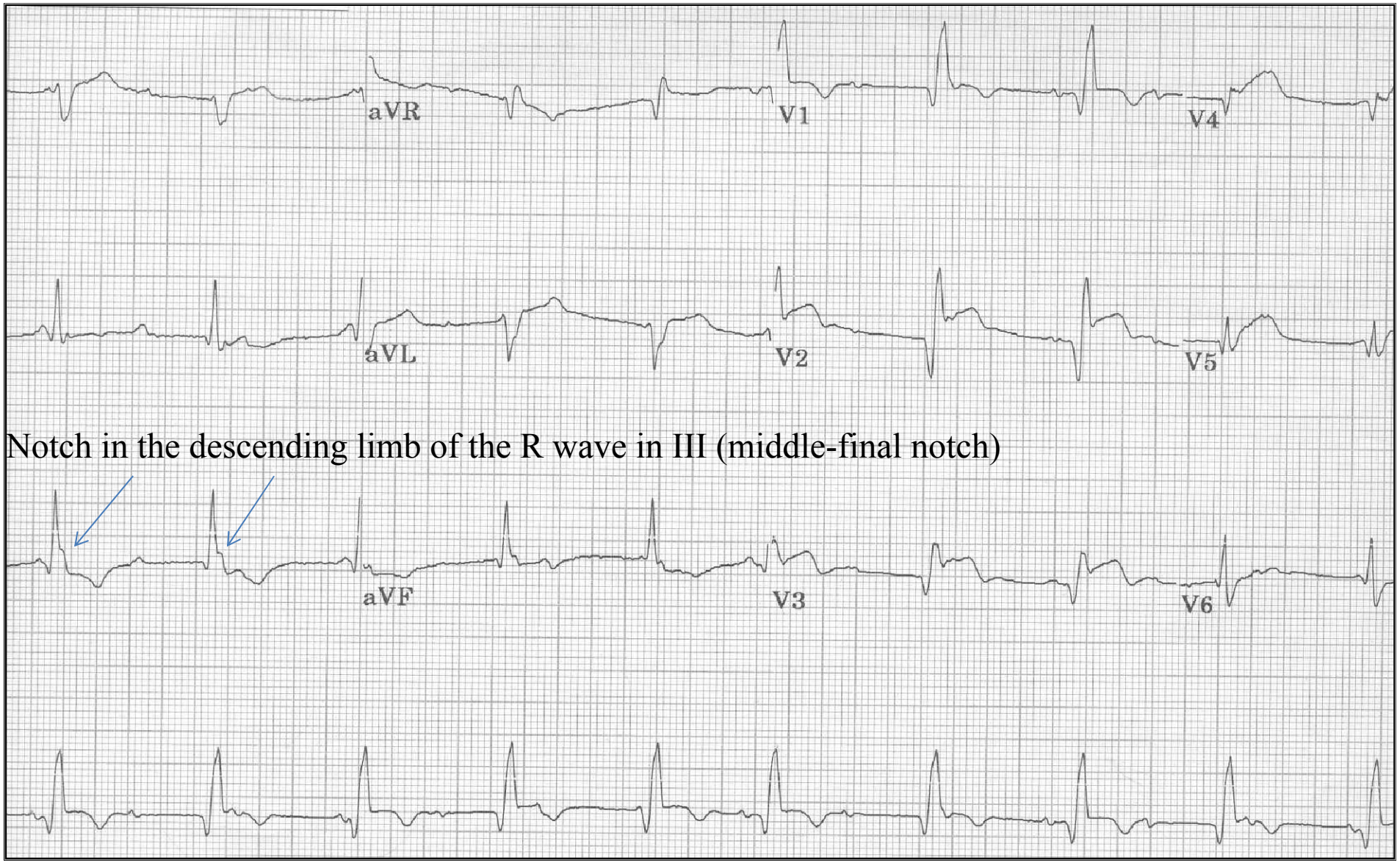
Elderly woman (76 yo.) with recent complicated Segment Elevation Myocardial Infarction (STEMI)

1. ***Which is ECG diagnosis?***
2. ***Which is the culprit artery?***



Notch in the descending limb of the R wave in III (middle-final notch)

This represents a proximal LAD occlusion (ST elevations also in I and aVL). There are Q waves and ST segment is still elevated followed by positive T waves in the leads with ST elevations, which indicates suboptimal reperfusion at the myocardial cell level. According Dr Samuel Sclarowsky the presence of this pattern is indicative of evolving MI without reperfusion (**1;2**).
The reason for suboptimal myocardial reperfusion could be:

1. The artery is still occluded and collateral flow is insufficient, or \
2. Distal embolization spontaneously or
3. Induced by PCI(2)

1. **Eskola MJ, Holmvang L, Nikus KC Sclarovsky S, et al. The electrocardiographic window of opportunity to treat vs. the different evolving stages of ST-elevation myocardial infarction: correlation with therapeutic approach, coronary anatomy, and outcome in the DANAMI-2 trial. Eur Heart J.2007 Dec;28(24):2985-91.**
2. **Nikus commets**

RBBB and LPFB pattern may be pre-existing or, much possible, they may be induced by the acute coronary occlusion being proximal in the left anterior descending artery (LAD).
There is a high-degree or advanced AV block; which in acute LAD scenario indicates at least 2-vessel disease occlusion, because either the right coronary artery (RCA) or the left circunflex (LCX), which provide irrigation to the LPF, are affected. It could even be that LAD was the vessel, which supplied collateral flow to a chronically occluded RCA, and when also LAD was occluded, advanced AV block developed.

