

**Name:** FSS  
**Weight:** 83Kg  
**Date:** 11/02/2008

**Sex:** M  
**Height:** 1,68m  
Time: 5:50PM

**Age:** 53yo.  
chest pain 3 hours onset

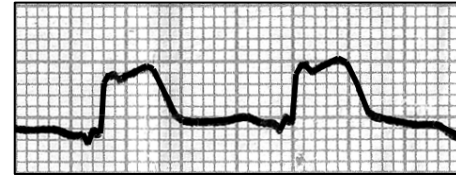
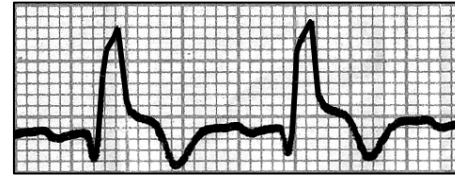
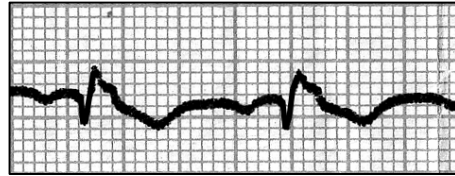
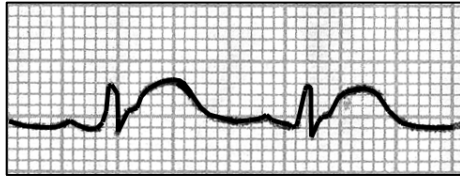
**Race:** Caucasian

DI

aVR

V1

V4

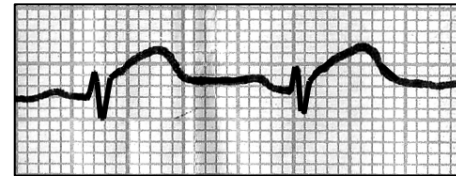
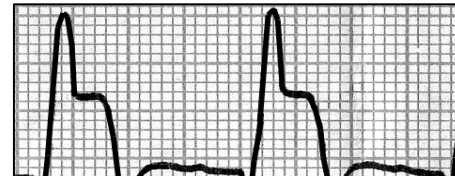
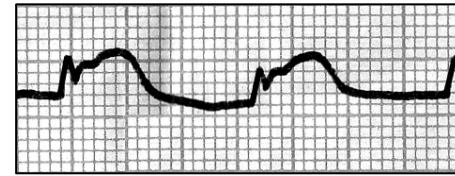
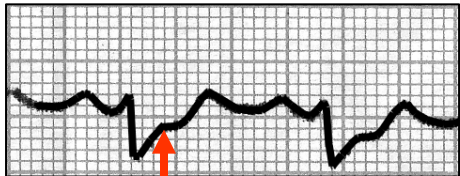


DII

aVL

V2

V5



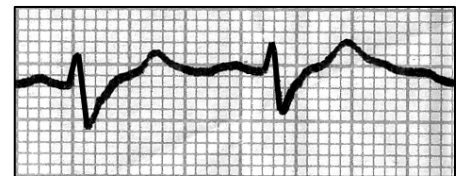
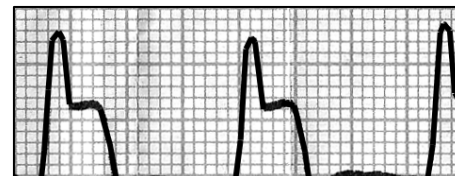
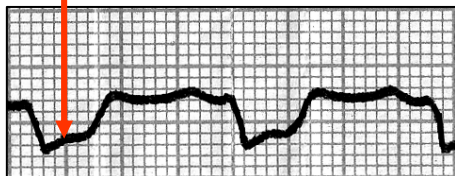
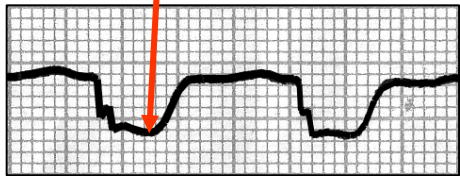
**MIRROR IMAGE IN INFERIOR LEADS**

DIII

aVF

V3

V6



Extensive transmural anterior myocardial infarction ( V1 to V6 + DI and aVL.) complicate with Complete RBBB.  
**Treatment:** Streptokinase intravenously within 4 hours 1,500,000 IU within 60 min.

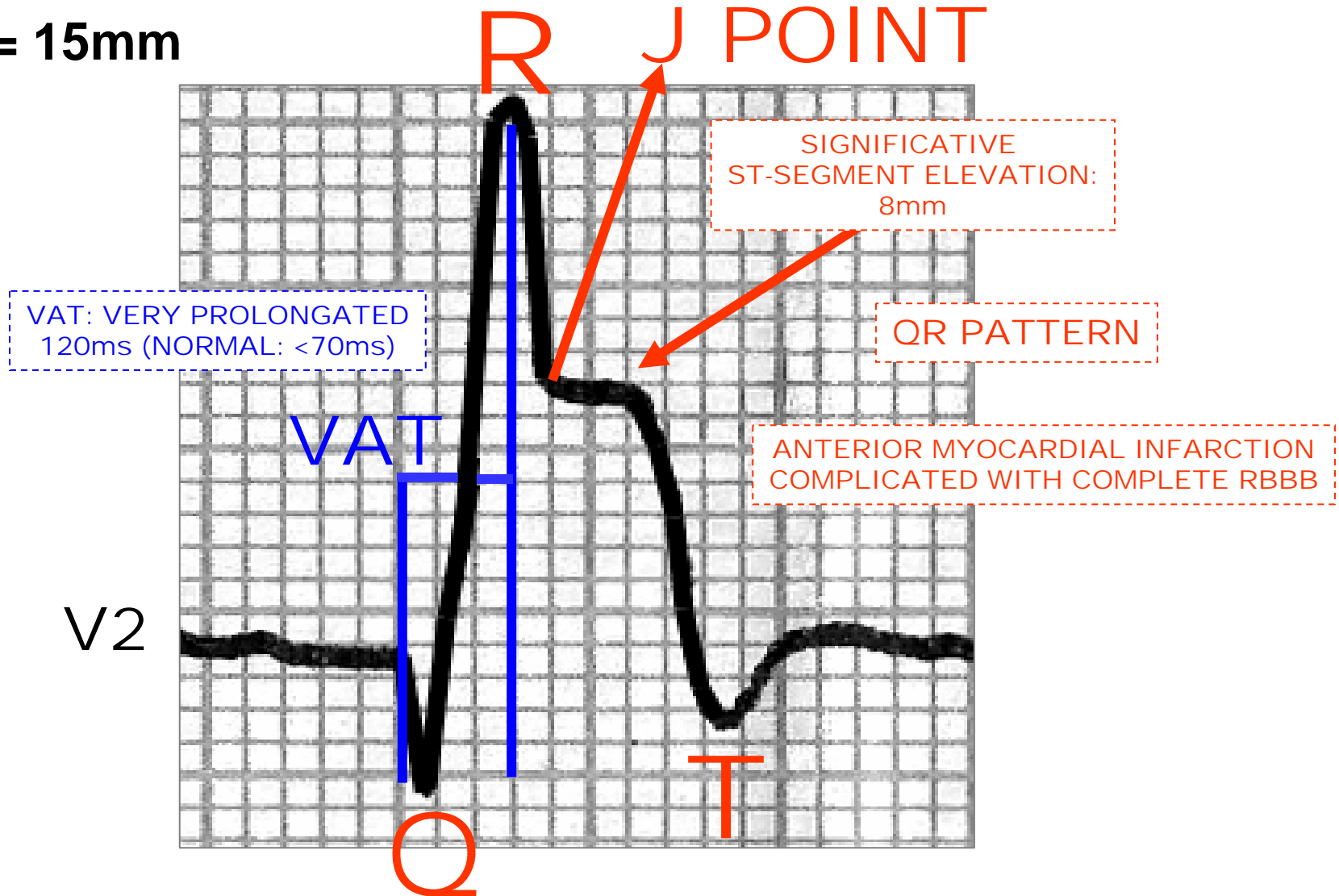
Name: FSS  
Weight: 83Kg  
Date: 11/02/2008

