BB case report sequential ECGs

I did not tell you my own diagnosis

I do not think tracings 1 + 3 are TRUE LBBB

I rather think there is some LEFT ANTERIOR FASCICULAR BLOCK

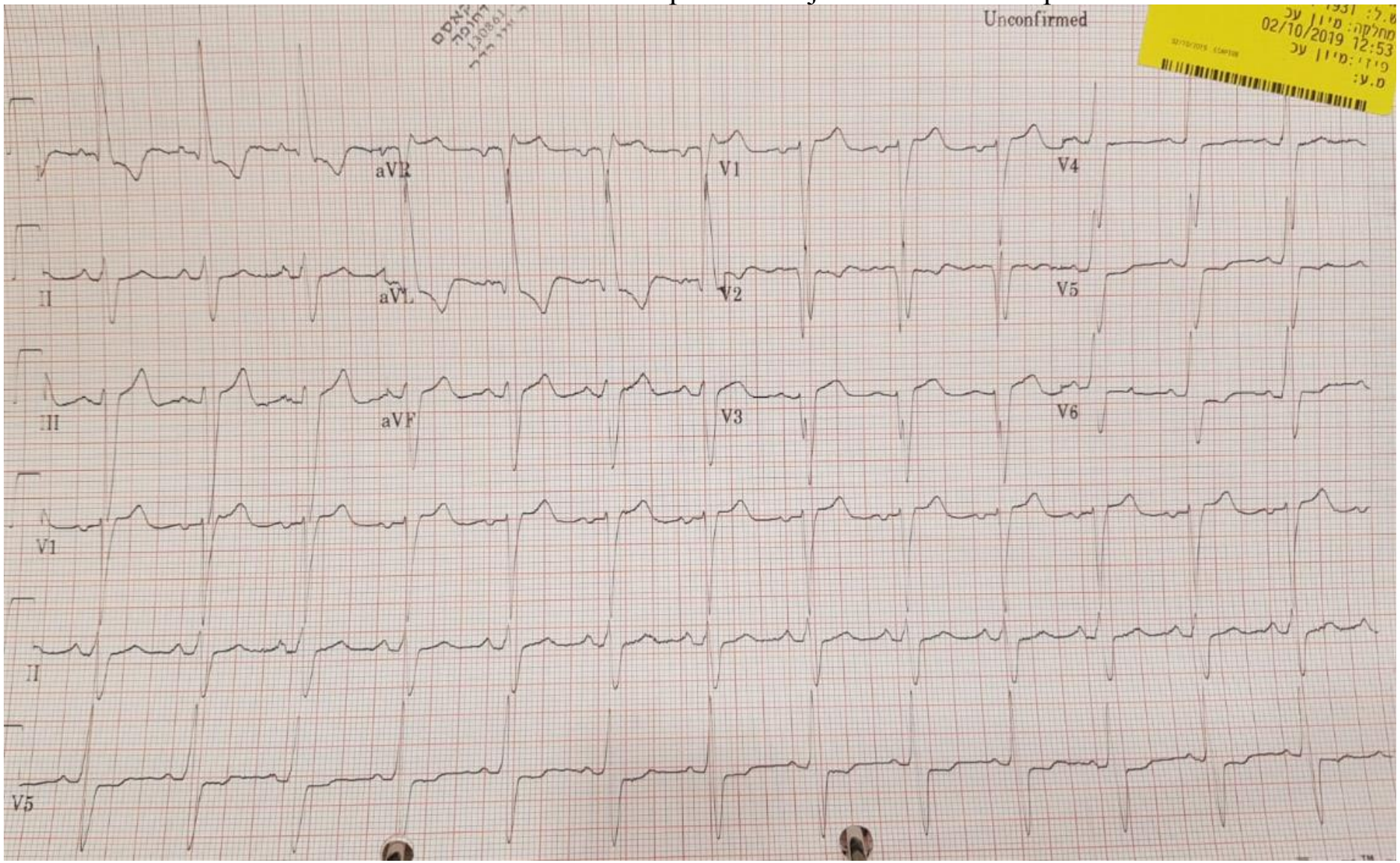
I am only wondering if we can rule out an additional LEFT SEPTAL BLOCKECG 1 with “LBBB” 2.10.19 at 14.46

ECG 2 with “RBBB” 2.10.19 at 16.53

BOTH ECG 1 and ECG 2 were performed just before PCI to proximal LAD

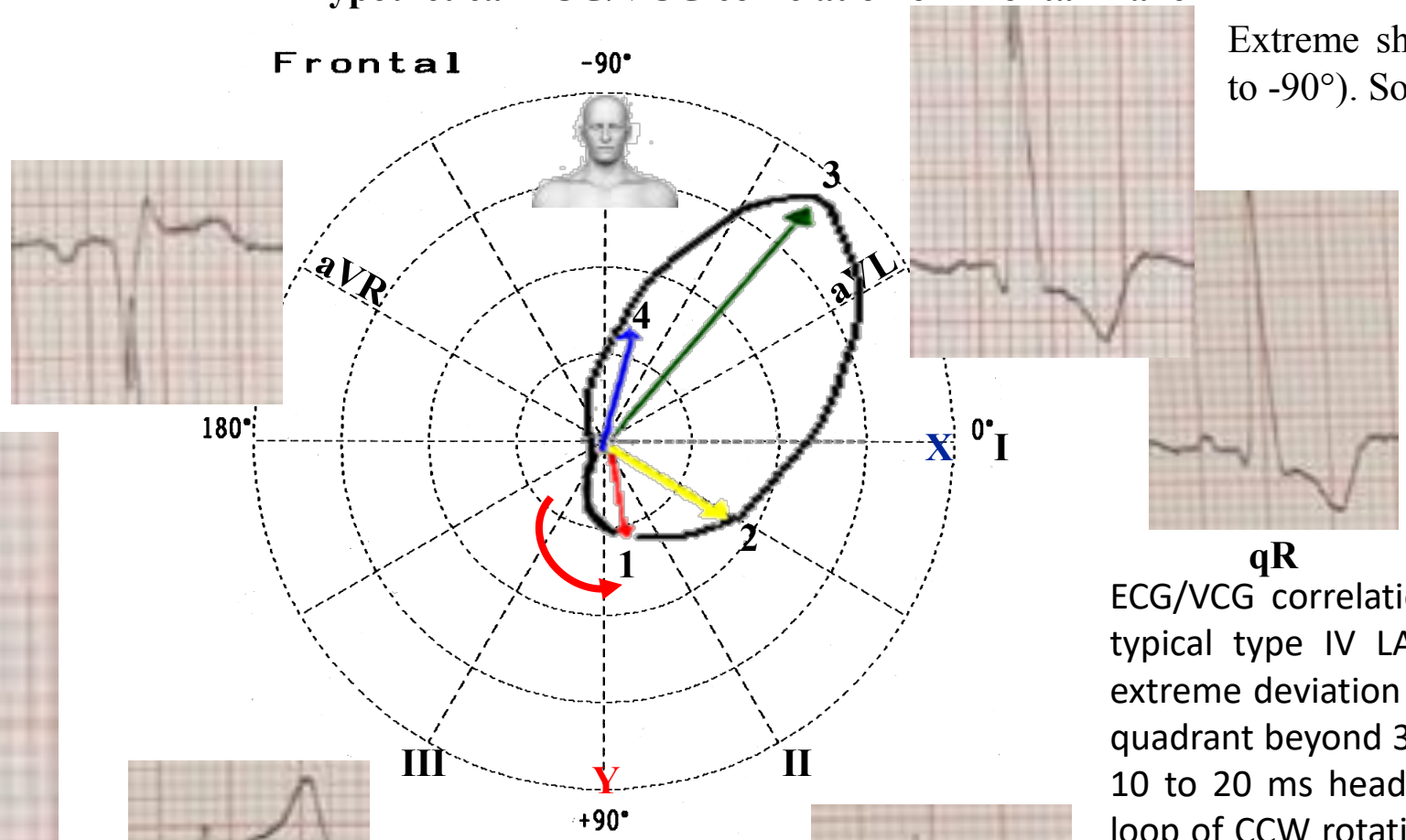
ECG 3 with “LBBB” performed this morning 4.10.19

Both ECG 1 and ECG 2 were performed just before PCI to proximal LAD



ECG diagnosis: sinus rhythm, heart rate 78bpm, P-duration 120ms, PR interval 160ms, QRS duration 150ms, QRS axis with extreme left axis deviation, rS in II, III and aVF, SIII>SII, SIII > 15mm(23mm), qR in I and aVL, strain pattern of repolarization with asymmetric negative T wave, **Conclusion:** left atrial enlargement, left ventricular hypertrophy with strain pattern of repolarization, minimal ST depression lateral wall (V5-V6), Rosembaum type IV left anterior fascicular block (SIII>15mm), anteroseptal myocardial infarction, wide fragmented QRS in V2-V3.

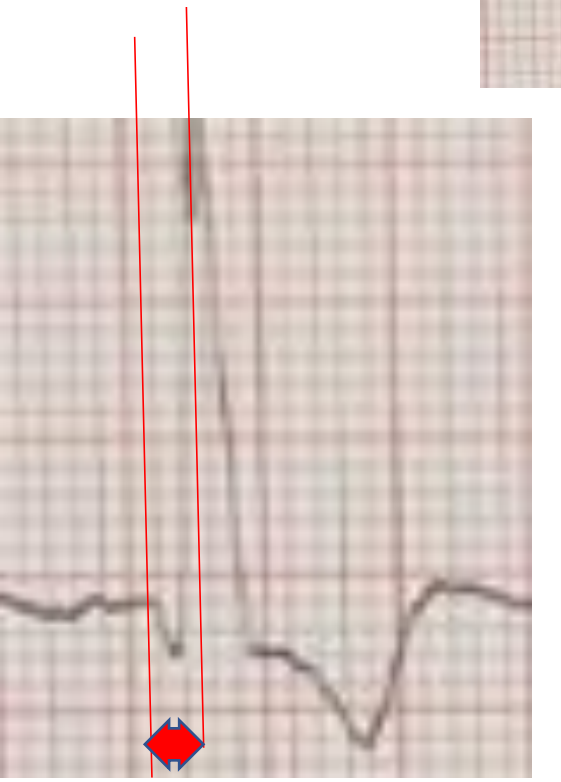
Hypothetical ECG/VCG correlation on Frontal Plane



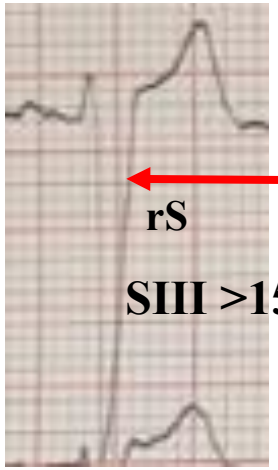
Extreme shift of ÂQRS (beyond -30° up to -90°). Some authors accept ≥45°.

qR

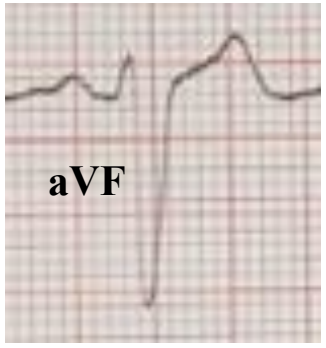
ECG/VCG correlation in the typical type IV LAFB. The following stand out: extreme deviation of QRS axis in the left superior quadrant beyond 30º (or -45º); vector from initial 10 to 20 ms heading below and rightward; QRS loop of CCW rotation; rS pattern in inferior leads; SIII > SII; SIII > 15mm; qR pattern in I and aVL and R-Wave Peak Time or intrinsicoid deflection in aVL ≥ 45 ms;

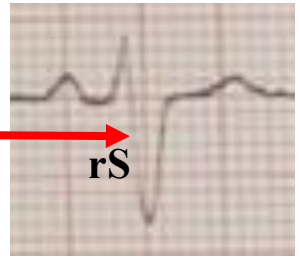


R-Wave Peak Time, Ventricular activation time or intrinsicoid deflection in aVL ≥ 45 ms;