The following are criteria of advanced AV block:

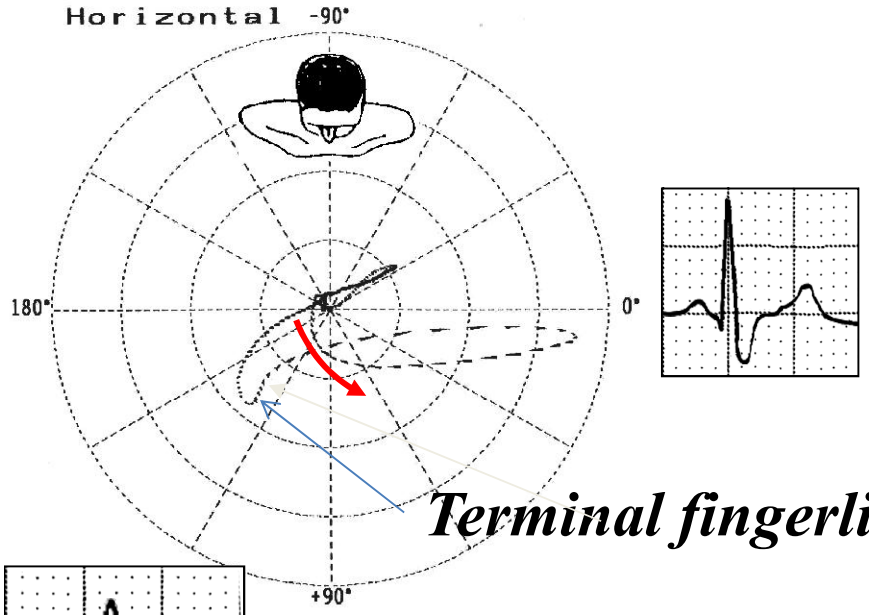
1. Two or more successive P waves are blocked;
2. AV ratio $\geq$ 3:1;
3. Constant PR interval in all conducted beats;
4. Atrial rate greater than the ventricular one;
5. The clinical significance is comparable to complete AV block;
6. The phenomenon of concealed conduction may be responsible by the ventricular response failure.

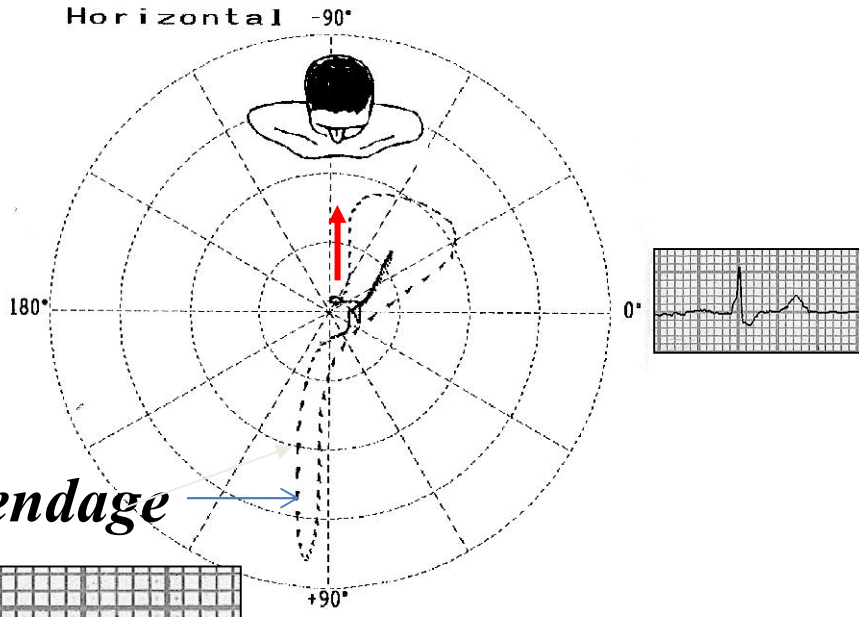
Comparison between Uncomplicated RBBB and RBBB associated with anterior MI ECG and VCG