Sex: M  
Height: 1,68m  
Time: 5:50PM

Age: 53yo.  
chest pain 3 hours onset

Race: Caucasian

R = 15mm



VAT: VENTRICULAR ACTIVATION TIME

# POSSIBLE CAUSES OF QR/qR PATTERN IN RIGHT PRECORDIAL LEADS

- 1) Severe Right Ventricular Enlargement<sup>1</sup> (Supra-systemic Intraventricular pressure inside right ventricle)
- 2) Right Atrial Enlargement: qR pattern in V<sub>1</sub> may be an indirect sign of RAE
- 3) Complete RBBB complicated with anterior Myocardial Infarction<sup>2;3</sup>.
- 4) Ebstein's anomaly: bizarre and low voltage RBBB with initial q wave<sup>4</sup>.

## References

- 1) Gandhi MJ, Dattey KK, Kulkarni TP, Hansoti RC. Genesis of qR pattern in right precordial leads in right ventricular overload. J Assoc Physicians India. 1962; 10: 217-223.
- 2) Sodi-Pallares D, Bisteni A, Herrmann GR. Some views on the significance of qR and QR type complexes in right precordial leads in the absence of myocardial infarction.
- 3) Rudiakov Lal. On The Diagnostic Significance Of The qR Type QRS Complex In Right Electrocardiographic Leads. Kardiologija. 1964; 18: 72-73.
- 4) Kumar AE, Fyler DC, Miettinen OS, Nadas AS. Ebstein's anomaly. Clinical profile and natural history. Am J Cardiol. 1971; 28: 84-95.

# POSSIBLE CAUSES OF QR/qR PATTERN IN RIGHT PRECORDIAL LEADS

- 1) Congenitally Corrected Transposition: Secondary to inversion of septan activation, RAE, by progressive tricuspid regurgitation that occurs with age and associated with deterioration of RV function<sup>5;6</sup>
- 2) Endomyocardiofibrosis<sup>7</sup>
- 3) Anterior MI or ischemia / injury associated with LSFB. S-T elevation and increase in R-wave voltage “giant R waves” also displayed concomitant shift of the frontal QRS axis toward the locus of injury<sup>8;9;10;11;12;13;14;15;16</sup>.

## References

- 5) Warnes CA. Transposition of the great arteries. *Circulation* 2006; 114: 2699-2709.
- 6) Ruttenberg HD, Elliott LP, Anderson RC, Adams P Jr, Tuna N. Congenital corrected transposition of the great vessels. Correlation of electrocardiograms and vector cardiograms with associated cardiac malformations and hemodynamic states. *Am J Cardiol.* 1966; 17: 339-354.
- 7) Tobias NM, Moffa PJ, Pastore CA, Barretto AC, Mady C, Arteaga E, Bellotti G, Pileggi F. The electrocardiogram in endomyocardial fibrosis *Arq Bras Cardiol.* 1992; 59: 249-253.
- 8) David D, Naito M, Michelson E, et al. Intramyocardial conduction: a major determinant of R wave amplitude during acute myocardial ischemia. *Circulation* 1982; 65:161-166.
- 9) Deanfield JE, Davies G, Mongiardi F, et al. Factors influencing R wave amplitude in patients with ischemic heart disease. *Br Hear J* 1983; 49:8-12.
- 10) Schick EC Jr, Weiner DA, Hood WB Jr, Ryan TJ. Increase in R-wave amplitude during transient epicardial injury (Prinzmetal type). *J Electrocardiol.* 1980;13:259-266.
- 11) Feldman T, Chua KG, Childers RW. R wave of the surface and intracoronary electrogram during acute coronary arterial occlusion. *Am J Cardiol* 1986; 58: 885-900.
- 12) Hassapoyannes CA, Nelson WP. Myocardial ischemia-induced transient anterior conduction delay. *Am Heart J* 1991; 67:659-660.
- 13) Madias JE. The “giant R waves” ECG pattern of hyperacute phase of myocardial infarction. *J Electrocardiol* 1993;26:77-80.
- 14) Moffa PJ, Pastore CA, Sanches PCR et al. The left-middle (septal) fascicular block and coronary heart disease. In Liebman J, ed. *Electrocardiology'96—From the cell to body surface.* Cleveland, Ohio, Word Scientific, 1996;547-550.
- 15) Moffa PJ, Ferreira BM, Sanches PC, Tobias NM, Pastore CA, Bellotti G. Intermittent antero-medial divisional block in patients with coronary disease *Arq Bras Cardiol* 1997; 68:293-296
- 16) Uchida AH, Moffa PJ, Riera AR, Ferreira BM. Exercise-induced left septal fascicular block: an expression of severe myocardial ischemia. *Indian Pacing Electrophysiol J.* 2006;6:135-138

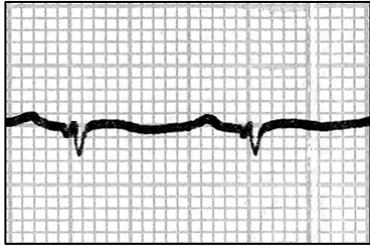
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**Sex:** M  
**Height:** 1,68m