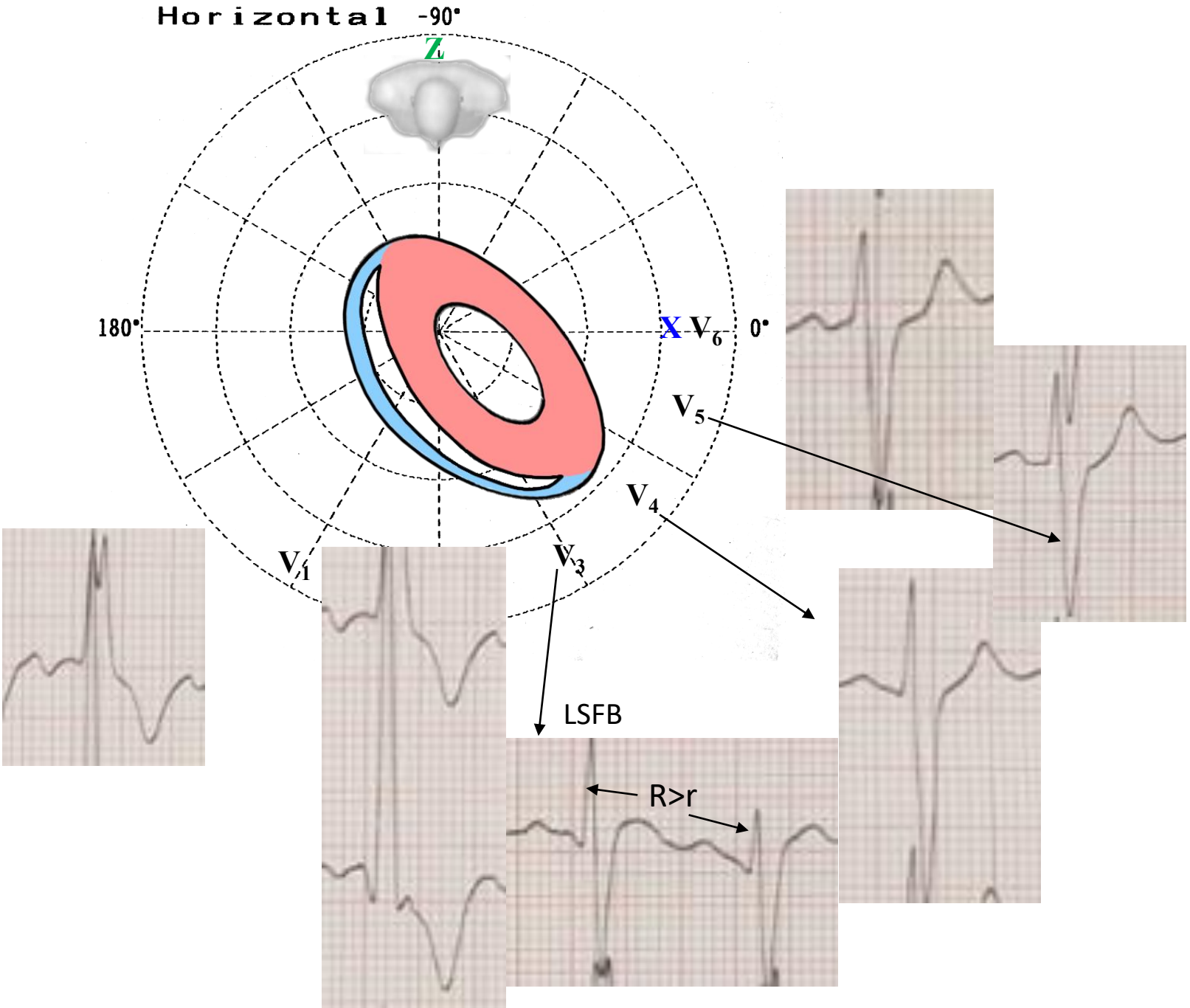


SIII > SII





Acute Anterior MI consequence of proximal LAD occlusion before S_1 complicated with RBBB.



Left atrial enlargement, complete right bundle branch block, initial embryonic q wave in anteroseptal wall,(from V1 to V3, R wave voltage of V1 ≥ than 5 mm; very high R wave voltage in V2 > 15 mm (37mm!!); the R wave voltage of the first beat of V3 > the second beat (transient left septal fascicular block)

R>r 10mm verses 4mm in V3): Intermittent prominent anterior QRS forces (PAF) during hyperacute phase of myocardial infarction (only in the first beat) ; absence of q wave in left precordial leads V_5, V_6(by absence of vector 1_{AM}).

LSFB pattern with a prolonged QRSDuration indicates the presence of additional conduction disturbances such as other fascicular blocks, such as LAFB, RBBB, MI, focal block, or a combination of these.

Conclusion

1) LAE+ LVH+ LAFB+ LSFB(transient): see R wave in V3 with and without LSFB+ CRBBB+ Acute anterior MI

The electrocardiographic LAFB types of Rosenbaum

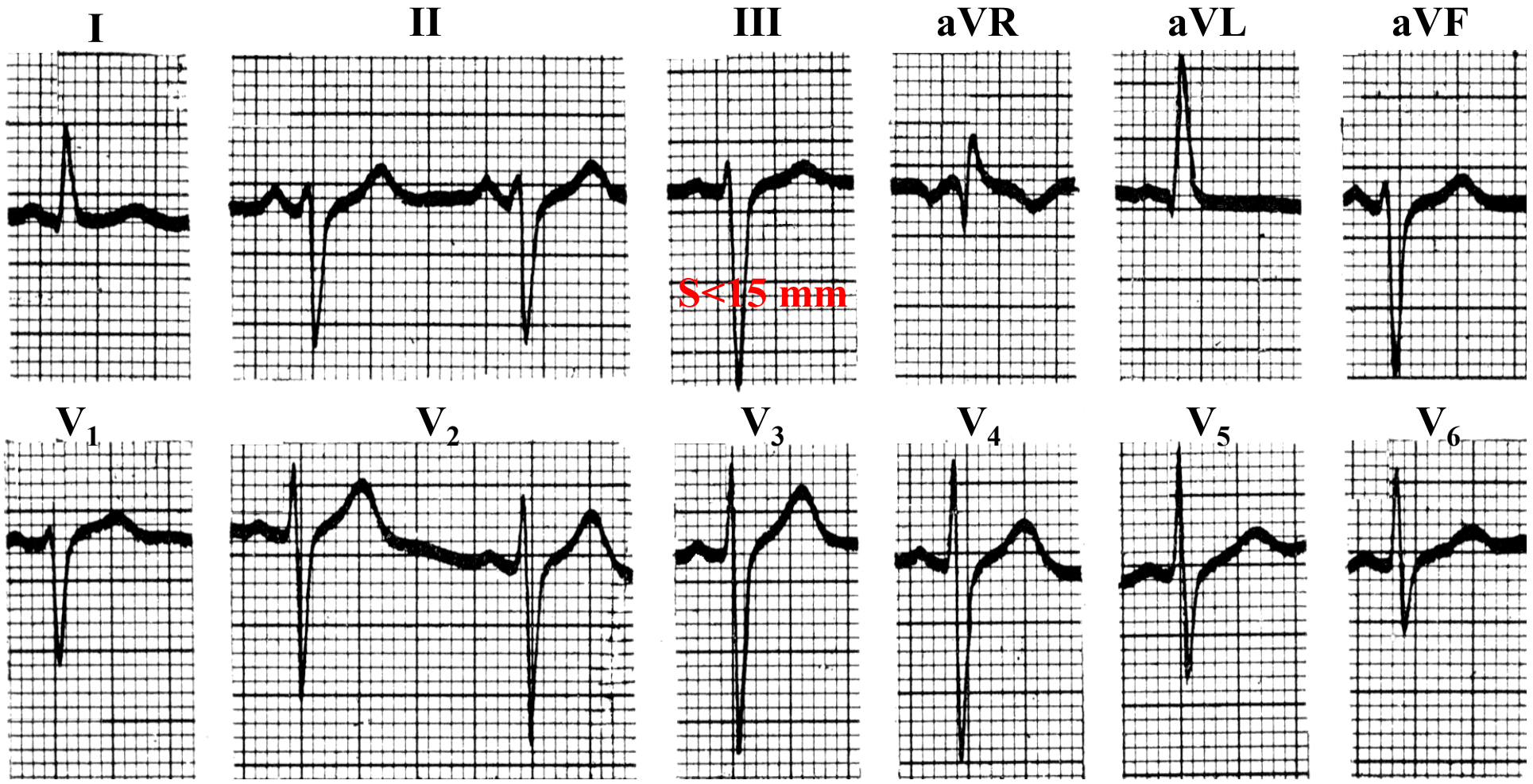
- LAFB TYPE I OR “STANDARD”.

- LAFB TYPE II: horizontal heart with clockwise rotation.

- LAFB TYPE III: ectomorph, vertical heart.

- LAFB TYPE IV: association of LAFB + LVH.

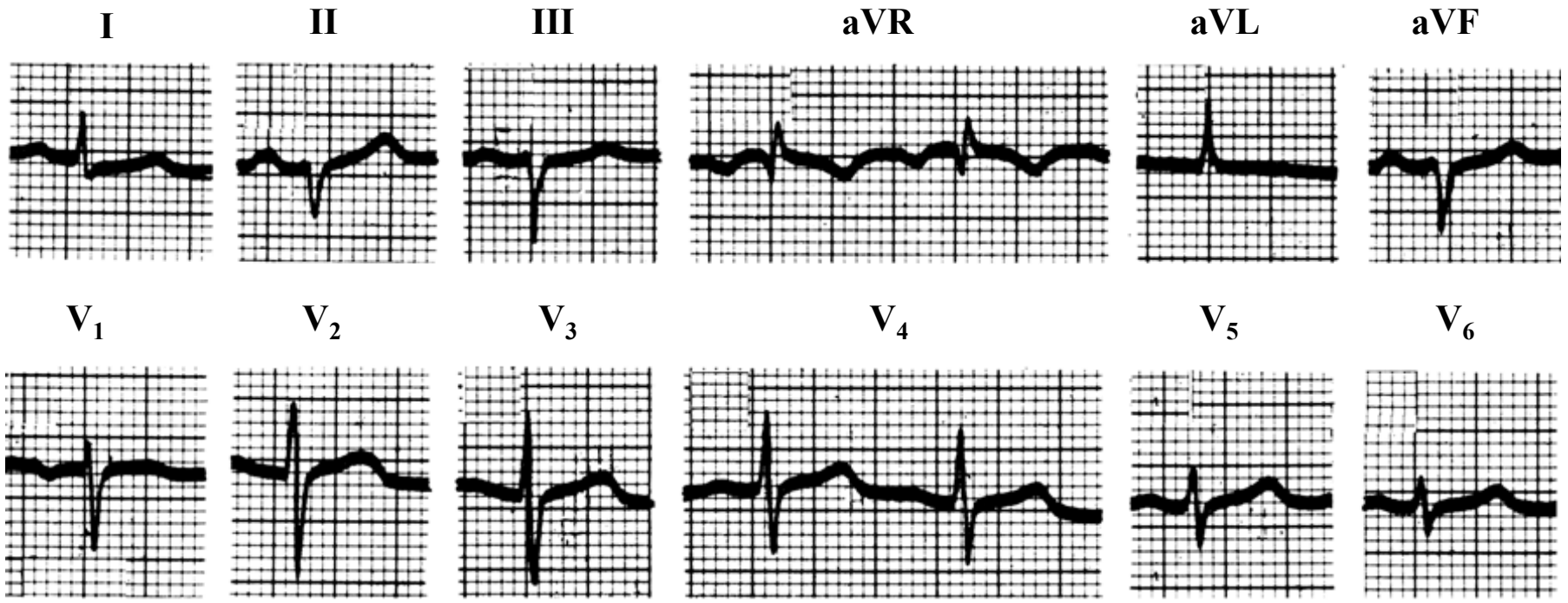
LAFB type I or “standard”



This variety was called Type I or Standard by Rosenbaum.

SÂQRS near –60°; q wave without s wave in I and no r’ complex in II. S wave of III <15 mm. Tendency to isodiphasism in aVR. This variety of LAFB is the most frequent (50% of the cases).

