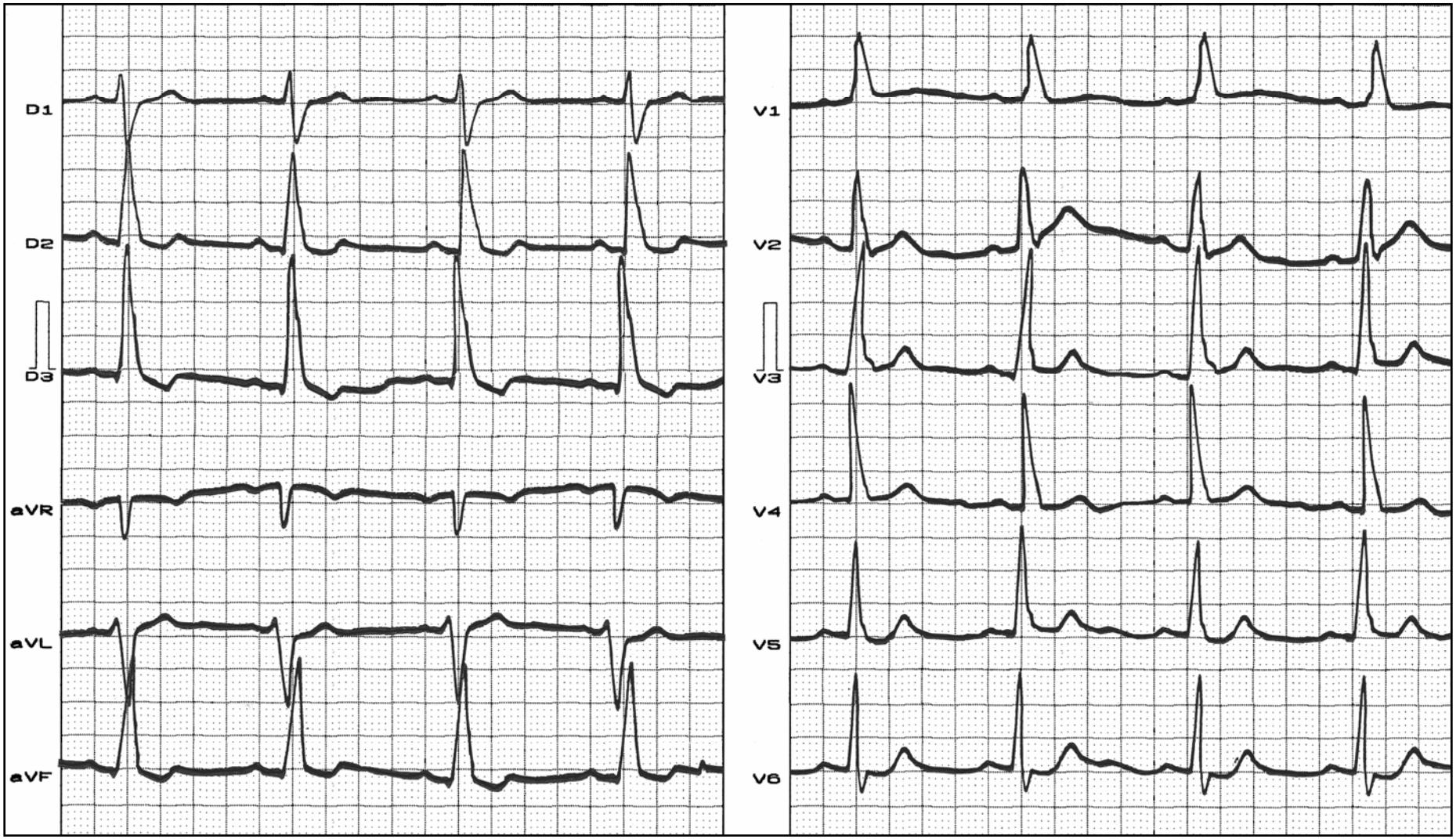
Dear colleagues,
This trace belongs to a old centripetal obese man with moderate to severe high blood pressure. The last Month he had two episodes of syncope.
Which is the ECG diagnosis?
Which is the appropriated approach?
Hug.
Andrés.

Prezados colegas,
Este traçado pertence a um homem portador de obesidade centrípeta e hipertensão arterial sistêmica moderada a severa. No último mês ele teve dois episódios de síncope.
Qual é o diagnóstico? E qual a conduta adequada neste caso?
Abraço,
Potro.