	Uncomplicated RBBB	***RBBB asscociated with anterior MI***
The initial 40ms defection	*It is written to the right and anteriorly, just as normally.*	*It is directed to back.*
Right precordial leads QRS pattern V2-V3:	*Triphasic rSR`.*	*Biphasic QR or qR.*
Final 60-80 ms QRS forces	*On right anterior quadrant. These Late forces project a prominent wide and slurred terminal R´ on leads V_{3R} and V1 and a wide, shallow terminal S wave on leads I and V6*	*On right anterior quadrant Idem*
Terminal VCG forces on HP	*Terminal finger-like appendage of the QRS loop, which is written slowly to the rigth and anteriorly*	*Idem*

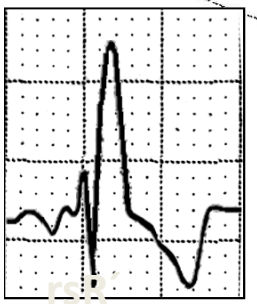
Uncomplicated RBBB

RBBB asscociated with anterior MI

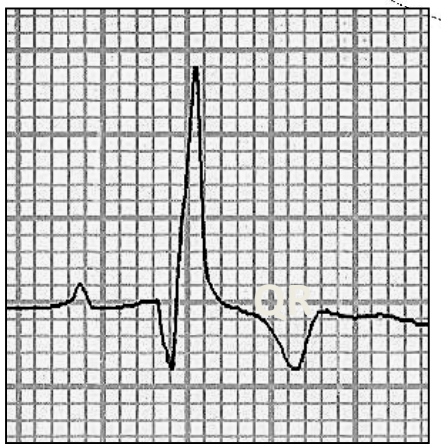




Terminal fingerlike appendage



Triphasic QRS pattern rsR’



Biphasic QRS patter QR or qR

Am I sure that the pattern is consequence of LPFB?
Answer: Yes.
Why?
Because the shape of R wave in III is characteristic: Notch in the descending limb of the R wave in III (middle-final notch)

1) Frontal plane axis between +90 and 180 degree in adults;
2) rS pattern in leadsI and aVL
3) qR pattern in III, aVF and II: Q wave is always present in III and may be small or absent in II or aVF.
4) Notch in the descending limb of the R wave in DII (middle-final notch);
5) RIII > RII: SAQRS closer to $+120^0$ (DII) than $+60^0$ (II), when closer to the latter, it would indicate an incomplete form of LPFB.
6) The q wave in III is always greater than the q wave in II and aVF. If there is association with inferior infarction, the Q wave > 40 ms.
7) QRS duration less than 120ms if isolated(without RBBB)

Left Posterior Fascicular Block (LPFB) Possible causes

It is the most rare block of all intraventricular blocks. Very rare without association with others blocks.

1) Coronary insufficiency: it constitutes the main cause in the first world, associated or not to infarction, especially inferior or inferolateral myocardial infarction.(ancient dorsal MI)
   (2a) During the acute phase of ischemia.
   (2b) During the acute phase of infarction: 0.2% to 0.4%
3) Lenègre disease, progressive cardiac conduction defect (PCCD) or "idiopathic" sclerosis of the intraventricular His system: by mutation in the SCN5A gene, the same one affecting Brugada Syndrome(Allelic diseases).
4) Lev disease or progressive idiopathic sclerosis of the "cardiac skeleton". With a clinical behavior similar to Lenègre disease, however, it occurs in elderly patients;
5) Aortic insufficiency: attributed to the mechanical effect of jet regurgitation on the posterior portion of the left septum, the site that the thick LPF goes through (LV inflow tract);

6. Aortic stenosis;
7. Aortic stenosis associated with aortic insuficience
8. Supravalvar aortic stenosis;
9. Coarctation of the aorta;
10. Dissecting aortic aneurysm;
11. Massive calcification of the “cardiac skeleton”;
12. Chronic chagasic myocarditis: the most frequent one in Latin America.
13. Others Cardiomyopathies;
14. Myocarditis;
15. Infiltrative myocardial diseases;
16. Systemic hypertension;
17. Interventricular septum tumor;
18. Hyperpotassemia;
19. Transitorily, during contrast injection in the right coronary artery and in
20. Acute pulmonary embolism.?