LAFB type II: Horizontal heart with clockwise rotation

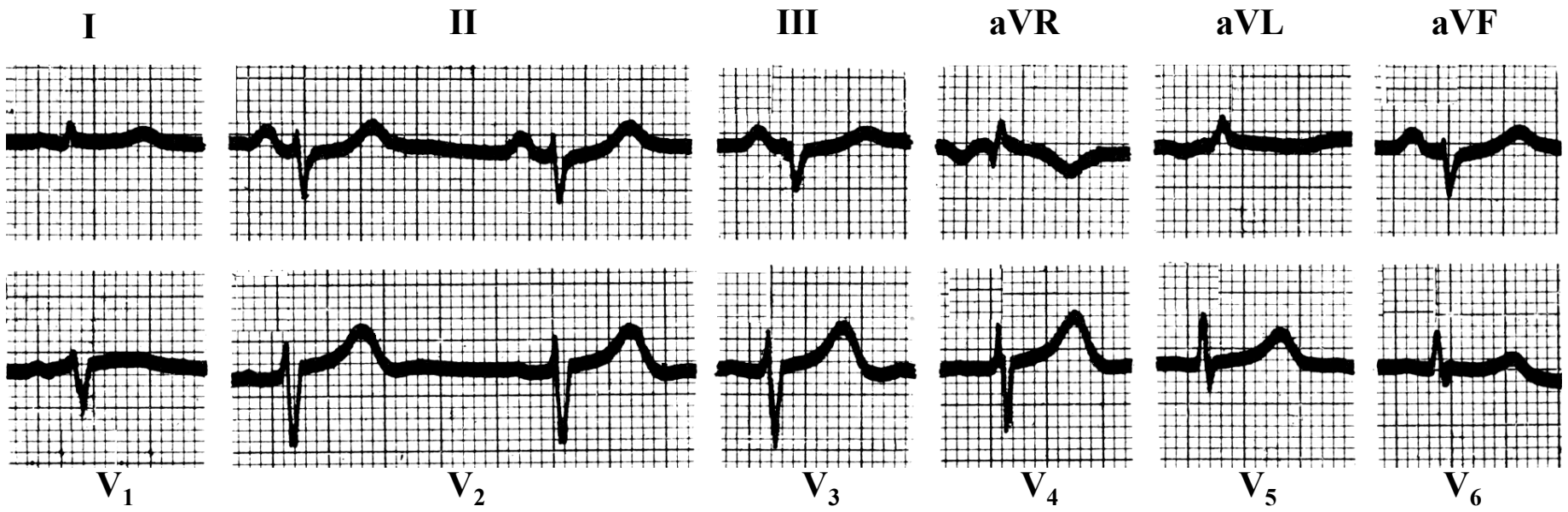


Observed in pregnant women, and obese and endomorphic people.

SÂQRS: around –60º; Voltage of QRS in the FP reduced by the posterior orientation of the final vectors; III: rSr'; I: Rs of low voltage. RS from V2 to V6.

Typical ECG of LAFB Rosenbaum type II (**Rosenbaum 1973**) of horizontal hearts with clockwise rotation, observed in the presence of rounded stomach: obese, pregnant women and endomorphic people.

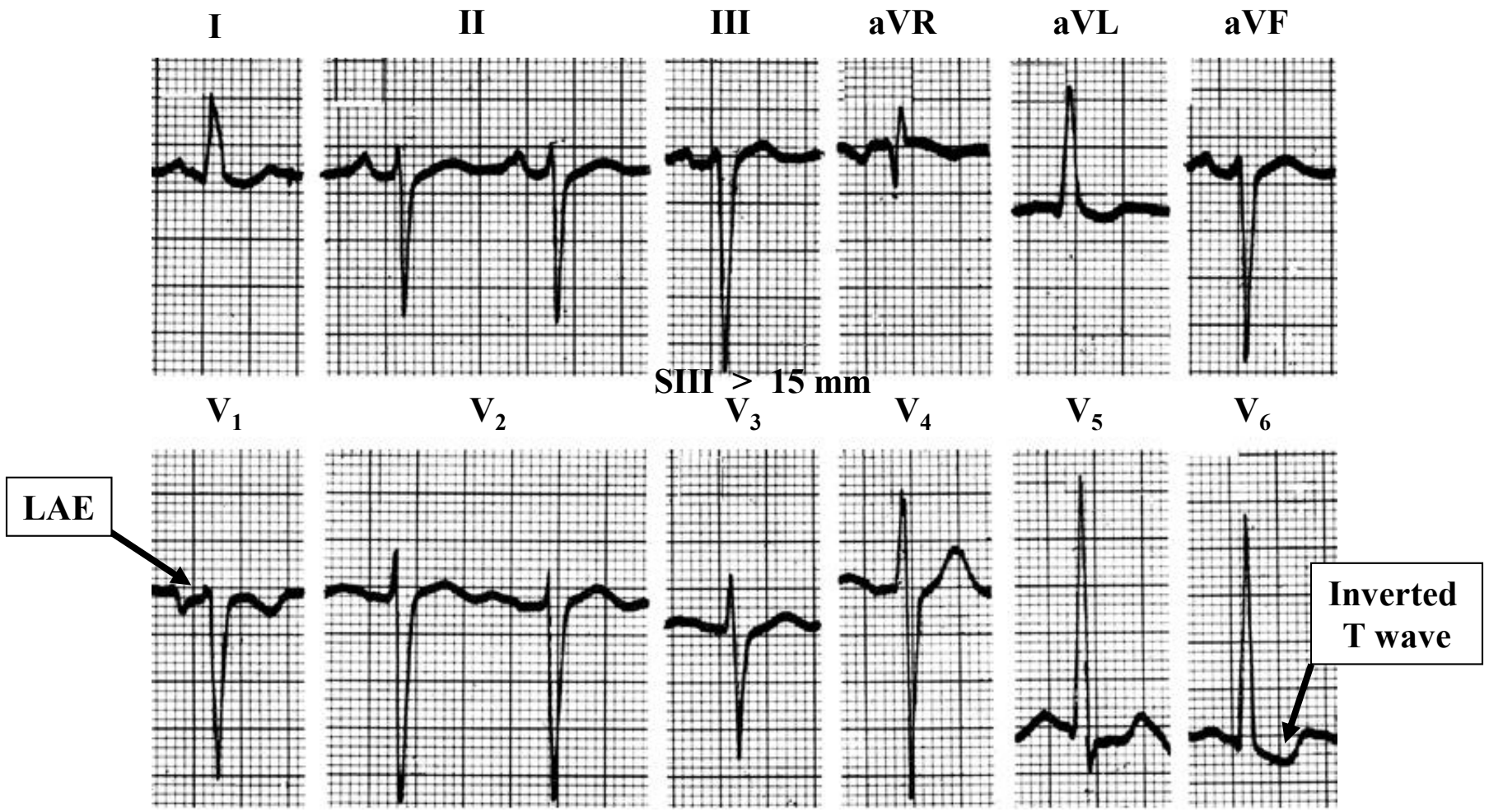
LAFB type III: Ectomorphic vertical heart



Observed in COPD.

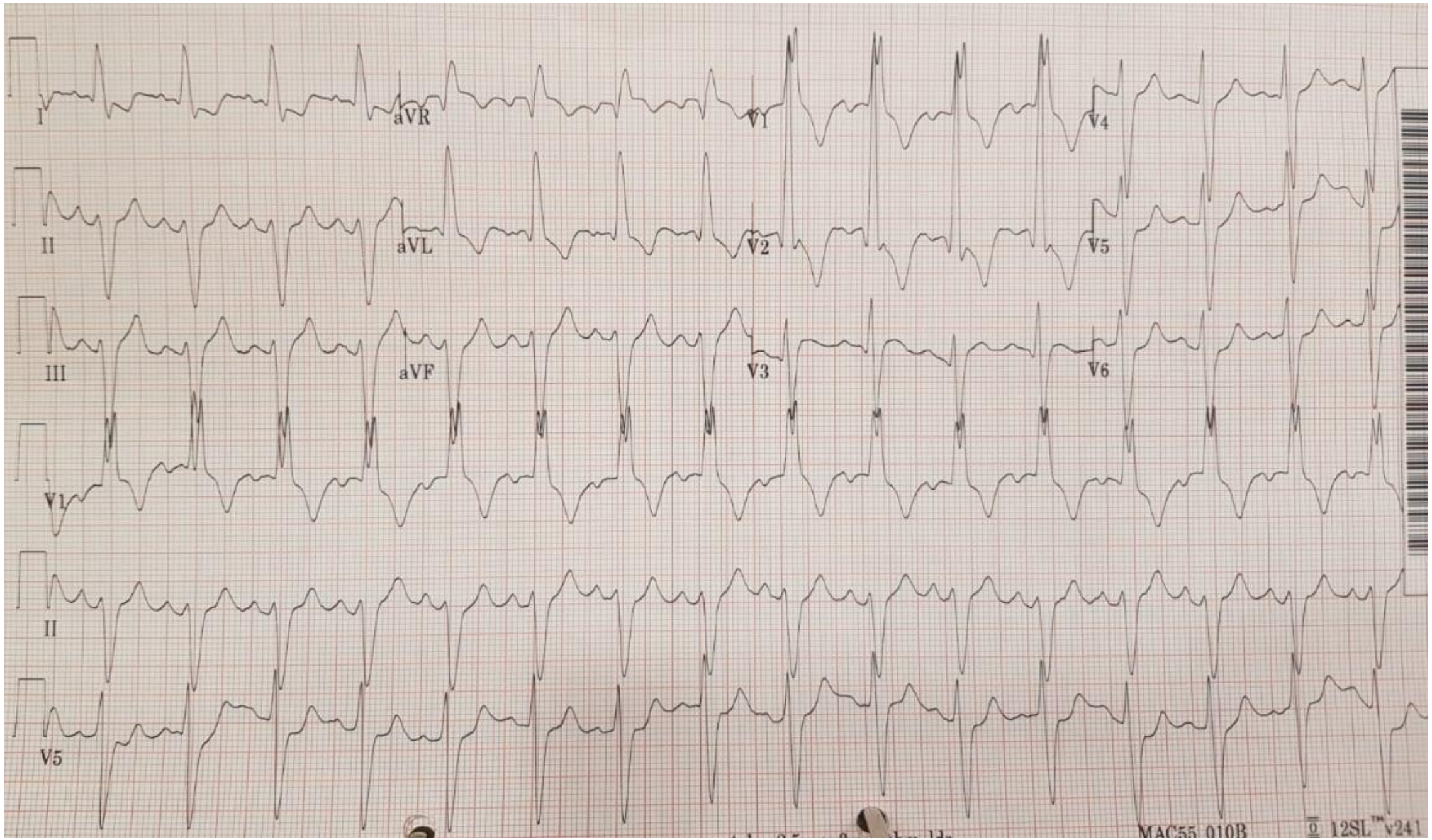
Possible presence of “P pulmonale” with SÂP to the right of +75º, SÂQRS: beyond –60º near –90º; I: R wave of low voltage; aVR predominantly positive (axis of QRS to the right of –60º).

LAFB type IV: Association of LAFB + LVH



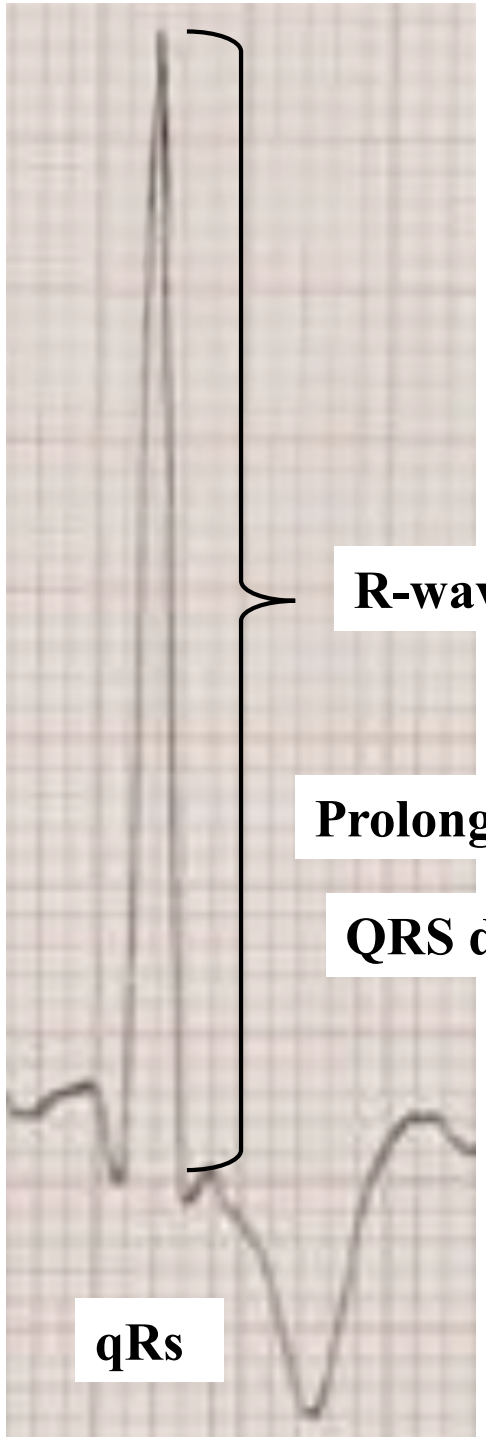
Frequent presence of LAE, S III > 15 mm, inverted T wave in one or more of the left leads: I, aVL, V_5 and V_6. Voltage of R wave in I and increased S wave of II and S of III.