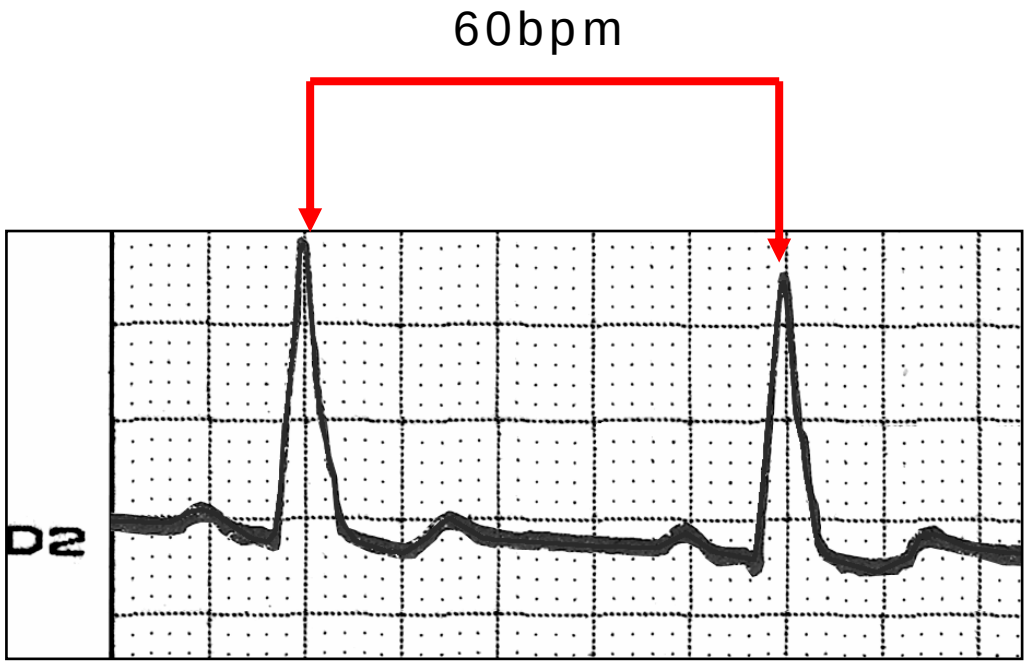
Name: HPP; **Date:** 11/08/2009; **Age:** 74 Y0; **Gender:** M; **Ethnic group:** Caucasian; **Weight:** 78 Kg; **Height:** 1.55 m;
Biotype: Endomorph ; **Medication in use:** anlodipino 5+ clortalidona 12,5+ Enalapril 20 2 x



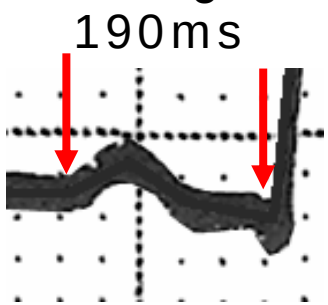
Clinical diagnosis: Hypertensive cardiopathy with two episodes of syncope.
ECG Diagnosis: ?