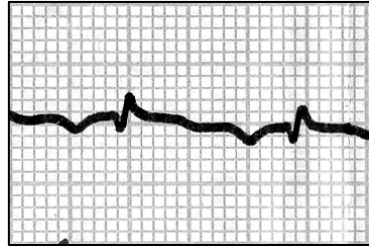
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**Race:** Caucasian  
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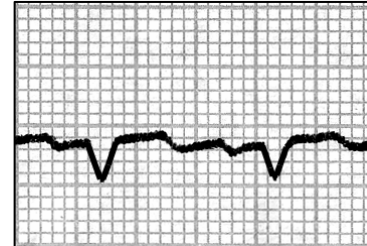
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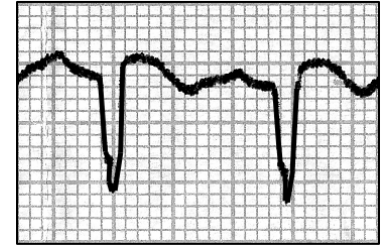
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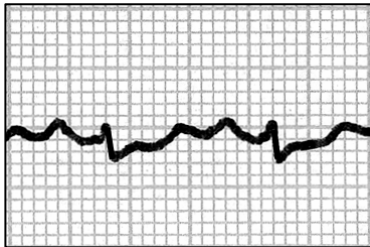
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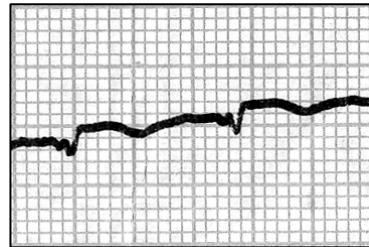
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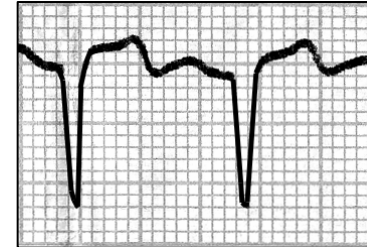
DII



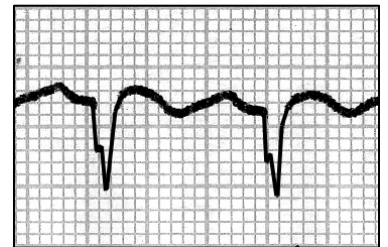
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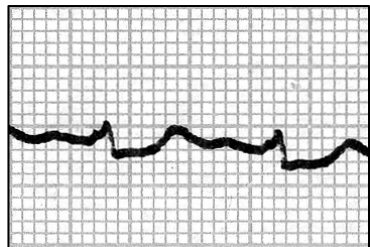
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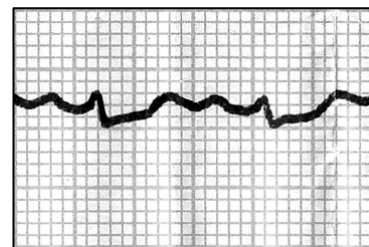
V5



DIII



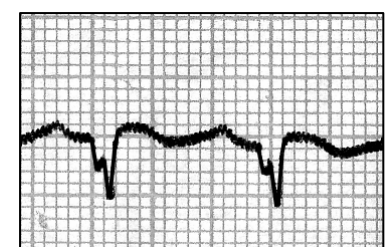
aVF



V3



V6



**ECG 18 hours later:** Thrombolytic therapy without success. Extensive transmural anterior myocardial infarction (  $V_1$  to  $V_6$  + DI and aVL.). Low QRS voltage on frontal plane. Absence of complete RBBB pattern or other dromotropic disorder.

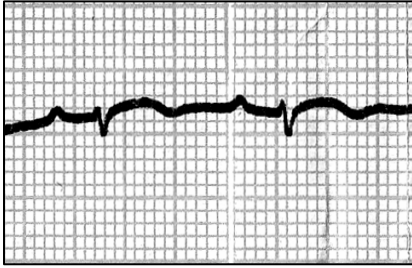
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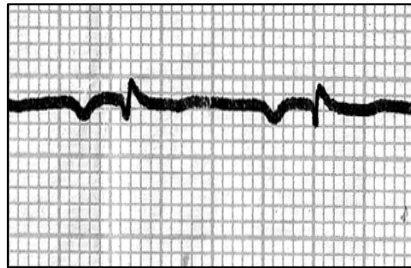
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**Race:** Caucasian

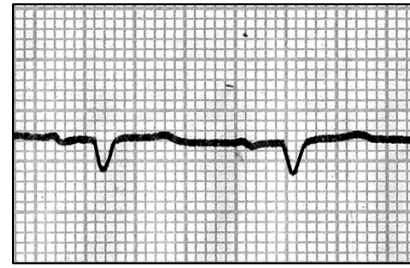
DI



aVR



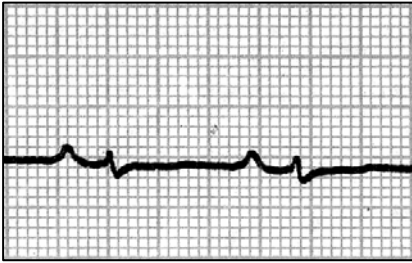
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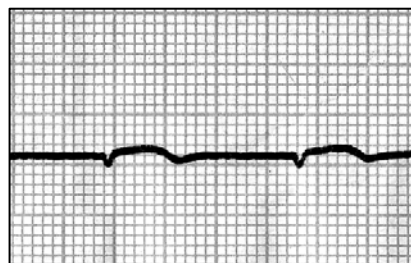
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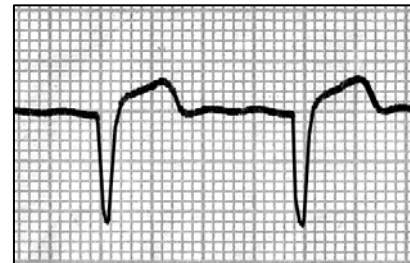
DII



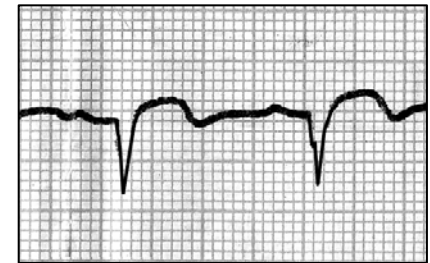
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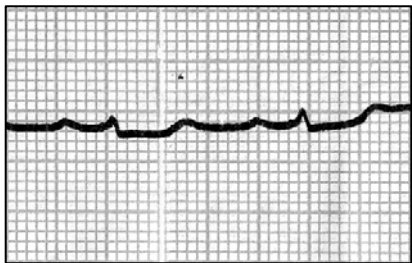
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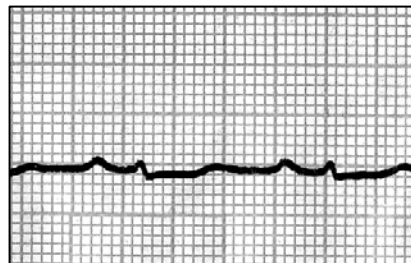
V5