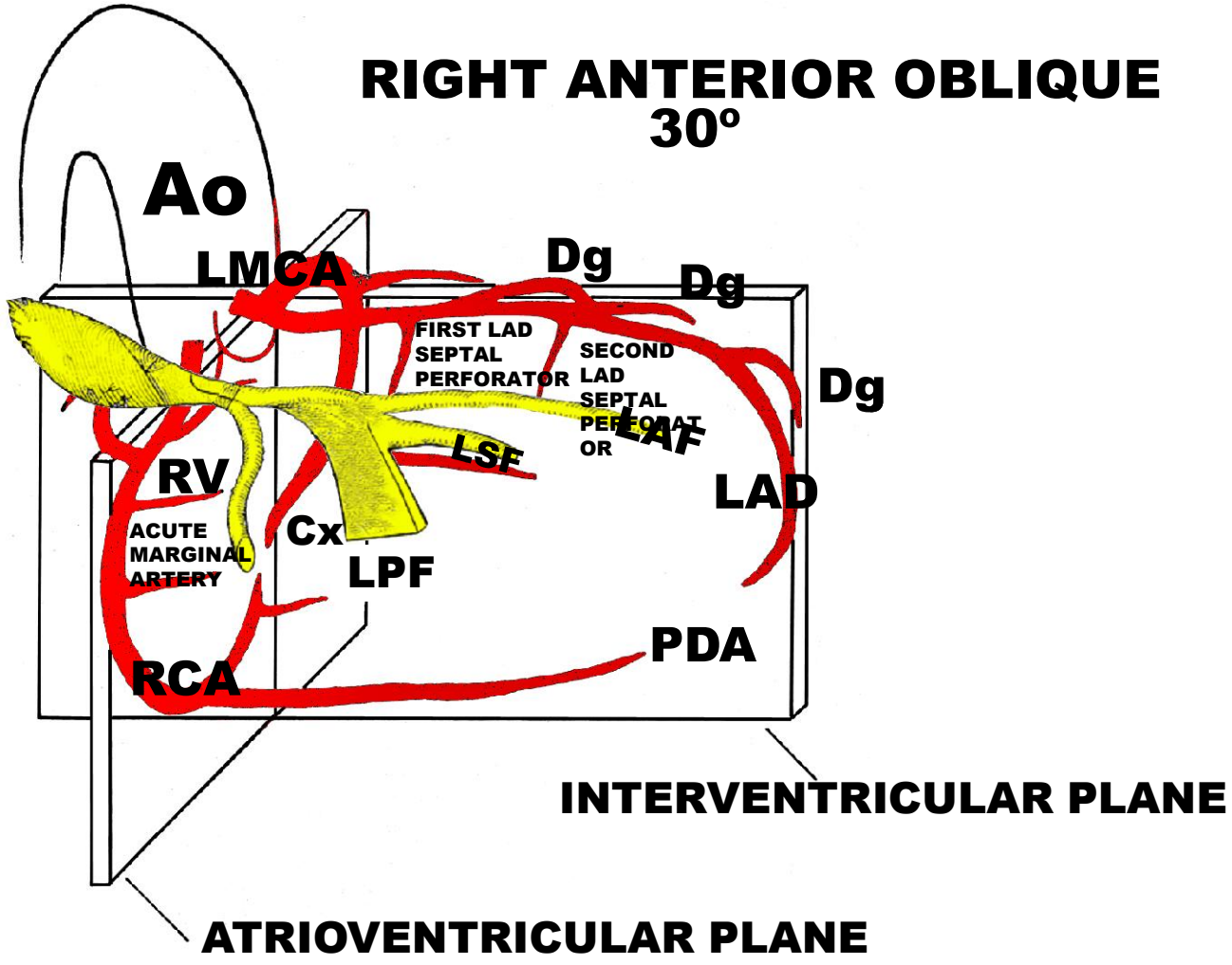
BLOOD SUPPLY OF LEFT BRANCH FASCICLES

Responsible system	LAF	LPF	LSF
Branches of the LAD:	40 %	10 %	100 %
Double irrigation (LAD & RCA)	50 %	40 %	0 %
Branches of the RCA	10 %	50 %	0 %

LAD – Anterior Descending Artery
RCA – Right Coronary Artery

Table that shows the percentage irrigation of the left branch fascicles.

INTRAVENTRICULAR HIS SYSTEM IRRIGATION



Outline that shows the irrigation of the intraventricular His system by the coronary system.

CAUSES OF GREATER VULNERABILITY OF THE LEFT ANTERIOR FASCICLE (LAF) IN COMPARISON TO THE LEFT POSTERIOR FASCICLE (LPF)

1) ANATOMICAL:

a) Less diameter (LAF: 3 mm; LPF: 6 mm).

b) Greater extension (LAF: 35 mm; LPF: 30 mm).

2) ELECTROPHYSIOLOGICAL:

As a consequence of its greater extension and less diameter, the depolarization and repolarization of LAF is slower than LPF, i.e. the "QT of LAF" is greater than the one of LPF, a fact that makes it more vulnerable.

3) VASCULAR.

Posterior fascicle always irrigated by the two systems of the LAD and RCA.

4) TOPOGRAPHIC.

The LPF runs through a more protected area, with less pressure mechanic impact.