ECG Diagnosis

Rhythm: sinus
HR = 60bpm

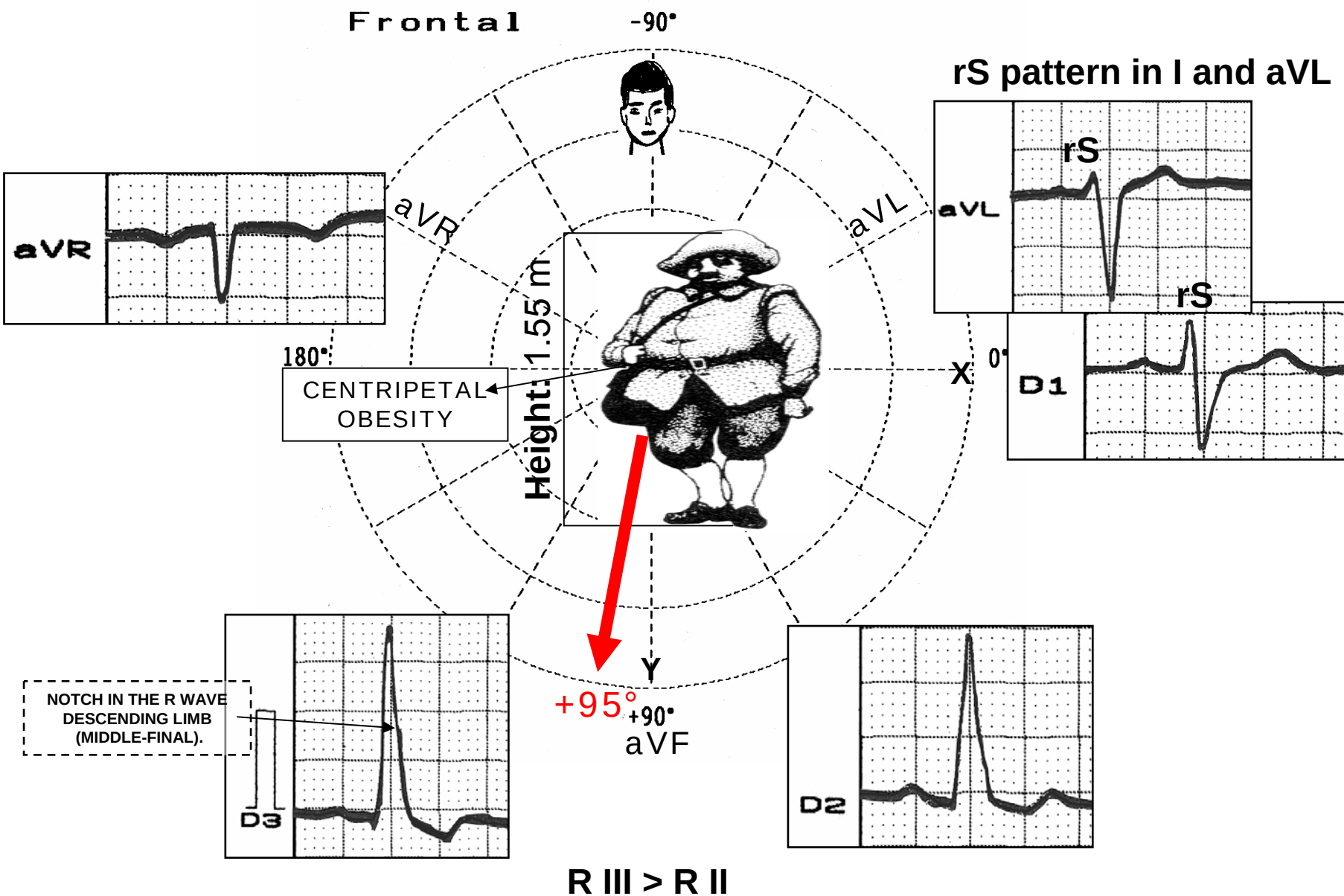


P-wave: duration: 95ms; voltage:1mm; shape: round; P-axis:+55° to front (positive in V2)
PR interval = 190ms