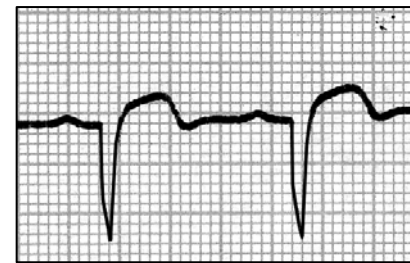
DIII



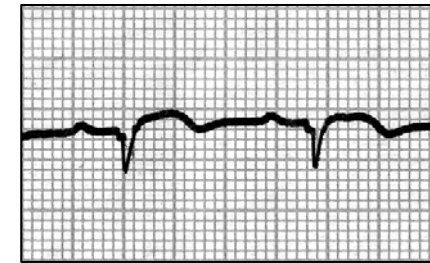
aVF



V3



V6



**ECG 10 days later:** Thrombolytic therapy. Extensive transmural anterior myocardial infarction. Low QRS voltage on frontal plane

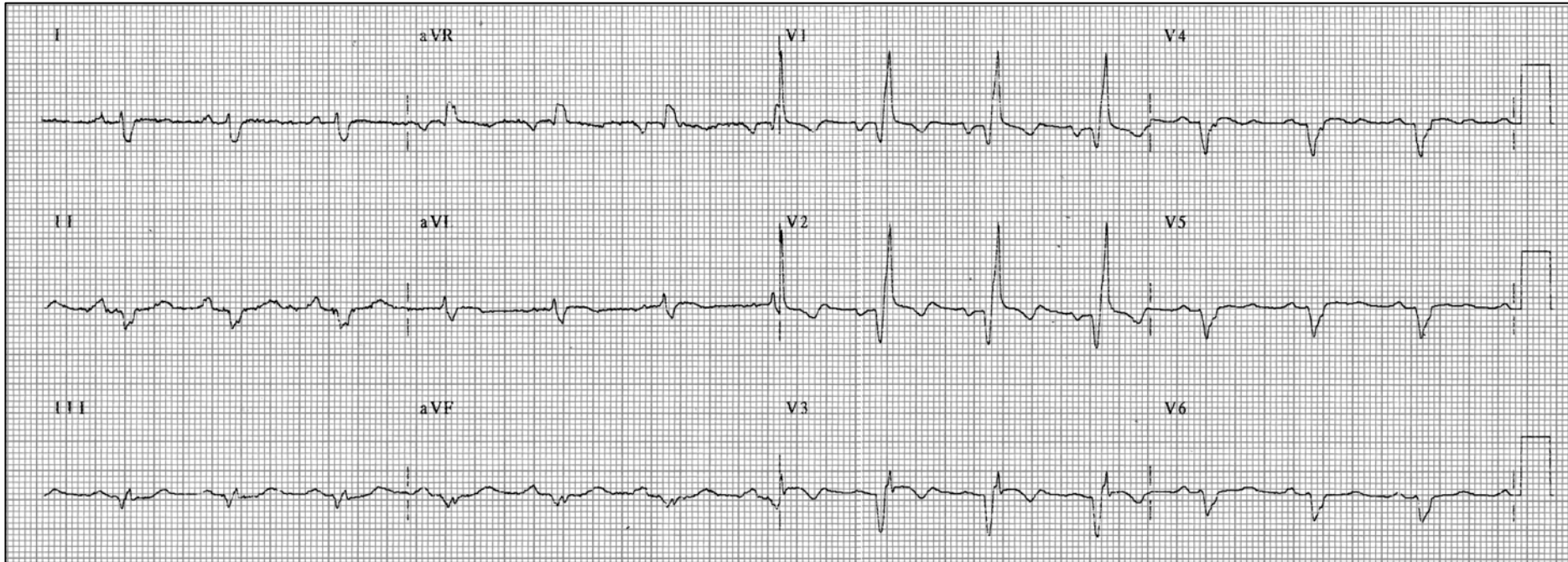
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**Sex:** M  
**Height:** 1,68m

**Age:** 53yo.  
**Date:** 16/04/2008

**Race:** Caucasian  
**Time:** 08:16

**Medications in use:** Carvedilol 25mg 2 times/day + Enalapril 20mg + Furosemide 40mg + Spironolactone 25mg + Sinvastatin 20mg + Aspirin 100mg.



