBB



Hypothetical QRS loop in the present case
LAFB+ Inferiro MI





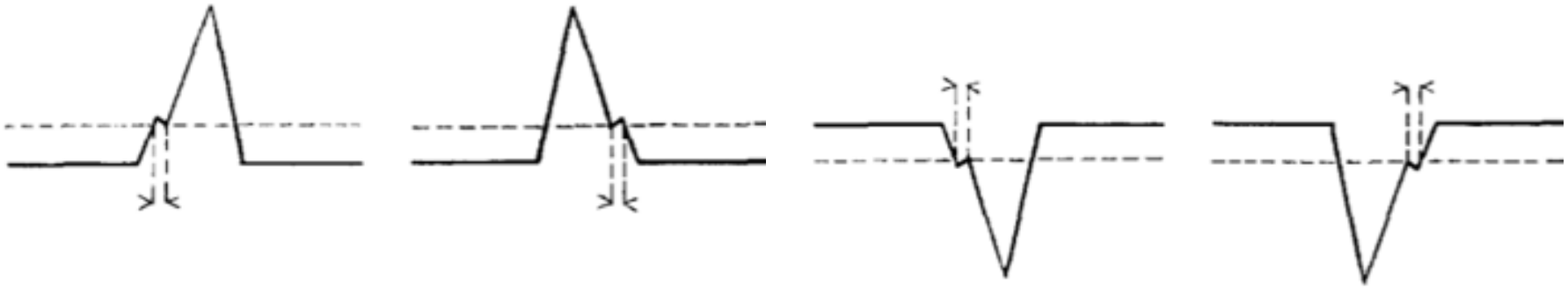


A: **QRS-loop in the isolated LPFB**

B: QRS-loop in the LPFB+RBBB

C: QRS-loop in the LPFB+ Inferior MI

Low-frequency notching in the QRS complex has been ignored by most electrocardiographs. One reason for this loss of interest may be that notching is often entirely obscured at the usual paper speed of 25 mm. per second. Raunio et al (1) have studied the QRS complex with attention to the timing of the notch during the QRS complex as well as to its magnitude In these studies the notches during the initial and terminal 40ms. of the QRS complex have been studied separately. Such a division is useful from the clinical point of view, because most disorders that alter QRS forces seemed to affect the first and the last QRS forces differently. A notch appearing during the first or last 40ms of the QRS complex has been regarded as significant if it was present in two or more of the 12 leads and if its duration was ≥ 20ms in at least one lead, with the exception that an initial QRS notch confined to the precordial leads has been regarded as significant even if it occurred in only one of the precordial leads provided it has a duration of ≥20ms ("significant initial QRS notch" and "significant terminal QRS notch"; see Fig.). In all of these studies the paper speed of 50 mm.(used in Finland) per second was used in the ECG recording. The initial and terminal QRS notches were frequently found in the ECG's of healthy children and in those of young adults below 30 years of age. The significant initial QRS notch was seen in 22% of 100 children, in 6 % of 110 young adults, and in 12 % of 43 champion-class sportsmen. In the same series the significant terminal QRS notch was seen in 8, 3, and 9 %, respectively. Preponderance of the right ventricle, frequently found in children, young adults, and sportsmen, may be considered to be the cause of the notching in these cases.



Various manifestations of a significant initial and terminal notch in the QRS complex. The interval between the arrows is ≥20ms..

1. **H Raunio, V M Anttonen. Initial and terminal notching of the QRS complex in the conventional electrocardiogram. Am Heart J. 1972 May;83(5):717-9. doi: 10.1016/0002-8703(72)90415-2.**

Chronic Obstructive Pulmonary Disease/Emphysema	Hypertension
Right P wave Axis +75°	Prolonged P-duration 120ms
Right atrial enlargement	Left atrial enlargement
Rightward shift of the P wave axis with prominent P waves in the inferior leads and flattened or inverted P waves in leads I and aVL	Absence of the expected persistent S wave in V6.
Clockwise rotation of the heart with delayed R/S transition point in the precordial leads	
	Absence of expected of low QRS voltages in the left-leads (I, aVL, V5-6).
	Q waves in the inferolateral leadsProbable sequalae of inferolateral fibrosis ? Hipertension+ CAD?