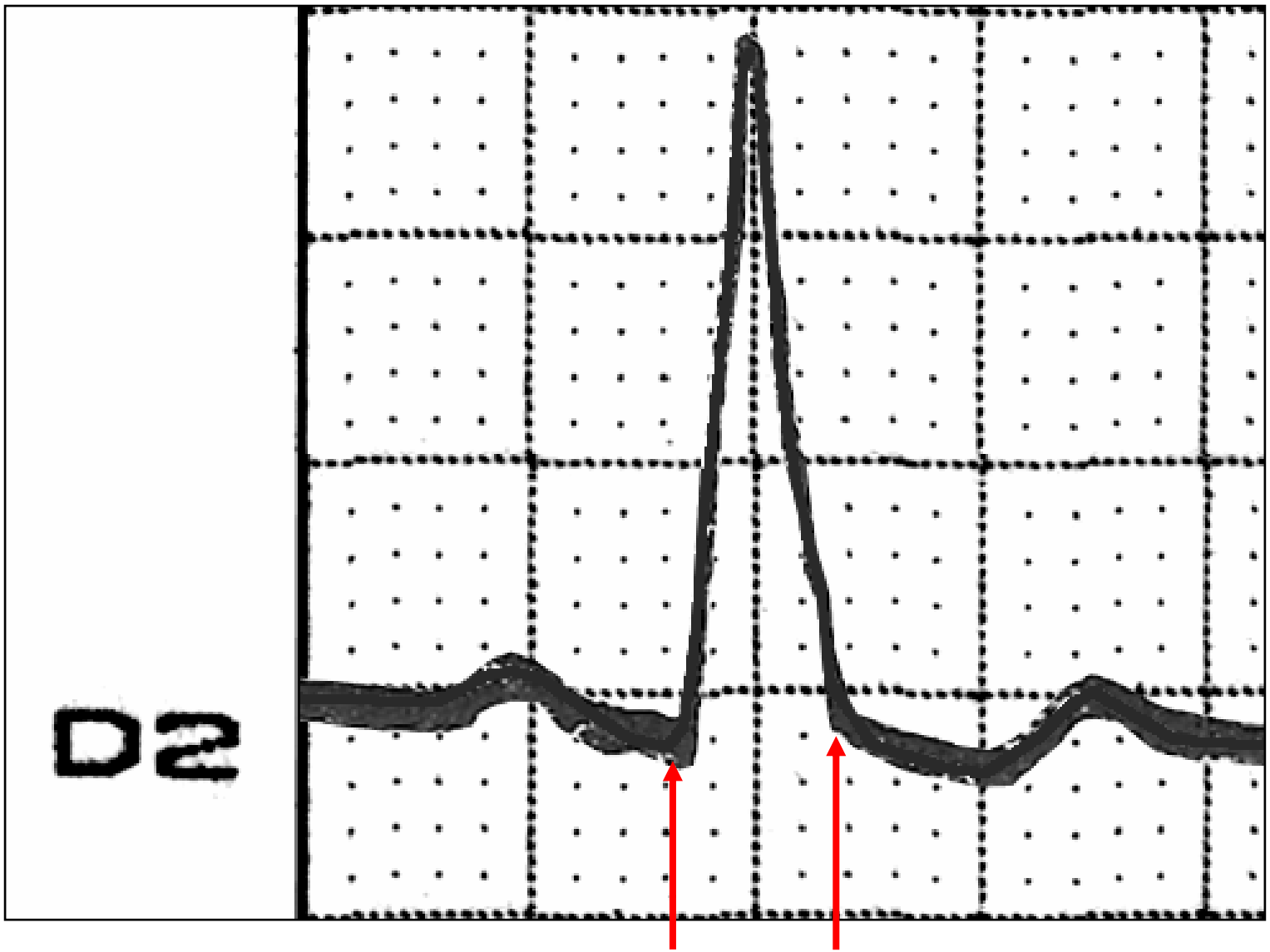


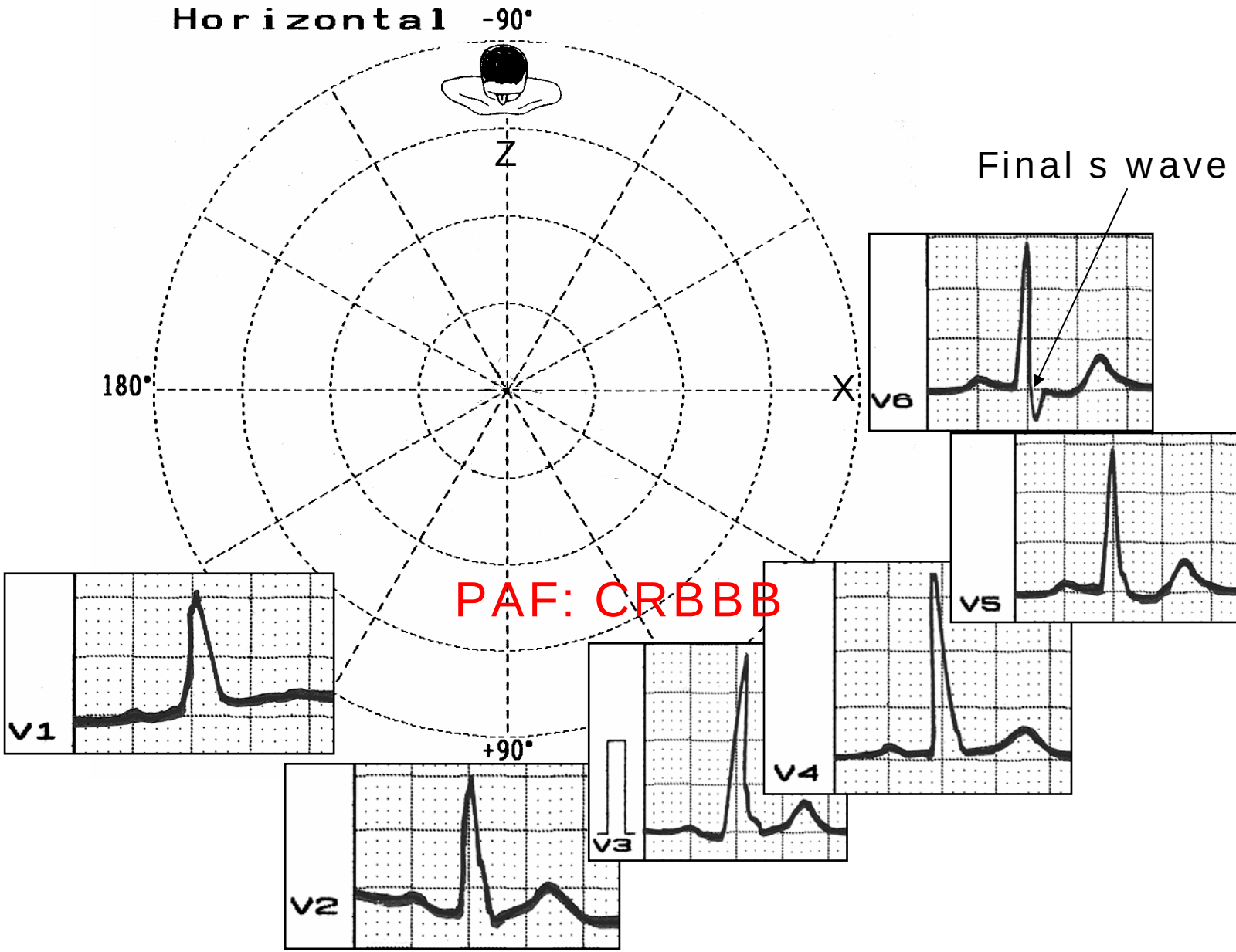
QRS AXIS = +95°

Why right axis deviation in a short centripetal obese hypertensive man?