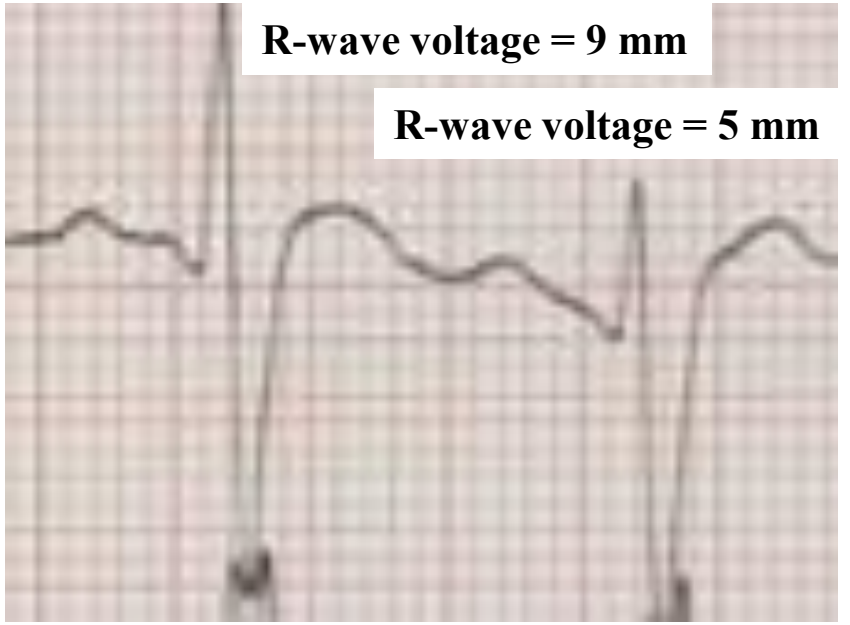
ECG 2 performed just before PCI to proximal LAD



ECG-2

As far as the slight change in QRS in V3 (tracing 2) that you attributed to possible LEFT SEPTAL BLOCK.. Don't you think it may be merely due to some minimal move of the V3 electrode.. there are strictly no changes in the QRS recorded in the closely placed electrodes. In addition it is obvious that the isoelectric line is not stable. Finally the amplitude of R wave in the first 3 complexes in V3 is different.





R-wave voltage 1st beat is 80% > 2nd beat. I do not think this could be an artefact.

What condition could give a voltage of R in V2 of 38 mm?

The QRS duration is very wide due to the association of RBBB + LSFB + LAFB + anterior MI.

There is no isolated right bundle branch block with this huge R voltage. Therefore my diagnosis is association with LSFB.

Additionally, qR pattern in V2 is typical of LSFB.

Do you have different arguments from these? Please let me know…

Possible causes for Prominent QRS Anterior Forces (PAF)

1. Normal subjects: PAF are observed in only 1% of normal subjects.
   Normal variant with marked counterclockwise rotation of the heart around the longitudinal axis of the heart resulting in a shifting of the transition area (R=S) early, i.e. to the right of the precordial lead V_2.
   Athlete's heart.
2. Misplaced precordial leads.
3. Ancient strictly posterior, dorsal, high posterobasal MI. Actual lateral MI;
4. Right ventricular hypertrophy (RVH): vectorcardiographic types A and B;
5. Diastolic LVH, volumetric or eccentric LVH, secondary to septal hypertrophy (magnitude of increase of 1_{AM} vector) and CCW heart rotation around the longitudinal axis;
6. Combined or biventricular hypertrophy;
7. CRBBB;
8. Pre-excitation variant of Wolff-Parkinson-White type A;
9. HCM: both obstructive and non-obstructive forms;
10. Progressive muscular dystrophy of childhood, Duchenne's cardiomyopathy, Duchenne's muscular dystrophy, X-linked muscular dystrophy, pseudo-hypertrophic muscular dystrophy, childhood muscular dystrophy;
11. Endomyocardial fibrosis;
12. Dextroposition. Example: left pneumonectomy.
13. LSFB;
14. A combination of the above.

Note: None of these 14 causes except LSFB can show the R pattern in V2.

The normal amplitudes of R waves in lead V_2 are:

Normal amplitudes of R wave in lead V_2 (mm)

Age	Mean in women	Mean in men	Range in women	Range in men
20-30	7.4	4.6	1.7-13.9	1.1-9.2
30-40	5.4	3.7	0.6-12.1	0.4-10.1
40-60	4.6	3.6	0.6-12.0	0.2-9.1

From 20 to 30 years old, R wave > 13.9 mm in women and > 9.2 mm in men is considered a criterion for PAF.
From 30 to 40 years old, R wave > 12.1 mm in women and > 10.1 mm in men is considered a criterion for PAF.
From 40 to 60 years old, R wave > 12.0 mm in women and > 9.1 mm in men is considered a criterion for PAF.

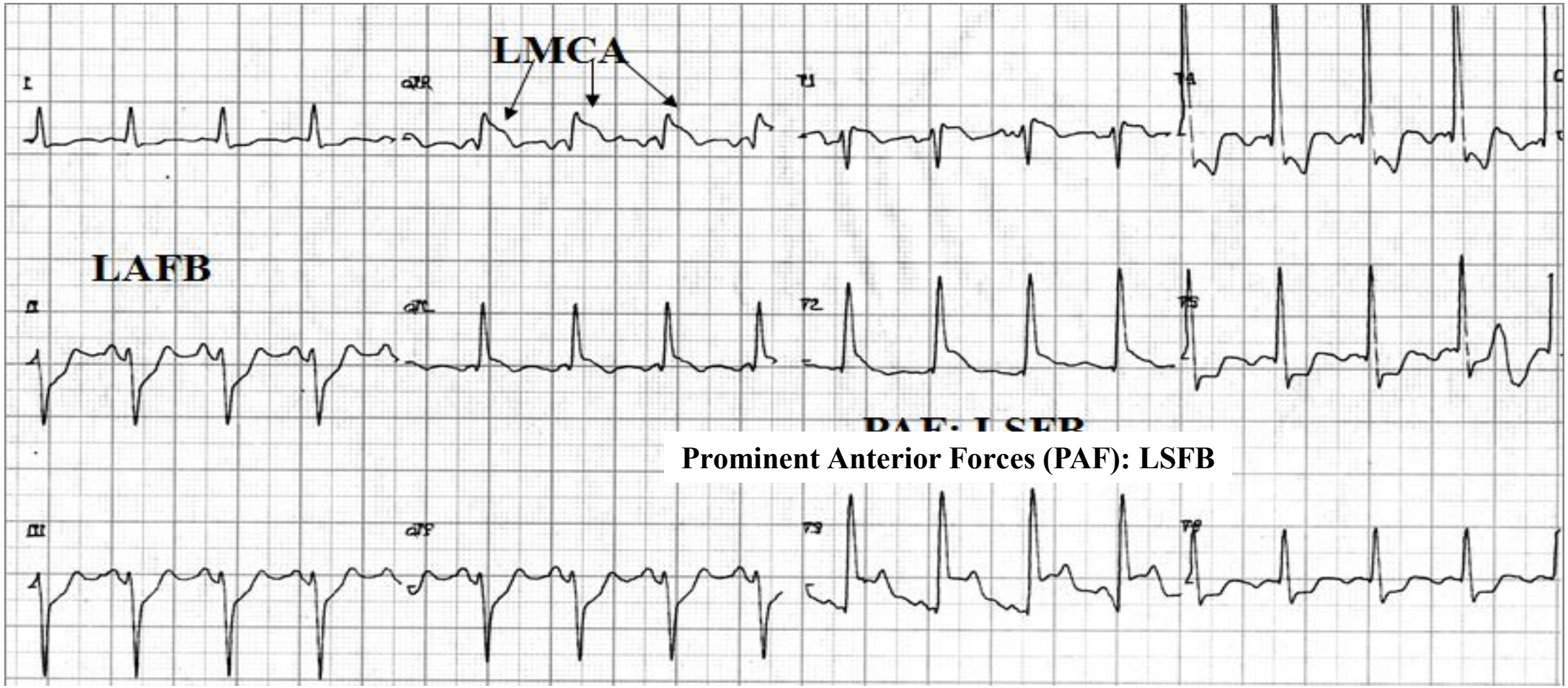
Electrocardiographic characterization of LSFB

- Normal QRS duration or with a minor increase (up to 110 ms). When associated with other fascicular or bundle blocks it could be ≥ 120 ms.
- FP leads with no modifications: normal QRS, if not associated with LAFB.
- Increased ventricular activation time or intrinsic deflection V1 and V2: ≥ 35 ms.
- R wave voltage of V1 ≥ than 5 mm;
- R/S ratio in V1 > 2;
- R/S ratio in V2 > 2;
- S wave depth in V1 < 5 mm;
- Possible small (embryonic) q wave in V2 and V3 or V1 and V2;
- R wave of V2 > 15 mm;
- RS or Rs pattern in V2 and V3 (frequent rS in V1) with R wave "in crescendo" from V1 through V3 and decreasing from V5 to V6;
- Absence of q wave in left precordial leads V_5, V_6 and I (by absence of vector 1_{AM}). One first needs to exclude ILBBB, CLBBB and WPW;
- Intermittent PAF during hyperacute phase of myocardial infarction**,** or during an exercise stress test in patients with severe myocardial ischemia and during early atrial extrastimuli with some degree of ventricular aberration
- Appearance of intermittent, rate-dependent q wave in V_1 and V_2**.**
- The last Brazilian Guidelines for Interpreting Rest Electrocardiogram provided the following criteria for ECG diagnosis of LSFB:
- QRS duration < 120 ms, in general, close to 100 ms. The appearance of LSFB does not increase QRSD by more than 25 ms, due to multiple interconnections between the fascicles of the LBB ("passageway zone" of Rosenbaum). The QRS complex is slightly prolonged between 100 ms to 115 ms. Thus, LSFB pattern with a prolonged QRSD indicates the presence of additional conduction disturbances such as other fascicular blocks, RBBB, MI, focal block, or a combination of these;
- ≥15 mm voltage R waves in V2 and V3 or from V1;
- Increasing for all intermediary precordial leads and decreasing from V5 to V6;
- "r" wave jump may occur from V1 to V2 ("rS" in V1 for R in V2);
- Absence of QRS axis shift on frontal plane;
- T wave polarity most of the times, negative in right precordial leads.

Note: all these criteria are valid in absence of RVH, septal hypertrophy or posterior-wall MI and other causes of PAF.

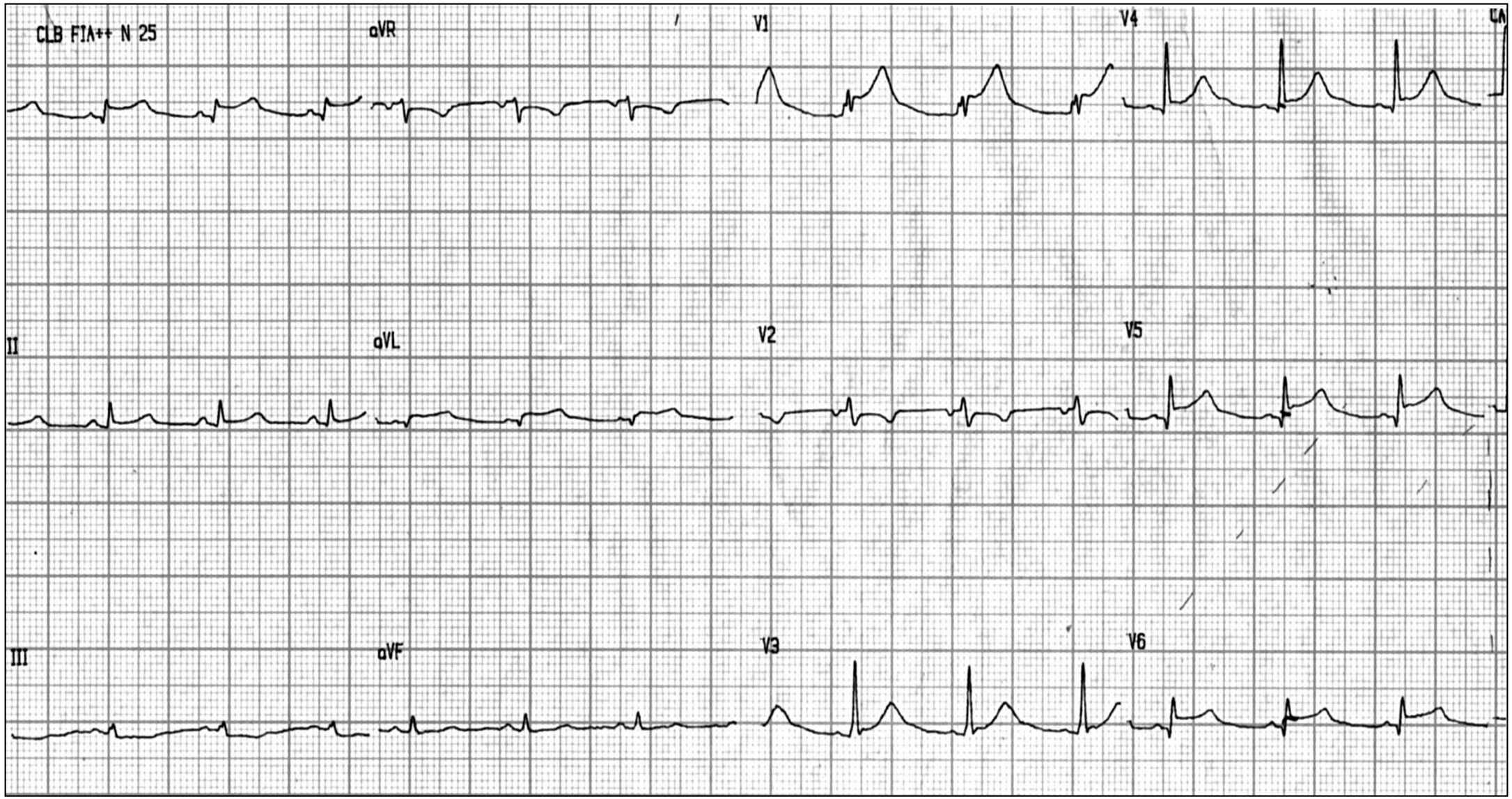
Other examples: A transient form of LSFB was observed in an acute coronary syndrome scenario in a 72-year-old male patient, admitted in the emergency room with typical precordial pain that yielded after the administration of IV nitroglycerin. Figures 16A, 16B and 16C. The coronary angiography revealed LMCA spasm + proximal critical lesion of the LAD. **Management:** The patient was urgently revascularized, successfully. (Coronary Artery Bypass Graft).