Chronic Obstructive Pulmonary Disease + Hipertension

Biatrial enlargement

Association Clockwise rotation of the heart with delayed R/S transition +Absence of the expected persistent S wave in V6.

PVC with two focus form RVOT and from LV

VCG characteristics of type C or special or III RVH

Etiology:

Acquired causes:

The great cause is chronic obstructive pulmonary disease (COPD). We only find chronic Cor Pulmonale in those patients with significant disease of airways with or without emphysema. (**Walsh 1960**)

Congenital causes:

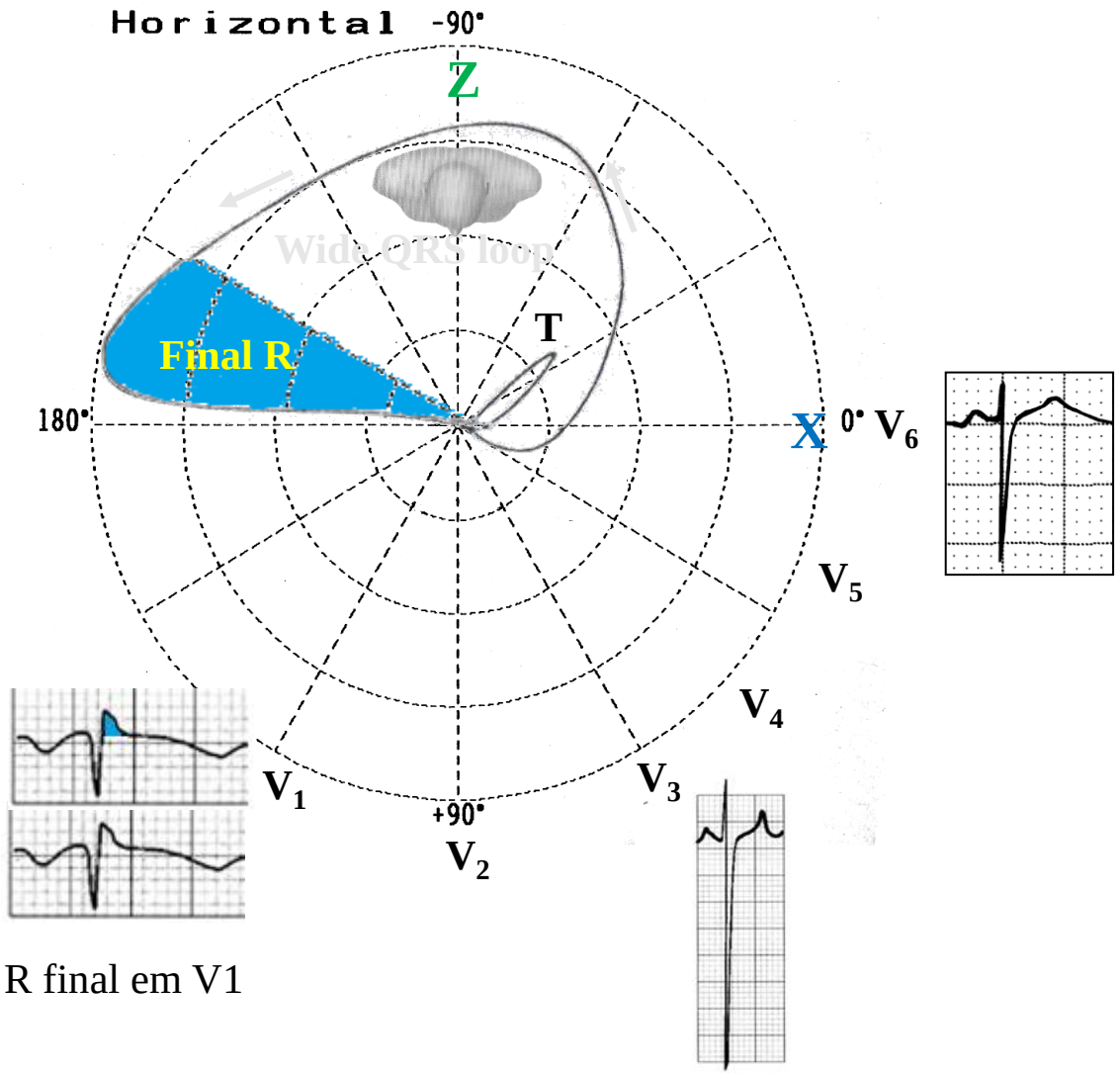
- Atrial Septal Defect (ASD).
- Rarely, Pulmonary Stenosis (PS).
- Tetralogy of Fallot (TOF) (only 10% of them).(**Pileggi 1960**).