QRS DURATION = 150ms



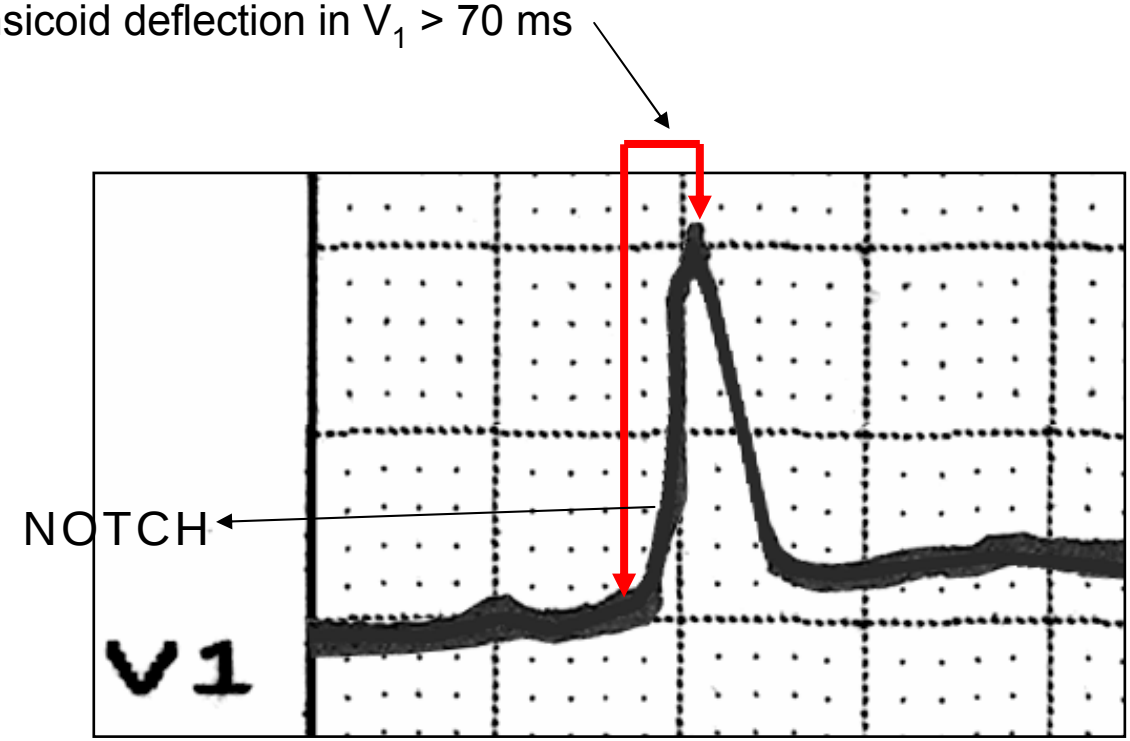


PAF: Prominent Anterior Forces CRBBB: Complete Right Bundle Branch Block

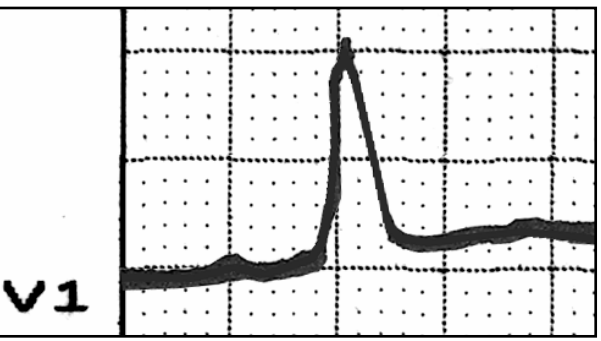
ECG diagnosis: bifascicular block: CRBBB associated to LPFB.

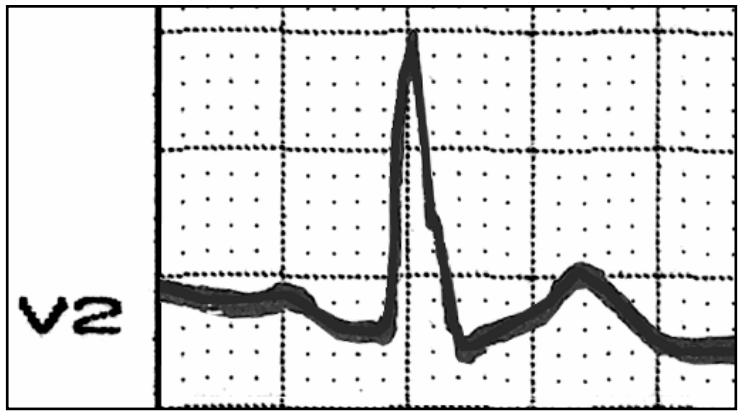
CRBBB diagnosis is made by the following criteria:

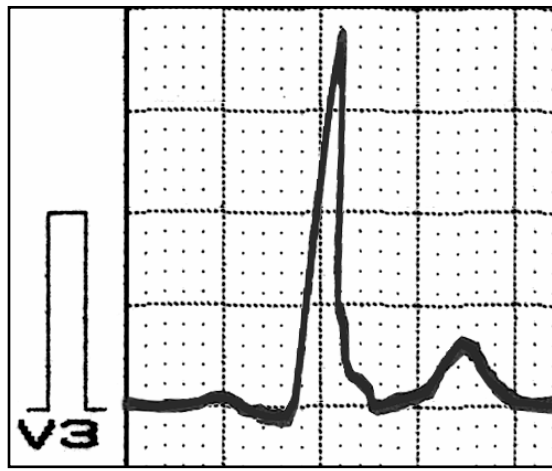
- QRS duration > 120 ms in the presence of supraventricular command
- V_1 lead with monophasic R wave with notch in ascending ramp; equivalent to rsR' CRBBB
- Intrinsicoid deflection in V_1 > 70 ms



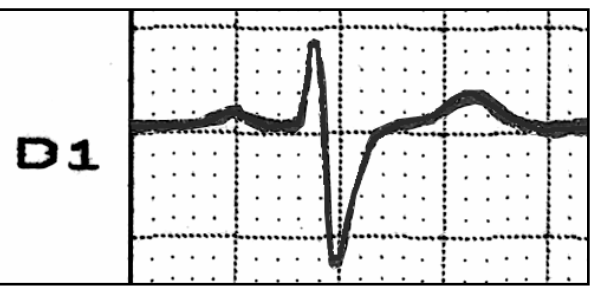
•Ventricular repolarization (ST-T) is not opposite to QRS complex: primary repolarization in anteroseptal wall?