Name: AR.; **Date:** 02/01/2009.; **Age:** 72 yo; **Gender:** Male; **Ethnic Group:** Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours ago.



ECG diagnosis: 1) LAFB + 2) LSFB: PAF + Injury block + aVR lead with ST segment elevation suggestive of obstruction in the LMCA. **Laboratory:** There was no increase of necrosis markers (CK-MB/troponin).

Name: AR; **Date:** 05/01/2009; **Age:** 72 yo; **Gender:** Male; **Ethnic:** Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours ago.



Electrocardiogram conducted on the third day after successful surgery.
Both fascicular or divisional blocks have disappeared: the extreme shift of QRS electric axis to the left in the frontal plane (LAFB) is not seen, and prominent anterior forces (LSFB) have disappeared.

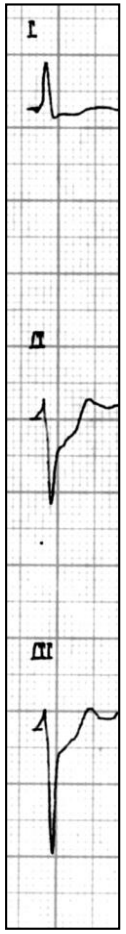
Date:
02/01/2009

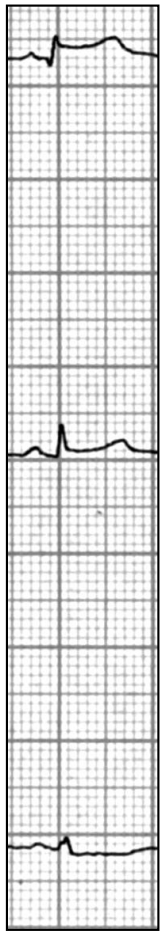
Date:
05/01/2009

I

II

III





With LAFB

Without LAFB

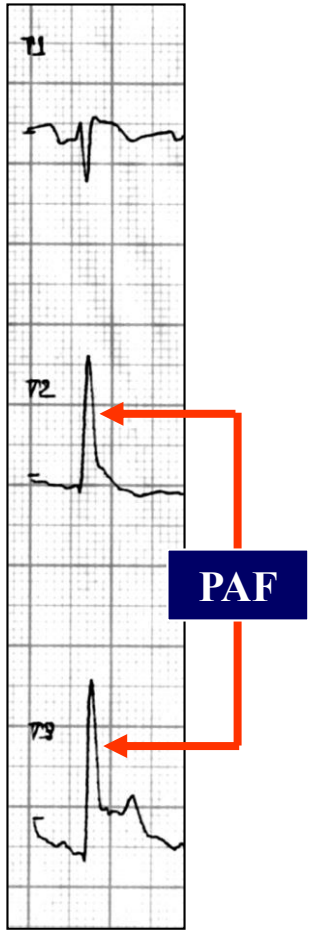
Date:
02/01/2009

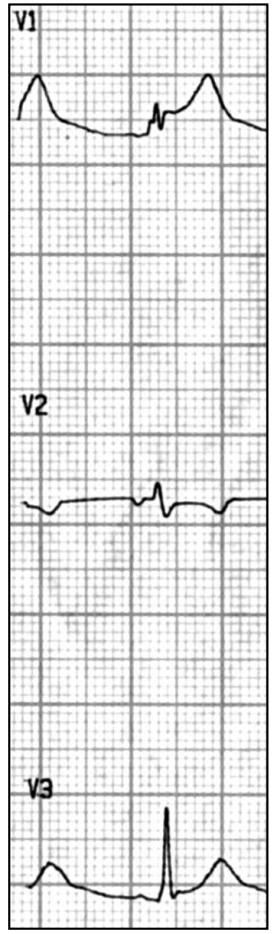
Date:
05/01/2009

V_1

V_2

V_3





With PAF: LSFB

Without PAF: LSFB

PAF: Prominent Anterior Forces

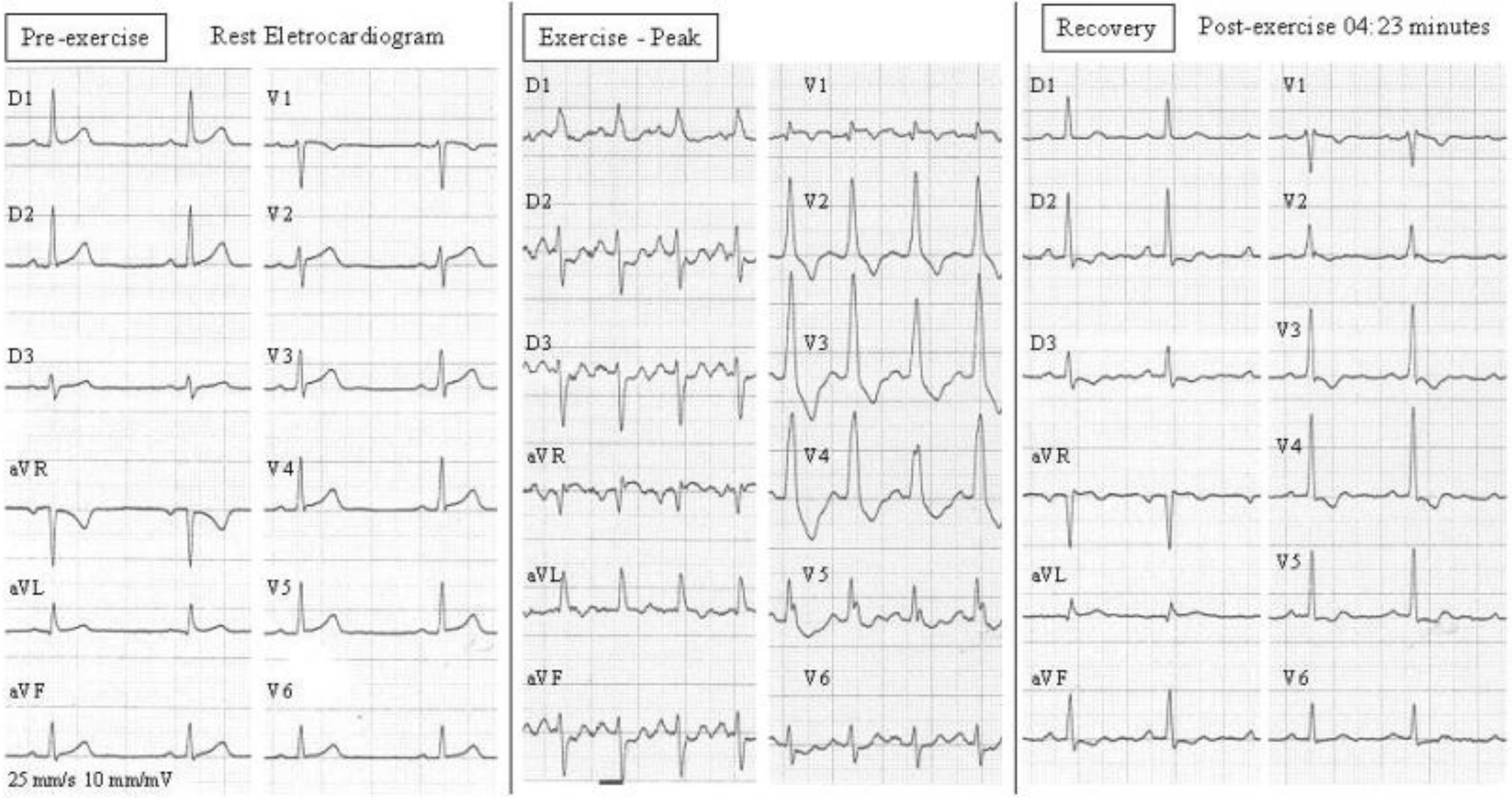
Comments about the evidence that reinforces the trifascicular nature of the Left His System

The following evidence and arguments reinforce the trifascicular theory of the left intraventricular His system, unlike the classical bifascicular concept coined in a conclusive way in the work by Rosenbaum and his associates.

The electrophysiological demonstration of the activation of the middle third of the left septal surface 5 ms before the antero-superior and postero-inferior regions, was made in 1970 by Durrer and colleagues. In Dr. Durrer's studies, the activation of the middle-septal region occurs in most cases from the anterior "false tendons" that originate from the LSF. For the authors, the LSF in its final portion opens as a fan, and the anterior pseudo-tendons are those resulting in the activation of the middle-septal region.

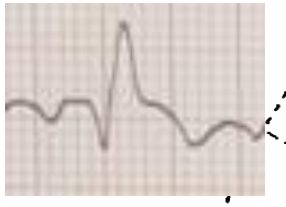
We know that one of the anatomical variations of the LSF (type 3) is precisely, the one that depends on the LPF (2.4% of cases).

Another piece of evidence of the trifascicular nature of the left Hisian system is determined by the electrical or electrovectorcardiographic recording of the anterior ventricular depolarization, sometimes intermittent or transitory, translated in ECG by R waves with great voltage in intermediary precordial leads V3 and V4. This is represented in the VCG by anterior and leftward shift of QRS loop in the HP. This may occur in association with a critical proximal lesion of LAD before S1. It also requires the absence of other factors capable of causing prominent anterior forces (PAF), such as dorsal or posterior wall infarction, RVH, cardiomyopathies, RBBB, WPW and others. In some patients studied angiographically, coronary disease was isolated to the LAD and ventricular dysfunction confined to the LV anterior wall. PAF were observed intermittently together with LAFB. These observations, in addition to serial studies following surgery, strongly suggest that the mechanism for PAF in these cases is conduction delay in the LSF. We present a 54-year-old male, with a history of systemic hypertension, hyperlipidemia and typical chest pain during exercise. During the exercise stress test, ECG demonstrated abrupt prominent anterior forces, an increase in R wave amplitude from V1 to V4, extreme left axis deviation and minor ST segment depression in II, III and aVF. The post-exercise period showed progressive return of the QRS axis in both frontal and horizontal planes and the ST depression worsened by 1 mm. Coronary angiogram showed a critical proximal LAD critical occlusion.

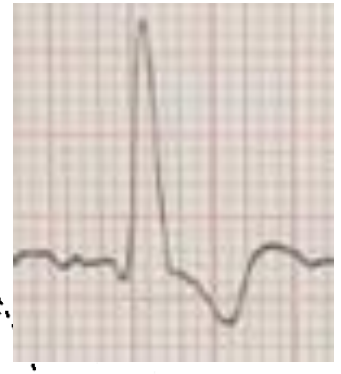


During peak of exercise we observe tall R waves in V1-V4. It is a hallmark ECG finding in LSFB. The proposed ECG criteria for LSFB are: prominent R waves in V1-V3, minimal QRS prolongation (QRS < 120 ms), T wave morphologic alteration (flat or inversion), frequent initial embryonic q wave in right and/or middle precordial leads, absence of initial q wave in left leads consequence of absence of 1_{AM} vector and clinical absence of other causes of prominent anterior forces. During exercise peak we also observe LAFB, consequently we have left bifascicular block (LAFB + LSFB).