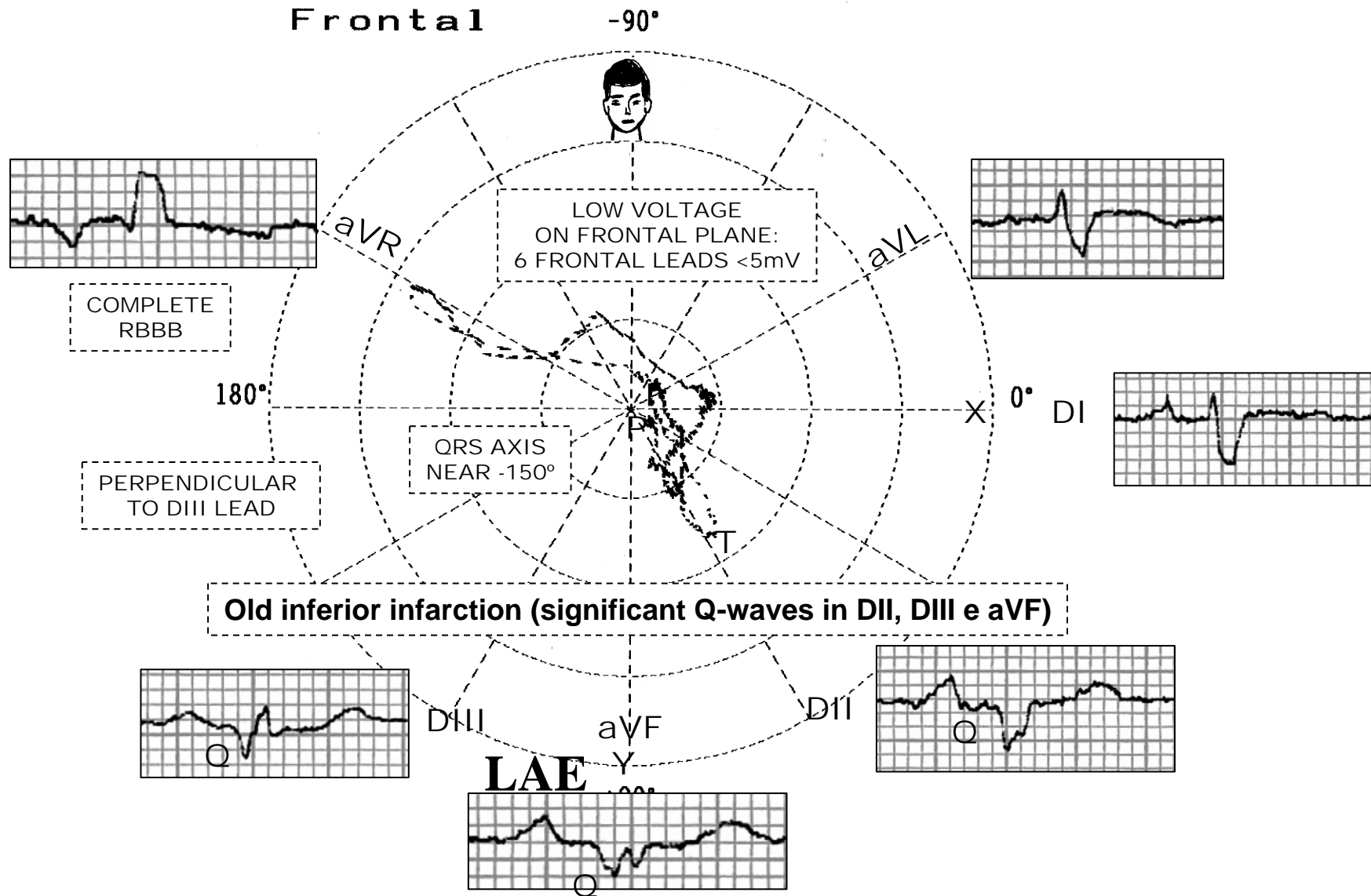
**ECG diagnosis** Sinus rhythm, HR: 81bpm, P axis  $+60^\circ$ , P wave: duration 120ms, prominent negative final component in lead  $V_1$ : Left Atrial Enlargement (LAE).

PR interval: Normal 181ms.

QRS axis  $-150^\circ$ , (right axis deviation), QRSd: 129ms, low voltage in frontal leads, old inferior myocardial infarction (significant Q-waves in DII, DIII and aVF), extensive anterior myocardial infarct associated with complete RBBB? (qR pattern from  $V_1$  to  $V_3$ ), QTc: 491ms.

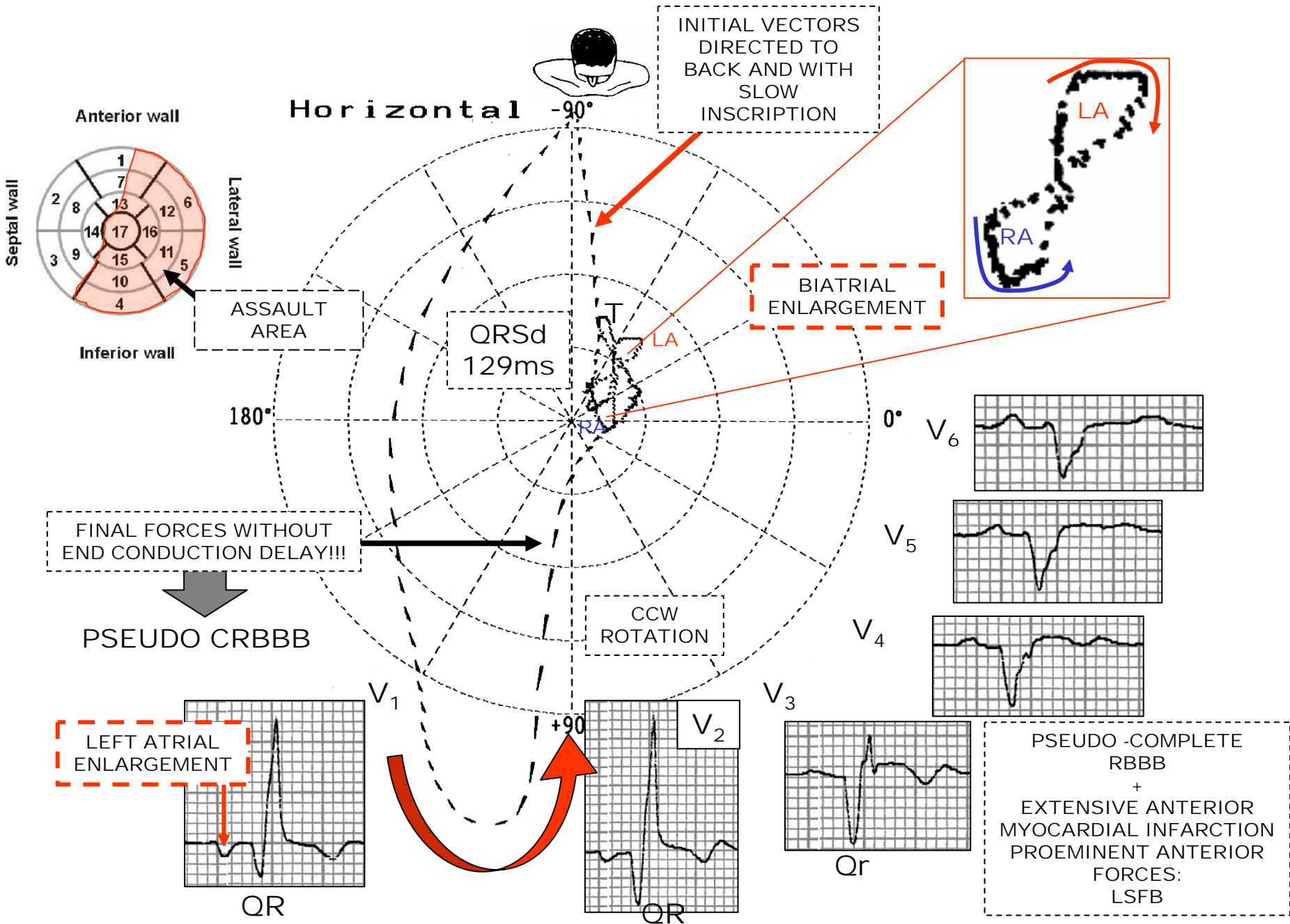
Prominent Anterior Forces (PAF): R waves with great voltage and sharp-pointed in  $V_2$ , progressive decrease of R wave voltage from  $V_4$  to  $V_6$ , absence of initial q wave in  $V_5$ - $V_6$ .: Left Septal Fascicular Block (LSFB).