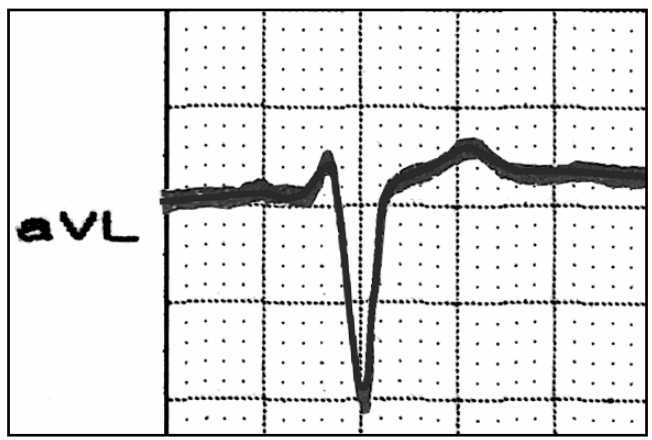


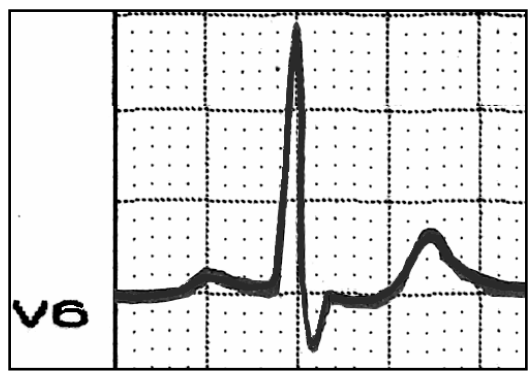




•Final S wave in left leads I, aVL and V6.

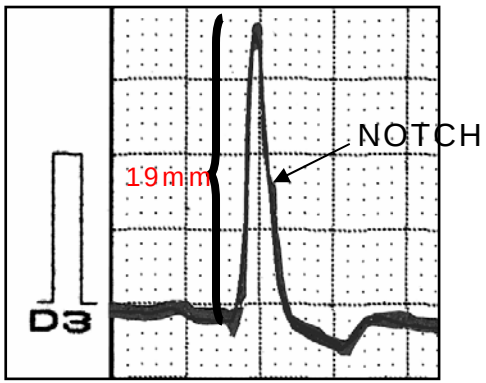


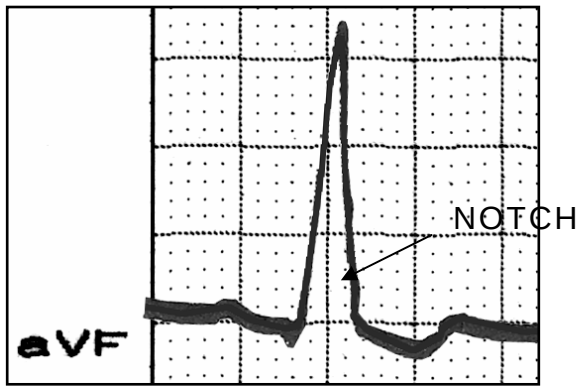


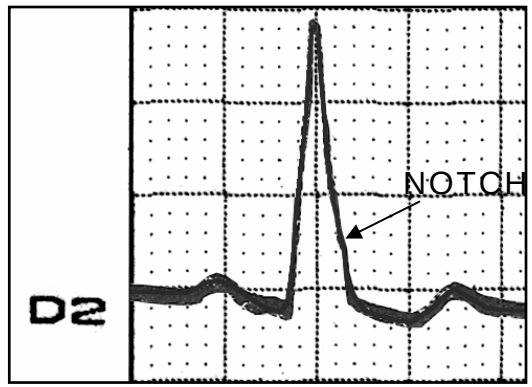


The diagnosis of LPFB is based on the following criteria:

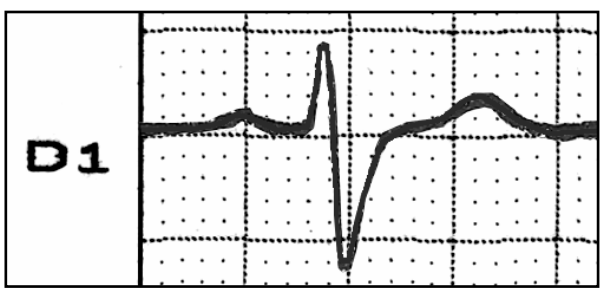
- Shift of QRS axis to the right : $+95^0$ in absence of vertical heart or RVH. We have LVH, obesity and horizontal heart. The diagnosis of LPFB is always clinicoeletrocardiographic.
- Inferior leads III, aVF and II with characteristic qR pattern

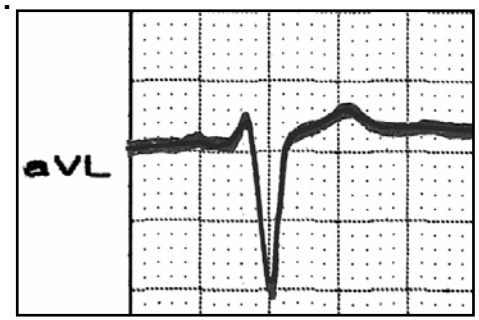






- SI-QIII pattern
- R wave in inferior leads of increased voltage
- R wave voltage in III >15 mm
- R III > RII;
- R wave of III, II and aVF with notch in descending ramp
- Complexes of the rS type in I and aVL.





In an elderly patient without right ventricular enlargement, endomorph biotype, centripetal obesity this pattern is evidence of disease of the right bundle brunch and the left posteroinferior fascicle of the left bundle.

This situation is often accompanied by transient heart block or the development of permanent heart block and, in a patient with a history of syncope episodes, indicates the need for a cardiac pacemaker implantation.