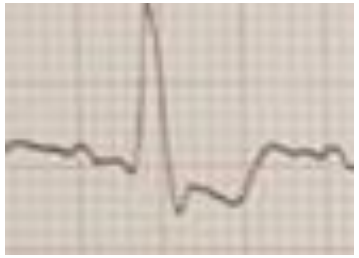
The ST injury vector pointing to up and rightward aVR: proximal LAD or LMCA obstruction



Discrete ST segment elevation



ST segment depression in I and minimal aVL



Limitations of the ST injury vector and the location of myocardial ischemia

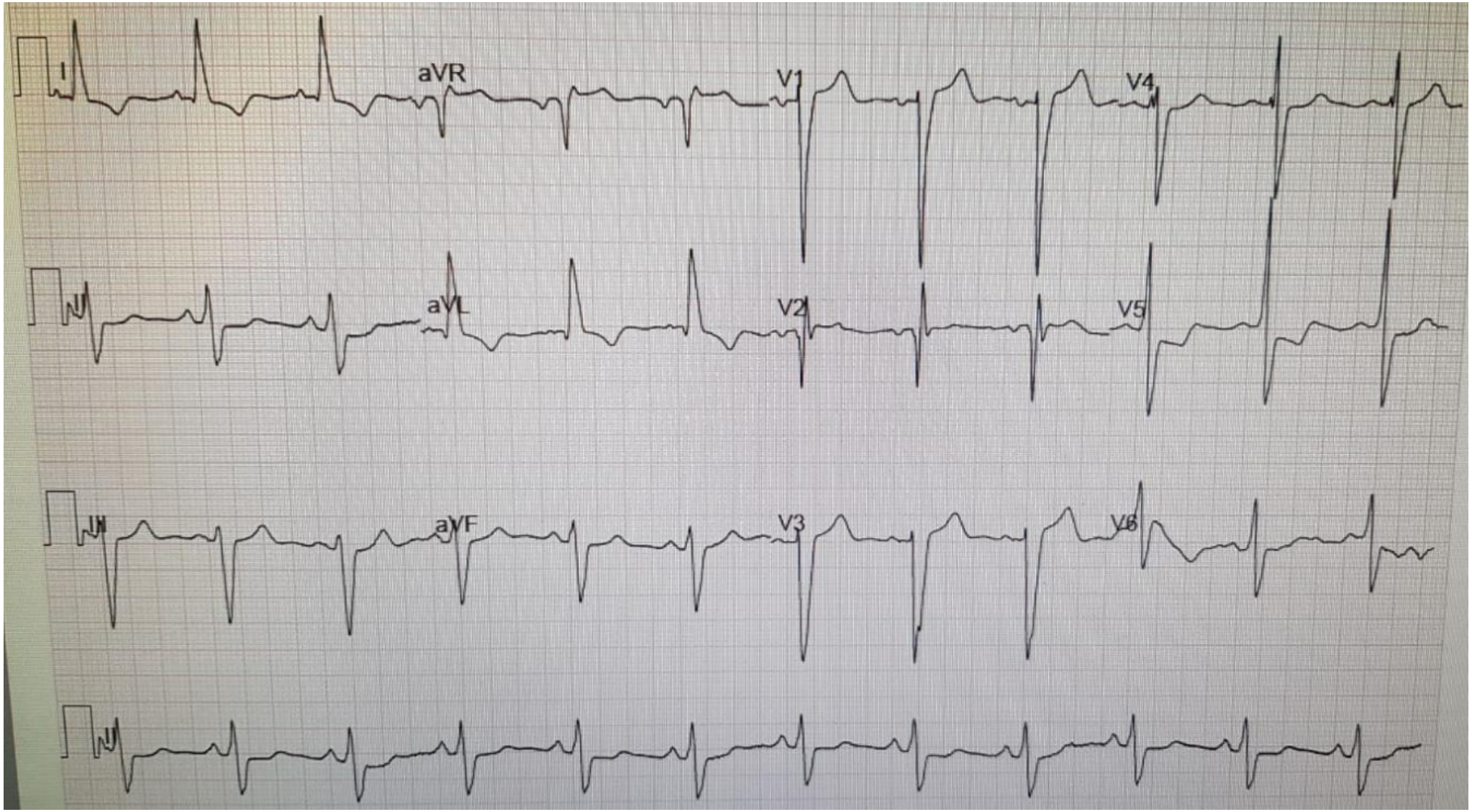
Specificity: high[1]
Predictive accuracy: high
Sensitivity: quite low

Clinical situations where the deviation of the ST segment is limited

1. Presence of a previous infarction
2. Preexisting abnormalities of the ST segment
3. Left Bundle Brach Block/Right Bundle Branch Block isolated or associated with fascicular block
4. Ventricular Preexcitation
5. Multivessel disease
6. Abnormal site of origin of a coronary artery
7. Dominance or underdevelopment of the coronary arteries.

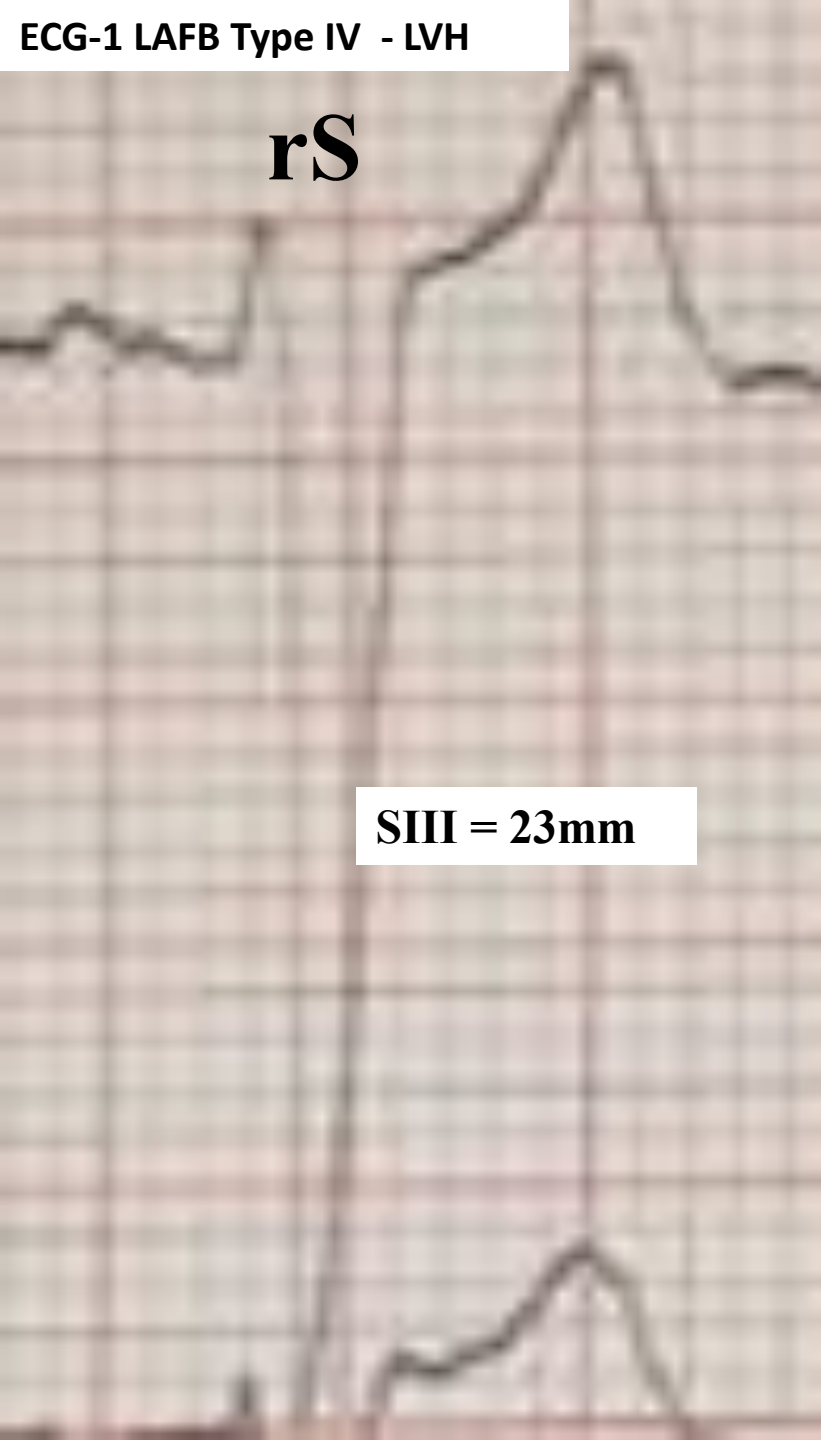
1. **Andersen MP, Terkelsen CJ, Sørensen JT, Kaltoft AK, Nielsen SS, Struijk JJ, The ST injury vector: electrocardiogram-based estimation of location and extent of myocardial ischemia. J Electrocardiol. 2010 Mar-Apr;43:121-131.**

ECG 3 performed this morning 4.10.19 after PCI?

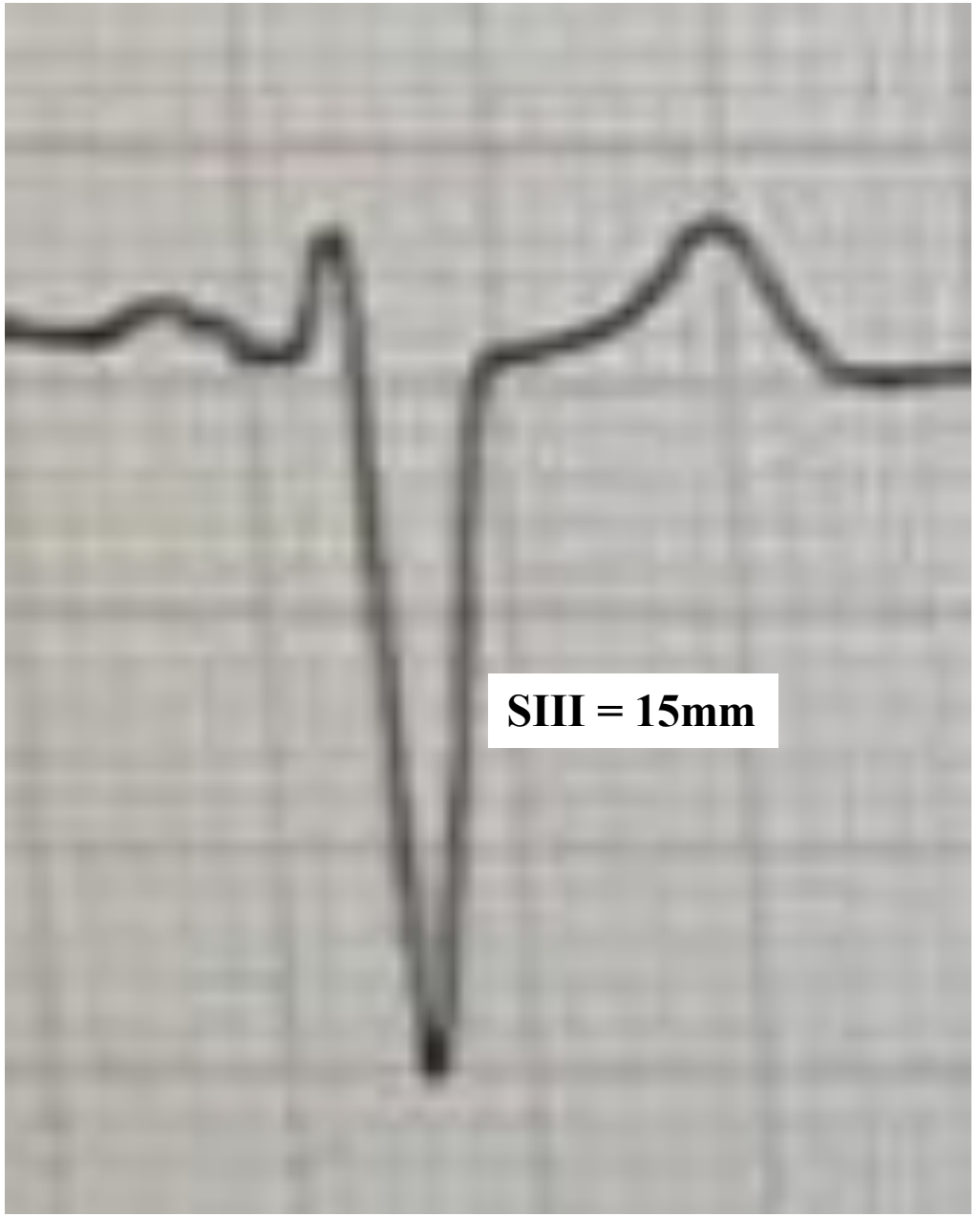


ECG diagnosis: LAE + LVH with strain pattern, LAFB Rosenbaum type I or “standard” (SIII ≤15mm) sequela septal MI in V2 Qrs) lateral ST segment depression