# ECG/VCG FRONTAL PLANE CORRELATION

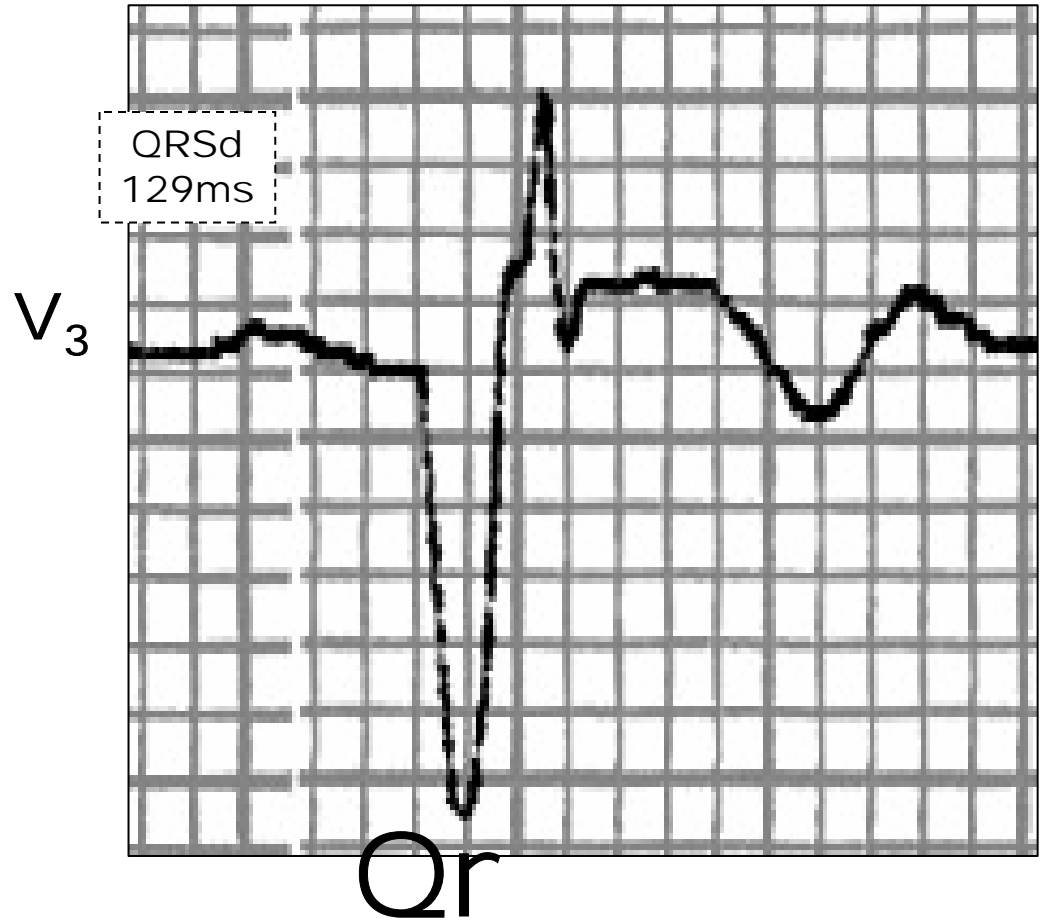
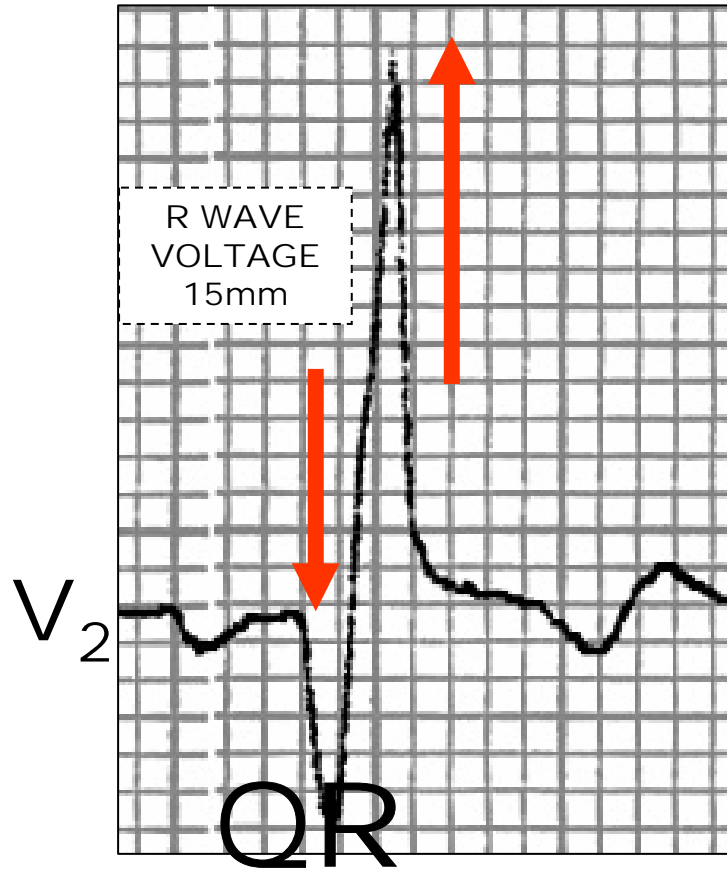