Factors responsible for ECG/VCG modifications:

- Selective hypertrophy of the posterior portion of the RV outflow tract (crista supraventricularis);
- Clockwise rotation in the longitudinal axis;
- Verticalization of the organ and possible inferior dislocation by diaphragm descent;
- Withdrawal of exploring electrodes by pulmonary hyperinsufflation. As the air is a poor conductor, electrical forces generated in the heart have a lower voltage, especially in the frontal plane, since the mentioned forces are heading backward, perpendicularly to this plane;
- Possible RV dilatation and right-sided heart failure;
- Possible RA dilatation;

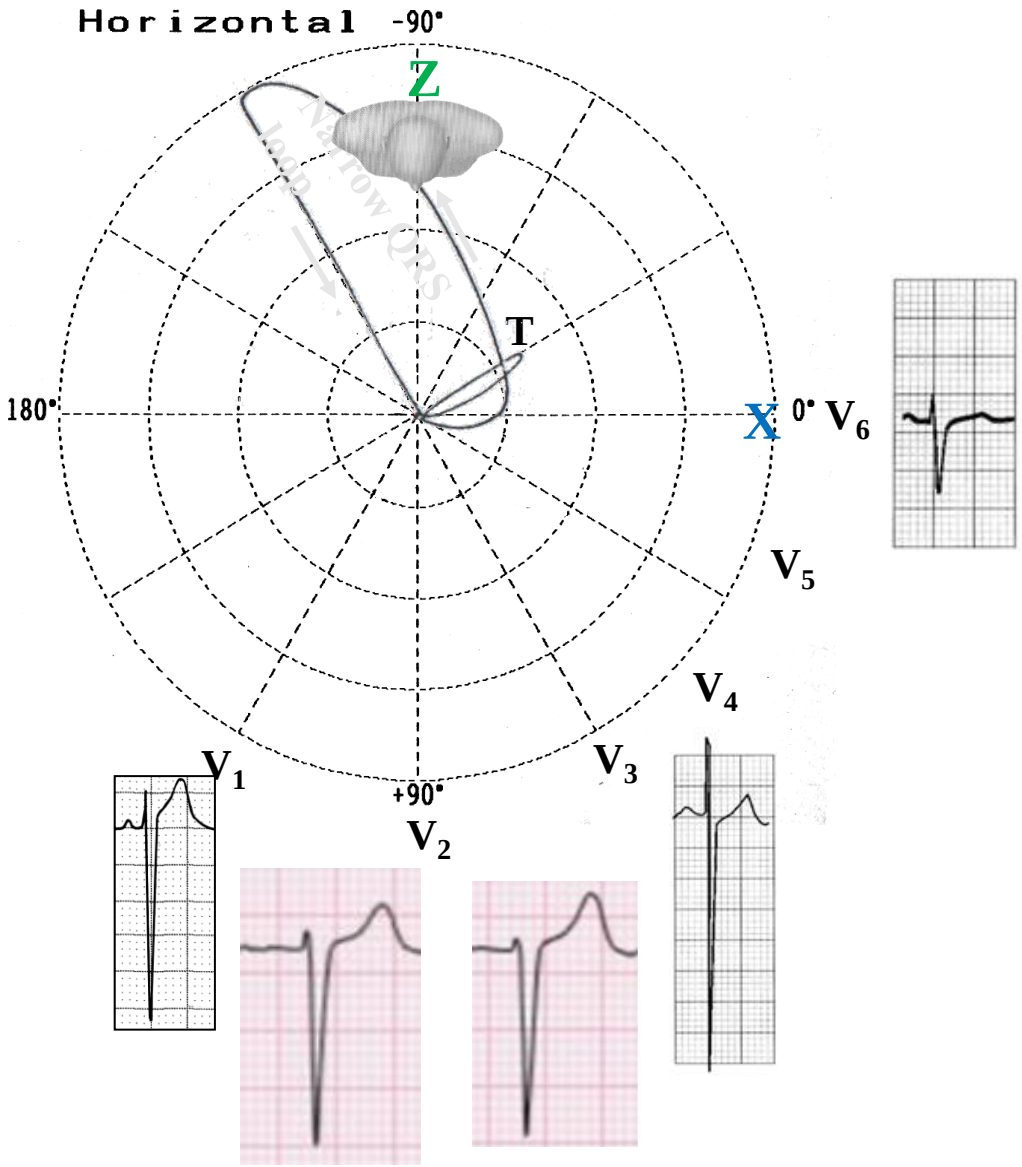
Type C or special RVH classification by VCG criteria in the HP