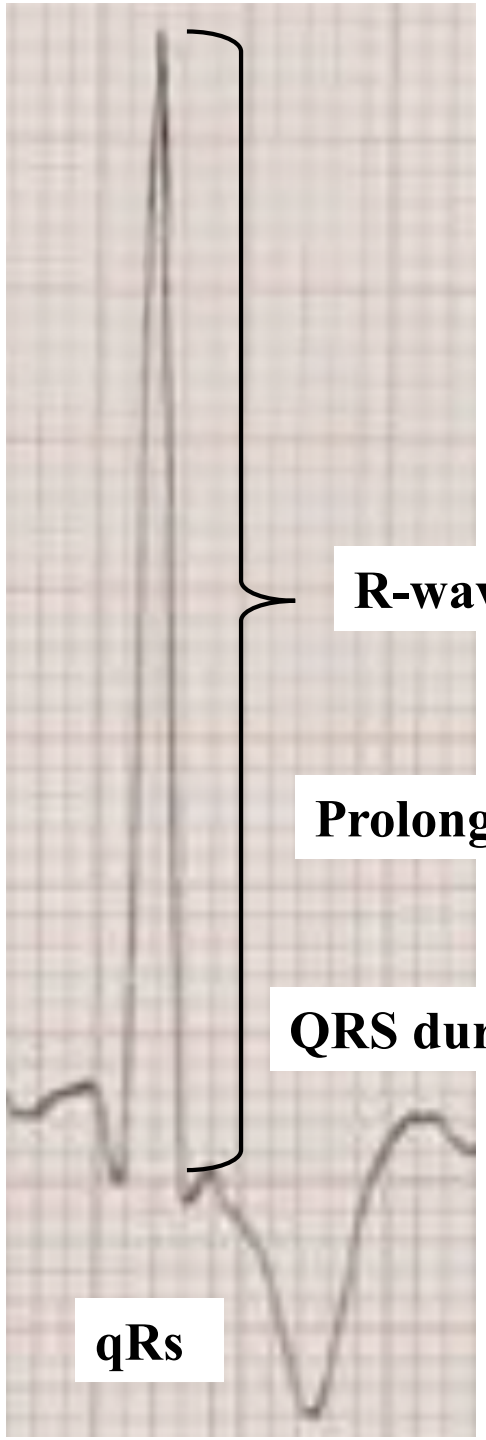
ECG-1 LAFB Type IV - LVH



ECG-3 LAFB Rosenbaum type I or “standard”



ECG-2



What condition could give a voltage of R in V2 of 38 mm?
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There is no isolated right bundle branch block with this huge R voltage. Therefore my diagnosis is association with LSFB.
Additionally, qR pattern in V2 is typical of LSFB.
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   Normal variant with marked counterclockwise rotation of the heart around the longitudinal axis of the heart resulting in a shifting of the transition area (R=S) early, i.e. to the right of the precordial lead V_2.
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7. CRBBB;
8. Pre-excitation variant of Wolff-Parkinson-White type A;
9. HCM: both obstructive and non-obstructive forms;
10. Progressive muscular dystrophy of childhood, Duchenne's cardiomyopathy, Duchenne's muscular dystrophy, X-linked muscular dystrophy, pseudo-hypertrophic muscular dystrophy, childhood muscular dystrophy;
11. Endomyocardial fibrosis;
12. Dextroposition. Example: left pneumonectomy.
13. LSFB;
14. A combination of the above.

Note: None of these 14 causes except LSFB can show the R pattern in V2.

The normal amplitudes of R waves in lead V_2 are:

Normal amplitudes of R wave in lead V_2 (mm)

Age	Mean in women	Mean in men	Range in women	Range in men
20-30	7.4	4.6	1.7-13.9	1.1-9.2
30-40	5.4	3.7	0.6-12.1	0.4-10.1
40-60	4.6	3.6	0.6-12.0	0.2-9.1

From 20 to 30 years old, R wave > 13.9 mm in women and > 9.2 mm in men is considered a criterion for PAF.
From 30 to 40 years old, R wave > 12.1 mm in women and > 10.1 mm in men is considered a criterion for PAF.
From 40 to 60 years old, R wave > 12.0 mm in women and > 9.1 mm in men is considered a criterion for PAF.

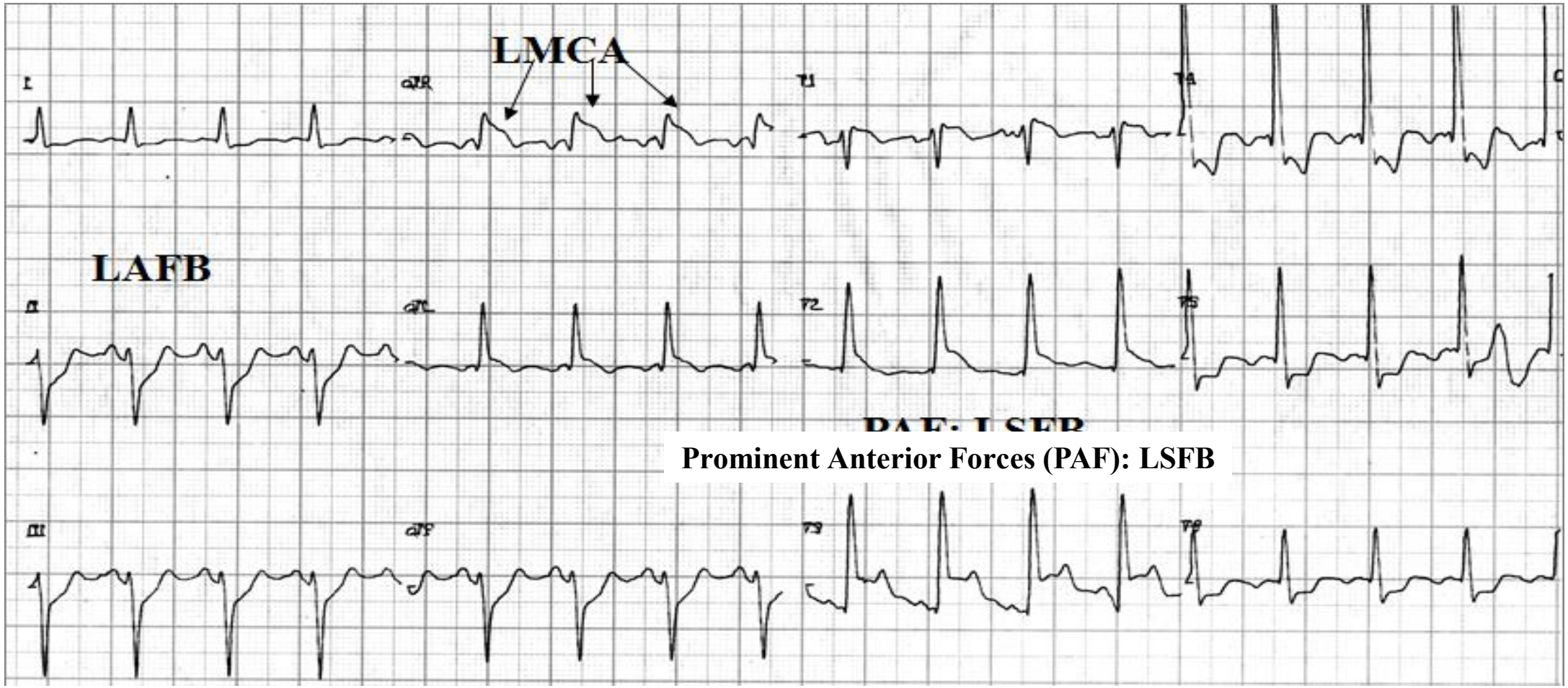
Electrocardiographic characterization of LSFB

- Normal QRS duration or with a minor increase (up to 110 ms). When associated with other fascicular or bundle blocks it could be ≥ 120 ms.
- FP leads with no modifications: normal QRS, if not associated with LAFB.
- Increased ventricular activation time or intrinsic deflection V1 and V2: ≥ 35 ms.
- R wave voltage of V1 ≥ than 5 mm;
- R/S ratio in V1 > 2;
- R/S ratio in V2 > 2;
- S wave depth in V1 < 5 mm;
- Possible small (embryonic) q wave in V2 and V3 or V1 and V2;
- R wave of V2 > 15 mm;
- RS or Rs pattern in V2 and V3 (frequent rS in V1) with R wave "in crescendo" from V1 through V3 and decreasing from V5 to V6;
- Absence of q wave in left precordial leads V_5, V_6 and I (by absence of vector 1_{AM}). One first needs to exclude ILBBB, CLBBB and WPW;
- Intermittent PAF during hyperacute phase of myocardial infarction**,** or during an exercise stress test in patients with severe myocardial ischemia and during early atrial extrastimuli with some degree of ventricular aberration
- Appearance of intermittent, rate-dependent q wave in V_1 and V_2**.**
- The last Brazilian Guidelines for Interpreting Rest Electrocardiogram provided the following criteria for ECG diagnosis of LSFB:
- QRS duration < 120 ms, in general, close to 100 ms. The appearance of LSFB does not increase QRSD by more than 25 ms, due to multiple interconnections between the fascicles of the LBB ("passageway zone" of Rosenbaum). The QRS complex is slightly prolonged between 100 ms to 115 ms. Thus, LSFB pattern with a prolonged QRSD indicates the presence of additional conduction disturbances such as other fascicular blocks, RBBB, MI, focal block, or a combination of these;
- ≥15 mm voltage R waves in V2 and V3 or from V1;
- Increasing for all intermediary precordial leads and decreasing from V5 to V6;
- "r" wave jump may occur from V1 to V2 ("rS" in V1 for R in V2);
- Absence of QRS axis shift on frontal plane;
- T wave polarity most of the times, negative in right precordial leads.

Note: all these criteria are valid in absence of RVH, septal hypertrophy or posterior-wall MI and other causes of PAF.