The possible causes of LPFB (the more rare intraventricular block) are(1;2):

- **Coronary insufficiency:** it constitutes the main cause in the first world, associated or not to infarction, especially inferior or inferodorsal myocardial infarction(MI) or inferolateral MI.
  - (2a) During the acute phase of ischemia(2;3).
  - (2b) During the acute phase of inferior myocardial infarction: 0.2% to 0.4%(4)

15 cases of LPFB associated with AMI were studied. In 5 cases the LPFB was the only intraventricular conduction defect, while in the other 10 cases it was associated with CRBBB. LPFB proved to be an early complication, appearing within a few hours from the onset of the acute episode, and an ominous sign, since hospital mortality rate was 87%. Cause of death was mainly HF. In most of these cases there was ECG evidence of infarction involving both anterior and inferior ventricular walls. Infarction of most or all of the ventricular septum was a common finding in the cases examined anatomically. Histologically, acute changes involving mainly the posterior septal and midseptal fibres were observed in 6 of the 8 cases studied. (5;).

1. **Elizari MV, Acunzo RS, Ferreiro M. Hemiblocks revisited. Circulation. 2007 Mar 6;115:1154-1163**
2. **Patanè S, Marte F, Mancuso A, Di Bella G. Transient right axis deviation with left posterior hemiblock and junctional rhythm during acute myocardial infarction.Int J Cardiol. 2009 Jul 10;135(3):e69-72.**
3. **Madias JE, Knez P.Transient left posterior hemiblock during myocardial ischemia-eliciting exercise treadmill testing: a report of a case and a critical analysis of the literature. J Electrocardiol. 1999 Jan;32:57-64.**
4. **Ciuraszkiewicz K, Janion M, Sielski J, Dudek D, Gawor Z. Post-myocardial Infarction Intraventricular Conduction Defects and B-type Natriuretic Peptide Levels. Clin Cardiol. 2009 Jun;32(6):E12-7**
5. **Godat FJ, Gertsch M. Isolated left posterior fascicular block: a reliable marker for inferior myocardial infarction and associated severe coronary artery disease. Clin Cardiol. 1993;16:220-226.**
6. **Rizzon P, Rossi L, Baissus C, Demoulin JC, Di Biase MLeft posterior hemiblock in acute myocardial infarction. Br Heart J. 1975 Jul;37:711-720.**

There have been few reports of exercise induced left fascicular blocks. In order to assess its frequency and significance, a retrospective study of 8684 patients was undertaken by Marcadet et al.

24 cases (11 anterior and 13 posterior) were recessed. 19 of these patients had typical effort angina, 3 had a history of MI and 3 had bypass surgery. 20 patients developed ST-T wave abnormalities included 11 ST segment depressions. 4 patients refused coronary angiography: 3 of these patients had probable coronary artery disease (CAD) (typical effort angina, positive exercise stress tests and in 1 case, inferior wall hypofixation during myocardial scintigraphy). 20 patients underwent coronary angiography.

In 2 patients, the exercise stress test was performed under Class IC antiarrhythmic therapy; 1 had a normal coronary angiogram and the other had patient coronary bypass graft. A control exercise stress test after withdrawal of drug therapy was negative in these 2 cases. The other 18 patients had significant CAD. **The recording of left fascicular block during exercise stress testing would seem to indicate severe coronary artery narrowing** (greater than or equal to 90% in 15 cases; greater than or equal to 80% in 3 cases) and LAFB is indicative of left main coronary or proximal left anterior descending artery disease.

In this series, medical therapy did not make exercise-induced left fascicular block regress, in contrast to coronary bypass surgery and angioplasty.

The presence of LPFB in patients with CAD is an ominous ECG finding, and is associated with extensive CAD(2).

1. **Marcadet DM, Genet P, Haddad A, Assayag P, Valère PE. Significance of hemiblock of the left branch during exercise Arch Mal Coeur Vaiss. 1991 Sep;84(9):1339-44.**
2. **Papa LA, Scariato A, Gottlieb R, Duca P, Kasparian H. Coronary angiographic assessment of left posterior hemiblock. J Electrocardiol. 1983 Jul;16:297-301.**

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.

Lev disease or progressive idiopathic sclerosis of the "cardiac skeleton". With a clinical behavior similar to Lenègre disease, however, it occurs in elderly patients(our case?)

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 Left Ventricular inflow tract(LVIT)

Aortic stenosis

Aortic stenosis associated with aortic insufficiency

Supravalvar aortic stenosis

Coarctation of the aorta

Sistemic Arterial hypertension

Cardiomyopathies, myocarditis and infiltrative myocardial diseases. Chronic chagasic cardiomyopathy It is the most frequent one in Latin America.

- **Dissecting aortic aneurysm**
- **Massive calcification of the “cardiac skeleton”**
- **Interventricular septum tumor (1)**
- **Hyperpotassemia (2)**
- **Transitorily, during contrast injection in the right coronary artery and in acute pulmonary embolism (1)**
- **Left bundle branch block and right axis deviation(3).**

In view of its anatomy and the fact that it receives a dual blood supply, the posterior fascicle of the left bundle branch appears to be less vulnerable than the anterior fascicle or the right bundle. Mechanical disruption of the posterior fascicle can produce isolated left posterior fascicular block **(4).**

1. **Cola H, Hoffman R, Borrega NG, Lazzar i JO. Left posterior hemiblock related to an interventricular septum tumor. First case in the literature. Eur Heart J. 1992 Apr;13:574-575.**
2. **Bashour T, Hsu I, Gorfinkel HJ, Wickramesekaran R, Rios JC. Atrioventricular and intraventricular conduction in hyperkalemia. Am J Cardiol. 1975 Feb;35:199-203.**
3. **Cheng TO. Intermittent right axis deviation in the presence of complete left bundle branch block. Int J Cardiol. 2006 Nov 18;113(3):406-7.**
4. **Rokey R, Chahine RA.Isolated left posterior fascicular block associated with acquired ventricular septal defect. Clin Cardiol. 1984 Jun;7:364-369.**

Changing axis deviation has been reported during atrial fibrillation(AF) or atrial flutter. Changing axis deviation has been also reported during acute myocardial infarction(AMI) associated with AF too or at the end of AF during AMI.