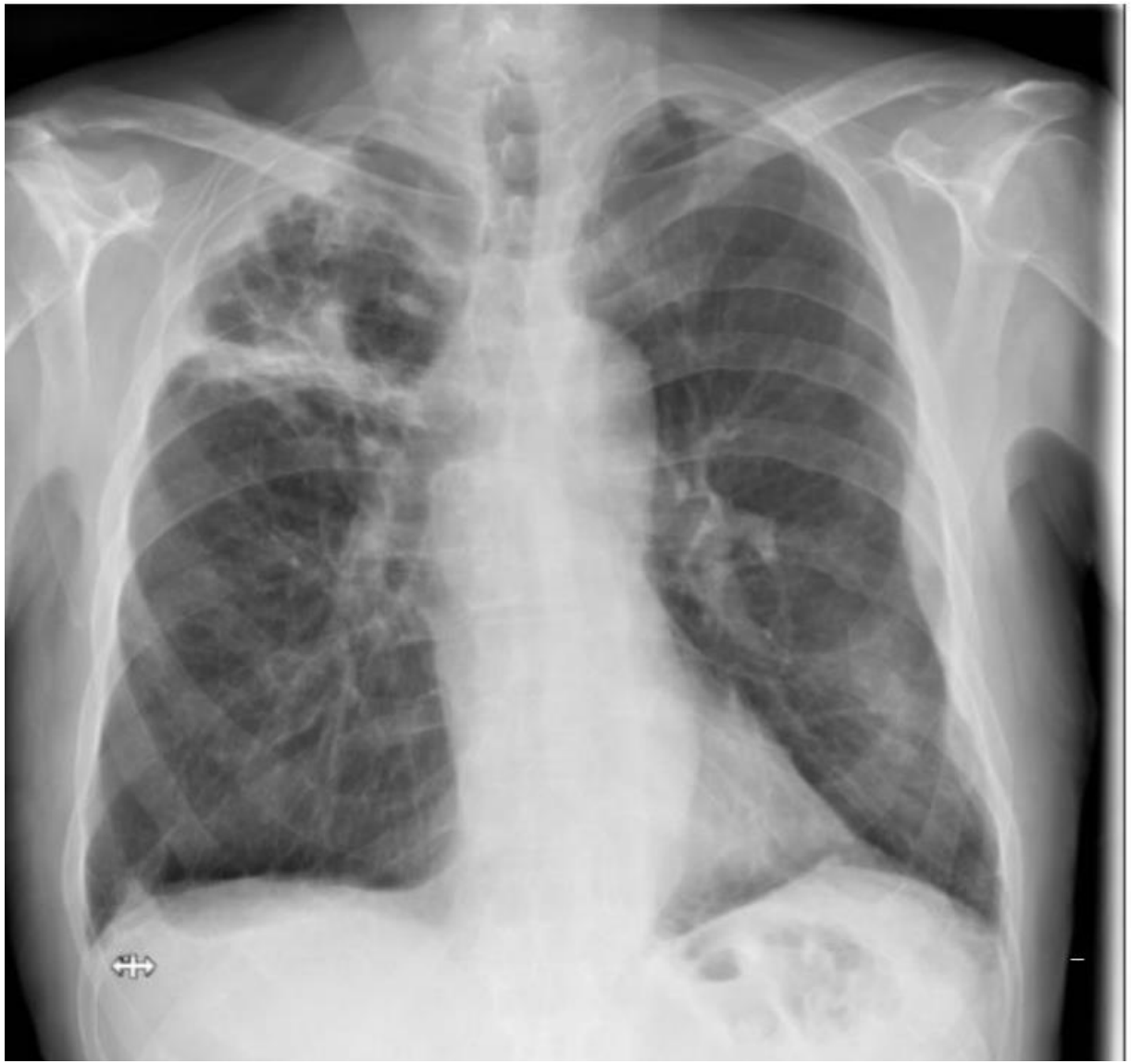
Type C-I

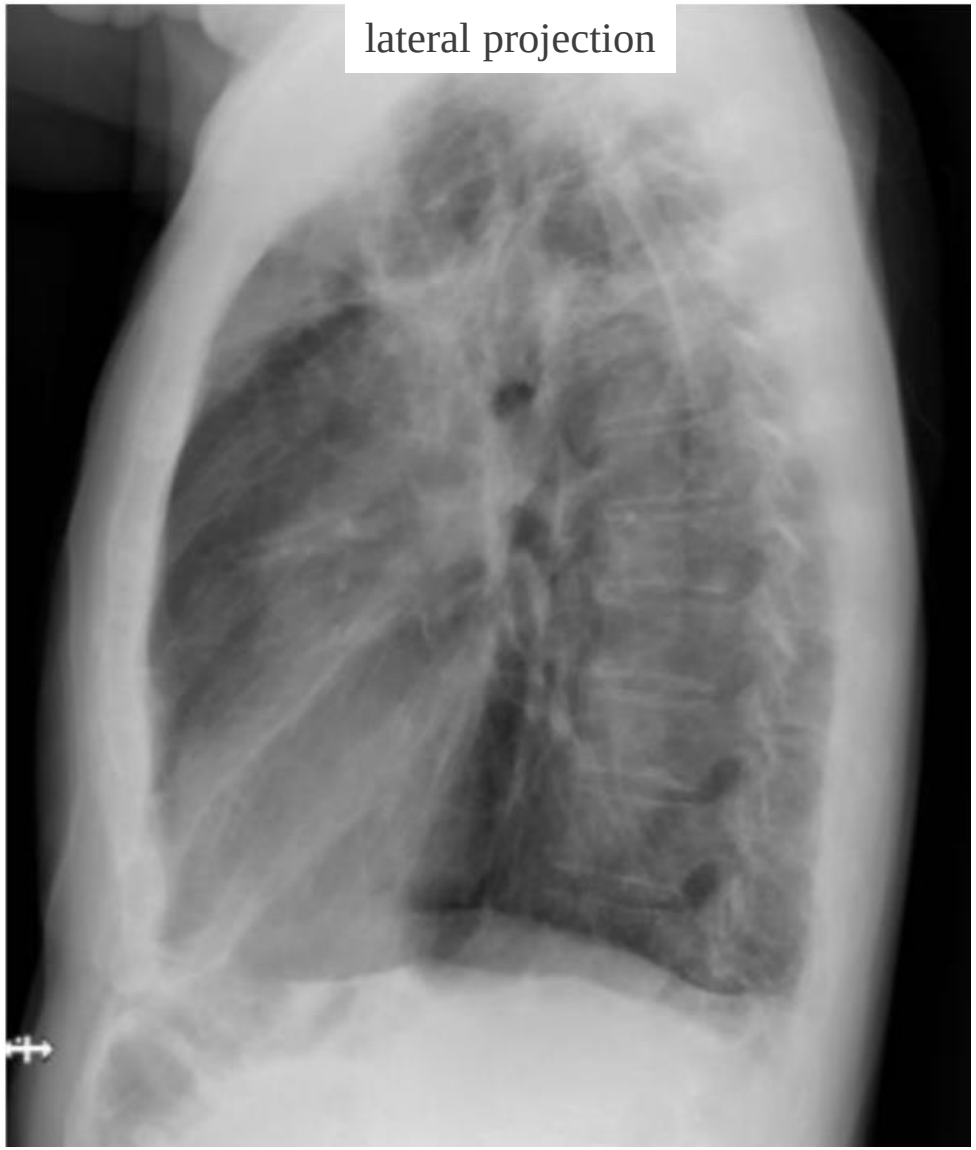


R final em V1

Type C-II







PA Chest X-ray projection shows prominent aortic knob (long time hypertensinn) Irregular opacities with a fine reticular pattern. Additional evidence of calcified or noncalcified pleural plaques. Emphysema presence of centrilobular dot-like or branching opacities, subpleural lines and parenchymal bands (They are more common in asbestosis than in idiopathic pulmonary fibrosis) Widespread ill-defined opacities projecting over both lungs. On lateral projection some of these lesions are seen to be plurally based. No convincing calcification.