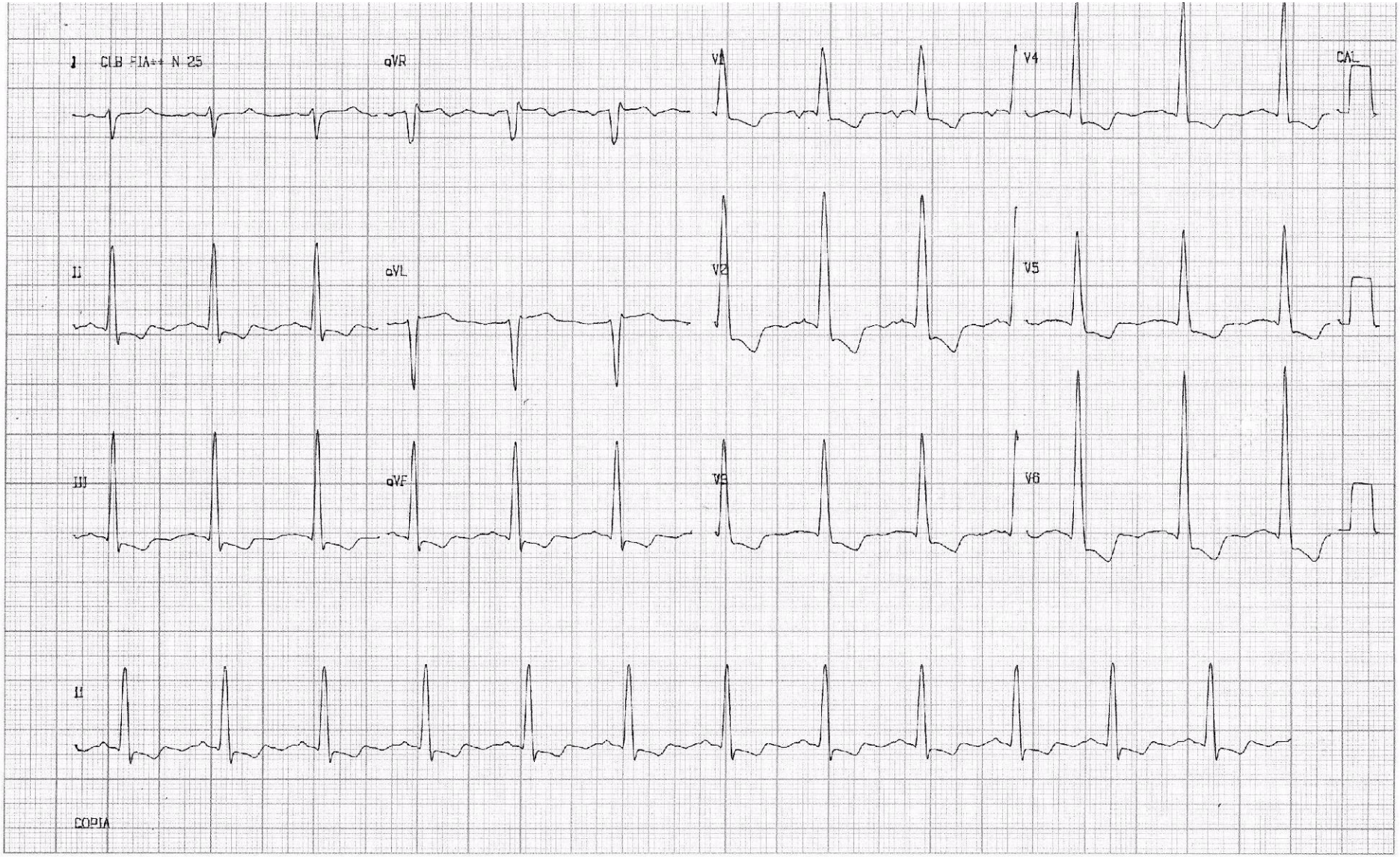
Anatomical, electrophysiological, vascular and topography causes for the greater vulnerability of the LAF compared to the LPF.

TOPOGRAPHIC CAUSE, RESPONSIBLE FOR THE GREATER VULNERABILITY OF THE LAF IN COMPARISON TO THE LPF

The LAF runs diagonally through the Left Ventricle Outflow Tract (LVOT) by the subendocardium. This region is subject to a great turbulence and high pressure, which justifies the greater vulnerability of the LAF when compared to the LPF, which runs through an area in the LV Inflow Tract (LVIT), which is much less exposed to turbulence, which explains the rarity of the LPFB.

Explanation of the greater topographic vulnerability of the LAF compared to the LPF.

Examples of
Left Posterior Fascicular Block
In association with Left Septal Fascicular Block