Left bundle branch block(LBBB) is usually associated with normal or left axis deviation.

Rarely the ECG shows a LBBB with changing QRS morphology and right axis deviation. There are several possible explanations for the intermittent shift in the QRS axis to the right in the presence of complete LBBB. The most plausible explanation is the coexistence of LPFB and predivisional LBBB(1) or complete LPFB and incomplete LAFB.(divisional LBBB).

The intermittent positive aspect of the neglected lead aVR indicates an intermittent right axis deviation in the presence of complete LBBB(2).

1. **Patanè S, Marte F, Dattilo G, Sturiale M. Acute myocardial infarction and left bundle branch block with changing axis deviation. Int J Cardiol. 2009 May 4. [Epub ahead of print]**
2. **Patanè S, Marte F, Di Bella G. Atrial fibrillation with left bundle branch block and intermittent right axis deviation during acute myocardial infarction. Int J Cardiol. 2008 Jun 23;127:e1-2.**

CAUSES OF GREATER VULNERABILITY OF THE LAF IN COMPARISON TO THE 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: LPF always irrigated by the two systems of the LADA and RCA.

4) TOPOGRAPHIC. The **LPF** runs through a more protected area, with less pressure mechanic impact. 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.

LPFB ELECTROCARDIOGRAPHIC CRITERIA FRONTAL PLANE

- QRS axis between : +90° and +-180° in adults
- rS pattern in leads I and aVL
- qR pattern in III, aVF and II: q wave is always present in III and may be small or absent in II or aVF
- Notch in the descending limb of the R wave in III (middle-final notch)
- RIII > RII: QRS closer to +120° (III) than +60° (II), when closer to the latter, it would indicate an incomplete form of LPFB
- 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 duration > 40 ms
- QRS duration < 120ms if isolated (without RBBB) or≥120°
- R-peak time or intrinsicoid deflection (ID) in aVF, V5 and V6: ≥ 40 ms(1). (increased) Time of appearance of R wave apex: "R-peak time". The delayed inscription time of the intrinsecoid deflection in aVF (or V6) in absolute and relative to aVL permits the diagnosis if intrinsecond deflection in aVL exceeds 35ms(1)

1. **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.**

PRECORDIAL LEADS –HORIZONTAL PLANE

- V1 and V2: rS pattern, QS rarely.
- S wave of V2 -V3 very deep by posterior dislocation and to the right of the final forces.
- Scant progression of growth of r wave in precordial leads: dislocation to the left of the transition area.
- V5 and V6: qRs or Rs patterns.
- Increased intrinsicoid deflection of V5 and V6 (> 45 ms to 50 ms)
- Disappearance of q wave in V5 and V6 when LPFB occurs.

LPFB VECTOCARDIOGRAPHIC CRITERIA

FRONTAL PLANE

Vector of initial 10 to 20 ms heading above and to the left (near -45º) with possible delay (initial 10 to 25 ms). If associated to inferior myocardial infarction, superior initial forces of 25 ms or more (more than 12.5 dashes above the orthogonal X lead. 1 dash = 2 ms).

Broad QRS loop, with clockwise rotation. Cooksey, Dunn and Massie wrote that occasionally, it may be in “eight” with a counterclockwise terminal portion (10%).

Maximal vector near +110º (+80º to +140º)

Almost all the loop is located below the X line (0 to ±1800) in the inferior quadrants

20% of the loop located in the right inferior quadrant. If there is association to CRBBB, 40% or more

Afferent limb heading below and slightly to the left, and the efferent one to the right.

Middle-terminal portion of the QRS loop (vector of 60 ms to 100 ms) with delay. It may possibly reach the right superior quadrant

QRS loop duration up to 110 ms if in isolation. In association to Complete RBBB > 120 ms

Normal ST-T vectors in isolated LPFB: T loop with clockwise rotation, heading below and to the left. If in association to Complete RBBB: alteration secondary to ventricular repolarization.

HORIZOTAL PLANE

ISOLATED LPFB CECTORCARDIOGRAPHIC CRITERIA

Vector from the initial 10 to 20 ms heading to the front and the left or right

Frequent RS in left leads V5 and V6: Precordial transition zone dislocated to the left, similar to right ventricular enlargement and intrinsecoide deflection of left leads V5 and V6 ≥ 45ms

QRS loop very similar to RVE of type C . Deep S wave in V2 or V2 and V3

QRS loop of counterclockwise rotation

≥ 20% of the QRS loop area in HP located in the right posterior quadrant. Greater area of QRS loop located in the left posterior quadrant

Maximal vector of QRS around -60° to -110°

Final portions of QRS loop with delay (60 ms to 100 ms) and located in the right posterior quadrant

T loop directed to the front and the left (+60°) and clockwise rotation.