Asbestosis

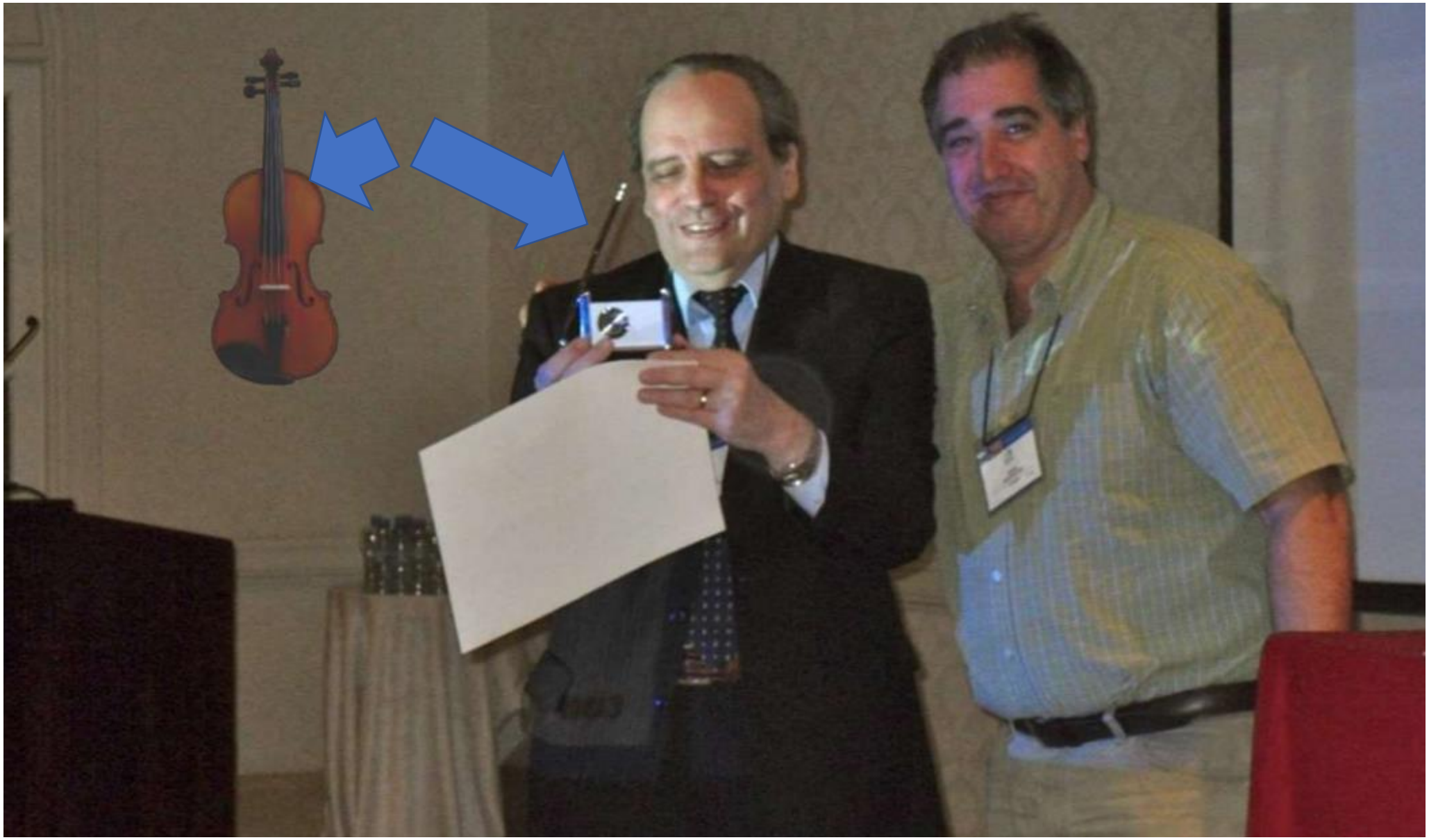
Asbestosis is a rare pneumoconiosis secondary to inhalation of asbestos fibers Asbestosis refers to later development of diffuse interstitial fibrosis secondary to asbestos fiber inhalation and should not be confused with other asbestos related diseases. Typically occurs 10-15 years following the commencement of exposure to asbestos and is dose related. Heavy asbestos exposure is predominantly encountered among men, as most exposures are occupational in the setting of construction, mining, or ship/automotive industries. Clinical presentation is insidious and nonspecific with shortness of breath prompting imaging. Alternatively, the presence of asbestosis may become evident when a patient presents with other asbestos related diseases. Pathology Asbestosis is histologically very similar to usual interstitial pneumonia with the addition of asbestos bodies. Typical changes on a high-resolution CT scan combined with relevant asbestos exposure is essential for the diagnosis. High resolution CT is the most performance investigation in particular in presence of asbestosis either minimal or of recent origin. Asbestosis and silicosis hospitalizations showed worsening trends such as increasing mortality and decreasing percentage of programmed hospitalizations.