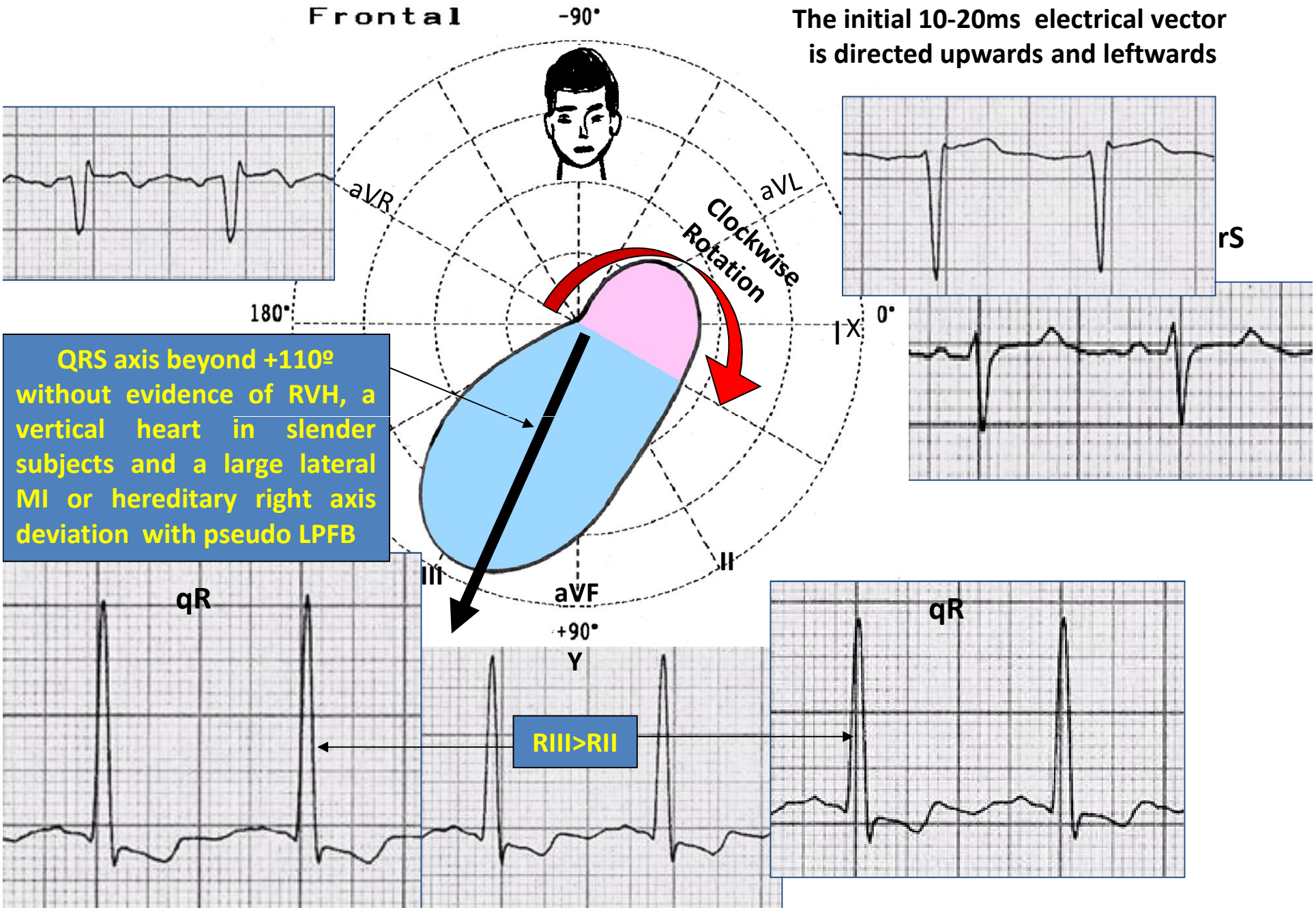


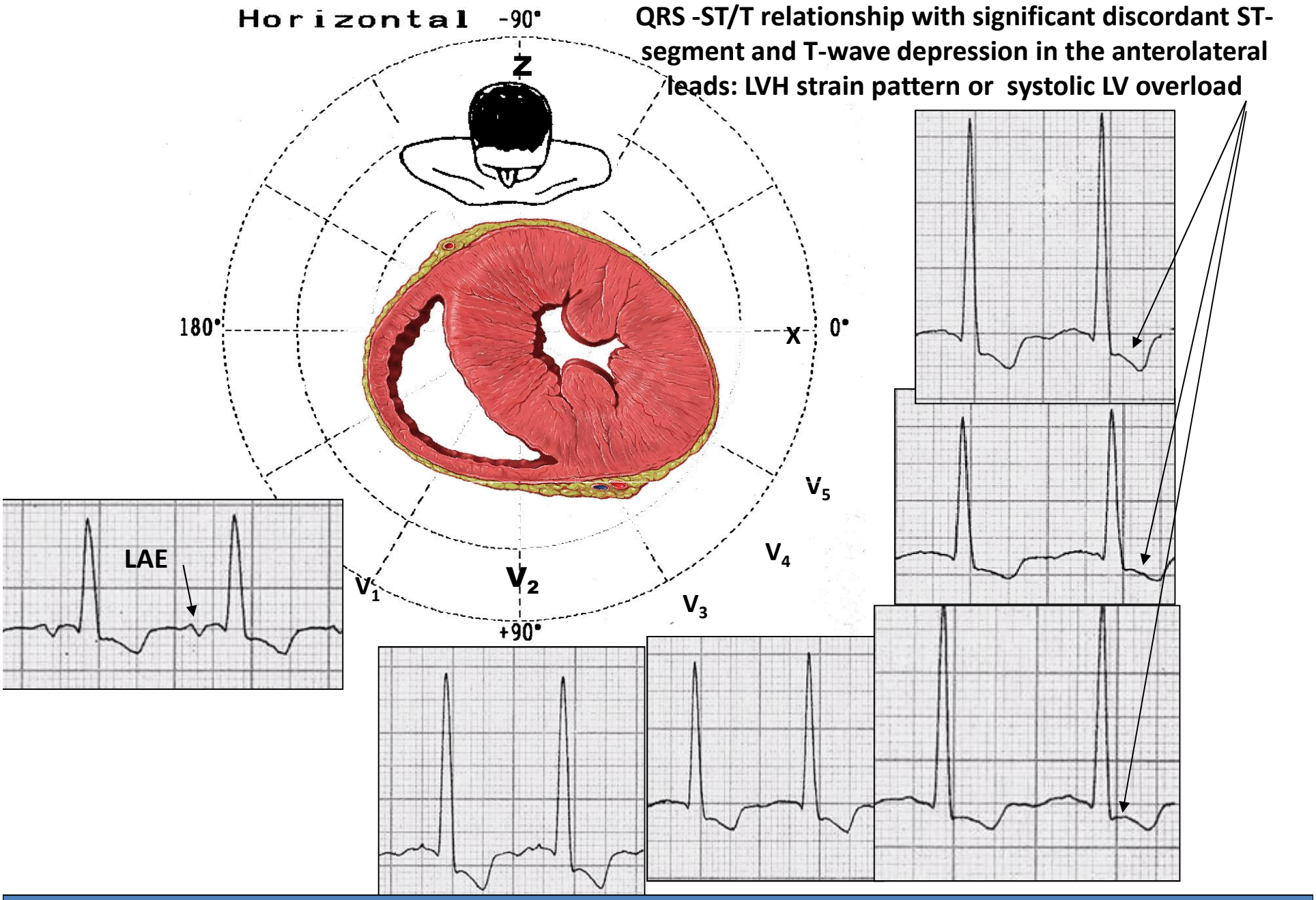
ECG-1 preformed in October 22/2012, 10:50h A.M. Few minutes before surgery of aortic valve substitution by severe congenital calcified bicuspid aortic valve disease in October 2012 (double aortic lesion of the stenosis type). The echocardiogram showed moderate to severe left ventricular hypertrophy (septal and free wall thickness 15 mm) with normal right chambers. The strategy was valve replacement by biological prosthesis. The surgery was successfully made on October 22, 2012, at 10:50 h.