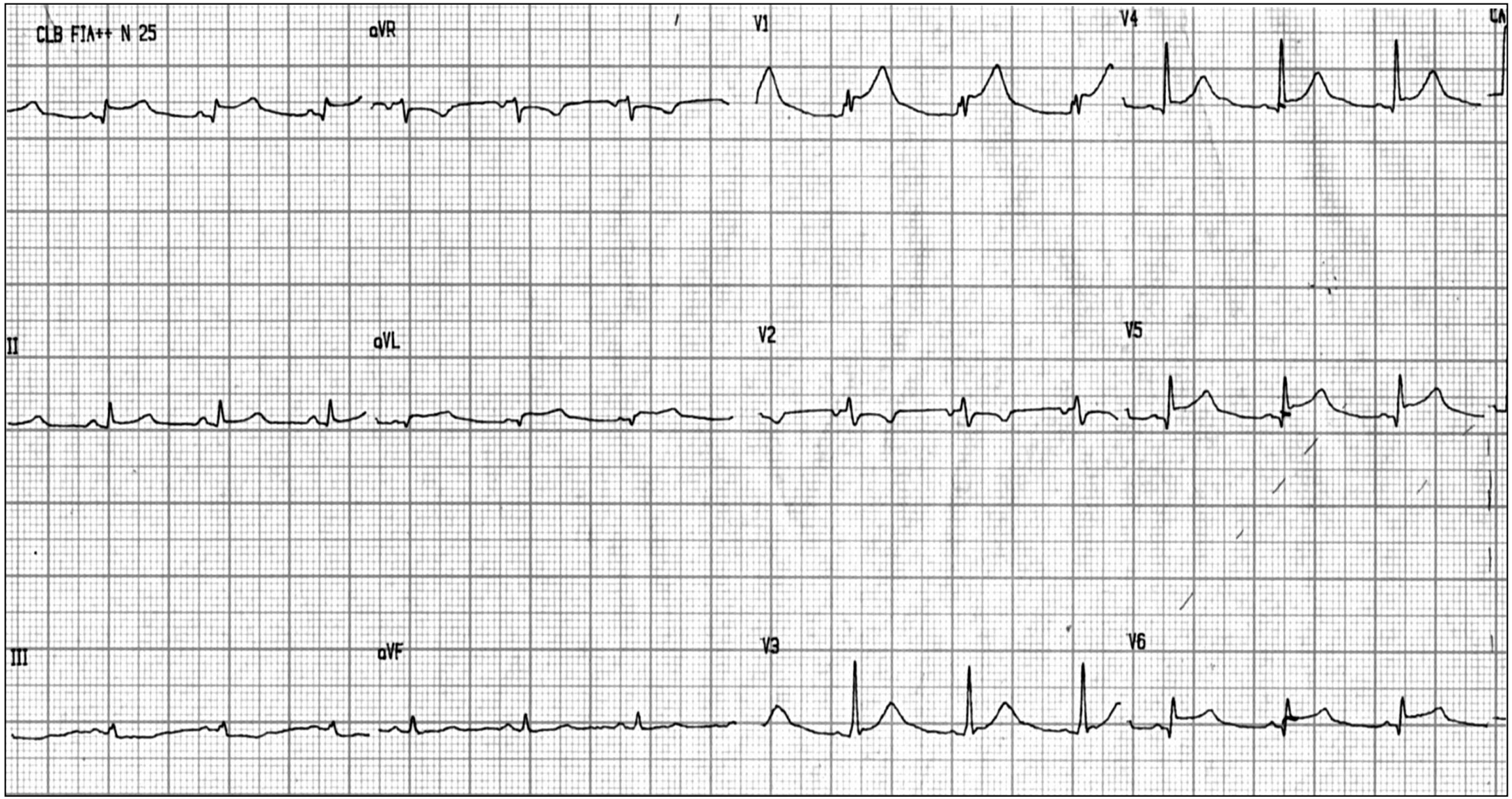
A transient form of LSFB was observed in an acute coronary syndrome scenario in a 72-year-old male patient, admitted in the emergency room with typical precordial pain that yielded after the administration of IV nitroglycerin. Figures 16A, 16B and 16C. The coronary angiography revealed LMCA spasm + proximal critical lesion of the LAD. **Management:** The patient was urgently revascularized, successfully. (Coronary Artery Bypass Graft).

Name: AR.; **Date:** 02/01/2009.; **Age:** 72 yo; **Gender:** Male; **Ethnic Group:** Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours ago.



ECG diagnosis: 1) LAFB + 2) LSFB: PAF + Injury block + aVR lead with ST segment elevation suggestive of obstruction in the LMCA. **Laboratory:** There was no increase of necrosis markers (CK-MB/troponin).

Name: AR; **Date:** 05/01/2009; **Age:** 72 yo; **Gender:** Male; **Ethnic:** Caucasian; **Weight:** 72 Kg; **Height:** 1.74 m; **Biotype:** Mesomorphic; **Management:** Coronary Artery Bypass Graft (CABG) 72 hours ago.



Electrocardiogram conducted on the third day after successful surgery.
Both fascicular or divisional blocks have disappeared: the extreme shift of QRS electric axis to the left in the frontal plane (LAFB) is not seen, and prominent anterior forces (LSFB) have disappeared.

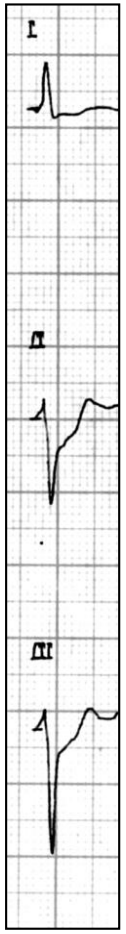
Date:
02/01/2009

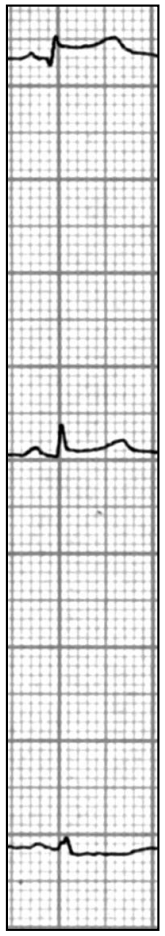
Date:
05/01/2009

I

II

III





With LAFB

Without LAFB

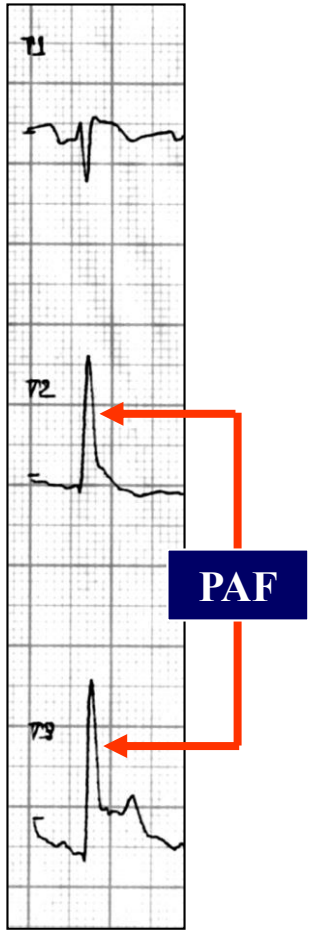
Date:
02/01/2009

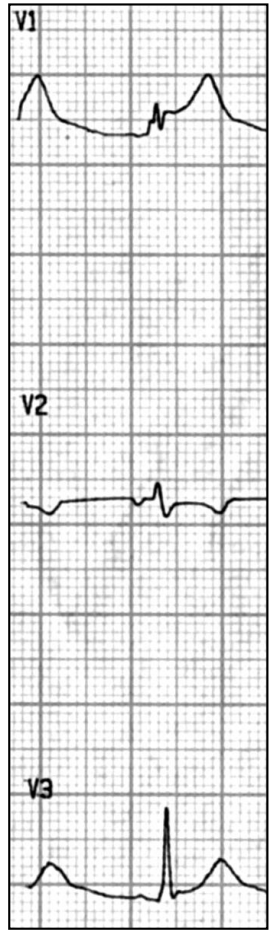
Date:
05/01/2009

V_1

V_2

V_3





With PAF: LSFB

Without PAF: LSFB

PAF: Prominent Anterior Forces

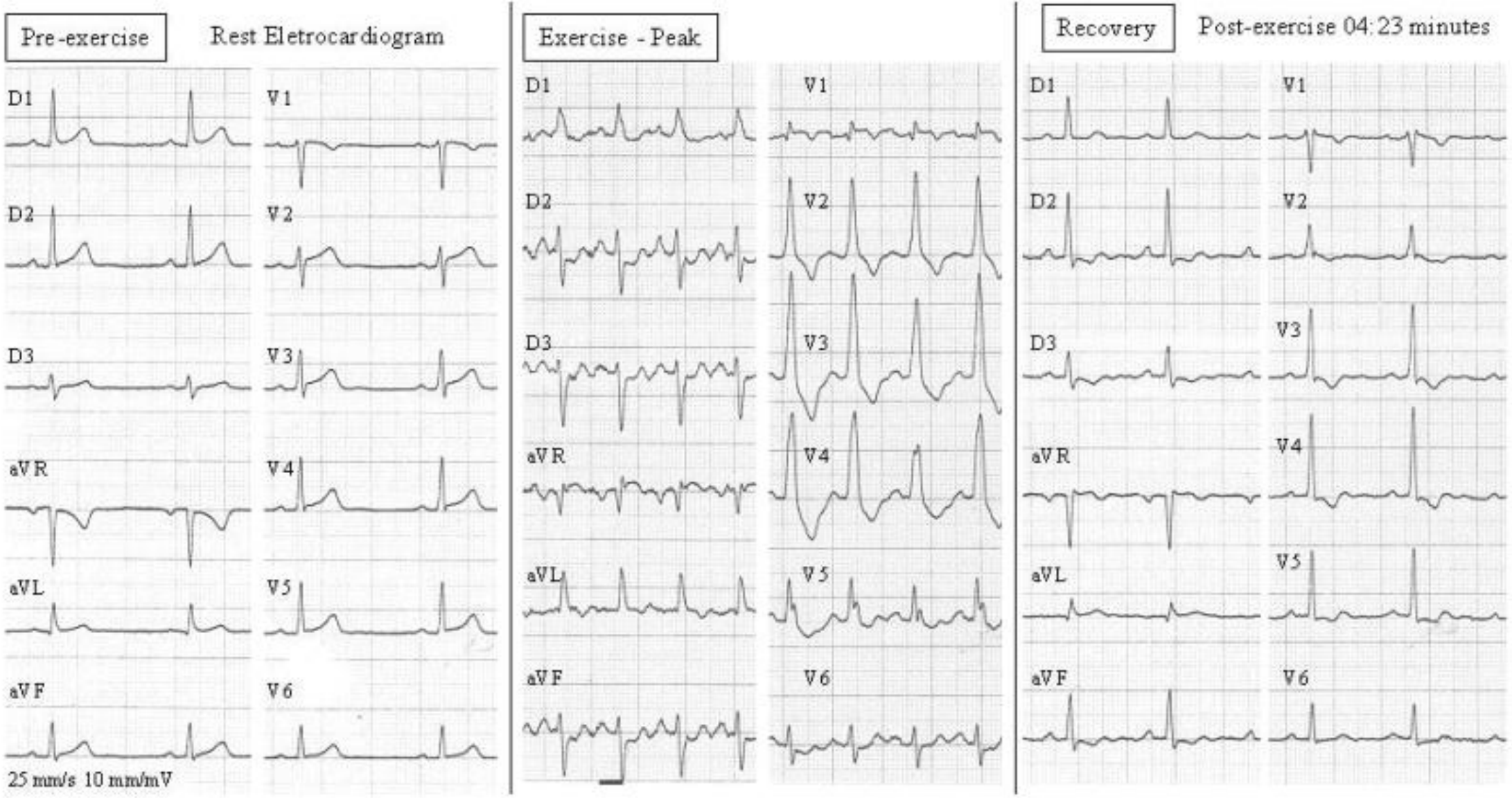
Comments about the evidence that reinforces the trifascicular nature of the Left His System

The following evidence and arguments reinforce the trifascicular theory of the left intraventricular His system, unlike the classical bifascicular concept coined in a conclusive way in the work by Rosenbaum and his associates.

The electrophysiological demonstration of the activation of the middle third of the left septal surface 5 ms before the antero-superior and postero-inferior regions, was made in 1970 by Durrer and colleagues. In Dr. Durrer's studies, the activation of the middle-septal region occurs in most cases from the anterior "false tendons" that originate from the LSF. For the authors, the LSF in its final portion opens as a fan, and the anterior pseudo-tendons are those resulting in the activation of the middle-septal region.

We know that one of the anatomical variations of the LSF (type 3) is precisely, the one that depends on the LPF (2.4% of cases).

Another piece of evidence of the trifascicular nature of the left Hisian system is determined by the electrical or electrovectorcardiographic recording of the anterior ventricular depolarization, sometimes intermittent or transitory, translated in ECG by R waves with great voltage in intermediary precordial leads V3 and V4. This is represented in the VCG by anterior and leftward shift of QRS loop in the HP. This may occur in association with a critical proximal lesion of LAD before S1. It also requires the absence of other factors capable of causing prominent anterior forces (PAF), such as dorsal or posterior wall infarction, RVH, cardiomyopathies, RBBB, WPW and others. In some patients studied angiographically, coronary disease was isolated to the LAD and ventricular dysfunction confined to the LV anterior wall. PAF were observed intermittently together with LAFB. These observations, in addition to serial studies following surgery, strongly suggest that the mechanism for PAF in these cases is conduction delay in the LSF. We present a 54-year-old male, with a history of systemic hypertension, hyperlipidemia and typical chest pain during exercise. During the exercise stress test, ECG demonstrated abrupt prominent anterior forces, an increase in R wave amplitude from V1 to V4, extreme left axis deviation and minor ST segment depression in II, III and aVF. The post-exercise period showed progressive return of the QRS axis in both frontal and horizontal planes and the ST depression worsened by 1 mm. Coronary angiogram showed a critical proximal LAD critical occlusion.



During peak of exercise we observe tall R waves in V1-V4. It is a hallmark ECG finding in LSFB. The proposed ECG criteria for LSFB are: prominent R waves in V1-V3, minimal QRS prolongation (QRS < 120 ms), T wave morphologic alteration (flat or inversion), frequent initial embryonic q wave in right and/or middle precordial leads, absence of initial q wave in left leads consequence of absence of 1_{AM} vector and clinical absence of other causes of prominent anterior forces. During exercise peak we also observe LAFB, consequently we have left bifascicular block (LAFB + LSFB).