The literature on ECG alterations consistent with RVH and functional chronic disorders of pulmonary circulation is abundant.

The ECG abnormalities most often encountered are: **P wave changes, right axis deviation of the QRS, rS pattern in the chest leads, clockwise rotation of the QRS, and ventricular conduction defects (RBBB and IRBBB**) Low prevalence of high right R wave and right ventricular conduction defects, which are usually considered to be the most reliable indicators of RVH suggestive of cor pulmonale(type C VCG RVH) . (1) have demonstrated, the occurrence of concomitant left heart disease may affect the usefulness of the ECG variables in the diagnosis of pulmonary heart disease, particularly for the precordial variables. the P wave variables, transitional zone (clockwise rotation), and RV 5,6 amplitude, while SV56 amplitude failed to show any association with ventilatory capacity. (1)

1. S Punsar, K Kokkola.ECG pattern of cor pulmonale and ventilatory function in tuberculous patients with left heart disease. Ann Clin Res. 1974 Apr;6(2):86-92.

Together 2 great colleagues, Pablo Ambrosio Chiale (in memorian) and Adrian Baranchuk (current editor in chief Journal of Electro cardiology)



Dear Pablo, we want to tell you that we miss you more and more each passing day. Adrián, how gratifying to have your friendship. Andrés, Raimundo and Nikus. https://www.youtube.com/watch?v=AUZ3UhxCMA0 Enjoy with his fiddle !!