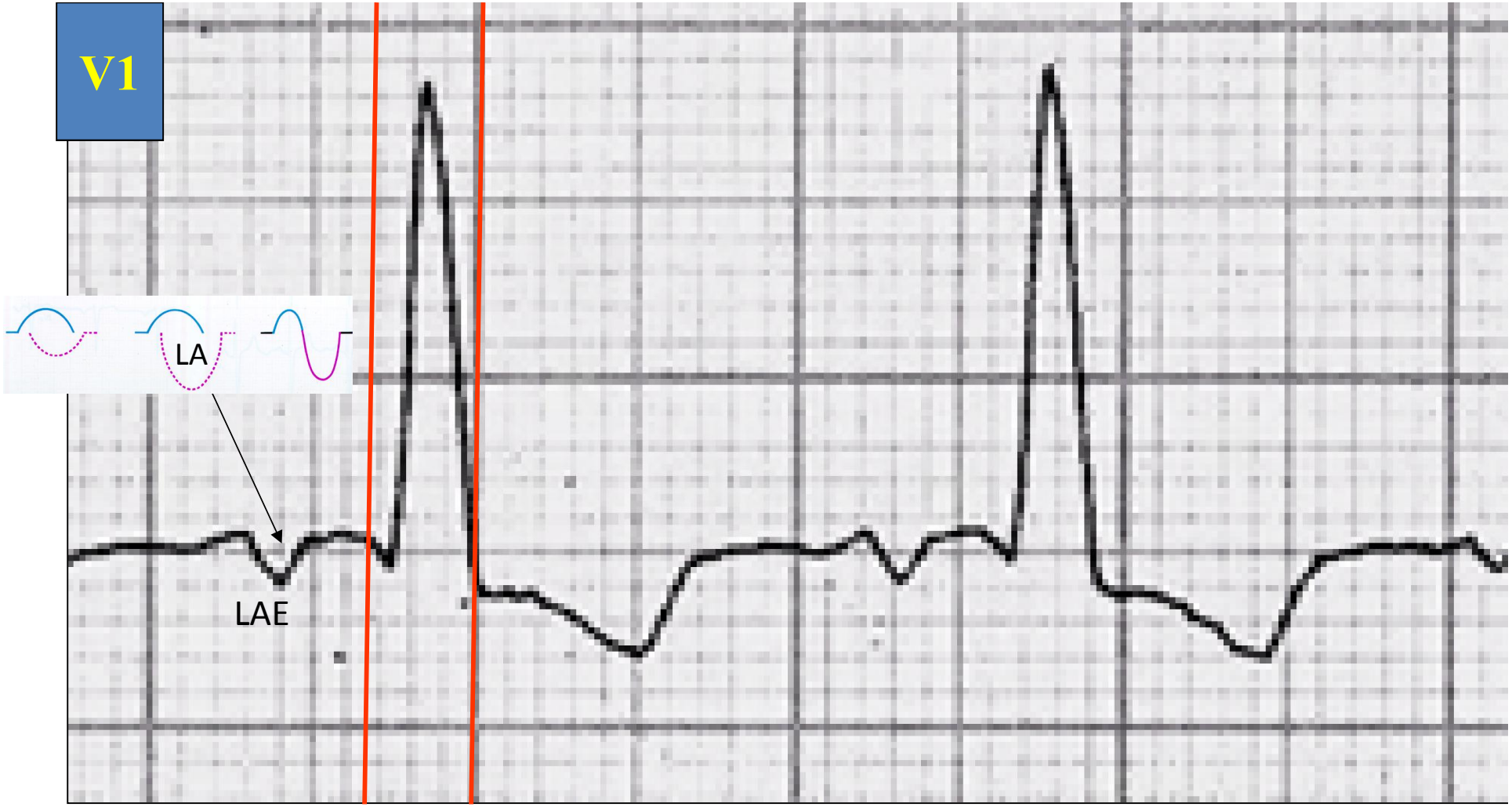
ECG-1 diagnosis: Sinus rhythm, heart rate 73bpm, P axis + 60º and to front in PH, LAE in V1, PR interval 130ms, QRS axis + 115º, (beyond +110º in adults). QRS duration =130ms, absence of broad final S waves in left leads and absence of final wide R wave in aVR, rS pattern in leads I and aVL (small initial r waves followed by deep S waves), qR pattern in inferior leads (II, III and aVF) with initial small q waves, RIII>RII. Clearly, the initial 10-20ms electrical vector is therefore directed upwards and leftwards (as excitation spreads outwards from endocardium to epicardium), causing small initial r waves in the high and low lateral leads (I and aVL-V5-V6) and concomitant small initial q waves in the inferior leads (II, III and aVF), right QRS axis deviation on FP without evidence of right ventricular hypertrophy, a vertical heart in slender subjects and a large lateral infarction. **(Elizari 2007) (1).** Additionally, hereditary right axis deviation with pseudo left posterior fascicular block and incomplete right bundle branch block **(2)** **(Lorber 1988)** . In the frontal plane, the main and terminal forces of the QRS loop are oriented inferiorly and to the right (>100°) with a wide-open clockwise-rotated QRS loop Actually, the ECG and VCG of LPFB is the exact mirror picture of LAFB in the standard and unipolar leads. Because isolated LPFB is extremely rare, when present it is almost always associated with RBBB or as in the present case LSFB. Never described before in literature**. (Elizari 2007) (1)**
Prominent QRS anterior forces (PAF) across all precordial leads, with qR pattern from V1 to V6.
QRS -ST/T relationship with significant discordant ST-segment and T-wave depression in the anterolateral leads: left ventricular strain pattern or systolic LVH overload **(Cabrera 1952) (2;3;4).** The ST segment and the T wave in the direction opposite to the main QRS vector causes widening QRS amplitude and wide QRS/T angle

Conclusions

1. **Left ventricular enlargement/hypertrophy with strain pattern of repolarization**
2. **Left Posterior Fascicular Block (LPFB)**
3. **Left Septal Fascicular Block (LSFB)**
4. **Left Bifascicular Block. A LPFB can occur in the setting of a bifascicular block as well.**


Frontal
-90°
aVR
aVL
Clockwise Rotation
180°
0°
I
II
III
aVF
+90°
Y
rS
qR
qR
RIII>RII
QRS axis beyond +110º without evidence of RVH, a vertical heart in slender subjects and a large lateral MI or hereditary right axis deviation with pseudo LPFB
The initial 10-20ms electrical vector is directed upwards and leftwards


Horizontal
-90°
Z
180°
X
0°
+90°
V1
V2
V3
V4
V5
LAE
QRS -ST/T relationship with significant discordant ST-segment and T-wave depression in the anterolateral leads: LVH strain pattern or systolic LV overload
Prominent QRS anterior forces (PAF) across all precordial leads, with qR pattern from V1 to V6:
Left Septal Fascicular Block + Left Ventricular Hypetrophy/Left Ventricular Enlargement



QRS duration 130ms

QRS duration = 130ms. Why? Because Normal QRS duration = 90ms +2ms of LPFB =110ms + 2ms of LSFB = 130ms.

Final diagnosis