# ECG/VCG HORIZONTAL PLANE CORRELATION

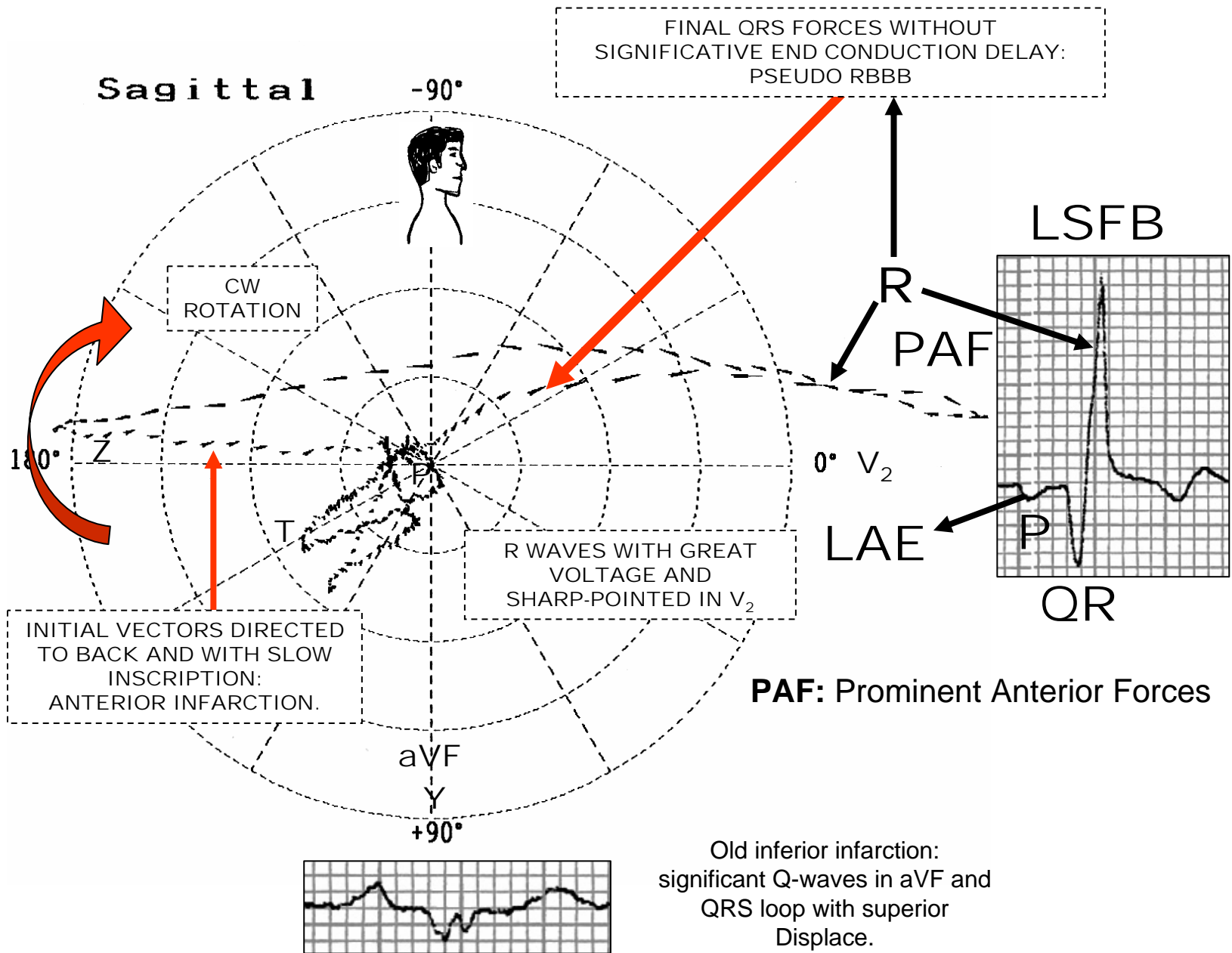


$$R-V_2 > R V_3$$



R waves with great voltage and sharp-pointed in V<sub>2</sub> (PAF)  
 Intrinsicoid deflection in V<sub>2</sub> > 50% of total QRSd and final forces without delay: Pseudo Complete RBBB  
 Progressive decrease of R wave voltage from V<sub>4</sub> to V<sub>6</sub>  
 Absence of initial q wave in V<sub>5</sub>-V<sub>6</sub>: Left Septal Fascicular Block.

# RIGHT SAGITTAL PLANE ECG/VCG CORRELATION



**PAF: Prominent Anterior Forces**

# FINAL CONCLUSIONS

- 1) BIATRIAL ENLARGEMENT: **ONLY VCG**
- 2) EXTENSIVE ANTERIOR MYOCARDIAL INFARCTION
- 3) OLD INFERIOR MYOCARDIAL INFARCTION
- 4) PAF: SECONDARY TO LSFB WITHOUT COMPLETE RBBB: **ONLY VCG**
- 5) ABSENCE OF COMPLETE RBBB: **ONLY VCG**

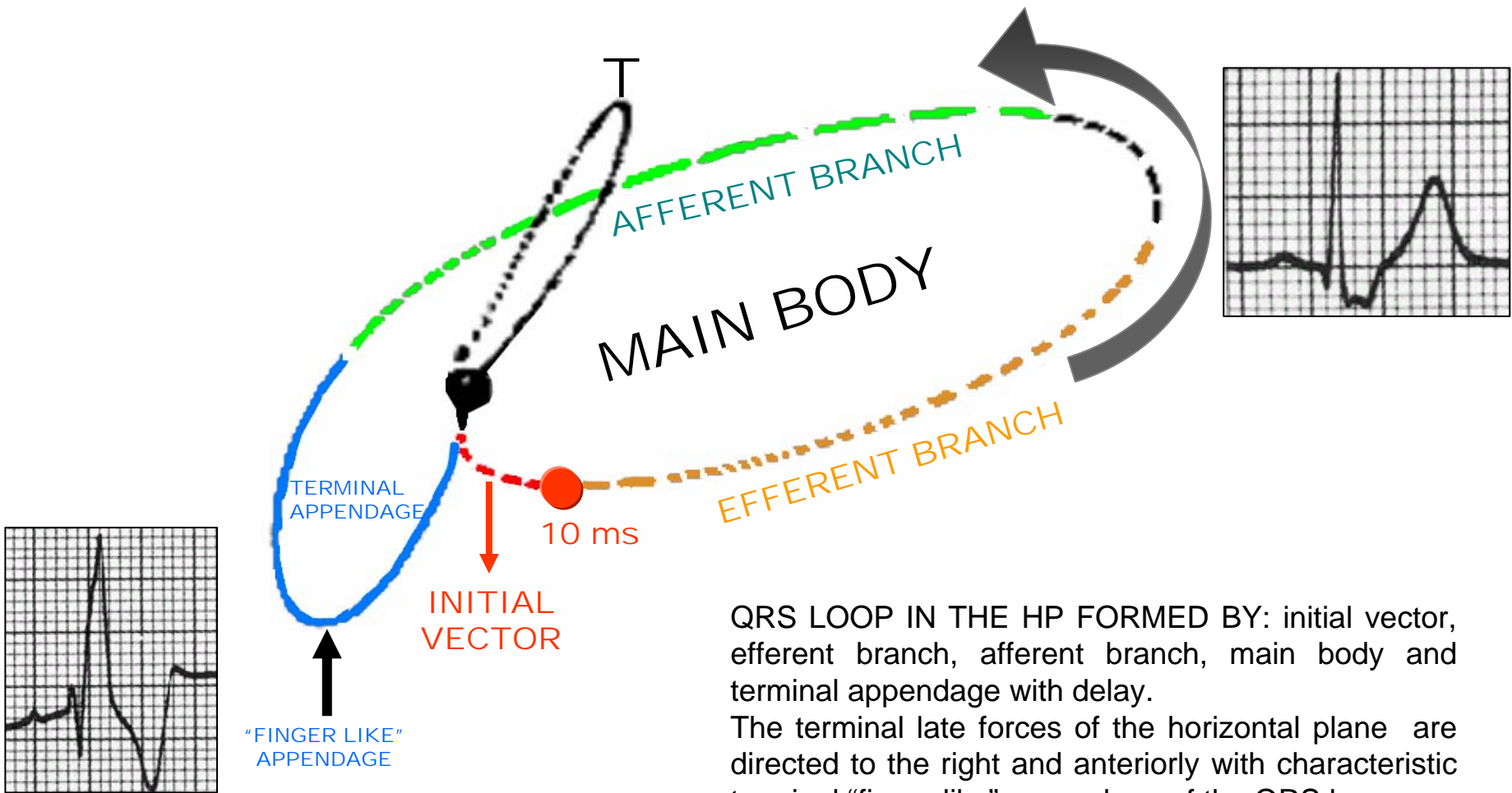
**COMMENTARIES:** IN THIS CASE VCG IS SUPERIOR TO ECG FOR THE APPROPRIATE DIAGNOSIS.

THEORICAL EXPLANATIONS IN NEXT SLIDE

# THEORETICAL EXPLANATIONS

- The coexisting RBBB and MI are individually recognizable in the VCG and ECG because the electrical effects of two conditions appear at different times in the QRS interval. The vector loop of RBBB, therefore, can be divided into an initial portion representing the activation of the left ventricle (LV) and a terminal portion representing activation the right ventricle (RV). Since most infarctions involve the LV and produce changes during the initial portion of the QRS complex/loop, their recognition is not hampered (with exception of lateral infarction: In the near past named strictly dorsal).
- In truly complete RBBB associated with anterior MI the terminal late forces of the horizontal plane are directed to the right and anteriorly with characteristic terminal “finger like” appendage of the QRS loop, whose average orientation is along the  $+120^\circ$  (between  $+140^\circ$  to  $+100^\circ$ ) axis of the horizontal reference frame which is writing slowly: **A CONDUCTION DELAY REPRESENTED BY THE CLOSE SPACING OF THE TIME DASHES IN THE TERMINAL PART OF THE QRS LOOP**. This late final forces are correspondent to the activation of basal wall of RV and /or septum.

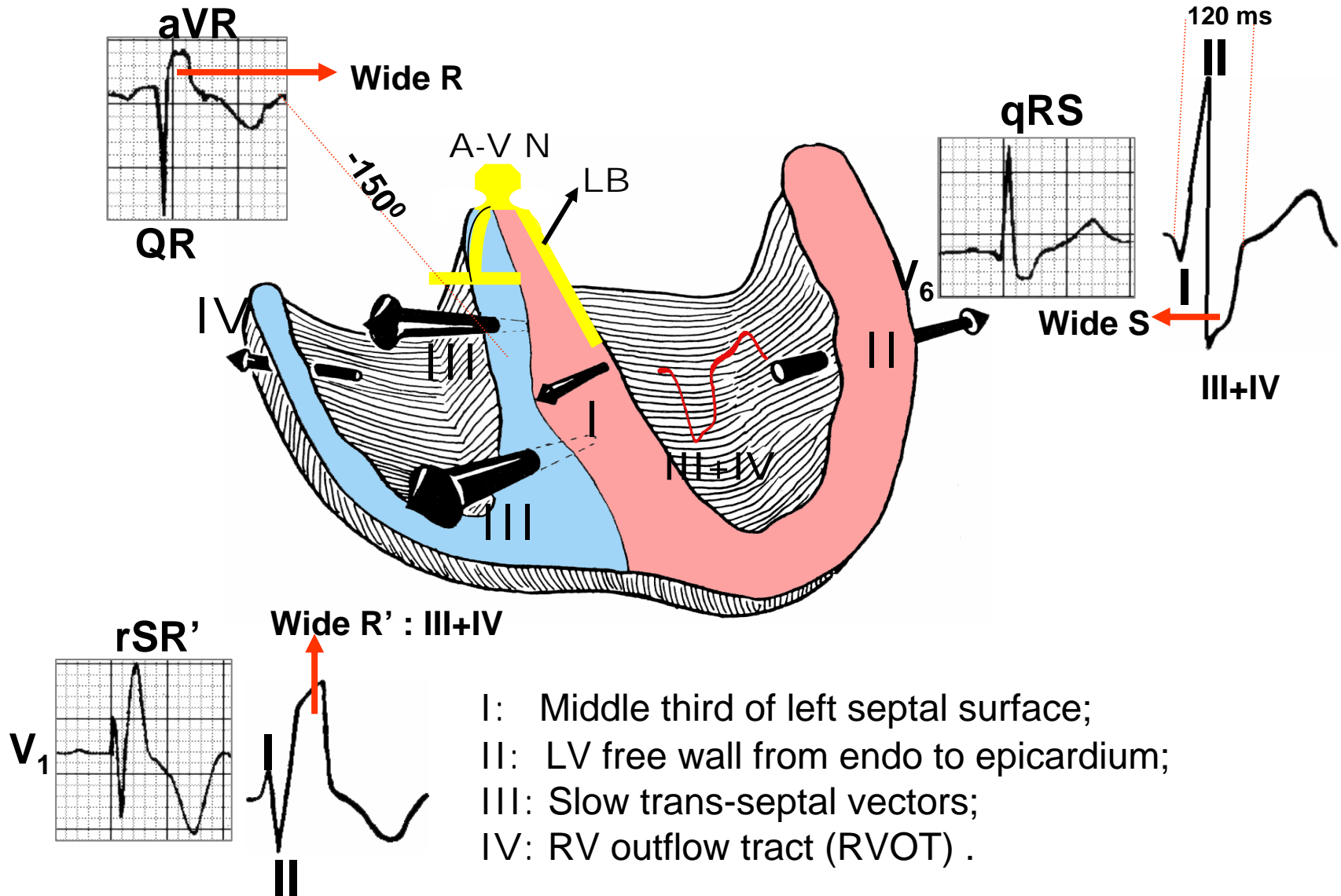
# VCG CRITERIA OF UNCOMPLICATED COMPLETE RBBB



QRS LOOP IN THE HP FORMED BY: initial vector, efferent branch, afferent branch, main body and terminal appendage with delay.

The terminal late forces of the horizontal plane are directed to the right and anteriorly with characteristic terminal "finger like" appendage of the QRS loop,

# UNCOMPLICATED COMPLETE RBBB VENTRICULAR ACTIVATION



# COMPLETE RBBB COMPLICATED WITH EXTENSIVE ANTERIOR MYOCARDIAL INFARCTION

